# MINISTRY OF SCIENCE AND HIGHER EDUCATION OF THE RUSSIAN FEDERATION Federal State Autonomous Institution of Higher Education "National Research Lobachevsky State University of Nizhny Novgorod"

Institute of Biology and Biomedicine

T.V. Lavrova

# **Biology**Part 1. **Parasitology**: otozoans and flat worms

protozoans and flat worms
Lecture notes

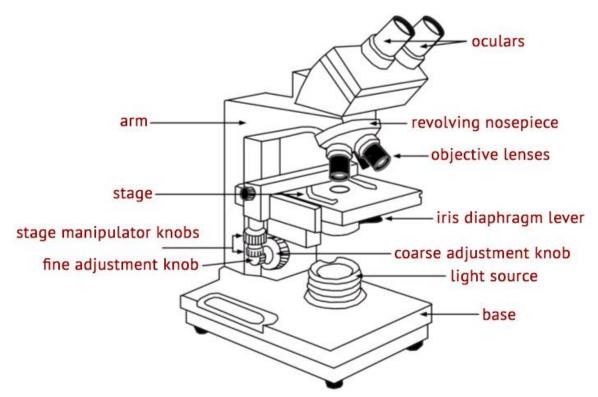
Recomended for students of Specialist's Degree Programme "General Medicine"

Nizhny Novgorod 2019

# Introduction

This lecture note is useful to students of health science, medicine and other students and academicians. It is believed to provide basic knowledge to students on medical parasitology. It also serves as a good reference to parasitologists, graduate students, biomedical personnel, and health professionals. It aims at introducing general aspects of medically important parasites.

Students preparing to provide health care in their profession need solid foundation of basic scientific knowledge of etiologic agents of diseases, their diagnosis and management. To face the fast growing trends of scientific information, students require getting education relevant to what they will be doing in their future professional lives.



Parts of the light microscope

#### **Basic definitions**

**A PARASITE** is a living organism, which takes its nourishment and other needs from a host; **the HOST** is an organism which supports the parasite.

#### DIFFERENT KINDS OF PARASITES

**Ectoparasite** – a parasitic organism that lives on the outer surface of its host.

**Endoparasites** – parasites that live inside the body of their host.

<u>Obligate Parasite</u> - This parasite is completely dependent on the host during a segment or all of its life cycle.

<u>Facultative parasite</u> – an organism that exhibits both parasitic and non-parasitic modes of living and hence does not absolutely depend on the parasitic way of life, but is capable of adapting to it if placed on a host.

<u>Accidental parasite</u> – when a parasite attacks an unnatural host and survives. <u>Erratic parasite</u> - is one that wanders in to an organ in which it is not usually found.

#### **DIFFERENT KINDS OF HOSTS**

<u>Definitive host</u> – a host that harbors a parasite in the adult stage or where the parasite undergoes a sexual method of reproduction.

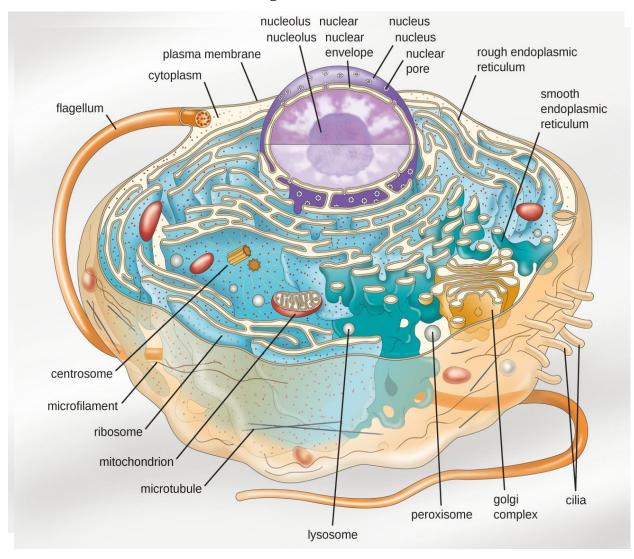
<u>Intermediate host</u> - harbors the larval stages of the parasite or an asexual cycle of development takes place. In some cases, larval development is completed in two different intermediate hosts, referred to as first and second intermediate hosts.

<u>Paratenic host</u> – a host that serves as a temporary refuge and vehicle for reaching an obligatory host, usually the definitive host, i.e. it is not necessary for the completion of the parasites life cycle.

<u>Reservoir host</u> – a host that makes the parasite available for the transmission to another host and is usually not affected by the infection.

<u>Natural host</u> – a host that is naturally infected with certain species of parasite. <u>Accidental host</u> – a host that is under normal circumstances not infected with the rasite.

# **Kingdom Protista**



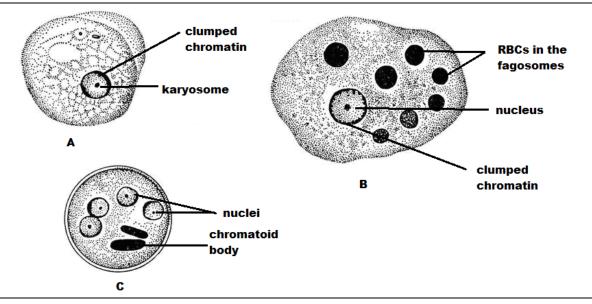
General characteristic: organelles of eucariotic cell						
Organelle	Function					
Membrane	Separation and transport control					
Membrane bound organelles						
Nucleus	DNA Storage					
Mitochondrion	Energy production					
Smooth Endoplasmic Reticulum (SER)	Lipid production; Detoxification					
Rough Endoplasmic Reticulum (RER)	Protein production; in particular for export out of the cell					
Golgi apparatus	Protein modification and export					
Peroxisome	Lipid Destruction; contains oxidative enzymes					
Lysosome	Protein destruction					
	Cytoskeleton					
Microtubules Cilia and fl	lagella movement, pathways for secretory vesicles, cell division					
Intermediate Filaments	Structurizing the nuclear envelope and anchor organelles					
Microfilaments	Cell movement					
Other						
Nucleolus	Ribosome assembly					
Ribosome	Protein production					
Cilia and flagella	Locomotion					

# Kingdom Protista

## Phylum Rhizopoda

The name 'Rhizopoda' refers to the process by which the cytoplasm flows within the cell to cause projections of the body wall called **pseudopodia** (**false feet**) that are used for both movement and the ingestion of food. Food is ingested in 2 processes: **phagocytosis** - the process by which a cell uses its plasma membrane to engulf a large particle ( $\geq 0.5 \, \mu m$ ) and results in the food item enclosed within a membrane-bound vesicle called **a food vacuole** (**phagosome**); and **pinocytosis** (**fluid endocytosis**) is a mode of endocytosis in which small particles suspended or dissolved in extracellular fluid are brought into the cell through an invagination of the cell membrane.

# Order Gymnoamoebina Family Entamoebidae *Entamoeba histolytica*



#### **STAGES:**

**Trophozoite** - actively growing and feeding stage

- 12–60 μm in size
- clear granular outer cytoplasm and more densely granular inner cytoplasm
- aggregated karyosome centrally located within the nucleus
- no contractile vacuole
- lacks mitochondria
- organelle called a 'mitosome' (thought to represent the remnants of mitochondria).
- reproduction by binary fission (cell division)

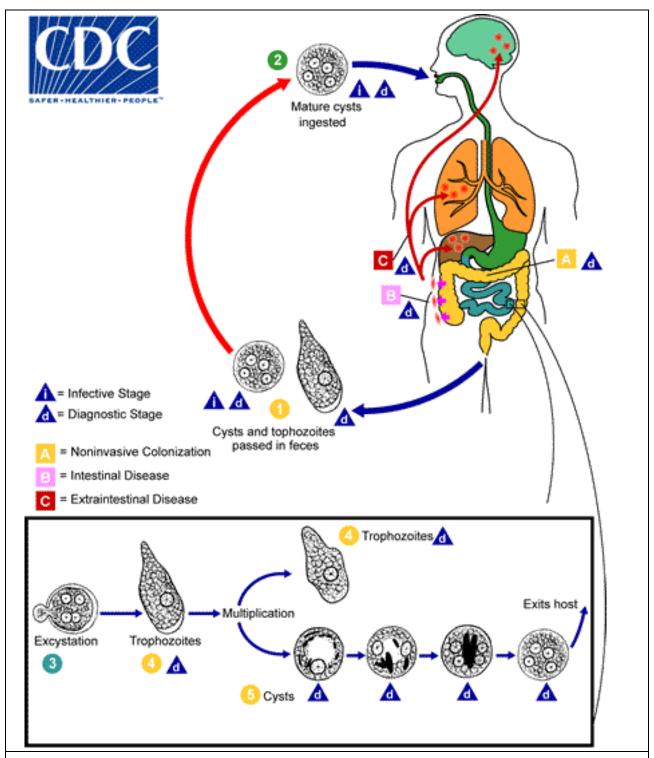
**Avirulent strains** (A) remain in the lumen of the colon, feed by pinocytosis and cause no harm.

**Virulent strains** (B) attack and ingest the epithelial cells lining the gut wall by phagocytosis and then proceed to spread through underlying layers. Ingested red blood cells inside the food vacuoles are usual to find.

#### Cyst (C) - transmission stage

- 10–15 µm in diameter
- has protective covering
- contains four nuclei (when mature)

bar-shaped chromatoidal bodies (agregations of ribosomes)



**Life cycle of** *Entamoeba histolytica* (from https://www.cdc.gov/dpdx)

Cysts and trophozoites (1) are passed in feces. (Cysts are typically found in formed stool, whereas trophozoites are typically found in diarrheal stool.)

Infection occurs by ingestion of mature cysts [2] in contaminated food, water, or hands. Excystation [3] occurs in the small intestine. Trophozoite (4) releases and undergoes a series of divisions to produce eight trophozoites.

Then it migrates  $\rightarrow$  to the large intestine to multiply by binary fission and produce cysts (5). Both stages are passed in the feces (1).

A: <u>noninvasive infection</u> - of individuals who are asymptomatic carriers, passing cysts in stool; B: <u>intestinal disease</u> - trophozoites invade the intestinal mucosa;

**C:** <u>extraintestinal disease</u> - trophozoites invade extraintestinal sites through the bloodstream (e.g. the liver, brain, and lungs)

#### **PATHOGENESIS:**

Fask-shaped ulcers are formed in the intestine wall which can become widespread with consequent bleeding over large areas of the intestine. The loss of functional mucosal surface within the patient's gut and the loss of blood and fluid may cause dehydration and emaciation.

The ulcers in the intestine often suffer secondary invasion by bacteria.

When the ulcers start to heal, they are replaced with fibrous scar tissue and this reduces gut elasticity and, if extensive, may impair peristalsis in the colon and even cause a gut blockage.

Secondary ulcers occur as a result of the amoebae damaging the lining of blood vessels and then being swept up in the bloodstream and setting up infections elsewhere in the body (most commonly located in the liver, although the lungs and brain may also be affected).

#### **CLINICAL FEATURES**

Diarrhoea, flatulence, gastric pain 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. Pain over the liver with hepatomegaly and elevation of the diaphragm is observed.

