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This manual is designed according to syllabus of biology for students of Medical faculty. It will be a guide to action for students during their practical work.

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UNIT 5. ANIMAL PARASITES. INTRODUCTION

Animal parasites belong to:

1. Parasitic protozoa (e.g. Malaria).
2. Flat worms (e.g. Schistosoma).
3. Round worms (e.g. Ancylostoma).
4. Parasitic arthropods, mainly insects (e.g. mosquitoes and tsetse flies).

Four groups of animals are of major importance in medical parasitology:

- 1) Protozoa,
- 2) Helminths,
- 3) Arthropods,
- 4) Molluscs.

These organisms are classified according to the rules of zoological Nomenclature (International Commission on Zoological Nomenclature).

Types of parasites:

1. Obligatory parasites are those parasites that cannot exist without a host e.g. Malaria.
2. Facultative parasites are those parasites that are able to exist in soil and water independently of their host, when the environmental conditions are suitable leading a free living life e.g. *Strongyloides stercoralis*.
3. Accidental parasites are those parasites which enter accidentally and can live in a host different from their normal one e.g. *Dipylidium caninum*, *Hymenolepis dimenuta*.
4. Temporary parasite is an occasional parasite, it only visits its host for blood meals (e.g. blood-sucking insect as mosquitoes, sand flies and tsetse flies).
5. Periodic parasite passes a definite part of its life cycle as a parasite (e.g. *Cordylobia hominis*, *Hypoderma bovis*).
6. Specific parasite occurs in a particular host, i.e. there is a specificity in the host parasite relationship (e.g. *Trichinella spiralis* and *Taenia solium* are specific for pigs).

Parasites can also be divided according to their habitat into endoparasites which live inside their host e.g. *Ancylostoma* worms, and ectoparasites which are found attached to the skin of their host or its superficial tissue e.g. *Pediculus*.

Types of hosts:

1. Definitive host: Is the host in which the adult stage of the parasite lives or in which sexual reproduction takes place e.g. man is definitive host for *Ascaris*, *Anopheles pharoensis* for *Plasmodium* sp. parasites and pigs for *Taenia solium*.

2. Intermediate host: Harbours the larval (immature stages) or asexual stages of a parasite (e.g. *Culex* mosquito is an intermediate host for *Wuchereria bancrofti*, *Pirenella conica* is the intermediate host for *Heterophyes heterophyes*, *Biomphalaria* sp. for intestinal Bilaharziasis ... etc.).

However the parasite may pass its larval stages in two intermediate hosts;

a) first (Primary) intermediate host which harbours the first immature stages of the parasite (e.g. *Prinella conica* for *Heterophyes*);

b) while the second (Transporting) intermediate host harbours the second immature stages of the same parasite, after leaving the first intermediate host.

This second host carries the parasite to man (e.g. *Mugil cephalus* and *Tilapia nilotica*).

Vector is a host that transmits parasites from one host to another. Vectors are usually arthropods e.g. *Anopheles* mosquitoes are vectors of malaria. They transmit the disease from one man to another through their bite.

- A mechanical vector: If the transmitter is not essential in the life cycle.
- A biological vector: If the transmitter is essential in the life cycle.

Points of practical value when studying parasitology:

I. Mode of infection by the parasite

Mode of infection: means the portal of entry of the parasite into the body and this may occur from one or more of the following routes of entry:

1) Infection by mouth, through ingested contaminated water or food:

a. Infection by drinking water containing the infective stage, examples: Intestinal amoebas and flagellates, and cercaria of schistosomes through swimming or contact contaminated water.

b. Infection by ingesting food containing the mature egg or larval stage- examples: *Ascaris lumbricoides*, *Trichuris trichiura*, *Enterobius vermicularis*, *Taenia solium*, *T. saginata*, *Trichinella spiralis*, *Diphyllobothrium latum*, intestinal flukes, and liver flukes.

2) Zoonosis is the term given to the disease of animals which are transmissible to man ex: arthropods: blood sucking arthropods transmit filaria.

II. Portal of exit

For the continuation of the life cycle of the parasite it must have a portal of exit from its host and this can occur via:

- 1) Faeces: as eggs of most Helminthes and cysts of intestinal protozoa.
- 2) Urine: as eggs of *Schistosoma haematobium*.
- 3) Sputum: as eggs of lung flukes.
- 4) Blood: as Malaria and Trypanosomes.
- 5) Genital tract: as *Trichomonas vaginalis trophozoites*.

III. The life cycle of the parasite

In other words its route of migration inside the human or the insect body is important. Some parasites, undergo a certain cycle inside the human body as *Ancylostoma*, *Strongyloides* and *Ascaris*, while other restrict their development in the intestine as *Enterobius* and *Trichuris*. In the insect host the parasite may undergo a cycle in the insect body, e.g. *Malaria* and *Wuchereria* and others do not as those which are transmitted by direct or indirect mechanical means.

IV. The infective stage

This may be can

- egg as *Enterobius*, *Ascaris*, *Trichuris*, *Hymenolepis*,
- a larva as *Ancylostoma*,
- a cercaria as *Schistosoma*,
- a cyst as *Entamoeba histolytica* and intestinal protozoa or
- a cysticercus as *C. Celluosae* and *C. bovis*.

V. Pathogenesis of parasitic infection

The way parasites damage their hosts occurs through different mechanisms including the following:

1. Mechanical: the parasite may obstruct a normal passage e.g. *Ascaris lumbricoides* may cause intestinal obstruction or bile duct obstruction, *Enterobius vermicularis* may cause appendicitis.

2. Traumatic: when the parasite invades the skin as in scabies or myiasis. Internal damage can also occur as in hookworms, which attach themselves by their buccal capsule to the intestinal mucosa producing ulcers.

3. Toxic: circulation of certain toxic byproducts of parasites produces generalized manifestations as in hookworms producing butterfly pigmentation of the face. Also, in *Hymenolepis nana* and *Ascaris lumbricoides* infection nervous manifestations appear. Scorpion stings produce severe toxicity in man.

4. Necrosis: enzymes elaborated by the parasite produce necrosis of tissue as in *Entamoeba histolytica*.

5. Stimulation of the host immune response: parasitic antigens stimulate both a cellular and humoral immune response provoking tissue reactions consisting of cellular proliferation and infiltration at the site of parasite antigens, or deposition of circulating immune complex in the tissues e.g. *Schistosoma granuloma* and *Plasmodium malaria* nephrosis.

6. Cellular destruction: destruction of red blood corpuscles occurs in malaria, reticuloendothelial cells in *Leishmania donovani* and other tissue cells in *Trypanosoma cruzi*.

7. Allergic manifestations: allergic reactions occur with insect bites.

8. Neoplastic formation: parasitic infections may contribute to tumour formation. *Schistosoma haematobium* can cause cancer bladder.

VI. The symptomatology

The symptomatology of the disease usually runs parallel with the pathological changes caused by the invading parasite.

VII. Diagnosis of parasitic infections

The diagnosis of parasitic infections has two methods of approach. Clinical and laboratory.

1. Clinical diagnosis:

Depends on the characteristic signs and symptoms related to the parasitic infection, e.g. Nocturnal perianal itching is suggestive of infection with *Enterobius vermicularis*.

2. Laboratory diagnosis can be achieved by:

a) Direct methods: which can detect the diagnostic stages of the parasite by microscopical examination of the excreta, blood, tissues or smears. Culture and animal inoculations can help in diagnosis of some parasitic infections.

d) Indirect methods: These methods on the detection of antigens or antibodies in the patients' serum. Indirect methods of diagnosis are mainly resorted to when parasites are present in tissues, e.g. *Toxoplasma gondii*, or in cases of closed chronic infection e.g. *Schistosoma mansoni* when no eggs can be detected in the faeces.

c) Molecular biological methods: These include DNA probes and the polymerase chain reaction (PCR).

VIII. The therapy and control measures

Concerning the therapy and control measures, here it may be said that an ideal drug is that which kills the parasite properly within the limits of tolerance of the patient. It then follow, that adequate information about the specific drug, its toxicity and contra-indications must be always borne in mind.

Concerning the methods of control or eradication of the parasite within a community, here one has to follow two ways:

1. The first: is that which kills the parasite in man by internal medication.

2. The other way: is that which attacks the parasite in the arthropod hosts or the reservoir. We have to know every detail about the bionomics of the insect host, and the surrounding climatic conditions so as to reach to a proper and adequate control, e.g. Malaria control.

IX. Prognosis

Lastly prognosis of the disease or its accurate history, paves the way in front of the physician to estimate with considerable accuracy the probability of rapid or prolonged recovery or fatal end.

Testing your knowledge
GENERAL CHARACTERISTICS OF THE RELATIONSHIP
BETWEEN PARASITE AND HOST

Task 1. Fill in the gaps in the sentences

1. Parasites that cannot exist without a host are called _____.
2. Parasites that are able to exist in soil and water, regardless of the host under favorable environmental conditions are called _____.
3. Parasites which enter accidentally and can live in a host different from their normal one are called _____.
4. Parasite which only visits its host for food is called _____.
5. Parasite which passes only a definite part of its life cycle as a parasite is called _____.
6. Parasites which are found attached to the skin of their host or its superficial tissue are called _____.
7. The organism, in which the adult stage of the parasite lives or where sexual reproduction takes place is called _____.
8. The organism, in which occur the larval stage of the parasite and its asexual reproduction is called _____.
9. The host in which the parasite does not undergo any development but in which it remains alive in the larval stage and can be infective to another host is called _____.
10. The organism is able to accumulate a parasite as reservoir in addition to the main host is called _____.

Task 2. Properly connect the type of action of the parasite with the characteristic action

Pathogenesis of parasitic infection

Type of parasite action	Characteristics of the parasite action
Mechanical	Destruction of red blood corpuscles occurs in malaria, reticuloendothelial cells in <i>Leishmania donovani</i> and other tissue cells in <i>Trypanosoma cruzi</i>
Traumatic	Allergic reactions occur with insect bites
Toxic	The parasite may obstruct a normal passage e.g. <i>Ascaris lumbricoides</i> may cause intestinal obstruction or bile duct obstruction, <i>Enterobius vermicularis</i> may cause appendicitis
Necrosis	Parasitic infections may contributed to tumour formation. <i>Schistosoma haematobium</i> can cause cancer bladder
Stimulation of the host immune response	Parasitic antigens stimulate both a cellular and humoral immune response provoking tissue reactions consisting of cellular proliferation and infiltration at the site of parasite antigens, or deposition of circulating immune complex in the tissues
Cellular destruction	Enzymes elaborated by the parasite produce necrosis of tissue as in <i>Entamoeba histoytica</i>
Allergic manifestations	Circulation of certain toxic byproducts of parasites produces generalized manifestations as in hookworms producing butterfly pigmentation of the face
Neoplastic formation	When the parasite invades the skin as in scabies or myiasis. Internal damage can also occur as in hookworms, which attach themselves by their buccal capsule to the intestinal mucosa producing ulcers

UNIT 6. PROTISTS

6.1. Malaria

The causative agent of malaria is *Plasmodium falciparum*. The main host – the anopheles mosquito of genus *Anopheles*. Intermediate host – the man.

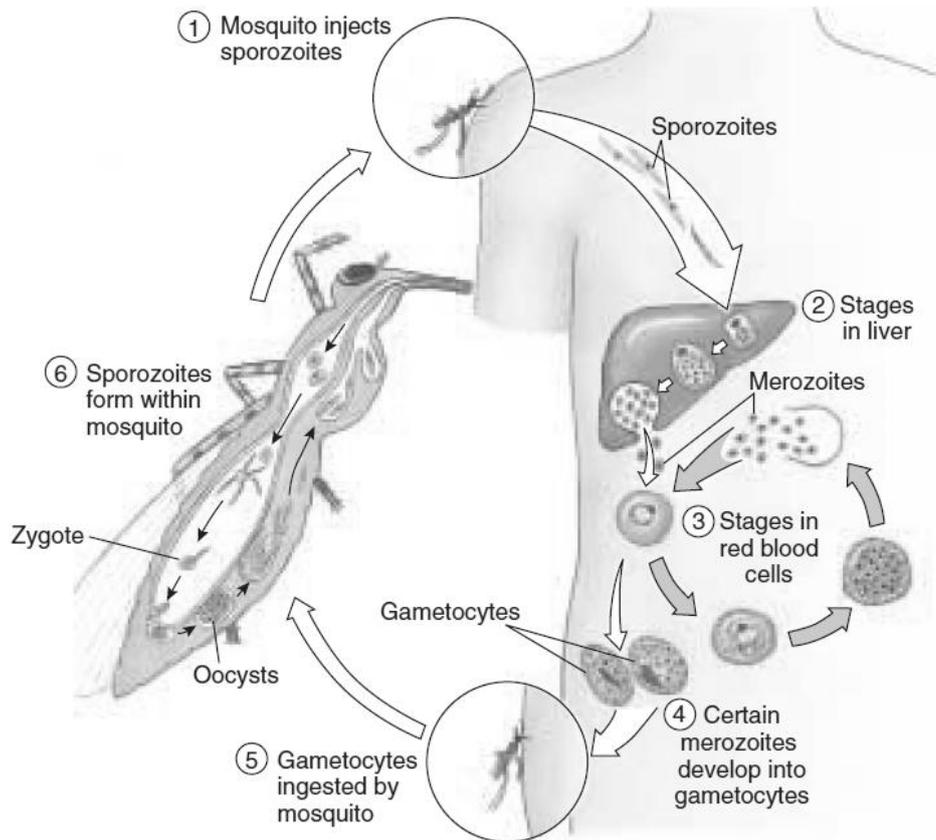
The life cycle of *Plasmodium falciparum*

Infection in humans begins with the bite of an infected female *Anopheles* mosquito. *Plasmodium* sporozoites released from the salivary glands of the mosquito enter the bloodstream during feeding, quickly invading liver cells (hepatocytes). The immune system clears the sporozoites from the circulation within 30 minutes.

Sporozoites invade liver cells and undergo schizogony to produce merozoites.

Merozoites invade circulating RBCs. Each merozoite produces as many as 36 new merozoites through schizogony in RBCs. Merozoites rupture RBCs to invade other RBCs. Simultaneous lysing of RBCs causes the sudden chills due to septicaemia because of release of haemozoin granules and very high fever typical of malaria.

The length of this erythrocytic stage depends on the parasite species: an irregular interval for *P. falciparum*, 48 hours for *P. vivax* and *P. ovale* and 72 hours for *P. malariae*.



The clinical manifestations of malaria, fever and chills, are associated with the synchronous rupture of the infected erythrocytes. The released merozoites invade additional erythrocytes. Not all of the merozoites divide into schizonts; some differentiate into sexual forms, male and female gametocytes. These gametocytes are taken up by a female *Anopheles* mosquito during a blood meal. Within the mosquito midgut, the male gametocyte undergoes a rapid nuclear division, producing eight flagellated microgametes that fertilize the female macrogamete. The resulting ookinete traverses the mosquito gut wall and encysts on the exterior of the gut wall as an oocyst. Oocyst forms Sporozoites by sporogony. Soon, the oocyst ruptures, releasing hundreds of sporozoites into the mosquito body cavity, where they eventually migrate to the mosquito salivary glands. The cycle is repeated again.

6.2. Amoebiasis

Amoeba sp. are primitive unicellular microorganisms with a relatively simple life cycle which can be divided into two stages:

- Trophozoite – actively motile feeding stage.
- Cyst – quiescent, resistant, infective stage.

Their reproduction is through binary fission, e.g. splitting of the trophozoite or through the development of numerous trophozoites within the mature multinucleated cyst.

Motility is accomplished by extension of pseudopodia (“false foot”).

Entamoeba histolytica

Morphological features

(a) Trophozoites. Viable trophozoites vary in size from about 10-60 μm in diameter. Motility is rapid, progressive, and unidirectional, through pseudopods. The nucleus is characterized by evenly arranged chromatin on the nuclear membrane and the presence of a small, compact, centrally located karyosome. The cytoplasm is usually described as finely granular with few ingested bacteria or debris in vacuoles. In the case of dysentery, however, RBCs may be visible in the cytoplasm, and this feature is diagnostic for *E. histolytica*.

(b) Cyst. Cysts range in size from 10-20 μm . The immature cyst has inclusions namely; glycogen mass and chromatoidal bars. As the cyst matures, the glycogen completely disappears; the chromatoidals may also be absent in the mature cyst.

Life cycle. Intestinal infections occur through the ingestion of a mature quadrinucleate infective cyst, contaminated food or drink and also by hand to mouth contact. It is then passed unaltered through the stomach, as the cyst wall is resistant to gastric juice. In terminal ileum (with alkaline pH), excystation takes place. Trophozoites being actively motile invade the tissues and ultimately lodge in the submucous layer of the large bowel. Here they grow and multiply by binary fission. Trophozoites are responsible for producing lesions in amoebiasis. Invasion of blood vessels leads to secondary extra intestinal lesions. Gradually the effect of the parasite on the host is toned down together with concomitant increase in host tolerance, making it difficult for the parasite to continue its life cycle in the trophozoite phase. A certain number of trophozoites

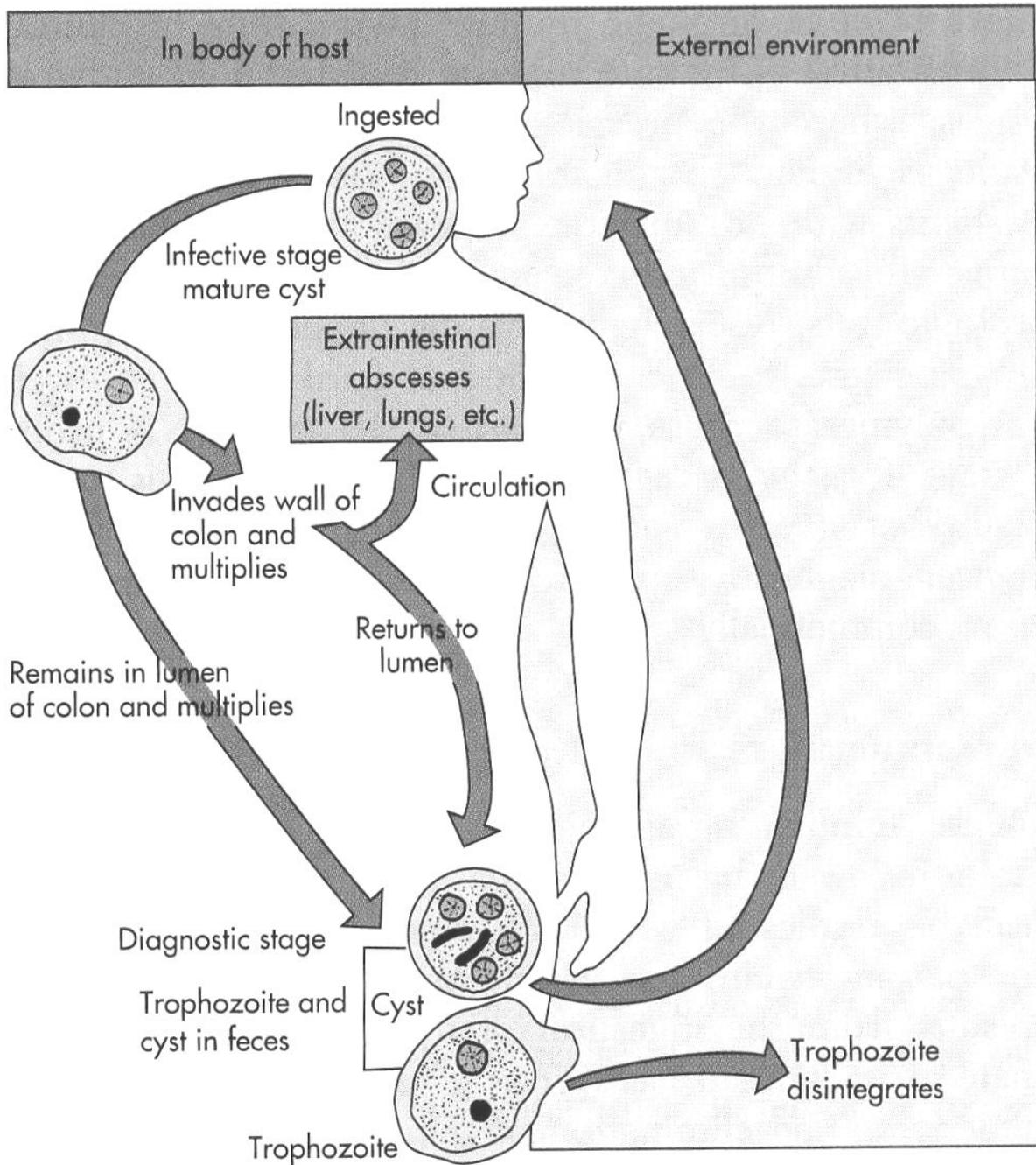
come from tissues into lumen of bowel and are first transformed into pre-cyst forms. Pre-cysts secrete a cyst wall and become a uninucleate cyst. Eventually, mature quadrinucleate cysts form. These are the infective forms. Both mature and immature cysts may be passed in faeces. Immature cysts can mature in external environments and become infective.

Pathogenesis. Trophozoites divide and produce extensive local necrosis in the large intestine. Invasion into the deeper mucosa with extension into the peritoneal cavity may occur. This can lead to secondary involvement of other organs, primarily the liver but also the lungs, brain, and heart. Extraintestinal amebiasis is associated with trophozoites. Amoebas multiply rapidly in an anaerobic environment, because the trophozoites are killed by ambient oxygen concentration.

Epidemiology. *E. histolytica* has a worldwide distribution. Although it is found in cold areas, the incidence is highest in tropical and subtropical regions that have poor sanitation and contaminated water. About 90% of infections are asymptomatic, and the remaining produces a spectrum of clinical syndrome. Patients infected with *E. histolytica* pass non-infectious trophozoites and infectious cysts in their stools. Therefore, the main source of water and food contamination is the symptomatic carrier who passes cysts. Symptomatic amebiasis is usually sporadic. The epidemic form is a result of direct person-to-person faecal-oral spread under conditions of poor personal hygiene.

Clinical features. The outcome of infection may result in a carrier state, intestinal amebiasis, or extraintestinal amebiasis. Diarrhoea, flatulence, and cramping are complaints of symptomatic patients. More severe disease is characterised by the passing of numerous bloody stools in a day. Systemic signs of infection (fever, leukocytosis, rigors) are present in patients with extraintestinal amebiasis. The liver is primarily involved, because trophozoites in the blood are removed from the blood by the portal veins. The right lobe is most commonly involved, thus pain over the liver with hepatomegaly and elevation of the diaphragm is observed.

Immunity. *E. histolytica* elicits both the humoral and cellular immune responses, but it is not yet clearly defined whether it modulates the initial infection or prevents reinfection.



Laboratory diagnosis. In intestinal amoebiasis:

- Examination of a fresh dysenteric faecal specimen or rectal scraping for trophozoite stage. (Motile amoebae containing red cells are diagnostic of amoebic dysentery).
- Examination of formed or semiformed faeces for cyst stage. (Cysts indicate infection with either a pathogenic *E. histolytica* or non-pathogenic *E. dispar*.)

Extraintestinal amoebiasis:

- Diagnosed by the use of scanning procedures for liver and other organs.
- Specific serologic tests, together with microscopic examination of the abscess material, can confirm the diagnosis.

Treatment. Acute, fulminating amebiasis is treated with metronidazole followed by iodoquinol, and asymptomatic carriage can be eradicated with iodoquinol, diloxanide furoate, or paromomycin. The cysticidal agents are commonly recommended for asymptomatic carriers who handle food for public use. Metronidazole, chloroquine, and diloxanide furoate can be used for the treatment of extra intestinal amoebiasis.

Prevention. Introduction of adequate sanitation measures and education about the routes of transmission.

Table 1. Difference between *E. histolytica* and *E. coli*

	Differentiating features	<i>E histolytica</i>	<i>E coli</i>
	Trophozoite		
1.	Size	18-40 µm	20-40 µm
2.	Motility	Actively motile	Sluggishly motile
3.	Cytoplasm	Clearly demarcated into endoplasm and ectoplasm	Endoplasm and ectoplasm are not clearly demarcated
4.	Cytoplasmic inclusions	Red blood cells (RBCs), leucocyte and tissue debris. No bacteria is seen.	Bacteria & tissue debris is seen but no Red blood cells (RBCs),or leucocytes are inclusions seen.
5.	Nucleus	Not visible in unstained preparation	Visible in unstained preparation
6.	Karyosome	Central	Eccentric
7.	Nuclear membrane	Delicate and is lined by fine chromatin	Thick and is lined by coarse chromatin
	Cyst		
8.	Size	6-15 µm	15-20 µm
9.	Nucleus	1-4,	1-8
10.	Karyosome	Central karyosome	Eccentric karyosome
11.	Chromatid bars	Rounded	Filamentous

6.3. Pathogenic flagellates

Flagellates are unicellular microorganisms. Their locomotion is by lashing a tail-like appendage called a flagellum or flagella and reproduction is by simple binary fission.

