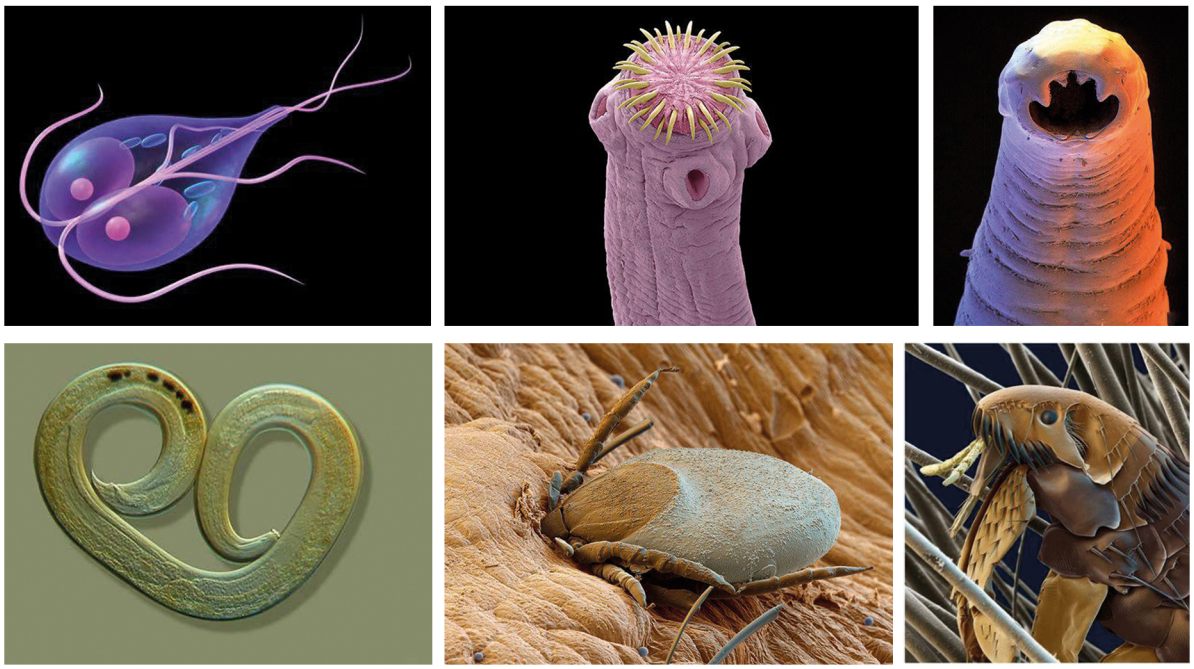
Basrah University
College of Veterinary Medicine
Department of Veterinary Microbiology
and Parasitology

ASSISTANTIN PRACTICAL VETIERNARY PARASITOLOGY

FOR THE THIRD STAGE STUDENTS



Second Edition

By

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Collection, Diagnosis, Preservation of Samples

Introduction:

Examination for parasites should always be performed and examination for many samples, like, feaces, blood, urine, nasopharyngeal, genital tract.

How to collect the feacal samples?

1-should collect in clean wide mouthed vials

2-shouldn't contaminated with anything (urine, water, grass, -----)

3-should handled carefully

4-numbered vials with all information with time, type, date

5- different liquids as preservation for any time examination

Condition for examiner

1-the examiner must with self cure, mask, gloves, special dress

2-have a good tools and instruments, like, microscope, glasses, centrifuge

3-the place must clean with water sources

Shipment of samples:

Sometime need for more diagnosis and need to packages to another city or country, so, it must be prepared for mailing as a regulated method.

Types of Examination

1-Physical Description

It is important to determine the:

1-Consistency of the feaces (formed, semi formed, soft or liquid)

2-With or without blood or mucus or any foreign materials

3-Color of the samples

2-Chemical Description

Consist of Macroscopic and Microscopic Examination

1- Macroscopic Examination:

All the available information regarding quantity, form, consistency, colour, reaction and presence of blood /mucus in faeces should be noted. For this, 15-20 gm of faeces is spread in a large Petri dish. Tapeworm segments / nematodes / immature trematodes are searched with the camel hairbrush. The recovered parasites stages are identified with or without staining.

