# lec 16 MEDICAL HELMINTHOLOGY

## LEARNING OBJECTIVES

At the end of this session, students should be able to:

- Understand medically important helminthes including their life cycles, modes of transmissions, clinical features, diagnosis, treatment and prevention.
- Describe blood, intestinal, liver and lung flukes.
- Understand common round worms.
- Understand different species of Cestodes.

## INTRODUCTION

Medical helminthology is concerned with the study of helminthes or parasitic worms. Helminthes are trophoblastic metazoa (multi-cellular organisms). Helminthes are among the common parasitic causes of human suffering. They are the cause of high morbidity and mortality of people worldwide. They cause different diseases in humans, but few helminthic infections cause life- threatening diseases. They cause anemia and malnutrition. In children they cause a reduction in academic performance. Helminthes also cause economic loss as a result of infections of domestic animals. There is age dependent distribution of infections from geo-helminthes and schistosomes. As a result of predisposing behavioral and immunological status, children disproportionately carry the burden of schistosomes and geo-helminthes.

The sources of the parasites are different. Exposure of humans to the parasites may occur in one of the following ways:

1. Contaminated soil (Geo-helminthes), water (cercariae of blood flukes) and food (Taenia in raw meat).
2. Blood sucking insects or arthropods (as in filarial worms).
3. Domestic or wild animals harboring the parasite (as in echinococcus in dogs).
4. Person to person (as in *Enterobius vermicularis, Hymenolopis nana*).
5. Oneself (auto-infection) as in *Enterobius vermicularis.*

They enter the body through different routes including: mouth, skin and the respiratory tract by means of inhalation of airborne eggs.

The helminthes are classified into three major groups. These are:

1. Trematodes (Flukes)
2. Nematodes (Round worms)
3. Cestodes (Tape worms)

| | Cestodes | Trematodes | Nematodes |
|---|---|---|---|
| Shape | Tape-like, segmented | Leaf-like unsegmented | Elongated, cylindrical, unsegmented |
| Head end | Suckers present; some have attached hooks | Suckers are present but no hooks | Hooks and sucker absent. Well- developed buccal capsule with teeth or cutting plates seen in some species |
| Alimentary canal | Absent | Present but incomplete, no anus | Complete with anus |
| Body cavity | Absent, but inside is filled with spongy undifferentiated mesenchymatous cells, in the midst of which lie the viscera | Same as cestodes | Present and known as **pseudocele**. Viscera remains suspended in the pseudocele |
| Sex | Not separate: hermaphrodite (monecious) | Not separate: hermaphrodite except *Schistsoma* | Separate (diecious) |
| Life cycle | Requires 2 host except *Hymenolepis* (1 host) and *Diphyllobothrum* (3 host) | Requires 3 host except schistosomes (2 host) | Requires 1 host except filarial worms (2 host) and *Dracunculus* (2 host) |

**Phylum**: platy-helminthes

**class** :cestoda

**genus** : Taenia

**species** : **<u>Taenia -saginata , Taenta –solium</u>**

Common name :(beef tapeworm) , (pork tapeworm ) Disease
: taenasis

*Taenia.saginata* , *Taenia-solium* the largest of species in the genus of taenia . the adult normally 4 -10-M in length, but can become very large over 22M long are reported.

It zoonotic disease , in small intestinal parasite where the human harboring the adult as definitive host and cattle are intermediated host where larval development occur .

typical of cestoda the body ribbon-like ,white in color , is flattened.

-dorsoventrally and heavily segment the body consists three portion.

1- scolex ( head )

2- neck

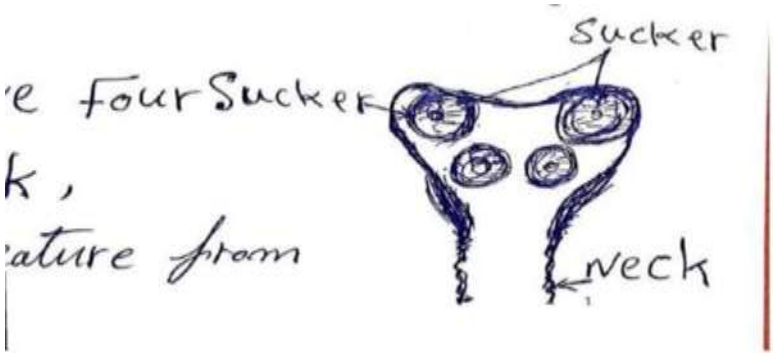
3- Trunk ,proglottid ( segment ) strobili Taenia –saginata

-the scolex (head ) have four sucker i n T - s a g i n a t a without hook identifying feature from other taenia ,the sucker used for attachment in wall of small intestine,

Taenia -solium have four sucker and rostellum armed with hook

**The neck** in both are short consist from the germ layer and immature proglottids (segment)

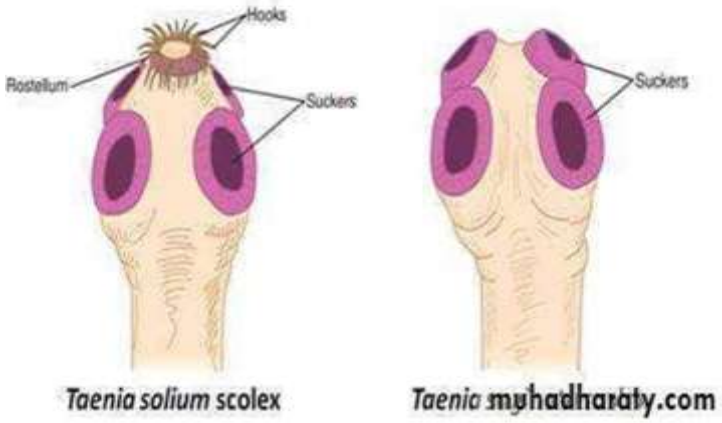
The last body consist from mature segment and gravid proglottids, and have about 1000-2000 proglottids i n **Taenia –saginata and Taenia- solium** about 800- 1000 segment , doesn't contain digestive system from mouth to anus , it derives the nutrient from the host by tegument cells




Tegument cells cover with absorptive hair for absorption of nutrient . and each segments have good development reproduction system consist from testes and ovary, uterus, vagina and genital pore and vitellaria ( yolk –gland) its (hermaphrodite ) each proglottids carries a set of female and male

reproduction organs

also the T-saginata and T-solium have nerve center composed from ganglion in scolex and the small fiber nerve supply the general body




<u>The mature proglottids</u>

In both worm composed from uterus , ovary ,vagina , and genital pore ( female organ ) and vitellaria(yolk)

The male organ . testes ,seminal duct

Differences between heads and proglottids of various Cestodes

| | *Taenia solium* | *Taenia saginata* | *Hymenolepis nana* | *Hymenolepis diminuta* | *Diphyllobothrium latum* | *Echinococcus granulosus* |
|---|---|---|---|---|---|---|
| **Heads** |  |  |  |  |  |  |
| **Proglottids** | 4 suckers<br>2 rows of hooks | 4 suckers<br>No hooks | 4 suckers<br>single row of 20–30 hooks | 4 suckers<br>No hooks | 2 Suctorial grooves or bothria No suckers. No hooks | 4 suckers<br>2 rows of hooks |
| |  |  |  |  |  |  |
| | Longer than broad<br>7–12 uterine branches on each side | Longer than broad<br>15–30 uterine branches on each side | Broader than long | Broader than long | Broader than long<br>Uterus coiled | Longer than broad |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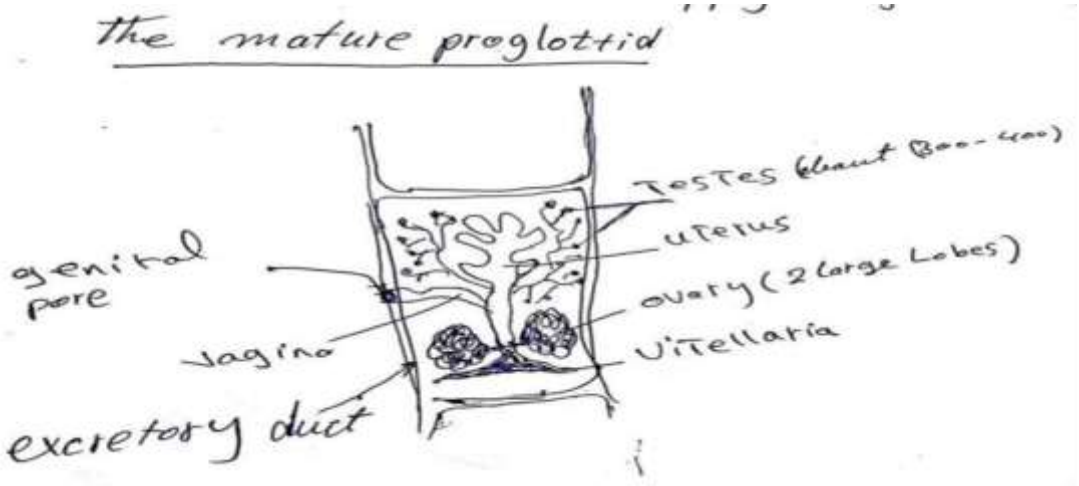
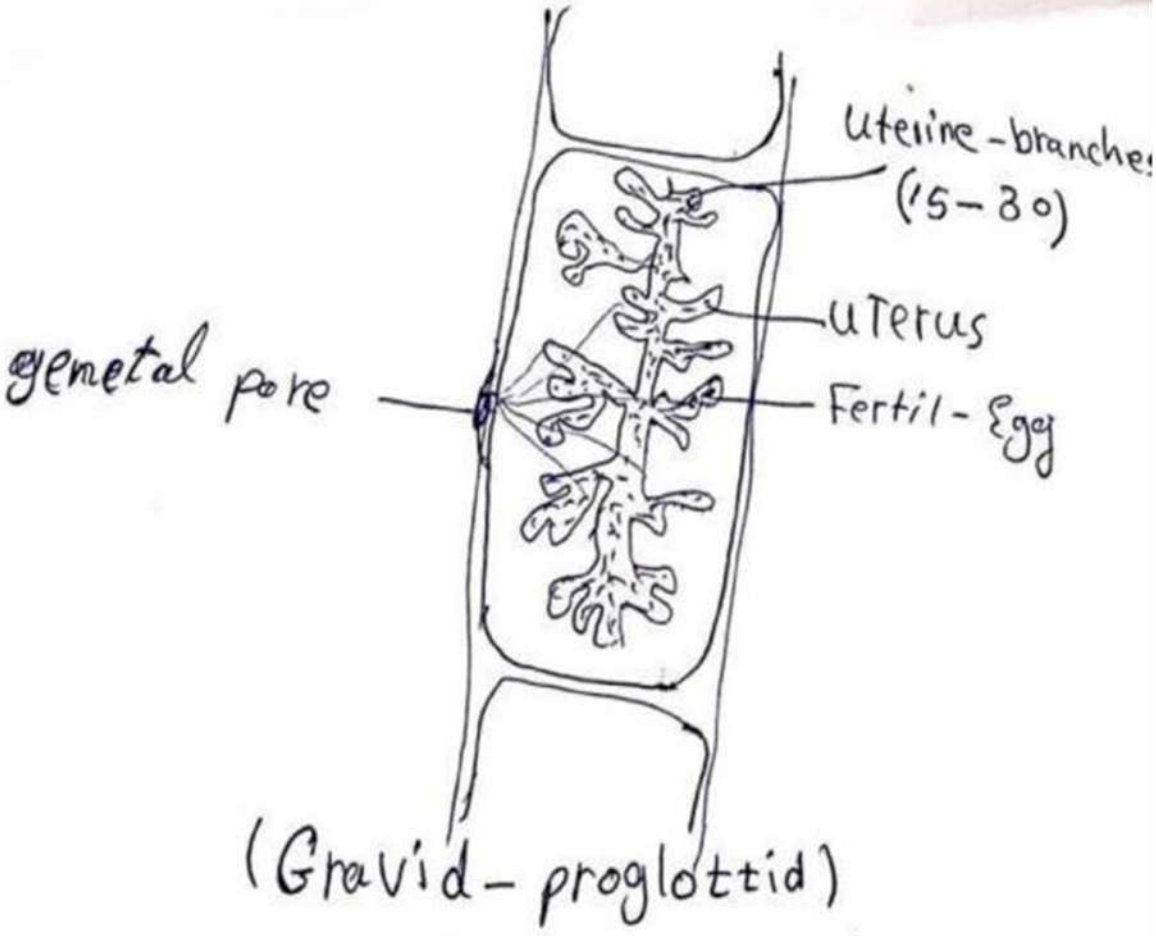
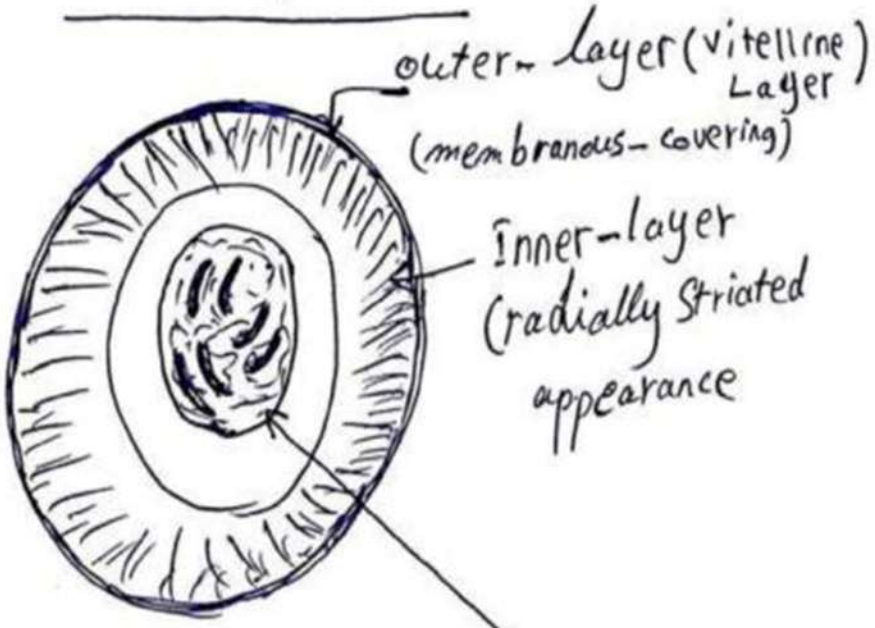

the mature proglottid
Testes (about 800 - 400)
uterus
ovary (2 large Lobes)
Vitellaria
genital pore
Vagina
excretory duct




(Gravid-proglottid)



**Infective stage :**

1- Larva stage ( cysticercus -bovis ) in the infected meat of cattle

2- Larval stage (cysticercus-cellulosae)in the infected meat of pig ,also They cause cysticercusis disease in humane ( T- solium )

1- Human acquired the eggs of Taenia .Solium by

a- Auto infection. Gravid segment which rupture with in the definitive host, in the small intestine, hatching the eggs which contain hexacnthembryo , produce Encosphere , which migrate to the host tissue via blood stream to form cysticercusis disease in human specially in eye ,brain, and muscular tissue

b-Accidental ingestion eggs of **Taenia -solium** with contaminated food and water with eggs, the eggs loss their outer covering in digestive system(i n s m a l l i n t e s t i n e )to developing the encosphere, migrat by perforation the wall of small intestine ,via blood stream to form cysticrecus in all body specially in brain ,eye and muscular system cause cysticercus disease in human .

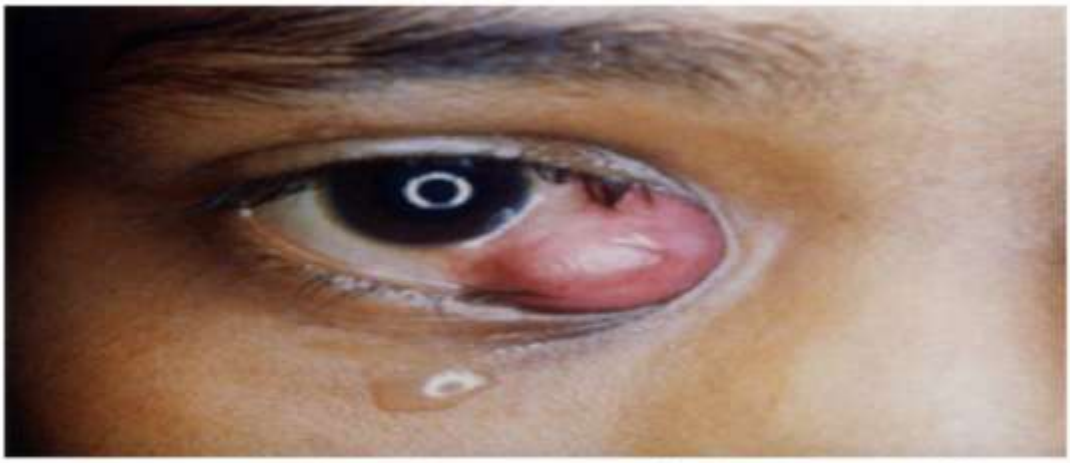
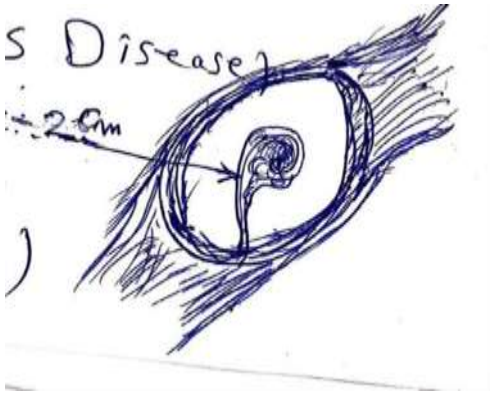


Fig. 1: Rt eye showing smooth, horizontally oval subconjunctival swelling.




**Life cycle**

The life cycle -**Taenia -saginata and Taenia -Solium** are indirect, human are definitive host

*cysticercus in muscle of cattle or pig as intermediate host .