#### TRANSMITTION

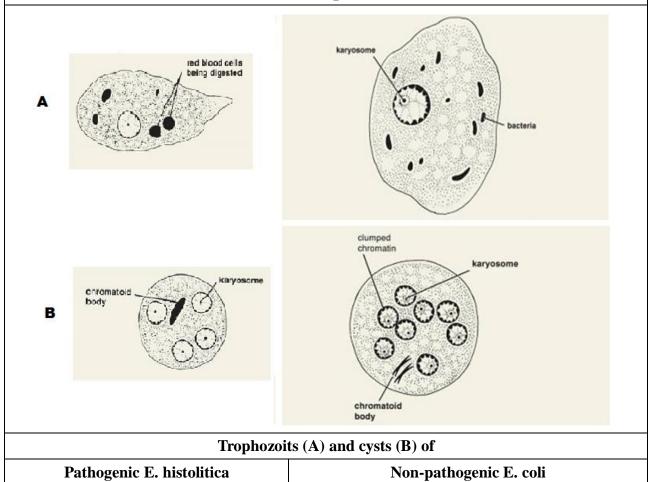
The cysts are usually transmitted via drinking water, contamination on vegetables grown on land fertilised with human faeces or via the bodies of insects that have moved between faeces and human food.

#### **PREVENTION**

Introduction of adequate sanitation measures and education about the routes of transmission. Avoid eating raw vegetables grown by sewerage irrigation and feaces.

Entamoeba coli life cycle stages include trophozoite, precyst, cyst, metacyst, and metacystic trophozoite. Typically the movements of trophozoites are sluggish, with broad short pseudopodia and little locomotion. The cysts are remarkably variable in size. Entamoeba coli is transmitted in its viable cystic stage through faecal contamination. E. coli is non-pathogenic and produces no symptoms. The mature cyst (with more than four nuclei) is the distinctive stage to differentiate E.coli from the pathogenic E.histolytica. Specific treatment is not indicated. Prevention depends on better personal hygiene and sanitary disposal of human excreta.

#### **Comapre:**



Morphology of trophozoits

Species	Size (diameter	Motility	Nucleus			Cytoplasm	
	or length)		Number	Peripheral Chromatin	Karyosomal chromatin	Appearance	Inclusions
Entamoeba histolytica	10-60 μm: usual range. 15- 20 μm - commensal form over20 μm - invasive form	Progressive with hyaline, finger- like pseudopods	One: not visible in unstained preparations	Fine granules: usually evenly distributed and uniform in size	Small, discrete: usually centrally located, but occasionally eccentrically located	Finely granular	Erythrocytes occasionally: non- invasive organisms may contain bacteria
Entamoeba coli		Sluggish, non progressive, with blunt pseudopods	One: often visible in unstained preparations	Coarse granules, irregular in size and distribution	Large, discrete, usually eccentrically located	Coarse, often vacuolated	Bacteria yeasts, other materials

Morphology of cysts

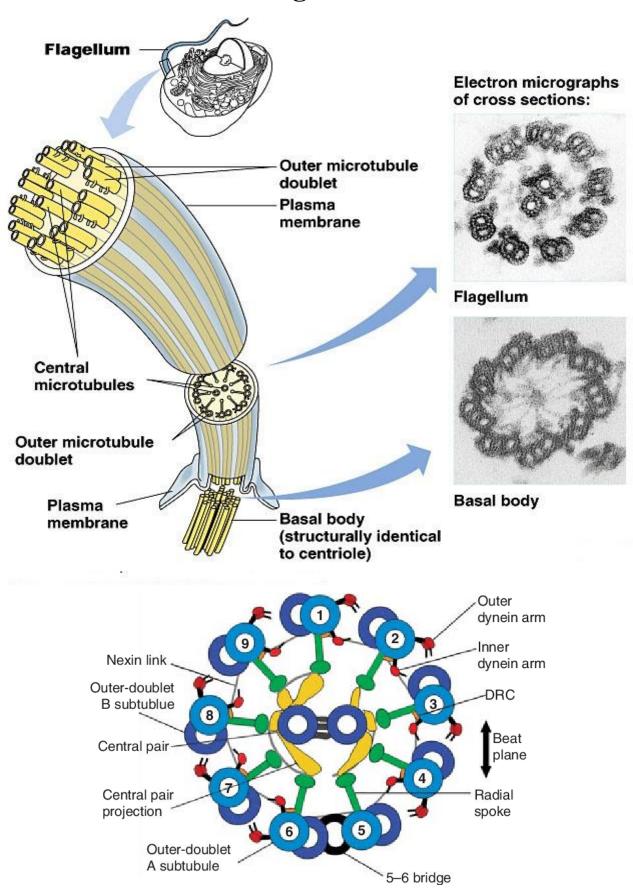
Species	Size	Shape	Nucleus			Cytoplasm		
			Number	Peripheral Chromatin	Karyosomal chromatin	Chromatoid bodies	Glycogen and other features	
Entaboeba histolytica	10-20 μm: usual range, 12–15μm	Usually spherical	immature cysts with 1 or 2 occasionally	Peripheral chromatin present: fine, uniform granules, evenly distributed		Present: elongated bars with bluntly rounded ends	Usually diffuse: concentrated mass often in young cysts; stains reddish brown with iodine	
Entaboeba coli	usual range, 15-25 μm	spherical: sometimes oval,	cyst: occasionally, super nucleate cysts with 16 rarely seen; immature cysts with 2 or more	present: coarse granules irregular in	usually eccentrically, but occasionally	E.histolytica; usually	Usually diffuse but occasionally well-defined mass in immature cysts; stains reddish brown with iodine	

#### **Assignment:**

On the slide find <u>trophozoites of Entamoeba histolytica</u> that looks like debris but with a smooth edge, either bluish or gray, with a nucleus containing a small, centrally located nucleolus and <u>cysts of Entamoeba histolytica</u> - spherical, gray or purple, with 1, 2, or 4 nuceli. Some young cysts may have large, blunt ended chromotoidal bars. Older cysts should have the 4 nuclei, but the chromotoidal bars may have disappeared.

<u>Trophosoites of Entamoeba coli</u> (may be found on the same slide) - are large blobs with smooth margins, either purple or gray, with single nucleus containing an nucleolus that is usually off-center. Note that the trophozoites are larger than *E. Histolytica*, the peripheral chromatin in the nucleus is less evenly dispersed. <u>Entamoeba coli cysts</u> are large, spherical or ellipsoidal, gray-purple; 8, 16, or 32 nuclei. Some may have chromatoidal bars, most of which should be sharply pointed.

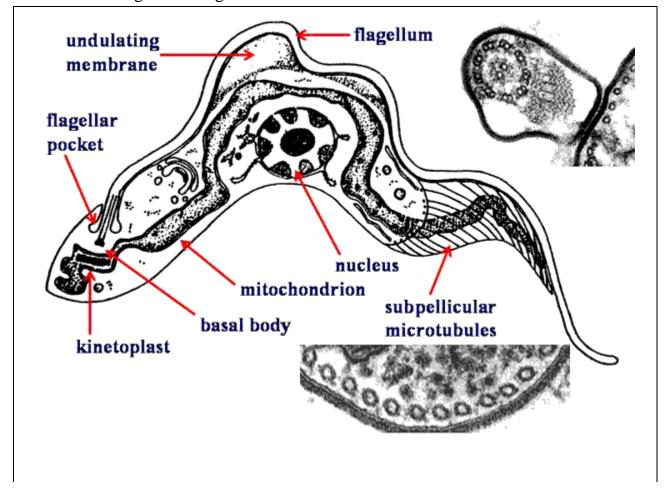
# **Flagellates**

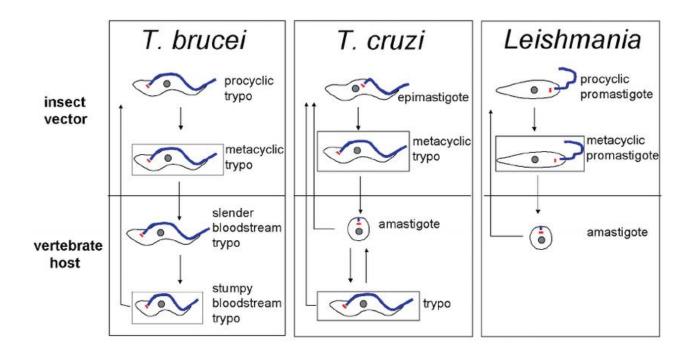


A schematic diagram of axoneme of eucarioteic cilium or flagellum

#### Typical morphology of trypanosoma

- elongated spindle-shaped body that more or less tapers at both ends
- centrally situated nucleus,
- <u>kinetoplast</u> (a highly modified portion of DNA in the mitochondria)
- <u>undulating membrane</u> arising from the kinetoplast and proceeding forward along the margin of the cell membrane
- single free flagellum at the anterior end





#### Morphological forms of kinetoplastids:

#### trypomastigote -

elongate

kinetoplast is posterior to the nucleus.

flagellum runs along the surface of the organism anteriorly

long undulating membrane.

#### epimastigote

kinetoplast is located between the nucleus and anterior end.

short undulating membrane lies along the proximal portion of the flagellum;

#### promastigote

elongate

the flagellum extending forward

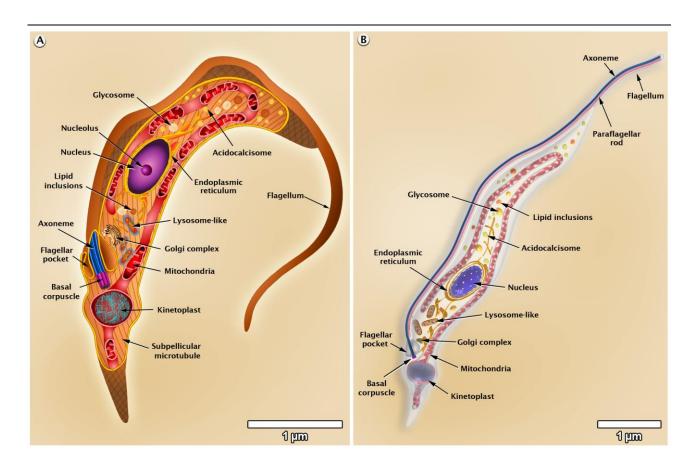
kinetoplast is located anterior to the nucleus

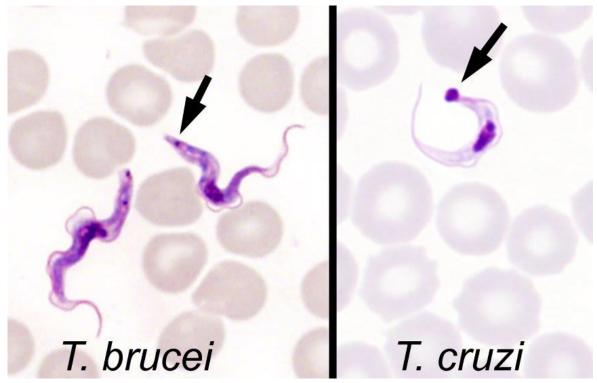
no undulating membrane is present.

amastigote (mainly within infected cells).

small and ovoid

short flagellum projecting only slightly beyond the organism (if at all).





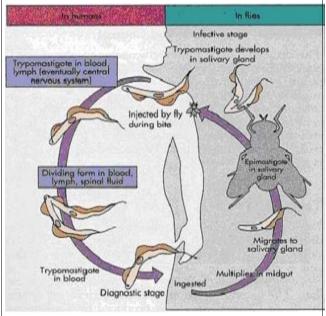
T. brucei cause African sleeping sickness. In the blood they are long, slender trypomastigotes, with a small kinetoplast, prominent nucleus, and undulating membrane.