There are three groups of flagellates:

- Luminal flagellates:

Giardia lamblia

Dientmoeab fragilis

- Hemoflagellates:

Trypanosoma species.

Leishmania species.

- Genital flagellates:

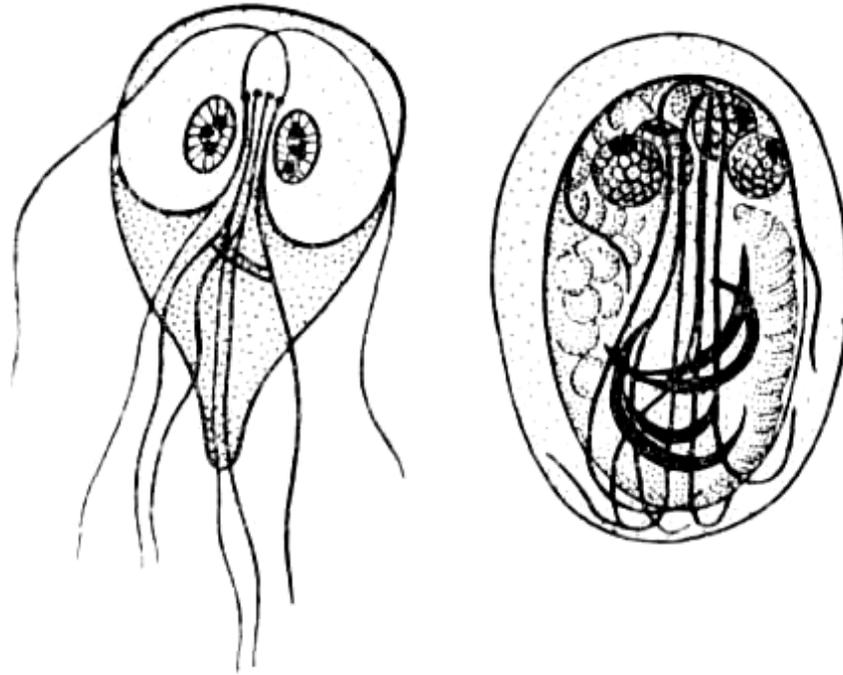
Trichomonas vaginalis

Giardia lamblia

Important features – the life cycle consists of two stages, the trophozoite and cyst. The trophozoite is 9-12 μm long and 5-15 μm wide anteriorly. It is bilaterally symmetrical, pear-shaped with two nuclei (large central karyosome), four pairs of flagella, two axonemes, and a suction disc with which it attaches to the intestinal wall. The oval cyst is 8-12 μm long and 7-10 μm wide, thick-walled with four nuclei and several internal fibers. Each cyst gives rise to two trophozoites during excystation in the intestinal tract. Transmission is by ingestion of the infective cyst.

Pathogenesis

Infection with *G. lamblia* is initiated by ingestion of cysts. Gastric acid stimulates excystation, with the release of trophozoites in duodenum and jejunum. The trophozoites can attach to the intestinal villi by the ventral sucking discs without penetration of the mucosa lining, but they only feed on the mucous secretions. In symptomatic patients, however, mucosa-lining irritation may cause increased mucous secretion and dehydration. Metastatic spread of disease beyond the GIT is very rare.



Giardia lamblia

Epidemiology

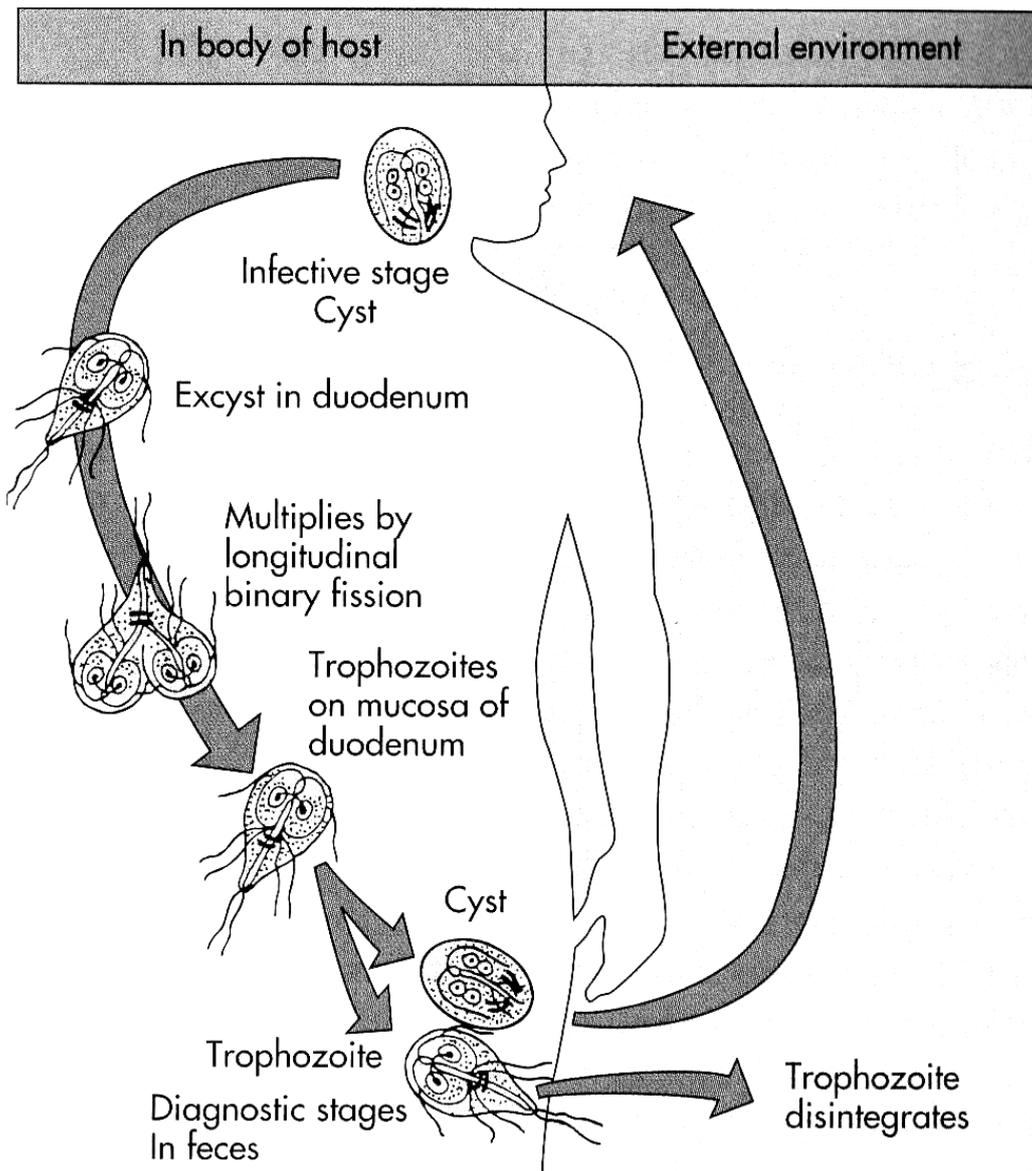
Giardia lamblia has a worldwide distribution, particularly common in the tropics and subtropics. It is acquired through the consumption of inadequately treated contaminated water, ingestion of contaminated uncooked vegetables or fruits, or person-to-person spread by the faecal-oral route. The cyst stage is resistant to chlorine in concentrations used in most water treatment facilities. Infection exists in 50% of symptomatic carriage, and reserves the infection in endemic form.

Clinical features

Clinical disease: Giardiasis. Symptomatic giardiasis ranges from mild diarrhea to severe malabsorption syndrome. Usually, the onset of the disease is sudden and consists of foul smelling, watery diarrhea, abdominal cramps, flatulence, and streatorrhoea. Blood & pus are rarely present in stool specimens, a feature consistent with the absence of tissue destruction

Immunity

The humoral immune response and the cellular immune mechanism are involved in giardiasis. Giardia – specific IgA is particularly important in both defense against and clearance of parasite.



Life cycle of *Giardia lamblia*

Laboratory diagnosis

Examination of diarrhoeal stool- trophozoite or cyst, or both may be recovered in wet reparation. In examinations of formed stool (e.g. in asymptomatic carriers) only cysts are seen. *Giardia* species may occur in “showers”, i.e. many organisms may be present in the stool on a given day and few or none may be detected the next day. Therefore one stool specimen per day for 3 days is important.

If microscopic examination of the stool is negative in a patient in whom giardiasis is highly suspected duodenal aspiration, string test (entero-test), or biopsy of the upper small intestine can be examined.

In addition to conventional microscopy, several immunologic tests can be implemented for the detection of parasitic antigens.

Treatment

For asymptomatic carriers and diseased patients the drug of choice is quinacrine hydrochloride or metronidazole.

Prevention

- Asymptomatic reservoirs of infection should be identified & treated.
- Avoidance of contaminated food and water.
- Drinking water from lakes and streams should be boiled, filtered and/or iodine treated.
- Proper waste disposal and use of latrine.

Trichomonas vaginalis

Important features – it is a pear-shaped organism with a central nucleus and four anterior flagella; and undulating membrane extends about two-thirds of its length. It exists only as a trophozoite form, and measured 7-23 μm long & 5-15 μm wide. Transmission is by sexual intercourse.

Pathogenesis

The trophozoite is found in the urethra & vagina of women and the urethra & prostate gland of men. After introduction by sexual intercourse, proliferation begins which results in inflammation & large numbers of trophozoites in the tissues and the secretions. The onset of symptoms such as vaginal or vulval pruritus and discharge is often sudden and occurs during or after menstruation as a result of the increased vaginal acidity. The vaginal secretions are liquors, greenish or yellowish, sometimes frothy, and foul smelling. Infection in the male may be latent, with no symptoms, or may be present as self limited, persistent, or recurring urethritis.

Epidemiology

This parasite has worldwide distribution, and sexual intercourse is the primary mode of transmission. Occasionally, infections can be transmitted by fomites (toilet articles, clothing), although this transmission is limited by liability of the trophozoite. Rarely Infants may be infected by passage through

the mother's infected birth canal. The prevalence of this flagellate in developing countries is reported to be 5-20% in women and 2-10% in men.

Clinical features

Clinical disease – trichomoniasis.

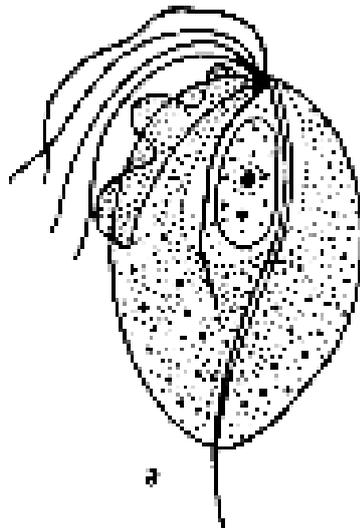
Most infected women at the acute stage are asymptomatic or have a scanty, watery vaginal discharge. In symptomatic cases vaginitis occurs with more extensive inflammation, along with erosion of epithelial lining, and painful urination, and results in symptomatic vaginal discharge, vulvitis and dysuria.

Immunity

The infection may induce humoral, secretory, and cellular immune reactions, but they are of little diagnostic help and do not appear to produce clinically significant immunity.

Laboratory diagnosis

- In females, *T. vaginalis* may be found in urine sediment, wet preparations of vaginal secretions or vaginal scrapings.
- In males it may be found in urine, wet preparations of prostatic secretions or following massage of the prostate gland.
- Contamination of the specimen with faeces may confuse *T. vaginalis* with *T. hominis*.



Trichomonas vaginalis

Treatment

Metronidazole is the drug of choice. If resistant cases occur, re-treatment with higher doses is required.

Prevention

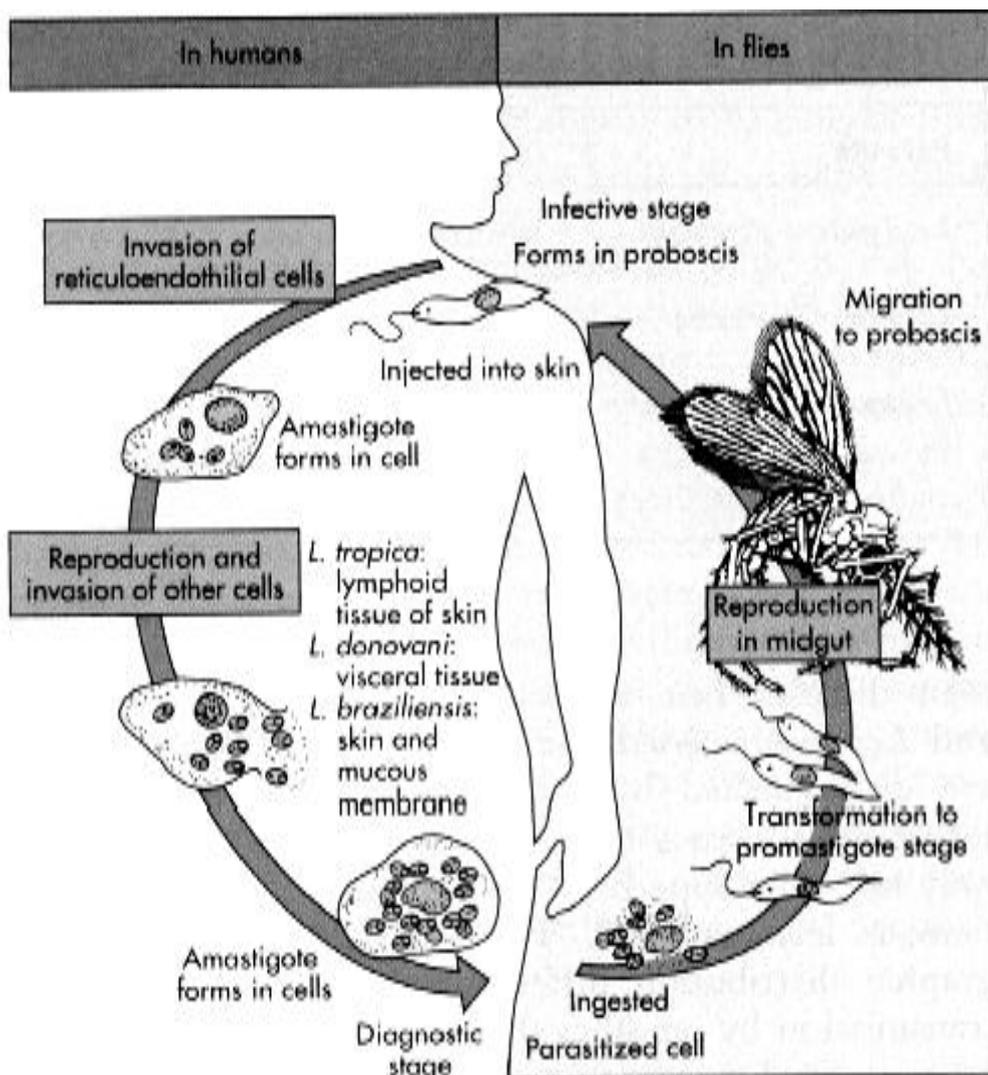
- Both male & female sex partners must be treated to avoid reinfection
- Good personal hygiene, avoidance of shared toilet articles & clothing.
- Safe sexual practice.

Haemoflagellates

LEISHMANIA SPECIES

Clinical disease – visceral leishmaniasis; cutaneous leishmaniasis; mucocutaneous leishmaniasis.

The species of leishmania exist in two forms, amastigote (aflagellar) and promastigote (flagellated) in their life cycle. They are transmitted by certain species of sand flies (Phlebotomus & Lutzomyia).



VISCERAL LEISHMANIASIS

Leishmania donovani

Important features – the natural habitat of *L. donovani* in man is the reticuloendothelial system of the viscera, in which the amastigote multiplies by simple binary fission until the host cells are destroyed, whereupon new macrophages are parasitized. In the digestive tract of appropriate insects, the developmental cycle is also simple by longitudinal fission of promastigote forms.

The amastigote stage appears as an ovoidal or rounded body, measuring about 2-3 μm in length; and the promastigotes are 15-25 μm lengths by 1.5-3.5 μm breadths.

Pathogenesis

In visceral leishmaniasis, the organs of the reticuloendothelial system (liver, spleen and bone marrow) are the most severely affected organs. Reduced bone marrow activity, coupled with cellular distraction in the spleen, results in anaemia, leukopenia and thrombocytopenia. This leads to secondary infections and a tendency to bleed. The spleen and liver become markedly enlarged, and hypersplenism contributes to the development of anaemia and lymphadenopathy also occurs. Increased production of globulin results in hyperglobulinemia, and reversal of the albumin-to-globulin ratio.

Epidemiology

L. donovani donovani, infection of the classic kala-azar (“black sickness”) or dum dum fever type occurs in many parts of Asia, Africa and Southeast Asia. Kala-azar occurs in three distinct epidemiologic patterns. In Mediterranean basin (European, Near Eastern, and Africa) and parts of China and Russia, the reservoir hosts are primarily dogs & foxes; in sub-Saharan Africa, rats & small carnivores are believed to be the main reservoirs. In India and neighboring countries (and Kenya), kala-azar is anthroponosis, i.e. there is no other mammalian reservoir host other than human. The vector is the Phlebotomus sand fly. Other variants of *L. donovani* are also recognized: *L. donovani infantum* with similar geographical distribution, reservoir host and vector; with *L. donovani donovani*. *L. donovani chagasi* is found in South America, Central America, especially Mexico, and the West Indies. Reservoir hosts are dogs, foxes, and cats, and the vector is the Lutzomyia sand fly.

Clinical features

Symptoms begin with intermittent fever, weakness, and diarrhea; chills and sweating that may resemble malaria symptoms are also common early in the infection. As organisms proliferate & invade cells of the liver and spleen, marked enlargement of the organs, weight loss, anemia, and emaciation occurs. With persistence of the disease, deeply pigmented, granulomatous lesion of skin, referred to as post-kala-azar dermal leishmaniasis, occurs. Untreated visceral leishmaniasis is nearly always fatal as a result of secondary infection.

Immunity

Host cellular and humoral defence mechanisms are stimulated.

Laboratory diagnosis

- Examination of tissue biopsy, spleen aspiration, bone marrow aspiration or lymph node aspiration in properly stained smear (e.g. Giemsa stain).
- The amastigotes appear as intracellular and extra cellular *L. donovani* bodies.
- Culture of blood, bone marrow, and other tissue often demonstrates the promastigote stage of the organisms.
- Serologic testing is also available.

Treatment

The drug of choice is sodium stibogluconate, a pentavalent antimonial compound.

Alternative approaches include the addition of allopurinol and the use of pentamidine or amphotercin B.

Prevention

- Prompt treatment of human infections and control of reservoir hosts.
- Protection from sand flies by screening and insect repellents.

OLD WORLD CUTANEOUS LEISHMANIASIS (ORIENTAL SORE)

Clinical disease

L. tropica minor – dry or urban cutaneous leishmaniasis

L. tropica major – wet or rural cutaneous leishmaniasis

L. aethiopica – cutaneous leishmaniasis

Important features

These are parasites of the skin found in endothelial cells of the capillaries of the infected site, nearby lymph nodes, within large mononuclear cells, in neutrophilic leukocytes, and free in the serum exuding from the ulcerative site. Metastasis to other site or invasion of the viscera is rare.

Pathogenesis

In neutrophilic leukocytes, phagocytosis is usually successful, but in macrophages the introduced parasites round up to form amastigote and multiply. In the early stage, the lesion is characterized by the proliferation of macrophages that contain numerous amastigotes. There is a variable infiltration of lymphocytes and plasma cell. The overlying epithelium shows acanthosis and hyperkeratosis, which is usually followed by necrosis and ulceration.

Epidemiology

Cutaneous leishmaniasis produced by *L.tropica* complex is present in many parts of Asia, Africa, Mediterranean Europe and the southern region of the former Soviet Union. The urban Cutaneous leishmaniasis is thought to be an anthroponosis while the rural cutaneous leishmaniasis is zoonosis with human infections occurring only sporadically. The reservoir hosts in *L. major* are rodents. *L. aethopica* is endemic in Ethiopia and Kenya. The disease is a zoonosis with rock & tree hyraxes serving as reservoir hosts. The vector for the old world cutaneous leishmaniasis is the Phlebotomus sand fly.

Clinical features

The first sign, a red papule, appears at the site of the fly's bite. This lesion becomes irritated, with intense itching, and begins to enlarge & ulcerate. Gradually the ulcer becomes hard and crusted and exudes a thin, serous material. At this stage, secondary bacterial infection may complicate the disease. In the case of the Ethiopian cutaneous leishmaniasis, there are similar developments of lesions, but they may also give rise to diffuse cutaneous leishmaniasis in patients who produce little or no cell mediated immunity against the parasite. This leads to the formation of disfiguring nodules over the surface of the body.

Immunity

Both humoral and cell mediated immunity are involved.

Treatment

The drug of choice is sodium stibogluconate, with an alternative treatment of applying heat directly to the lesion. Treatment of *L.aethopica* remains to be a problem as there is no safe and effective drug.

Prevention

- Prompt treatment & eradication of ulcers
- Control of sand flies & reservoir hosts.

NEW WORLD CUTANEOUS AND MUCOCUTANEOUS LEISHMANIASIS (AMERICAN CUTANEOUS LEISHMANIASIS)

Clinical disease

Leishmania mexicana complex – cutaneous leishmaniasis.

Leishmania braziliensis complex – mucocutaneous or cutaneous leishmaniasis.

Important features

The American cutaneous leishmaniasis is the same as oriental sore. But some of the strains tend to invade the mucous membranes of the mouth, nose, pharynx, and larynx either initially by direct extension or by metastasis. The metastasis is usually via lymphatic channels but occasionally may be the bloodstream.

Pathogenesis

The lesions are confined to the skin in cutaneous leishmaniasis and to the mucous membranes, cartilage, and skin in mucocutaneous leishmaniasis. A granulomatous response occurs, and a necrotic ulcer forms at the bite site. The lesions tend to become superinfected with bacteria. Secondary lesions occur on the skin as well as in mucous membranes. Nasal, oral, and pharyngeal lesions may be polypoid initially, and then erode to form ulcers that expand to destroy the soft tissue and cartilage about the face and larynx. Regional lymphadenopathy is common.

Epidemiology

Most of the cutaneous & mucocutaneous leishmaniasis of the new world exist in enzootic cycles of infection involving wild animals, especially forest rodents. *Leishmania mexicana* occurs in South and Central America, especially in the Amazon basin, with sloths, rodents, monkeys, and raccoons as reservoir

hosts. The mucocutaneous leishmaniasis is seen from the Yucatan ninsula into Central and South America, especially in rain forests where workers are exposed to sand fly bites while invading the habitat of the forest rodents. There are many jungle reservoir hosts, and domesticated dogs serve as reservoirs as well. The vector is the *Lutzomyia* sand fly.

Clinical features

The types of lesions are more varied than those of oriental sore and include Chiclero ulcer, Uta, Espundia, and Disseminated Cutaneous Leishmaniasis.

Laboratory diagnosis

- Demonstration of the amastigotes in properly stained smears from touch preparations of ulcer biopsy specimen.
- Serological tests based on fluorescent antibody tests.
- Leishman skin test in some species.

Immunity

The humoral and cellular immune systems are involved

Treatment

The drug of choice is sodium stibogluconate.

Prevention

- Avoiding endemic areas especially during times when local vectors are most active.
- Prompt treatment of infected individuals.

Trypanosomiasis

Etiologic agents

Trypanosoma brucei complex – African trypanosomiasis (sleeping sickness).

Trypanosoma cruzi – American trypanosomiasis (Chagas' disease).

