Medical Helminthology علم الديدان الطبية

Introduction:

The field of medicine that relates to helminths (worms) which are capable of causing infectious disease to humans and/or animals.

Helminthes are trophoblastic metazoa (multicellular) eukaryotic animals spend part or entire life cycle in a human or host. They generally possess digestive, circulatory, nervous, excretory, and reproductive systems. Some are free-living in soil and water, while the helminthes of medical importance are parasitize on human or animal. Helminthes parasites are more complex than free-living helminthes, because they have evolved mechanisms to deal with the different environments of their various hosts and living conditions.

Helminthes cause different diseases in humans worldwide, on a global basis it is estimated that up to 500 million people may be infected at any given time with pinworms. Although 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.

The sources of the parasites are different. Exposure of humans to the worms 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 cats.

4- Person to person as in Enterobius vermicularis, Hymenolopis nana.

5- Oneself (autoinfection) as in Enterobius vermicularis.

Helminthes are studied in microbiology because they cause infectious diseases and most are diagnosed by microscopic examination of eggs or larvae (main infective stages of worms).
Worms do harm their hosts in the following ways:

1- By causing irritation, as in *Fasciola*.
2- By absorbing the food intended for the host, as in tapeworms.
3- By sucking blood or lymph, as in hookworms.
4- By feeding on the tissues of the host, as in *Ascaris*.
5- By causing mechanical obstruction and pressure, as in microfilaria.
6- By causing the growth of nodules and tumors and by perforating vessels, as in *Opisthorchis felineus* and *Wuchereria bancrofti*.
7- By causing wounds through the site of infection, as in *Ascaris* or by reducing the resistance of the host to other infections, as in lungworm.
8- By secreting toxins or harmful substances, *e.g.* anti-coagulants secreted by hookworms, anti-digestive enzymes secreted by intestinal worms.
9- By transmitting casual agents of infectious disease, such as bacteria, viruses, blood protozoa and spirochaetes.

**Signs and symptoms of worm’s infection:**

Helminthes inhabit most areas of the world and produce a large proportion of the parasitic infections suffered by humans. Signs and symptoms are many and varied and are dependent upon the type of worm with which the victim is infected. People with intestinal worms infections are usually undernourished and weak, and may be concurrently infected with viruses, fungi, or bacteria. The malnourished state may also lead to certain types of chemical and metal poisoning due to toxins excreted by the parasites.

There is no difference in age or gender of those who experience parasitism, as human intestinal parasites can be present with any disease, in any person, and at any age. Worm infections may show a broad and confusing array of symptoms, many of which are related to the gastrointestinal tract. The most commonly reported complaints are periods of diarrhea, alternating with periods of constipation. This may be accompanied by bloating and possibly edema, nausea, loss of appetite, other signs of an irritated and possibly behavioral changes. Furthermore, clinical signs may appear as anemia, irritation of the gastrointestinal mucosa, dehydration and blockage of the organs as common complaints.

Certain intestinal worms called flukes may cause severe disease of the gastrointestinal tract, bladder, or liver and may destroy large numbers of blood cells. Some parasitic worms have the ability to fool bodies into thinking they
are normal part of the tissue or organ and the immune system will not fight off the intruders. A number of worm caused infections can cause physical trauma by perforating (burrowing) into the intestines, the circulatory system, the lungs, the liver, or the skin of body. They can break down, damage, or block organs of the body by forming clumps as balls or tumors, and sometimes are even mistaken for cancer tumors. A number of species are also able to travel to the brain, heart, and lungs, where they invade the tissues of these vital organs.

**Eosinophilia:**

An important clinical finding that is sometimes discovered in a common blood test is found in the CBC (complete blood count), which includes a count of the percentages of certain white blood cells that might show an increase in a white blood cell called the eosinophil. Eosinophils are white blood cells that normally comprise only a small percentage of up to 3% of the leukocytes in the blood. They increase significantly in many allergic responses and in some worm infection which invade the tissues of the host. Some of the kinds of worms and medical conditions that may elicit a significant increase in eosinophils include: Trichinosis, Toxocariasis, Filariasis, Echinococcosis, Strongyloides, Ascariasis, Shistosomiasis.

**Life Cycle and Hosts:**

Parasitic helminthes are highly modified compared to free-living helminthes. They often lack sense organs such as eyes, and may even lack a digestive system. Their reproductive system, however is often complex, which ensures infection of new hosts. Some flukes can produce 25,000 eggs per day.

Adult helminthes may be Dioecious; male reproductive organs are in one individual, and female reproductive organs are in another. In those species, reproduction occurs only when two adults of the opposite sex are in the same host. Adult helminthes may also be Monoecious, or Hermaphroditic (one worm has both male and female reproductive organs). Two hermaphrodites may copulate and simultaneously fertilize each other.
Clinical diagnosis methods of helminthes:

Parasitic infections are usually diagnosed from samples of faeces, urine, blood and tissues; it can be direct or indirect.

**Direct identification:**

1- Evidence of intestinal parasitism, apart from the general clinical signs, is obtained from faecal examination. It can be investigated by:
   a- Direct saline smear: This procedure provides only an indication of the parasites present and cannot be used quantitatively. To prepare a direct faecal smear a drop of saline is placed in the centre of a microscope slide and a 2 mg faecal sample is suspended in this drop without spreading it and covered with coverslip.
   b- Stained smears: This type of smear is essential for accurate diagnostic and is suitable for long-term storage for record purposes. The two stains generally used are haematoxylin and trichrome.
   c- Parasite concentration in faeces by flotation: This is used for the identification of helminth eggs.

2- Urine examination of urine sediment is used mainly for the identification of *Schistosoma* eggs.

3- Blood testing is used to identify the various stages of blood parasites and is routinely applied to diagnose malaria, trypanosomiasis and most types of filariasis.
   a- Thin blood films are useful for studying morphological changes of blood cells and blood parasites.
   b- Thick blood films contain 6 to 20 times as much blood per unit area as thin films. The thick film is suited for rapid diagnosis of parasitaemia that is too low to be detected with thin films.

4- Tissue recovery of helminths from biopsy material is often an important aid for diagnosis. Lymph node, spleen, liver, lung, bone marrow or spinal fluid biopsies are used to diagnose a variety of worms.

5- Post-mortem: Post-mortem examination is an effective way to diagnose some helminth infection.
**Indirect identification:** indirect methods, must be used if the parasite density in the specimen is below the sensitivity of the method employed, or if the parasite cannot be directly demonstrated due to the life cycle in the host, such as, Complement fixation test (CFT), Indirect fluorescent antibody test (IFAT), Radioimmunoassay (RIA), Enzyme Linked Immunosorbent Assay (ELISA), Rapid card agglutination test (CAT).

**Nucleic acid-based diagnosis:** The use of nucleic acid probes in the diagnosis of parasitic infections, is based on the fact, that every organism carries unique DNA sequences. This technique proved specificity and sensitivity of diagnosis, such as Polymerase Chain Reaction (PCR).

**Intermediate host:** The host that carry the immature reproductive (non-sexual) stage of the worm. Human can serve as intermediate host for the dog tapeworm.

**Definitive host:** The host that carry the mature reproductive (sexual) stage of the worm. Human can serve as the definitive host for beef, pork, and fish tapeworms.

**Infective stage:** The larval stage of the worm, which attacks the final host to complete its life cycle.

**Helminthes Classification:**

1- Phylum Platyhelminthes, Trematodes and Cestods.
2- Phylum Nemathelminths, Nematodes.

Phylum Platyhelminthes (Flatworms) general characteristics:

1- Bilaterally symmetrical, dorsoventrally flattened, triploblastic and acoelomate.
2- Body shape generally worm-like but varies from moderately elongated shape to long flat ribbons and leaf-like.
3- Small to moderate in size varying from microscopic to extremely elongated forms measuring up to the 10-15 meters.
4- White, colorless, some derive color from the ingested food.
5- Anterior end of the body is differentiated into the so called head.
6- Presence of great variety of adhesive secretions, organs of attachment and adhesion (hooks and suckers).
7- Parasitic trematodes and cestodes, epidermis is lacking and the body is covered with cuticle.
8- Body is smooth due to lacking of endo and exoskeleton.
9- Body space between the various organs is filled with a mesenchyme usually called parenchyma.
10- Digestive system, if present, is incomplete.
11- Sexes are united (hermaphrodite) with very few exceptions.
12- Reproductive system is highly evolved in most of the forms.

**Classification of Platyhelminthes:**

**First:** Class: Turbellaria: Most are free-living.

**Second:** Class: [Trematoda](https://www.biology.wisc.edu/zoology/9999/9999Platyhelminthes.html) (having pores), general characteristics:
- 1- Ectoparasitic or endoparasitic forms, commonly called flukes.
- 2- Body shape usually Leaf-like, dorsoventrally flattened.
- 3- Body wall without epidermis and cilia, most are hermaphrodite.
- 4- Body undivided and covered with a cuticle, most are hermaphrodite.
- 5- Well-developed suckers usually present, ventral sucker sometimes termed the acetabulum
  - **Monostome** is used to describe worms with one sucker (no ventral sucker) e.g. *Notocotyulus*.