1-Human infected by ingestion raw or undercooked infected meat

. contain the cysticercus

2- Due to digestive enzymes In small intestine release the scolex (head) and attaches to small intestine

3- After 2-3 month become adult in small intestine

4- Eggs or gravid segment (proglottids ) passed with feces

5- The eggs can survive days to months in the environment

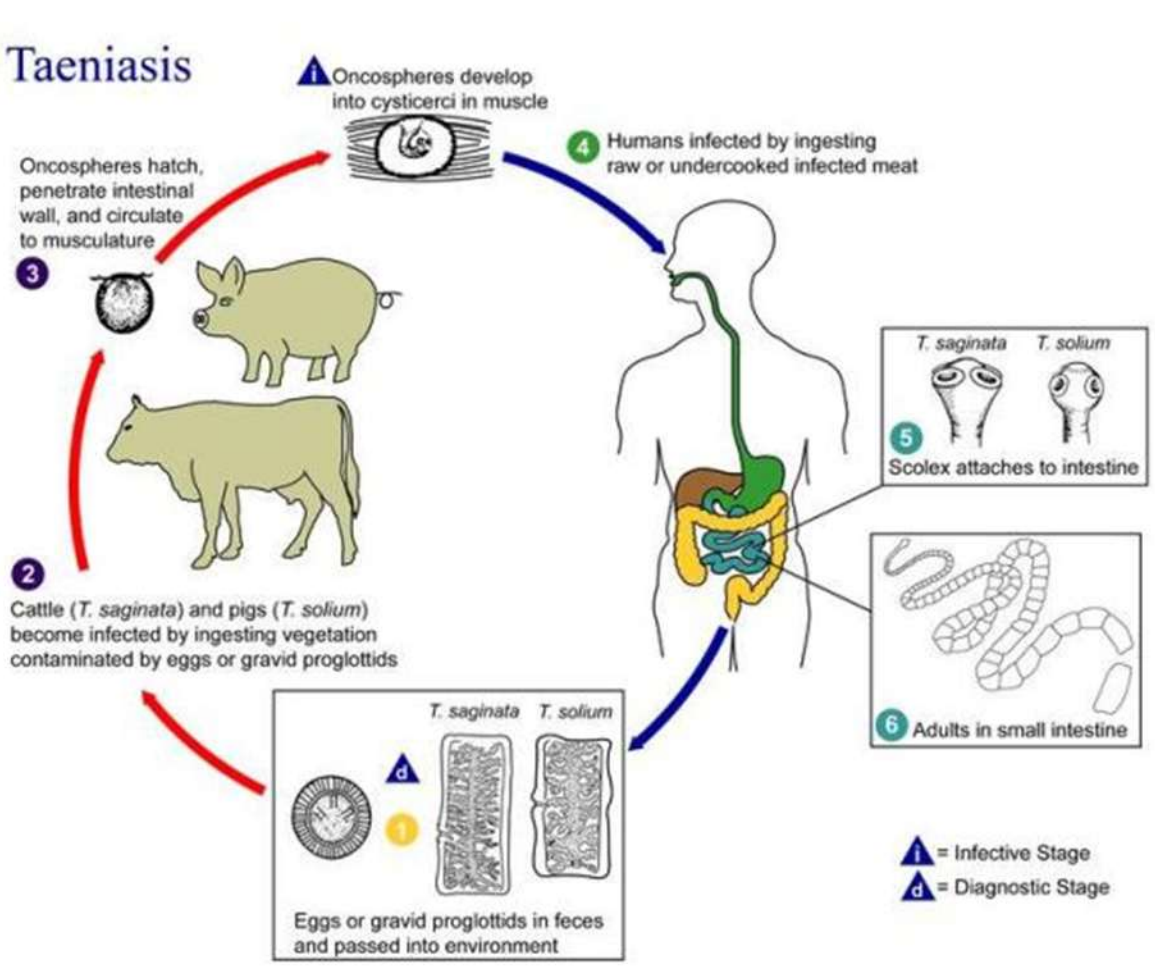
6- Cattle and pigs, become infected ingesting vegetation (plants ) contamination with eggs or gravid proglottids, , in the small intestine the eggs hatch, hexacnth embryo invade the intestinal wall and migrate to the striated muscles , where they develop hinto cysticercus

7-(cysticercus - bovis ) develop in muscular system or organ of the cattle (cysticercus - bovis ),

(cysticercus - cellulosa ) in muscular system of the pig ,
cysticercus can survive for several years in the animal
8-Human become infected by ingesting raw or undercooked infected meat contain the cysticercusis

In the human. in the intestine the cysticercus develops over 2- 3 month into adult tapeworm , which can survive years

the gravid proglottids are released with feces, in the T.saginata may produce up to 100, 000 eggs and in the T.Solium 50,000 eggs per proglottids


Taeniasis
Oncospheres develop into cysticerci in muscle
Humans infected by ingesting raw or undercooked infected meat
Oncospheres hatch, penetrate intestinal wall, and circulate to musculature
3
T. saginata
T. solium
5
Scolex attaches to intestine
2
Cattle (T. saginata) and pigs (T. solium) become infected by ingesting vegetation contaminated by eggs or gravid proglottids
6
Adults in small intestine
T. saginata
T. solium
d
1
Eggs or gravid proglottids in feces and passed into environment
= Infective Stage
= Diagnostic Stage

The different between T-saginata and T-solium

| | *Taenia saginata* | *Taenia solium* |
|---|---|---|
| Length | 5–10 m | 2–3 m |
| Scolex | Large quadrate | Small and globular |
| | Rostellum and hooks are absent | Rostellum and hooks are present |
| | Suckers may be pigmented | Suckers not pigmented |
| Neck | Long | Short |
| Proglottids | 1,000–2,000 | Below 1,000 |
| Measurement (gravid segment) | 20 mm × 5 mm | 12 mm × 6 mm |
| Expulsion | Expelled singly | Expelled passively in chains of 5 or 6 |
| Uterus | Lateral branches15–30 on each side; thin and dichotomous | Lateral branches 5–10 on each side; thick and dendritic |
| Vagina | Present | Absent |
| Accessary lobeof ovary | Absent | Present |
| Testes | 300–400 follicles | 150–200 follicles |
| Larva | Cysticercus bovis; present in cow not in man | Cysticercus cellulosae; present in pig and alsoin man |
| Egg | Not infective toman | Infective to man |
| Definitive host | Man | Man |
| Intermediate host | Cow | Pig, occasionally man |
| Disease | Causes intestinal taeniasis | Causes intestinal taeniasis and cysticercosis |
| | | |

**<u>Clinical Symptom</u>** :

The main symptom tapeworm infection in human :

1- cause digestive problem , abdominal pain loss of appetite , weight loss, anemia , intestinal obstruction .

2- the most visible symptom of taeniasis in active passing of proglottids through the anus and in feces .

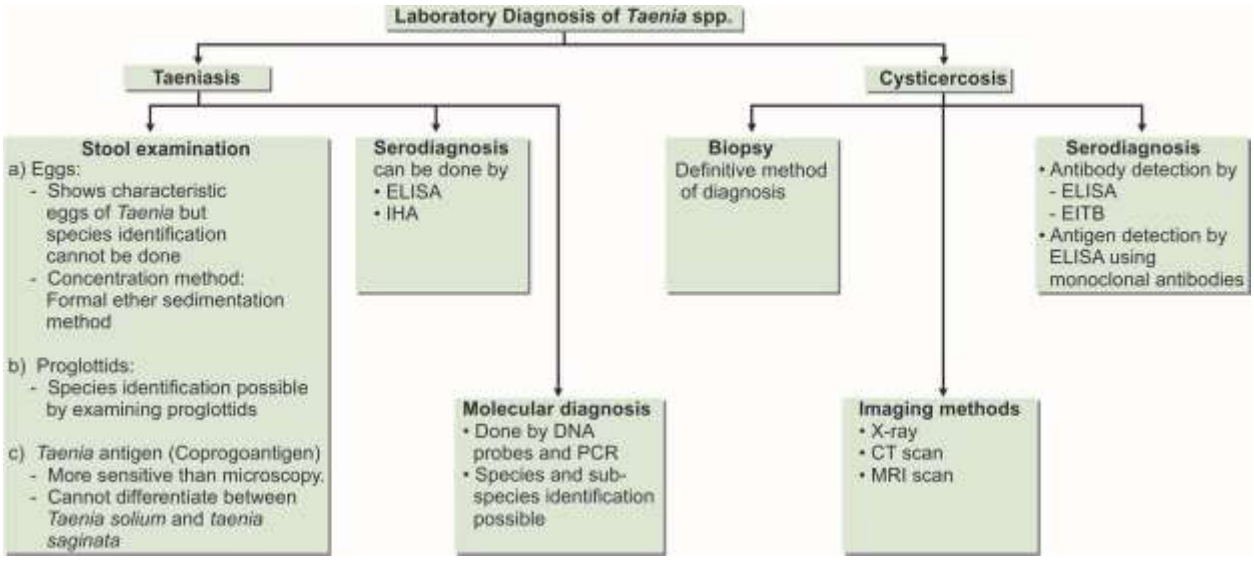
3- infection with Taenia-Solium can result in human **cysticercosis disease** , which can be cysticercus- c e l l u l o s a .

The larva Stage me be subcutaneous, muscular or in all organs of the body also in ,neurocycticercosis ,and ocular (loss of vision ) and conjunctivitis

**very serious disease infected the eye or brain damage**

## diagnosis

Laboratory diagnosis of *Taenia* spp.




## Treatment

Single dose of praziquantel (10–20 mg/kg) is the drug ofchoice.

- Niclosamide (2 g), single dose, is another effective drug.
- Purgation is not considered necessary.

**Lec17**

# Hymenolepis Nana

***Common name:*** Dwarf tape worm

## Habitat

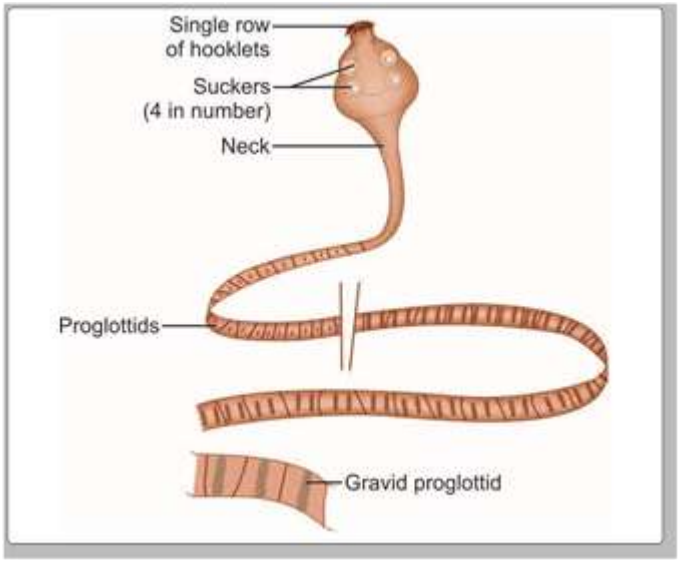
The adult worm lives in the proximal ileum of man. *H. nana var. fraterna* is found in rodents like mice and rats, where they are found in the posterior part of the ileum.

## Morphology

*Adult Worm*

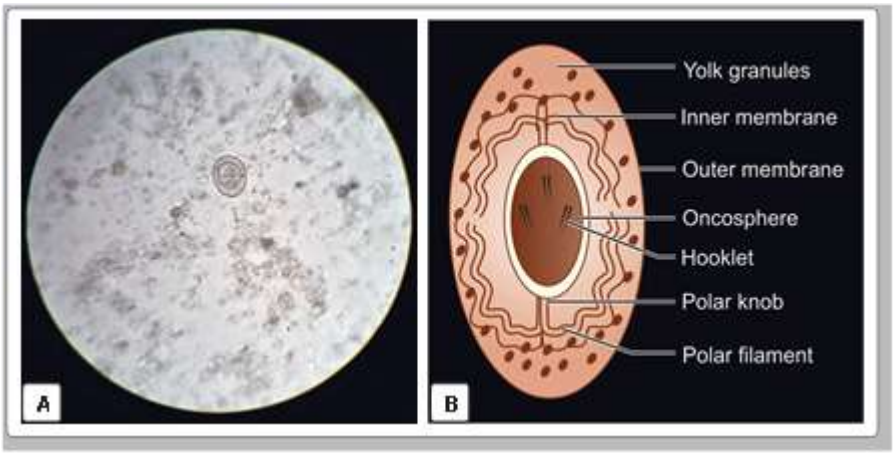
*H. nana* is the smallest intestinal cestode that infects man.

- It is 5–45 mm in length and less than 1 mm thick. The *scolex* has 4 suckers and a retractile rostellum with a single row of hooklets
-




Adult worm of *Hymenolepis nana*

Egg of *Hymenolepis nana.* **A.** As seen under microscope; **B.** Schematic diagram



## Life Cycle

**Host:** Man.

- There is no intermediate host.
- **Mode of transmission:** Infection occurs by ingestion of the food and water contaminated with eggs.
  - **Internal autoinfection** may also occur when the eggs released in the intestine hatch there itself (Fig. 12.25).
  - **External autoinfection** occurs when a person ingest own eggs by fecal oral route.

- *H. nana* is unusual in that it undergoes multiplication in the body of the definitive host.
- When the eggs are swallowed, or in internal autoinfection, they hatch in the small intestine.
- The **hexacanth embryo** penetrates the intestinal villus and develops into the cysticercoid larva.
- This is a solid pyriform structure, with the vesicular anterior end containing the invaginated scolex and a short conical posterior end.
- After about 4 days, the mature larva emerging out of the villus evaginates its scolex and attaches to the mucosae.
- It starts strobilization, to become the mature worm, which begins producing eggs in about 25 days.

A different strain of *H. nana* infects rats and mice. The eggs passed in rodent feces are ingested by rat fleas (*Xenopsylla cheopis* and others), which acts as the intermediate host. The eggs develop into cysticercoid larvae in the hemocele of these insects. Rodents get infected when they eat these insects. The murine strain does not appear to infect man. However, the human strain

may infect rodents, which may, therefore, constitute a subsidiary reservoir of infection forthe human parasite.

## Clinical Features

**Hymenolopiasis** occurs more commonly in children.

- There are usually no symptoms but in heavy infections, there is nausea, anorexia, abdominal pain, diarrhea, andirritability.
- Sometimes pruritus may occur due to an allergic response.

### Laboratory Diagnosis

The diagnosis is made by demonstration of characteristic eggs in feces by direct microscopy. Concentration methods like salt flotation and formalin ether may be readily used. ELISA test has been developed with 80% sensitivity.

### Treatment

Praziquantel (single dose of 25 mg/kg) is the drug of choice, since it acts both against the adult worms and thecysticercoids in the intestinal villi.

- Nitazoxanide 500 mg BD for 3 days may be used as alternative.

## Hymenolepis Diminuta

differs from *Hymenolepis nana* in that:

- The adult worm measures about 10-60 cm
- The rosetellum on the head has no hooks
- In the mature segment, there are two testes at one side and another testis on the other side.

**Life cycle**

The adult worms are present in the small intestine of man and rats. Eggs

passed in stool are similar to the eggs of *H. nana* but are brown in color with no polar filaments arising from the polar thickening. The eggs are ingested by the rat flea where they develop to cysticercoid stage. Infection to man takes place accidentally by food or contaminated hands by cysticercoid stage.

**Pathogenecity**

Most infections are asymptomatic, but occasionally, patients may present with nausea, anorexia and diarrhea.

**Treatment**

same as *Hymenolepis nana*.

# Dipylidium Caninum

This common tapeworm of dogs and cats, it may accidentally cause human infection, mainly in children.

## Morphology

- The adult worm in the intestine is about 10–70 cm long
- The scolex has 4 prominent suckers and a retractile rostellum with upto 7 rows of spines (Fig. 12.26).
- The mature proglottid has 2 *genital pores*, 1 on either side, hence the name *Dipylidium* (*dipylos—2 entrances*).
- Gravid proglottids are passed out of the anus of the host singly or in groups.

## Life Cycle

**Definitive host:** Dogs, cats, and rarely man.

**Intermediate host:** Fleas.

- Man acquires infection by ingestion of flea harboring cysticercoid larva.
- The eggs or proglottids passed in feces of dogs and cats are eaten by larval stages of dog and cat fleas, *Ctenocephalus canis* and *C. felis*.

The embryo develops into a tailed cysticercoid larva

## Clinical Features

Human infection is generally asymptomatic, but the actively motile proglottids passed in stools may raise an alarm.

## Diagnosis

The diagnosis is made by detection of proglottids or eggs in stool.

## Treatment

The drug of choice is praziquantel.

## *Class Cestoda*

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## *Diphyllobotherium latum*

General information's

1-life cycle: in direct life cycle •

2-final host: Man •

3-Intermediat host : need 2 •

First one ___cyclop •

Second one _____Fish(fresh water fish ) •

4-infective stage :plerocercoid •

5-Disease name: Fish tape worm disease •

Morphology: •

A-Scolex elongated •

B-has no rostlellum and no hooks •

C-Large tape worm may reach to 10 M •

D-egg is oval in shape operculated and has hexacanth embryo, the color of • worm is Ivory

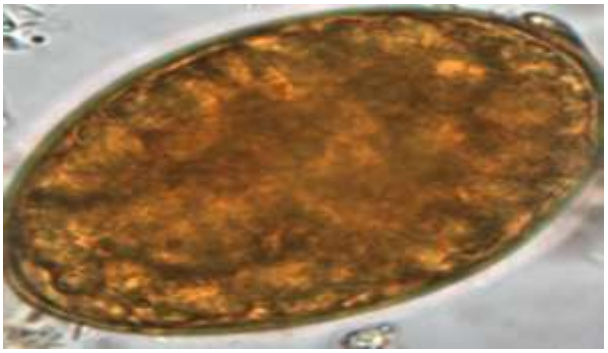
•

Microscopy

Eggs

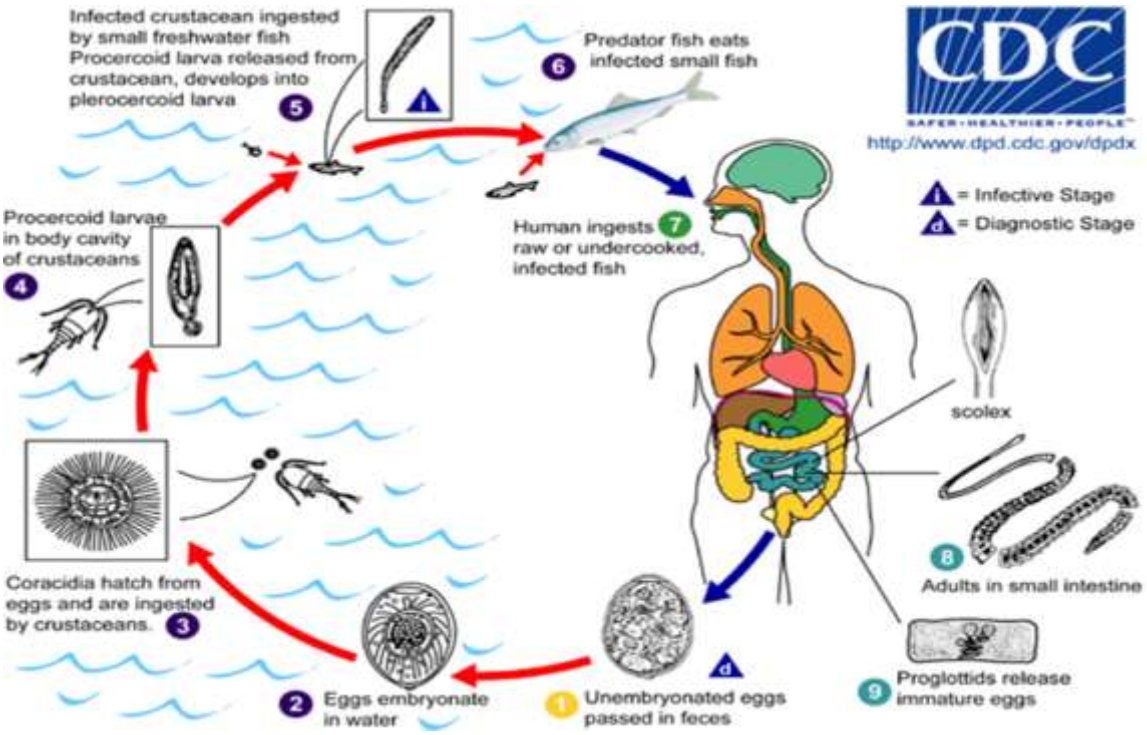
Diphyllobothrium spp. eggs are oval or ellipsoidal and range in size from 55 to 75 µm by 40 to 50 µm. There is an operculum at one end that can be inconspicuous, and at the opposite (abopercular) end is a small knob that can be barely discernible. The eggs

are passed in the stool unembryonated



The cestode *Diphyllobothrium latum* (the fish or broad tapeworm), the largest human tapeworm. Several other *Diphyllobothrium* species have been reported to infect humans, but less frequently; they include *D. pacificum*, *D. cordatum*, *D. ursi*, *D. dendriticum*, *D. lanceolatum*, *D. dalliae*, and *D. yonagoensis*.

**Life Cycle:**



Pathogenesis

. **1-GIT disturbance such as (diarrhea , abdominal pain ,vomiting ,Intestinal obstruction and may occurs appendicitis.**

**2-cause decrease of Vit.B12 in the blood.**

**3-cause Megaloplastic anemia or called (Cephalous anemia)**

**4-worme may secrete toxic material .**

## Diagnosis

Laboratory Diagnosis:

Microscopic identification of eggs in the stool is the basis of specific diagnosis. Eggs are usually numerous and can be demonstrated without concentration techniques. Examination of proglottids passed in the stool is also of diagnostic value.

Diagnostic findings

Microscopy

Morphologic comparison with other intestinal parasites

## Lec 19 Echinococcus Granulosus

***Common name:*** Dog tape worm

### Habitat

- The adult worm lives in the jejunum and duodenum of dogs and other canine carnivora (wolf and fox).
- The larval stage (hydatid cyst) is found in humans and herbivorus animals (sheep, goat, cattle and horse).

### Morphology

*Adult Worm*

It is a small tapeworm, measuring only 3–6 mm in length.

- It consists of a scolex, a short neck, and strobila.
- The scolex is **pyriform,** with 4 suckers and a prominentrostellum bearing 2 circular rows of hooklets (25–30).

- The neck is short than the rest of the worm (3 mm ×6 mm).
- The strobila is composed of only 3 proglottids, the anterior immature, the middle mature, and the posteriorgravid segment (Fig. 12.15).
- The terminal proglottid is longer and wider than the restof the worm and contains a branched uterus filled witheggs.
- The adult worm lives for 6–30 months.

*Egg*

- The eggs of *Echinococcus* are indistinguishable fromthose of *Taenia* species.
- It is ovoid in shape and brown in color.
- It contains an embryo with 3 pairs of hooklets.

*Larval Form*

The larval form is found within the hydatid cyst developing inside various organs of the intermediate host.

- It represents the structure of the scolex of adult worm and remains **invaginated** within a vesicular body.
- After entering the definitive host, the scolex with suckers and rostellar hooklets, becomes **exvaginated** and develops into adult worm.

## Life Cycle

**Definitive hosts:** Dog (optimal host), wolf, jackal, and fox

**Intermediate host:** Sheep and Cattle. Sheep is the ideal intermediate host.

- Man acts as an accidental intermediate host (dead end).
- The larval stage of the parasite is passed in intermediate hosts, including man, giving rise to hydatid cyst.