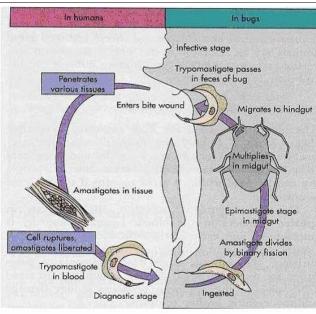
(*T. rhodesiense* and *T. gambiense*) are identical morphologically.

The American trypanosome, *Trypanosoma cruzi*, is causing <u>Chagas' disease</u>.

Trypomastigotes in the blood are C- or ?-shaped. They have a large kinetoplast at one end and a prominent nucleus.

# Life cycle





**T. brucei** (A) is transmitted by the bite of any one of a number of *Glossina* spp. (tsetse flies).

T. cruzi (B) are engulfed by phagocytic cells and then transform into amastigotes, which undergo binary fission and destroy the host cells. Groups of these amastigotes in the tissues are termed pseudocysts.

T. cruzi is generally transmitted in the feces of reduviid bugs, which defecate when they bite. The parasite enters the wound when the host rubs the contaminated feces into the lesion.

#### **Pathogenesis**

The typical somnolence (sleeping sickness) usually progresses to coma as a result of demyelinating encephalitis.

The amastigotes can kill cells and cause inflammation, consisting mainly of mononuclear cells.

#### **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.

- an occasional ulcer at the site of the fly bite
- fever, myalgia, arthralgia, and lymph node enlargement

Chronic disease: lethargy, tremors, meningoencephalitis, mental retardation, and general deterioration.

The final stages: convulsions, hemiplegia, and incontinence.

Early stages: erythematous and indurated area at the site of the bug bite ("chagoma"), - rash and edema around the eyes and face; Acute infection: fever, chills, malaise, myalgia, and fatigue.

Chronic disease: hepatosplenomegaly, myocarditis, enlargement of the esophagus and colon; granulomas in the brain with cyst formation and a meningoencephalitis.

In *T.rhodesiense*, the disease caused is a more acute, rapidly progressive disease (usually fatal). - lymphadenopathy

- lethargy, anorexia, and mental disturbance.
- kidney damage & myocarditis.

#### **Prevention**

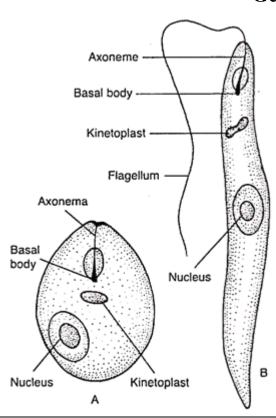
- Control of breeding sites of tsetse flies and use of insecticides.
- Treatment of human cases to reduce transmission to flies.
- Avoiding insect bite by wearing protective clothing & use of screen, bed netting and insect repellants
- Bug control, eradication of nests
- Treating infected person & exclusion of donors by screening blood.
- Development of vaccine.

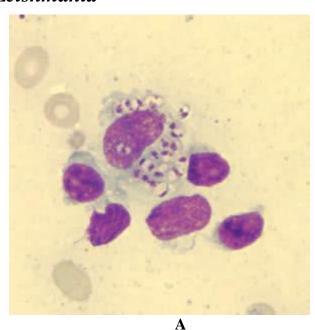
#### **Assignment:**

In <u>Trypanosoma brucei</u> blood smear find numerous purple trypomastigotes among blood cells. Note the irregular manner in which the trophozoites have died, the small kinetoplast at one end, and the undulating membrane associated with the flagellum.

In <u>Trypanosoma cruzi</u> blood smear find purple trypomastigotes among blood cells. Note that they are smaller than the African trypanosomes and die in a C-or ?-shape. The kinetoplast is slightly larger in proportion to the body of the trypomastigote when compared to the African trypanosomes.

#### Genus Leishmania



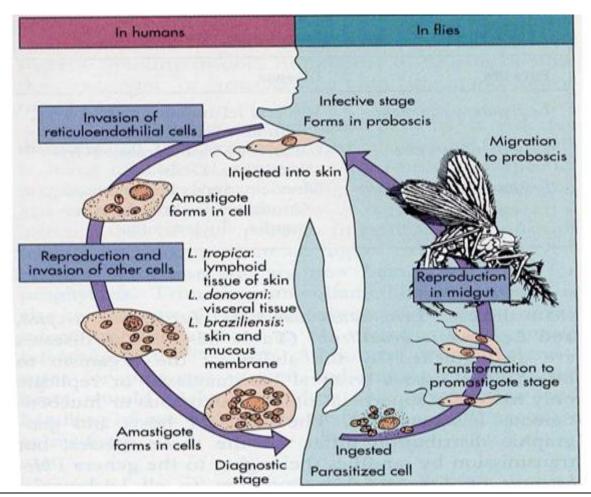


#### <u>amastigote (A)</u> form -- in vertebrate hosts

- ovoidal or rounded body
- 2-4 μm in length
- oval or round nucleus in the middle of the cell or along the side of the cell membrane.
- kinetoplast lies tangentially or at right angles to the nucleus.
- axoneme delicate filament extending from the kinetoplast to the margin of the body.

#### **promastigote** (**B**) -- in invertebrate host

- 15-25μm lengths by 1.5-3.5μm breadths
- a single large spherical nucleus lies eccentrically or in the middle of the body
- colorless homogeneous cytoplasm (not differentiated into ectoplasm and endoplasm)
- kinetoplast lies transversely near the anterior end.



#### Life cycle

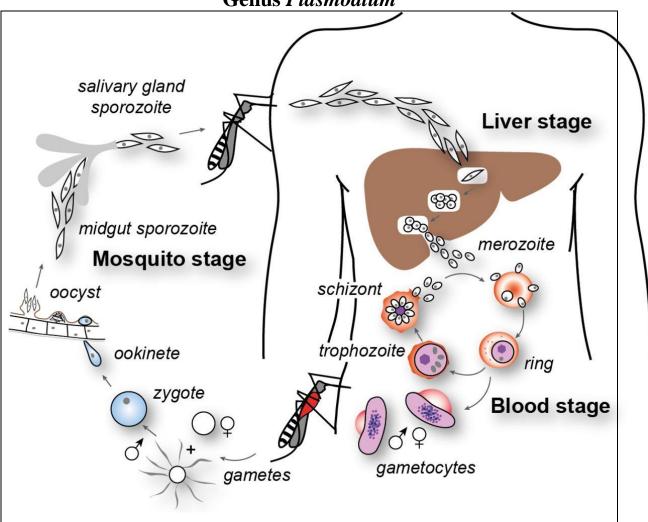
*Leishmania* spp. spend their lives in the vertebrate host only as an amastigote where they multiply in the gut as promastigotes and invade large numbers in phagocytic cells. Transmitted by the bite of sandflies (*Phlebotomus* and *Lutzomyia sp.*).

#### Assignment

#### Leishmania sp., amastigotes in spleen smear

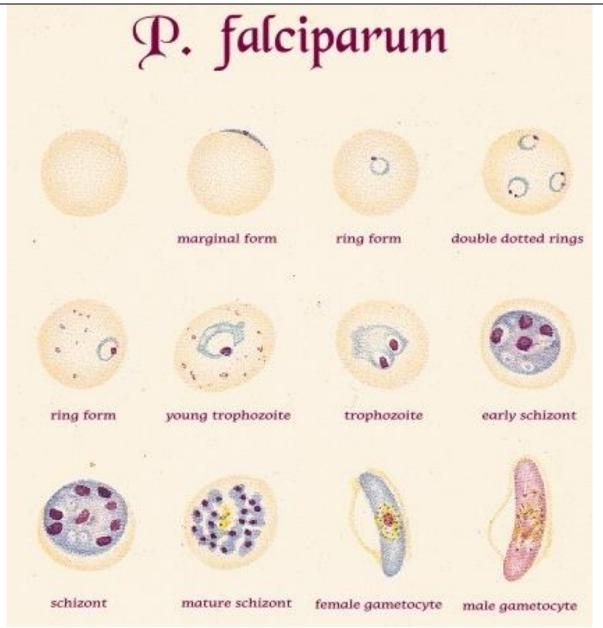
The amastigotes represent numerous small, dark dots with a nucleus and adjacent kinetoplast. You should be able to easily see the numerous parasites using a 40x objective lens, then go to oil immersion (100x) to take a closer look.

# Phylum Apicomplexa Genus *Plasmodium*



The life cycle of malaria is passed in two hosts (alternation of hosts) and has exual and asexual stages (alternation of generations).

Sporozoites injected with the bite of mosquitos of the genus *Anopheles* make their way to the liver and multiply in hepatocytes. Once this <u>exoerythrocytic cycle</u> is complete, <u>merozoites</u> (the stages resulting from multiple fission) enter red blood cells and begin <u>schizogony</u> (merogony). New merozoites formed within these cells (<u>segmenters</u>) rupture out, invade new cells and keep this <u>erythrocytic cycle</u> going. Parasites in red blood cells that are early in development and resemble an amorphous mass are termed <u>trophozoites</u>. Some appear ring-like or band-shaped and are termed <u>rings</u> or <u>bands</u>. Some merozoites grow and form <u>gametocytes</u> (gametes) within red blood cells. Using the plates in your textbook, see if you can identify segmenters, gametocytes, trophozoites, and rings in your blood smears. If gametes are ingested by the appropriate mosquito, some form flagella and undergo <u>exflagellation</u> (male gamete formation). These male gametes (<u>microgametes</u>) fuse with female gametes (<u>macrogametes</u>) to form a motile zygote termed an <u>ookinete</u>. Ookinetes penetrate the gut wall and form oocysts containing sporozoites on the hemocoel side of the gut. Sporozoites eventually rupture out of oocysts and migrate to the salivary glands.



## Stages in human

**Clinical features:** The initial symptoms of malaria are flu-like and include:

- high temperature of 38°C or above;
- cough;
- feeling hot and shivery;
- headaches and muscle pains;
- nausea, vomiting and diarrhoea;
- generally feeling unwell.

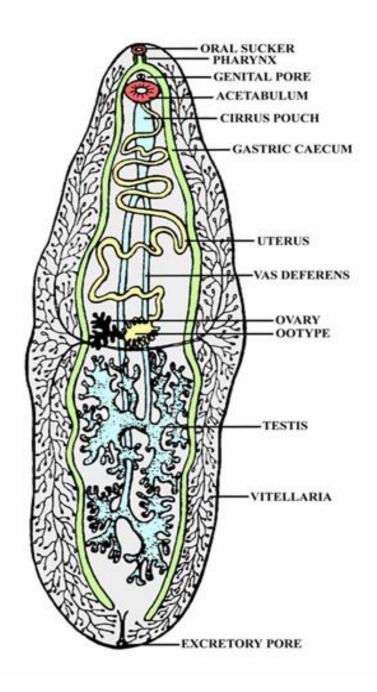
Symptoms occur in 48-hour cycles (in P. vivax, P. ovale, and P. falciparum) or -hour cycle (in P. malaie) and last between 6 and 12 hours.

#### **Prevention**

- Chemoprophylaxis and prompt diagnosis and treatment.
- Control of mosquito breeding
- Protection of insect bite by screening, netting and protective clothing
- Use of insect repellents.