  **Distomes** is used to describe worms with two suckers (oral and ventral) e.g: *Fasciola*.

  **amphistomes** is used to describe worms with oral sucker and an acetabulum at the posterior end of the body, e.g: *Paramphistomum*.

- 6- Digestive tract incomplete consisting of mouth, pharynx and two forked or many branched intestine; anus absent.
- 7- Protonephridia excretory system consisting of flame cells.

**Order- Monogenea:**

- 1- Ecto-or endoparasitic worms on vertebrates.
- 2- Oral sucker either weak or absent.
- 3- Anterior end provided with a pair of adhesive structures.
- 4- Posterior end provided with an adhesive disc(opisthaptor )
- 5- Free- swimming ciliated larva called [onchomiracidium](https://www.biology.wisc.edu/zoology/9999/9999Platyhelminthes.html)
- 6- Only one host in life cycle. e.g. *Gyrodactylus*
Order-Aspidobothria:

1- Endo-parasites in the gut of fish and reptiles.
2- Oral sucker is absent.
3- The anterior end without an adhesive structure.
4- Life cycle simple, no alternation of hosts. *e.g. Aspidogaster*

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**Monogenea:** *Gyrodactylus*

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Aspidobothria: *Aspidogaster*

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Order-Digenea:

1- Endo-parasites of vertebrates and invertebrates.
2- Mostly with two suckers without hooks.
3- Life cycle is complicated involving many larval stages.
4- One or more intermediate hosts in life cycle.

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General life cycle of class trematoda
Class: Cestoda

1- Endo-parasites in the intestine of vertebrates, called tapeworms.
2- Body without epidermis and cilia but covered with cuticle.
3- Body is divided into few to many segments (proglottids).
4- Anterior end (scolex) is provided with adhesive structures (hooks or suckers) except in subclass Cestodaria.
5- Mouth and digestive tract totally absent.
6- Excretory system consists of protonephridia.
7- Nervous system usually comprises a pair of ganglia and two lateral longitudinal nerve cords.
8- Each segment contains one or two sets of complete hermaphroditic reproductive system.
9- Life cycle complicated usually involving two or more hosts.
10- Embryos possess hooks.

Parasitic adaptation: Parasitic Fitness of the parasite to its environment. It is the characteristic which results in suitable and convenient morphological and functional correlation between an organism and its environment.

Parasitic adaptation of Platyhelminthes: Platyhelminthes have undergone profound adaptation of morphological and physiological nature.
A- Morphological adaptations:

1- Body covering: thick tegument frequently provided with scales affords suitable protection to the parasite. This thick protoplasmic layer is continually renewed by mesenchymal cells forming it.

2- Organs of adhesion: for a firm grip on/in the host’s body, some special organs of adhesion are needed. Flatworms are variously armed with suckers, hooks and/or spines.

3- Organs of locomotion: food of parasite comprises readily available and digested/semi digested food of the host. Elaborate organs of nutrition not needed. They have an incomplete gut and in most cases a suctorial pharynx for sucking food. In cestodes, worms freely bathes in digested food of host, which is absorbed directly, thus, total absence of alimentation in tapeworms.

4- Neurosensory system: need for quick & efficient “response to stimuli” is associated with free active life and not with a quiet parasitic life in a safe environment. In parasitic worms therefore, there is preferred reduction of nervous system and a total absence of sense organs but the free-living miracidium possesses eyespot.

5- Reproductive system: best developed system in helminth parasites, designed and preferred to meet the need for tremendous egg production. Parasitic flatworms with a few exceptions (like Schistosoma) are monoecious (hermaphrodite). Hermaphroditism is of distinct advantage to the parasite because:
   • It ensures copulation even when a few individuals are present.
   • After copulation, both individuals lay eggs, doubling the rate of production.
   • In the absence of a companion, the parasite can reproduce an offspring. In cestodes, reproductive system is much more elaborate and each mature proglottid possesses 1 or 2 complete sets of male and female genitalia. In gravid proglottid, all other organs of the system degenerate to make room for the uterus which becomes highly enlarged and branched to accommodate large number of eggs.
B- Physiological adaptations: تأقلم وظيفي

1- Protective mechanism: inside the alimentary canal the parasites have to protect themselves from the action of digestive juices of host. Tapeworms accomplish this by:
   - Stimulating walls of gut to secrete mucus, which then forms a protective clothing around parasite.
   - Secreting antienzymes to neutralize the digestive enzymes of host.
   - Continually renewing their protective body covering, e.g. tegument.

2- Anaerobic respiration: environment in gut and bile ducts is devoid of free oxygen. Flatworms inhabiting these places, therefore, they respire anaerobically by breaking down glycogen.

3- Osmoregulation: osmotic pressure of endoparasite’s body fluids, especially in case of trematodes is almost the same as that of host. This reduces unnecessary osmoregulation. But in intestinal tapeworms, osmotic pressure is little higher, this permits ready absorption of host’s digested food by tapeworms.

4- High fertility: eggs produced by a parasitic flatworm face a very uncertain future while passing through the complex life cycle; these potential off-springs face several hazards as a result of which a very small percentage of total eggs can grow and reach adulthood. Consequently, many worms may produce eggs in millions and their reproductive organs are accordingly developed.
Phylum Platyhelminthes

Platyhelminthes can be divided, according to the worms’ habitat in the final host, into:

1- Liver flukes مخرجات كبدية
2- Intestinal flukes مخرجات معوية
3- Blood flukes مخرجات دموية
4- Lung flukes مخرجات رئوية

First: Liver flukes

Fasciola hepatica (sheep liver fluke) de Brie, 1883

Fasciola gigantica (large liver fluke) Cobbold, 1856

Fascioliasis or liver rot is a disease caused by two species of parasitic flatworms or trematodes that mainly affect the liver. It belongs to the group of water borne and foodborne trematode infections and is a zoonosis, meaning an animal infection that may be transmitted to humans.

Human cases occurred occasionally but are now increasingly reported from Europe, the Americas and Oceania (where only *F. hepatica* is transmitted) and from Africa and Asia (where the two species overlap). WHO estimates that at least 2.4 million people are infected in more than 70 countries worldwide, with several million at risk. No continent is free from fascioliasis, and it is likely that where animal cases are reported, human cases also exist.

1- *Fasciola hepatica* (sheep liver fluke)

Morphology:

*Adult Worm*: averaging 30 mm in length and 13 mm in width, *F. hepatica* is one of the largest flukes in the world. The adult worm has a very characteristic leaf shape with the anterior end being broader than the posterior end and an anterior cone-shaped projection.

The fluke possesses a powerful oral sucker at the end of the anterior cone and a ventral sucker (acetabulum) at the base of the cone, which allow it to attach to the lining of the biliary ducts.

Each worm possesses ovaries and testes, which are highly branched and allow for individual flukes to produce eggs independently.
**The Egg:** is operculated and average 140 μm in length and 75 μm in width, egg of *F. hepatica* is un-embryonated when released.

**Body wall:** The wall of *F. hepatica* lacks a cellular layer of epidermis. However, it consists of a thick layer of cuticle made of a homogeneous layer of scleroprotein to protect the fluke from the juices of the host, a thin basement membrane.

Smooth muscle layers consist of an outer layer of circular muscle fibers, middle layer of longitudinal muscle fibers and an inner layer of diagonal muscle fibers.

Mesenchyme below the muscles with numerous loosely arranged uninucleate and binucleate cells with syncytial network of fibers having fluid filled spaces. Mesenchyme helps in the transport of nutrients and wastes substances.
**Digestive system:** The alimentary canal of *F. hepatica* is incomplete. The digestive system begins with mouth surrounded by the oral sucker, highly muscular pharynx, esophagus and intestine but does not terminate in anus that is why it is referred as incomplete. The intestine immediately forks to form right and left limbs or branches that run along both side of the body up to the posterior extremity and terminate blindly. These branches of the intestine are called as crura or intestinal caeca.

Digestive system from the mouth up to the esophagus is lined by cuticle. This region of the alimentary canal is the fore gut region that serves as efficient suctorial apparatus. The intestine is lined by columnar epithelial cells that are endodermal in origin. Numerous secretory gland cells surrounded by a thin muscular layer consisting of circular and longitudinal muscle fibers are present in the caecal epithelium.

The fluke often migrates into the bile ducts and the capillaries of the host for nourishment. It feeds on blood, lymph, inflammatory exudates and bile juice which it sucks from the wall of the host’s bile passages.