- The adult worm lives in the small intestine of dogs and other canine animals. These animals discharge numerous eggs in the feces.
- Intermediate hosts (sheep and cattle) ingest them while grazing.
- Human infection follows ingestion of the eggs due to intimate handling of infected dogs or by eating raw vegetables or other food items contaminated with dog feces.
- The ova ingested by man or by sheep and cattle are liberated from the chitinous wall by gastric juice liberating the **hexacanth embryos** which penetrate the intestinal wall and enter the **portal venules**, to be carried to the liver along the portal circulation.
- These are trapped in hepatic sinusoids, where they eventually develop into hydatid cyst. About 75% of hydatid cyst develop in liver, which acts as the first

filterfor embryo.

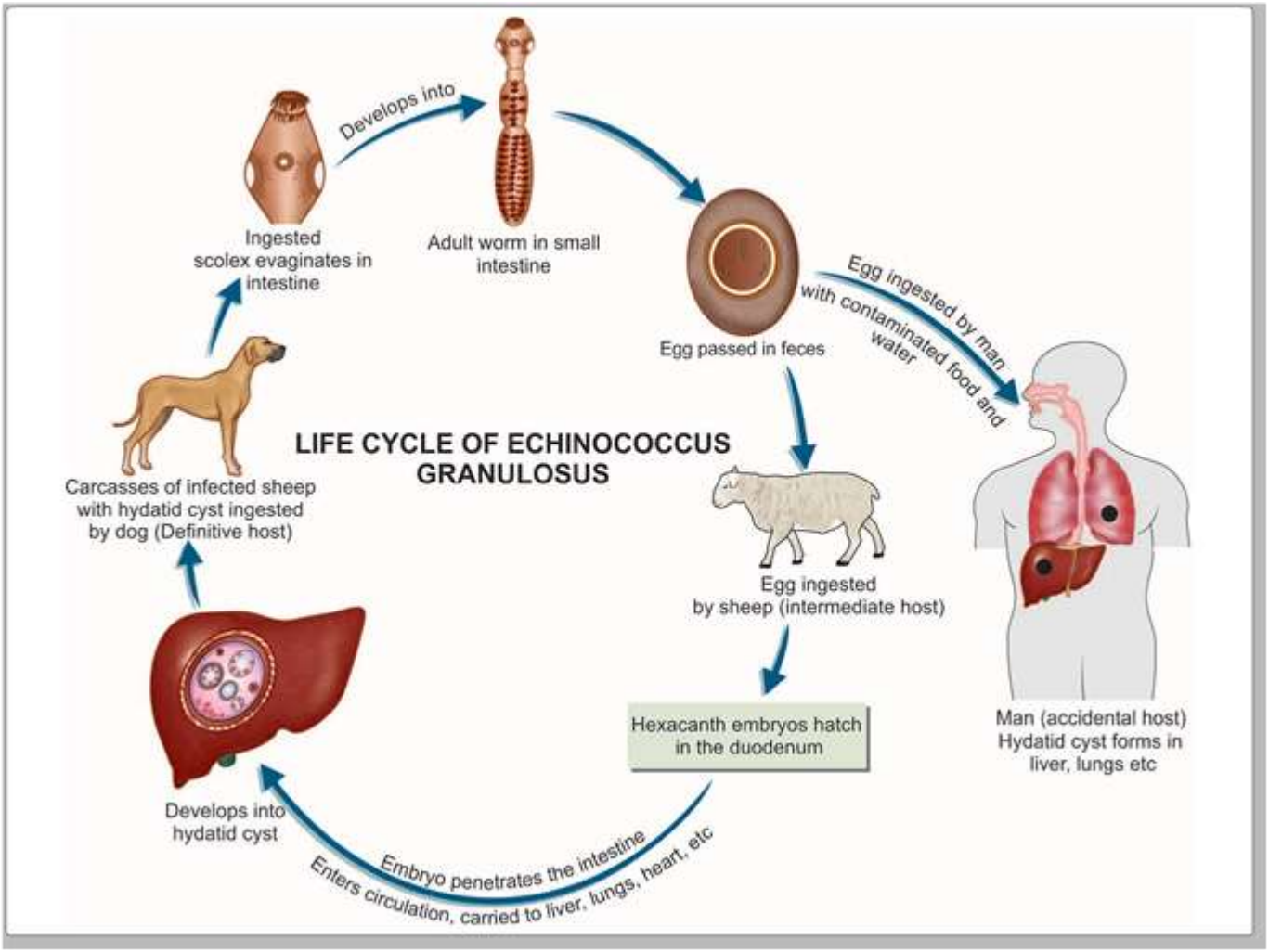
- However, some embryo which pass through the liver, enter the right side of heart and are caught in **pulmonary capillaries** (forming pulmonary hydatid cysts), so that the lung acts as the **second filter**.
- A few enter the systemic circulation and get lodged in various other organs and tissues such as the spleen, kidneys, eyes, brain, or bones.
- When sheep or cattle harboring hydatid cysts die or are slaughtered, dogs may feed on the carcass or offal. Inside the intestine of dogs, the scolices develop into the adult worms that mature in about 6–7 weeks and produce eggs to repeat the life cycle.
- When infection occurs in humans accidentaly, the cycle comes to a dead end because the human hydatid cystsare unlikely to be eaten by dogs.

## Pathogenesis

*Evolution of Hydatid Cyst*

At the site of deposition, the embryo slowly develops intoa hollow bladder or cyst filled with fluid .This becomes the hydatid cyst (Greek *hydatis: a drop of water*).

- It enlarges slowly and reaches a diameter of 0.5–1 cmin about 6 months. The growing cyst evokes host tissue reaction leading to the deposition of fibrous capsule around it.




Life cycle of *Echinococcus granulosus*

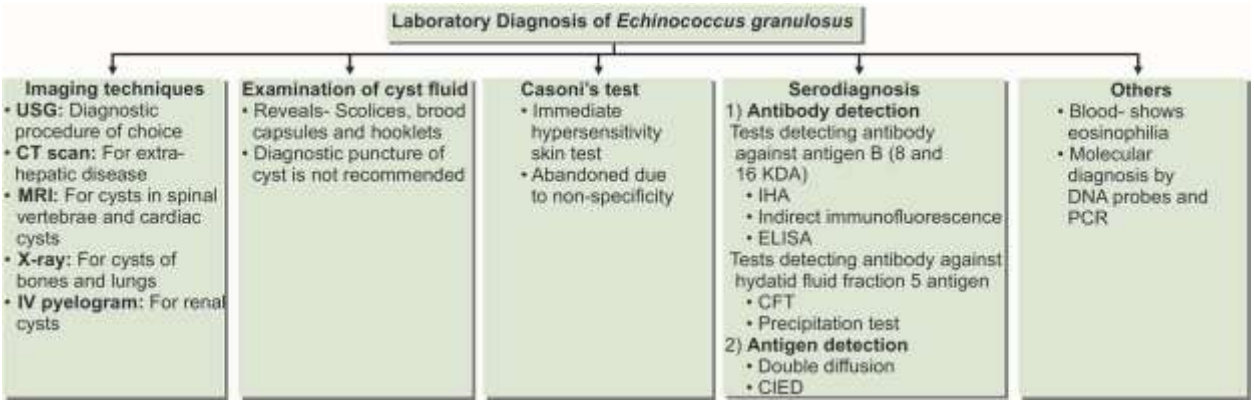
## Clinical Features

- Most of the times infection is asymptomatic and accidentally discovered.
- Clinical disease develops only when the hydatid cyst has grown big enough to cause obstructive symptoms. Disease results mainly from pressure effects caused bythe enlarging cysts.
- In about half the cases, the primary hydatid cyst occurs in **liver** (63%) mostly in the right lobe. Hepatomegaly, pain, and obstructive jaundice are the usual mainfestations.

- The next common site is the **lung** (25%) (most common being the lower lobe of the right lung). Cough, hemoptysis, chest pain, pneumothorax, and dyspnea constitute the clinical picture.
- In the kidney (2%), hydatid cyst causes pain and hematuria.
- **Other sites** affected include spleen (1%), brain (1%), pelvic organs, orbit, and bones (3%).

    - Cerebral hydatid cysts may present as focal epilepsy
    - When hydatid cyst is formed inside the **bones**, the laminated layer is not well developed because of confinement by dense osseous tissues. The parasite migrates along the bony canals as naked excrescences that erode the bone tissue. This is called osseous hydatid cyst. Erosion of bone may lead to pathological fractures.

- Apart from pressure effects, another pathogenic mechanism in hydatid disease is **hypersensitivity** to the echinococcal antigen. The host is sensitized to the antigen by minute amounts of hydatid fluid seeping through the capsule. Hypersensitivity may cause **urticaria**. But if a hydatid cyst ruptures spontaneously or during surgical interference, massive release of hydatid fluid may cause severe, even **fatal anaphylaxis.**



## Treatment

**Traditionally** surgical removal was considered as the thebest mode of treatment of cysts. Currently, ultrasound staging is recommended and management depends on thestage.

In early stages, the treatment of choice is punture, aspiration, injection, and reaspiration (PAIR).

*Puncture, Aspiration, Injection, and Reaspiration(PAIR)*

PAIR, considered as a controversial procedure earlier, is now widely used in early stages of the disease.

# lec 20 Trematodes: Flukes

**Trematodes** are unsegmented helminths, which are flat and broad, resembling the leaf of a tree or a flatfish (hence the name *Fluke*, from the *Anglo-saxon* word '*floc'* meaning'*flatfish'*). The name **Trematode** comes from their having large prominent suckers with a hole in the middle (Greek*trema: hole, eidos*: *appearance*).

## Classification Based on Habitat

Based on habitat, trematodes can be classified as

i. Blood flukes
ii. Liver flukes
iii. Intestinal flukes
iv. Lung flukes

**Zoological Classification of Trematodes**

| Superfamily | Family | Genus | Species |
|---|---|---|---|
| Schistosomatoidea | Schistosomatidae | *Schistosoma* | *S. haematobium*<br>*S. mansoni*<br>*S. japonicum*<br>*S. mekongi*<br>*S. intercalatum* |
| Paramphistomatoidea | Zygocotylidae | *Gastrodiscoides*<br>*Watsoniu s* | *G. hominis*<br>*W. watsoni* |
| Echinostomatoidea | Fasciolidae | *Fascio la*<br>*Fasciol opsis* | *F. hepatica*<br>*F. buski* |
| Opisthorchioidea | Opisthorchiidae<br><br><br>Heterophyidae | *Opisthorchis*<br><br>*Clonorc his*<br>*Heterop hyes*<br>*Metagon imus* | *O. felineus*<br>*O. viverrini*<br>*C. sinensis*<br>*H. heterophyes*<br>*M. yokogawai* |
| Plagiorchioidea | Paragonimidae | *Paragonimus* | *P. westermani* |
| | | | |

**Classification based on Habitat**

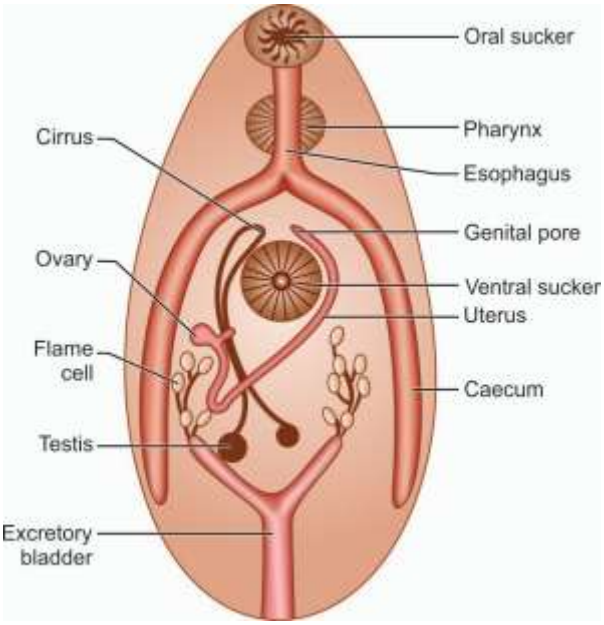
| Habitat | Trematodes |
|---|---|
| Blood (Blood fluke) | *Schistosoma haematobium* (In the vesical and pelvic venous plexuses)<br>*Schistosoma mansoni* (In the inferior mesenteric vein)<br>*Schistosoma japonicum* (In the superior mesenteric vein) |
| Biliary tract (Liver fluke) | *Clonorchis sinensis*<br>*Fasciola hepatica*<br>*Opisthorchis* spp. |
| Gastrointestinal tract (Intestinal fluke)<br>Small intestine<br><br><br><br><br>Large intestine | <br>*Fasciolopsis buski*<br>*Heterophyes heterophyes*<br>*Metagonimus yokogawai*<br>*Watsonius watsoni*<br>*Gastrodiscoides hominis* |
| Respiratory tract (Lung fluke) | *Paragonimus westermani* |

**Flukes: General Characteristics**

They vary in size from the species just visible to the naked eye, like *Heterophyes* to the large fleshy flukes, like *Fasciola* and *Fasciolopsis*.

- A conspicuous feature of flukes is the presence of 2 muscular cup-shaped suckers (hence called **distomata**)—**the oral sucker** surrounding the mouth at the

anterior end and the **ventral sucker** or **acetabulum** in the middle, ventrally




- The body is covered by an integument which often bears spines, papillae, or tubercles.
- They have no body cavity, circulatory and respiratory organs.
- The alimentary system consists of the mouth surrounded by the oral sucker, a muscular pharynx and the esophagus, which bifurcates anterior to the acetabulum to form 2 blind caeca, that reunite in some species. The alimentary canal, therefore appears like an **inverted Y**. The anus is absent (Fig. 13.1).
- The excretory system consists of flame cells and collecting tubules, which lead to a median bladder opening posteriorly (Fig. 13.1).
- There is a rudimentary nervous system consisting of paired ganglion cells.
- The reproductive system is well-developed. Flukes are **hermaphroditic** (**monoecious**) except for schistosomes, in which the sexes are separate (**dioecious**).
- The hermaphroditic flukes have both male and female structures, so that **self-fertilization** takes place, though in many species **cross-fertilization** also occurs.

In the schistosomes, the sexes are separate, but the male and female live in close apposition (***in copula***), the female fitting snugly into the folded ventral surface of the male, which forms the **gynecophoric canal**.

- Trematodes are **oviparous** and lay eggs, which are **operculated**, except in the case of schistosomes.

### Life Cycle

Medically important members of the class Trematoda belong to the subclass Digenea, as they are digenetic, i.e. require 2 hosts to complete their life cycle.

## BLOOD FLUKES

### Schistosomes

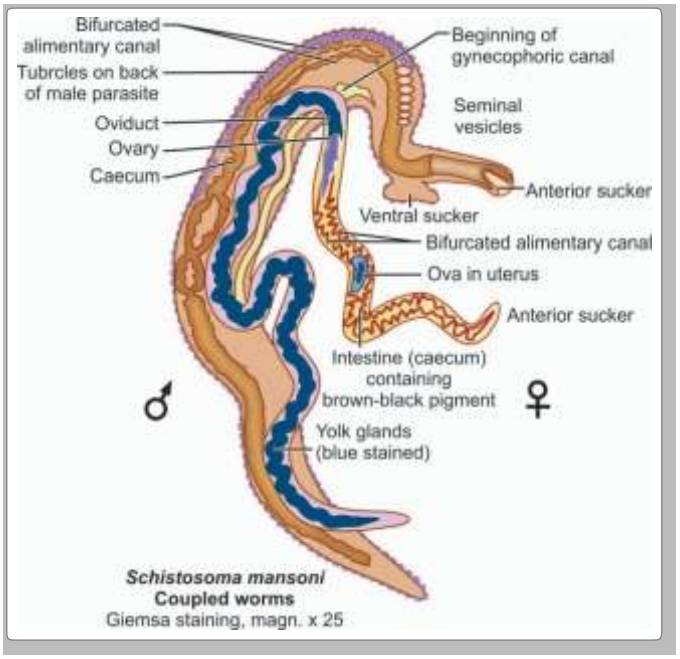
Schistosomes are **dioecious**, (sexes are separate) trematodes, which lead to **Schistosomiasis** (bilharziasis).

- Schistosomiasis is a water-borne disease constituting an important public health problem and affecting millions of persons in Africa, Asia, and Latin America.
- It is estimated that over 100 milion people are infected with *S. haematobium*, *S. mansoni*, and *S. japonicum* each. Two other species of *Schistosoma* parasitizing humans are *S. mekongi* and *S. intercalatum.*
- The male worm is broader than the female and its lateral borders are rolled ventrally into a cylindrical shape, producing a long groove or trough called the **gynecophoric canal**, in which the female is held. It appears as though the body of the male is split longitudinally to produce this canal; hence the name schistosome (Greek *schisto*: *split* and *soma*: *body*)
- Schistosomes were formerly called *Bilharzia* after **Theodor Bilharz** who in 1851, first observed the worm in the mesenteric veins of an Egyptian in Cairo.

## Features distinguishing schistosomes from othertrematodes

Schistosomes differ from the hermaphroditic trematodesin many aspects.

- They are unisexual (diecious).
- They lack a muscular pharynx.
- Their intestinal caeca reunite after bifurcation to forma single canal.
- They produce non-operculated eggs.
- They have no redia stage in larval development.
- The cercariae have forked tails and infect by penetratingthe unbroken skin of definitive hosts.



# Schistosoma Haematobium

Habitat

.The adult worms live in the vesical and pelvic plexuses of veins

## Morphology

*Adult worm*

- The male is 10–15 mm long by 1 mm thick and coveredby a finely tuberculated cuticle.
- It has 2 muscular suckers, the oral sucker being small and the ventral sucker large and prominent. Beginning immediately behind the ventral sucker and extending to the caudal end is the gynecophoric canal, in which the female worm is held (Fig. 13.3).
- The adult female is long and slender, 20 mm by 0.25 mm with the cuticular tubercles confined to the two ends.
- The gravid worm contains 20–30 eggs in its uterus atone time and may pass up to 300 eggs a day.

*Egg*

The eggs are ovoid, about 150 µm by 50 µm, nonoperculated, with a brownish yellow transparent shell carrying a **terminal spine** at one pole; the terminal spine being

characteristic of the species 

Egg of *Schistosoma haematobium*

## Life Cycle

*S. heamatobium* passes its life cycle in 2 hosts.

**Definitive host:** Humans are the only natural definitive hosts. No animal reservoir is known.

**Intermediate host:** Fresh water snails.

**Infective form:** Cercaria larva

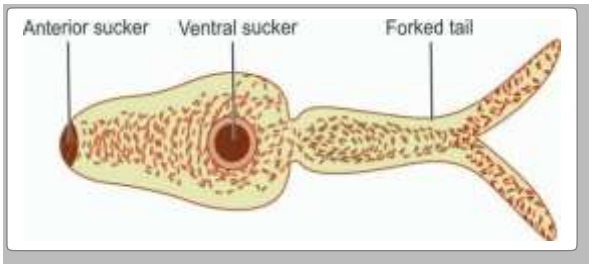
- The eggs that are passed in urine are embryonated and hatch in water under suitable conditions to release the free living **ciliated miracidia**.
- Miracidia swim about in water and on encountering a suitable intermediate host, penetrate into its tissues and reach its liver (Fig. 13.6). The intermediate hosts are snails of *Bulinus* species in Africa. In India, the intermediate host is the limpet, *Ferrisia tenuis*.

### Development in Snail

Inside the snail, the miracidia lose their cilia and in about 4–8 weeks, successively pass through the stages of the **first and second generation sporocysts** (Fig. 13.6).

- Large number of **cercariae** are produced by asexual reproduction within the

second generation sporocyst.




The cercaria has an elongated ovoid body and **forked tail** (*furcocercous cercaria*) (

- The cercariae escape from the snail.
- Swarms of cercariae swim about in water for 1–3 days. If during that period they come into contact with persons bathing or wading in the water, they penetrate through their unbroken skin. Skin penetration is facilitated by lytic substances secreted by penetration glands present in the cercaria.