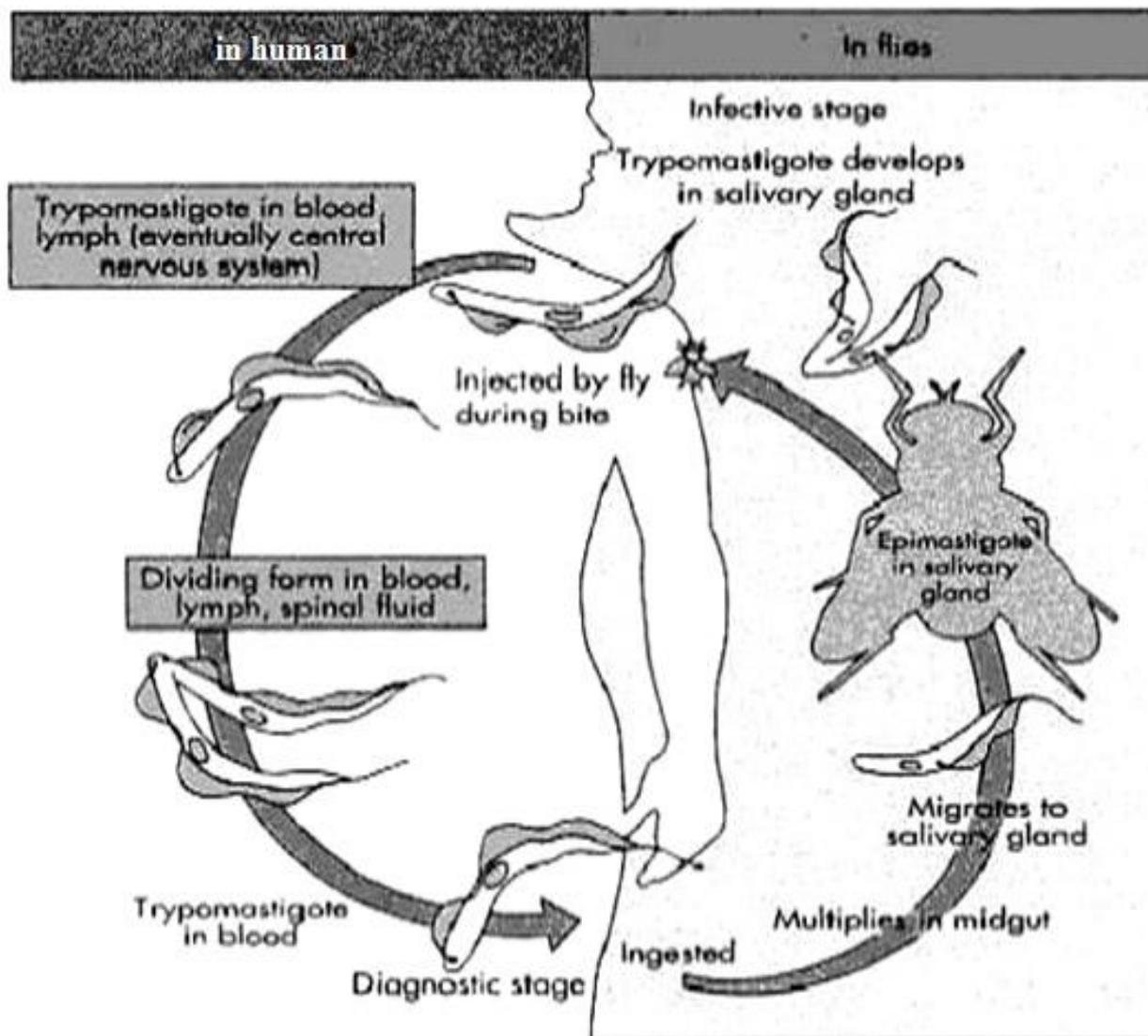
Important features

These species may have amastigote, promastigote, epimastigote, and trypomastigote stages in their life cycle. In human trypanosomes of the African form, however, the amastigote and promastigote stages of development are

absent. Typical trypanosome structure is an elongated spindle-shaped body that more or less tapers at both ends, a centrally situated nucleus, a kinetoplast posterior to nucleus, an undulating membrane arising from the kinetoplast and proceeding forward along the margin of the cell membrane and a single free flagellum at the anterior end.

AFRICAN TRYPANOSOMIASIS

Trypanosoma gambiense and *Trypanosoma rhodesiense* are causative agents of the African trypanosomiasis, transmitted by insect bites. The vector for both is the tsetse fly.



Life cycle of *Trypanosoma brucei*

Pathogenesis

The trypomastigotes spread from the skin through the blood to the lymph node and the brain. The typical somnolence (sleeping sickness) usually progresses to coma as a result of demyelinating encephalitis. In acute form, cyclical fever spike (approximately every 2 weeks) occurs that is related to antigenic variation. As antibody mediated agglutination and lysis of the trypomastigotes occurs, the fever subsides. With a few remains of antigenic variants new fever spike occurs and the cycle repeats itself over a long period.

Epidemiology

T. burcei gambiense is limited to tropical west and central Africa, correlating with the range of the tsetse fly vector. The tsetse flies transmitting *T.b. gambiense* prefer shaded stream banks for reproduction and proximity to human dwellings. People who work in such areas are at greatest risk of infection. An animal reservoir has not been proved for this infection. *T. burcei rhodeseinse* is found primarily in East Africa, especially the cattle-raising countries, where tsetse flies breed in the brush rather than along stream banks. *T.b. rhodeseines* also differs from *T.b. gambiense* in that domestic animal hosts (cattle and sheep) and wild game animals act as reservoir hosts. This transmission and vector cycle makes the organism more difficult to control than *T.b. gambiense*.

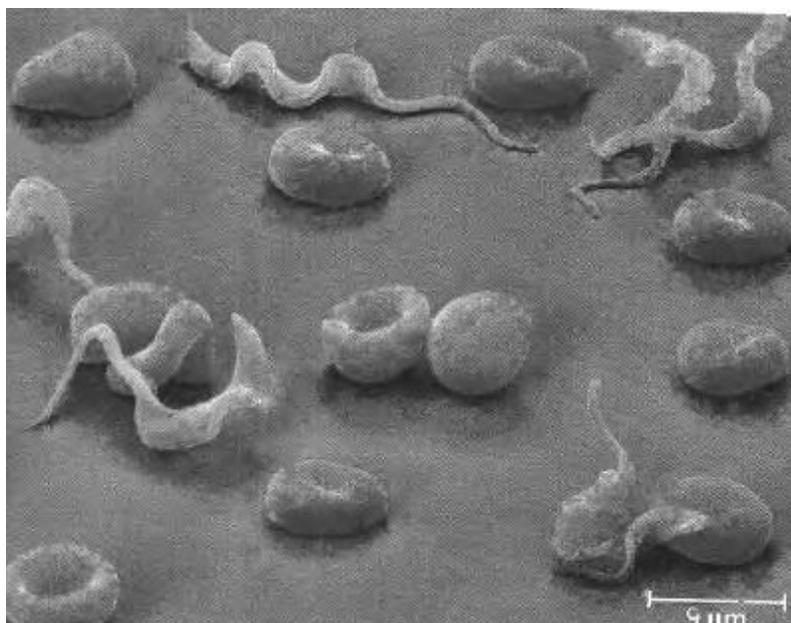
Clinical features

Although both species cause sleeping sickness, the progress of the disease is different. *T.gambiense* induced disease runs a low-grade chronic course over a few years. One of the earliest signs of disease is an occasional ulcer at the site of the fly bite. As reproduction of organisms continues, the lymph nodes are invaded, and fever, myalgia, arthralgia, and lymph node enlargement results. Swelling of the posterior cervical lymph nodes is characteristic of Gambian sleeping sickness and is called winterbottom's sign. Chronic disease progresses to CNS involvement with lethargy, tremors, meningoencephalitis, mental retardation, and general deterioration. In the final stages, convulsions, hemiplegia, and incontinence occur. The patient becomes difficult to arouse or obtain a response from, eventually progressing to a comatose state. Death is the result of CNS damage and other infections, such as pneumonia. In *rhodesiense*, the disease caused is a more acute, rapidly progressive disease that is usually fatal. This more virulent organism also develops in greater numbers in the blood. Lymphadenopathy is uncommon, and early in the infection, CNS invasion

occurs, resulting in lethargy, anorexia, and mental disturbance. The chronic stages described for *T.gambiense* are not often seen, because in addition to rapid CNS disease, the organism produces kidney damage and myocarditis, leading to death.

Immunity

Both the humoral and cellular immunity involve in these infections. The immune responses of the host to the presence of these parasites, however, is faced with antigenic variation, in which organisms that have changed their antigenic identity can escape the host immune response and initiate another disease process with increased level of parasitemia.



Trypomastigote stage of *Trypanosoma burcei complex*

Laboratory

Examination of thin and thick films, in concentrated anticoagulated blood preparations, and in aspiration from lymph nodes and concentrated spinal fluid. Methods for concentrating parasites in blood may be helpful approaches including centrifugation of heparinized samples and an ion-exchange chromatography. Levels of parasitosis vary widely, and several attempts to visualize the organism over a number of days may be necessary.

Treatment

The same treatment protocol is applied for these parasites. For the acute stages of the disease the drug of choice is suramin with pentamidine as an

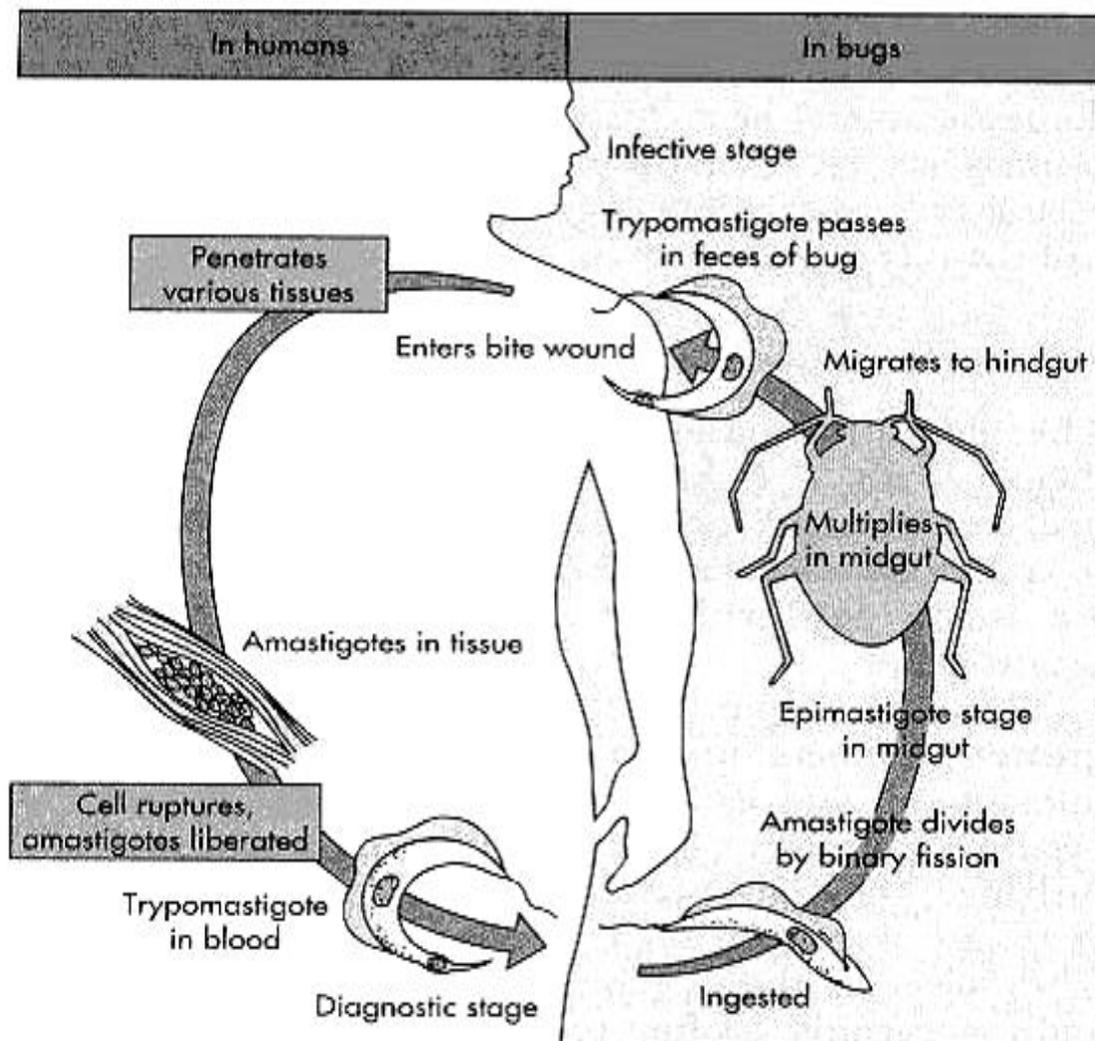
alternative. In chronic disease with CNS involvement, the drug of choice is melarsoprol. Alternatives include trypanamide combined with suramin.

Prevention

- Control of breeding sites of tsetse flies and use of insecticides.
- Treatment of human cases to reduce transmission to flies.
- Avoiding insect bite by wearing protective clothing and use of screen, bed netting and insect repellants.

AMERICAN TRYPANOSOMIASIS

Trypanosoma cruzi is a pleomorphic trypanosome that includes an additional form of amastigote in its life cycle. The vector for transmission are reduviid bugs.



Life cycle of *Trypanosoma cruzi*

Pathogenesis

During the acute phase, the organism occurs in blood as a typical trypomastigote and in the reticuloendothelial cells as a typical amastigote. The amastigotes can kill cells and cause inflammation, consisting mainly of mononuclear cells. Cardiac muscle is the most frequently and severely affected tissue. In addition, neuronal damage leads to cardiac arrhythmias and loss of tone in the colon (megacolon) and esophagus (megaesophagus). In the chronic phase, the organism persists in the amastigote form.

Epidemiology

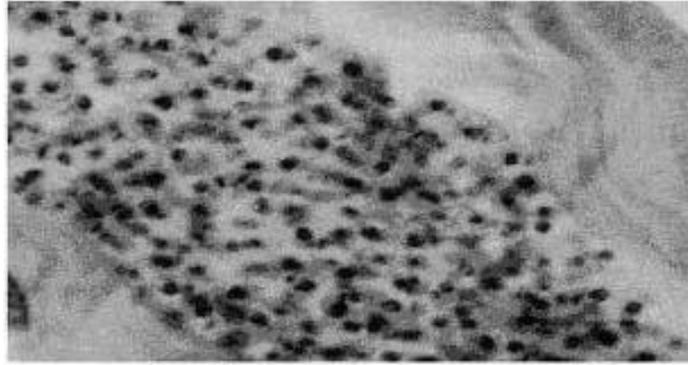
T. cruzi occurs widely in both reduviid bugs and a broad spectrum of reservoir animals in North, Central, and South America. Human disease is found most often among children in South and Central America, where there is direct correlation between infected wild animal reservoir hosts and the presence of infected bugs whose nests are found in human dwellings.

Clinical features

Chagas' disease may be asymptomatic acute or chronic disease. One of the earliest signs is development at the site of the bug bite of an erythematous and indurated area called a chagoma. This is often followed by a rash and edema around the eyes and face; in young children frequently an acute process with CNS involvement may occur. Acute infection is also characterized by fever, chills, malaise, myalgia, and fatigue. The chronic Chagas' disease is characterized by hepatosplenomegaly, myocarditis, and enlargement of the esophagus and colon as a result of the destruction of nerve cells (E.g. Auerbach's plexus) and other tissues that control the growth of these organs. Involvement of the CNS may produce granulomas in the brain with cyst formation and a meningoencephalitis. Death from chronic Chagas' disease results from tissue destruction in the many areas invaded by the organisms, and sudden death results from complete heart block and brain damage.

Laboratory diagnosis

Examine thin or thick stained preparations for trypomastigotes. Wet preparations should also be examined to look for motile organisms that leave the blood stream and become difficult to find. Biopsy of lymph nodes, liver, spleen, or bone marrow may demonstrate organisms in amastigote stage.



Amastigote stage of *Trypanosoma cruzi* in skeletal muscle

Xenodiagnosis – which consists of allowing an uninfected, laboratory-raised reduviid bug to feed on the patient and, after several weeks, examining the intestinal contents of the bug for the organism.

Immunity

Unlike African trypanosomiasis, the antigenic variation is less common in *T. cruzi* infection. Therefore, the humoral and cellular immune responses function in the immune system.

Treatment

The drug of choice is nifurtimox. Alternative agents include allopurinol & benzimidazole.

Prevention

- Bug control, eradication of nests
- Treating infected person & exclusion of donors by screening blood.
- Development of vaccine.

6.4. Medically important Ciliates

The intestinal protozoan *Balantidium coli* is the only member of the ciliate group that is pathogenic for humans. Disease produced by *B. coli* is similar to amebiasis, because the organisms elaborate proteolytic and cytotoxic substances that mediate tissue invasion and intestinal ulceration.

Life cycle

The life cycle of *B. coli* is simple, involving ingestion of infectious cysts, excystation, and invasion of trophozoites into the mucosal lining of the large intestine, caecum, and terminal ileum. The trophozoite is covered with rows of hair like cilia that aid in motility. Morphologically more complex than amebae, *B. coli* has a funnel-like primitive mouth called a cytostome, a large (macro) nucleus and a small (micro) nucleus involved in reproduction.

Epidemiology

B. coli are distributed worldwide. Swine and (less commonly) monkeys are the most important reservoirs. Infections are transmitted by the faecal-oral route; outbreaks are associated with contamination of water supplies with pig faeces. Person-to-person spread, including through food handlers, has been implicated in outbreaks. Risk factors associated with human disease include contact with swine and substandard hygienic conditions.

Clinical features

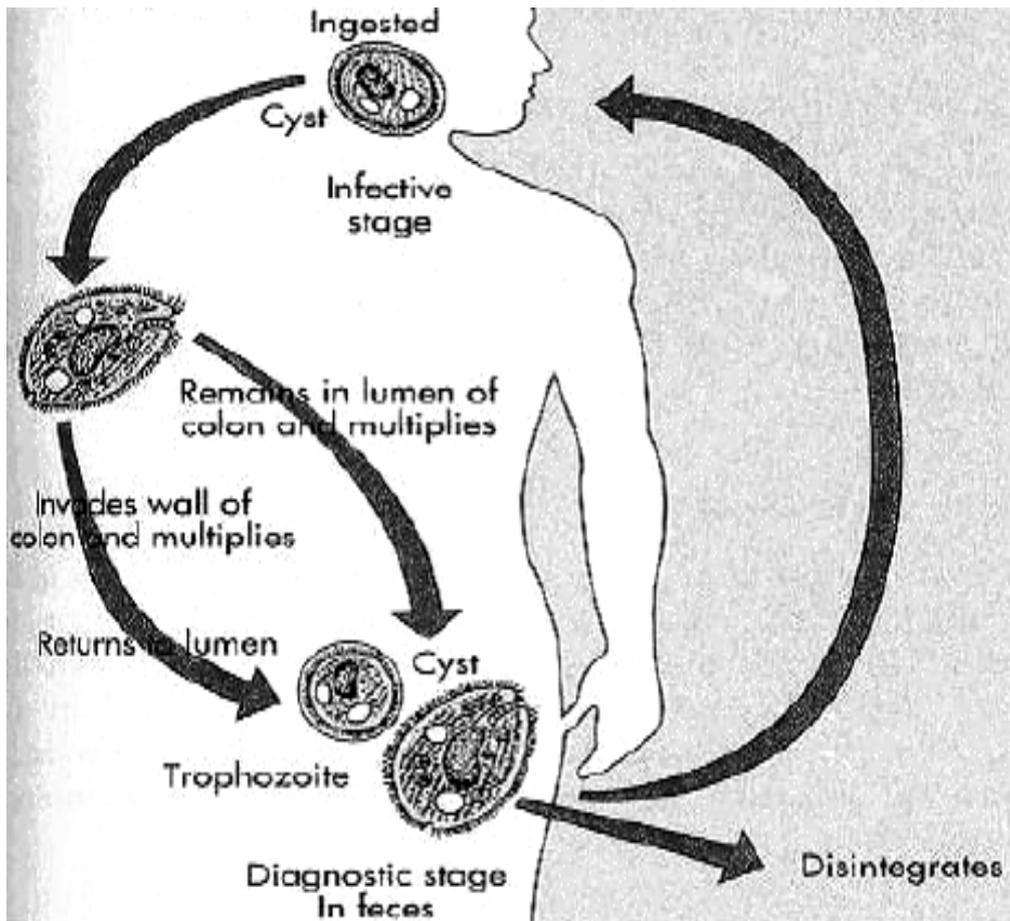
As with other protozoan parasites, asymptomatic carriage of *B. coli* can exist. Symptomatic disease is characterized by abdominal pain, tenderness, tenesmus, nausea, anorexia, and watery stools with blood and pus. Ulceration of the intestinal mucosa, as with amebiasis, can be seen; a secondary complication caused by bacterial invasion into the eroded intestinal mucosa can occur. Extra intestinal invasion of organs is extremely rare in balantidiasis.

Laboratory Diagnosis

Microscopic examination of faeces for trophozoite and cysts is performed. The trophozoite is very large, varying in length from 50 to 200 μm and in width from 40 to 70 μm . The surface is covered with cilia.

Treatment

The drug of choice is tetracycline; iodoquinol and metronidazole are alternative agents.



Life cycle of *Balantidium coli*

Testing your knowledge

PARASITES – REPRESENTATIVES OF PROTOZOA

1. The main host of *Plasmodium falciparum*
 - a) trophozoite
 - b) man
 - c) anopheles mosquito of genus *Anopheles*
 - d) fish

2. Intermediate host of *Plasmodium falciparum*
 - a) mollusc
 - b) man
 - c) anopheles mosquito of genus *Anopheles*
 - d) fish

3. Who enters the the human body through the bite of a mosquito with malaria
 - a) merozoites
 - b) sporozoites
 - c) schizonts
 - d) ookinete
4. Who forms of the erythrocytes after schizogony
 - a) merozoites
 - b) sporozoites
 - c) schizonts
 - d) ookinete
5. Sporozoites are formed as a result of the process
 - a) schizogony
 - b) sporogony
 - c) fertilization
 - d) gametogenesis
6. Who fall into into the stomach of a mosquito when it feeds on the blood of human malaria patient
 - a) sporozoites;
 - b) merozoites;
 - c) gametocytes;
 - d) oocyst
7. Cyst of dysentery amoeba has
 - a) two nuclei
 - b) three nuclei
 - c) four nuclei
 - d) eight nuclei
8. Trophozoites of dezintery amoeba destroys mucous membrane and feed on
 - a) blood
 - b) kidney tissue
 - c) lymph
 - d) protozoa
9. A person infected with dysentery amoeba;
 - a) through unwashed hands;
 - b) through the meat of sick animals;
 - c) through blood;
 - d) sexually transmitted

10. *Giardia lamblia* trophozoites are located in the
- duodenum;
 - blood;
 - skin;
 - heart
11. *Giardia* is characterized by the presence of
- radial symmetry
 - two pairs of flagella
 - pear-shaped with two nuclei
 - four axonemes
12. The trophozoites of *Giardia lamblia*
- can attach to the intestinal villi by the ventral sucking discs
 - feed on blood
 - destroy mucosa
 - penetrate the muscular layer of the intestinal wall
13. The trophozoite is found in the urethra and vagina of women and the urethra and prostate gland of men
- in life cycle of *Giardia lamblia*.
 - in life cycle of *Trypanosoma brucei*
 - in life cycle of *Trypanosoma cruzi*
 - in life cycle of *Trichomonas*
14. The vector is the tsetse fly
- in life cycle of *Giardia lamblia*.
 - in life cycle of *Trypanosoma brucei*
 - in life cycle of *Trypanosoma cruzi*
 - in life cycle of *Trichomonas*
15. The vector for transmission are reduviid bugs
- in life cycle of *Giardia lamblia*.
 - in life cycle of *Trypanosoma brucei*
 - in life cycle of *Trypanosoma cruzi*
 - in life cycle of *Trichomonas*
16. The parasite that causes the formation of deep skin ulcers
- Trypanosoma brucei*
 - Giardia lamblia*
 - Leishmania donovani*
 - Leishmania tropica minor*

17. The most severely affected organs are the organs of the reticuloendothelial system (liver, spleen and bone marrow) in lesions

- a) *Trypanosoma brucei*
- b) *Giardia lamblia*
- c) *Leishmania donovani*
- d) *Leishmania tropica minor*

18. This disease is accompanied by lethargy, tremors, meningoencephalitis, mental retardation, and general deterioration

- a) Gambian sleeping sickness
- b) mucocutaneous Leishmaniasis
- c) trichomoniasis
- d) amoebiasis

19. The vector for transmission of African trypanosomiasis is

- a) tsetse fly
- b) dogs
- c) mosquitoes
- d) clams

20. The reservoir hosts in *Leishmania major* are

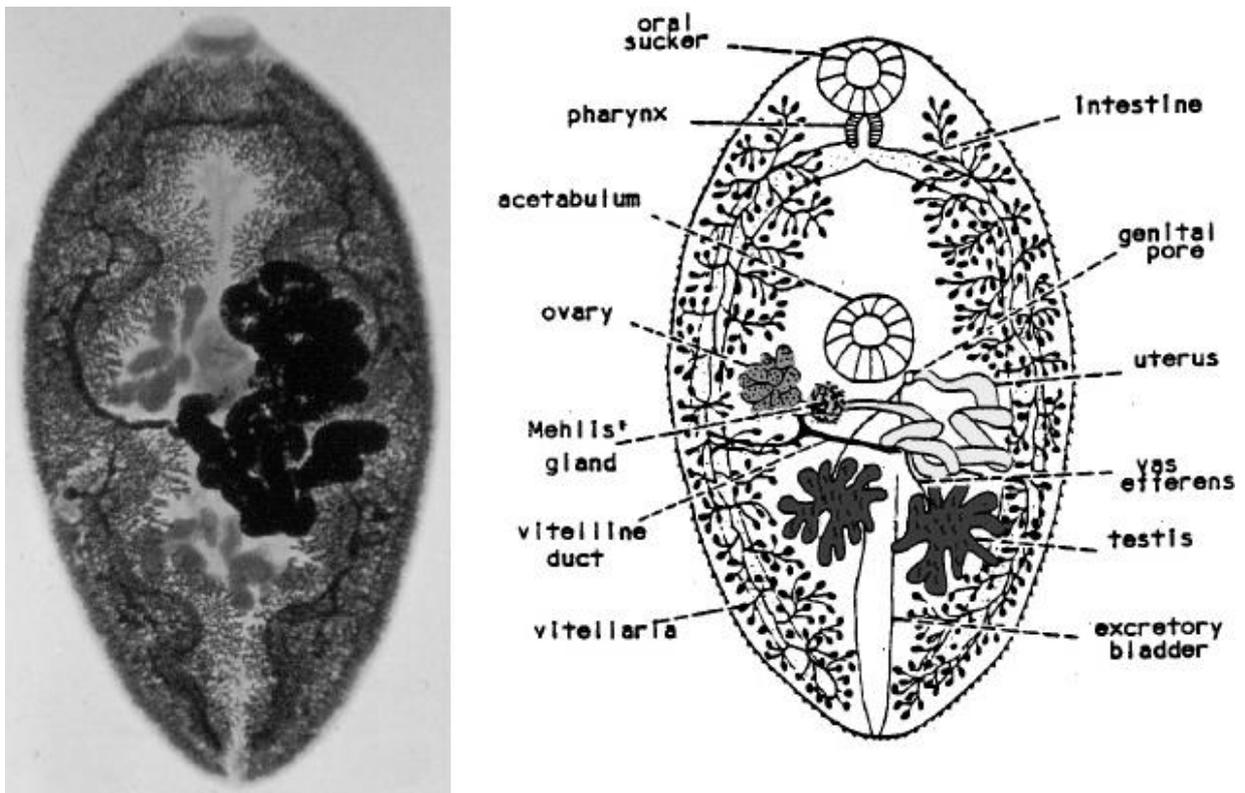
- e) rodents
- f) antelope
- g) tsetse fly
- h) dogs

UNIT 7. TREMATODES

7.1. *Paragonimus westermani*

Paragonimus westermani is the major species of lung fluke to infect humans, causing paragonimiasis. Paragonimiasis is a food-borne parasitic infection caused by the lung fluke. It may cause a sub-acute to chronic inflammatory disease of the lung.