**Excretory system:** The excretory system is concerned with excretion as well as osmoregulation. It consists of a large number of flame cells or flame bulbs or protonephridia connected with a system of excretory ducts to the excretory pore at the posterior end. Flame cells are irregular in shape bulb like bodies; each has a thin elastic wall with pseudopodia like processes and nucleus. An intracellular cavity has many long cilia arising from basal granules. The cilia vibrate like a flickering flame, hence, the name flame cell.

**Nervous system:** A nerve ring surrounds the esophagus, it has a pair of cerebral ganglia and a ventral ganglion below the esophagus, and small nerves are given out anteriorly from the ganglia. Posteriorly three pairs of longitudinal nerve cords arise the ganglia (dorsal, lateral and ventral nerve cords), the lateral nerve cords are best developed, and they run to the posterior end and give out many small branches. Sense organs are lost in adult fluke.
Respiration: Mode of respiration is anaerobic or anoxybiotic. Glycogen is metabolized to carbon dioxide and fatty acids releasing energy in the form of heat. The carbon dioxide, thus, produced is diffused out through general body surface and the fatty acids are excreted through the excretory system.

Reproductive system: Fasciola hepatica is hermaphrodite. The gonads are well developed and the male and female genital ducts open into a common chamber, the genital atrium. It is situated anteriorly in the body and opens to the exterior through the common genital aperture or gonopore, located ventrally in front of the acetabulum.

Male Reproductive Organs:
- **Testis:** greatly ramified, in the middle of the body, one behind the other.
- **Vasa deferentia:** A narrow delicate duct, the vas deferens, emerges out from each testis and rims forward to meet the fellow of opposite side forming the common sperm duct just below the ventral sucker.
- **Seminal vesicle:** large muscular pear-shaped sac, lies in front of ventral sucker.
- **Ejaculatory duct:** fine convoluted tube from the seminal vesicle runs forward in a zigzag fashion through the cirrus to open into the genital chamber through the male genital pore.
- **Cirrus and cirrus sac:** The cirrus or penis is a muscular cylindrical structure traversed internally by the passage of ejaculatory duct. It can be pushed out
and drawn in through the genital pore and thus helps in copulation. The cirrus and the seminal vesicle both are enclosed in a bag-like cirrus sac.

f- **Prostate glands**: unicellular prostate glands around the ejaculatory duct.

**Female Reproductive Organs:**

a- **Ovary**: large, highly branched tubular structure on the right, in front of testes.

b- **Oviduct**: short, narrow and convoluted duct runs down ward to join the uterus.

c- **Uterus**: long wide and highly convoluted tube that extends up to the genital atrium, opening into it through the female genital aperture, close to male genital pore on the left side. It contains a large number of capsules containing fertilized eggs.

d- **Mehlis gland**: also called shell glands but they do not play any role in shell-formation, surrounding the base of the oviduct, function of this gland includes secretion helps in lubricating uterus for smooth passage of eggs and in activating sperms.

e- **Laurer’s canal**: The Laurer’s canal arises from the oviduct and acts as a sperm duct. It develops a temporary opening on the dorsal body surface during breeding season. It may serve as vagina.

f- **Vitellaria (Vitelline glands and vitelline ducts)**: provide eggs with yolk.

**Life cycle of *Fasciola hepatica***:

Unembryonated eggs pass through feces, following the passage of the eggs in the stool, micacidia develop within 2 weeks, escape from the eggs, and infect the *Lymnaea* snail, which acts as the first intermediate host penetrated by the miracidia.

Inside the snail, miracidia reach the digestive gland and within 14 days, the second larval stage is produced, called sporocyst. Further development occurs when the germ balls in the sporocyst give rise to the next generation of redia. *F. hepatica* has two generations of redia, mother and daughter redia. Active daughter redia are eventually transform to the cercaria, which they leave the snail to the outer environment.
Cercaria are released into fresh water where feces is dumped or runs into the water. There, they encyst and form metacercariae on freshwater vegetation called watercrass, which is considered the second intermediate host. Humans and other mammals are infected after eating the contaminated vegetation. The metacercariae excyst in the duodenal portion of the intestine and the larvae penetrate the intestinal wall and enter the gallbladder and bile ducts of the human host. The adult worms attach themselves in the large bile ducts and the gallbladder of humans rather than in the intestines as other parasites do, but eggs are passed from the bile duct into the intestine and are excreted in feces.

Symptoms and signs:
After the larvae are ingested with contaminated food or water, a symptomless incubation period starts, lasting for a few days to a few months. This is followed by an acute and a chronic clinical phase:

*Acute phase:* The acute phase, lasting 2-4 months, begins when the immature worms penetrate the intestinal wall and the peritoneum, the protective membrane surrounding the internal organs. From here, they puncture the
liver's surface and eat their way through its tissues until they reach the bile ducts. This invasion kills the liver's cells and causes intense internal bleeding. Typical symptoms include fever, nausea, a swollen liver, skin rashes and extreme abdominal pain.

**Chronic phase:** The chronic phase begins when the worms reach the bile ducts, where they mature and start producing eggs. These eggs are released into the bile and reach the intestine, where they are evacuated in faeces, thereby completing the transmission cycle. Symptoms include intermittent pain, jaundice and anaemia. Pancreatitis, gallstones and bacterial super-infections may also occur. Patients with chronic infections experience hardening of the liver (fibrosis) as a result of the long-term inflammation.

In animals, *F. hepatica* is responsible of a disease called “liver rot”, which is the main infection in animals, this occurs by mechanical and toxic destruction of liver tissue by passage of immature worms necrosis, fibrosis, hepatitis, and hepatomegaly. Obstructive jaundice also occurs when the adult worms reside in the bile ducts which leads to irritation and thickening of the ducts and stone formation.

**Laboratory diagnosis:**
The characteristic eggs of *F. hepatica* in feces. Entero-test string procedure may also be helpful. The Entero-test string procedure is a simple and non-invasive method useful in sampling duodenal fluid. Eggs are recovered from duodenal aspirates containing bile fluid and can be diagnosed as *F. hepatica* eggs reside exclusively in the bile duct and the gallbladder. Serological methodology is also available for early diagnosis of the liver fluke infection.

**Treatment and prevention:** Bithinol, which is a halogenated phenol, is used for treatment of the infection. Triclabendazole is also effective as treatment for this worm. Prevention of infection is accomplished by avoiding raw vegetation growing in watery environments in endemic regions. Adherence to good sanitary practices where raw human sewage is properly treated and disposed of will also prevent infection.
2- *Fasciola gegantica* (large liver fluke)

*Fasciola gegantica*, is the largest trematode of the human flukes. It measures up to 75 mm in length, 12 mm in width. It tends to be more oblong with a longer rounded posterior end as compared to broadly pointed posterior end of *F. hepatica*. It has a shorter cephalic cone, a larger ventral sucker and a more anterior position of the testes.

The eggs of *F. gigantica* are larger (180μm x 80μm) than those of *F. hepatica* (140μm x 80μm) and has a shorter anterior cone. It lives in the bile duct of herbivorous mammals, including human. It has been reported from Africa, Asia, Hawaii, Russia, Vietnam and Iraq. The life cycle is similar to that of *F. hepatica*, but *F. gigantica* employs different snails as intermediate hosts. Development is slower, and metacercariae are more susceptible to desiccation. Pathology is similar to those of *F. hepatica*.

*F. gigantica* may also be found in ectopic locations, when metacercariae enter the circulation and are distributed in abnormal sites e.g. peritoneum, lungs, brain, eyes and cause fibrosis.

The prepatent period between infection and the presence of adult worms in the bile ducts is 9 to 12 weeks. Patients may experience fever, nausea, vomiting, abdominal pain, hepatomegaly hepatic tenderness, and eosinophilia. Abscess or tumor like reactions have also been reported to occur in subcutaneous tissues or in the liver.
<table>
<thead>
<tr>
<th>Parameter</th>
<th><em>F. gigantica</em></th>
<th><em>F. hepatica</em></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Length</strong></td>
<td>3-7 X 1 cm</td>
<td>2-3 X 1.3 cm</td>
</tr>
<tr>
<td><strong>Cephalic cone</strong></td>
<td>Smaller</td>
<td>Larger</td>
</tr>
<tr>
<td><strong>Lateral sides</strong></td>
<td>Parallel</td>
<td>Converging</td>
</tr>
<tr>
<td><strong>Inner intestinal branches</strong></td>
<td>T and Y shaped</td>
<td>rudimentary</td>
</tr>
<tr>
<td><strong>Suckers</strong></td>
<td>Ventral larger than oral</td>
<td>Equal</td>
</tr>
<tr>
<td><strong>Final host</strong></td>
<td>Mainly cattle, Buffalo</td>
<td>Sheep</td>
</tr>
</tbody>
</table>
Phylum: Platyhelminthes, Class: Trematoda, Liver flukes

- *Opisthorchis viverrini*, Southeast Asian liver fluke- Poirier, 1886
- *Opisthorchis felineus*, Cat liver fluke - Sebastiano Rivolta, 1884
- *Dicrocoelium dendriticum*, Lancet liver fluke- Looss, 1899
- *Clonorchis sinensis*, Chinese liver fluke- Looss, 1907

**Taxonomy and geographical distribution:**

*Opisthorchis viverrini* and *Clonorchis sinensis*, which infect human and *Opisthorchis felineus*, which infects cats, are pathologically important foodborne members of the genus *Opisthorchis*; family, Opisthorchiidae; order, Digenea; class, Trematoda; phylum, Platyhelminthes.