**Development in Man**

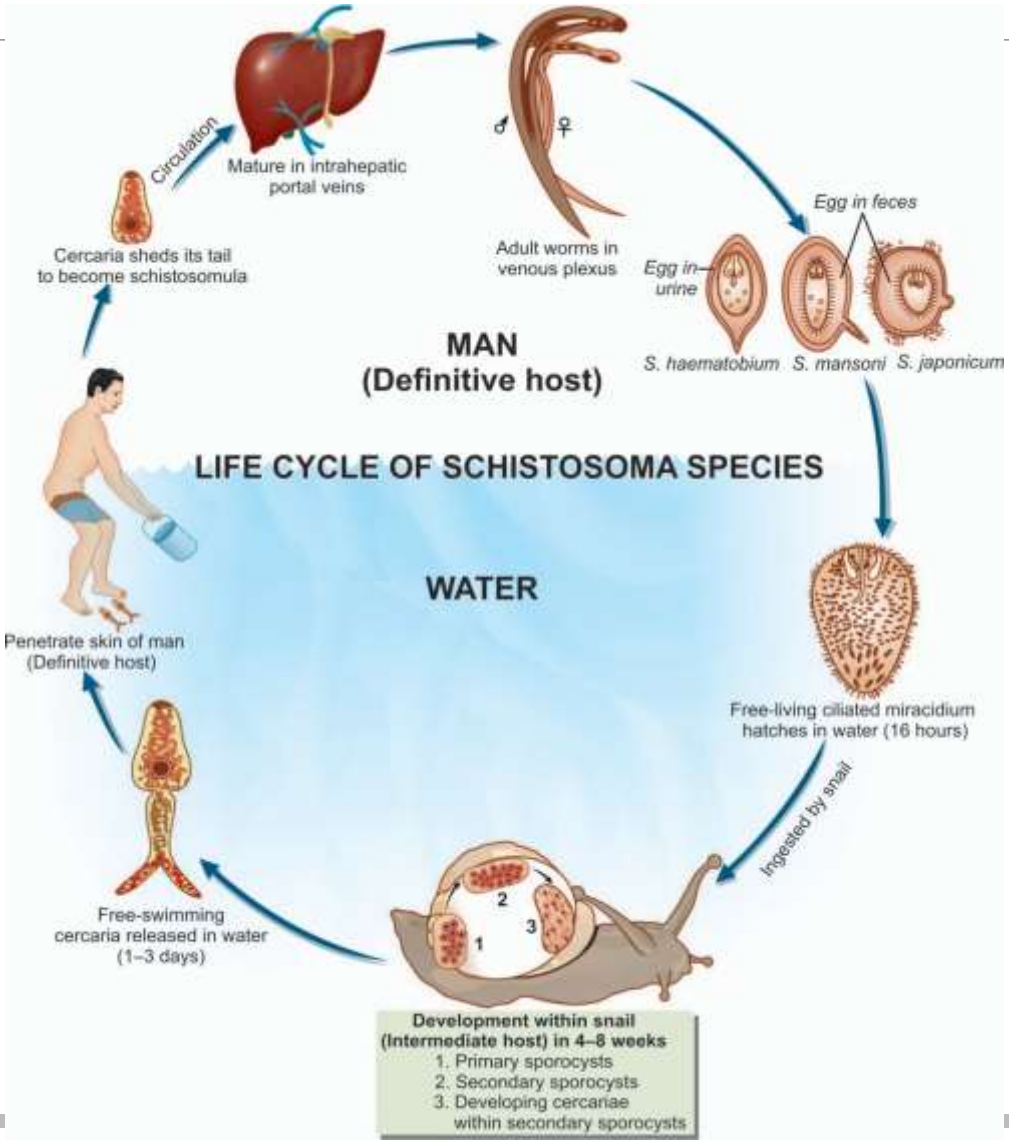
On entering the skin, the cercariae shed their tails and become **schistosomulae** which enter the peripheral venules (Fig. 13.6).

- They then start a long migration, through the vena cava into the right side of the heart, the pulmonary circulation, the left side of the heart, and the systemic circulation, ultimately reaching the liver.
- In the intrahepatic portal veins, the schistosomulae grow and become sexually differentiated adolescents about 20 days after skin penetration.
- They then start migrating against the blood stream into the inferior mesenteric veins, ultimately reaching the **vesical** and **pelvic venous plexuses,** where they mature, mate, and begin laying eggs.
- Eggs start appearing in urine usually 10–12 weeks after cercarial penetration.
- The adult worms may live for 20–30 years.

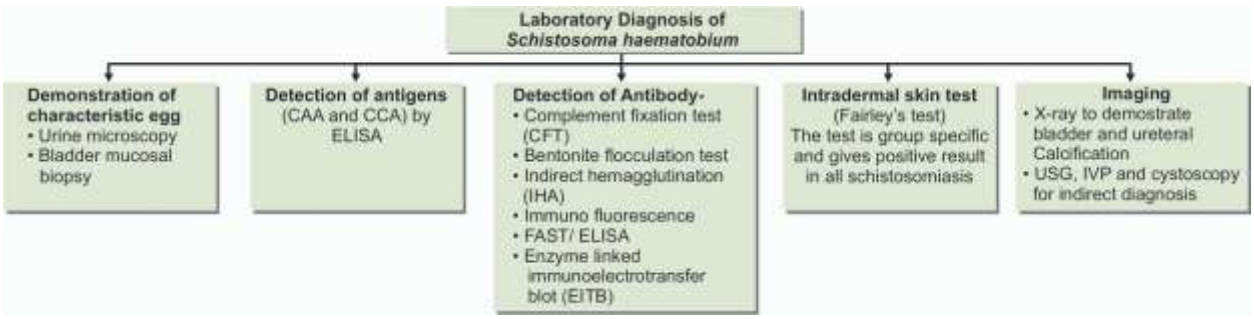
## Pathogenicity and Clinical Features

Clinical illness caused by schistosomes can be classified depending on the stages in the evolution of the infection, as follows:

- Skin penetration and incubation period
- Egg deposition and extrusion
- Tissue proliferation and repair.
- **The clinical features during the incubation period** may be **local cercarial dermatitis** or general **anaphylactic** or **toxic** symptoms.
    - Cercarial dermatitis consists of transient itching and petechial lesions at the site of entry of the cercariae (**swimmer's itch**).
    - This is seen more often in visitors to endemic areas than in locals who may be immune due to repeated contacts.
    - It is particularly severe when infection occurs with cercariae of nonhuman schistosomes.
    - Anaphylactic or toxic symptoms include fever, headache, malaise, and urticaria.
    - This is accompanied by leucocytosis, eosinophilia, enlarged tender liver, and a palpable spleen. This condition is more common in infection with *S. japonicum* (***Katayama fever***).
    -




## Laboratory diagnosis of *Schistosoma haematobium*



## Treatment

Praziquantel is the drug of choice (40mg/kg for 1 day). Metriphonate is the alternative drug of choice in schisto-somiasis due to *S. haematobium*. (7.5 mg/kg. weekly for 3weeks).

| | *Schistosoma haematobium* | *Schistosoma mansoni* | *Schistosoma japonicum* |
|---|---|---|---|
| **Habitat** | Veins of the vesical and pelvic plexuses, less commonly in portal vein and its mesenteric branches | Inferior mesenteric vein and its branches | Superior mesenteric vein and its branches |
| **Morphology** Size:<br>*Male*<br>*Female* | 1.5 cm × 1 mm 2 cm × 0.22 mm | 1 cm × 1 mm<br>1.4 cm × 0.25 mm | 1.2–2 cm × 0.5 mm<br>2.6 cm × 0.3 mm |
| Integument | Finely tuberculated | Grossly tuberculated | Non-tubercular |
| Number of testes | 4–5 in groups | 8–9 in a zigzag row | 6–7 in a single file |
| Ovary | In the posterior one-third of the body | In the anterior half of the body | In the middle of the body |
| Uterus | Contains 20-30 eggs | 1–3 eggs | 50 or more eggs |
| Egg | Elongated with terminal spine | Elongated with lateral spine | Round with small lateral knob |
| Cephalic glands in Cercariae | 2 pairs oxyphilic and 3 pairs basophilic | 2 pairs oxyphilic and 4 pairs basophilic | 5 pairs oxyphilic, no basophilic |
| Distribution | Africa, Near East, Middle | Africa and south America | China, Japan, far east |

| | | | |
|---|---|---|---|
| | East, India | | (oriental) |
| **Definitive host** | Man | Man | Man (mainly) domestic animals & rodents (which act as reservoir of infection |
| **Intermediate host** | Snail of Genus *Bulinus* | Snail of Genus *Biomphalaria* | Amphibian snail of Genus *Oncomelania* |

**Lec 21 Fasciola Hepatica**

***Common name:*** Sheep liver fluke

## History and Distribution

*F. hepatica* was the first trematode that was discovered more than 600 years ago in 1379 by Jehan de Brie.

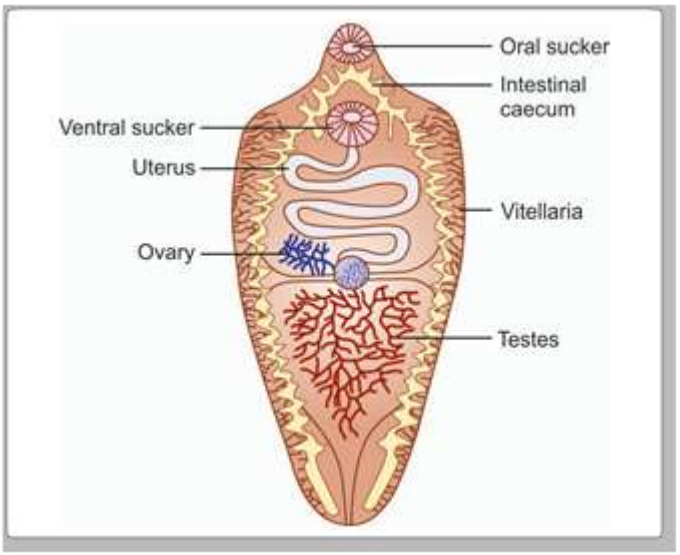
- It was named by Linneus in 1758.
- It is the largest and most common liver fluke found in humans, but its primary host is the sheep and to a less extent, cattle.
- It is worldwide in distribution, being found mainly in sheep-rearing areas.
- It causes the economically-important disease, **'liver rot',** in sheep.

**Habitat /** The parasite resides in the liver and biliary passages of the definitive host.

## Morphology

*Adult Worm*

- It is a large leaf-shaped fleshy fluke, 30 mm long and 15 mm broad, grey or brown in color.




- It has a conical projection anteriorly containing an oral sucker and is rounded posteriorly (Fig. 13.10).
- The adult worm lives in the biliary tract of the definitive host for many years—about 5 years in sheep and 10 years in humans.
- Like all other trematodes, it is hermaphrodite.

*Egg*

The eggs are large, ovoid, operculated, bile-stained, and about 140 µm by 80 µm in size.

- Eggs contain an immature larva, the miracidium
- Eggs do not float in saturated solution of common salt
- Eggs of *F. hepatica* and *Fasciolopsis buski* cannot be differentiated
- Eggs are unembryonated when freshly passed.

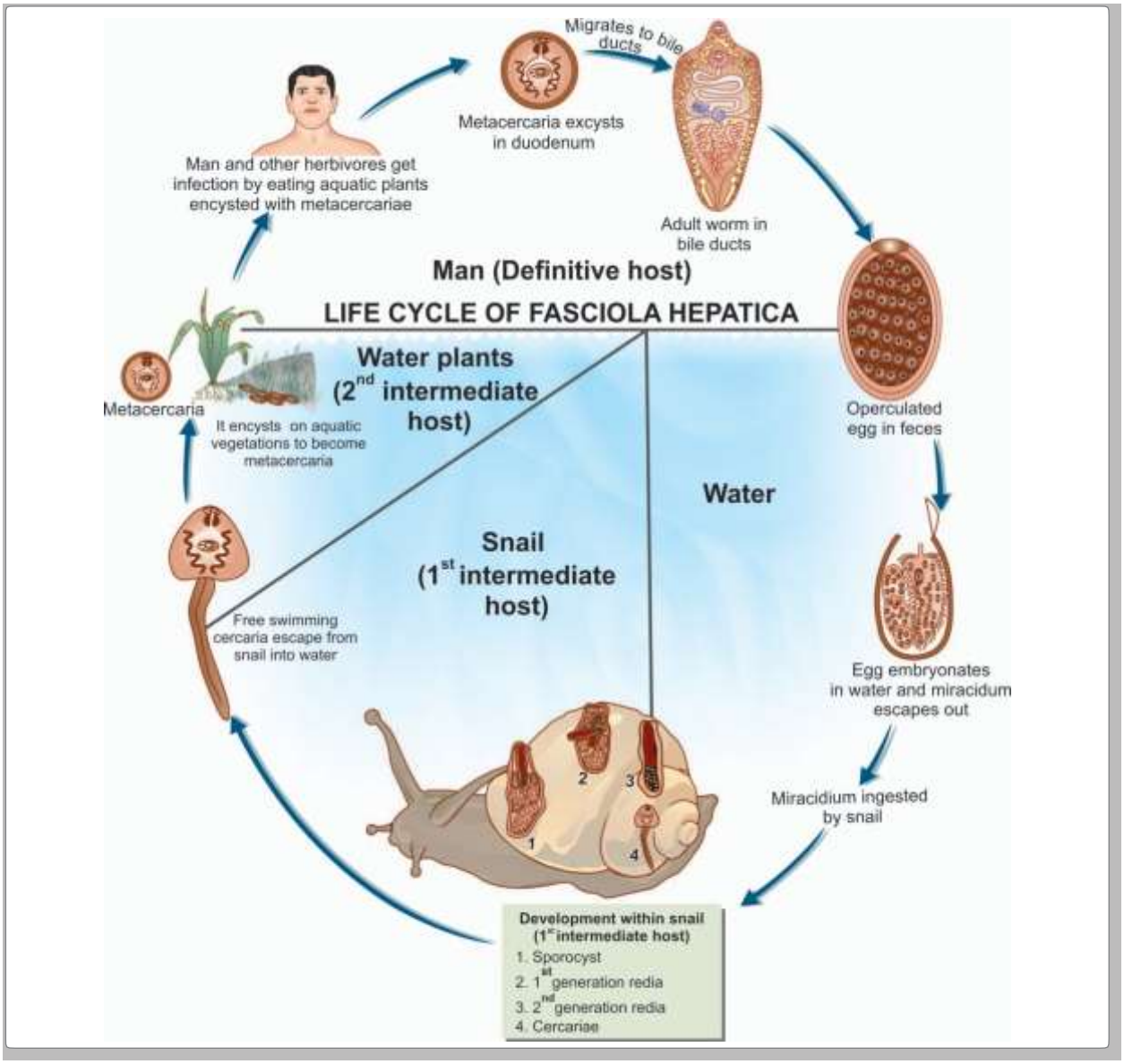
## Life Cycle

*F. hepatica* passes its life cycle in 1 definitive host and 2intermediate hosts.

**Definitive host:** Sheep, goat, cattle, and man. **Intermediate host:** Snails of the genus *Lymnaea* and*Succinea*. Encystment occurs on aquatic plants, which act as **second intermediate host**.

**Mode of infection:** The definitive host, sheep and man, get infection by ingestion of **metacerceriae** encysted onaquatic vegetation.

- Adult worm lives in the **biliary passage** of sheep or man. Eggs are laid in the biliary passages and are shedin feces.
- The embryo matures in water in about 10 days and the **miracidium** escapes. It penetrates the tissues of first intermediate host, snails of the genus *Lymnaea* (Fig. 13.11).
- In snail, the miracidium progresses through the**sporocyst** and the first and second generation **redia** stages to become the cercariae in about 1–2 months.
- The cercariae escape into the water and encyst on aquatic vegetation or blades of grass to become metacercariae, which can survive for long periods.
- Sheep, cattle, or humans eating watercress or other watervegetation containing the metacercaria become infected.
- The metacercariae excyst in the duodenum of the definitive host and pierce the gut wall to enter the peritoneal cavity.




- They penetrate the Glisson's capsule, traverse the liver parenchyma, and reach the biliary passages, where they mature into the adult worms in about 3–4 months (Fig. 13.11).

## Pathogenicity

- **Fascioliasis** differs from clonorchiasis in that *F. hepatica* is larger and so causes more mechanical damage. In traversing the liver tissue, it causes parenchymal injury. As humans are not its primary host, it causes more severe inflammatory response. Some larvae penetrate right through the liver and diaphragm ending

up in the lung.

- In acute phase during the migration of the larva, patients present with fever, right upper quadrant pain, eosinophilia, and tender hepatomegaly. The symptoms subside as parasites reach their final destination.

## Diagnosis

*Stool Microscopy*

Demonstration of eggs in feces or aspirated bile from duodenum is the best method of diagnosis. Eggs of *F. hepatica* and *F. buski* are indistiguishable.

*Blood Picture*

It reveals eosinophilia.

*Serodiagnosis*

Serological tests such as immunofluorescence, ELISA, immunoelectrophoresis, and complement fixation are helpful in lightly-infected individuals for detection of specific antibody. ELISA becomes positive within 2 weeks of infection and is negative after treatment. In chronic fascioliasis, *Fasciola* copro-antigen may be detected in stool.

*Imaging*

USG, CT scan, Endoscopic Retrograde Choangiopancreato- graphy (ERCP) and percutaneous cholangiography may behelpful in diagnosis.

## Treatment

Oral triclabendazole (10 mg/kg once) is the treatment ofchoice.

- Alternative drug is bithionol (30–50 mg for 10–15 days)
- Prednisolone at a dose of 10–20 mg/kg is used to controltoxemia.

## Lec 22 Nematodes:General Features

Nematodes are said to be the most worm-like of all helminths. This is because they generally resemble the common earth worm in appearance, which is considered to be the prototype of 'worms'. However, taxonomically earthworms are not nematodes as they are segmentedworms of the Phylum Annelida.

- Nematodes are elongated, cylindrical, unsegmented worms with tapering ends. The name '*nematode*' means '*thread-like*', from '*nema*' meaning '*thread*'.
- Unlike trematodes and cestodes, all of which are parasitic, most nematodes are free-living forms foundin soil and water.
- Several species are parasites of plants and are of greateconomic importance. Many nematodes parasitize invertebrate and vertebrate animals.

The largest number of helminthic parasites of humansbelong to the class of nematodes. There are an estimated500,000 species of nematodes

**General Characteristics**

They are cylindrical, or filariform in shape, bilaterally symmetrical with a secondary **triradiate symmetry at the anterior end**.

- The adults vary greatly in size, from about a millimeter (*Strongyloides stercoralis*) to a meter (*Dracunculis medinensis*) in length. Male is generally smaller than female and its posterior end is curved or coiled ventrally.

- Their body is covered with a **tough outer cuticle**, whichmay be smooth, striated, bossed, or spiny. The **middle** layer is hypodermis and the **inner layer** is the somaticmuscular layer. They move by **sinuous flexion** of thebody.
- The body cavity is a **pseudocele**, in which all the visceraare suspended.

- The digestive system is complete, consisting of a anteriorly placed mouth leading to the esophagus, which characteristically varies in shape and structure in different groups. The intestine is lined with a single layer of columnar cells and leads to the rectum, opening through the anus. In the male, the rectum and the ejaculatory duct open into the **cloaca**.
- Nematodes have simple excretory and nervous systems.

**Types of female nematodes**

The female nematodes may be divided as follows:

- **Oviparous** (laying eggs):
  - Unsegmented eggs: *Ascaris, Trichuris*
  - Segmented eggs: *Ancylostoma, Necator*
  - Eggs containing larvae: *Enterobius*
- **Viviparous** (producing larvae): *Trichinella, Wuchereria, Brugia, Dracunculus*.
- **Ovoviviparous** (laying eggs containing fully formed larvae, which hatch out immediately): *Strongyloides*.

- The nematodes are **diecious** i.e. the sexes are separate.
- The male reproductive system consists of a single delicate tubule differentiated into testis, vas deferens, seminal vesicle, and ejaculatory duct, which opens into the cloaca. It also includes copulatory structures such as spicules or bursa or both.
- The female reproductive system consists of the ovary, oviduct, seminal

receptacle, uterus, and vagina.

- Female nematodes may produce eggs (**oviparous**) or larvae (**viviparous**). Some lay eggs containing larvae,which immediately hatch out (**ovoviviparous**).

## Classification of Nematodes on the Basis of the Habitat of Adult Worms

| Intestinal Human Nematodes | Somatic Human Nematodes |
|---|---|
| **Small Intestine** | **Lymphatics** |
| · *Ascaris lumbricoides* (Common round worm) | · *Wuchereria bancrofti* |
| · *Ancylostoma duodenale* (Old world Hook worm) | · *Brugia malayi* |
| · *Necator americanus* (American or New World Hook worm) | · *Brugia timori* |
| · *Strongyloides stercoralis* | **Skin/subcutaneous tissue** |
| · *Trichinella spiralis* | · *Loa loa* |
| · *Capillaria philippinensis* | · *Onchocerca volvulus* |
| **Large intestine** | · *Dracunculus medinensis* (Guinea worm) |
| · *Trichuris trichiura* (Whip worm) | **Mysentery** |
| · *Enterobius vermicularis* (Thread or pin worm) | · *Mansonella ozzardi* |
| | · *Mansonella perstans* |
| | **Conjunctiva** |
| | · *Loa loa* |

## Lec 23 *Enterobius Vermicularis*

***Common name:*** Pinworm, Seatworm, Threadworm

* *E. vermicularis* is considered to be **world's most common** parasite, which specially affects the children.