In size, shape, and color, *P. westermani* resembles a coffee bean when alive. Adult worms are 7.5 to 12 mm long and 4 to 6 mm wide. The thickness ranges from 3.5 to 5 mm.



The skin of the worm (tegument) is thickly covered with scalelike spines. The oral and ventral suckers are similar in size, with the latter placed slightly pre-equatorially. The excretory bladder extends from the posterior end to the pharynx. The lobed testes are adjacent from each other located at the posterior end, and the lobed ovaries are off-centered near the center of the worm (slightly postacetabular). The uterus is located in a tight coil to the right of the

acetabulum, which is connected, to the vas deferens. The vitelline glands, which produce the yolk for the eggs, are widespread in the lateral field from the pharynx to the posterior end. *P. westermani* eggs range from 80 to 120 μm long by 45 to 70 μm wide. They are yellow-brown, ovoid or elongate, with a thick shell, and often asymmetrical with one end slightly flattened.



Egg of *Paragonimus westermani*

Paragonimus has a quite complex **life-cycle** that involves two intermediate hosts as well as humans. **Eggs** first develop in water after being expelled by coughing (unembryonated) or being passed in human feces. In the external environment, the eggs become embryonated. In the next stage, the parasite **miracidia** hatch and invades the first intermediate host such as a species of freshwater snail. Miracidia penetrate its soft tissues of the snail. Within the snail mother **sporocyst** form and produce many **mother rediae**, which subsequently produce many **daughter rediae** which shed crawling cercariae into fresh water. **Cercariae** next invade the second intermediate host such as crabs or crayfish and encyst to develop into **metacercariae** within 2 months. Infection of humans or other mammals (definitive hosts) occurs via consumption of raw or undercooked crustaceans. Human infection with *P. westermani* occurs by eating inadequately cooked or pickled crab or crayfish that harbor metacercariae of the parasite. The metacercariae excyst in the duodenum, penetrate through the intestinal wall into the peritoneal cavity, then through the abdominal wall and diaphragm into the lungs, where they become encapsulated and develop into

adults. The worms can also reach other organs and tissues, such as the brain and striated muscles, respectively. However, when this takes place completion of the life cycles is not achieved, because the eggs laid cannot exit these sites.

Diagnosis is based on microscopic demonstration of eggs in stool or sputum, but these are not present until 2 to 3 months after infection.

7.2. *Fasciola hepatica*

Fasciola hepatica is a parasitic fluke that lives in the liver. In addition to humans it infects cows and sheep. It is known as the common liver fluke and causes a disease called fascioliasis.

Fasciola hepatica is one of the largest flukes of the world, reaching a length of 30 mm and a width of 13 mm (*Fasciola gigantica*, on the other hand, is even bigger and can reach up to 75 mm). It is leaf-shaped, pointed at the back (posteriorly) and wide in the front (anteriorly). The oral sucker is small but powerful and is located at the end of a cone-shape projection at the anterior end. The acetabulum is a larger sucker than the oral sucker and is located at the anterior end.

The outer surface of the fluke is called the tegument. This is composed of scleroprotein and its primary function is to protect the fluke from the destructive digestive system of the host. Its also used for renewal of the surface plasma membrane and the active uptake of nutrients.

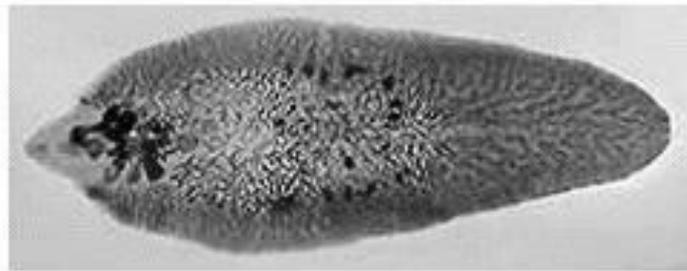
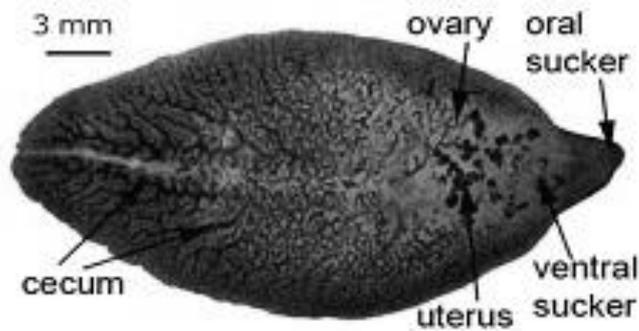
The alimentary canal of *F. hepatica* has a single mouth which leads into the blind gut; it has no anus. The mouth is located within the anterior sucker on the ventral side of the fluke. This mouth leads to the pharynx, which is then followed by a narrow oesophagus. The oesophagus, which is lined with a thin layer of epithelial cells, then opens up into the large intestine. As there is no anus, the intestine branches, with each branch ending blindly near the posterior end of the body. It has been shown that flukes migrate into smaller capillaries and bile ducts when feeding within the host. They use their mouth suckers to pull off and suck up food, bile, lymph and tissue pieces from the walls of the bile ducts. *F. hepatica* relies on extracellular digestion which occurs within the intestine of the host. The waste materials are egested through the mouth. The

non-waste matter is adsorbed back in through the tegument and the general surface of the fluke. The tegument facilitates this adsorption by containing many small folds to increase the surface area.

F. hepatica has no respiratory organs: the adult flukes respire anaerobically (without oxygen). *F. hepatica's* excretory system contains a network of tubules surrounding one main excretory canal. This canal leads to the excretory pore at the posterior end of the fluke. This main canal branches into four sections within the dorsal and ventral regions of the body. The role of *F. hepatica's* excretory system is excretion and osmoregulation. Each tubule within the excretory system is connected to a flame cell, otherwise known as protonephridia. These cells are modified parenchyme cells. In *F. hepatica* their role is to perform excretory, but more importantly, osmoregulatory functions. Flame cells are therefore primarily used to remove excess water.

The nerve system of *F. hepatica* consists of a pair of nerve ganglia, each one is located on either side of the oesophagus. Around the oesophagus is a nerve ring. This nerve ring connects the two nerve ganglia together. The nerves stem off from this ring, reaching all the way down to the posterior end of the body. At the posterior end, one pair of nerves become thicker than the others, these are known as the lateral nerve cords. From these lateral nerve cords, the other nerves branch. Sensory organs are absent from *F. hepatica*.

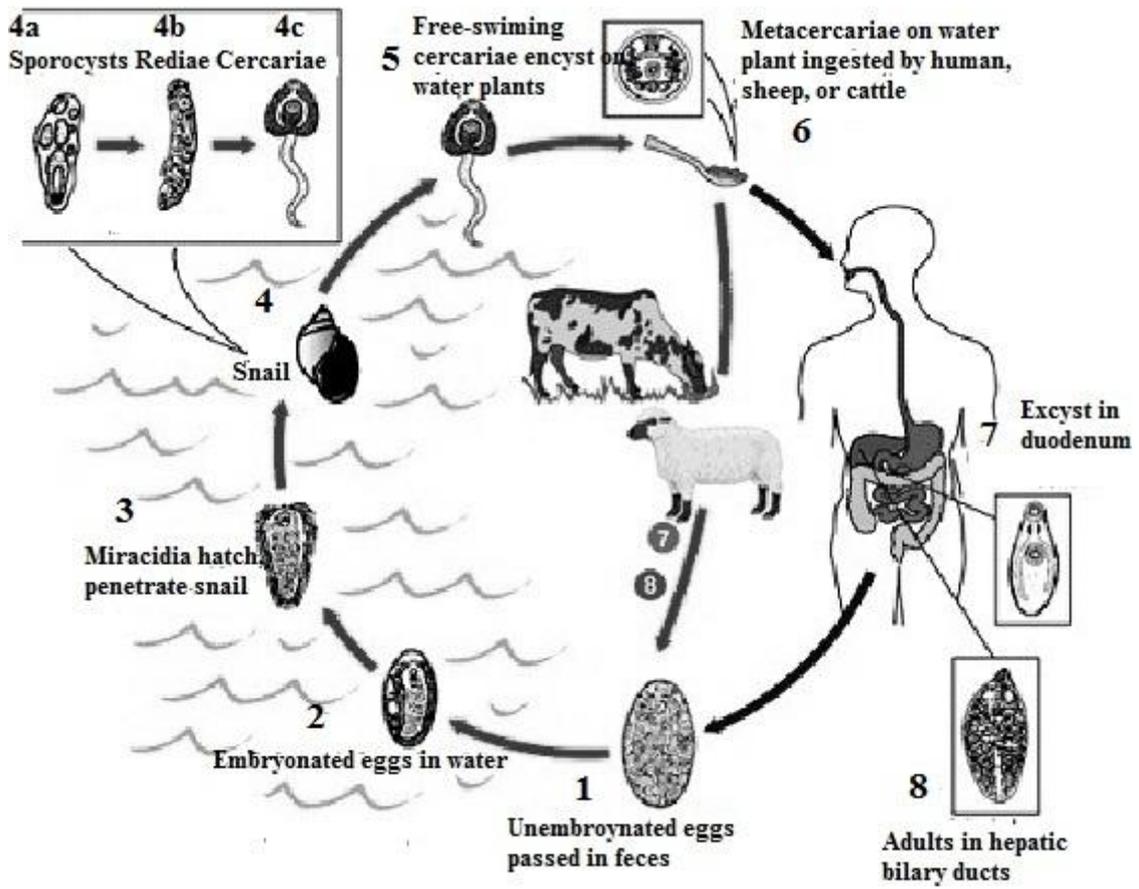
F. hepatica adult flukes are hermaphrodite, this means each fluke contains both male and female reproductive organs. The male and female reproductive organs open up into the same chamber within the body, which is called the genital atrium. The genital atrium is an ectodermal sac which opens up to the outside of the fluke via a genital pore. The testes are formed of two branched tubules, these are located in the middle and posterior regions of the body. From the epithelium lining of the tubules sperm is produced. The sperm then passes into the vas deferens and then into the seminal vesicle. From the seminal vesicle projects the ejaculatory duct and this is what opens up into the genital atrium, many prostate glands surround this opening. On the right hand side of the anterior testis there is a branched, tubular ovary. From here, a short oviduct passes to the vitelline duct. This duct connects, via a junction, the ovaries, the uterus and the yolk reservoir. From this junction, the uterus opens into the genital atrium, this opening is surrounded by Mehlis glands.



The life cycle of *Fasciola hepatica* starts when a female lays **eggs** in the liver of an infected human. Immature eggs are discharged in the biliary ducts and taken out in the feces. If landed in water, the eggs become embryonated and develop larvae called **miracidia**. Within the aquatic snail mother **sporocyst** form and produce many **mother rediae**, which subsequently produce many **daughter rediae** which shed crawling **cercariae**, a larva that is capable of swimming with its large tail. The cercaria exits and finds aquatic vegetation where it forms a cyst called **metacercaria**. A human eats the raw freshwater plant containing the cyst. The metacercaria excysts in the first part of the small intestine, duodenum. It then penetrates the intestinal wall and gets into the peritoneal cavity. It finds the liver and starts eating liver cells. This happens only a few days after the initial contact with the parasite. Usually the larva spends a few weeks just browsing and eating the liver. Then it relocates to the bile duct where it begins its final stage and becomes an adult. It takes about three months for the metacercaria to develop into an adult. Adults are about 3 cm long and 1 cm wide. Adult females can produce up to 25000 eggs per day.

In the chronic phase of fascioliasis adults in the large biliary ducts cause liver inflammation and obstruction of the biliary fluid. During the migration of the larvae (this acute phase of the disease lasts many weeks) symptoms include: diarrhea, eosinophilia (high number of white blood cells), fever, nausea, stomach ache, vomiting.

F. hepatica is found in areas where cattle and sheep are raised.



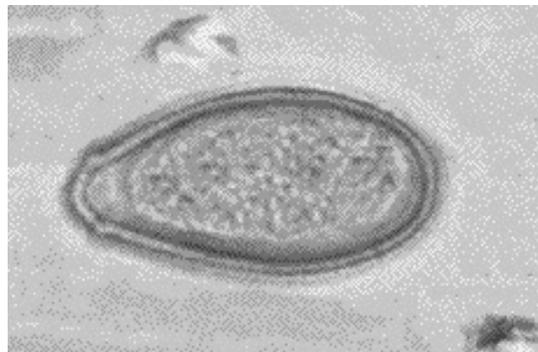
Egg of *Fasciola hepatica*

7.3. *Dicrocoelium dendriticum*

Dicrocoelium dendriticum, *Dicrocoelium hospes*, *Eurytrema pancreaticum* (pathogen – Less Common Liver Trematodes).

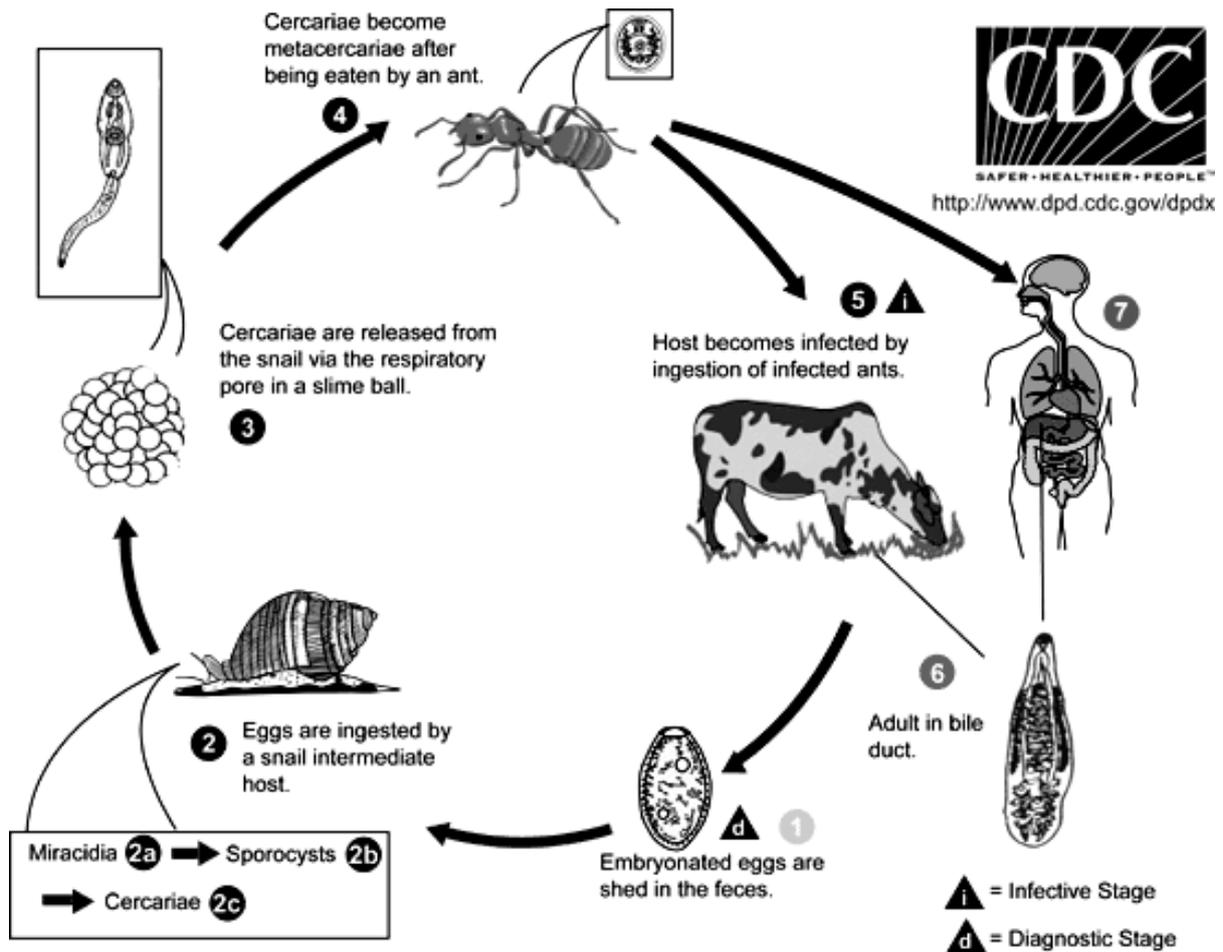


The adult worms of *D. dendriticum* are lancet-shaped, flat, and transparent and measure 5 to 15 mm long by 1.5 to 2.5 mm wide. The eggs are thick-shelled, operculate, deep golden brown, and measure 38 to 45 μm by 22 to 30 μm ; the eggs of the two flukes cannot be differentiated. The eggs are embryonated when passed and are resistant to drying.



Egg of *Dicrocoelium dendriticum*

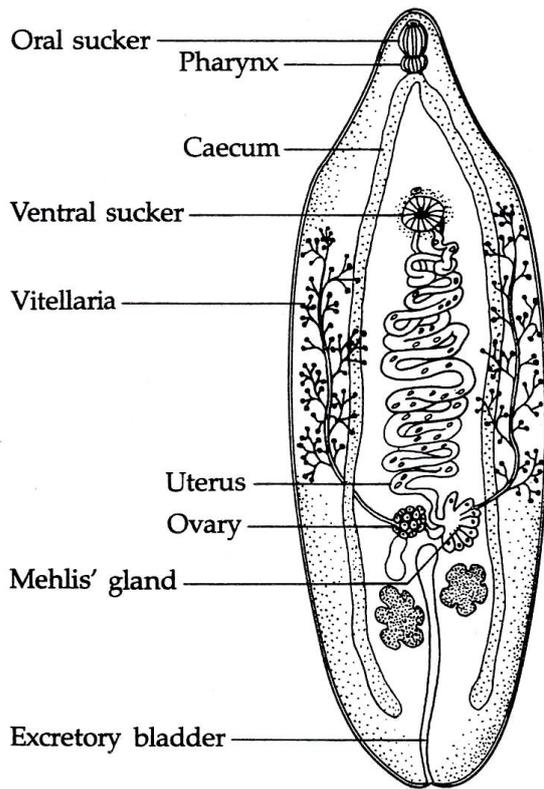
Life Cycle. The life cycle is similar to that of the other liver trematodes. However, in this case, the snail intermediate host is a land snail. The *cercariae* are released from the snail after rains follow a long period of dry weather. They are released from the snail's respiratory chamber as slime balls that are left behind on grass as the snail crawls along the ground or on plants. The ant is the required second intermediate host for *D. dendriticum*. Human infection is acquired through accidental ingestion of ants, primarily on fresh herbs or plants used for human consumption. The *metacercariae* excyst and migrate to the biliary passage for *D. dendriticum*, where they then become adult flukes.



Although the life cycle is similar to that caused by *F. hepatica*, the pathogenic effects are less severe and patients may report mild symptoms. Symptoms include chronic constipation and flatulent dyspepsia. In heavy infections, there may be jaundice with an enlarged liver. There may also be vomiting and diarrhea, as well as systemic toxemia. Eosinophilia tends to be absent in this infection.

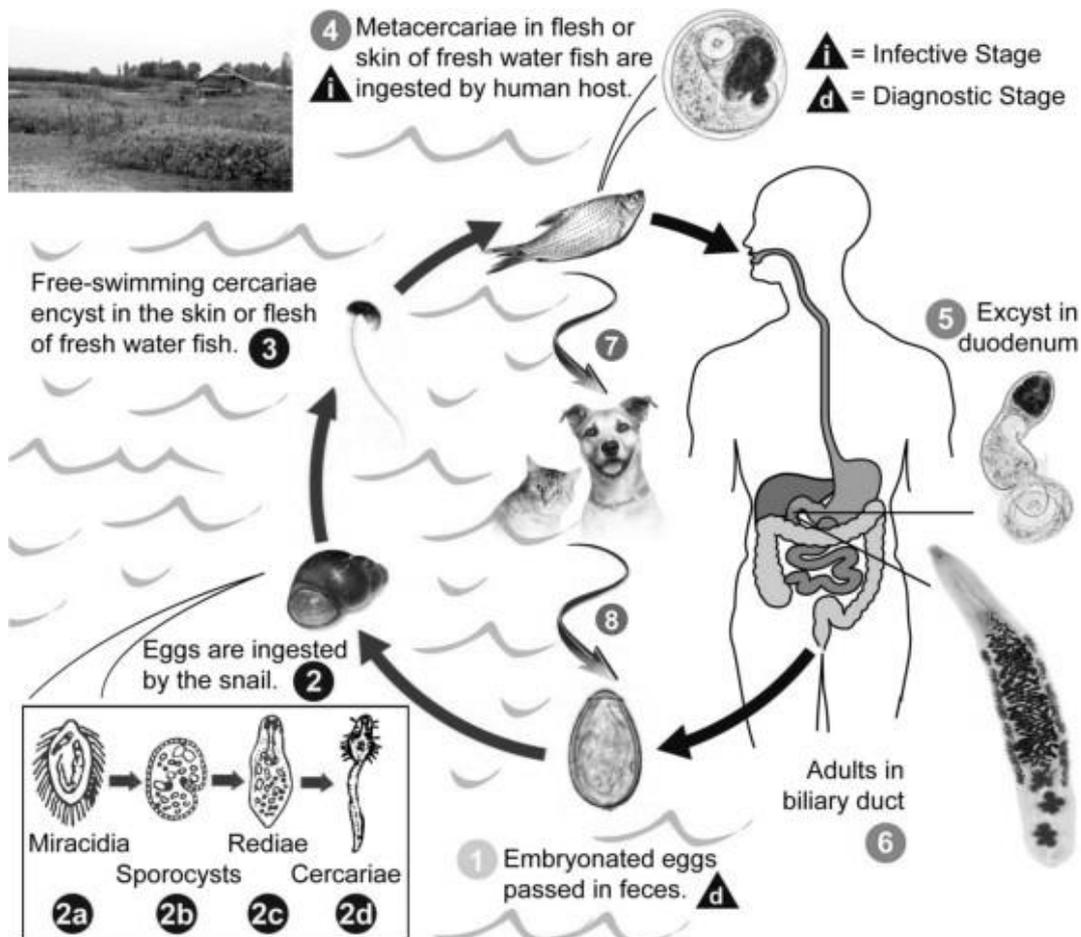
7.4. *Opisthorchis viverrini*

The adult flukes deposit fully developed eggs that are passed in the feces. After ingestion by a suitable snail (first intermediate host), the *eggs* release *miracidia*, which undergo in the snail several developmental stages (*sporocysts*, *rediae*, *cercariae*). Cercariae are released from the snail and penetrate freshwater fish (second intermediate host), encysting as *metacercariae* in the



muscles or under the scales. The mammalian definitive host (cats, dogs, and various fish-eating mammals including humans) become infected by ingesting undercooked fish containing metacercariae. After ingestion, the metacercariae excyst in the duodenum and ascend through the ampulla of Vater into the biliary ducts, where they attach and develop into adults, which lay eggs after 3 to 4 weeks. The adult flukes (*O. viverrini*: 5 mm to 10 mm by 1 to 2 mm; *O. felineus*: 7 mm to 12 mm by 2 mm to 3 mm) reside in the biliary and pancreatic ducts of the

mammalian host, where they attach to the mucosa.