2- Microscopic Examination:

First: Qualitative examination

a. Direct smear examination:

a. Comminute 5-6 faecal balls or 2-4 gm of faecal with pestle and mortar.

b. Transfer a loop-full of the material to a slide with the matchstick or a toothpick, and a drop of water to form a uniform suspension.

c. Spread it on the slide and apply a cover slip.

d. Examine the slide microscopically under low power.

e. Use high power in case of doubt.

f. This method is successful in heavy infections.

b. Concentration Methods:

the basis of these method is the differences in specific gravity. They are more sensitive than direct smears. It includes the following techniques:

1. Sedimentation Techniques:

this technique is useful in demonstrating majority of the trematoda eggs and can be performed with / without centrifugation as below:

A. Emulsify 2-5 gm of faecal sample in a pestle and mortar with sufficient quantity of water.

B. Strain the material through a sieve (40 mesh per sq. inch) and pour it into a plastic container of 30 ml capacity.

C. Fill the tube up to the brim with water.

D. Keep it for 15-20 min. or centrifuge it at 1000 r.p.m. for 2-5 minutes.

E. Pour off 2/3 rd supernatant solution.

F. Fill the tube again up to the brim and repeat the above process till supernatant is clear.

G. Throw off the supernatant layer.

H. A loop-full of sedimentation with or without centrifugation is kept on a slide, it is covered with a cover slip and examined under low and high power of microscope.

2.Floatation Technique:

this technique is useful in demonstration of strongyles, *Strongyloides*, Ascarids, oxyurids, *Trichuris* and cestode egg. For this purpose, some of the saturated salt solutions like copper nitrate, sodium nitrate, zinc sulphate, magnesium sulphate, sodium chloride and sugar have been tried. Of these saturated solutions, magnesium sulphate is of general utility. The followed procedure is:

a. Get a flat-bottom glass tube of about 7.5 cm length and of 2.5 cm internal diameter.

b. Obtain 5-6 faecal balls or about 4 gm of faeces and comminute it in pestle and mortar.

c. To avoid debris, one or two washings are done with tap water in presence/absence of a centrifugal machine.

d. Add about 15 ml of saturated magnesium sulphate solution in the glass tube. Add more salt solution and fill up to the brim.

e. Keep a slide on the top and leave it undisturbed for about 25 min.

f. Remove the slide in a horizontal position, turn it quickly, apply a cover slip and examine microscopically. The examination must be conducted between 25-35 min. after preparation otherwise the ova/oocyst may start distorting to osmosis.

Second: Quantitative Examination

It is done to obtain the accurate information regarding the severity of infection. Eggs per gram (EPG) of faeces are determined by the following technique:

1. Modified Mc Master technique.
2. Feaces egg counting method.
3. Boray and Pearson technique.

3. Coproculture:

The eggs of some worms species may not or may hardly be differentiated, faeces are cultured to obtained L3 of nematodes, so it is useful in identification of specific nematode problems. for example, various types of *Trichonema* in horses can only be identify after coproculture and in cattle or sheep the trichostrongyloids can be differentiated after the larvae have been incubated.

a. Charcoal Culture:(Figure 1)

1. Comminute 20-40 gm faecal material in sufficient volume to make a thick suspension.
2. Mix sufficient quantity of charcoal powder in it.
3. Fill this mixture into a small wide mouthed bottle loosely up to 2/3 rd level.
4. Cover with loose lid and incubate at 27 C^{o} for 5-7 days.
5. Maintain sufficient moisture in the faecal material.
6. Fill the bottle with water up to the brim and then invert it in a glass Petri dish lid to cover infective larvae.

7. Put a layer of water (1.0 cm) around the bottle in the lid and keep this assembly undisturbed for 1-2 h.

8. Collect the layer around bottle.

9. For species identification transfer the larvae in 70% alcohol or 10% simmering formalin and examine.

Figure (1): Charcoal culture

b. Filter paper strip culture:

This method used with dogs and cats (method of Harada and Mori for *Ancylostoma* and *Uncinaria*. And it performed as following:(Figure 2)

1. Add 3-4 ml distilled water in a centrifuge tube of 15 ml capacity.
2. Smear approximately 0.5-1.0 gm faeces in a thin film to the centre of each filter strip.
3. Insert the strip in the centrifuge tube and incubate it at 27C^{o} for 8-10 days.
4. Check the tube daily for the original level of water and if required add more water.