#### **Helminths: TREMATODES (FLUKES)**

Trematodes belong to the **phylum platyhelminthes**. They are found in a wide range of habitats. The great majority inhabit the alimentary canal, liver, bile duct, ureter and bladder of vertebrate animals.



Hypothetical trematode

#### **General morphology:**

Most of digenetic trematodes are dorso-ventrally flattened and possess a muscular <u>oral sucker</u> that surrounds the mouth. In addition, the majority also possess a midventral or posterior <u>acetabulum</u> (ventral sucker).

The body of a fluke **is covered** by a living layer of cells termed **a <u>tegument</u>**, which functions in nutrient absorption. Thus, flukes can digest and absorb nutrients not only across the gut wall, but also across the outer body. Ornamentation, such as spines, are often present within the tegument and can often be seen with the light microscope.

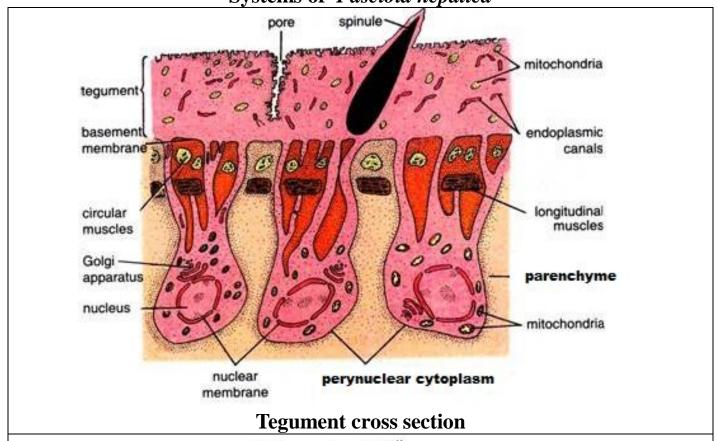
The digestive tract of a fluke normally consists of a short, muscular <u>esophagus</u> (often surrounded by a muscular <u>pharynx</u>), which then splits into a pair of blind <u>intestinal cecae</u>. Generally, tissues are drawn into the mouth which are then eroded by the strong pumping action of the pharynx. Species such as the schistosomes, which live in the blood vessels and suck blood, do not have a pharynx.

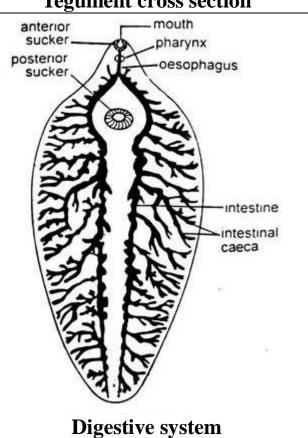
**The excretory system** is <u>protonephric</u> type. (Vessels branches are repeatedly ended with the "flame cells" with a bundle of flagella near the nucleus). A longitudinal excretory duct opens posteriorly by a an excretory pore.

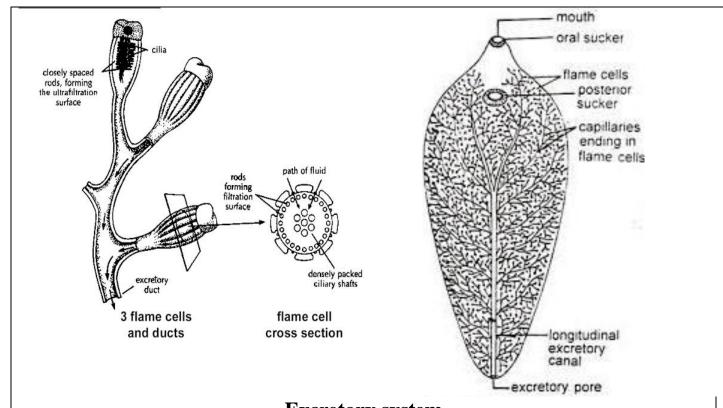
Most trematodes are <u>hermaphroditic</u> (except the schistosomes) and many self-fertilize. **The male reproductive system** usually consists of two <u>testes</u> (1-several hundred), each of which has a <u>vas efferens</u> that connect to form a common duct, the <u>vas deferens</u>. The vas deferens leads to the <u>genital pore</u>, which usually has associated structures such as an <u>internal seminal receptacle</u> for sperm storage, a <u>prostate gland</u> that may add secretions to the sperm, and a <u>cirrus</u>, the male copulatory organ.

The female reproductive system is more complicated than the male system and consists of a single ovary, an oviduct, a seminal receptacle for sperm storage, vitelline glands along the lateral margins of the body that provide material for egg shell formation, a series of glandular structures that aid in egg shell maturation (i.e Mehlis gland, ootype, Lauer's canal, etc.), a uterus which may be filled with eggs, and perhaps a muscular modification of the end of the uterus termed a metraterm.

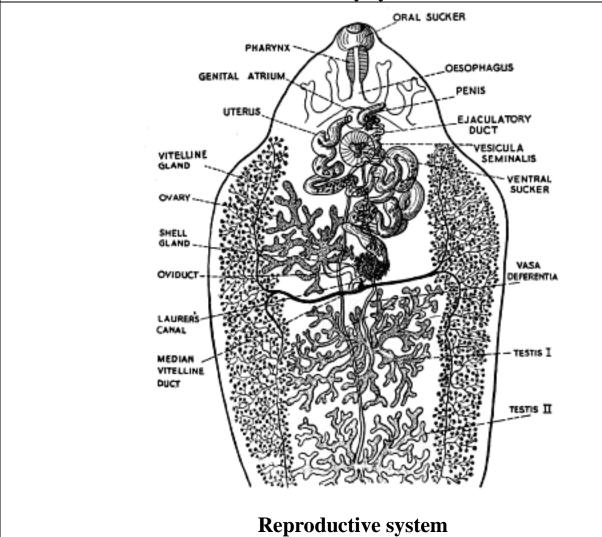
Systems of Fasciola hepatica







# **Excretory system**



#### **Clinical features**

During the **early phase** of the infection (the period when the larval fluke is migrating:

- inflammation, tissue destruction, and toxic/allergic reactions.
- nonspecific symptoms/ (abdominal pain, nausea, vomiting, hepatomegaly, malaise, fever, cough) laboratory abnormalities (peripheral eosinophilia, elevated transaminase levels) During the **chronic phase**, clinical manifestations, if any, may develop months to years postexposure and include
- inflammation or blockage of bile ducts
- intermittent gallbladder (cholangitis, cholecystitis)
- inflammation of the.

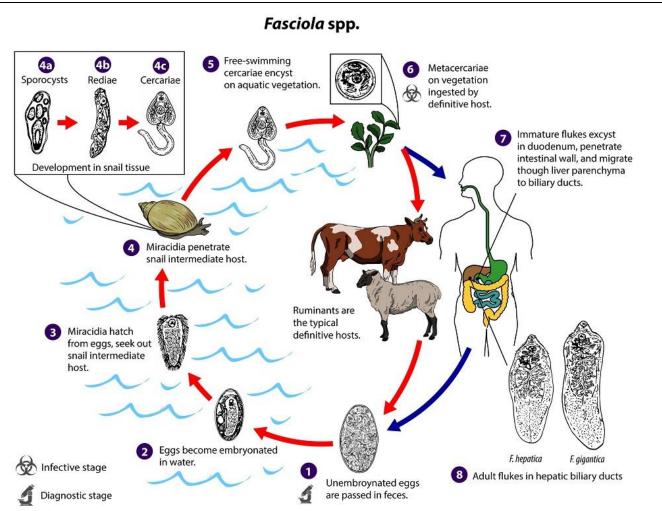
**Prevention by** not eating raw watercress and other water plants, especially from Fasciola-endemic grazing areas.

#### **Assignment:**

Note the large size of the worm. Oral and ventral suckers are relatively small in proportion to the body and located anteriorly. Internal details are often difficult to discern because of the dendritic nature of many key structures.

Use lens to distinguish each system separately on stained films.

#### LIFE CYCLE OF TREMATODES



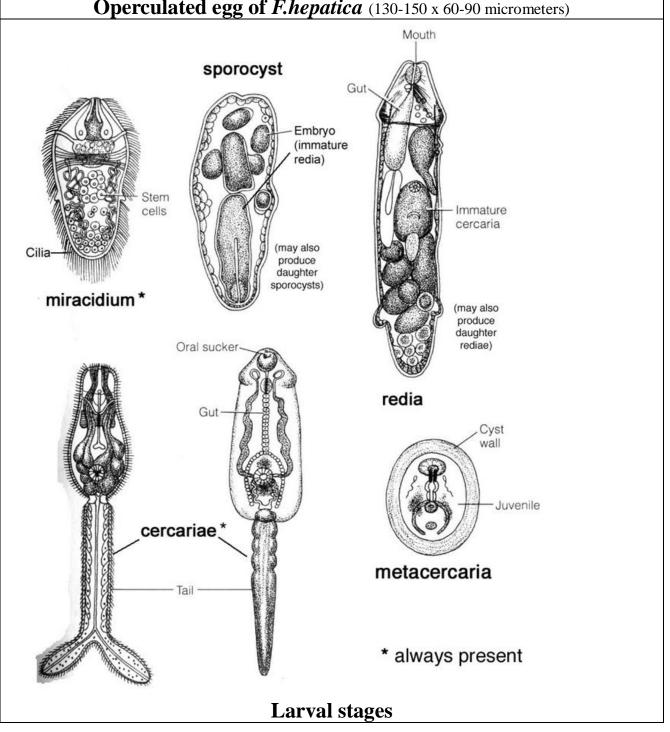
**Life cycle:** Immature eggs are discharged in the biliary ducts and passed in the stool (1). Eggs become embryonated in freshwater over about 2 weeks (2); eggs release miracidia (3), which invade a snail intermediate host (4). In the snail, the parasites undergo several developmental stages (sporocysts (4a), rediae (4b), cercariae (4c)). The cercariae are released from the snail actively (5) and encyst as metacercariae on aquatic vegetation or other substrates nearby. Humans and other mammals become infected by ingesting metacercariae-contaminated vegetation (6). After ingestion, the metacercariae excyst in the duodenum (7) and penetrate through the intestinal wall into the peritoneal cavity. The immature flukes then migrate through the liver parenchyma into biliary ducts, where they mature into adult flukes and produce eggs (8). In humans, maturation from metacercariae into adult flukes usually takes about 3–4 months (development of *F. gigantica* may take longer). **Definitive hosts**: domestic and wild ruminants (most commonly, sheep, cattle, and goats; also, camelids, cervids, and buffalo).

**Intermediate hosts:** snails in the family Lymnaeidae

**Assignment:** On demonstration slides find all larval stages of *Fasciola hepatica*. Eggs are relatively large and easy to see under low power of microscope. They do not possess shoulders and are passed unembryonated in the feces. The eggs are almost identical to those of *Fasciolopsis buski*, the giant intestinal fluke of Asia.