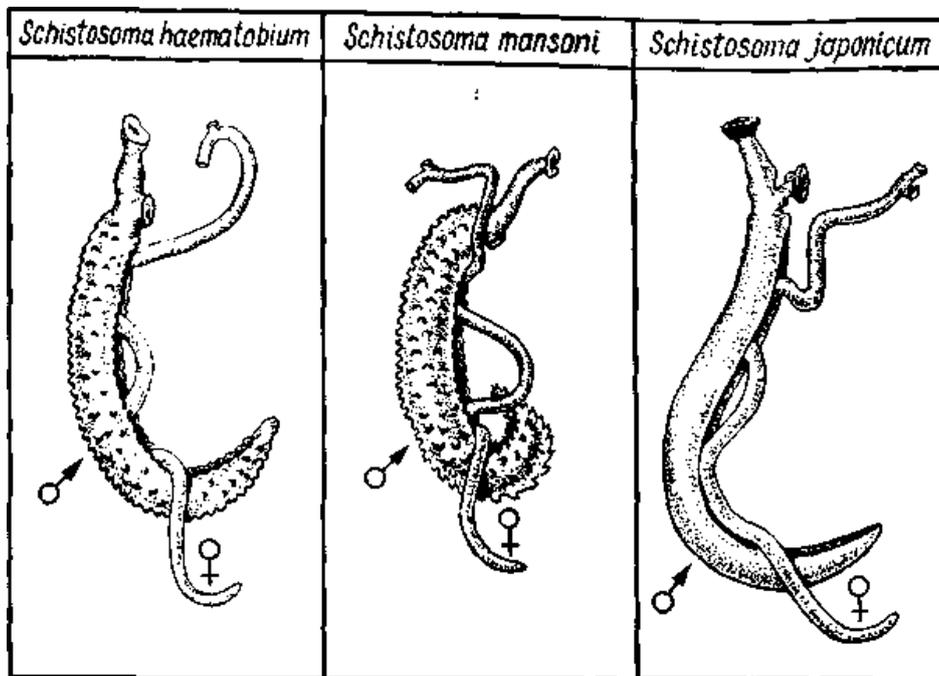


7.5. Schistosomiasis (bilharziasis)

Schistosome is the only fluke with separate sexes. The female worm lies in the gynecophoral canal of the male. This condition is important for transportation.

There are five medically important species:

1. *Schistosoma mansoni*: causes intestinal schistosomiasis.
2. *Schistosoma haematobium*: causes vesical (urinary) schistosomiasis.
3. *Schistosoma japonicum*: causes intestinal schistosomiasis.
4. *Schistosoma intercalatum*: causes intestinal schistosomiasis.
5. *Schistosoma mekongi*: causes intestinal schistosomiasis. This seems to cause milder disease in man. It causes disease in other vertebrate hosts.



The first two schistosomes (*S. mansoni* and *S. haematobium*) are prevalent in Ethiopia.

Schistosoma mansoni

Habitat. This species lives in the veins of the intestine.

Geographical distribution. It is found in Africa, South America, Middle East (some Arab countries) etc. Stream and lake-based transmission is common. The snail hosts that harbor *S. mansoni* are the genera: *Biomphalaria* (*B. glabrata*) and *Trochicorbis*. These have oval shells.

Morphology

Male. The male ranges in size from 1-1.4 cm in length and the body is covered by coarse tubercles. It has 6-9 testes.

Female. The female is 1.5-2.0 cm in length. The ovary is present in the anterior third and Vitelline glands occupy the posterior two-thirds. It lays about 100-300 eggs daily. The uterus is short containing few ova.

Urinary schistosomiasis

Etiology. *Schistosoma haematobium*

Habitat. The worm lives in the veins of the bladder of humans. The peak prevalence is the 10-14 year age group. The snail hosts that harbor *S. haematobium* are the genera *Bulinus* (*Bulinus africanus*, *B. truncatus*) and *Physopsis*.

Morphology

Male. The male ranges in size from 1-1.5 cm in length. The body is covered by fine tubercles. It has 4-5 testes.

Female. The female ranges in size from 2-2.5 cm in length. The ovary is present in the posterior third. Vitelline glands occupy the posterior thirds. Uterus is long containing many ova. It lays about 20-200 eggs daily.

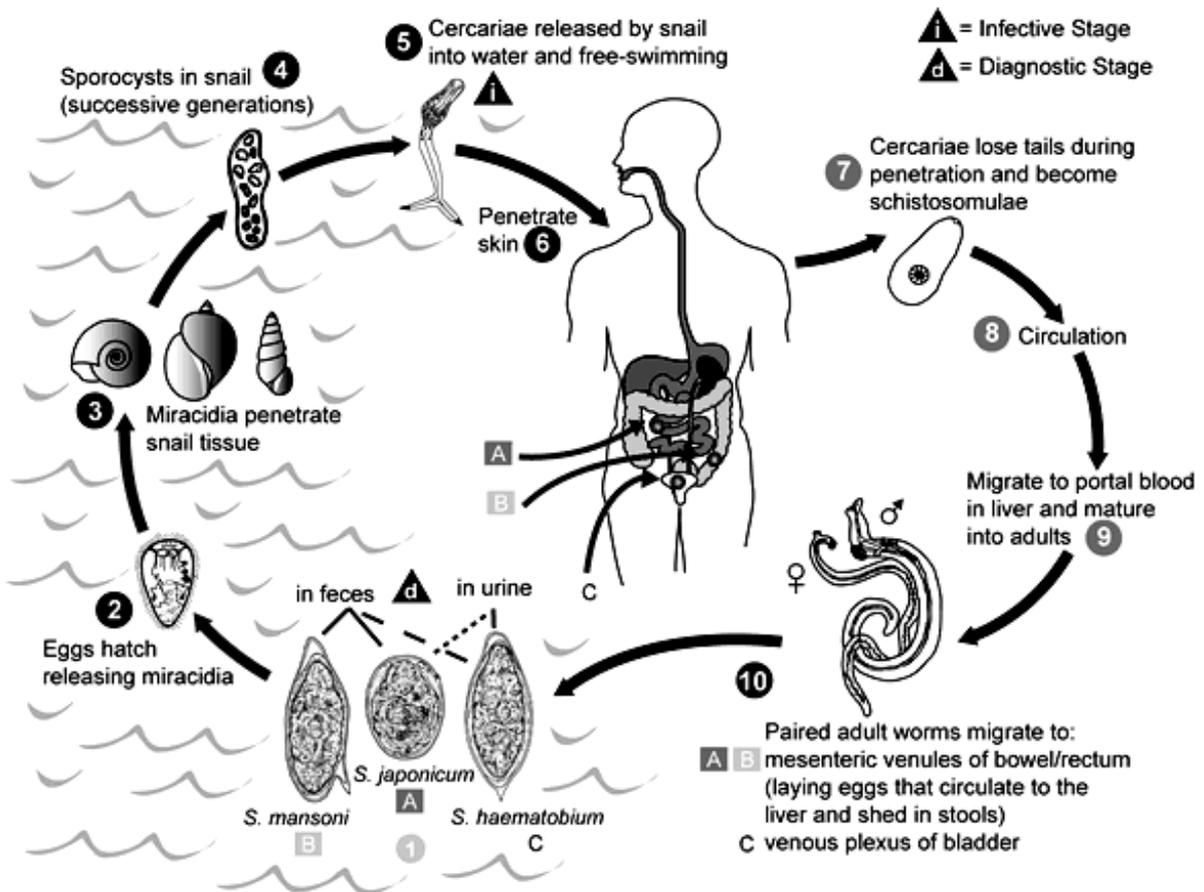
Distribution. In Ethiopia, *S. haematobium* is found in the Lower Awash Valley in the east and in Benshangul-Gumuz (Assossa) regional state in the west in low altitudes below 1000 meters above sea level.

Schistosoma japonicum. The female adult worm lays about 500-3500 eggs daily. The eggs are ovoid, bearing only a minute lateral spine or a small knob postero-laterally. It is found in Japan, China, and Philippines, etc.

Schistosoma intercalatum. This is the rarest and least pathogenic schistosome that matures in man. It is found in Western and Central Africa. The daily egg output is about 300. The eggs have a terminal spine.

Life cycle of Schistosomes. Adult worms reside in pairs: the female lying in the gynecophoral canal of the male. After fertilization, **eggs** are passed into the venules. A larval form – the **miracidium** - develops within the egg. Its lytic enzymes and the contraction of the venule rupture the wall of the venule liberating the egg into the perivascular tissues of the intestine (*S. mansoni*) or urinary bladder (*S. haematobium*). The eggs pass into the lumens and organs and are evacuated in the feces (*S. mansoni*) or the urine (*S. haematobium*).

On contact with fresh water the miracidia hatch from the eggs and swim about until they find the appropriate snail, which they penetrate. After two generations of *sporocyst* development and multiplication within the snail, the fork-tailed *cercariae* emerge. Infection to man takes place during bathing or swimming. The cercariae penetrate the skin, are carried into the systemic circulation and pass through to the portal vessels. Within the intrahepatic portion of the portal system, the worms feed and grow to maturity.



Egg of schistosomes

Symptoms and complications

Patients infected with *S. haematobium* suffer from terminal haematuria and painful micturition. There is inflammation of the urinary bladder (cystitis), and enlargement of spleen and liver.

Patients infected with *S. mansoni* suffer from cercarial dermatitis (swimmers itch) and dysentery (mucus and blood in stool with tenesmus) as well as enlargements of the spleen and liver.

S. haematobium causes squamous cell carcinoma in the bladder.

Laboratory Diagnosis

S. mansoni

- Microscopic examination of the stool for eggs after concentration by sedimentation method. The egg has characteristic lateral spine.

- Rectal snip.

S. haematobium

- Examination of the urine after allowing it to sediment in a conical urinalysis glass. A drop from the sediment is taken and examined for eggs. Egg has terminal spine.

- Biopsy from bladder.

Testing your knowledge

TREMATODES

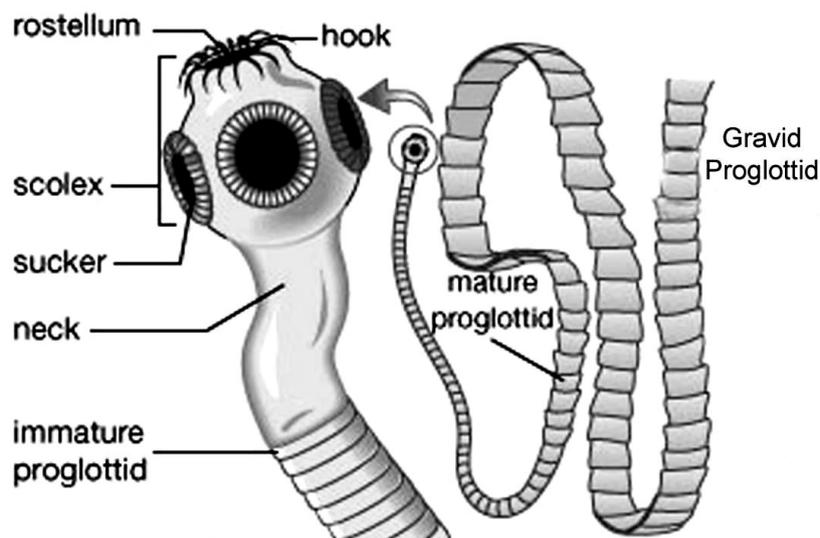
1. The definitive main host of the *Fasciola hepatica* is _____.
2. Larva of *Fasciola hepatica*, which comes out of the eggs and introduced into the mollusk is called _____.
3. Within the aquatic snail mother sporocyst of the *Fasciola hepatica* form and produce many mother _____, which subsequently produce many daughter _____ which shed crawling _____, a larva that is capable of swimming with its large tail.
4. Liver fluke disease causes _____.
5. _____ is the reason Paragonimiasis.

6. The second intermediate host of lung fluke (*Paragonimus*) is _____.
7. The second intermediate host of *Opisthorchis viverrini* is _____.
8. The second intermediate host of *Dicrocoelium dendriticum* is _____.
9. The adult worms of *Opisthorchis viverrini* lives in the _____.
10. The adult worms of _____ lives in the lungs.
11. The adult form of *Dicrocoelium dendriticum* lives in the _____.
12. The larvae of *Opisthorchis viverrini* developed in the following sequence: miracidia, _____, _____, metacercariae.
13. Under the ventral sucker of *Dicrocoelium dendriticum* is _____.
14. Under the ventral sucker of *Opisthorchis viverrini* is _____.
15. Adult worms of schistosomes reside in pairs: the female lying in the _____ of the male.
16. Adult worms of schistosomes lives in the _____.
17. Stage rediae absent in the life cycle of _____.
18. Ant is the second intermediate host of _____.
19. Crabs are the second intermediate host of _____.
20. The intermediate host of _____ is a land snail.

UNIT 8. CESTODES (TAPEWORMS)

Introduction

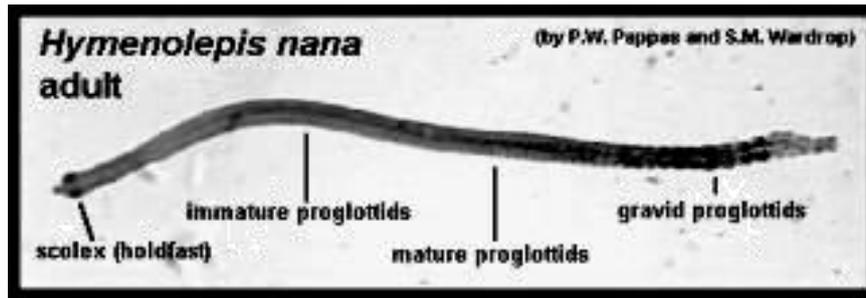
The tapeworms are hermaphroditic and require an intermediate host. The adult tapeworms found in humans have flat body, white or grayish in color. They consist of an anterior attachment organ or scolex and a chain of segments (proglottids) also called strobilla. The strobilla is the entire body except the scolex. The scolex has suckers or grooves. It has rostellum, which has 1 or 2 rows of hooks situated on the center of the scolex. Adult tapeworms inhabit the small intestine, where they live attached to the mucosa. Tapeworms do not have a digestive system. Their food is absorbed from the host's intestine.



8.1. *Hymenolepis nana* (dwarf tapeworm)

Morphology

Adult worm measures 1-3 cm in length. It is made up of head (scolex), neck and segmented body. The head carries four suckers and a rostellum armed with one row of hooks. The segments of the body are divided into mature and gravid segments. In the mature segment, there are three testes in the middle.

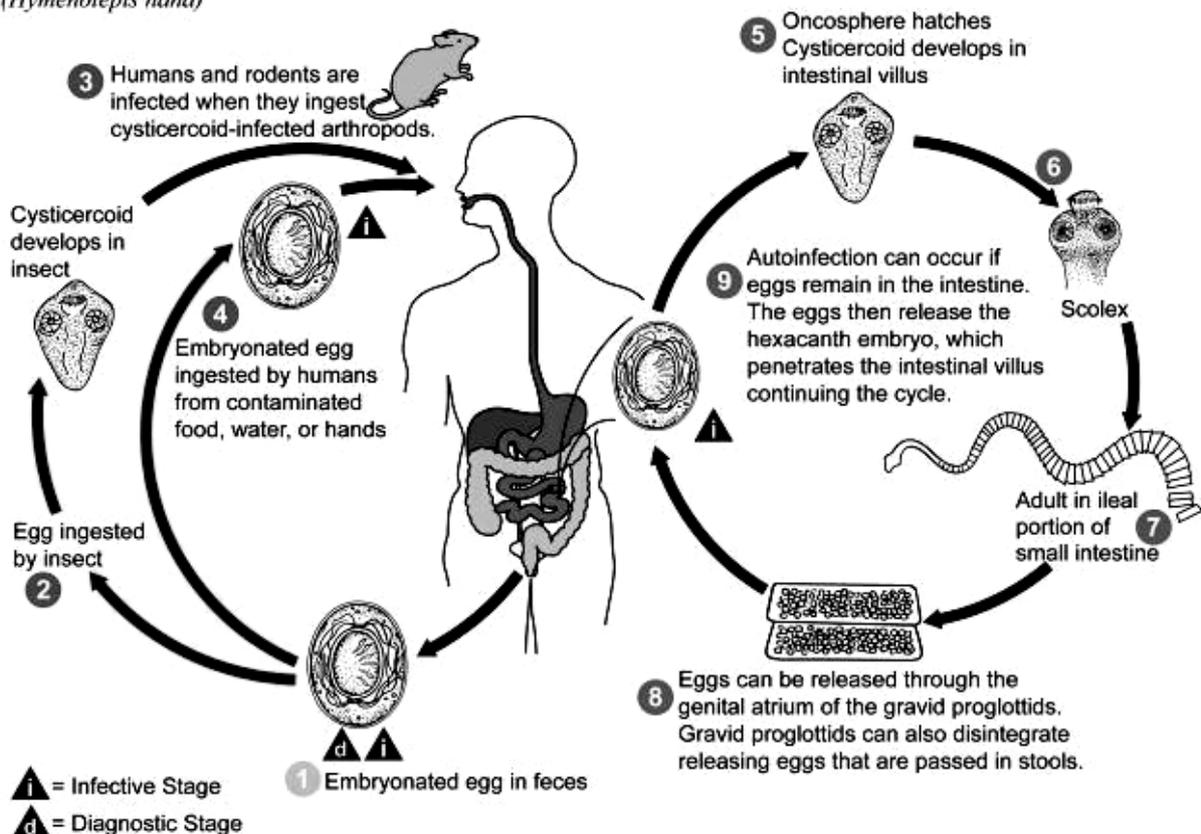


Infective stage and mode of infection

The egg, which is immediately infective when passed by the patient, is rounded, about 40 microns in diameter. It contains a six-hooked oncosphere within a rigid membrane (the embryosphere). This embryosphere has two polar thickening or knobs from which project 4-8 long, thin filaments called polar filaments.

Hymenolepiasis

(*Hymenolepis nana*)



Infection takes place by:

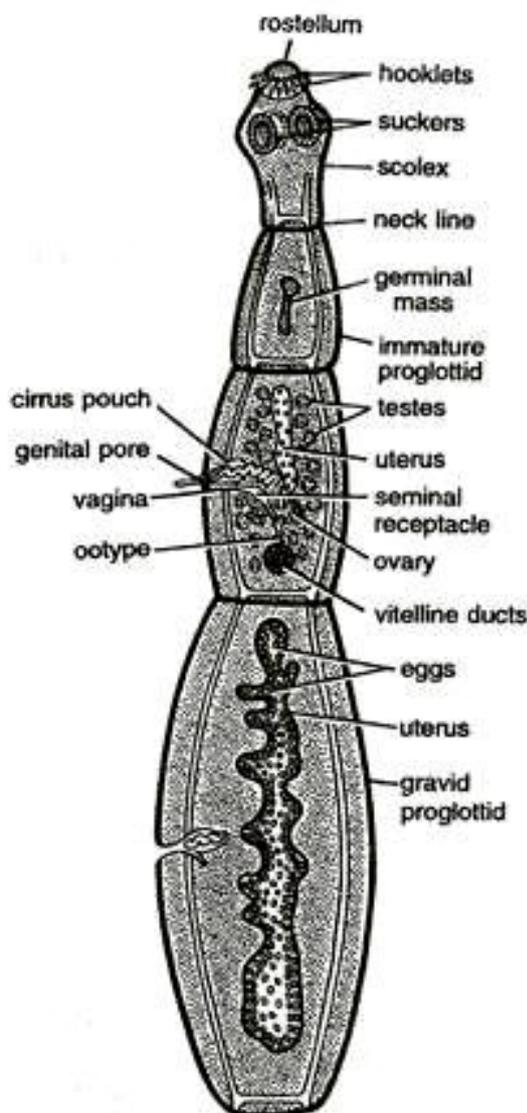
1. Ingestion of egg with contaminated raw vegetables.
2. Direct infection from a patient

3. Auto infection: the eggs of *H. nana* are infective as soon as they are passed with feces by the patient. If the hands of the patient are contaminated by these eggs, she/he infects herself/himself again and again.

Pathogenecity

Light infections produce no symptoms. In fairly heavy infections, children may show lack of appetite, abdominal pain and diarrhea.

8.2. Echinococcus



There are two different species. These are: *Echinococcus granulosus* and *Echinococcus multilocularis*

Echinococcus granulosus

(dog tape worm)

Responsible for most cases of echinococcosis. Echinococcosis is caused by larval tapeworms. The disease is common in East Africa (the highest prevalence is seen in Kenya: 10-15%).

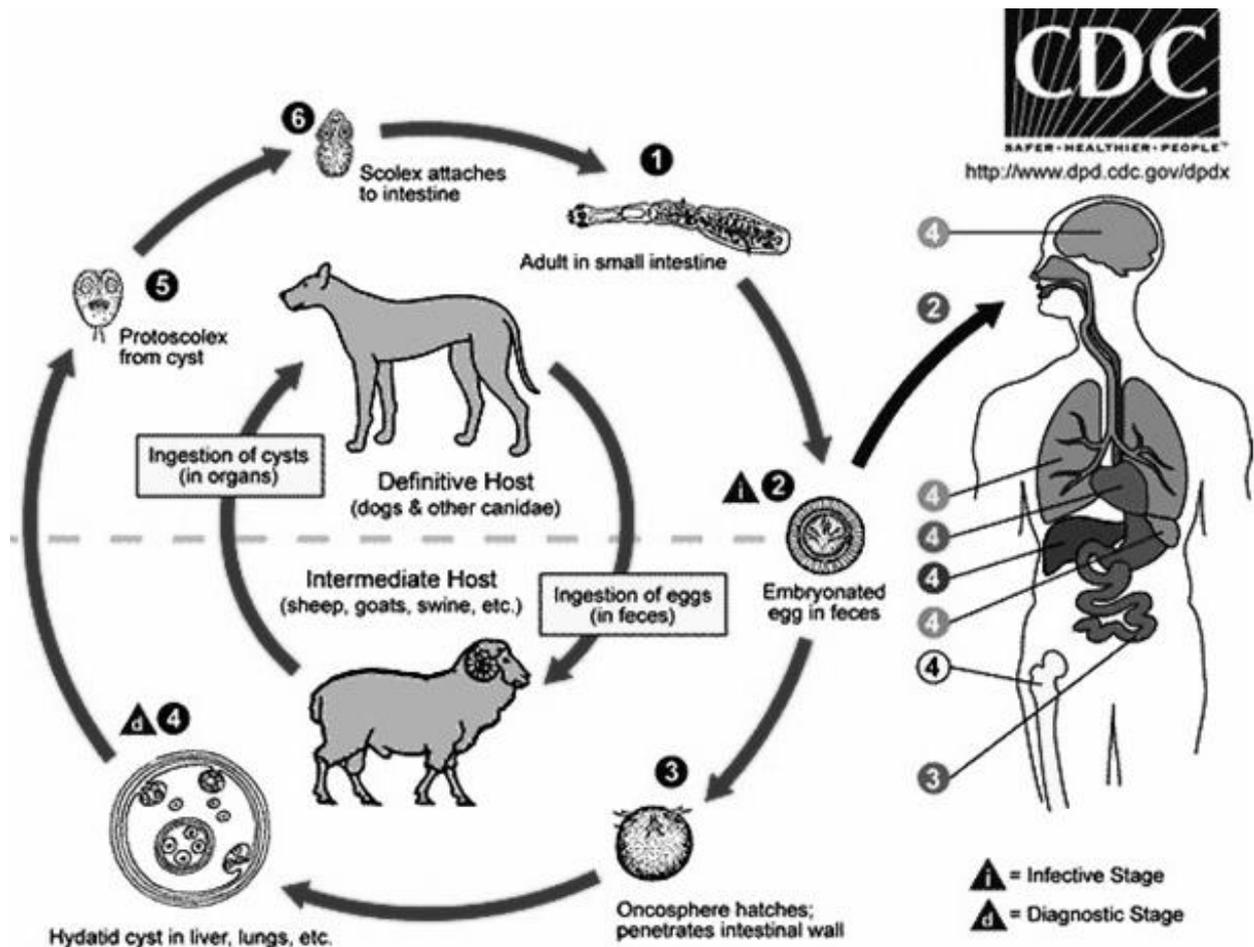
Morphology

The adult worm measures 3-6 mm in length (up to 1 cm). It has scolex, neck and strobilla. Adult worms live in small intestine of definitive host (dog). Man is an intermediate host – carrying the hydatid cyst (larva). Man contracts infection by swallowing eggs in excreta of definitive host.