Current estimated total number of fish-borne liver fluke infections is 45 million people in Asia and Europe, of which 9 million are infected with *Opisthorchis viverrini*, 1.2 million with *Opisthorchis felineus* and 35 million with *Clonorchis sinensis*. As many as 680 million people worldwide are at risk of infection. Infection hot spots for *O. viverrini* are northeast Thailand, Lao PDR, Cambodia and to a lesser extent, south Vietnam. As many as 67 million people in Southeast Asia are at risk of infection. *Clonorchis sinensis* is found in North Vietnam, China, Taiwan, and Korea, and previously in Japan. To date, *O. felineus* has been reported to occur in all European countries except Great Britain, Ireland, Finland, and Scandinavian. Among the republics of the former USSR, it is present in the Ukraine, Belarus, Baltic countries, Moldova, and Kazakhstan.

**Morphology:** The three liver flukes are morphologically very similar. *Clonorchis sinensis* was isolated into a separate monotypic genus where the main distinctions from the *Opisthorchis* species are the branched shape of its testes (vs. lobed testes in *Opisthorchis*) and the position of testes and vitelline glands. The liver flukes are hermaphroditic, dorso-ventrally flattened, and leaf like in shape. Adult worms of *O. felineus* have a lancet-shaped body narrowed at the front end with a rounded tail. The size of adult flukes measures 5.5–10 x 0.77–1.65 mm with *C. sinensis* largest (10–25 x 3–5 mm) and *O. felineus* smallest (7–12 x 2–3 mm). The body size varies depending on the definitive
The body has two muscular suckers, the oral sucker situated anteriorly and the ventral sucker at the mid-body. All three species of liver fluke eggs have distinct opercular shoulders surrounding the operculum at one end and a small knob or comma shape appendage at the abopercular end. The ovary is smooth-edged or, more rare, weakly lobed, and occurs in front of the testes in the middle of the body. The uterus spans from the ovary to the front edge of the ventral sucker but does not overlap it. The vitelline glands form bunches located in the middle body third behind the ventral sucker. The seminal receptacle is located somewhat behind the ovary. The testes have four or five lobes and are located diagonally one after another. The genital pores appear at the anterior side of the ventral sucker. The egg shell surface is rough and irregular or seen as musk-melon patterns by electron microscopy. Eggs of the three species of liver flukes share similar morphologies and are difficult to differentiate.

3- **Opisthorchis viverrini**, human carcinogenic liver fluke

*O. viverrini* is a prevalent human worms particularly in the far east and south east of Asia, highly prevalent was recorded in Thailand and Laos. Over 100 flukes were recovered from the gallbladder of one patient. It was first described in the post-mortem examination of two prisoners from a jail in Chiangmai, northern Thailand, in 1911 by Leiper. This worm causes a disease called opisthorchiasis. In humans, it may cause cancer.

**Adult worm:** This worm usually reside in the medium-sized or small intrahepatic bile ducts. In heavy infections, adult worms may be found in the gallbladder, the extrahepatic bile duct, and the pancreatic duct. The adult liver flukes are lancet shaped, thin and transparent, length range is 7.5 x 1.5 mm. It has a pair of diagonal, deeply lobed testes, single multi-lobed ovary in front of testes. Vitelline glands look as many follicles located between the ventral sucker and testes.

**The egg:** Yellowish brown, size 27 x 15 μm, oval and thick, it has a special character of opercular shoulder and terminal knob. This egg is mature when released (contains miracidium).
**Life Cycle:** Fish-borne trematodes have a complicated life cycle with two intermediate hosts, starting from a human host, the adult worms deposit fully developed eggs that are passed in the feces. The eggs from adult flukes are passed out with the feces. The eggs must get into water in order to hatch and be able to infect their first intermediate host, a freshwater snail. After being ingested by a suitable snail, the eggs release miracidia which undergo in the snail several developmental stages: sporocysts, rediae, cercariae. The snail intermediate hosts are *Bithynia goniompharus*, cercariae are released from the snail.

Once exposed to appropriate fish intermediate hosts, the free-swimming cercariae shed their tails, penetrate into the tissues or the skin of freshwater fish (Cyclocheilichthys spp.). Metacercariae penetrate subcutaneous adipose and muscle tissues to a depth of no more than 1–2 mm. Several unusual sites for clustering of metacercaria are known, such as pectoral fins, gills, and gut walls. Development of cercaria into metacercaria takes from 3 weeks to 2 months depending on the fish species and water temperature.

Cats, dogs, and various fish-eating mammals including humans are the definitive host. They become infected by ingesting undercooked fish containing infective metacercariae (Koi pla meal in Thailand).
In infected definitive host, the metacercaria excyst in the duodenum and ascend into the biliary ducts, where they attach and develop to adults. Eggs exit the bile ducts and are excreted in the feces after 3 to 4 weeks. The adult worms reside in the biliary system of the mammalian host, where they attach to the mucosa. The life span of *O. viverrini* in human is not known, however, it may be over 25 years as recorded in *C. sinensis*.

**Pathology and pathogenesis of *O. viverrini***:

Pathological changes in liver fluke infection are confined mainly to bile ducts, liver, and gall bladder in both humans and in animal models. The magnitude of the pathology depends on the intensity, duration, and susceptibility of hosts. For light infections, the liver appears grossly normal, whereas, in heavy infections localized dilation of the thickened peripheral bile ducts can be seen on the surface beneath the fibrotic capsule of the liver. Major microscopic changes are confined to the large- and medium-sized bile duct where the
flukes are found. The gross and microscopic characteristics of human opisthorchiasis are well established within 7–15 years after *O. viverrini* infection. Clinical manifestation is infrequent and often nonspecific and related to abdominal discomfort as a result of indigestion. Community based morbidity rates are approximately 5% among infected people.

However, preclinical pathology can be detected by ultrasonography which has revealed various hepatobiliary abnormalities as a result of chronic opisthorchiasis. This chronic infection condition leads to bile duct inflammation and may eventually induce cholangiocarcinoma (CCA). Although pathogenesis of opisthorchiasis is complex, recent studies have revealed that the pathogenetic features in opisthorchiasis are largely a consequence of immunomodulation during the acute and chronic phase of infection. For pathogenesis of CCA, chronic inflammation plays a central role in carcinogenesis through oxidative and nitrative DNA damage which initiates tumorigenesis.

**Opisthorchus and cancer:**
Cholangiocarcinoma accounts for 15% of liver cancers worldwide. In the absence of flukes or endemic infections, development of CCA is quite rare although incidence is increasing in many high income countries for unknown reasons. The northeast of Thailand has the highest CCA incidence in the world. At present, *O. viverrini* and *C. sinensis* have been classified as type 1 carcinogens. A recent study suggested that host genetic background may play a role in the development of CCA and may help explain the high incidence of CCA in northeast Thailand. Additionally, past exposure to infection in terms of elevated *O. viverrini* antibody levels may be a risk factor for CCA development. Liver cirrhosis, chronic infection with the Hepatitis C virus, heavy alcohol consumption, high fluke egg density in stools, obesity, consumption of nitrate-containing foods, history of familial cancer, and gallstones may also be risk factors.
Laboratory diagnosis:

Laboratory diagnosis: The most widely used methods of opisthorchiasis diagnostics are traditionally based on the detection of *O. viverrini* and *O. felineus* eggs in feces and duodenal contents under the microscope. The eggs of the parasite become detectable during the fourth week after infection. For liver pathology, diagnosis of opisthorchiasis utilizes clinical and instrumental tools used to diagnose gastroenterological diseases. The most widespread methods are X-ray diagnostics, ultrasonic examination, and computer tomography. Immunological methods for opisthorchiasis diagnostics are currently available; however, they are nonspecific and lack sensitivity, and they are used as an addition to parasitological methods. Detection of *O. felineus* in fish and snails is also performed by microscopy. In addition, molecular diagnosis by PCR is very sensitive and specific.

Treatment and prevention:
Opisthorchiasis is effectively cured by treatment with praziquantel. Praziquantel treatment eliminates not only *O. viverrini* but also reduces associated morbidities and risk of CCA. Prevention starts with avoiding raw fish and maintain proper cooking. Mass treatment of animal reservoir and by interrupting life cycle of the worm (snail control).