# Life cycle stages of trematodes Operculum Fertilised egg or zygote Yolk cells Egg shell

**Operculated egg of** *F.hepatica* (130-150 x 60-90 micrometers)



# Life cycle stages of trematodes

Stage	Description/ role in the life cycle	Reproduction	Motile	Feeding
Adult	Lives in the definitive host. Usually hermaphrodites but sexes separate in some species. Produces eggs	Yes. Usually sexual reproduction but may be parthenogenic.	Yes	Yes. Has mouth and gut though may also absorb nutrients across the body surface.
Egg	Covered with the protected wall. Contains the miracidium. May hatch in the environment or within gut of the first intermediate host.	No	No	No
Miracidium	Infective stage. Covered in cilia. Invades the first intermediate host	No	Yes	No
Sporocyst	Lacks a mouth and gut; Reproduces within first intermediate host	Yes. Asexual reproduction to form daughter sporocysts or rediae	No	Yes. Absorbs nutrients across body wall
Redia	Has a mouth and gut. Reproduces within first intermediate host. Evidence of caste system in some species	Yes. Asexual reproduction to form daughter sporocysts or rediae	Yes	Yes
Cercaria	Infective stage with a propulsive 'tail'. Often leaves first intermediate host and invades second intermediate host or definitive host	No	Yes	No
Metacercaria	Infective stage. Encysted and covered with protective wall. Develops in the environment or within the second intermediate host	No	No	No

# OTHER MEDICALLY IMPORTANT TREMATODES (FLUKES) Dicrocolelum lanceatum Cercariae encyst to metacercariae after being eaten by an ant intermediate host. Incidental human infection Definitive host becomes infected by ingestion of infected ants. Cercariae are released from the snail via the respiratory pore in a slime ball. Cercariae Miracidia - Sporocysts 6 Adult in bile duct

Embryonated eggs are shed in the feces.

Eggs are ingested by

host.

a snail intermediate

Infective stage

Diagnostic stage

#### Life cycle

Embryonated eggs containing miracidia are shed in feces of definitive hosts, which are typically ruminants (1). The eggs are then ingested by the first intermediate host (snail) (2). When the miracidia hatch (2A), they migrate through the gut wall and settle into the adjacent vascular connective tissue, where they become mother sporocysts (2B). The sporocysts migrate to the digestive gland where they give rise to several daughter sporocysts. Inside each daughter sporocyst, cercariae are produced (2C). Cercariae migrate to the respiration chamber where they are shed in slime ball from the snail (3). After a slime ball is ingested by the second intermediate host (ant), the cercariae become free in the intestine and migrate to the hemocoel where they become metacercariae (4). When the infected ant is eaten by a suitable definitive host (5), the metacercariae excyst in the small intestine. The worms migrate to the bile duct where they mature into adults (6). Humans can serve as definitive hosts after ingesting infected ants (e.g. on contaminated food items) (7).

#### **Hosts**

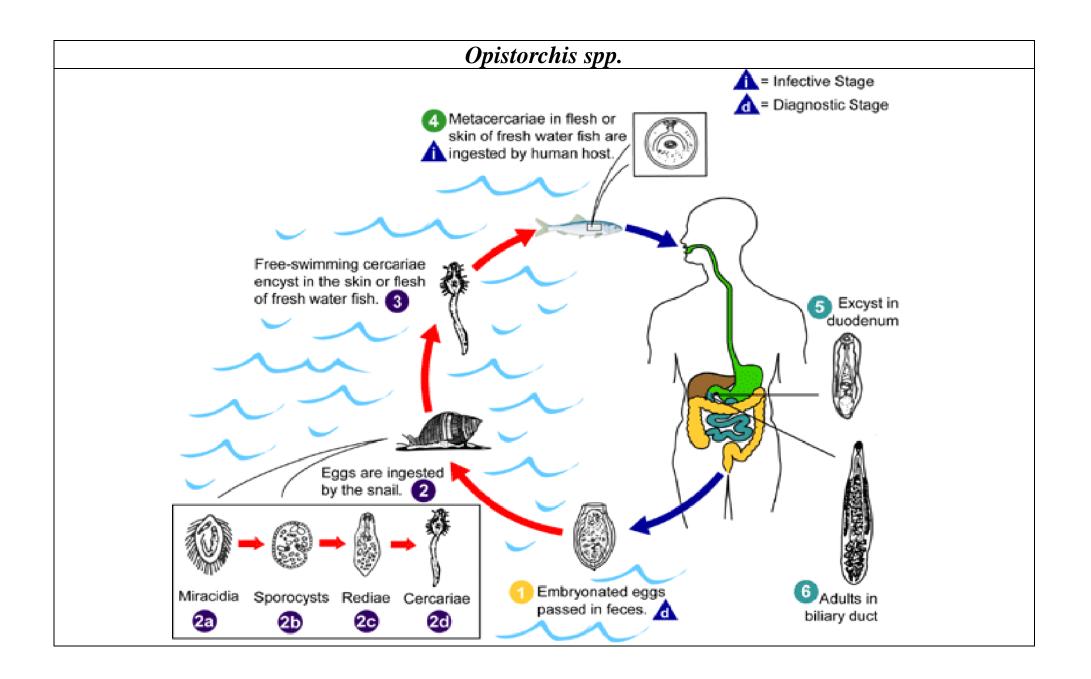
Ruminants, especially cattle and sheep, aberrant in primate species and domestic dogs.

First intermediate hosts: air-breathing land snail species (Pulmonates).

Second intermediate hosts: ants (especially members of the genus *Formica*).

#### **Clinical features**

Cholecystitis, liver abscesses, and generalized gastrointestinal/abdominal distress.



#### Life cycle

The adult flukes deposit fully developed eggs that are passed in the feces (1). After ingestion by a suitable snail (first intermediate host) (2), the eggs release miracidia (2a), which undergo in the snail several developmental stages (sporocysts (2b), rediae (2c), cercariae (2d)). Cercariae are released from the snail (3) and penetrate freshwater fish (second intermediate host), encysting as metacercariae in the muscles or under the scales (4). The mammalian definitive host (cats, dogs, and various fisheating mammals including humans) become infected by ingesting undercooked fish containing metacercariae. After ingestion, the metacercariae excyst in the duodenum (5) 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 (6).

The adult flukes (O. viverrini: 5 mm to 10 mm by 1 mm 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.

#### **Hosts**

Definitive hosts: cats, dogs.

First intermediate hosts: sreshwater snails Second intermediate hosts: freshwater fish

#### **Clinical features**

Inflammation and intermittent obstruction of the biliary ducts;

In mild cases: dyspepsia, abdominal pain, diarrhea, or constipation;

With infections of longer duration: hepatomegaly and malnutrition.

Most infections are asymptomatic.

#### **Prevention**

Avoiding raw or undercooked freshwater fish, lightly salted, smoked, or pickled fish

#### Shistosoma spp. Free-swimming cercariae released Cercariae from snail into water penetrate skin Sporocysts develop in snail (successive Cercariae lose tails during generations) penetration and become schistosomulae (0) 8 Circulation Miracidia penetrate snail tissue Migration to portal blood in liver and maturation into adults 1 Eggs shed from infected human: in feces in urine Eggs hatch and release miracidia 10 Paired adult worms migrate to: Mesenteric venules of bowel/rectum S. japonicum (laying eggs that circulate to the liver and shed in stools) S. mekongi S. haematobium S. mansoni Α Infective stage Venous plexus of bladder; eggs shed Diagnostic stage C in urine

**Life cycle:** *Schistosoma* eggs are eliminated with feces or urine, depending on species (1). Under appropriate conditions the eggs hatch and release miracidia (2), which swim and penetrate specific snail intermediate hosts (3). The stages in the snail include two generations of sporocysts (4) and the production of cercariae (5). Upon release from the snail, the infective cercariae swim, penetrate the skin of the human host (6), and shed their forked tails, becoming schistosomulae (7). The schistosomulae migrate via venous circulation to lungs, then to the heart, and then develop in the liver, exiting the liver via the portal vein system when mature, (8, 9). Male and female adult worms copulate and reside in the mesenteric venules, the location of which varies by species (with some exceptions) (10). For instance, *S. japonicum* is more frequently found in the superior mesenteric veins draining the small intestine (A), and *S. mansoni* occurs more often in the inferior mesenteric veins draining the large intestine (B). However, both species are capable of moving between sites. *S. intercalatum* and *S. guineensis* also inhabit the inferior mesenteric plexus but lower in the bowel than *S. mansoni*. *S. haematobium* most often inhabitsin the vesicular and pelvic venous plexus of the bladder (C), but it can also be found in the rectal venules. The females (size ranges from 7–28 mm, depending on species) deposit eggs in the small venules of the portal and perivesical systems. The eggs are moved progressively toward the lumen of the intestine (*S. mansoni*, *S. japonicum*, *S. mekongi*, *S. intercalatum/guineensis*) and of the bladder and ureters (*S. haematobium*), and are eliminated with feces or urine, respectively (1).

#### Hosts

Various animals such as cattle, dogs, cats, rodents, pigs, horses, and goats, serve as reservoirs for *S. japonicum*, and dogs for *S. mekongi. S. mansoni* is also frequently recovered from wild primates in endemic areas but is considered primarily a human parasite and not a zoonosis.

Intermediate hosts are snails of the genera *Biomphalaria*, (*S. mansoni*), *Oncomelania* (*S. japonicum*), *Bulinus* (*S. haematobium*, *S. intercalatum*, *S. guineensis*). The only known intermediate host for *S. mekongi* is *Neotricula aperta*.

#### **Clinical features**

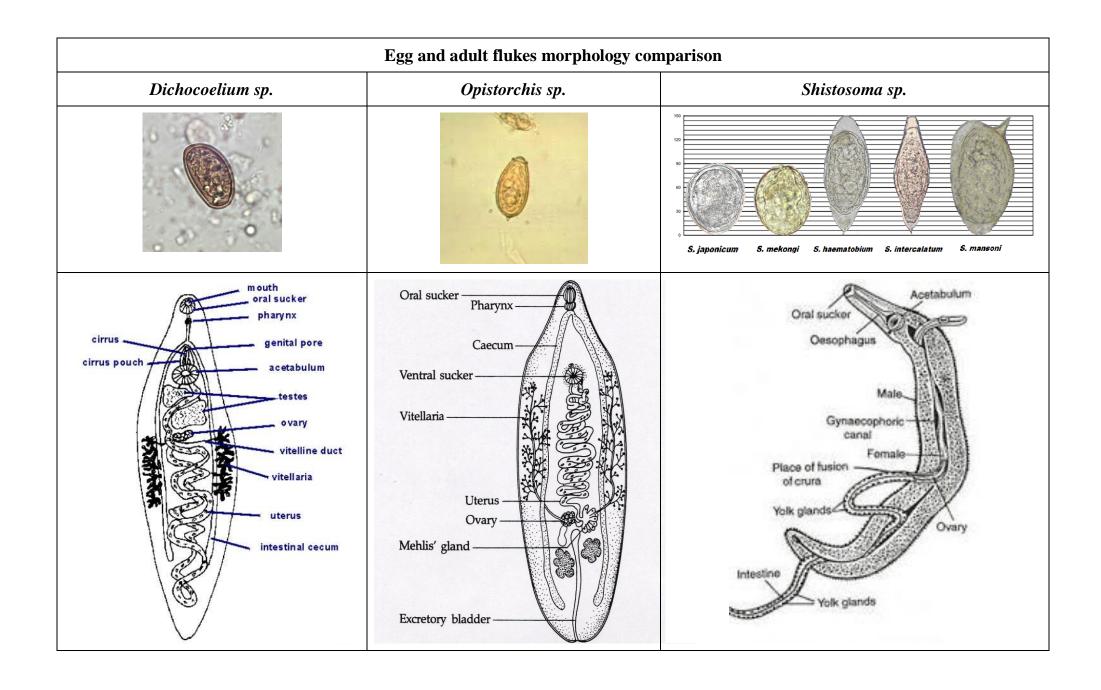
Symptoms are caused by the the body's reaction to the eggs. Many infections are asymptomatic.