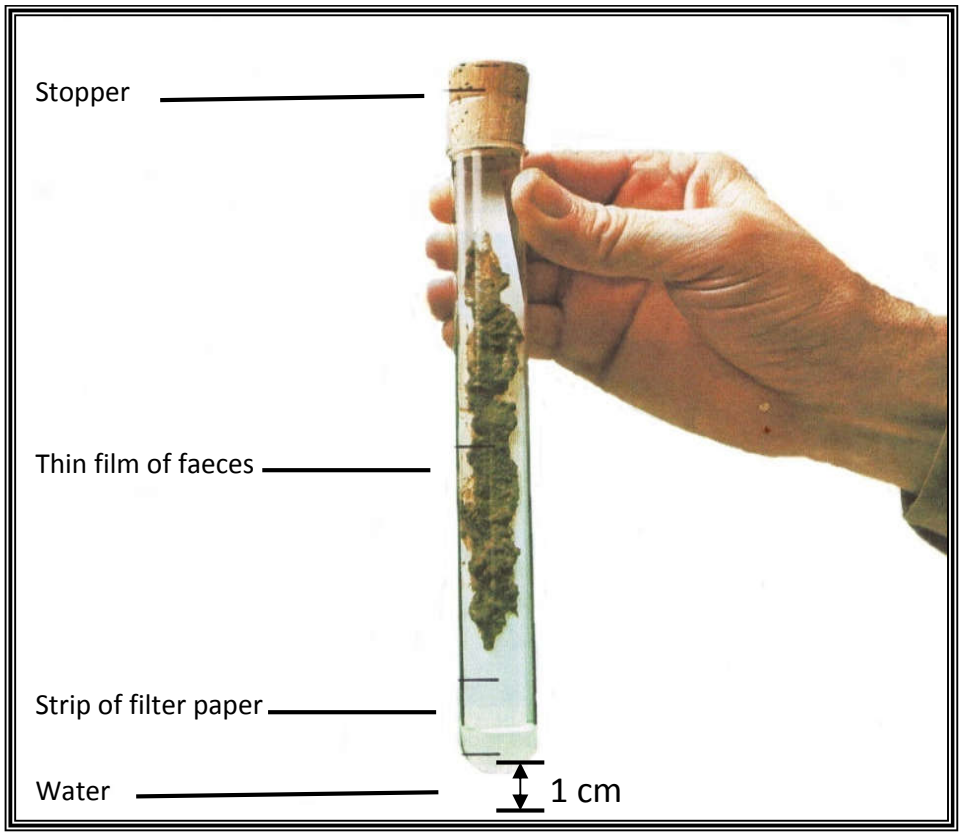


Figure (2): Filter paper strip culture

c. Petri Dish Culture:

This method used for cattle and pigs faeces and it performed as following: (Figure 3)

1-The faeces are mixed with charcoal or sphagnum in a 1: 1 proportion.

2-The mixture placed on the filter paper, in a Petri dish.

3-The Petri dish is covered and placed in an incubator at 25-27 C^{o} for 7 days (depend on the embryonating period of the worm's eggs) .

4-Each day the humidity is checked but the mixture is not shaken.

5-The presence of the hatching larvae on the tops of the sphagnum is checked.

6-When water is added to this mixture and it is placed in a Baermann-apparatus, the larvae can be collected the following day.

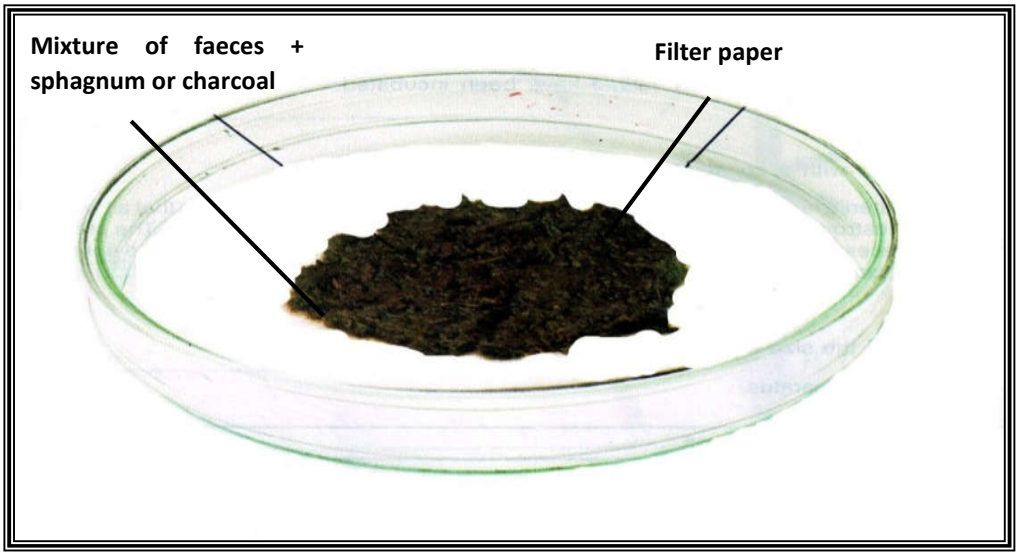


Figure (3): Petri Dish Culture

4. Baermann's Technique:

This method is useful for the examination of faeces / herbage suspected for nematode infective larvae. Active larvae migrate from the faecal material / herbage through the wire gauze and muslin cloth into the water.