Life cycle and Pathogenecity

Oncosphere hatch in duodenum or small intestine into embryos (oncosphere) which:

- penetrate wall
 - enter portal veins
 - migrate via portal blood supply to organs: eg: lungs, liver, brain etc., thus, causing extra intestinal infections. In these organs, larvae develop into hydatid cysts. The cysts may be large, filled with clear fluid and contain characteristic protoscolices (immature forms of the head of the parasite).
- These mature into developed scolices, which are infective for dogs.



Mode of human infection

Ingestion of eggs by the following ways:

- Ingestion of water or vegetables polluted by infected dog feces.
- Handling or caressing infected dogs where the hairs are usually contaminated with eggs.



Clinical features

Asymptomatic infection is common, but in symptomatic patients

- It may cause cough – with hemoptysis in lung hydatid disease.
- Hepatomegaly – with abdominal pain and discomfort.
- Pressure – from expanding cyst.
- Rupture of cyst – severe allergic reaction – anaphylaxis.

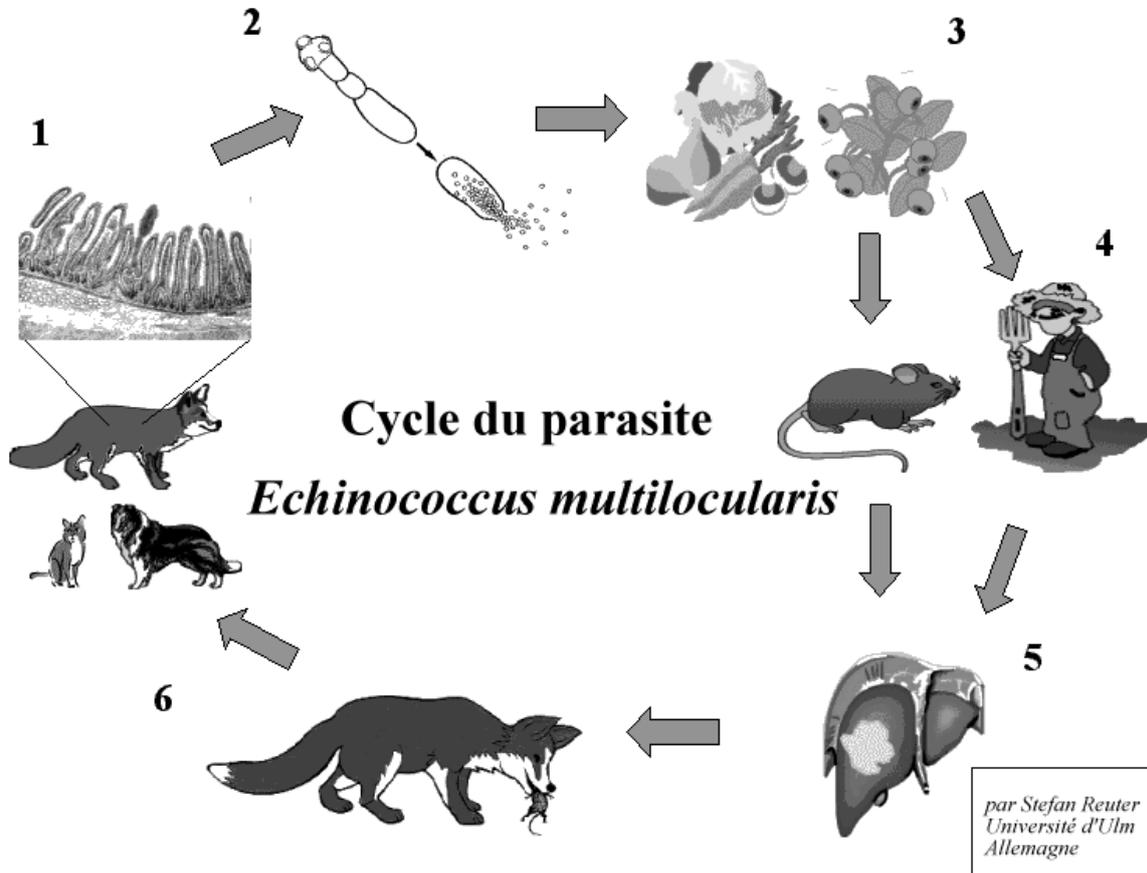


Diagnosis:

- X-ray or other body scans.
- Demonstration of protoscolices in cyst after operation.
- Serology.

Echinococcus multilocularis

Foxes are the definitive hosts, while various rodents such as mice serve as intermediate hosts.

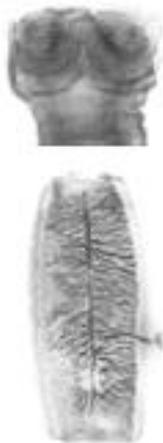


8.3. *Taenia saginata* (beef tapeworm)

In adult stage, *T. saginata* inhabits the upper jejunum where it may survive for as long as 25 years. It causes intestinal infection, Taeniasis. It has worldwide distribution. These are one of the true and segmented tapeworms. Their body is divided into three regions:

1. Scolex: the hold fast organ
2. Neck: posterior to the scolex
3. Stobilla: the main bulk, made up of proglottids.

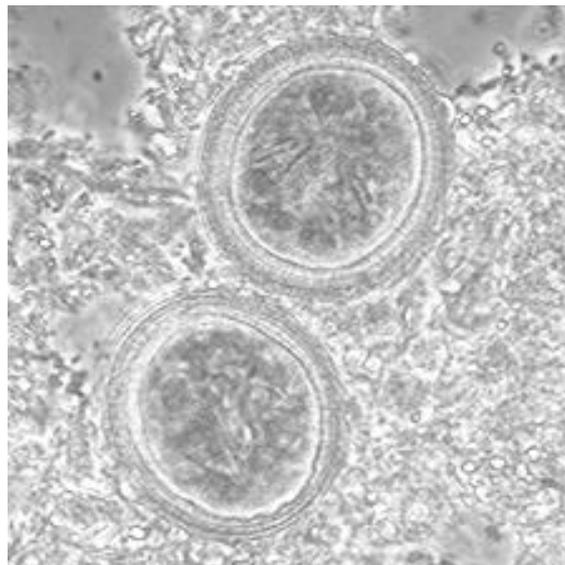
Taenia saginata



Taenia solium



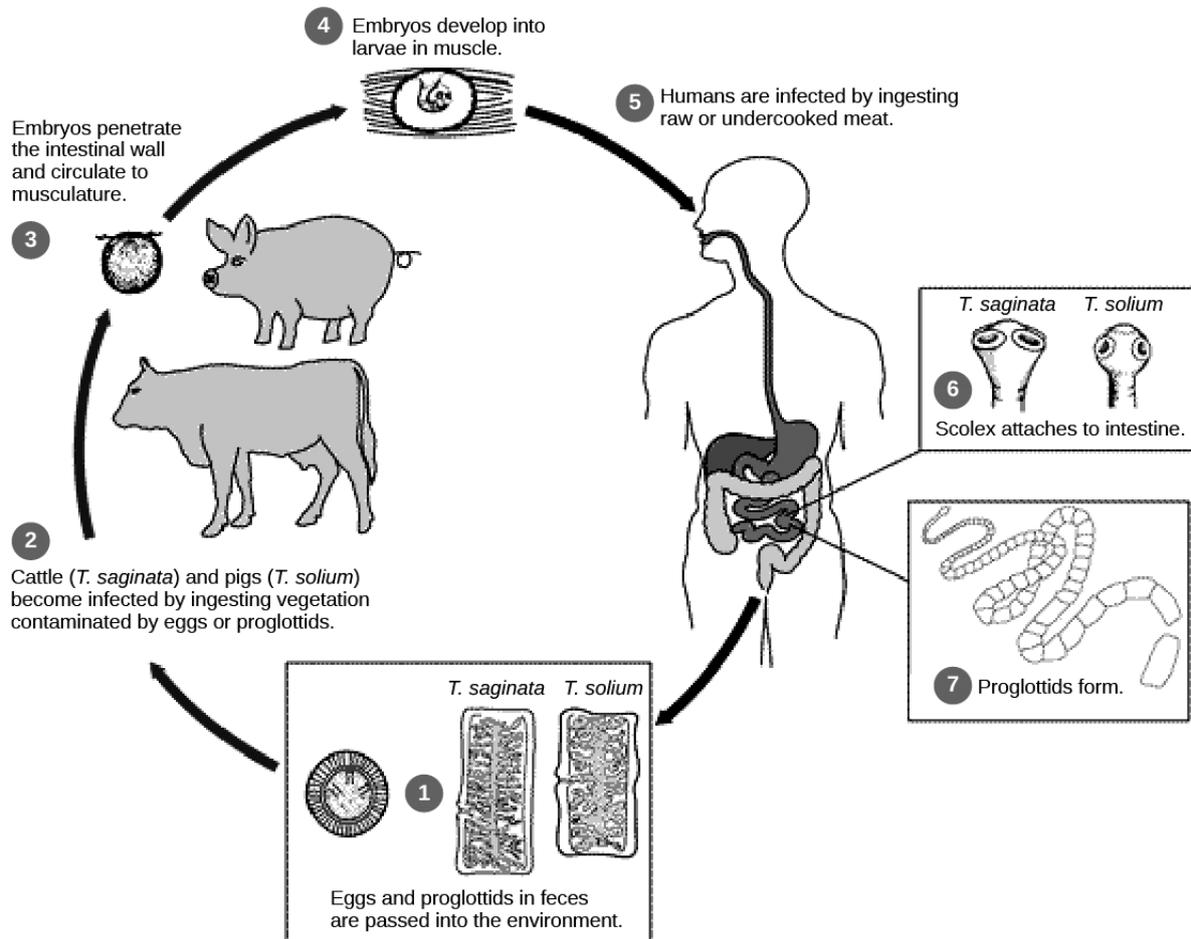
Adult worm measures 5-10 meters in length. The pyriform scolex has 4 suckers but no rostellum. The mature segments have irregularly alternate lateral genital pores. Each of the terminal segments contains only a uterus made up of a median stem with 15-30 lateral branches.



Life cycle

The adult worm lives in the small intestine of man. Gravid segments pass out in the stool and become disintegrated and eggs come out to the soil. The gravid proglottid uterus contains about 100,000 eggs. The egg of *T. saginata* is round, about 40 microns in diameter. The 6-hooked embryo is enclosed in a radially striated embryophore. Eggs are ingested by an intermediate host, cattle. The 6-hooked embryo escapes from its shell, penetrates through the intestinal

wall into the blood vessels and is carried to the muscles where it develops into a larval stage, cysticercus bovis (made up of an invaginated /inverted head and spherical body). Infection to man takes place by the ingestion of raw or insufficiently cooked beef. In the small intestine of man, the head of the cysticercus gets invaginated and the body becomes segmented.



Pathogenicity

Infected persons may complain of epigastric pain, abdominal discomfort, diarrhea, weight loss, hunger sensation, vomiting, etc.

Diagnosis

Recovery of the gravid segments or the eggs from the stool

Prevention:

- Thorough cooking of meat (above 570°C).
- Proper disposal of human excret

8.4. Taenia solium (pork tapeworm)

The adult worms of *T. solium* reside or inhabit the upper jejunum. Infection has worldwide distribution.

Morphology

Adult worm measures about 3 meters in length. The globular scolex has rostellum with 2 rows of hooklets. There are <1000 proglottids. Gravid proglottid liberates about 30,000-50,000 eggs.

Life cycle

Embryonated eggs passed with stool are ingested by pig and the embryo is released. It penetrates the intestinal wall and is carried by vascular channels to all parts of the body. After a period of 2-3 months of development the encysted larval stage called cysticerci or bladder worm occurs in the striated muscles of the tongue, neck, trunk brain, eye, and the nervous system. The cysticercus survives for 5 years. Humans become infected by eating pork containing larvae, *cysticercus cellulosae*. When improperly cooked cysticercus infected meat is eaten by man, the scolex remains undigested and attaches itself to the intestinal wall and chain of proglottids begin to grow to adult worm.

Clinical manifestations

Resembles that of *T. saginata* infection.

Diagnosis

Demonstration of eggs in stool specimen.

Prevention:

- Treatment of infected persons.
- Thorough cooking of pork and proper processing
- Proper disposal of human excreta (good hygiene/sanitation).

8.5. Diphyllotrium latum (fish tapeworm or broad tapeworm)

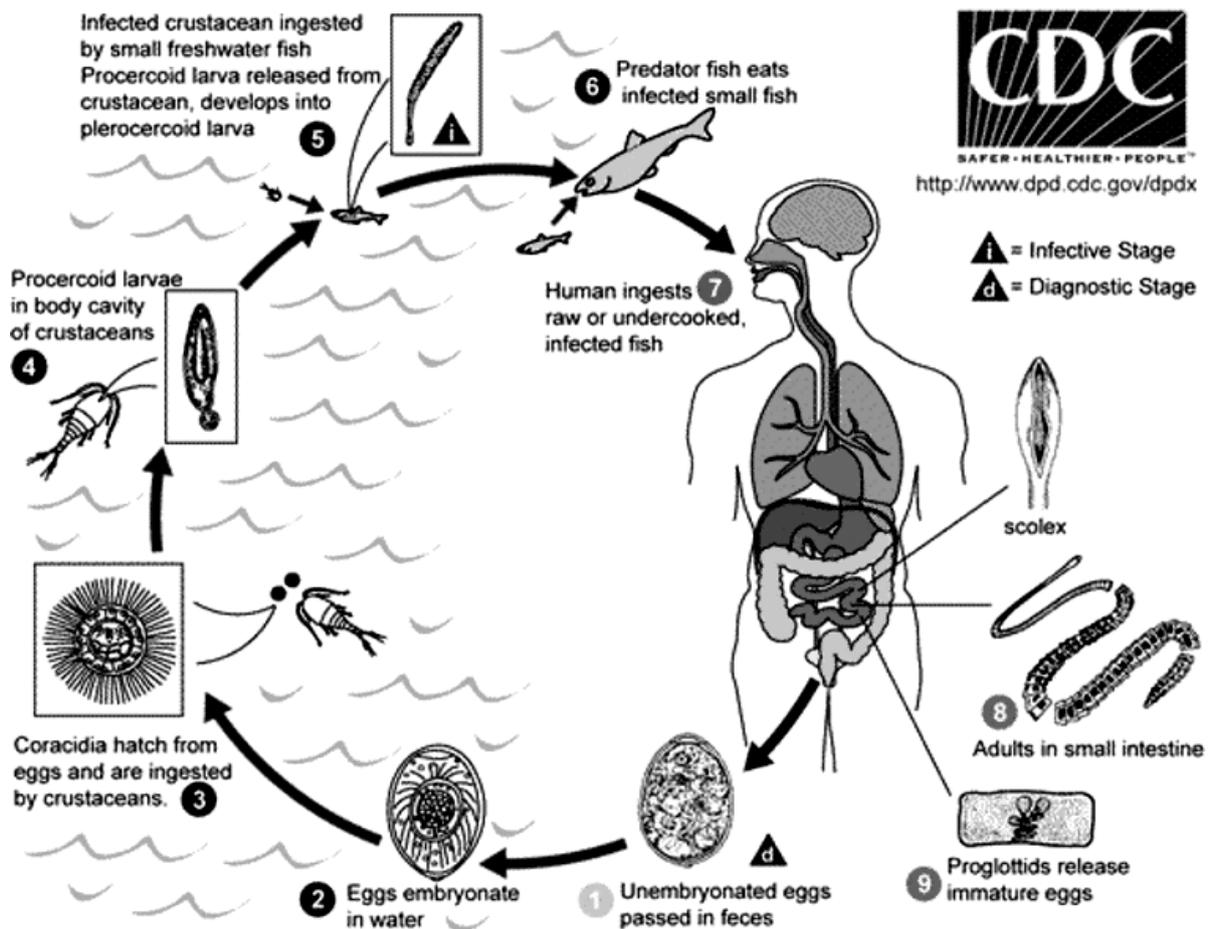
The broad tapeworm infecting man has worldwide distribution, occurring in areas where improperly cooked or raw fresh water fish is prominent in diet.

Morphology

Diphyllobotrium latum is the broadest and longest tapeworm. The adult worm measures up to 30 feet with 3000-4000 proglottids, which are wider than they are long. The tapeworm has no rostellum hooks or suckers.

Life cycle

Unlike *Taenia*, the gravid segments are retained by the worm. Operculated eggs passed in feces hatch into small ciliated coracidium larvae which swim about freely. These are eaten by crustaceans – Cyclops or Diaptomus – in which the larvae develop into second stage larvae- the proceroid. When the crustaceans are swallowed by fresh water fish, the larvae migrate into the flesh of the muscle fish and develop to plerocercoid or sparganum larvae. Humans are infected by ingesting raw or improperly cooked fish. The tapeworm matures in the intestine and after 3 weeks, the adult worm discharges eggs. The life cycle requires two intermediate hosts. So far there is no report of the parasite in Ethiopia.



Clinical manifestation

Most infections are asymptomatic. Rarely, it causes severe cramping, abdominal pain, vomiting, weakness and weight loss. Pernicious anemia can also result, due to interference of vitamin B12 absorption in jejunum.

Diagnosis

Eggs in stool: single shell with operculum at one end and a knob on the other.

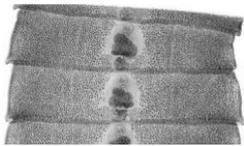
Prevention

Prohibiting the disposal of untreated sewage into fresh water /lakes.
Personal protection: cooking of all fresh water fish.

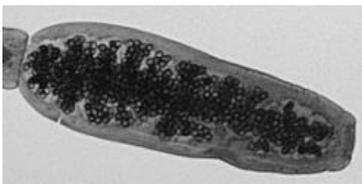
Testing your knowledge CESTODES (TAPEWORMS)

1. Tapeworms of cestodes do not have _____ system.
2. Adult worm of *HYMENOLEPIS NANA* is made up of _____, neck and _____.
3. The head of *HYMENOLEPIS NANA* carries _____ suckers and a rostellum armed with one row of _____.
4. Oncosphere of *HYMENOLEPIS NANA* penetrate the villi of the small intestine, where it forms _____.
5. The adult worm of *Echinococcus granulosus* measures _____ cm in length.
6. Definitive host of *Echinococcus granulosus* is _____.
7. Adult worms of *Echinococcus granulosus* live in _____ of definitive host.
8. Man is an _____ host of *Echinococcus granulosus* - carrying the hydatid cyst (larva).
9. Definitive host of *TAENIA SAGINATA* is/are _____.
10. Intermediate host of *TAENIA SAGINATA* is/are _____.

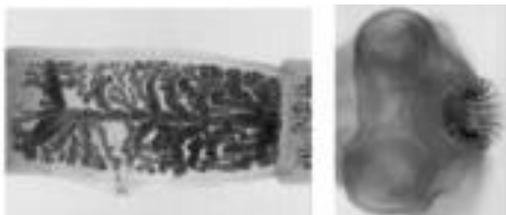
11. Each of the terminal segments of TAENIA SAGINATA contains only _____ made up of a median stem with 15-30 lateral branches.
12. 6-hooked embryo escapes from egg of TAENIA SAGINATA, penetrates through the intestinal wall into the blood vessels and is carried to the muscles where it develops into a larval stage, _____ (made up of an invaginated /inverted head and spherical body).
13. Humans become infected TAENIA SOLIUM by eating pork containing larvae – _____.
14. The globular scolex of TAENIA SOLIUM has rostellum with _____.
15. The first larvae of DIPHYLLOBOTRIUM LATUM which hatch from the eggs in the water is called _____.
16. The second larvae of DIPHYLLOBOTRIUM LATUM which develops in the Cyclops is called _____.
17. The second intermediate host of DIPHYLLOBOTRIUM LATUM is/are _____. The third larva that develops in it is called _____.
18. The figure shows the segment of _____.



19. The figure shows the segment of _____.



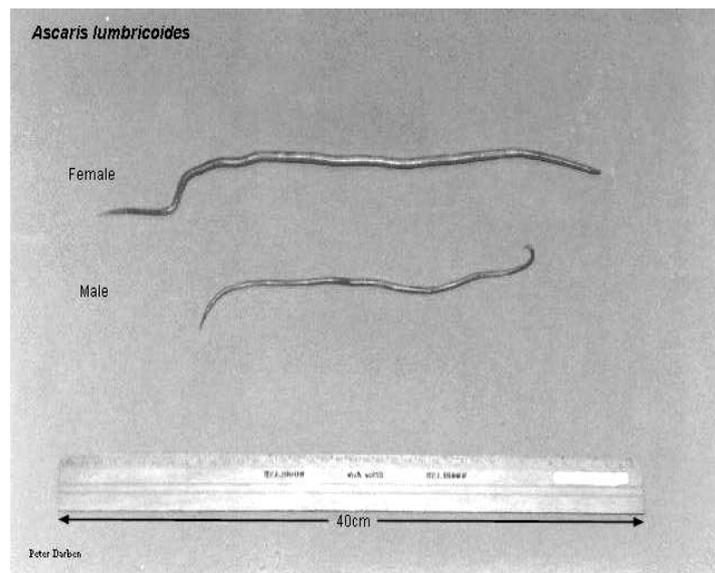
20. The figure shows the segment of _____.



UNIT 9. NEMATODES (TAPEWORMS)

9.1. General characters

1. Non-segmented cylindrical worms tapering at both ends.
2. Possess cuticle.
3. Sexes are separate (diecious), male is smaller than female & its posterior end is curved ventrally.
4. Females are either:
 - Viviparous (produce larvae/ embryos).
 - Oviparous (lay eggs) or
 - Ovo-viviparous (lay eggs which hatch immediately).
5. Live in intestinal tract or tissues.



Classification of Intestinal Nematodes

Small Intestine only:

- *Ascaris lumbricoides* (round worm);
- *Necator americanus* (american hook worm);
- *Ancylostoma duodenale* (hook worm);
- *Strongyloides stercoralis*;
- *Trichinella spiralis* (trichina worm);
- *Capillaria philippinensis*.

Caecum and Vermiform appendix:

- Enterobius vermicularis (pin worm);
- Trichuris trichiura (whip worm).

Lymphatic:

- Wuchereria bancrofti;
- Brugia timori.

Subcutaneous:

- Loa loa (african eye worm);
- Onchocerca volvulus (blinding filaria);
- Dracunculus medinensis (thread worm).

Conjunctiva:

- Loa loa.