A local cutaneous hypersensitivity reaction (small, itchy maculopapular lesions).

Acute schistosomiasis (Katayama fever) is a systemic hypersensitivity reaction that may occur weeks after the initial infection, especially by *S. mansoni* and *S. japonicum*. Manifestations include systemic symptoms/signs including fever, cough, abdominal pain, diarrhea, hepatosplenomegaly, and eosinophilia.

#### **Prevention**

Avoiding contact with fresh water infested with **Schistosome** parasites, i.e.swimming, wading, or any other aquatic activities in these bodies.



# **CESTODES (TAPEWORMS)**

The adult tapeworms have flat body, white or grayish in color. Tapeworms vary in length from 2 to 3 mm to 10 m, and may have three to several thousand segments.

They consist of an anterior attachment organ or <u>scolex</u> and a chain of segments (<u>proglottids</u>) also called <u>strobilla</u>. The strobilla is the entire body except the scolex. The scolex is characterized by holdfast organs consist of <u>a rostellum</u>, <u>bothria</u>, or <u>acetabula</u>. A <u>rostellum</u> is a retractable, conelike structure that is located on the anterior end of the scolex, and in some species is armed with hooks. <u>Bothria</u> are long, narrow, weakly muscular grooves. <u>Acetabula</u> are suckers like those of digenetic trematodes.

The strobila elongates as new proglottids form in the neck region. The segments nearest the neck are **immature** (sex organs not fully developed) and those more posterior are **mature**. The terminal segments are **gravid**, with the egg-filled uterus as the most prominent feature.

The scolex contains the <u>cephalic ganglion</u>, or "brain" of the tapeworm **nervous** system.

A characteristic feature of adult tapeworm is the **absence of an alimentary canal** (there is neither a mouth nor a digestive tract), since all of these adult worms inhabit the small intestine - substances enter the tapeworm across the **tegument**. This structure is well adapted for transport functions, since it is covered with numerous **microvilli** resembling those lining the lumen of the mammalian intestine.

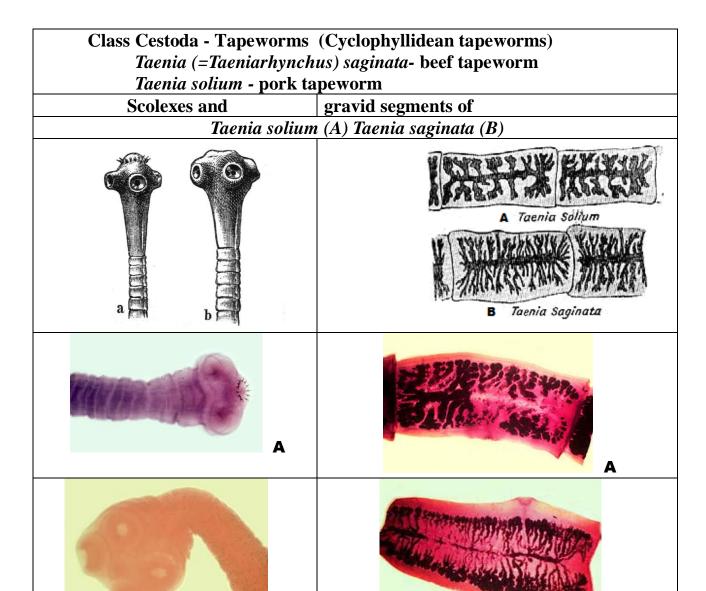
Tapeworms also lack a circulatory system and an organ specialized for gas exchange.

The excretory system is of the **flame cell** type.

Cestodes are hermaphroditic, each proglottid possessing male and female reproductive system. They are usually self-fertilizing.

Eggs exit through a uterine pore in the center of the ventral surface (of pseudophyllidean tapeworms) or released only when the tapeworms shed gravid proglottids into the intestine.

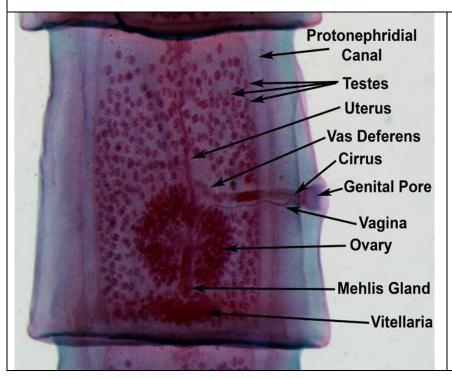
The eggs of pseudophyllidean tapeworms are **operculated**, but those of cyclophyllidean species are not. Eggs of all tapeworms contain at some stage of development an embryo or **oncosphere**. The oncosphere of *pseudophyllidean* tapeworms is ciliated externally and is called a **coracidium**. The coracidium develops into a **procercoid** stage in its micro-crustacean first immediate host and then into a **plerocercoid** larva in its next intermediate host which is a vertebrate. The plerocercoid larva develops into an adult worm in the definitive (final) host. The oncosphere of *cyclophyllidean* tapeworms, depending on the species, develops into a **cysticercus larva, cysticercoid larva, coenurus larva,** or **hydatid larva (cyst)** in specific intermediate hosts. These larvae become adults in the definitive host.



В

В

# Reproductive system (mature proglottid) Ventral excretory canal **0 0** Uterus Dorsal excretory canal Sperm duct Vasa efferentia Seminal vesicle Cirrus sac Testes Genital atrium - Vagina Ovary -Seminal receptacle Mehlis' gland Ootype Vitelline gland



The male and female systems share a common genital pore - a large aperture on either the right or left side of the proglottid. It opens into a shallow, cuplike genital atrium. The male and female systems both open into the atrium via its own gonoduct.

There are two ducts joining the medial border of the genital atrium. The anterior duct is the thicker and is the <u>male gonoduct</u>. The gonoduct is regionally specialized. The wide portion of the gonoduct attached to the atrium is the muscular <u>cirrus sac</u>. Inside the sac is the convoluted, eversible, tubular <u>cirrus (penis)</u>, which is the intromittent organ. The next region of the male gonoduct is the tubular <u>sperm duct</u>, also convoluted, which extends to the <u>testes</u>. Its entire length is not visible. The testes are numerous small spheres scattered throughout the parenchyma. Each is drained by a tiny <u>tributary of the sperm duct</u>, but these cannot be seen. (There is no seminal vesicle and autosperm are stored in the coils of the sperm duct.)

The smaller and more posterior of the two ducts entering the genital atrium is the **female gonoduct**. The first region is the **vagina**. It extends medially and posteriorly to the small **seminal receptacle**. This is a clear, unstained, oval chamber where allosperm received by the vagina are stored. It is usually easily visible. A short duct exits the posterior end of the seminal receptacle and joins the oviduct.

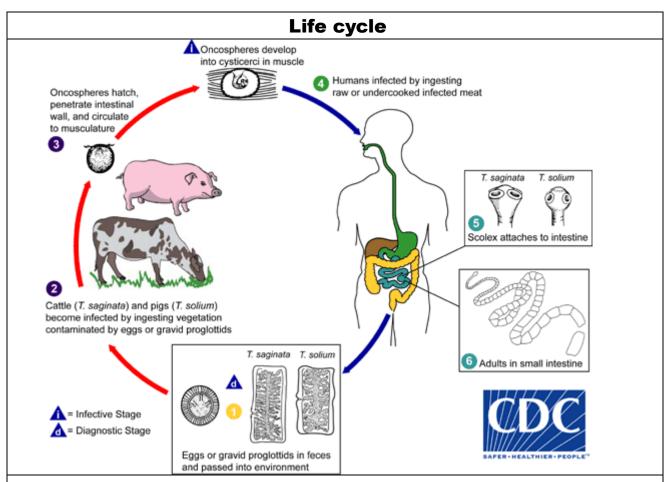
The <u>germarium (= ovary)</u> is divided into large right and left lobes lying on either side of the seminal receptacle. It is the site of oogenesis and produces large numbers of small, yolkless oocytes. The two lobes of the germarium are connected across the midline by a short, wide, transverse <u>isthmus</u>.

The follicles of the germarium open into small ducts which drain into the isthmus. The narrow <u>oviduct</u> arises from the isthmus and extends posteriorly for a short distance before receiving the duct from the seminal. The isthmus is usually easy to see but the oviduct is often obscured by the seminal receptacle and is harder to find. After receiving the duct from the seminal receptacle the oviduct continues posteriorly to the <u>ootype</u>. Fertilization occurs in the oviduct.

Yolk cells are produced by the single <u>vitellarium</u> at the posterior end of the proglottid. A short <u>vitelline duct</u> exits the vitellarium and extends anteriorly to join the oviduct at the ootype. <u>Mehlis's gland</u> (lubricates the passage of uterus through which ova move, provides the eggshell formation) surrounds the ootype. The ootype, Mehlis's gland, and associated ducts cannot be seen in most slides.

A small <u>uterine duct</u> extends from the ootype to the <u>uterus</u>. Shelled eggs move from the ootype through the uterine duct into the uterus. Within the shell meiosis is completed, a zygote forms, and development proceeds to the oncosphere larval stage. The uterus is a blind sac with lateral branches in which embryonated eggs are stored.

As the proglottid ages the accumulating eggs cause the uterus to become larger, darker, and more visible

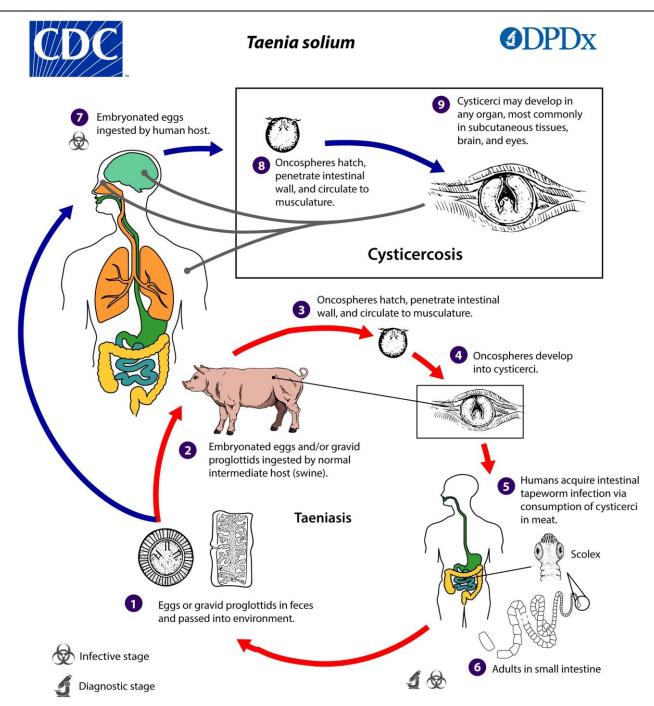


Taeniasis is the infection of humans with the adult tapeworm of *Taenia saginata*, *T*. solium or T. asiatica. Humans are the only definitive hosts for these three species. Eggs or gravid proglottids are passed with feces 0; the eggs can survive for days to months in the environment. Cattle (*T. saginata*) and pigs (*T. solium* and *T. asiatica*) become infected by ingesting vegetation contaminated with eggs or gravid proglottids 2. In the animal's intestine, the oncospheres hatch 3, invade the intestinal wall, and migrate to the striated muscles, where they develop into cysticerci. A cysticercus can survive for several years in the animal. Humans become infected by ingesting raw or undercooked infected meat **1**. In the human intestine, the cysticercus develops over 2 months into an adult tapeworm, which can survive for years. The adult tapeworms attach to the small intestine by their scolex 5 and reside in the small intestine 6. Length of adult worms is usually 5 m or less for T. saginata (however it may reach up to 25 m) and 2 to 7 m for T. solium. The adults produce proglottids which mature, become gravid, detach from the tapeworm, and migrate to the anus or are passed in the stool (approximately 6 per day). T. saginata may produce up to 100,000 and T. solium may produce 50,000 eggs per proglottid respectively.