Material:

Baermann- apparatus consist of a glass funnel clamped to standard ;to the bottom of the funnel a rubber tubing is fitted (10 cm) ending in a small pipette (dropper) . The rubber tube can be pinched, for instance by means of a clip. The funnel is covered with a fine sieve (for instance, a piece of wire gauze, mashes 0.6-0.7 mm), which

is pushed down a little in the middle, so that it partly hangs in the funnel. The sieve is covered with a double layer of gauze. (Figure 4)

Method:

Apply to the gauze ±20 g of fresh faeces gently fill the funnel with tap water (max. 30C^{o}) so that the faeces are completely immersed . If the faeces are vary watery , more layer of gauze will be used . Let the whole settled at room temperature into the funnel neck , where they are concentrated at the bottom . When the clip is release , the first 3-4 drops (not more) may be collected on microscope slide and ,without cover glass , be examined under low magnification . The nematode will be swimming actively. Instead of collecting the first drops on an object glass 5-10 ml of the liquid may be drained off into a centrifuge tube. The liquid is centrifuged and the supernatant is gently decanted. The larvae will be in the sediment. Even minor infection may be detected by means of the Baermann-apparatus.

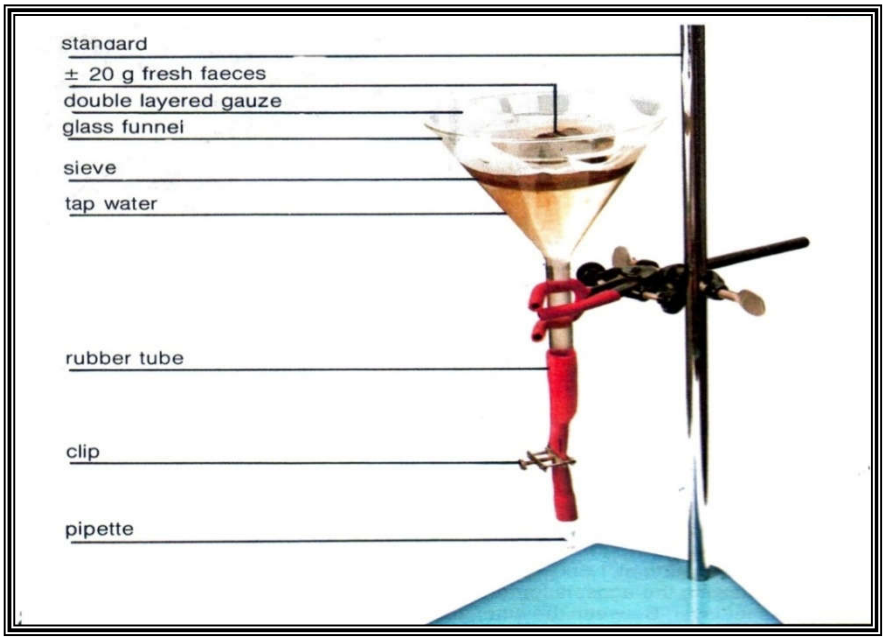


Figure (4): Baermann-apparatus

5. Sporulation of Oocysts:

This means formation of spores or breaking up into spores. The oocyst of coccidian parasite is identified on the basis of number of sporocyst per oocyst and number of sporozoite per sporocyst after sporulation under natural or laboratory conditions. The faecal samples are processed as following:

1. Mix a little quantity of faecal material in 2.5% potassium dichromate or 0.5% chromic solution.

2. Layer this mixture thinly in Petri dish and incubate at about 27 C^{o} for 1-3 days.

3. Examine the oocyst for the formation of sporocyst and sporozoite under microscope.

4. Dispatch the sporulated oocyst in 2.5% potassium dichromate.

Skin Examination

A. Skin scrapings Examination:

Skin scrapings are examined for the recovery of parasitic mites. The scraping must be obtained in a manner that takes into account both the nature of the lesion and location of the mites. These should be taken from moist part near the edge of the lesion a voiding the inclusion of large amount of dry crust, hair or wool. In some cases, if the animal gets furious, it is advised to go local anesthesia.

Procedure:

1. Dip the scalpel blade in glycerin or liquid paraffin.

2. Pick up a fold of animal skin at the edge of the suspected area pinching it firmly between thumb and forefinger.

3. By holding the blade at right angle to the skin, scrub the crust of the fold several times till blood begins to seep through the abrasions. The scraping will adhere to the blade.