**Habitat**

Adult worms are found in the caecum, appendix, and adjacent portion of ascending colon.

**Morphology**

**Adult Worm**

The adults are short, white, fusiform worms with pointed ends, looking like bits of white thread.

* The mouth is surrounded by 3 wing-like cuticular expansions (cervical alae), which are transversely striated.

* The esophagus has a double-bulb structure, a feature unique to this worm.

**Female Worm**

The female is 8–13 mm long and 0.3–0.5 mm thick.

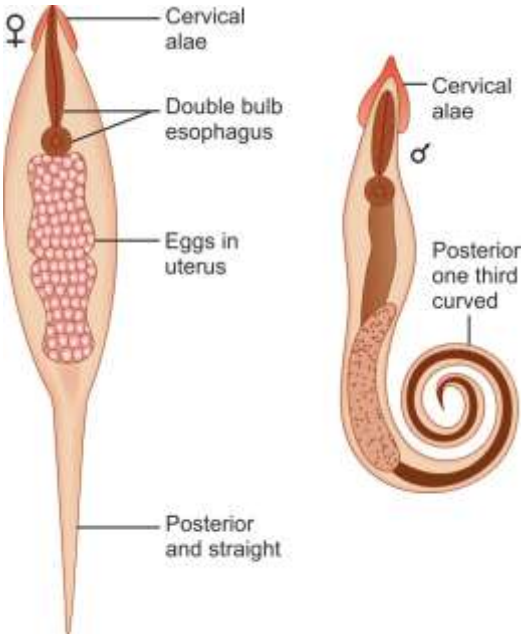
* The worm is **oviparous.**

* Females survive for 5–12 weeks.

**Male Worm**

The male worm is 2–5 mm long and 0.1–0.2 mm thick.

*Males live for about 7–8 weeks.



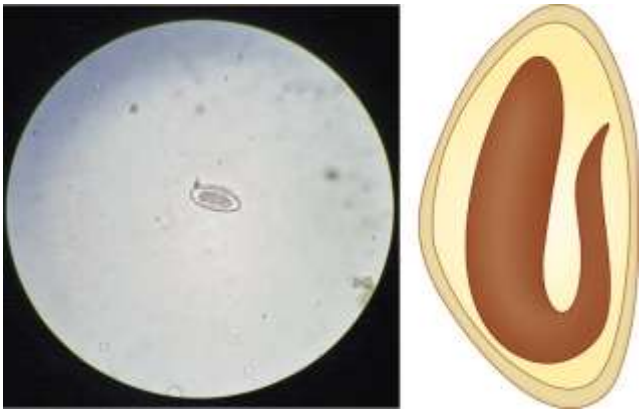

**Adult worm of *Enterobius vermicularis* (male and female)**

## Egg

The egg is colorless and not bile-stained.

* It floats in saturated salt solution.

* It has a characteristic shape, being elongated ovoid, attended on one side, and convex on the other



(**planoconvex**), measuring 50–60 µm by 20–30 µm

## Life Cycle

*E. vermicularis* is monoxenous, passing its entire life cycle in the human host. It has no intermediate host and does not undergo any systemic migration.

**Natural host:** Man

**Infective form:** Embryonated eggs.

* **Mode of infection:** Man acquires infection by ingesting embryonated eggs containing larva by means of

* Contaminated fingers

* Autoinfection

* Eggs laid on perianal skin containing infective larvae are swallowed and hatch out in the intestine.

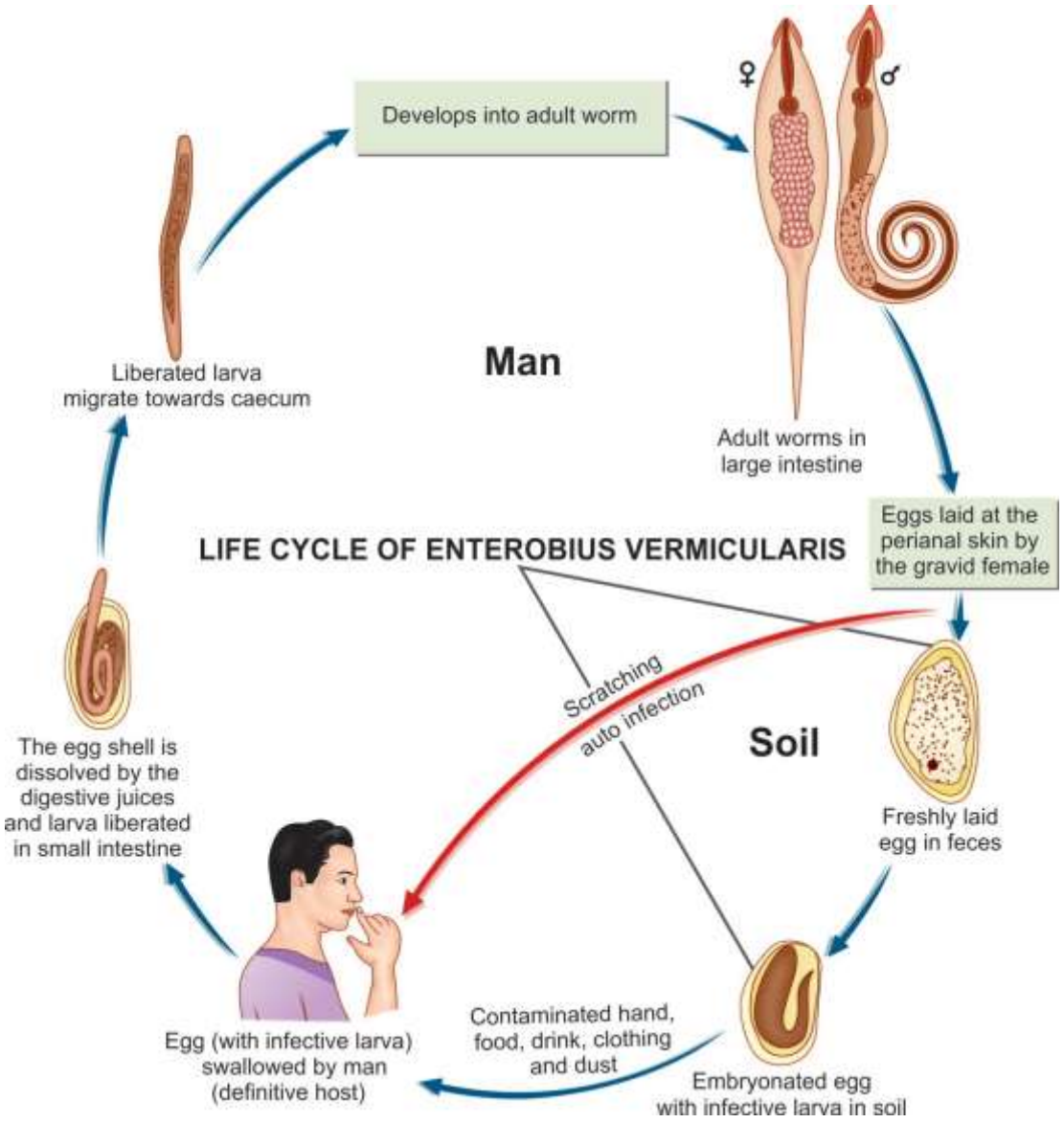
*They moult in the ileum and enter the caecum, where they mature into adults.

* It takes from 2 weeks to 2 months from the time the eggs are ingested, to the development of the gravid female, ready to lay eggs.

* The gravid female migrates down the colon to the rectum. At night, when the host is in bed, the worm comes out through the anus and crawls about on the perianal and perineal skin to lay its sticky eggs. The worm may retreat into the anal canal and come out again to lay more eggs.

*The female worm may wander into the vulva, vagina and even into the uterus and fallopian tubes, sometimes reaching the peritoneum.

* The male is seldom seen as it does not migrate. It usually dies after mating and is passed in the feces.




*__Autoinfection:__ Ingestion of eggs due to scratching of perianal area with fingers leading to deposition of eggs under the nails. This type of infection is mostly common in children. This mode of infection occurs from anus to mouth.

* __Retroinfection:__ In this process, the eggs laid on the perianl skin immediately hatch into the infective stage larva and migrate through the anus to develop into worms in the colon. This mode of infection occurs from anus to colon.

## Pathogenicity and Clinical Features

Enterobiasis occurs mostly in children. It is more common in females than in males. About one-third of infections are asymptomatic.

*The worm produces intense irritation and pruritus of the perianal and perineal area (**pruritis ani**), when it crawls out of the anus to lay eggs. This leads to scratching and excoriation of the skin around the anus.

* As the worm migrates out at night, it disturbs sleep. **Nocturnal enuresis** is sometimes seen.

* The worm crawling into the vulva and vagina causes irritation and a mucoid discharge. It may migrate upto the uterus, fallopian tubes and into the peritoneum. This may cause symptoms of **chronic salpingitis**, cervicitis, peritiontis, and recurrent urinary tract infections.

* The worm is sometimes found in surgically removed appendix and has been claimed to be responsible for **appendicitis**.

## Laboratory Diagnosis

Pinworm infestation can be suspected from the history of perianal pruritus. Diagnosis depends on the demonstration of the eggs or adult worms.

## Demonstration of Eggs

* Eggs are present in the feces only in a small proportion f patients and so feces examination is not useful in diagnosis.

* They are deposited in large numbers on the perianal and perineal skin at night and can be demonstrated in swabs collected from the sites early morning, before going to the toilet or bathing. Swabs from perianal folds are most often positive.

* The eggs may sometimes be demonstrated in the dirt collected from beneath the finger nails in infected children.

## NIH Swab Method

The **NIH swab** (named after National Institutes of Health, USA) has been widely used for collection of specimens. This consists of a glass rod at one end of which a piece of transparent cellophane is attached with a rubber band. The glass rod is fixed on a rubber stopper and kept in a wide test tube. The cellophane part is used for swabbing by rolling over the perianal area (Fig. 19.4). It is returned to the test tube and sent to the laboratory, where the cellophane piece is detached, spread over a glass side and examined microscopically.

**Scotch Tape Method**

Another method for collection of specimens is with scotch tape (adhesive transparent cellophane tape) held sticky side out, on a wooden tongue depressor. The mounted tape is firmly pressed against the anal margin, covering all sides.

The tape is transferred to a glass slide, sticky side down, with a drop of toluene for clearing and examined under the microscope.

**Demonstration of Adult Worm**

The adult worms may sometimes be noticed on the surface of stools.

* They may occasionally be found crawling out of the anus while the children are asleep.

*They may be detected in stools collected after an enema and may be in the appendix during appendicetomy.

**Treatment**

Pyrantel pamoate (11 mg/kg once, maximum 1 g), Albendazole

(400 mg once) or mebendazole (100 mg once) can be used for single dose therapy, while piperazine has to be given daily for one week.

# *Ascaris Lumbricoides*

***Common name:*** Roundworm

## History and Distribution

*Ascaris lumbricoides* has been observed and described from very ancient times, when it was sometimes confused with the earthworm.

*Its specific name *lumbricoides* is derived from its resemblance with earthworm (*Lumbricus*, meaning *earthworm* in Latin).

*It is the most common of human helminths and is distributed worldwide. A billion people are estimated to be infected with roundworms. The individual worm burden could be very high, even up to over a thousand.

An editorial in the *Lancet* in 1989 observed that if all the roundworms in all the people worldwide were placed end-to-end they would encircle the world 50 times.

*The incidence may be as high as 80–100% in rural areas with poor sanitation.

## Habitat

Adult worms live in the small intestines (85% in jejunum and 15% in ileum).

## Morphology

## Adult Worm

They are large **cylindrical** worms, with **tapering** ends, the anterior end being more pointed than the posterior.

*They are pale pink or flesh colored when freshly passedin stools, but become white outside the body.

*The mouth at the anterior end has **3** fi nely toothed lips, 1 **dorsal** and **2 ventrolateral.**

## Male Worm

*The adult male worm is little **smaller** than female. It measures 15–30 cm in length and 2–4 mm in thickness.

*Its posterior end is curved ventrally to form a **hook** and carries 2 **copulatory spicules.**

## Female Worm

The female is **larger** than male, measuring 20–40 cm in length and 3–6 mm in thickness.

*Its posterior extremity is **straight** and **conical**.

A single worm lays up to 200,000 eggs per day. The eggs are passed in feces.

## Egg

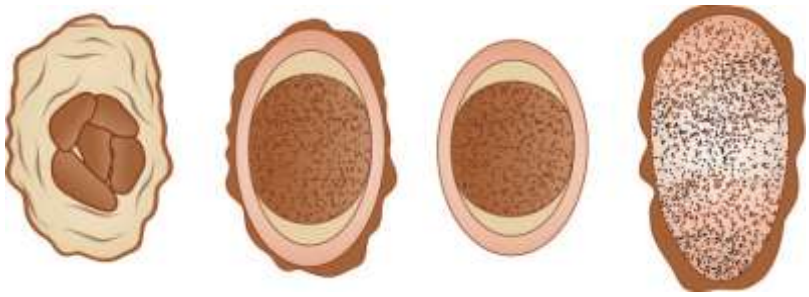
Two types of eggs are passed by the worms; fertilized and unfertilized.

*The fertilized eggs, laid by females, inseminated by mating with a male, are embryonated and develop into the infective eggs.

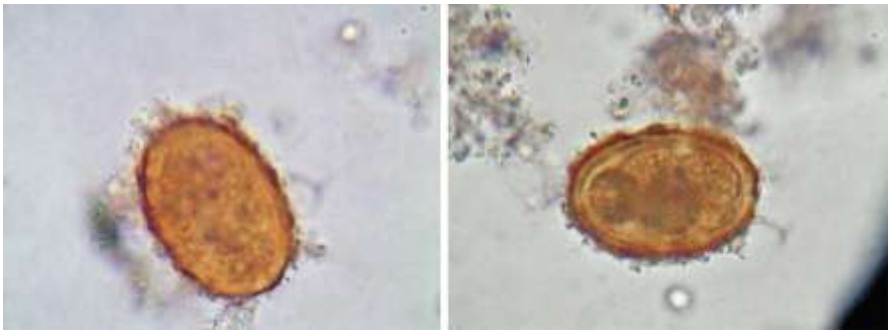
*The unfertilized eggs, are laid by un inseminated female. These are non-embryonated and cannot become infective.

**Features of Round Worm Egg**

| Type of egg | Main feature |
|---|---|
| Unfertilized egg (Fig. 20.4A) | • Ellipitical in shape<br>• Narrower and longer<br>• 80 µm × 55 µm in size<br>• Has a thinner shell with an irregular coating of albumin<br>• Contains a small atrophied ovum with a mass of disorganized highly refractile granules of various size<br>• Does not float in salt solution |
| Fertilized eggs (Fig. 20.4B) | • Round or oval in shape<br>• Size 60–75 µm × 40–45 µm<br>• Always bile-stained<br>• Golden brown in color<br>• Surrounded by thick smooth translucent shell with an outer coarsely mammiliated albuminous coat, a thick transparent middle layer and the inner lipoidal vitelline membrane<br>• Some eggs are found in feces without the outer mamillated coat. They are called the decorticated eggs (Fig. 20.3C)<br>• In the middle of the egg is a large unsegmented ovum, containing a mass of coarse lecithin granules. It nearly fills the egg, except for a clear crescentic area at either poles<br>• Floats in saturated solution of common salt |



**Types of *Ascaris* eggs found in stools. A. Fertilized egg surface focus, showing outer mamillary coat; B. Fertilized egg, median focus, showing unsegmented ovum surrounded by 3 layers of coats; C. Decorticated fertilized egg, the mamillary coat is absent; D. Unfertilized egg, elongated, with atrophic ovum**



**Unfertilized egg of *Ascaris*; B. Fertilized egg of *Ascaris***

**Note:** Stool samples may show both fertilized and unfertilized eggs, or either type alone.

## Life Cycle

Life cycle of *Ascaris* involves only 1 host.

**Natural host:** Man. There is no intermediate host.

**Infective form:** Embryonated eggs

***Mode of transmission:**

€ Infection occurs when the egg containing the **infective rhabditiform** larva is swallowed. A frequent mode of transmission is through fresh vegetables grown in fields manures with human feces (‘night soil’). Infection may also be transmitted through contaminated drinking water.

€ Children playing about in mud can transmit eggs to their mouth through dirty fingers (**geophage**), where soil contamination is heavy due to indiscriminate defecation, the eggs sometimes get airborne along with windswept dust and are inhaled.

The inhaled eggs get swallowed.

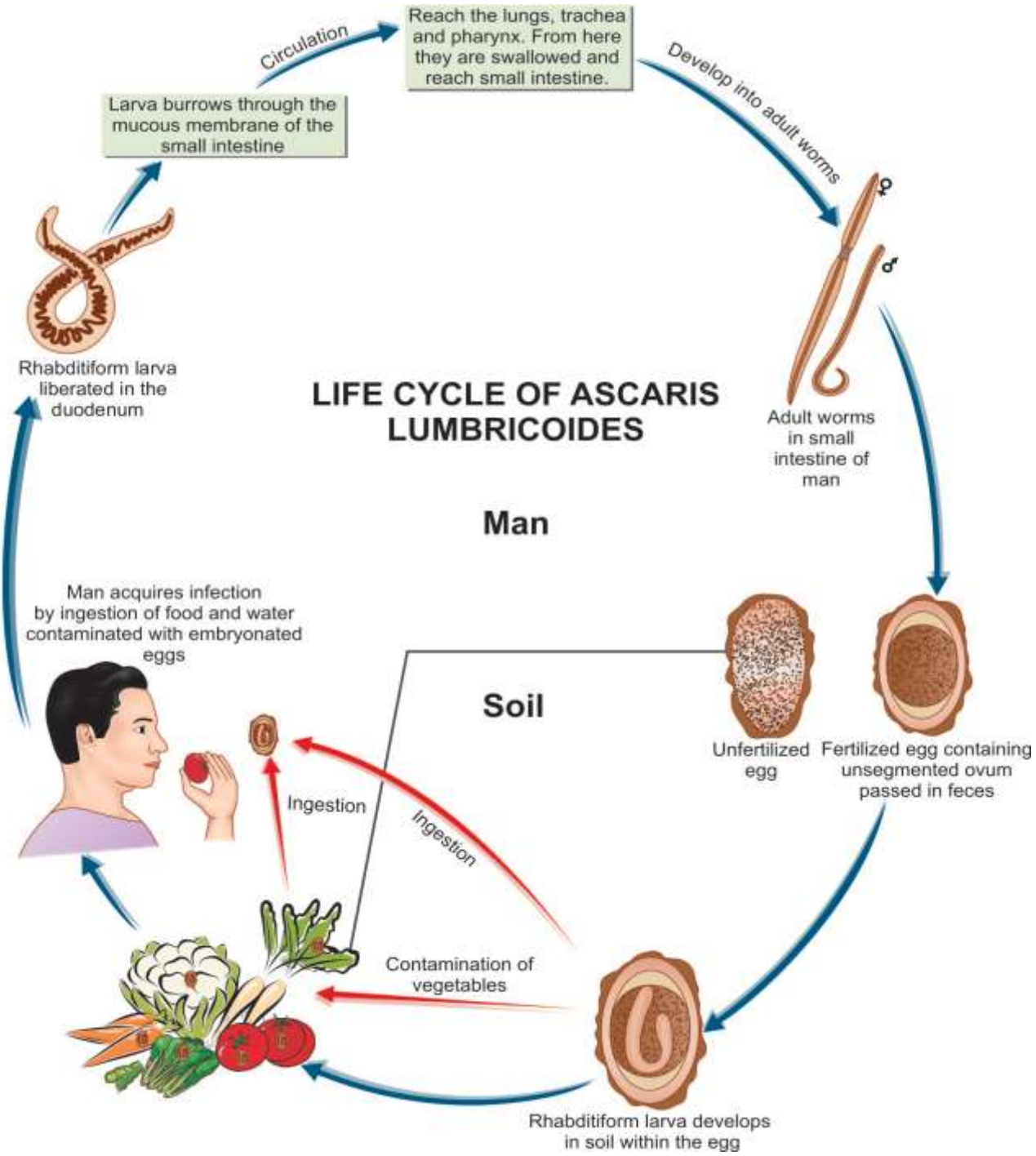
*They penetrate the intestinal mucosa, enter the **portal vessels** and are carried to the **liver**.

They then pass via the hepatic vein, inferior vena cava, and the right side of the heart and in about **4 days** reaches the **lungs**, where they grow and **moult twice**.

*After development in the lungs, in about **10–15 days**, the larvae pierce the lung capillaries and reach the alveoli. They crawl up or are carried up the respiratory passage to the throat and are swallowed.

*The larvae **moult finally** and develop into adults in the upper part of the **small intestine**. They become sexually mature in about **6–12 weeks** and the gravid females start laying eggs to repeat the cycle.

*The adult worm has a lifespan of 12–20 months.