#### **Clinical Features**

*Taenia saginata* taeniasis produces only mild abdominal symptoms. Occasionally, appendicitis or cholangitis can result from migrating proglottids. *Taenia solium* taeniasis is less frequently symptomatic than *Taenia saginata* taeniasis. The main symptom is often the passage (passive) of proglottids. The most important feature of *Taenia solium* taeniasis is the risk of development of cysticercosis.

#### **CYSTICERCOSIS**



Cysticercosis is an infection of both humans and pigs with the larval stages of the parasitic cestode, *Taenia solium*. This infection is caused by ingestion of eggs shed in the feces of a human tapeworm carrier . These eggs are immediately infectious and do not require a developmental period outside the host. Pigs and humans become infected by ingesting eggs or gravid proglottids , . Humans are usually exposed to eggs by ingestion of food/water contaminated with feces containing these eggs or proglottids or by person-to-person spread. Tapeworm carriers can also infect themselves through fecal-oral transmission (e.g. caused by poor hand hygiene). Once eggs or proglottids are ingested, oncospheres hatch in the intestine , 

invade the intestinal wall, enter the bloodstream, and migrate to multiple tissues and organs

where they mature into cysticerci over 60–70 days 4, 9. Some cysticerci will migrate to the central nervous system, causing serious sequellae (neurocysticercosis).

This differs from **taeniasis**, which is an intestinal infection with the adult tapeworm. Humans acquire intestinal infections with *T. solium* after eating undercooked pork containing cysticerci . Cysts evaginate and attach to the small intestine by their scolices. Adult tapeworms develop to maturity and may reside in the small intestine for years .

### **Clinical features**

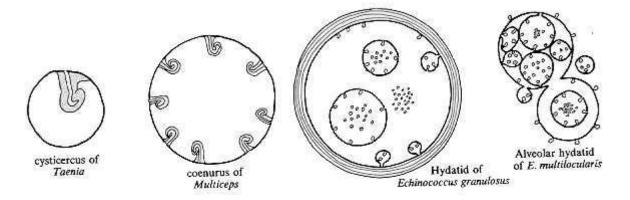
The symptoms of cysticercosis vary depending upon the location and number of cysticerci. Cysticerci may develop in skeletal and heart muscle, skin, subcutaneous tissues, the lungs, liver, and other tissues, including the oral mucosa. In most locations, cysticerci cause few symptoms and spontaneously degenerate.

Cysticerci can migrate to the central nervous system and cause neurocysticercosis (NCC), which is associated with serious neurological and epileptic manifestations. Death can occur suddenly.

### Prevention of taeniasis

- Treatment of infected persons
- Thorough cooking of meat (above 57°C)
- Proper disposal of human excreta

# **Tapeworm cyst types**

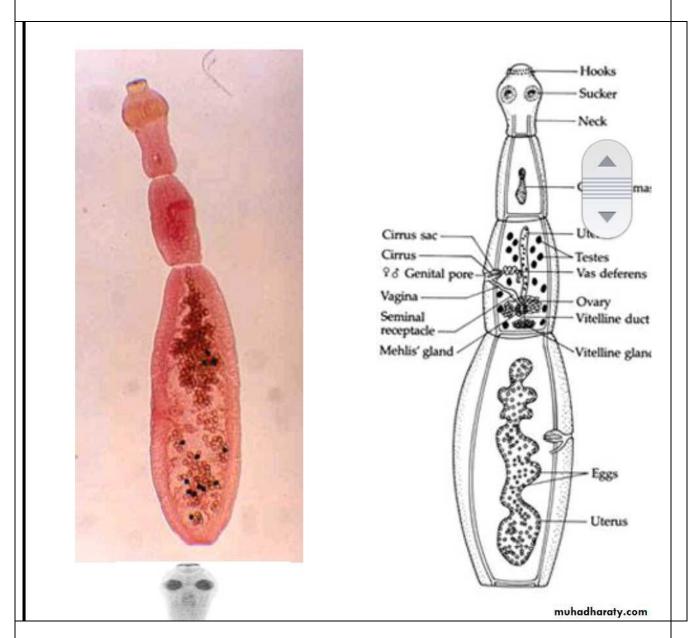


# Assignment

Find four suckers on each scolex of *Taenia* (=*Taeniarhynchus*) *saginata* and *Taenia solium*. The differentiation hooked scolex from the non-hooked scolex of *Taenia* (=*Taeniarhynchus*) *saginata* is of high priority in diagnostic labs. Both mature and gravid proglottids are present.

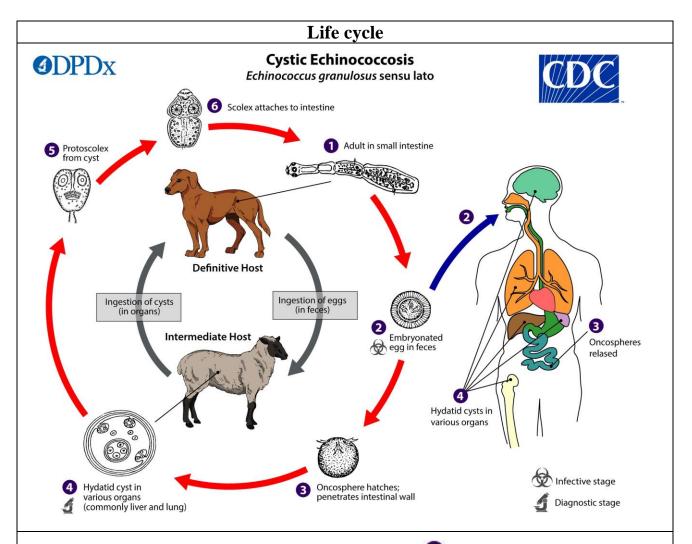
Compare the mature proglottid with pictures find the vitellarium, genital pore, ovary, vagina, testes, uterus, and sperm duct. The gravid proglottids should have a branched uterus filled with eggs, and you should become familiar with how to discern and count the lateral uterine branches in the proglottid. Note that the genital pore is single and lateral on each proglottid.

# Echinococcus granulosus - dog tape worm

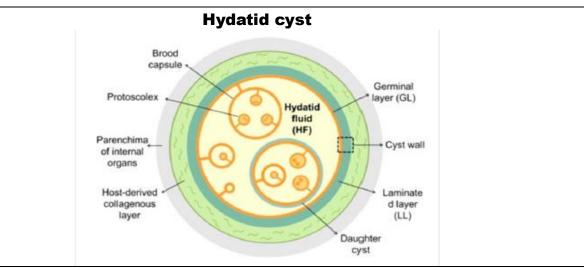


# Morphology

The adult worm measures 3-6 mm in length (up to 1 cm). It has scolex, short neck and strobilla. Scolex is pyriform, with 4 succers and a rostellum bearing 2 circular rows of hooklets. Strobilla composed of only 3 proglottids, the anterior immature, the middle mature and the posterior gravid segment. The terminal proglottid is longer and wider than the rest and contains a branced uterus filled with eggs. Adult worms live in small intestine of definitive host (dog) for 6-30 months. Man is an intermediate host - carrying the hydatid cyst (larva).



The adult *Echinococcus granulosus* (2—7 mm long) resides in the small intestine of the definitive host. Gravid proglottids pass eggs that are passed in the feces, and are immediately infectious. After ingestion by a suitable intermediate host, eggs hatch in the small intestine and release six-hooked oncospheres that penetrate the intestinal wall and migrate through the circulatory system (via portal blood supply) into various organs, especially the liver and lungs. In these organs, the oncosphere develops into a thick-walled hydatid cyst that enlarges gradually, producing protoscolices and daughter cysts that fill the cyst interior.



The definitive host becomes infected by ingesting the cyst-containing organs of the infected intermediate host. After ingestion, the protoscolices evaginate, attach to the intestinal mucosa and develop into adult stages in 32 to 80 days. Humans are aberrant intermediate hosts, and become infected by ingesting eggs. Oncospheres are released in the intestine and hydatid cysts develop in a variety of organs. If cysts rupture, the liberated protoscolices may create secondary cysts in other sites within the body.

**Hosts** are wild and domestic canids.

### **Clinical features**

*Echinococcus granulosus* infections often remain asymptomatic for years before the cysts grow large enough to cause symptoms in the affected organs.

Hepatic and pulmonary signs/symptoms are the most common clinical manifestations. Other organs (spleen, kidneys, heart, bone, and central nervous system, including the brain and eyes) can also be involved, with resulting symptoms. Rupture of the cysts can produce fever, urticaria, eosinophilia, and potentially anaphylactic shock.

### **Prevention**

Avoid handlig and carressing infected dogs and ingestion of water and vegetables polluted by infected dog feces

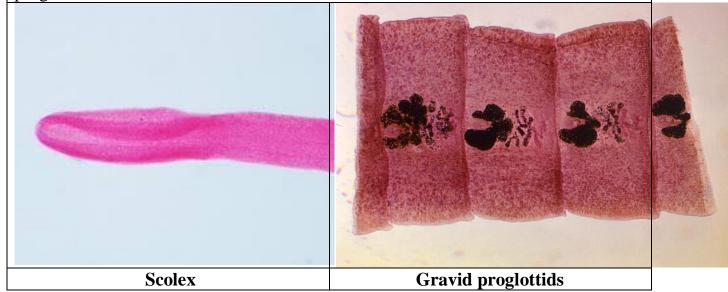
# Dyphillobothrium latum – broad tapeworm

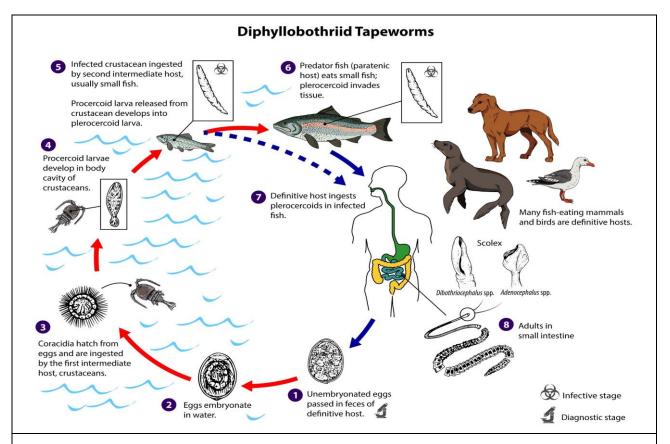
### Morphology

This is a large worm with a scolex with dorsal and ventral longitudinal grooves called bothria.