4. Boil the material in 10% potassium hydroxide to dissolve the debris/hairs etc.

5. After cooling pour the material into a centrifuge tube and centrifuge for 2 min. at 2000 r.p.m.

6. Examine a drop of the sediment for mites under the microscope.

B. Nasal Scrapings Examination:

This sample usually obtained by a rotatory motion of a spatula in the nasal tract. The fresh nasal discharge collected from the suspected

animals in normal saline solution, then centrifuged at 1500 r.p.m. for 5 min. and the sediment is examined under low power of microscope.

Sputum Examination

Sputum specimens should be a deep sputum rather than primarily saliva, and it should be delivered immediately to the laboratory. Microscopic examination should include saline wet-mount and permanent stain preparation. This sample can detect occasionally, intestinal parasites such as larvae of *Ascaris* species, *Strongyloides* species and hookworm; cestode hooklets; and intestinal protozoa such as *E. histolytica*.

Urine Examination

Examination of urine specimens may be useful in diagnosing of *Schistosoma haematobium* eggs. The important portion of the urine is the last few drops of it because the eggs may be trapped in the mucus or pus and are more frequently in this portion of the urine. the sample centrifuged and the sediment examined under microscope.

Urogential Examination

Urogential specimens are collected if infection with *T. vaginalis* is suspected. Identification is based on wet-mount preparation examination of vaginal and urethral discharges, prostatic secretion, or urine sediment. Specimens should be placed in a container with a small amount of 0.85% saline and sent immediately to the laboratory for examination.

Blood Examination

Introduction:

Examination for parasites should always be performed and examination for many samples, like, feaces, blood, urine, nasopharyngeal, genital tract. Blood examination used to identify blood parasites which may intracellular or extracellular and mostly protozoa, each animals with special site of blood collection which different from one to another as it's below:

Table (1): sites of blood collection from different species

Species	Site of Collection and Permitted Conditions
Mouse	cardiac, orbital sinus (anesthetized only), tail vein, saphenous vein
Rat	as with mouse, plus subclavian veins Guinea Pig cardiac, anterior vena cava/subclavian vein
Rabbit	cardiac, marginal ear vein. It is often helpful to apply a vasodilator on the ears of rabbits prior to collecting blood, such as oil of wintergreen (methyl salicylate).
Dog and Cat	cephalic, saphenous veins, femoral and jugular veins
Ruminants	jugular vein
Swine	jugular vein, anterior vena cava, ear veins
Chicken	brachial vein, right jugular vein, cardiac

Generally, there are two ways to performed blood smear:

a. Direct Examination:

1. Place three drops of fresh blood, mixed with anticoagulant (EDTA) on a clean slide.
2. Put a cover slip over it and examine under microscope.
3. The live trypanosomes and microfilaria are identified in the basis of their characteristic shape and motility.

b. Staining Blood Smear:

1. Preparation of Blood Smear:

There are two kinds of blood smears

A. Thin Blood Film:

place a new clean glass slide (specimen slide) on a flat surface and add a small drop of blood approximately 1 mm from the end of the slide. while holding the other end of the specimen slide in place with the finger of one hand, another slide (spread slide) is placed in front of the drop of blood with the other hand. The spreader slide should be held as follows:

I. The middle finger should be below the spreader slide while the slide is stabilized by the edges of the adjoining fingers.
II. The spreader slide should be held at a 45-degree angle and slowly drawn backward into the drop of blood.
III. Once the edge of the spreader slide is in contact with the blood, the angle of the slide is lowered to about 25 degree to allow the blood to flow evenly across the edge of the spreader slide.

IV. Once the blood has spread evenly across the edge of the spreader slide , quickly pushed the slide across the entire length of the slide containing the blood.

V. As the spreader moves, a thin film of blood will be left on the specimen slide. ideally the blood smear should cover 2/3 to 3/4 of the slide when correctly prepared. the goal is to achieve a wedge-shaped smear with a thin edge.