## Pathogenicity and Clinical Features

Disease caused by *A. lumbricoides* is called as *ascariasis*.

*Clinical manifestations in ascariasis can be caused either by the migrating larvae or by the adult worms.

**Symptoms due to the Migrating Larvae**

The pathogenic effects of larval migration are due to allergic eaction and not the presence of larvae as such. Therefore, the initial exposure to larvae is usually asymptomatic, except when the larval load is very heavy.

*When reinfection occurs subsequently, there may be intense cellular reaction to the migrating larvae in the lungs, with infiltration of eosinophils, macrophages, and epithelioid cells.

*This **ascaris pneumonia** is characterized by low grade fever, dry cough, asthmatic wheezing, urticaria, eosinophilia, and mottled lung infiltration in the chest radiograph.

*The sputum is often **blood-tinged** and may contain **Charcot-Leyden** crystals.

***vitamin A deficiency**.

€ ***The mechanical effects** are the most important manifestations of ascariasis. Mechanical effects can be due to masses of worms causing luminal occlusion or even a single worm infiltrating into a vital area.

***Ectopic ascariasis (Wanderlust):**

***biliary obstruction** or **pancreatitis**. It may enter the liver parenchyma, where it may lead to liver abscesses.

**obstructive appendicitis**.

**Laboratory Diagnosis**

**Detection of Parasite**

**Adult Worm**

The adult worm can occasionally be detected in stool or sputum of patient by naked eye.

*Barium meal may reveal the presence of adult worm in the small intestine.

*A plain abdominal film may reveal masses of worms in gas-filled loops of bowel in patients with intestinal obstruction.

*Pancreaticobiliary worms can be detected by ultrasound (more than 50% sensitive) and endoscopic retrograde cholangiopancreatography (ERCP; 90% sensitive).

**Larvae**

In the early stages of infection, when migrating larvae cause Loeffler's syndrome, the diagnosis may be made by demonstrating the larvae in **sputum**, or more often in **gastric washings**.

*Presence of Charcot-Leyden crystals in sputum and an attendant eosinophilia supports the diagnosis. At this stage, no eggs are seen in feces.

*Chest X-ray may show patchy pulmonary infiltrates. by demonstrating the larvae in **sputum**, or more often in **gastric washings**.

*Presence of Charcot-Leyden crystals in sputum and an attendant eosinophilia supports the diagnosis. At this stage, no eggs are seen in feces.

*Chest X-ray may show patchy pulmonary infiltrates.

**Eggs**

Definitive diagnosis of ascariasis is made by demonstration of eggs in **feces**.

*Eggs may not be seen if only male worms are present, as may occasionally be the case. Fecal films often contain many artifacts resembling *Ascaris* eggs and care must be taken to differentiate them.

*Eggs may be demonstrative in **the bile** obtained by **duodenal aspirates**

**Treatment**

Several safe and effective drugs are now available for treatment of ascariasis. These include pyrantel pamoate (11 mg/kg once; maximum 1 g), albendazole

**Lec 24** **Trichuris Trichiura**

## History and Distribution

*Trichuris trichiura*, the human whipworm, was _ rst described by **Linnaeus** in **1771**.

*The name *Trichuris* means a '*hair-like tail*' (*Greek trichos— hair, oura—tail*). This name is not quite correct because it is the anterior end of the worm that is hair-like and not the tail. The name whipworm is more apt as the thick posterior part resembles the stock and the thin anterior end resembles the lash of a whip.

*The antiquity of the whipworm as a human parasite is indicated by the demonstration of its eggs in colonic contents of a young man, who died on the Alps some 5,300 years ago and whose well-preserved body was discovered in **1990**.

*It is worldwide in distribution, but is much more common in the tropics. The infection is widespread in tropical Africa, South America, and South-east Asia.

*Trichuris* infection is found throughout India.

*Some 800 million people are estimated to be infected with this worm.

*While whipworm infection is extremely frequent,

whipworm disease is relatively rare.

## Habitat

*T. trichiura* lives in the large intestine. The adult worms are found attached to the wall of the **caecum** and less commonly to the vermiform appendix, colon, and anal canal.

## Morphology

### Adult Worm

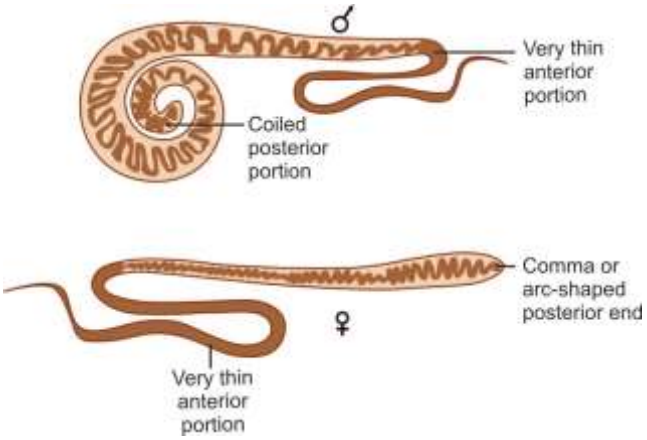
The male worm is 30–45 mm long, while the female is slightly larger, about 40–50 mm.

*The worm is _ flesh-colored. In shape, it resembles a whip, with the anterior three-fifth thin and thread-like and the posterior two- fifth thick and fleshy, appearing like the handle of a **whip.**

*The attenuated anterior portion, which contains the capillary esophagus, is embedded in the mucosa. The posterior part contains the intestines and reproductive organs.

*The posterior end of the male is coiled ventrally, while the hind end of the female is straight, blunt, and rounded.

*The worm has a lifespan of 5–10 years.




## Egg

The egg has a characteristic appearance.

*It is brown in color being **bile-stained**.

*It has a **triple shell**, the outermost layer of which is stained brown.

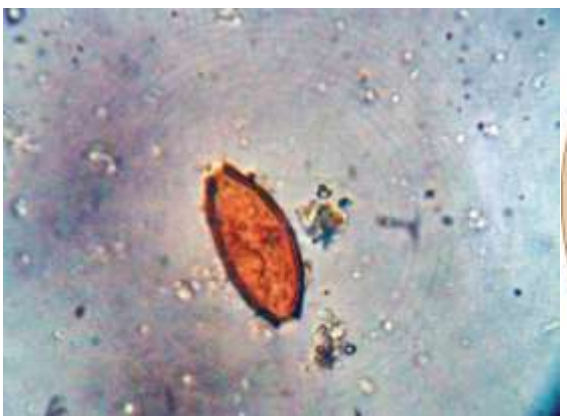
*It is **barrel-shaped** and about 50 µm long and 25 µm wide in the middle, with a projecting **mucus plug** at each pole containing an unsegmented ovum

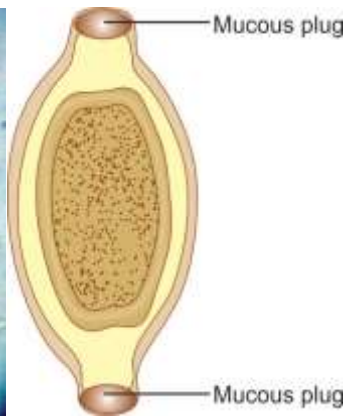
The plugs are colorless.

*The egg _ floats in saturated salt solution.

*When freshly passed, the egg contains an unsegmented ovum. At this stage, it is not infective for humans.

*The fertilized female lays about 5,000 eggs per day.





**Life Cycle**

**Natural host:** Man. No intermediate host is required.

**Infective form:** Embryonated eggs contaning Rhabditiform larva.

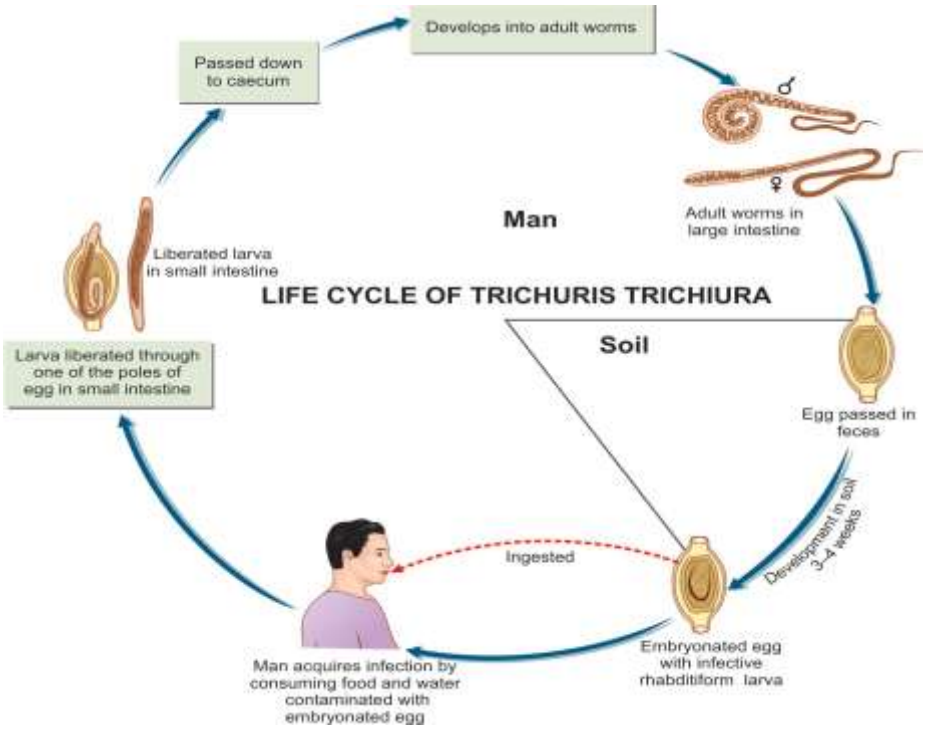
*Adult female worm lives in large intestine worm lays

eggs which are discharged in feces.

*The egg undergoes development in soil, optimally under warm, moist, shady conditions, when the **infective rhabditiform larva** develops within the egg in **3–4 weeks**. At lower temperatures, this may be delayed for 3 months or more. These embryonated eggs are infective to man.

***Mode of Transmission:** Infection occurs in humans when the mature embryonated eggs containing the infective larvae are swallowed in **contaminated food or water**.

*The eggs hatch in the **small intestine** and the larva, which emerges through the pole of the egg, passes down into the caecum.




*In about **2–3 months**, they become mature adults and lie embedded in the cecal wall, with the thread-like anterior portion piercing the mucosa and the thick posterior end projecting out.

*The gravid adult female lays eggs, which are discharged in feces and the cycle is repeated

*Eggs start appearing in feces usually about **3 months** after infection.

**Pathogenecity and Clinical Features**

Infection with *T. trichiura* (**trichuriasis, whipworm infection**, or **trichocephaliasis**) is asymptomatic, except when the worm load is heavy. Disease may result either due to mechanical effects or allergic reaction.

*The worms lie threaded into the cecal mucosa and even though it is not a blood feeder, oozing of blood may occur at the sites of attachment. The blood loss is **about 0.005 mL per worm per day**. Over a period of time, this may lead to anemia and malnutrition.

*It has been suggested that mechanical blockage of the appendiceal lumen by masses of whipworms may cause acute appendicitis. In heavy infection, the worm may be abundant on the colonic mucosa, even upto the rectum. Mucus diarrhea, chronic dysentery and abdominal pain, and weight loss are frequently seen in such cases. Some patients, particularly young children, may develop rectal prolapse.

**Laboratory Diagnosis**

**Stool Examination**

The characteristic barrel-shaped eggs are found in stools.

*The degree of infection can be assessed by egg counts. **Less than 10 eggs** per smear in direct stool preparation is considered **light infection** and **more than 50** per smear as **heavy infection.**

*Light infection is not considered to cause clinical disease.

**Sigmoidoscopy**

Sigmoidoscopy is useful as worms are found in the rectal mucosa in whipworm diarrhea and dysentery. **Charcot- Leyden crystals** are usually abundant in stools of patients **Flowchart 16.1:** Laboratory diagnosis of *Trichuris trichiura* with whipworm dysentery. In heavy infection, sigmoidoscopy may show white bodies of worm hanging from the inflamed mucosa, the so called **coconut cake rectum**.

## Blood Examination

Differential leukocyte count (DLC) may show upto 25% eosinophila in the early stage of the disease.

**Treatment**

Mebendazole (100 mg 12 hourly for 3–5 days) or Albendazole (single dose of 400 mg) are effective with cure rates of 70–90%.

**Lec 25**

## Trichinella Spiralis

***Common name:*** **Trichina worm**

**History and Distribution**

*Trichinella spiralis,* tissue nematode, is the causative agent of **trichinosis**.

*The name *Trichinella* is derived from the minute size of the adult. (Greek *trichos*—hair; *ella* sux for diminutive; *spiralis* refers to the spirally coiled appearance of larvae in muscles).

*It was _ rst observed in **1821** in the muscles of a patient at autopsy by James Paget, who was then a first year medical student at St. Bartholomew's Hospital, London.

*Owen, in **1835**, described the encysted larval form in muscles and named it *Trichina spiralis.*

*Virchow discovered its life cycle in **1859**.

*The major source of human infection was shown to be the **consumption of inadequately cooked pork**.

*Trichinosis is recognized as an important public health problem in Europe and America, but is much less common in the tropics and oriental countries.

*Human trichinosis had not been recorded in India till 1996, when thirst case was reported from Punjab.

**Habitat**

Adult worms live deeply buried in the **mucosa of small intestine (duodenum or jejunum)** of pig, bear, rat, or man. The encysted larvae are present in the **striated muscles** of these hosts. There are no free-living stages.

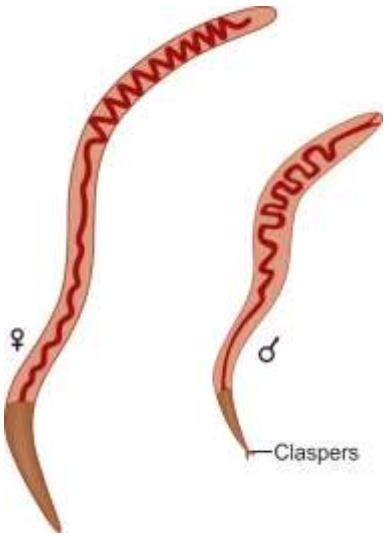
**Morphology**

**Adult Worm**

The adult *T. spiralis*, a small white worm just visible to the naked eye, is one of **the smallest nematodes** infecting humans.

*The **male** measures about 1.5 mm by 0.04 mm and the female about 3 mm by 0.06 mm (twice the length of male).

*The **anterior half** of the body is **thin** and **pointed**, welladapted for **burrowing** into the mucosal epithelium.




The **posterior end** of the male has a pair of pear-shaped **clasping papillae** (termed as claspers), one on each side of the **cloaca** that it uses to hold the female worm during mating.

*The female worm is **viviparous** and discharge larva instead of eggs.

*The life span of the adult worm is very short. The male worm dies soon after fertilizing the female and the female dies after 4 weeks to 4 months (16 weeks), the time required for discharging the larvae.

## Larvae

The larva becomes encysted in the striated muscles and at the time of encystment measures 1 mm in length by 36 μm in diameter.

*The larva in the cyst is **coiled** and hence, the name *spiralis.*

### Life Cycle

*Trichinella* is a parasite that has a direct life cycle, which means it completes all stages of development in one host. But only a **single cycle** occurs in one host and for continuation of the cycle and maintenance of the species, it is necessary for the infection to be transmitted to another

host of the same species or of different species.

***Optimum host:** Pig.

***Alternate host:** Man.

*Infection can pass from—Pig-to-pig (facilitated by the custom of feeding pigs with untreated household garbage, which may contain bits of pork with infective cysts), rat-to-rat, and pig-to-rat.

*Man is the **dead-end** of the parasite, as the cysts in human muscles are unlikely to be eaten by another host.

***Infective form:** Encysted larva found in the muscles of pigs and other animals.

***Mode of infection:** Man acquires infection mainly by eating raw or undercooked pork or inadequately processed sausages or other meat products containing the viable larvae.

*When such meat is eaten without adequate cooking, the cysts are digested by the gastric juice and viable larvae are released (excystation) in the stomach, duodenum, and jejunum.

*The larvae immediately penetrate the mucosal epithelium.

*They molt **4 times** and rapidly develop into adults, either male or female, by the second day of infection. Within 5 days, they become sexually mature.

*The male dies after fertilizing the female. The fertilized females start **releasing motile larvae** by the **sixth day** of infection.

*Larvae continue to be discharged during the remaining part of the lifespan of the female worm, which ranges from **4 weeks to 4 months**.

*Each female gives birth to approximately **1,000 larvae.**

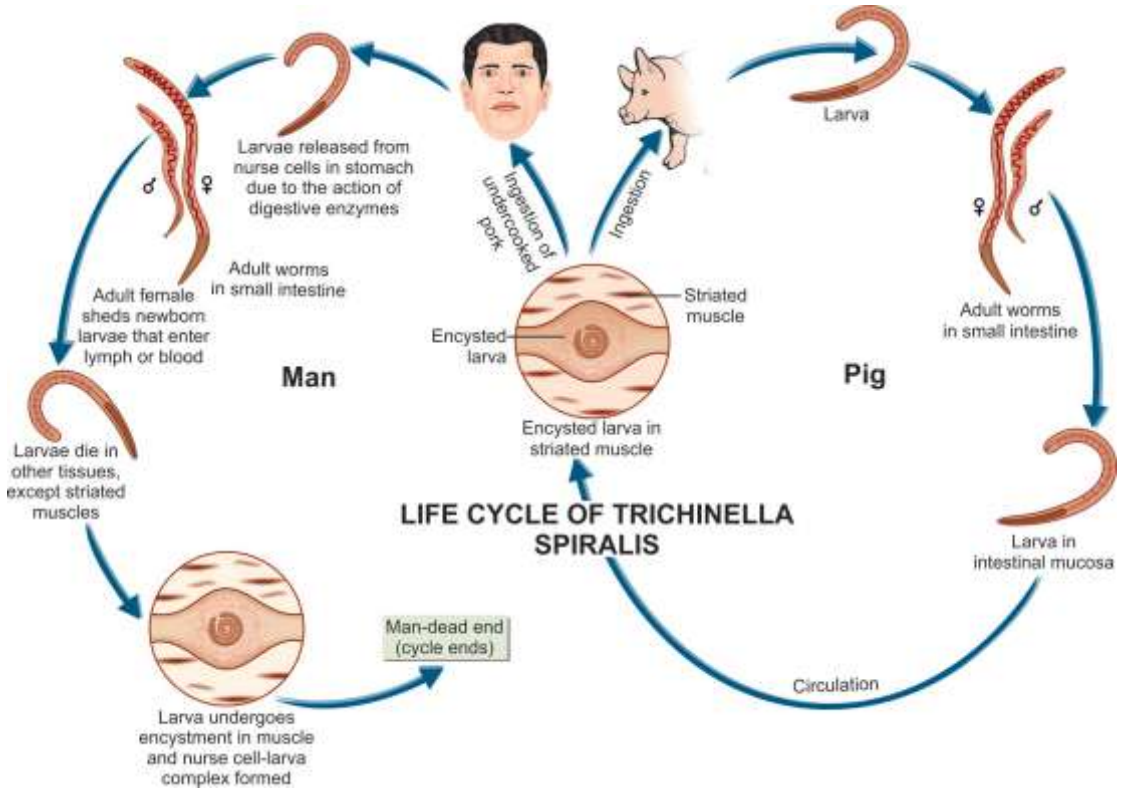
*These larvae **enter the intestinal lymphatics** or **mesenteric venules** and are transported in circulation to different parts of the body. They get **deposited in the muscles**, **central nervous system,** and **other sites.** The larva die in most other situations, except the **skeletal muscles**, where it grows.

***Deposition in the muscles** occurs mostly during the **second week** of infection. Larval development in muscles takes place during the next **3 or 4 weeks**.

*Within 20 days after entering the muscle cells, the larvae become encysted. A muscle cell carrying larva of *T. spiralis* is called as a **nurse cell**.

*Encysted larvae lie parallel to the muscles of host.

*Encysted larva can survive for months to years. In man, the life cycle ends here




**Pathogenicity and Clinical Features**

The disease caused by *T. spiralis* is called **trichinosis**.

*The manifestations vary from asymptomatic infection, which is very common, to an acute fatal illness, which is extremely rare.

*The pathology and clinical features vary according to the stage in the life cycle of the worm.

**Diagnosis**

Diagnosis of trichinosis can be made by direct and indirect methods.

## Direct Methods

i. Detection of spiral larvae in muscle tissue by performing muscle biopsy. Deltoid, biceps, gastrocnemius, or pectoralis muscles are usually selected for biopsy.

ii. Detection of adult worms and larvae in the stool during the diarrheic stage

iii. **Xenodiagnosis:** For xenodiagnosis, biopsy bits are fed to laboratory rats, which are killed a month or so, later. The larvae can be demonstrated more easily in the muscles of such infected rats.