Characteristically, the width of the mature and gravid proglottids is greater than their length. Terminal proglottids are characterized by the presence of an egg-filled uterus with a few short branches in the middle one-third of the segment, usually described by the central rosette. The proglottids are wider than long and have a characteristic rossette-shaped uterus centrally filled with eggs.

The adultworms can reach more than 10 m in length, with more than 3,000 proglottids.





# Life cycle

Eggs are passed unembryonated in feces ①. Under appropriate conditions, the eggs mature (approximately 18 to 20 days) 2 and yield oncospheres which develop into a coracidia 3. After ingestion by a suitable crustacean (first intermediate host) the coracidia develop into procercoid larvae 4. Procercoid larvae are released from the crustacean upon predation by the second intermediate host (usually a small fish) and migrate into the deeper tissues where they develop into a plerocercoid larvae (spargana), which is the infectious stage for the definitive host . Because humans do not generally eat these small fish species raw, the second intermediate host probably does not represent an important source of human infection. However, these small second intermediate hosts can be eaten by larger predator species that then serve as paratenic hosts 6. In this case, the plerocercoid migrates to the musculature of the larger predator fish; humans (and other definitive host species) acquire the parasite via consumption of undercooked paratenic host fish **7**. In the definitive host, the plerocercoid develops into adult tapeworms in the small intestine. Adult diphyllobothriids attach to the intestinal mucosa by means of two bilateral groves (bothria) of their scolex <sup>3</sup>. Immature eggs are discharged from the proglottids (up to 1,000,000 eggs per day per worm) and are passed in the feces. Eggs appear in the feces 5 to 6 weeks after infection.

### **Hosts**

Intermediate and paratenic hosts include a wide diversity of freshwater and marine fishes (e.g. perch, pike, salmon).

Definitive host specificity among the medically important diphyllobothriids is low. (In addition to humans, other carnivorous, fish-eating mammals and a few birds.)

#### **Clinical Features**

Diphyllobothriasis can be a long-lasting infection, up to 25 years. Most infections are asymptomatic but gastrointestinal symptoms may occur in some patients. Aberrant migration of proglottids can cause cholecystitis or cholangitis. Rarely, massive infections may cause intestinal obstruction.

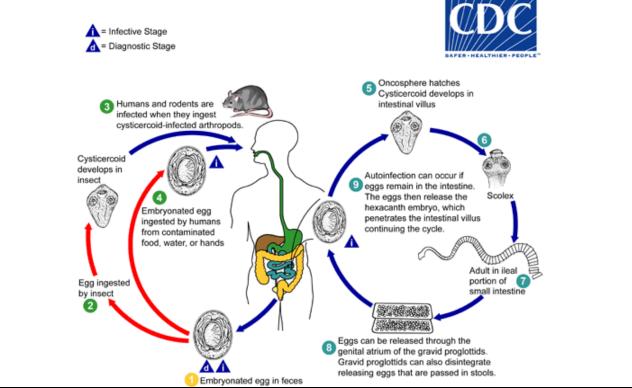
#### **Prevention**

By not eating raw or undercooked fish (for example, sushimi or tasting of fish while cooking).

### 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.



Eggs of *Hymenolepis nana* are immediately infective when passed with the stool and cannot survive more than 10 days in the external environment ①. When eggs are ingested by an arthropod intermediate host ② (various species of beetles and fleas may serve as intermediate hosts), they develop into cysticercoids, which can infect humans or rodents upon ingestion ③ and develop into adults in the small intestine. A morphologically identical variant, *H. nana* var. *fraterna*, infects rodents and uses arthropods as intermediate hosts. When eggs are ingested ④ (in contaminated food or water or from hands contaminated with feces), the oncospheres contained in the eggs are released. The oncospheres (hexacanth larvae)

penetrate the intestinal villus and develop into cysticercoid larvae ⑤. Upon rupture of the villus, the cysticercoids return to the intestinal lumen, evaginate their scoleces ⑥, attach to the intestinal mucosa and develop into adults that reside in the ileal portion of the small intestine producing gravid proglottids ⑦. Eggs are passed in the stool when released from proglottids through its genital atrium or when proglottids disintegrate in the small intestine ③. An alternate mode of infection consists of internal autoinfection, where the eggs release their hexacanth embryo, which penetrates the villus continuing the infective cycle without passage through the external environment ⑨. The life span of adult worms is 4 to 6 weeks, but internal autoinfection allows the infection to persist for years.

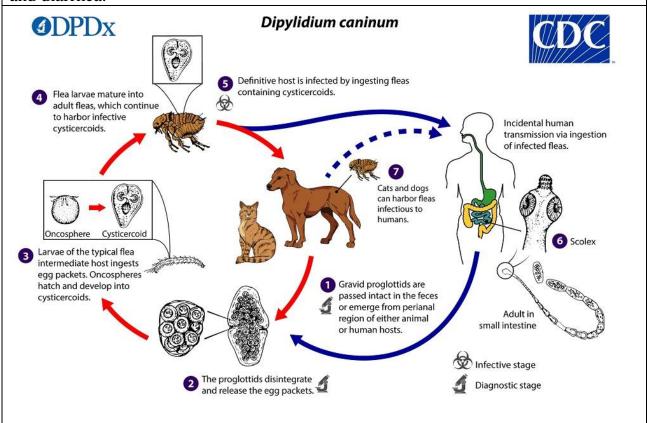
# 

Eggs of *Hymenolepis diminuta* are passed out in the feces of the infected definitive host (rodents, man) ①. The mature eggs are ingested by an intermediate host (various arthropod adults or larvae) ②, and oncospheres are released from the eggs and penetrate the intestinal wall of the host ③, which develop into cysticercoid larvae. Species from the genus *Tribolium* are common intermediate hosts for *H. diminuta*. The cysticercoid larvae persist through the arthropod's morphogenesis to adulthood. *H. diminuta* infection is acquired by the mammalian host after ingestion of an intermediate host carrying the cysticercoid larvae ④. Humans can be accidentally infected through the ingestion of insects in precooked cereals, or other food items, and directly from the environment (e.g., oral exploration of the environment by children). After ingestion, the tissue of the infected arthropod is digested releasing the cysticercoid larvae in the stomach and small intestine. Eversion of the scoleces ⑤ occurs shortly after the cysticercoid larvae are released.

Using the four suckers on the scolex, the parasite attaches to the small intestine wall. Maturation of the parasites occurs within 20 days and the adult worms can reach an average of 30 cm in length ©. Eggs are released in the small intestine from gravid proglottids © that disintegrate after breaking off from the adult worms. The eggs are expelled to the environment in the mammalian host's feces O.

### **Clinical features**

*Hymenolepis nana* and *H. diminuta* infections are most often asymptomatic. Heavy infections with *H. nana* can cause weakness, headaches, anorexia, abdominal pain, and diarrhea.



Gravid proglottids are passed intact in the feces or emerge from the perianal region of the host 
. In the environment, the proglottids disintegrate and release egg packets, which are also occasionally found free in the feces 
. The intermediate host (most often larval stages of the dog or cat flea *Ctenocephalides* spp.) ingests egg packets, and the oncosphere within is released into the larval flea's intestine. The oncosphere penetrates the intestinal wall, invades the insect's hemocoel (body cavity), and develops into a cysticercoid 
. The cysticercoid remains in the flea as it matures from a larva into an adult 
. The vertebrate host becomes infected by ingesting the adult flea containing the cysticercoid 
. In the small intestine of the vertebrate host, the cysticercoid develops into the adult tapeworm after about one month. The adult tapeworms (measuring up to 60 cm in length and 3 mm in width) reside in the small intestine of the host, where they each attach by their scolex 
. Gravid, double-pored proglottids detach from the strobila (body) and are shed in the feces. Humans also acquire infection by ingesting the cysticercoid

contaminated flea. Children are most frequently infected, possibly due to close contact with flea-infested pets 7.

#### Hosts

Canids and felids are the normal hosts for *Dipylidium caninum*. The intermediate host is usually the larval stages of the dog or cat flea (*Ctenocephalides* spp.) and occasionally *Trichodectes canis* (the dog louse).

### **Clinical features**

Most infections with *Dipylidium caninum* are asymptomatic. Mild gastrointestinal disturbances may occur. The most striking feature in animals and children consists of the passage of proglottids. These can be found in the perianal region, in the feces, on diapers, and occasionally on floor covering and furniture. The proglottids are motile when freshly passed and may be mistaken for maggots or fly larvae.

### **Assignment**

On the slide of *Diphyllobothrium latum* find scolex with dorsal and ventral longitudinal grooves called <u>bothria</u>. These grooves may be difficult to discern, and may only appear as longitudinal, lightly stained areas on the scolex.

Gravid proglottids are wider than long and have a characteristic rossette-shaped uterus centrally filled with eggs.

The **dwarf tapeworm** slide: note that it is small and has four suckers and 20-30 hooks on the scolex. Proglottids are wider than long, the genital pores unilateral, and there are three testes per segment

Diphylidium caninum: find the scolex with suckers and grooves. Gravid proglottids

# References

Addis Ababa, Ethiopia. Lynnes. & David A. **Diagnostic Medical parasitology** 3rded. Washington. D.C: ASM press, 1997.

Faller. **Medical microbiology**, 4th ed USA: Mosby, 2002.

Greenwood D. **Helminthes**. In: Greenwood D, Slack R, Peutherer J (eds). Medical Microbiology; 15th edn, Churchill Livingstone, Harcourt Publishing Ltd, London.1997; Pp 587-602,

Heynema D. In: Brooks GF, Butel JS, Morse SA. Jawetz, Menlik and Adelberg's. **Medical Parasitology**, Chapter 46; Appleton and Lang, Stanford, Connecticut, USA Pp 617-651; 21st ed, 1998.

Pavl C. Beaver & Rodney C. Jung & Eddie W. Cupp. Clinical parasitology. 9thed. Philadelphia: Lea & Febiger, 1984.

Shibru Tedla. **Introduction to Parasitology: Protozoan and Helminth parasites of man.** Addis Ababa University Press, 1986

# **Internet resources:**

Alan Gunn, Sarah J. Pitt. **Parasitology. An Integrated Approach**. Publisher: Wiley-Blackwell, 2012. 468 p.

Amirah Amir. **Medical Parasitology.** A Textbook, Springer. 2017. 198 p. DOI: <a href="https://doi.org/10.1007/978-3-319-68795-7\_1">https://doi.org/10.1007/978-3-319-68795-7\_1</a>

**Encyclopedia of Parasitology.** Heinz Mehlhorn (Editor), Springer-Verlag Berlin Heidelberg, 2016. DOI:10.1007/978-3-662-43978-4

Rohela Mahmud, Yvonne Ai Lian Lim, Dawit Assafa Ephrem Kibru, S. Nagesh, Solomon Gebreselassie, Fetene Deribe, Jemal Ali. Lecture notes. Medical parasitology, Ethiopia, 2004. 150 p.

Sures B., Nachev M. (2016) **Environmental Parasitology**. In: Mehlhorn H. (eds) Encyclopedia of Parasitology. Springer, Berlin, Heidelberg. 2016. 136 p. DOI: <a href="https://doi.org/10.1007/978-3-662-43978-4\_1076">https://doi.org/10.1007/978-3-662-43978-4\_1076</a>

https://www.cdc.gov/dpdx

https://www.academia.edu/27407475/LECTURE\_NOTES\_Medical\_Parasitology