VI. The blood smears should be air-dried. they should be labeled immediately with patient's name and date.

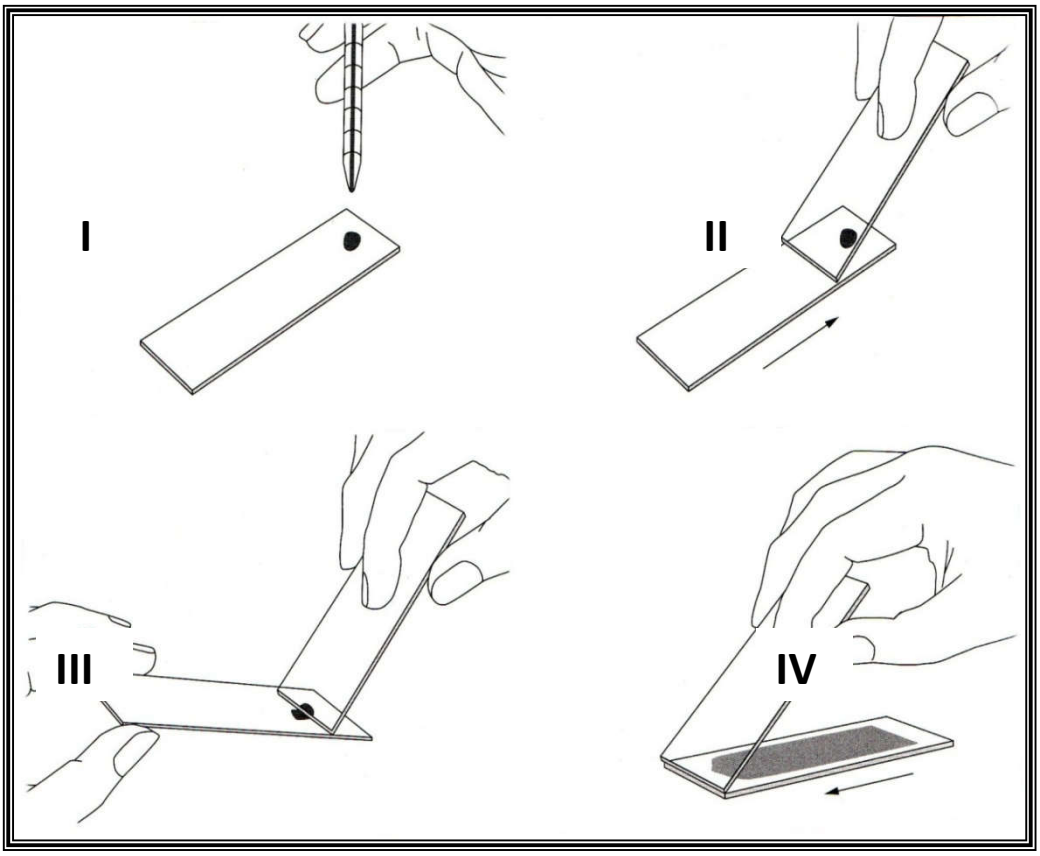


Figure (5): Steps of Blood Smear Preparation

B. Thick blood film:

Thick blood films are prepared to identified nematode microfilaria. these are soaked in water or in aqueous solution of stain (Giemsa) to leach out the hemoglobin out of the red cells. if the stain is alcoholic stain (Leishman) the films are immersed in water and agitated gently until colorless, the films are dried in air before staining.

2. Staining:

a. Giemsa Stain:

I. Preparation of Stain

Giemsa stain	**0.75 gm**
Glycerol	**25 ml**
Methanol	**75ml**

For preparation, the stain is placed in a mortar glycerol is added to make a paste with pestle. then methanol is added and stirred for mixing. Finally, stain is poured into a dark bottle and incubated at 37 C^{o} for 24 h. (giemsa working solution prepared by adding 1 part of giemsa stain solution to 10 parts of buffered water (pH= 7.2).

II. Procedure

1- Fixed the air dried film with methanol for 2 minutes.
2- Allow the smear to dry and then immerse it in Giemsa working solution for 45 min.
3- Discard the excess stain and rinse the slide in buffered water.
4- Drain the slide thoroughly in vertical position and allow them to air dry.

5- Wipe the bottom sides of the slide before drying.

6- Mount the stained smear in a neutral mounting median (Canada balsam or DPX).

b. Leishman Stain:

I. Preparation of Stain

Leishman stain	**150 gm**
Methanol (pure)	**100 ml**

For preparation, the stain and methanol are added into a dark bottle. the stain dissolve slowly and the bottle should be shaken from time to time during next two days.

II. Procedure

1-Cover the air-dried blood smear with 7-10 drops of stain.

2-Dilute the stain solution with twice the number of buffered water drops (pH=6.5) after one minute.

3-Commleave it for 15-20 min. and then drain off the excess stain.

4- Rinse the stained slide with buffered water and keep it vertically for air-drying.

Note:

There are also other stains to stain the blood films such as Wright's stain and Field's stain.

Examination of Blood-Stained Slide

The blood smear is first viewed under low power to find an area for viewing that is, an area where the cells are not overlapping but are in only one layer close to one another. this area is usually located near the wedge-shaped end of the blood smear. A drop of immersion oil is then placed on the selected site and the 100X lens is moved into the oil while observing from the side. the smears should be examined following a zigzag path as shown in (fig. 2). Moving toward the thicker part of the smear.