Modes of infection of nematodes

1. Ingestion of:

- Embryonated eggs contaminating food and drinks, e.g. *A. lumbricoides*, *E. vermicularis* and *T. trichiura*
- Growing embryos in an intermediate host (infected cyclops) e.g. *D. medinensis*
- Encysted embryos in infected pig's flesh e.g. *Trichinella spiralis*
- Penetration of skin – filariform larvae bores through the skin e.g. *A. duodenale*, *S. stercoralis*, *N. americanus*

2. By blood sucking insects e.g. filarial worms

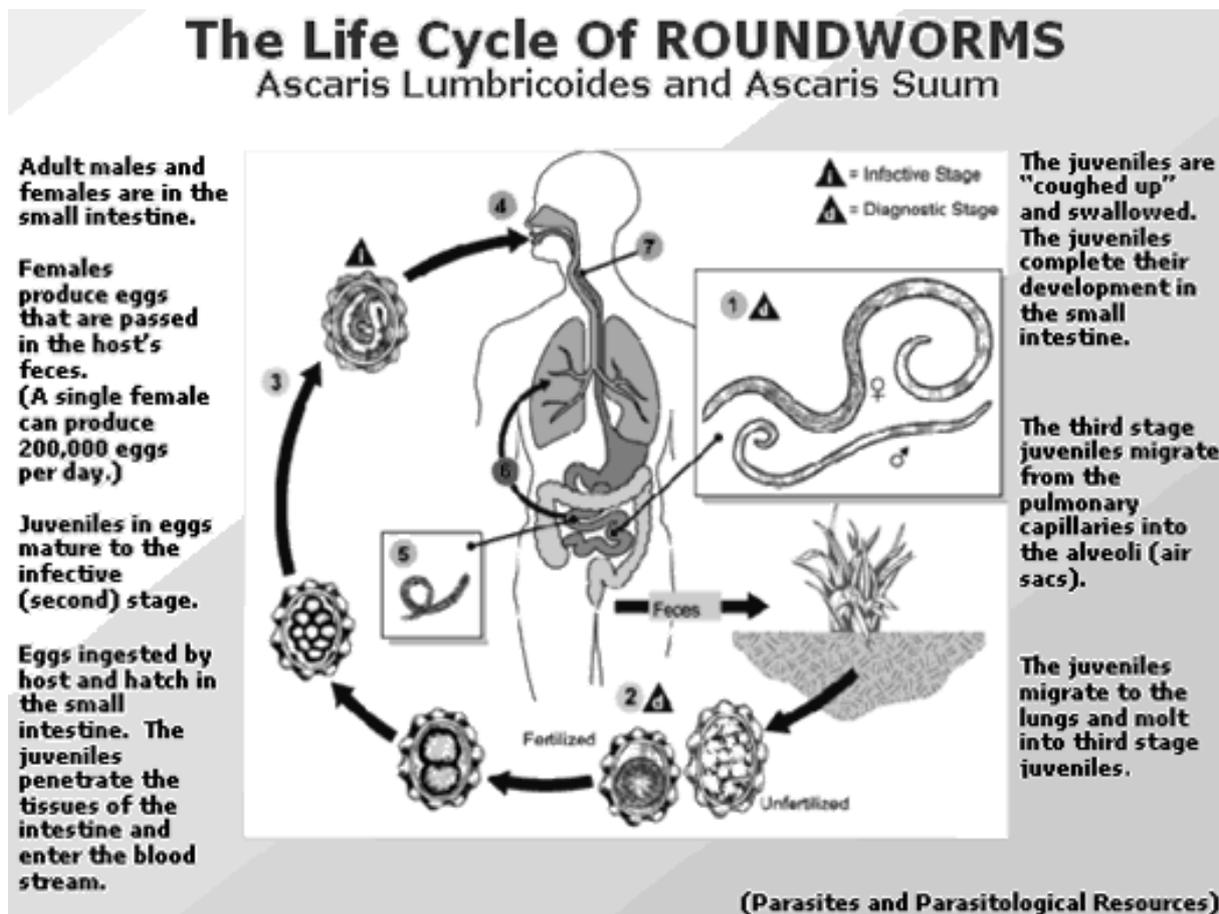
3. Inhalation of infected dust containing embryonated eggs e.g. *A. lumbricoides*, *E. vermicularis*

9.2. Ascaris lumbricoides (roundworm)

- Adult worms – Male 15 to 30 cms, Female 20 to 40 cms, oviparous.
- Eggs – 60 µ, bile stained, Albuminous coat with unsegmented ovum.
- Infective form – Embryonated eggs.
- Mode of transmission – Ingestion.
- Site of localization – Small intestine.



Egg of *A. lumbricoides*

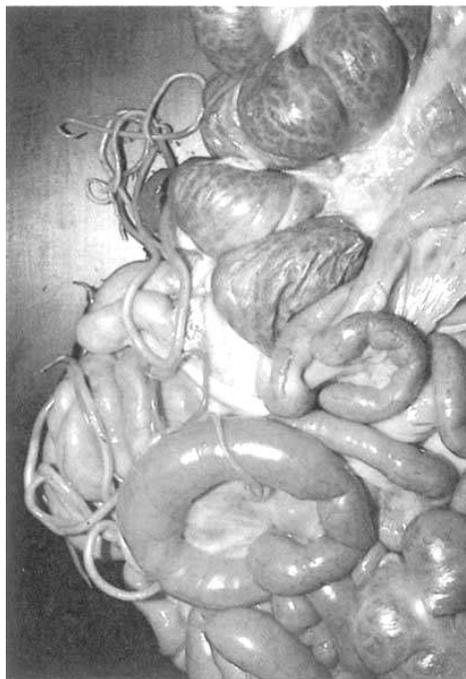


Pathogenicity and clinical features

- Ascariasis – infection of *A. lumbricoides*
- Majority of infections are asymptomatic
- Clinical disease is largely restricted to individuals with a high worm load
- Symptoms divided into two groups: those produced by
 1. Migrating larvae
 2. Adult worms

Symptoms and complications

1. Symptoms produced by Migrating larvae
 - Pneumonia (loeffler's syndrome) – fever, cough, dyspnoea, blood tinged sputum that may contain larva, urticarial rash & eosinophilia
 - Visceral larva migrans – if larvae enter systemic circulation (from pulmonary capillaries) to reach other organs like brain, spinal cord, heart, kidney.
2. Symptoms produced by Adult worms
 - Abdominal discomfort, anorexia, nausea & diarrhoea.
 - PEM, Vit. A deficiency (night blindness)
 - Intestinal obstruction (particularly in children 1-5 years), intussusception & volvulus
 - Penetration through intestinal ulcer (perforation) – peritonitis
 - Hypersensitivity reactions to worm Ags (toxic body fluids) – urticaria, edema of face, conjunctivitis, irritation of URT



Pathogenicity and clinical features

1. Symptoms produced by Adult worms:
Ectopic Ascariasis – due to migration of worm up into the stomach. It may
 - be vomited out,
 - pass up through the oesophagus at night & comes out through mouth or nose,

- enter larynx to cause asphyxia,
- migrate to other organs and cause appendicitis, cholecystitis, biliary colic, cholangitis, pancreatitis

Laboratory diagnosis

1. Macroscopic – Direct detection of worm/s in stool or vomit
2. Microscopic – direct examination of feces following floatation method: bile stained eggs. (eggs may not be seen at least 40 days after infection)
3. Blood examination – eosinophilia.

Other modes of diagnosis

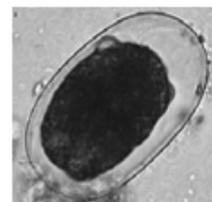
4. Imaging – large collections of worms in abdomen
5. USG – to diagnose hepatobiliary or pancreatic ascariasis
6. Serology (Ab detection) – mainly reserved for epidemiological studies.

Prevention

1. Good sanitation and personal hygiene
2. Mass treatments with single dose mebendazole or albendazole for all school-age children every three to four months – serves dual function:
 - treats the children and
 - reduces the overall worm burden in the community

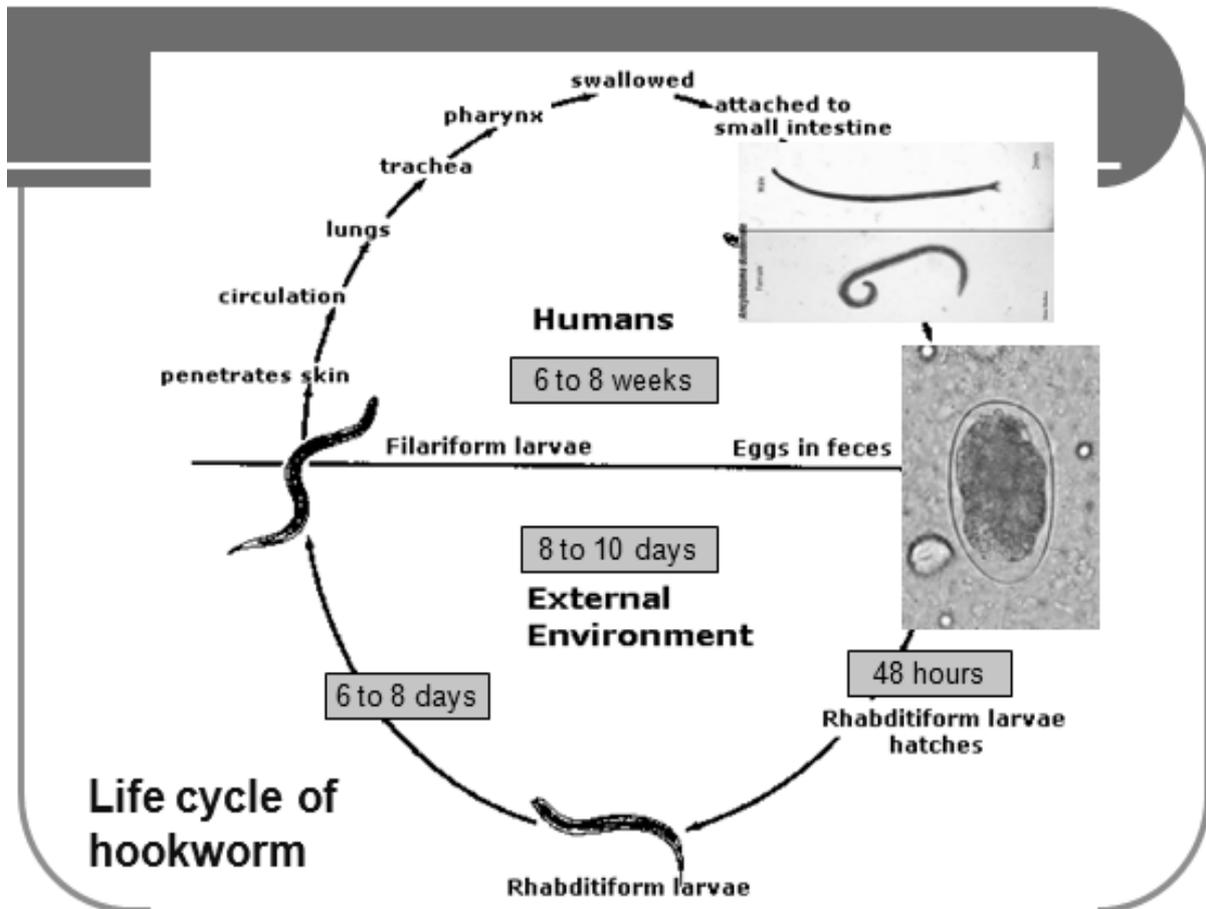
9.3. Ancylostoma duodenale (hook worm)

Adult worms	Male 8 -11mm Female 10-13 mm, oviparous
Eggs	60 μ , non bile stained (colorless) Segmented, 4 blastomeres
Infective form	3 rd stage filariform larva
Mode of infection	Penetration into skin
Site of localization	Small intestine

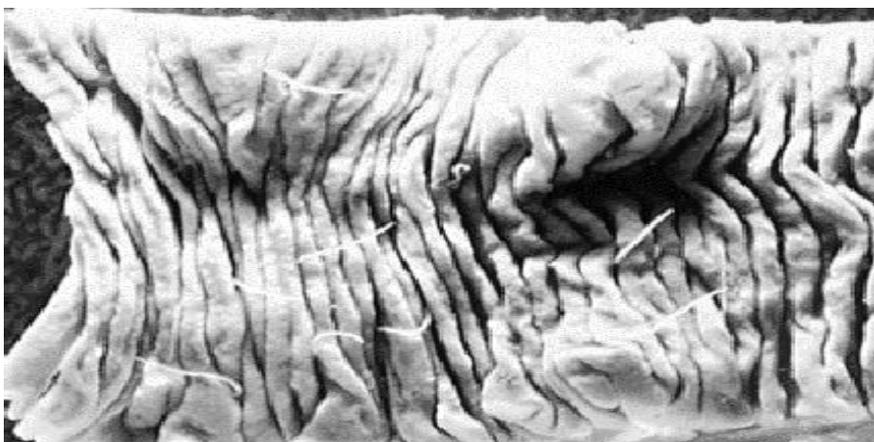


Sites of skin penetration:

1. Thin skin between toes
2. Dorsum of the feet
3. Inner side of the soles
4. Gardeners & miners – skin of hands



Hook worms in the intestine



Pathogenicity and clinical features

- Ancylostomiasis or hookworm disease, characterised by iron deficiency anaemia;
- Majority of infections are asymptomatic;
- Symptoms develop in heavy infections and divided into two groups: those produced by:
 1. Migrating larvae;
 2. Adult worms.

Symptoms produced by larvae

Lesions in the skin:

1. Ancylostome dermatitis or Ground itch – occurs at the site of entry (more common in necator), lasts for 2 to 4 weeks
2. Creeping eruption – reddish itchy papule along the path traversed by filariform larvae (larva migrans)
3. Lesions in the lungs – bronchitis & bronchopneumonia.



Symptoms produced by adult worm

1. Epigastric pain, diarrhoea & vomiting during early phase of infection.
2. Microcytic hypochromic (Iron deficiency) anaemia – due to chronic blood loss:
 - a single adult hookworm sucks 0.2ml of blood/ day
 - Hemorrhages from punctured sites

Clinical features of hookworm anemia

- Extreme pallor
- Abnormal appetite showing Pica or Geophagy – perverted taste for earth, mud or lime
- Epigastric tenderness with dyspepsia

- Constipation
- Puffy face with swelling of lower eyelids
- Pedal edema
- Growth retardation
- General appearance – pale plumpy with protuberant abdomen & dry lustreless hair.



Laboratory diagnosis

- Stool examination – microscopy: non bile stained egg, segmented
- Occult blood in stool – positive
- Blood examination – anaemia, eosinophilia

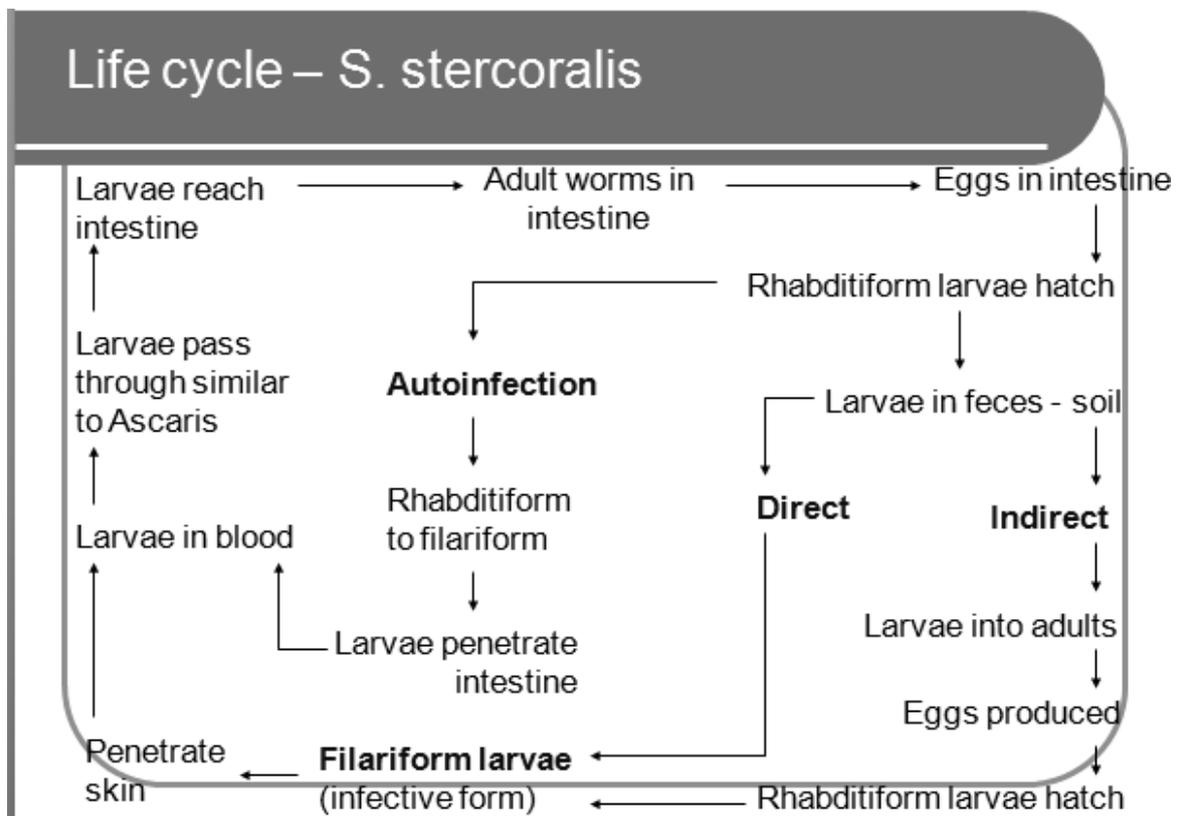


Prevention and control

- Proper sanitation measures & sewage disposal
- Personal hygiene
- Personal protection – wearing boots & gloves
- Simultaneous treatment of carriers & diseased with wholesale treatment of community

9.4. Strongyloides stercoralis

Adult worms	2 - 2.5mm, ovoviviparous, eggs laid in the tissues
Free living worms	Moist soil
Infective form	Filariform larvae 
Mode of transmission	Penetration / autoinfection
Site of localization	Wall of Small intestine, mainly duodenum & jejunum



Pathogenicity

1. Skin lesions (2 types) – “larva currens”
 - At the site of entry – urticarial rash
 - In the perianal region – linear, erythematous urticarial wheal

2. Pulmonary lesions – due to migrating larva

- Alveolar hemorrhages
- Bronchopneumonia

3. Intestinal lesions – “burrowing lesions”

- Epigastric pain
- Diarrhoea with blood & mucus
- Nausea
- Weight loss

Laboratory diagnosis

- Stool examination – rhabditiform larva
- Culture – larva
- ELISA – to detect Abs



Treatment and prevention

- Potentially life threatening disease – treat even if its asymptomatic
- Thiabendazole for 2 days
Disseminated strongyloidosis – 5 to 7 days.

9.5. *Trichinella spiralis* (trichina worm)

Adult worms (smallest nematode infecting man)

Male 1.4 – 1.6 mm
Female 3 - 4 mm, viviparous

Infective form

Encysted larvae (100 μ) in striated muscles of pig

Mode of transmission

Ingestion of improperly cooked pork

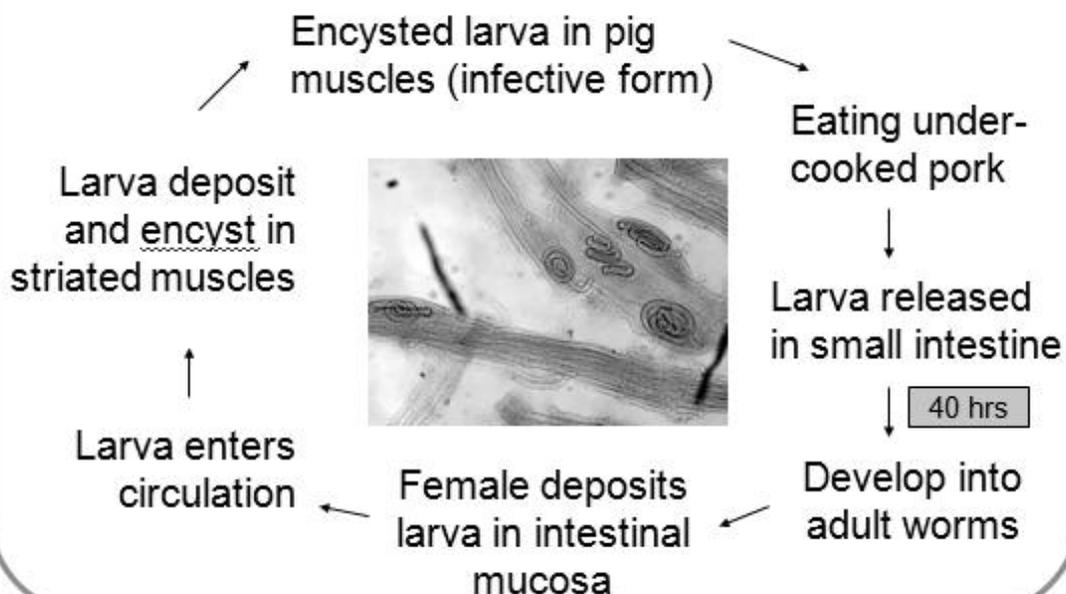
Site of localization

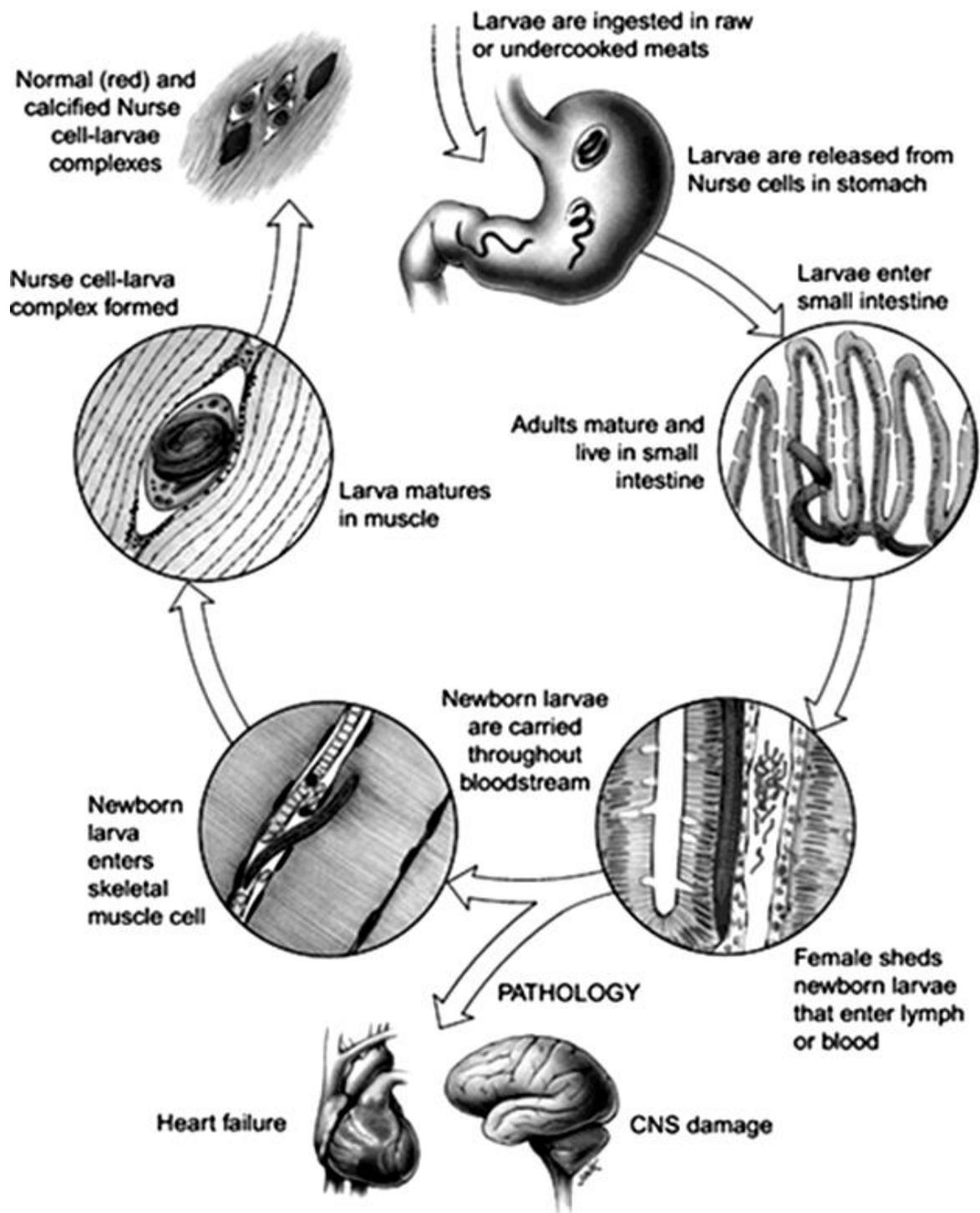
Small intestine

Commonly involved muscles

Diaphragm, Intercostals, Deltoid
Pectoralis major, Biceps

Life Cycle – *T spiralis*



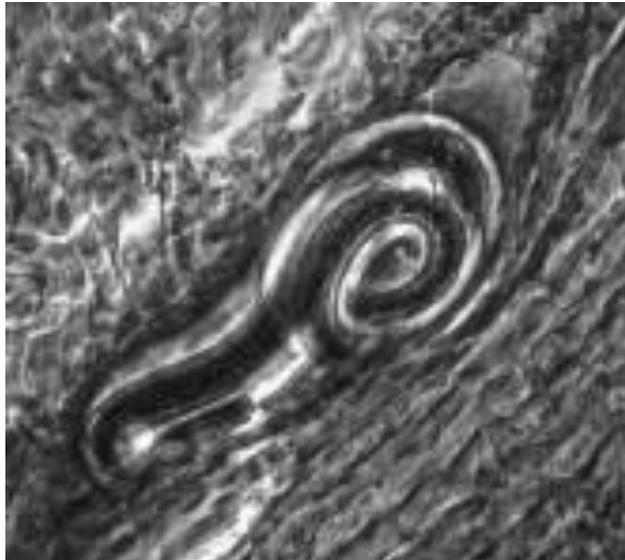


Pathogenicity

1. Trichinelliasis / Trichinosis – clinical features depends on the stage:
 - Stage of intestinal invasion: 5-7 days, pain in abdomen, nausea, vomiting, diarrhoea
 - Stage of larval migration: fever, urticarial rash, splinter hemorrhages, periorbital & facial edema
 - Stage of encystation: asymptomatic in light infections; myalgia, weakness in heavy infections
2. Complications – during migration:
 - myocarditis, encephalitis.