## Indirect Methods

i. History of consumption of raw or inadequately cooked or processed pork, about two weeks earlier along with a recent episode of **gastroenteritis**.

**Treatment:** Albendazole

*Lec 26* *Strongyloides stercoralis*

## Morphology

- The female worm is thin,, about 2.5 mm long and, the male is shorter and broader.
- Paired uteri lead to the vulva situated at the junction of the middle and posterior thirds of the body.
- The worm is ovoviviparous, the eggs laid in the mucosa hatch immediately, releasing rhabditiform (first stage) larvae.
- The rhabditiform larvae migrate into the lumen and pass down the gut to be released in feces. When reaching the soil, they moult twice to become the infective filariform (third stage) larvae.
- The filariform larvae are slender, its nonfeeding and can live in soil only for about 12 days.

## Life Cycle

The life cycle of *S. stercoralis* is a complex, It is unique among human nematodes in that it has,

in addition to the **parasitic cycle**, a **free-living soil cycle**, in which it can persist for long periods in soil, feeding on soil bacteria.

### A- Parasitic Phase

### 1- Direct development

- The adult worm is found in the human intestine in the mucosa of the duodenum and jejunum.
- Only the female worms are seen in the intestine. It was believed that they are parthenogenetic and can produce offspring without being fertilized by the

male. But it has since been established that parasitic males do exist. They are not seen in human infections because they do not invade the intestinal wall, However, the majority of females are probably parthenogenetic.

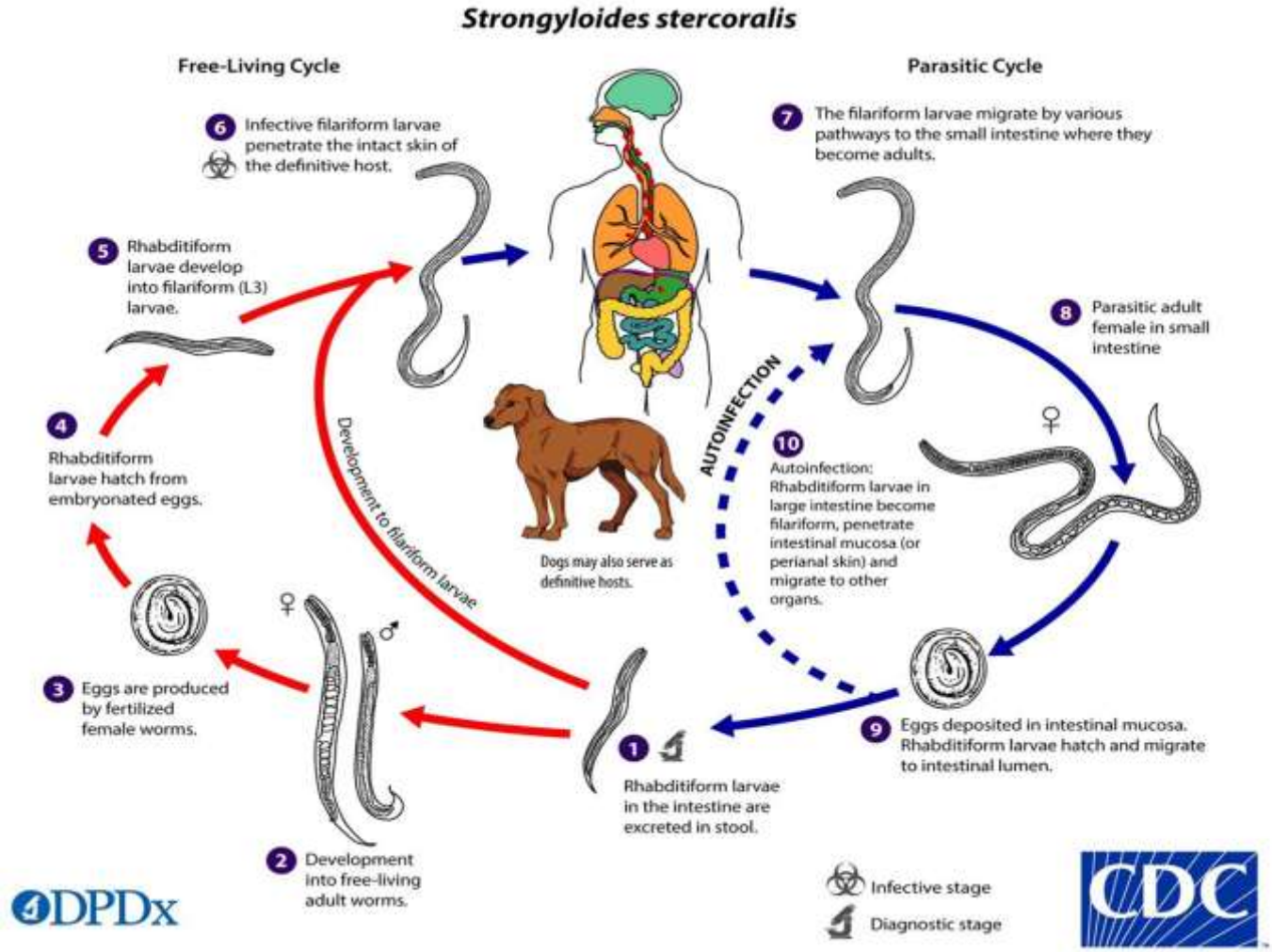
- When a person walks barefoot in soil, the infective filariform larvae will penetrate the skin, enter blood vessels and are carried along the venous circulation to the right side of the heart and to the lungs. Here migrate up the respiratory tract to the pharynx and are swallowed, reaching their final destination, the duodenum and jejunum, mature in 15-20 days and start laying eggs.

## 2-Autoinfection

- The worm also has a cycle of autoinfection. Here the rhabditiform larvae mature into the infective third stage larvae during their passage down the gut. These filariform larvae cause reinfection.
- The larvae wander in the dermis of the perianal region for sometime, causing a radiating perianal creeping eruption, a form of *cutaneous larva migrans*.
- They ultimately. enter the lymphatics or venules and are carried to the right heart and the lungs to complete the life cycle as above. This ability to cause autoinfection explains the persistence of the infection in patients for long periods, even 30 to 40 years.

## A- Free-living Phase

- The rhabditiform larvae passed in stools develop in moist soil into free-living males and females.
- They mate in soil. The fertilized female lays eggs which hatch to release the next generation of rhabditiform larvae. These may repeat the free-living cycle, or may develop into the filariform larvae which infect humans and initiate the parasitic phase.




## Pathogenesis and Clinical Features

Strongyloidosis is generally asymptomatic, But it may sometimes cause clinica manifestations, which may be severe and even fatal, particularly in those with defective immune response. The clinical disease may be classified as cutaneous, pulmonary and intestinal

### Cutaneous

- There may be a dermatitis, with erythema, itching, larva currens and haemorrhages at the site of penetration of the filariform larvae, when large numbers of larvae enter the skin.

- In those sensitised by prior infection, there may be an allergic response. Itching

### Pulmonary

During escape of the larvae from the pulmonary capillaries into the alveoli, small haemorrhages occur, along with cellular infiltration into alveoli and bronchioles.

### Intestinal

Mucus diarrhoea is often present. In heavy infection, the mucosa may be honeycombed with the worm and there may be extensive sloughing, causing dysenteric stools.

## Diagnosis

- Demonstration of the rhabditiform larvae in freshly passed stools, larva may sometimes be present in sputum and gastric aspirates.
- Stool culture when larvae are scanty in stools.
- Serological tests have been described, using strongyloides or filarial antigens. complement fixation, indirect haemagglutination and ELISA.
- Radiological appearances in intestinal infection are said to be characteristic and helpful in diagnosis.

# Nematoda

lec 27,28

## Hook worm

## *Ancylostoma duodenale*

## *&*

## Necator American

Morphology

- They are stout cylindrical worms, pale pink or greyish white, but may appear reddish brown due to ingested blood.
- The body is curved with the dorsal aspect concave and the ventral aspect convex.
- The anterior end is constricted and bent dorsally. This cervical curvature gave it the name hookworm.
- The mouth is not at the tip but directed dorsally. The prominent buccal capsule, reinforced with a hard chitin-like substance carries two pairs of hook-like teeth ventrally and a dental plate with a median cleft dorsally.

**Male** **Female**

## Habitat

The adult worms live in the small intestines of infected persons, mostly in the jejunum, less often in the—duodenum and infrequently in the ileum.

## Male

- Worm is about 8 to11 mm in length and about 0.4 mm thick. The posterior end of the male is expanded into a copulatory bursa supported by fleshy rays.

- The pattern of the rays helps in distinguishing between different species. The cloaca into which the rectum and genital canal open is situated within the bursa.

- There are two long retractile bristle-like copulatory spicules, the tips of which project from the bursa.

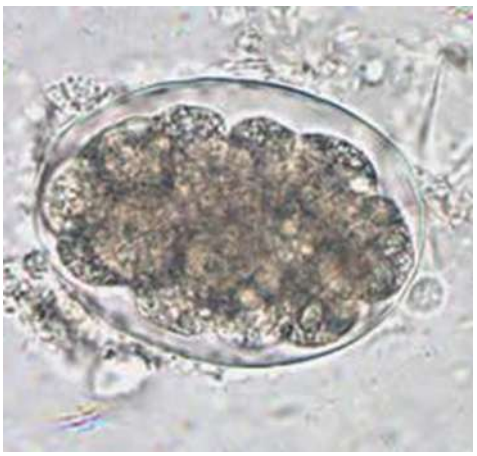
**Female**

- Is larger, 10 to 13 mm long and 0.6 mm thick.
  - Its hind end is conoid, with a sub terminal anus situated ventrally.
  - The vulva opens ventrally at the junction of the middle and posterior thirds of the body. The vagina leads to two intricately coiled ovarian tubes which occupy the hind and middle parts of the worm.

**Eggs**

- The eggs are oval or elliptical, measuring 60 µm by 40 µm, colourless, not bile stained, with a thin transparent hyaline shell membrane.
- When released by the worm in the intestine, the egg contains an unsegmented ovum, during its passage down the intestine, the ovum develops. When passed in feces, the egg contains a segmented ovum, usually with 4 or 8 blastomeres.
- There is a clear space between the segmented ovum and the egg shell.

## Eggs



**Filariform larvae**

## Transmutation

- When a person walks barefooted on soil containing the filariform larvae they penetrate the skin and enter the subcutaneous tissue. The common sites of entry are the skin between the toes
- Rarely infection may take place by the oral route, the filariform larvae being carried on contaminated vegetables or fruits

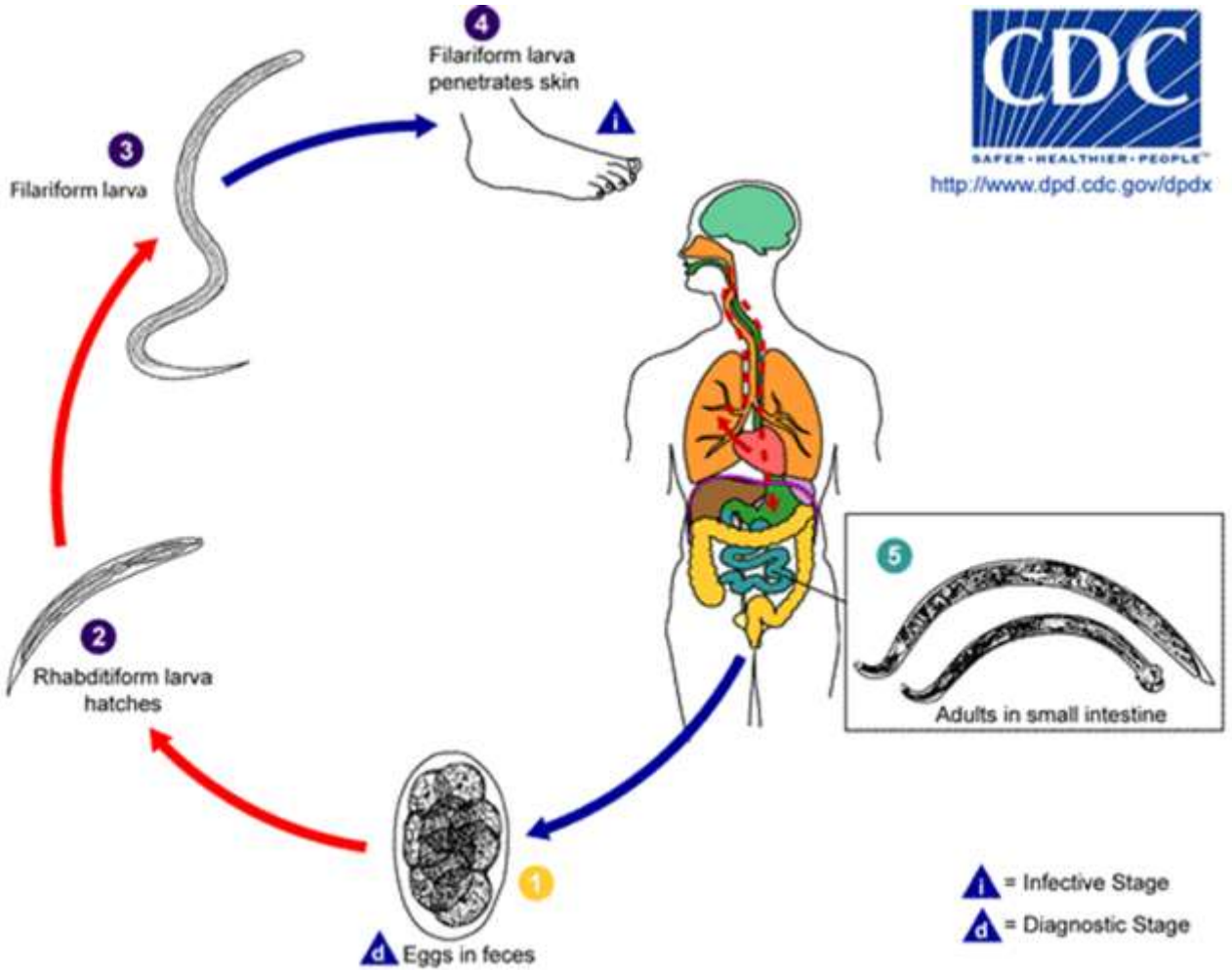
## Life Cycle

- Humans are the only natural host. Eggs freshly passed in feces are not infective for humans. When deposited in the soil, the embryo develops inside the eggs.
- In about 2 days, a **rhabditiform larva**, hatches out of the egg. It feeds on bacteria and other organic matter in the soil, grows in size and moults twice, on the 3rd and 5th days after hatching to become the third-stage infective **filariform larva**, with a sharp pointed tail.
- The filariform larvae are non-feeding. They can live in the soil, grass or other vegetation for about 5 weeks, waiting for their hosts.
- When a person walks barefooted on soil containing the filariform larvae they penetrate the skin and enter the subcutaneous tissue.
- In the subcutaneous tissue the larvae enter the venues and are carried in circulation to the right heart and to the lungs. In the lungs. they break out of the capillaries to reach the alveoli, from where they migrate up the respiratory tract to the epiglottis. They crawl over the epiglottis to the pharynx and are swallowed.

- During migration or on reaching the jejunum, they moult and develop a temporary buccal capsule by which they get attached to the gut mucosa. They feed and grow in size, undergo a fourth and final moulting, develop the buccal capsule and grow into adults.
- It takes usually about 6 weeks to 6 months from the time of infection for the adult worms to become sexually mature and start laying eggs.
- Rarely infection may take place by the oral route, the filariform larvae being carried on contaminated vegetables or fruits. The larvae may penetrate the buccal mucosa to reach the venous circulation and complete their migration via the lungs.

## Laboratory diagnosis

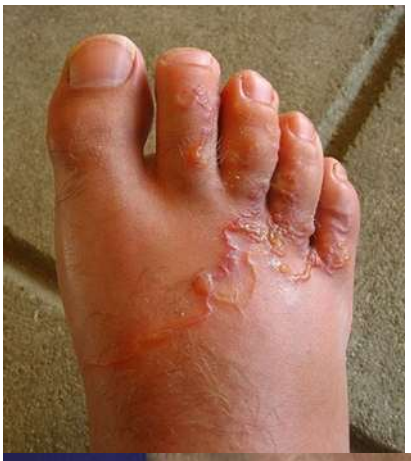
- Demonstration of the eggs in faeces by direct microscopy or by concentration methods is the diagnostic test. In stool samples examined 24 hours or more after collection, the eggs may have hatched and rhabditiform larvae may be present.

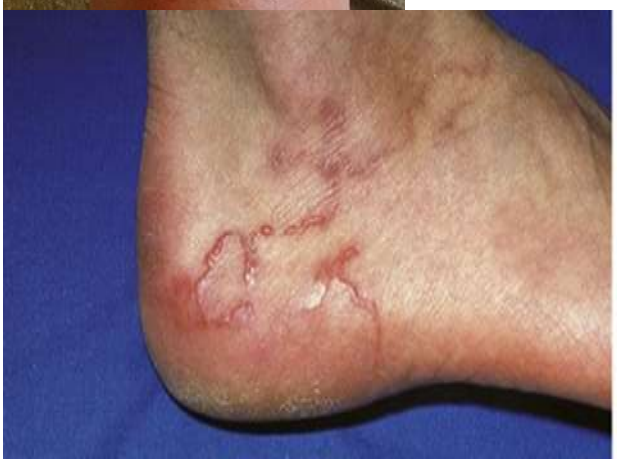


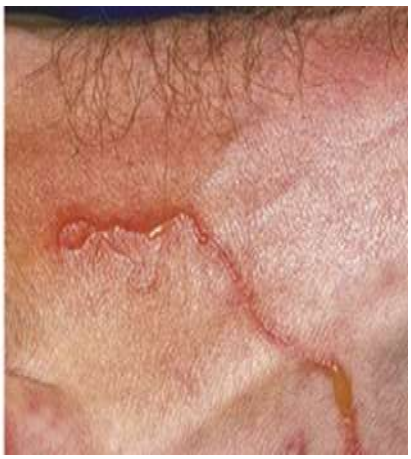
| Comparison between Necator american and *Ancylostoma duodenale* | |
|---|---|
| **Differences** | |
| **Necator american** | ***Ancylostoma duodenale*** |
| 1- The adult worms are slightly smaller | 1- Larger |
| 1- anterior curvature in opposite direction to body curve | 1- The anterior in adult female is curvature uniform with body curve; |
| 2- Vulva opens a little in front of the middle | 2- Vulva opens at junction of middle and posterior thirds |
| 3- have a smaller buccal capsule with 2 pairs of semilunar cutting plates instead of teeth | 3- has 2 pairs of hook-like teeth ventrally and a dental plate with median cleft dorsally |
| *4- Copulatory,* has a paired dorsal ray, making a total of 14 rays, copulatory spicules are fused at the tip | *4- Copulatory* Has a single dorsal ray with a split end making a total of 13 rays, copulatory spicules are separate. |
| 5- The lifespan is longer being about 4-20 years | 5- The lifespan is longer being about 4-20 years 2 to 7 years |
| **Similarity** | |
| The eggs are identical with those of *A. duodenale*. The life cycle is similar to that of *A. duodenale*. | |

## The clinical features

- Clinical disease may be due to larvae or adult worms.
- When the filariform larvae enter the skin, they cause severe local itching and secondary bacterial infection may follow.
- The more important manifestations of ancylostomiasis are caused by the adult worms in the intestine.
- The worms attach themselves to the gut mucosa by their buccal capsules, they suck into their mouth a portion of intestinal villi.
- adult *Ancylostome* can suck about 0.2 ml blood a day, this chronic blood loss leads iron deficiency anaemia.
- infection may cause epigastric pain, and vomiting, diarrhoea, the stool being reddish or black. This is more often seen in the acute stage, when the infection is heavy.







**lec28** **Filarial Worms**

- Slender thread-like worms (Latin, *filum*—thread) which are transmitted by the bite of blood-sucking insects.
- In the bodies of infected vertebrate hosts, they occur both as adults and the embryos, which are known as microfilariae. In some species, the microfilariae retain their egg membranes which envelope them as a sheath. These are known as ‘sheathed’ microfilariae, in contrast to others which rupture their egg membranes and come out as ‘unsheathed’ or naked microfilariae.