4- *Dicrocoelium dendriticum*, Lancet liver fluke

Common worm of bile passages of sheep and herbivorous and ruminant mammals, and humans, causing dicrocoeliasis. It is widespread in Europe, Asia and Africa, lesser extent in north and South America. *Adult morphology*: adult worms reside in the smaller bile ducts; it is lancet shape, flat, thin and transparent. *D. dendriticum* relatively small, measuring 5 to 15 mm by 1-2 mm and covered with a smooth cuticle. The most conspicuous features of the internal anatomy are the position of the two testes anterior to the ovary in the anterior half of the body, and distribution of the major portion of the long uterine coils in the median field of the posterior half of the body.
**The egg:** eggs are asymmetrical, ovoidal shape with thick shell, dark brown in color, they have a broad convex operculum and measure about 45 micron. The egg usually contains a mature miracidium when released from the definitive host.

**Life cycle:**
Ruminants such as cows and sheep are usually the definitive host, but humans and other herbivorous mammals can also serve as definitive hosts through ingestion of infected ants. Lancet liver fluke has an indirect life cycle with two intermediate hosts, a snail and an ant. The eggs shed by adult flukes reach the host’s gut with the bile and are expelled with the feces. Once outside the host, a terrestrial snail *Cionella* ingests eggs.

Inside the snail, miracidia hatch out of eggs in the gut of the snail and develop into sporocysts, which on their turn multiply asexually, each one producing about 100 daughter sporocysts (no redia stage). Each daughter sporocyst can produce up to 60 cercaria in 3-4 months. Accumulation of cercaria in the snail cause irritation, which results in expelling them in the form of slime balls that adhere to the vegetation, each slime ball, may contain about 100 cercaria.

Silky ants or *Formica fusca* act as the second intermediate host, these ants eat the slime balls on the vegetation. Inside the ants, most cercaria continue
developing to metacercaria that are infective for the final host (cattle, sheep, goats and humans).
Metacercaria remain in the hemocoele of the ant but few of them migrate to the sub-esophageal ganglion of the ant and manipulate its behavior by acting on the nerve cells. Towards the evening, instead of following the rest of the ant colony, the manipulated ants climbs on top of blade of grass and remains there the whole night through until dawn. Final host can then ingest the infected ants during feeding on grass in early morning grazing.
Once the final host ingests the infected ant, the metacercaria are released in the gut of the host and migrate to the liver through the common bile ducts (they do not migrate to the liver tissue as in *Fasciola*). Metacercaria complete the development to adults and start producing eggs in 8-12 weeks. Adult worms feed on bile but not liver tissue.

Pathogenesis of *D. dendriticum*:
Most infections have no symptoms or only slight ones, in case of heavy infections (up to 50,000 flukes in one animal) the bile duct become irritated and distended. Chronic infection can cause blood loss, anemia and end with cirrhosis. However, infection with *Dicrocoelium* is usually less harm than *F. hepatica* and *F. gigantica*. Economic damage is mostly due to condemnation of the livers at slaughter and to reduced productivity of affected livestock.
**Laboratory diagnosis:**
Detection of eggs in the feces or by identification of the flukes after necropsy. However, since the eggs are passed to the intestine only when the gall bladder is emptied, a negative fecal egg count is not conclusive, i.e. there can be false negatives.

**Prevention and control of *Dicrocoelium:***
The most important preventative measure is to keep the snail population as low as possible. The snails that act as intermediate hosts are terrestrial, but need humidity for development and survival. Effective drainage or anything else that keeps the pastures dry will reduce the snail population. Triclabendazole, Netobimin and praziquantel (20 mg/kg) are the drug of choice for infected animals.
Phylum Platyhelminthes, Class Trematoda, Order Digenea

Second: Intestinal Flukes: Flukes reside in the digestive tract of final host.

- *Echinostoma revolutum*, Frolich- 1802.
- *Paramphistomum cervi*, Zeder- 1790, Rumen fluke.

1- *Metagonimus yokogawai* is the smallest fluke that attack human intestine; it is widely spread in the Far East, U.S.S.R., Balkans, Siberia and Europe, it causes metagonimiasis in human.

*Adult Morphology*: The body of the adult worm is often described as leaf-shaped, similar to most trematodes. It is one of the smallest intestinal flukes; the size of the adult fluke does not exceed 2.5 mm length x 0.75 mm width. The ventral sucker is larger than the oral sucker. The most identified feature is that the ventral sucker and genital opening are fused and displaced to the right of the midline of the body. The testes are large and diagonal to each other while the smaller ovary is anterior to the testes and the uterus is filled with eggs. The uterus winds forward to the genital pore and is the largest organ in the body.

*Egg*: it is relatively small measures up to 26.5 micrometer, resembles the egg of *H. heterophyes*.
**Life cycle:** Adults release fully embryonated eggs each with a fully-developed miracidium, and eggs are passed in the host’s feces. After ingestion by the snail *Semisulcospira* (first intermediate host), the eggs hatch and release miracidia which penetrate the snail’s intestine. The miracidia undergo several developmental stages in the snail, sporocysts, rediae, and cercariae. Many cercariae are produced from each redia. The cercariae are released from the snail and encyst as metacercariae in the tissues of a suitable fresh brackish water fish (second intermediate host). The definitive host becomes infected by ingesting undercooked or salted fish containing metacercariae. After ingestion, the metacercariae excyst and attach to the mucosa of the small intestine and mature to adults. This worm can infect fish eating mammals (cats and dogs), humans and birds.

**Pathogenicity:** light infection usually asymptomatic. In heavy infections, abdominal pain and discomfort appear, patients often experience a chronic mucous diarrhea and eosinophilia. The eggs have the ability to escape into the lymphatic venules via intestinal wall penetration and to migrate to other areas of the body, such as the heart or brain; this will lead to granulomatous tissue in these areas.  
*Prevention and treatment:* Avoidance of consuming undercooked fish. Diagnosis by eggs in feces. The treatment of choice is praziquantel.
2- *Echinostoma revolutum*: worms found in America, Europe and Asia causes echinostomiasis in aquatic birds, carnivores and humans. This worm is much longer than wide, it measures an average of 6-8 mm long by 2 mm width, long is three times the width. The morphological characteristic feature is the cephalic collar of spines, the number of spines depends on the species,

*Eggs*: Eggs size varies 80-135 µm long by 55-80 µm wide. They have an unclear operculum and the abopercular end is often thickened. The larger eggs are very similar to *Fasciola* and *Fasciolopsis*. Eggs are passed unembryonated in feces.

*Life Cycle*: Unembryonated eggs are passed in feces and develop in the water. The miracidium takes on average 10 days to mature before hatching and penetrating the first intermediate host, the snail *Lymnaea*. The intramolluscan stages include a sporocyst, two generations of rediae, and cercariae. The cercariae leave the snail and encyst as metacercariae after penetrating the second intermediate host, another snail, fish or tadpoles. The definitive host becomes infected after eating infected second intermediate hosts. Metacercariae excyst in the duodenum and adults reside in the small intestine.
Pathogenicity: Catarrhal inflammation (increasing blood flow to the mucosa and edema) often occurs due to the penetration of the sharp-spined collar into the intestinal mucosa. In addition, nausea, vomiting, diarrhea, fever and abdominal pain may occur.

3- Paramphistomum cervi: The livestock rumen fluke is found in mostly tropical and subtropical regions, including Australia, Asia, Africa, Eastern Europe, and Russia. Adult Paramphistomum cervi live in the rumens of ruminants (goats, sheep, cattle, and buffaloes), they live in the rumen and cause paramphistomiasis or amphistomiasis.

Adult Morphology: Rumen flukes are small, not longer than 15 mm and 5 cm wide. The have a grayish to reddish color. Unlike many fluke species, their body is not flattened but ventrally concave pear-shaped, with the head at the narrowest anterior end. They have two suckers, an oral and a ventral one, the ventral sucker is much larger and close to the posterior end, tests are lobed and located anterior to the ovary.

Eggs: operculated (130x80 micrometers), very similar to those of Fasciola hepatica but slightly lighter, they are unembryonated when released.
Life cycle: adult flukes in the intestine lay eggs that are shed outside with the feces. About 2 weeks later miracidia hatch out of the eggs. They swim in the water until they find a suitable snail. They penetrate into the snail and continue development to sporocysts and mother rediae, which multiply asexually and produce daughter rediae. Each redia produces several cercariae. Cercariae swim around and attach to the vegetation where they encyst and become metacercariae, encysted metacercariae do not survive in dryness, but can survive and remain infective for up to 1 year in a humid and temperate environment. Livestock ingests metacercariae while grazing in contaminated pastures. Once in the small intestine the young flukes leave the cysts, attach to the intestinal mucosa and continue development. They feed on the tissues of the gut wall. Later on, they detach from the gut's wall and migrate to the rumen, where they complete development to adult flukes.
Pathogenicity: Paramphistomiasis causes enteritis and anaemia in livestocks mammals and result in economic losses. Adults attach to the villi in the rumens of the hosts and sap nutrients from the intestine. Pathological symptoms are produced by immature flukes, when the young flukes start to gather in the intestine, there is a watery and fetid diarrhea which is often associated with high mortality (even up to 80-90%) in ruminants, they attack the duodenal mucosa to induce acute enteritis. Surprisingly, the adult flukes are regarded as commensals and non-pathogenic. However, they do cause the intestinal villi to erode and instill inflammation. Paramphistomiasis is responsible for severe economic losses to milk, meat and wool production since the flukes take nutrients from their hosts, which leads to weight loss and physiological decline.