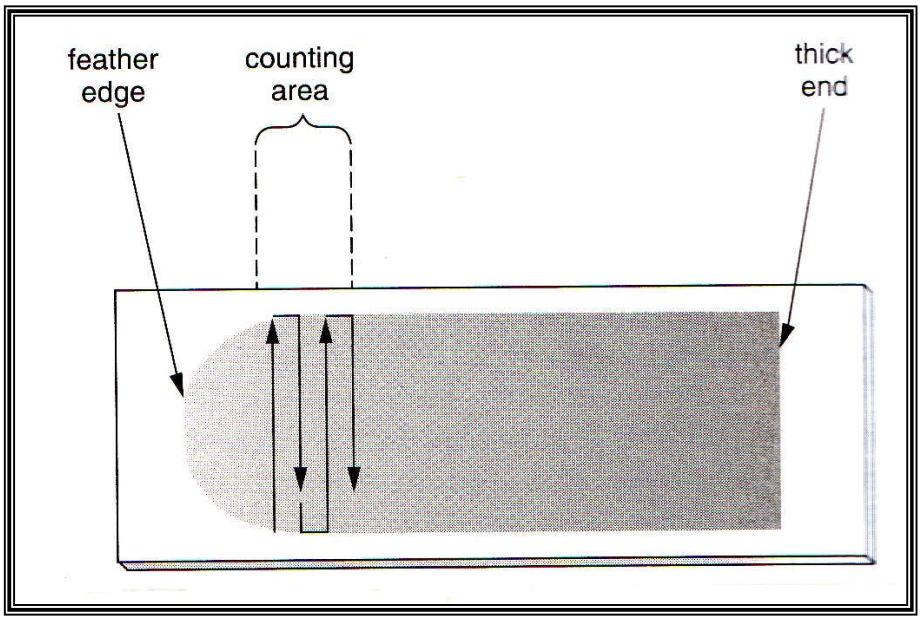


Figure (6): Examination of blood-stained slide following a zigzag path

PROTOZOA

CHARACTERISTICS OF THE PROTOZOANS

1-This kingdom contains the unicellular, or one-cell, organisms (better known as the protozoans).

2-Most protozoans are free-living organisms; however, those protozoans that are parasitic may produce significant pathology in domesticated animals and humans.

3-Within the kingdom Protista are several phyla, which contain:

(1) flagellated protozoans, (2) amoeboid protozoans, (3) apicomplexans, and (4) ciliated protozoans.

4-Protozoans vary greatly in size, form, and structure; most are microscopic, and a very few are macroscopic, that is, visible to the naked eye.

5-Binary fission, the most common form of reproduction, is asexual; multiple asexual division occurs in some forms. Both sexual and asexual reproduction occur in the Apicomplexa.

6-The stages of parasitic protozoa that actively feed and multiply are frequently called trophozoites; in some protozoa, other terms are used for these stages. Cysts are stages with a protective membrane or thickened wall. Protozoan cysts that must survive outside the host usually have more resistant walls than cysts that form in tissues.

7-All parasitic protozoa require preformed organic substances that is, nutrition is holozoic as in higher animals.

Histomonas meleagridis

Kingdom: Protozoa

Phylum: Parabasalia

Class: Trichomonadea

Order: Trichomonadida

Family: *Dientamoebidae*

Genus*: Histomonas meleagridis*

Name of Disease: Histomonosis, black head disease, necrotic enteritis, enterohepatitis syndrome.

Intermediate and final host and life cycle:

Birds become infected by ingestion of the embryonated egg of the caecal worm, *Heterakis gallinarum*, the flagellate being carried in the unhatched larva. When the egg hatches, the *Histomonas* are released from the larva and enter the caecal mucosa where they cause ulceration and necrosis. They reach the liver in the portal stream and colonize the liver parenchyma, producing circular necrotic foci, which increase in size as the parasites multiply in the periphery of the lesion. The next phase of the life cycle is not clear, but it is presumed that the *Heterakis* worms become infected with the caecal histomonads, possibly by ingestion, and that these subsequently reach the ovary of the worm. It is certainly established that the histomonads become incorporated in a proportion of the *Heterakis* eggs, and thus reach the

exterior. Infection of birds may also result from the ingestion of earthworms, which are transport hosts for *Heterakis* eggs and larvae.

Geographical Distribution: Epidemics are common and long lasting. Worldwide, in extreme large animal houses.