## Lymphatic filariasis

### *Wuchereria bancrofti*

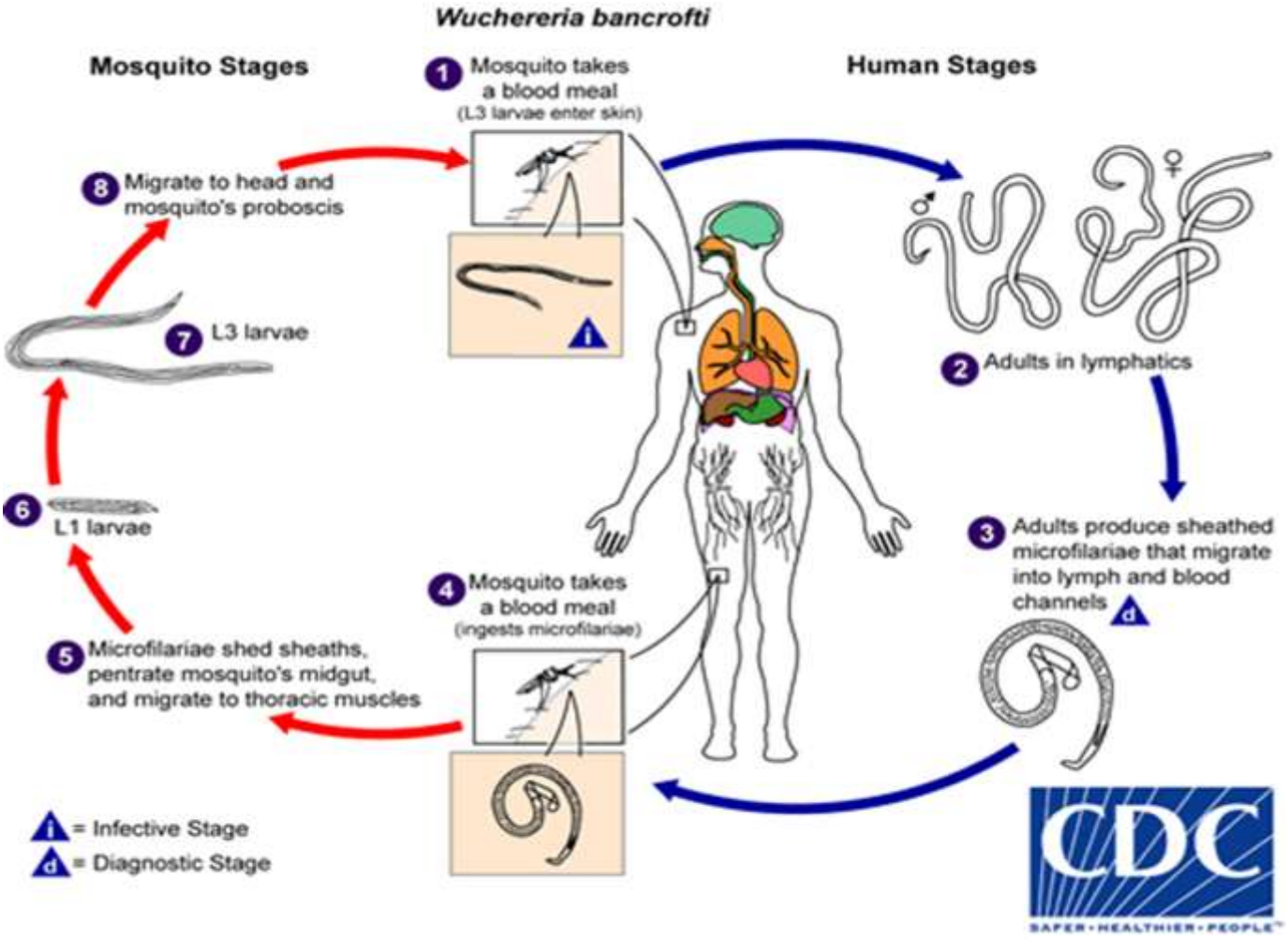
### Morphology

- The adults are whitish, translucent, thread-like worms with smooth cuticle and tapering ends.
- The female is larger than the male.
- Males and females remain coiled together usually in the abdominal and inguinal lymphatics and in the testicular tissues. The adult worms live for many years, probably 10 to 15 years or more.
- The worm is ovoviviparous. The embryo (microfilaria) is released encased in its elongated egg-shell, which persists as a *sheath*.
- The microfilaria has a colourless. it is actively motile and can move forwards and backwards within the sheath.

### Life Cycle

- Humans are the definitive host. No animal host or reservoir is known.
- The intermediate host is the female mosquito *Culex* sp.
- Microfilariae do not multiply or development in the human body. If they are not taken up by a female vector mosquito, they die.
- When a vector mosquito feeds on a carrier, the microfilariae are taken in with the blood meal and reach the stomach of the mosquito.
- They penetrate the stomach wall and migrate to the thoracic muscles where they development.
- During the next 2 days, they become **the first-stage** larva which is a sausage-shaped form with a spiky tail.

- Within a week, it moults once or twice, increases in size and becomes **the second-stage larvae**, In another week, it develops its internal structures and becomes the elongated **third-stage filariform larva** (actively motile). This is the infective larva. It enters the proboscis of the mosquito. When a mosquito with infective larvae in its proboscis feeds on a person, the larvae get deposited, usually in pairs, on the skin near the puncture site.
- The larvae enter through the puncture wound or penetrate the skin by themselves.
- After penetrating the skin, the third-stage larvae enter the lymphatic vessels and are carried usually to abdominal or inguinal lymph nodes, where they develop into adult forms. There is no multiplication at this stage and only one adult develops from one larva male or female.
- They become sexually mature in about 6 months and mate. The gravid female worm releases large numbers of microfilariae.
- They pass through the thoracic duct and pulmonary capillaries to the peripheral circulation


Wuchereria bancrofti
Mosquito Stages
Human Stages
1 Mosquito takes a blood meal
(L3 larvae enter skin)
2 Adults in lymphatics
3 Adults produce sheathed microfilariae that migrate into lymph and blood channels
4 Mosquito takes a blood meal
(ingests microfilariae)
5 Microfilariae shed sheaths, pentrate mosquito's midgut, and migrate to thoracic muscles
6 L1 larvae
7 L3 larvae
8 Migrate to head and mosquito's proboscis
i = Infective Stage
d = Diagnostic Stage
CDC
SAFER·HEALTHIER·PEOPLE™

## Pathogenicity

- Cause disease Lymphatic filariasis
  - The typical manifestations of filariasis are caused by the adult worms blocking lymph nodes and vessels, either mechanically or more commonly due to allergic inflammatory reactions to worm antigens and secretions.
  - The worms inside lymph nodes and vessels may cause granuloma formation and calcifiation.

## Diagnosis

- The diagnosis of filariasis depends on the clinical features.
- The laboratory tests that can be used for diagnosis include the following:

- Demonstration of microfilaria in peripheral blood.
- Demonstration of the adult worm in biopsy specimens.
-Skin tests with filarial antigens.
-Serological tests.

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Arthropods Transmitting disease

## 1- Suborder Nematocera

## Family Culicidae

### Morphology:

**Adult:** very slender insects,.

**Wing**: Scales cover wing veins and wing border, palps, thorax and abdomen..

**Abdomen**: They have a slender abdomen and long slender legs.

**Antennae** are long, of 14-15 segments, plumosed in males and pilosed in females.

**Mouth parts**: are adapted for piercing and sucking blood in females and sucking plant juices in males.

**Larva and pupa** are aquatic and very active.

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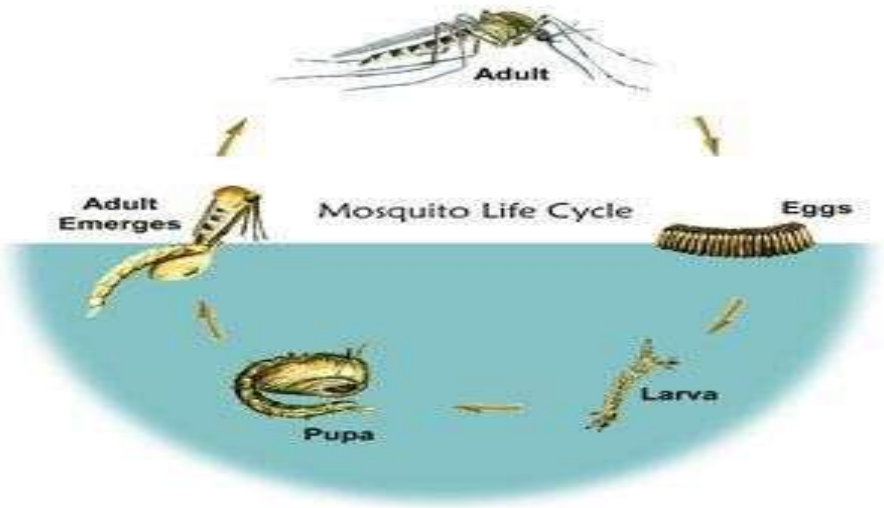
**Life cycle of Mosquitoes**

**Complete metamorphosis**

The fertilized female always needs a blood meal before egg laying. In a week, the female begins to lay its eggs.




Life cycle of Mosquitoes (*culex***)**

**Egyptian mosquitoes:**

***Anopheles pharoensis*:** The chief vector of malaria in Egypt. It is common in rice-growing areas, as it likes to breed in pools with a thick growth of weeds. It is more common in summer and autumn. It enters houses and prefers to bite by night.

***Anopheles sergenti*:** Found most commonly in Oasis, Sinai, and Fayum. Also breeds n rice fields and pools.

***Anopheles multicolor*:** Most common in Delta and Cairo. It breeds in small pools with or without weeds

***Anopheles gambia*:** This is not an Egyptian mosquito but it visited Egypt some years ago causing a severe epidemic of malaria.

**A) Culicine:**

1) ***Aedes aegypti:*** It was found throughout Egypt particularly in heavily inhabited districts. Due to the efforts of the health authorities, it has been extreminated. It is a domestic mosquito and can breed in "zeers", water tanks or barrels as well as in latrines or deep wells and "sakias". It prefers to bite through the day.
2) ***Culex pipiens*:** Very abundant species in Egypt. It can breed almost in any collection of water in and outside houses.

**Medical importance:**

**1. Anopheles mosquitoes:**

Transmit malaria and in some countries "Bancrofti's" **filariasis.**Not all anopheline mosquitoes are capable of transmitting **malaria.** Efficiency in transmission depends on the prevailing species and its feeding habits, which differs in preference for human or animal blood.

***Culex* mosquitoes** :

**a-Filaria of man (Wuchereria bancrofti).**

**b-Rift valley fever.**

**c-Encephalitis virus**

**d- yellow fever,**

**e-Dengue fever.**

***Aedes* mosquitoes:**

- **Filaria of man (*W. bancrofti*).**
- **Filaria of dog (*D. immitis*).**
- **Dengue virus.**
- **Rift valley fever.**
- **Yellow fever.**
- **Zika virus**

## Control of mosquitoes:

### 1- Measures against adult mosquitoes:

a) Use of wire screening and mosquito nets: The following points should be noticed in screening:
   - Size of mesh and quality of material.
   - Screened doors should be opened outwards, never inwards, -
     Better to use sliding than hinged or swinging windows.

b) Spraying with chemicals: Kerosene ***Pyrethrum*** extracts, "D.D.T." or "Gammexane" are good insecticides. Combination of two or more of these chemicals may be used. "Pyrethrum" has a knocking down effect but has no residual effect; while "D.D.T." and "Gammexane" are particularly of great value because of their prolonged residual killing effect (may be as long as three months).

c) Using of mosquito repellents: When applied to exposed parts of the skin, these substances repel mosquitoes from biting e.g. "oil of citronella", "indalone" and "dimethyl phthalate".

d) Animal barriers: This depends on the feeding habits of the local mosquitoes. Anthropophilic mosquitoes (feeding only on human blood) are not controlled by animal barriers which are effective against zoophilic mosquitoes (feeding on animal blood) could be attracted to animals rather than to man.

### 1. Measures against larval stages:

a) Draining off collections of water.

b) Filling up of ponds and water collections.

c) Changing the growth requirements of larvae e.g. development of shade where they require sunshine and production of turbidity in place of clear water.

d) Spraying oil on breeding places. This suffocates and poisons larvae and pupae.

e) Dusting with “Paris green”. This is a green salt, the particles of which float over water for many hours. It is a stomach poison and is particularly effective against ***Anopheles*** larvae because they are surface feeders. It has no effect against pupae as they do not feed. It is used diluted with suitable material as oven dust (one part in 99 of diluent).

f) Spraying of “D.D.T.” This is a contact toxic material, directly affecting the peripheral nervous system and also muscles causing irritability, paralysis and death. It has no effect on pupae.

g) Introduction of natural enemies as “*Gambusia”* fishes. These fishes are top feeders, feeding preferably on larvae. They possess marked fecundity and rapid rate of reproduction. They withstand handling and transport. These fishes are worthless as food for man.

## 2. Family Psychodidae

## (Sand flies)

**1- Genus *phlebotomus*:** occurs in Old World tropics,Mediterranean region and West Africa.

**Life cycle: Complete metamorphosis**

**Habits:**

The adults are poor fliers. They tend to "hop" for short distance.

They are active at night only and hide during the day in dark corners.

They can pass through ordinary mosquito nets because of their small size.

Only females are blood suckers and cause irritation to man, animals and birds.

Males feed on plant juices.

**Medical importance:**

- Leishmaniasis caused by ***Leishmania donovani. L.tropica (Phlepotomus) and L. braziliensis ( Lutzomyia).***
- Sand fly fever (papatasi fever or 3 days fever)
- Bartonellosis "Oroya fever" or "Carrion's disease".
- Sand fly bites produces local indurated lesion.

## 3. Family Simulidae

## *Simulium spp*. (black flies)

**Morphology**

The small species of this family are often called black flies, Coffee flies or buffalo gnats.

**The body** is stout, covered with short white,**colour:** yellow, orange or silvery hairs. Horn like antennae, small piercing proboscis. **Thorax** is humped with prominent anterior Wing veins and reduced posterior.

It breeds in rapidly running streams of water.

**Medical importance:**

1. The bite of ***Simulium*** species, to man is painful followed by an intense itching ,raised ulcerative lesions,and hemorrahgic spots at the site of bite due to the irritation produced by salivary secretion ,fever, nausia and headache(black fly fever).
2. ***Simulium*** transmits:***Onchocerca volvulus*** to man (river blindness).

## 4. Family Tabanidae

1. Genus ***Chrysops*** "Deer fly or Horse fly"

These are quite large flies with prominent brightly coloured eyes and a head thatis broader than the thorax. Females only are **blood sucking.**

**Medical importance:**

***Chrysops*** transmits ***Loa loa;*** while

## 5. Family Muscidae

### 1. *Musca domestica* "House fly" Morphology

This is non-biting insect, cosmopolitan**.**

**Size**: medium sized**.**

**Arista** are bilaterally plumosed up to its tip**.**

**Wing**: the 4$^{th}$ vein of the wing has a sharp curve and first posterior cell is closed ornearly so.

**Thorax** :

with 4 longitudinal dark stripes.

**Mouth parts** are adapted for lapping or sucking.

It either sucks fluid stuffs or regurgitates some saliva to dissolve solid substancesbefore sucking it.

**Life cycle:** complete metamorphosis

**Medical importance:**

(A)- **Disease transmission**: The adult fly transmits microorganisms to our food or body directly. Transmission may be either mechanical:

**1. Mechanically:**

- Contaminated legs and hairs of the fly.
- Through human faces, when ingested by the fly and passed with its faces

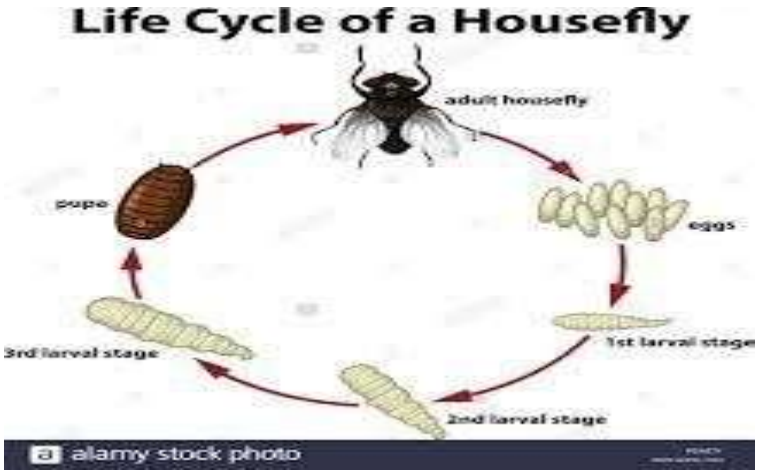
unchanged.

- By the vomit drop, as the fly sucks fluids until its diverticulum becomes filled; when such fly drops on sweet food, it vomits these fluids so as to dissolve it and sucks it again.

**Organisms transmitted in this way are:**

- **Bacteria:** ***Vibrio cholera, Streptococci. Shigella, Salmonella, Escherchia coli.***
- **Protozoa**: ***E. histolytica*** cysts, ***Giardia*** cysts, ***Balantidium*** cysts.
- **Helminth eggs:** as ***Taenia, Ascaris***, ***Trichuris, Oxyuris***, and ***Hymenolepis nana.***

**(B)-Disease causation:** Larvae of muscid flies may cause accidental intestinal myiasis, urogenital, aural or traumatic myiasis.




**Developmental stages of *Musca domestica***

**A-eggs B-larva C- D- shaped peritreme D-pupa**

## *2-* *Stomoxys calcitrans*

## "Stable fly"

**This fly differs from *Musca domestica* in the following:**

1. Proboscis is prominent; mouth- parts are adapted for piercing and sucking ofblood.
2. The 4$^{th}$ vein of the wing curves gently and 1$^{st}$ posterior cell is widely opened.
3. Thorax with 4 longitudinal stripes, lateral pair of which are narrow.
4. Arista carries hairs on dorsal surface only.
5. Posterior spiracles of larvae have three S-shaped slits.

**Medical importance:**

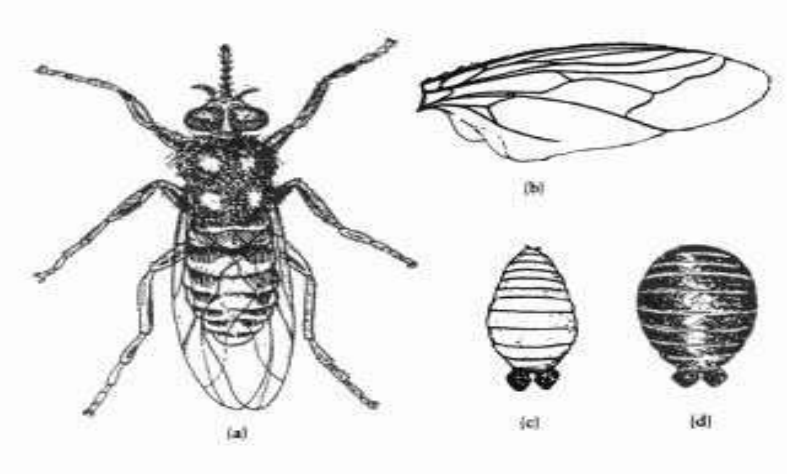
1. Painful bite.
2. Mechanical transmitters of trypanosomiasis.

## *3.* Genus *Glossina (G. palpalis & G. morsitans)*

These two species are the most important **blood sucking flies** since they transmit the two species of trypanosomes which cause sleeping sickness to man, which is a fatal disease. They occur only in Central Africa.

### Morphology:

**Size:**Comparatively big fly, about 2 or 3 times the size of the house fly. **Color** :differs according to species, yellowish brown or dark brown or blackish. **proboscis** is long, chitinised, horizontal in position and is protruding, with a bulbose base( differs than ***Stomoxys*** in; More chitinised .




**maxillary palps** are nearly as long as proboscis). The arista is provided with compound bristles (hairs) on its upper side.

**Wing**: The venation of the wing is characteristic. The 4th vein has a double curve before it ends near the apex, and the discal cell (between the 4th and 5th) is cleaver-shaped.

**The abdomen** is of seven apparent segments.

## Habits:

The mouth parts are adapted for piercing and sucking and both sexes suck blood,usually they are diurnal in habit.

The female are viviparous It gives one larva every 10-14 days.

The female carries its eggs in the uterus until they hatch and grow into mature larvae.

## Medical Importance:

The two species vary greatly in their adaptation to environment and consequently in their distribution.

***G. palpalis*** is essentially an West African species and it is the most important vector of Gambian(chronic) sleeping sickness. It occurs chiefly in Congo and West Africa. It prefers to live along water courses and lakes and is known as “Wet fly”. It lays larvae along shores in shade of trees.

***G. morsitans*** is the vector of Rhodesian (acute)sleeping sickness.It is

found in Sudan and Rhodesia. It lays larvae in dry open spaces.

**Control:**

No effective control has been devised. Extensive spraying and dusting in some parts of Africa, combined with control of grasses and other vegetations, particularly along lakes, rivers and streams has greatly reduced its prevalence. Repellents provide temporary protection and the use of insecticidal sprays and aerosols in homes and buildings provides partial protecti