Diagnosis and treatment: Symptoms are usually visible on the behavior of the host. Infected sheep and cattle become severely anorexic or digest food inefficiently. Continuous diarrhea is an obvious indication of heavy infection in the digestive system, the fluid faeces are examined to identify immature flukes. Treatments with reported success are resorantel, oxyclozanide, clorsulon, ivermectin and the combination of bithional and levamisole.

Third: Lung flukes

- Paragonimus westermani, Oriental lung fluke- Kerbert- 1878.

P. westermani is distributed in many parts of the world including Asia, Africa, India and South America.

Adult morphology: The typical shape is oval, red- to brown-colored adult of P. westermani measures 1 by 0.7 cm. The cuticle of possesses spines, ceca extend to the end of the body in a zig-zag manner to. This worm resides in lungs and causes lung infection called paragonimiasis or distomiasis.

Eggs: The average egg of ranges in size from 78 to 120 μm by 45 to 60 μm. They are oval and consists of an undeveloped miracidium protected by a thin smooth shell. An opercular rim (shoulders) surrounds the prominent operculum.
Life Cycle: *P. westermani* is transmitted in undercooked crayfish or crabs. On ingestion of such freshwater products, the immature flukes are released into the body, where they migrate through the intestinal wall, through the peritoneal cavity, into the diaphragm, and finally into lung tissue, where encystation occurs. Migration of immature flukes to other areas of the body, such as the brain or liver may take place.

Clinical symptoms: Paragonimiasis: Pulmonary Distomiasis. As the common name oriental lung fluke suggests, patients infected with *Paragonimus* typically experience symptoms associated with pulmonary discomfort, cough, fever, chest pain, and increased production of blood-tinged sputum. Individuals infected with this parasite may also experience chronic bronchitis, eosinophilia, and the production of fibrous tissue. These symptoms often mimic those seen in persons infected with tuberculosis. Patients who develop infections in areas other than the lung experience symptoms corresponding to the affected organ or tissue. One area is the brain, Cerebral Paragonimiasis. Migration of immature *P. westermani* reaching the brain may result in the development of a serious neurologic condition. Patients experience seizures, visual difficulties, and decreased precision of motor skills.

Laboratory Diagnosis: Diagnosis of *P. westermani* is accomplished by the recovery of eggs in sputum specimens. These eggs are commonly found in bloods samples. Occasionally, the eggs may be seen in stool samples when sputum is swallowed.
**Fourth: Blood flukes or Schistosomes**

*Schistosoma* spp., commonly known as blood-flukes and bilharzia, includes flatworms which are responsible for the most significant parasitic infection of humans by causing the disease schistosomiasis, and are considered by the World Health Organization as the second most important parasitic disease, next only to malaria, with hundreds of millions infected worldwide. Schistosomiasis often is a chronic illness that can damage internal organs and, in children, impair growth and cognitive development.

Schistosoma genus is the only trematodes that have separate sexes (diecious), typical female measures 2 cm in length and the male measures 1.5 cm, the male surrounds the female almost completely in a groove called gynicophoric groove or canal, facilitating copulation. *Schistosoma* cercaria with forked tail.

**Cercarial dermatitis** (swimmer’s itch or duck lice): Erythematous papules on the exposed areas of a swimmer. It is a skin rash caused by an allergic reaction to an infection with certain cercaria of Avian *Schistosoma* (*Austrobilharzia variglandis*). Cercaria released from infected snails who swim in fresh and salt water. Symptoms include burning, tingling, and itching of the infected skin. The pimples may develop into small blisters. Itching may last up to a week or more but will gradually go away.

Life cycle of *Schistotoma* spp.
<table>
<thead>
<tr>
<th>Parameter</th>
<th><em>S. haematobium</em></th>
<th><em>S. mansoni</em></th>
<th><em>S. japonicum</em></th>
</tr>
</thead>
<tbody>
<tr>
<td>Bilharziasis type</td>
<td>Urinary</td>
<td>Intestinal</td>
<td>Intestinal</td>
</tr>
<tr>
<td>Definitive host</td>
<td>Human</td>
<td>Human and rodents</td>
<td>Human and animals</td>
</tr>
<tr>
<td>Intermediate host</td>
<td><em>Bulinus</em></td>
<td><em>Biomphalaria</em></td>
<td><em>Oncomelania</em></td>
</tr>
<tr>
<td>Adult location</td>
<td>Urinary and vesicle veins, pelvic plexus</td>
<td>Inferior mesenteric veins and hemorrhoid plexus</td>
<td>Superior and gastric mesenteric veins</td>
</tr>
<tr>
<td>Ova spine</td>
<td>Terminal spine</td>
<td>Lateral spine</td>
<td>Small lateral knob</td>
</tr>
<tr>
<td>Ova excretion</td>
<td>With urine</td>
<td>With stool</td>
<td>With stool</td>
</tr>
<tr>
<td>Male tegument</td>
<td>Fine papillae</td>
<td>Coarse papillae</td>
<td>Smooth</td>
</tr>
<tr>
<td>Testes</td>
<td>4-5 large</td>
<td>6-9 small</td>
<td>6-8 small</td>
</tr>
<tr>
<td>Ceca union</td>
<td>2&lt;sup&gt;nd&lt;/sup&gt; half of body</td>
<td>1&lt;sup&gt;st&lt;/sup&gt; third of the body</td>
<td>Posterior third of the body</td>
</tr>
<tr>
<td>Female ovary location</td>
<td>2&lt;sup&gt;nd&lt;/sup&gt; half of the body</td>
<td>1&lt;sup&gt;st&lt;/sup&gt; half of the body</td>
<td>Middle of the body</td>
</tr>
<tr>
<td>Uterus</td>
<td>Long with 20-30 ova</td>
<td>Short with 1-4 ova</td>
<td>Long with 50-100 ova</td>
</tr>
</tbody>
</table>
Phylum Platyhelminthes, Class: Cestoda (tapeworms)

Cestodes or tapeworms are the most specialized of the Platyhelminthes parasites. All cestodes have at least one, and sometimes more than one intermediate host (invertebrates) as well as definitive host (vertebrates). In some cases, both hosts are vertebrates, as in the common Beef Tapeworm *Taenia saginata*, and in a few species there may be only a single host. Tapeworms infect human and animals causing mild to serious infections. There are more than 1,000 species of tapeworms.

The mature Cestode always live in the hosts intestines where it can obtain all its food. Cestodes have evolved to have no digestive system of their own, they simply absorb nutrients from their hosts guts. To aid this process the entire surface of their body is covered with microscopic projections, which greatly increase the surface area available for the absorption of nutrients. They have no need to travel and therefore have no locomotor organs (cilia). Their excretory and nervous systems are similar to other. Typical adult tapeworm consists of a knoblike head, or scolex, equipped with hooks for attaching to the intestinal wall of the final host, a neck region (germinative), and a series of flat, rectangular body segments (strobila), generated by the neck. The chain of proglottids ranges from a few number to more than 1000 segments.

1- **Subclass: Cestodaria**

The subclass Cestodaria consists of monozoic (unsegmented) tapeworms, with a single set of reproductive organs. No scolex is present and the shelled embryo contains ten hooks. All are parasites of fish.

2- **Subclass: Eucestoda**

The true tapeworms are usually polyzoic (segmented), comprised of a scolex which functions as a holdfast and a strobila that is divided into many proglottids. The scolex is the anterior end of the tapeworm and can be equipped with a variety of attachment structures. The scolex may bear suckers, grooves, hooks, spines or any 2 combination of any. In the strobila, each proglottid potentially contains one or more sets of both male and female reproductive organs, making the tapeworms hermaphroditic. Proglottids are
added on in the “neck” region (by a process called strobillation) between the scolex and strobila, and mature as they move down the body. The male system develops first and anterior proglottids will often contain only male organs. Older proglottids will contain both reproductive systems.

Ova may be released through the uterine pore or the entire gravid proglottid may break off (apolysis) releasing eggs as it breaks up. True tapeworms usually produce a hexacanth embryo (6-hooked). This subclass has 2 orders of tapeworms: Pseudophyllidae and Cyclophyllidea.