Laboratory diagnosis

1. Muscle biopsy – encysted larva
2. Blood – eosinophilia between 2nd and 4th week
3. Serology – to detect specific Abs by:
 - Bentonite flocculation test
 - Latex agglutination test



Prevention

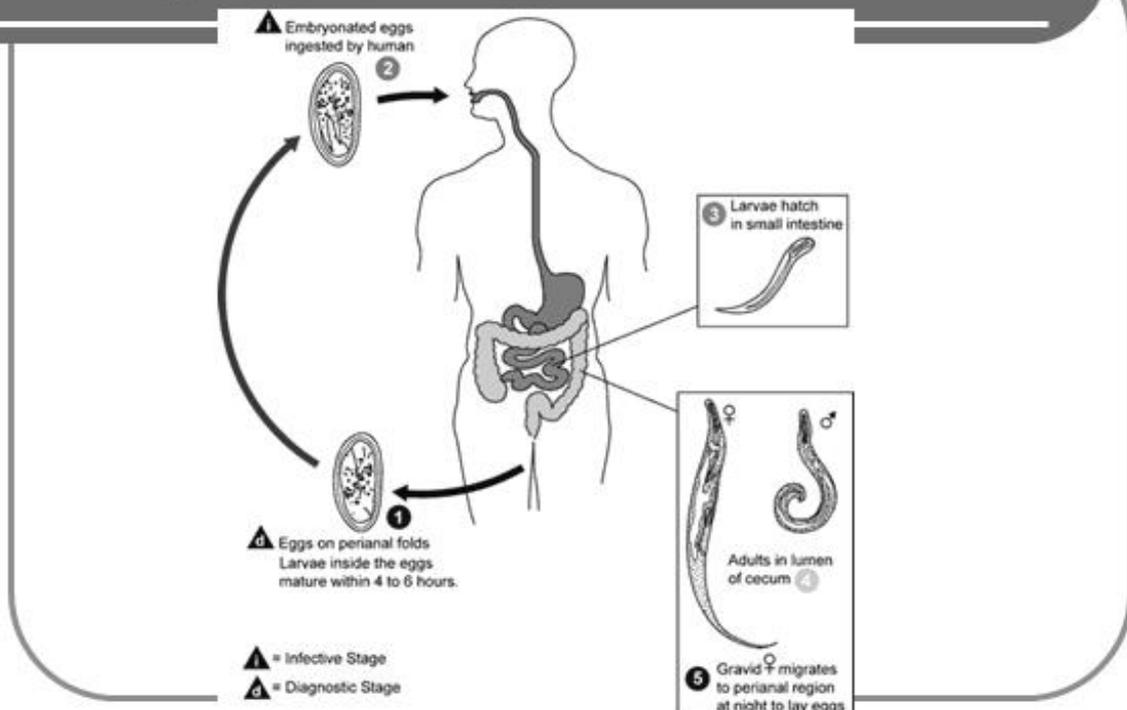
- Proper cooking of pork or proper storage
- Avoidance of feeding bits and refuse from slaughter houses and farms to pigs – breaks life cycle.

9.6. Enterobius vermicularis (pin worm, seatworm)

Adult worms	Male 2 - 5 mm Female 8 -13 mm, oviparous
Eggs	60 μ , non bile stained Plano-convex with coiled embryo
Infective form	Embryonated egg
Mode of transmission	Ingestion, Autoinfection
Site of localization	Large intestine – caecum & appendix



Life cycle – *E. vermicularis*

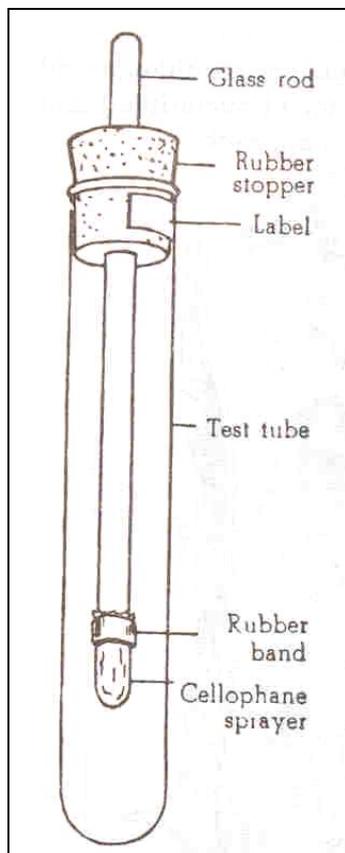
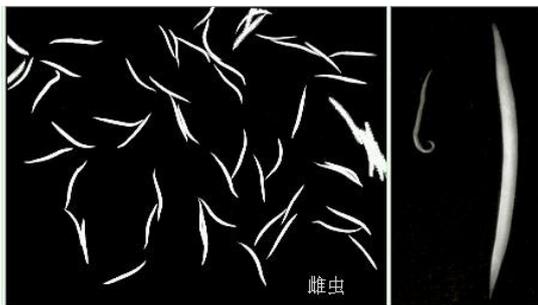


Clinical features

- Due to migration of worm - Perianal, perineal & vaginal itching (pruritis) worsens at night.
- Insomnia and restlessness
- Nocturnal enuresis

Laboratory diagnosis and treatment

- Detection of adult worms in feces or perianal region
- NIH swab – scrapings from perianal region
- Microscopy – non bile stained eggs
- Mebendazole, pyrantel pamoate



9.7. *Trichuris trichiura* (whio worm)

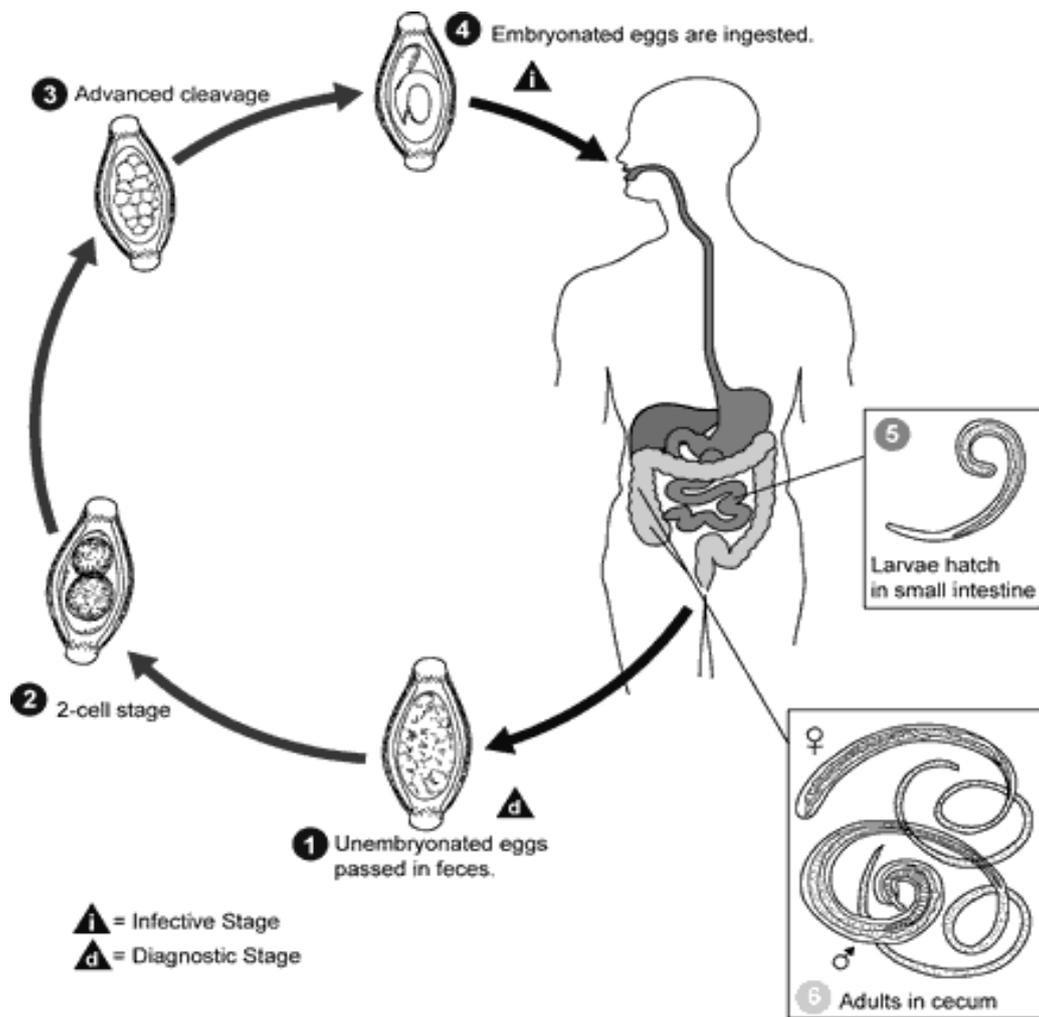
<i>Trichuris trichiura</i> (Whip Worm)		
Adult worm	30 – 50 mm	
Eggs	60 μ, bile stained Barrel-shaped with Mucus plug at each pole Unsegmented ovum	
Infective form	Mature embryonated eggs	
Mode of transmission	Ingestion	
Site of localization	Large intestine - caecum	

Clinical features

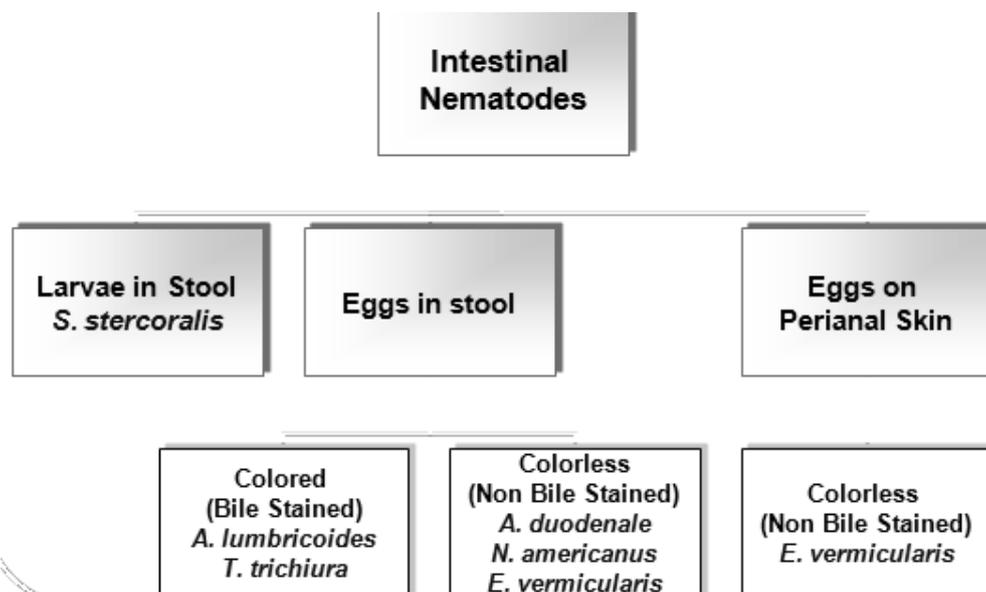
1. Infection – Trichuriasis
2. Symptoms depend on worm burden
3. Less than 10 worms – asymptomatic
4. Heavier infections –
 - chronic profuse mucus and bloody diarrhea with abdominal pains and edematous rectum
 - malnutrition, weight loss and anemia

Laboratory diagnosis and treatment

1. Stool examination – bile stained eggs with bipolar mucus plugs
2. Treatment – albendazole / mebendazole
3. Prevention:
 - Proper disposal of night soil
 - Prevention of consumption of uncooked vegetables & fruits.



DIAGNOSIS OF INTESTINAL NEMATODES



9.8. Filariae

1. The nematode genera of the superfamily Filarioidea will be here under the collective term *filariae*, and the diseases they cause are designated as *Filarioses*.
2. In the life cycle of filariae infecting humans,
3. *insects (mosquitoes, blackflies, flies etc.)*
4. *function as intermediate hosts and vectors.*
5. Filarioses are endemic in subtropical and
6. tropical regions; in other regions they are
7. observed as occasional imported cases.

The most important filariosis is ***onchocercosis***, the causative agents is *Onchocerca volvulus*, is transmitted by blackflies. Microfilariae of this species can cause severe skin lesions and eye damage, even blindness. Diagnosis of onchocercosis is based on clinical symptoms, detection of microfilariae in the skin and eyes, as well as on serum antibody detection.

Other forms of filarioses include lymphatic filariasis .The causative agent is *Wuchereria bancrofti*, *Brugia species* and *Loa loa*. *Loa loa*. *Dirofilaria* species from animals can cause lung and skin lesions in humans.



Filariae are threadlike nematodes. The length of the adult stages of the species that infect humans varies between 2-50 cm whereby the females are larger than the males. The females release embryonated eggs or larvae called microfilariae. The eggs are about 0.2-0.3 mm long, surrounded by an extended eggshell.

They can be detected in the skin or in blood. Based on the periodic appearance of microfilariae in peripheral blood, periodic filaria species are differentiated from the nonperiodic ones showing continuous presence.

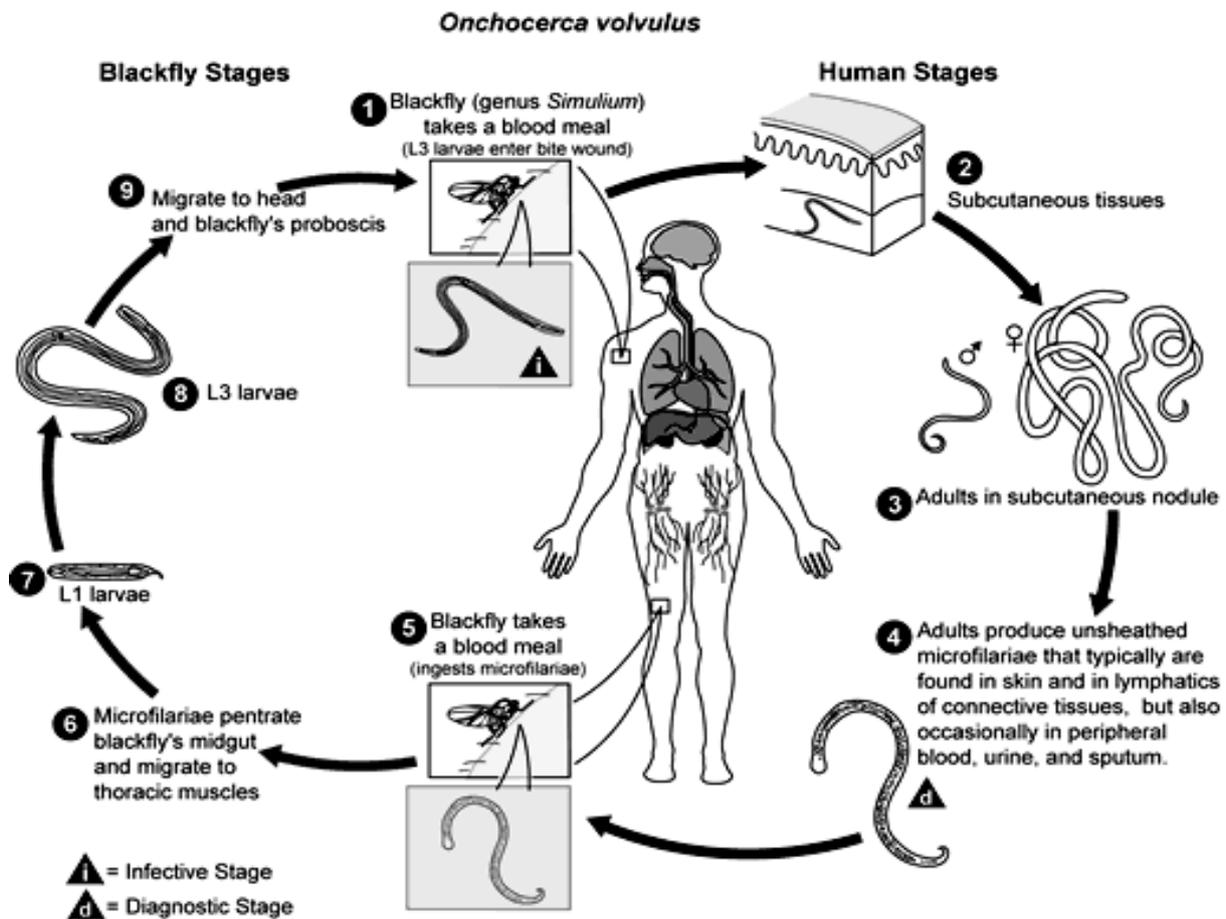
The periodic species produce maximum microfilaria densities either at night (nocturnal periodic) or during the day (diurnal periodic).

Different insect species, active during the day or night, function as intermediate hosts accordingly to match these changing levels of microfilariaemia.

Insect:

- **Ingestion of microfilaria with a blood meal**
↓
- **development in thoracic musculature with two moltings to become infective larva**
↓
- **migration to mouth parts and transmission into skin of a new host through puncture wound during the next blood meal.**

Human: migration to definitive localizations and further development with two more moltings to reach sexual maturity.

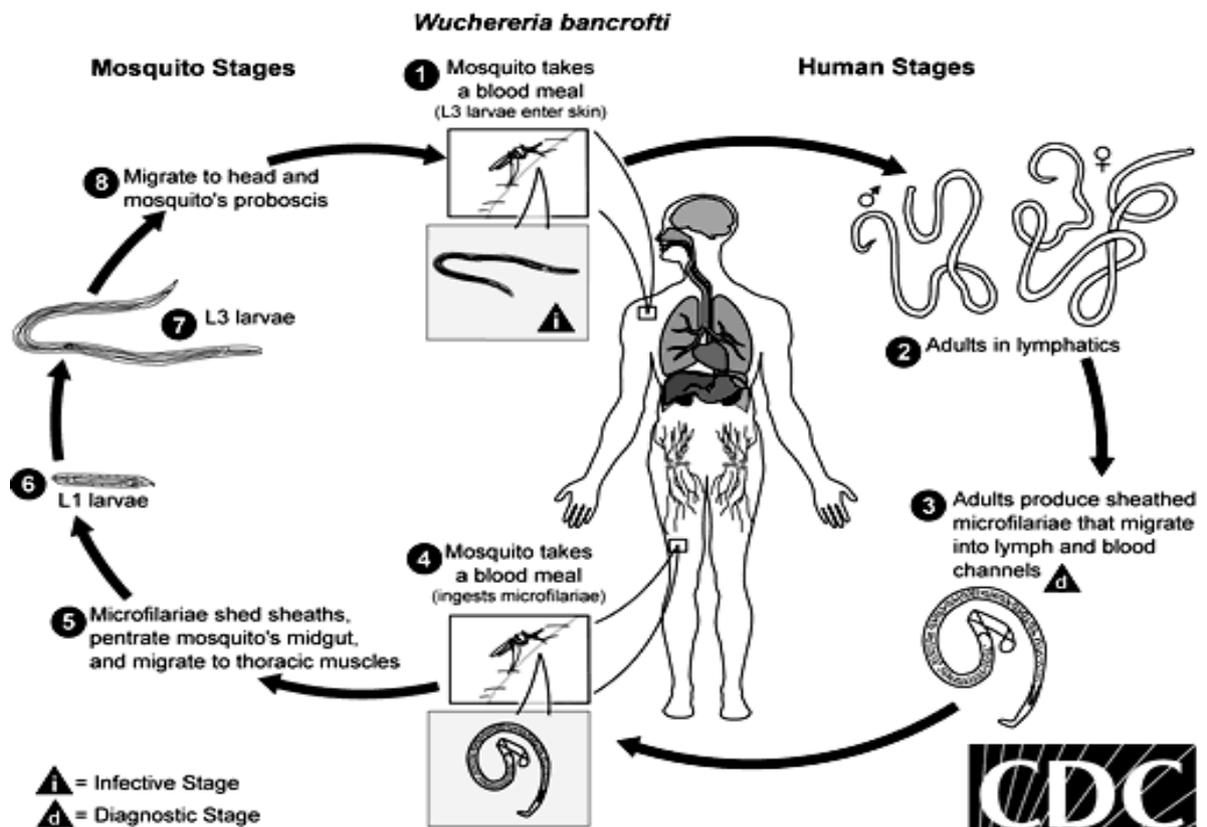


***Wuchereria bancrofti* causative agents of lymphatic filariasis**

About 120 million people in 80 countries suffer from *lymphatic filariasis* caused by *Wuchereria bancrofti* or *Brugia* species (One-third each in India and Africa, the rest in southern Asia, Pacific region, and South America).

Life cycle and epidemiology

- Humans are the only natural final hosts of *W. bancrofti*.
- The intermediate hosts of *W. bancrofti* are various diurnal or nocturnal mosquito genera.
- The development of infective larvae in the insects is only possible at high environmental temperatures and humidity levels;
- In *Wuchereria bancrofti* the process takes about 12 days at 28 °C.



Following a primary human infection, the filariae migrate into lymphatic vessels where they develop to sexual maturity.

Microfilariae appear in the blood after seven to eight months.

Pathogenesis and clinical manifestations

The initial symptoms can appear as early as 1 month, although in most cases the incubation period is 5 to 12 months or much longer.

Asymptomatic infection, is with microfilaremia that can persist for years.

Acute symptomatic infection:

Inflammatory and allergic reactions in the lymphatic system caused by filariae

Swelling of lymph nodes, lymphangitis, general malaise, swellings on legs, arms, scrotum, orchitis.

Chronic symptomatic infection

Chronic obstructive changes in the lymphatic system hindrance or blockage of the flow of lymph and dilatation of the lymphatic vessels (“lymphatic varices”). Indurated swellings caused by connective tissue proliferation in lymph nodes, extremities (especially the legs, “elephantiasis”), the scrotum. thickened skin, lymphuria, when lymph vessels rupture. This clinical picture develops gradually in indigenous inhabitants over a period of 10–15 years after the acute phase, in immigrants usually faster.

Tropical, pulmonary eosinophilia

Syndrome with coughing, asthmatic pulmonary symptoms, high-level blood eosinophilia, lymph node swelling and high concentrations of serum antibodies (*including IgE*) to filarial antigens. No microfilariae are detectable in blood, but sometimes in the lymph nodes and lungs. This is an allergic reaction to filarial antigens



1. A diagnosis can be based on clinical symptoms (frequent eosinophilia)
2. Finding of microfilariae in blood (sampling at night for nocturnal periodic species).
3. Microfilariae of the various species can be differentiated morphologically in stained blood smears and by DNA analysis.
4. Detection by ultrasonography, particularly in the male scrotal area.
5. Detection of serum antibodies (group-specific antibodies, specific IgE and IgG subclasses) and circulating antigens
6. The recent development of a specific ELISA and a simple quick test (the ICT filariasis card test is useful for serology).

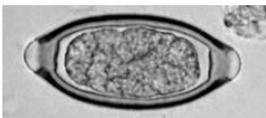
Therapy

Both albendazole and diethylcarbamazine have been shown to be at least partially effective against adult filarial stages. However, optimal treatment regimens still need to be defined.

Testing your knowledge

NEMATODES

1. *Ascaris lumbricoides* is localized in the _____ of man.
2. The infecting larvae in the egg of *Ascaris lumbricoides* develops in the soil within _____.
3. Definitive host of *Ascaris lumbricoides* is/are _____.
4. Migration on blood flow through the heart and lungs is typical for the larvae of _____, _____, _____.
5. The female of _____ comes out of the anus and lay eggs in the folds of the perineum.
6. Adult worms of *Ancylostoma duodenale* is localized in _____.
7. Filariform larvae of *Ancylostoma duodenale* penetrate the human body through _____.
8. Rabditioform larvae of _____ can produce a generation of free-living worms.
9. Intermediate host of *Enterobius vermicularis* is/are _____.
10. The intermediate hosts of *Wuchereria bancrofti* are _____.
11. Site of localization of _____ is large intestine – caecum.
12. The picture presented egg of _____.



13. The picture presented egg of _____.



14. The picture presented egg of _____.



15. Human is infested *Trichinella* eating _____.
16. The larva of *Trichinella* forms a capsule in meat, and can survive up to _____ days/months/years.
17. Adult worms of _____ are located in the lymph nodes, disrupting the flow of lymph.
18. Adult worms of *Trichuris trichiura* are located in _____.
19. Adult worms of *Strongyloides stercoralis* is localized in _____.
20. Rhabditiform larvae of *Strongyloides stercoralis* develops in _____ larvae which penetrate the human body through the skin.

List of recommended literature:

1. Cambell N. A. Biology / N. A. Cambell et al. // Benjamin Cummings. – 2013. – 1484 p.
2. Raven P. H. Biology / P. H. Raven, G. B. Johnson, K. A. Mason // MGH. – 2002. – 1239 p.

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GENERAL BIOLOGY

TRAINING TOOLKIT

PART B

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