**a- Order: Pseudophyllidae**

Adult pseudophyllidean tapeworms are parasites of all classes of vertebrates, but fish are their primary host. The life cycle involves a procercoid (larval stage found in copepods) and plerocercoid larval stage (found in fish). Tapeworms in this order are characterized by:

1- A bothriate scolex: possessing a dorsal and a ventral groove or bothrium.
2- A neck may be present or absent.
3- Testes and vitellaria are follicular and scattered throughout the proglottid.
4- The ovary is bilobed and posterior.
5- Genital pores are usually ventral and may be lateral or dorsal in some groups.
6- A uterine pore is present on the dorsal or ventral surface.
7- The eggs are usually operculate and contain a ciliated embryo, the coracidium.

*Diphyllobothrium latum*: Commonly called the broad fish tapeworm and causes diphyllobothriasis. This cestode is common in fish-eating carnivores in northern Europe, Russia, the Arctic, and the Great Lakes areas of North America. There appears to be little host specificity for adults have been recovered from the intestine of many canines, felines, pinnipeds, bears, and humans. In humans *D. latum* can be a serious pathogen, causing a pernicious anemia. This is due to the worm absorbing large amounts of vitamin B₁₂.

*Morphology*: The adult worm reaches a length of 10m or more and may contain up to 3000 proglottids. The scolex of *D. latum* is elongate, spoon-shaped, and has two long sucking grooves, one on the dorsal and the other on the ventral surface. The mature and gravid proglottids are wider than long,
with the main reproductive structures (mainly the uterus) located in the center of the gravid proglottid. Both eggs and proglottids may be found in the stool, all proglottids are wider than they long and the gential pore open mid-ventrally.

**Eggs:** about 40 x 60 µm in size. Broadly oval and operculated, there is a small nub on the abuperculated end, unembryonated when released. Ova are shed up to a million in one days.

**Life cycle:** After developing for 2 weeks in fresh water, the eggs hatch and the ciliated, coracidium larvae are ingested by the first intermediate host, the copepod (crustacean). Fish then ingest the copepods, containing the second larval stage (procercoid), the 2nd intermediate host fish contains many plerocercoid larvae which initiate the infection with the adult worm when ingested by human.

**Pathogenicity:** Most individuals infected with a fish tapeworm are asymptomatic. However, digestive disturbances including abdominal pain and cramps accompanied by weight loss may occur, but healthy individuals may experience few symptoms. Anemia, or pernicious anemia in heavy infection, follows due to a vitamin B₁₂ deficiency, due to the presence of the parasites infecting the intestine. Diagnosis by ova in stool.
Sparganosis: Helminthic disease caused by *Spirometra mansonides* (or *Dipyllobothrium mansonides*) which infects cats and dogs. It is caused by the plerocercoid of this worm when reaching the human, accidentally (not the final host) so the human serves as 2\textsuperscript{nd} or paratenic intermediate host. Spargana (plerocercoid) can migrate to many parts of the body, including subcutaneous tissue, breast, orbit, urinary tract, pleural cavity, lung, abdominal viscera and brain.

b- **Cyclophyllidea:** Members of this order are common parasites in the intestines of amphibians, reptiles, birds and mammals. They are characterized by:

1- The scolex usually contains four cup-shaped suckers.
2- The rostellum usually present or absent, and may or may not be armed.
3- Uterine pore is absent so gravid proglottids developed.
4- Genital pore is lateral to the proglottid.
5- Vitelline gland is single, compact, and usually posterior to the ovary.
6- Proglottids are often wider than long.

Gravid segments generally leave the host’s body individually, but may also be released in groups. Non-operculated eggs escape by rupture or disintegration of gravid proglottids and develop into some form of bladderworm in an invertebrate or vertebrate intermediate host. Infective eggs contain an oncosphere larva that bears 6 hooks.

They have a variety of intermediate host types, both invertebrate and vertebrate. Depending on the cestode species, the larval cestodiasis stage in the vertebrate intermediate host may be as cysticercus, strobilocercus, coenurus (multiceps), or hydatid cyst.

- **Cysticercus** has a bladder-like form with an invaginated scolex located at one end.

- **Strobilocercus** is similar to the adult worm, in which a scolex is visible, but has a terminal bladder end rather than proglottids.
- **Coenerus** is similar to the cysticercus but bears multiple scoleces.

- **Hydatid cyst**, brood pouches develop within the bladder and gives rise to multiple scolices (up to 20) and each hydatid cyst may contain thousands of scolices.

Larvae in invertebrate intermediate hosts (usually arthropods) are cysticercoids; these have no bladder but a simple invaginated scolex. The scolex has 4 prominent suckers and a terminal rostellum, which may or may not be armed with hooks.

**Genus Taenia spp.**

There are several species of *Taenia* that humans are likely to encounter. These include two species for which humans serve as the definitive host: *Taenia saginata*, the beef tapeworm; and *T. solium*, the pork tapeworm. Several species of *Taenia* also infect dogs and cats (e.g., *T. pisiformis*), and humans are likely to encounter these when they note the presence of these tapeworms' proglottids in their pets' feces.

All species of *Taenia* have similar life cycles. The adult tapeworm lives in the definitive host's small intestine. Proglottids, which contain eggs, break off the posterior end of the tapeworm, and these proglottids are either passed intact in the host's feces or they dissolve in the host's intestine and eggs are passed in the feces. When a suitable intermediate host ingests the eggs, the oncosphere larva is released and, with the aid of the embryonic hooks, penetrates the intestinal wall and enters the bloodstream. Upon reaching the liver, the oncosphere begins to develop into a cysticercus. Bladder-worms break out of the liver and attach to the mesenteries throughout the abdominal cavity. The definitive host is infected when it eats an intermediate host infected with cysticerci. Upon ingestion, the scolex evaginates, attaches to the intestinal lining, the bladder disintegrates, and the strobila is formed by the budding of the neck region.
As adults in the definitive host's small intestine, tapeworms rarely cause problems; in exceptional cases, the tapeworms might physically block the intestinal tract, due to their large size, or proglottids might become lodged in the appendix and result in appendicitis. The proglottids of *Taenia* are large and muscular. Occasionally single proglottids or long chains of proglottids might crawl out of the anus of an infected human. Larval stage of *T. solium* is *Cysticercus cellulosae*, which is the infective stage to humans, *T. saginata* infective stage to humans is *Cysticercus bovis*.

**Cysticercosis:** It is a systemic infection caused by dissemination of the larval form of the pork tapeworm, *Taenia solium*. A high prevalence has been reported from the developing countries because of the co-existence of poor sanitary conditions and domestic pig raising without proper veterinary control or surveillance systems. It occurs mainly in pork eating nations due to consumption of undercooked pork. Humans are the definitive hosts and carry the infective *Cysticercus* and intestinal adult tapeworm. Human cysticercosis occurs when *eggs* are ingested via faecal-oral transmission from a tapeworm host. The human then becomes an accidental intermediate or paratenic host, with development of cysticercosis within organs. Cysticercosis can affect various organs, such as brain, spinal cord, muscles, orbit, subcutaneous tissues and heart. The clinical manifestation of the patient varies depending upon the
site of larval encystment, number of cyst and the extent of associated inflammatory responses. CT scan and MRI are used for diagnosis.

*Taenia multiceps*: tapeworm of the small intestine of dogs and other canines, has a larva called coenurus, which develops in the brain of sheep, causing the dizzy, and exceptionally in the human brain then determining the coenurus.

*Cerebral coenurosis*: it is a parasitic infection caused by larvae of the tapeworm *T. multiceps*; it develops in the intermediate host and rarely infect humans. Cerebral coenurosis cysts cause ventricular obstruction and increased intracranial pressure. On CT scan, viable cysts appear as lesions.

**Treatment**: Praziquantel opens membrane calcium channels causing paralysis of the worm, aiding the body in expelling the parasite through peristalsis. Niclosamide, used to treat many different kinds of infections with trematodes and adult tapeworms, is quite effective.

**Echinococcosis:**

Echinococcosis is a zoonotic disease caused by Echinococcus spp. tapeworms. The definitive hosts, which include dogs, other canids, hyenas and cats, carry the adult tapeworms subclinically. Dogs are particularly important in zoonotic transmission due to their close relationships with humans. Intermediate hosts are initially asymptomatic; however, the growth of the larvae, which form cysts in vital organs such as the liver and lungs, can lead to illness and death. Echinococcosis is a major public health problem in some countries, approximately 2-3 million human cases are thought to occur worldwide.

Cystic echinococcosis (Hydatid cyst), the most common form of the disease in people and domesticated animals, is caused by *Echinococcus granulosus*. Because the larvae of this organism usually develop as discrete single cysts, it is the least severe and most treatable form. Nevertheless, large or multiple cysts may cause irreversible damage to organs, and the rupture or puncture of the cyst can seed multiple organs with larvae or cause anaphylactic reactions. Humans typically become symptomatic many years after infection. Cystic echinococcosis causes economic losses from the condemnation of internal
organs at meat inspection. In some cases, it may also result in decreased meat and milk production or decreased value of the fleece due to debilitation.

Alveolar echinococcosis, caused by *E. multilocularis*, is less common than cystic echinococcosis, but it is very serious and more difficult to treat. The larvae of this organism grow as multiple, budding cysts, which can infiltrate entire organs and disseminate to distant sites including the brain. As well as affecting people, alveolar echinococcosis is reported to cause serious disease in animal intermediate hosts including dogs. The occurrence of this organism in a wildlife cycle between foxes and small mammals makes it difficult to prevent. Polycystic echinococcosis, which is usually caused by *Echinococcus vogeli* in humans, is similar to alveolar echinococcosis in the growth of the larvae and its presence in wildlife hosts.

*Echinococcus granulosus* or hydatid worm:

**Morphology**: Tape-worms form three different developmental stages: eggs; larvae; and adults. Adult *E. granulosus* worms are small (2-6 mm long) and have a scolex with only three attached segments. The scolex has four lateral suckers and the rostellum is non-retractable and armed with a double crown of 28-50 recurved hooks. The anterior segment is immature, the middle segment is mature with functional testes and ovaries, and the posterior segment is gravid with the uterus filled with eggs. The eggs are typical for most taeniid species and are small and round (30-43µm in diameter), thick-shelled and contain a hexacanth (6-hooked) embryo (oncosphere). The encysted larval (metacestode) stage is known as a bladder-worm or hydatid, and it produces multiple infective stages (protoscoleces, apparent as invaginated scolices already containing suckers and hooks) either directly from the germinal layer of the cyst wall, or by forming brood sacs (hydatid sand) by endogenous (internal) or exogenous (external) budding of the germinal layer.
Types of hydatid cysts:

1- *E. granulosus* forms fluid-filled unilocular cysts with endogenous budding of brood capsules, fluid-filled sphere with germinal membrane proliferating endogenously to form brood capsules.

2- *E. vogeli* forms fluid-filled polycystic cysts with exogenous budding, fluid-filled with germinal membrane budding exogenously to form new cysts and endogenously to form septae.

3- *E. multilocularis* forms fluid-free multilocular or alveolar cysts with exogenous budding, germinal membrane budding exogenously to form multiple cysts with no free fluid.

Life Cycle: The definitive hosts for *E. granulosus* (canids, felids, and hyaenids) become infected when they ingest cysts (metacestodes) in the tissues of the intermediate hosts. Feeding the viscera of intermediate hosts to dogs perpetuates cycles in domesticated animals. The cysts develop into tapeworms, which mature in the host’s small intestine. Gravid proglottids or eggs are shed in the feces, and are immediately infective. *Echinococcus* eggs
have a sticky coat that will adhere to an animal’s fur and other objects. Insects such as flies and beetles, or birds, can also act as mechanical vectors.

Under ideal conditions, *E. granulosus* eggs remain viable for several weeks or months in pastures or gardens, and on fomites. The eggs survive for only short periods of time if they are exposed to direct sunlight and dry conditions. The intermediate hosts include a large number of domesticated and wild animals, particularly herbivores. Humans can also be infected. If an intermediate host ingests the eggs, the larvae are released, penetrate the intestinal wall, and are carried in blood or lymph to the target organs.

Parasites can develop into cysts in many different organs, but they are found most often in the liver and, less frequently, the lungs. The rate of development varies with the intermediate host and species of parasite, but the cysts usually grow slowly. Their diameter generally increases from less than 1 cm to 5 cm each year. Some cysts may persist unchanged for years. Most *E. granulosus* cysts are 1-7 cm in diameter when they are discovered, but some may eventually reach 20 cm. Each fluid-filled cyst is surrounded by a fibrous wall from the host and contains two walls derived from the parasite: an outer laminated membrane and an inner membrane called the germinal layer. Brood capsules develop from the germinal membrane. Each brood capsule contains one to several invaginated heads (protoscolices) that can develop into adult tapeworms if they are ingested by the definitive host. Capsules and protoscolices either float freely in the hydatid fluid or adhere to the wall with a peduncle; the capsules and protoscolices that float freely are known as “hydatid sand.” If a cyst ruptures, the hydatid sand can develop into new cysts. Some cysts are sterile; they either never produce brood capsules, or they become sterile after bacterial infection or calcification.
**Pathogenesis:** The adult stages are considered benign and do not cause disease in dogs, as the worms do not invade or feed on host tissues. Encysted larval stages generally do not cause clinical disease in domestic livestock as they are often confined to visceral tissues.

The symptoms of echinococcosis depend on the size, number and the location of the metacestodes. Until the cysts become large enough to damage adjacent tissues and organs, they are usually asymptomatic. The clinical signs are those of a mass lesion. Significant pathological changes occur in humans, or other intermediate hosts, when the slowly growing cysts put pressure on surrounding tissues and produce chronic space-occupying lesions. Cysts may grow around 1 mm per month and can become extremely large, up to 30 cm in diameter with liters of fluid containing thousands of protoscoleces. Organ enlargement may be accompanied by a variety of clinical signs depending on the size and location of the cysts. Compression of liver may result in jaundice, portal hypertension and abdominal distention. Cysts in the lung may cause haemoptysis (coughing up blood), dyspnoea (difficulty breathing) and chest pain. Cysts in the brain or spinal cord can provoke acute inflammatory responses and numerous neurological sequelae, including epilepsy and blindness. Cyst rupture has been associated with acute clinical signs (such as
peritonitis and pneumothorax), and the sudden release of hydatid fluid may cause severe allergic reactions (such as asthma and anaphylactic shock). Protoscolices released from ruptured cysts can regress and form new hydatid cysts throughout the body.

**Differential diagnosis:** Infections in dogs may be diagnosed by the detection of eggs, and occasionally worms, in faecal samples. Infections in intermediate hosts are diagnosed well after the larvae have encysted. Clinical symptoms of a slow-growing tumour accompanied by eosinophilia. Cysts can be visualized by computerized axial tomography (CAT) scans, X-rays or ultrasound.

**Hymenolepis nana** and *H. diminuta*:

*Hymenolepsis nana* is called the dwarf tapeworm and is the smallest tapeworm known to infect humans. The *H. nana* species is parasitic for both birds and mammals such as mice and rats. Rodents are known to harbor the organism and are capable of transmitting the disease, although no intermediate host is required during its life cycle. *Hymenolepsis diminuta* resembles that of *H. nana*. Hymenolepis worms are often found in rat intestines. Because the organism is common in warmer climates, infections are common in the southeastern United States. The intermediate host is the tribolium beetle, particularly those who consume rat feces.

<table>
<thead>
<tr>
<th><strong>H. nana eggs</strong></th>
<th><strong>H. diminuta egg</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Spherical transparent</td>
<td>spherical and yellow-transparent</td>
</tr>
<tr>
<td>30-47 µm in diameter</td>
<td>70-85 µm in diameter (larger)</td>
</tr>
<tr>
<td>Hyaline shell is thin</td>
<td>Hyaline shell are thick</td>
</tr>
<tr>
<td>Contain a 6-hooked oncosphere.</td>
<td>Same</td>
</tr>
<tr>
<td>Polar filaments (4-8) which are found between the developing embryo and the egg shell.</td>
<td>Polar filaments are absent.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>H. nana scolex</strong></th>
<th><strong>H. diminuta scolex</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Has 4 suckers with armed rostellum with hooks.</td>
<td>Has 4 suckers with but unarmed rostellum (no hooks)</td>
</tr>
</tbody>
</table>
**Life cycle:** Ingestion of *H. nana* eggs is the most common manner in which the infection is contracted. Larvae most often develop in beetles and fleas where grains are contaminated by rat feces, although no intermediate host is required in the life cycle of *H. nana*. The cysticercoid larvae mature in the small intestine where the scolex attaches itself to the intestinal mucosa; there the adult tapeworm thrives and reproduces. Gravid proglottids are passed from the body following disintegration and the infective eggs may contaminate foodstuffs and work areas. The infective eggs may then be ingested by animals, including humans, which develop into cysticercoid larvae and the infectious cycle begins. It is possible for the eggs to hatch in the intestine and in this case, cysticercoid larvae develop into adult forms without leaving the intestine of the host, resulting in a process called autoinfection.

**Hymenolepiasis:** Mild gastrointestinal distress may be encountered but the infected individuals are primarily asymptomatic. Some may experience mild diarrhea, weight loss, and abdominal cramps and mild pain. *H. nana* infections can grow worse over time because, unlike in most tapeworms, eggs of this species can hatch and develop without ever leaving the definitive host.

*Thanks you.................Dr. Hayder Z. Ali*