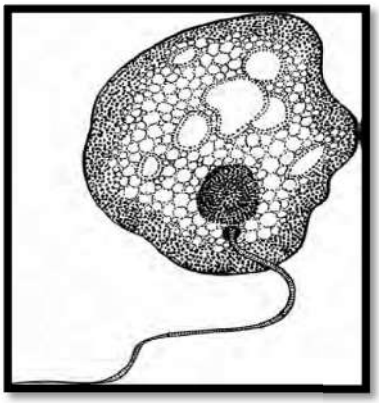
Site of Infection: In the caecum**,** the caecal mucosa and liver**.**

Diagnostic feature for parasite: A pleomorphic organism, the morphology depending on organ location and the stage of disease. In the caecum, the organism is round or oval, amoeboid, with clear ectoplasm and granular endoplasm, 6.0–20 µm in diameter, and bears a single flagellum, although this appears to be lost when in the mucosal tissue or the liver. The nucleus is vesicular and a flagellum arises from a small blepharoplast near the nucleus. In the caecal mucosa and liver, the organism is found singly or in clusters and is amoeboid, 8–15 µm in diameter, with no flagellum.

Diagnoses: Infections with *H. meleagridis* can be verified at early stages by microscopical examination of fresh feces. In cases of post mortem examinations, the liver shows pea-sized, defined yellowish necrosis sites. Squeezing preparation of the liver tissues shows amoeboid trophozoites.

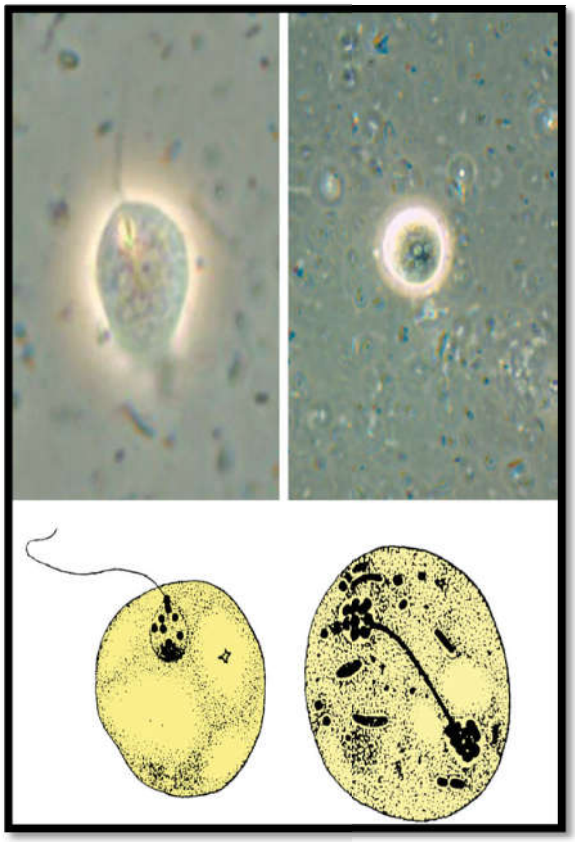
Clinical signs: Infection is often mild and asymptomatic in chickens. Turkey poults become dull, the feathers are ruffled and the faeces become sulphur- yellow in colour 8 days or more after infection. Unless treated, the birds usually die within 1 or 2 weeks.

Pathogenicity: The principal lesions of histomonosis appear in the caecum and liver. One or both caeca may be affected with small raised pin□point ulcers, which subsequently enlarge and may affect the whole mucosa, occasionally ulcerating and perforating the caecal wall causing peritonitis. The mucosa becomes thickened and necrotic and may be covered with a characteristic foul□smelling yellowish exudates that can eventually form hard dry caecal cores adhering to the caecal wall. The caeca are markedly inflamed and often enlarged. Liver lesions are pathognomonic and consist of circular, depressed, yellowish areas of necrosis and tissue degeneration, varying in size up to 1 cm or more and extending deeply into the liver. In older birds the lesions may be confluent and other organs such as the kidney and lung may occasionally be involved. The parasites can be readily found on histopathological examination. Affected lesions are hyperemic, hemorrhagic and necrotic with lymphocytic and macrophage infiltration and the presence of giant cells.



Histomonas meleagridis

Liver lesions due to *Histomonas meleagridis*.



***Histomonas meleagridis*. Light microscopic and diagrammatic representations of intestinal stages**

Eimeria spp.

Kingdom: Protozoa

Phylum: Apicomplexa

Class: Conoidasida

Order: Eucoccidiorida

Family: Eimeriidae

Genus: *Eimeria* spp.

Name of Disease: Coccidiosis**.**

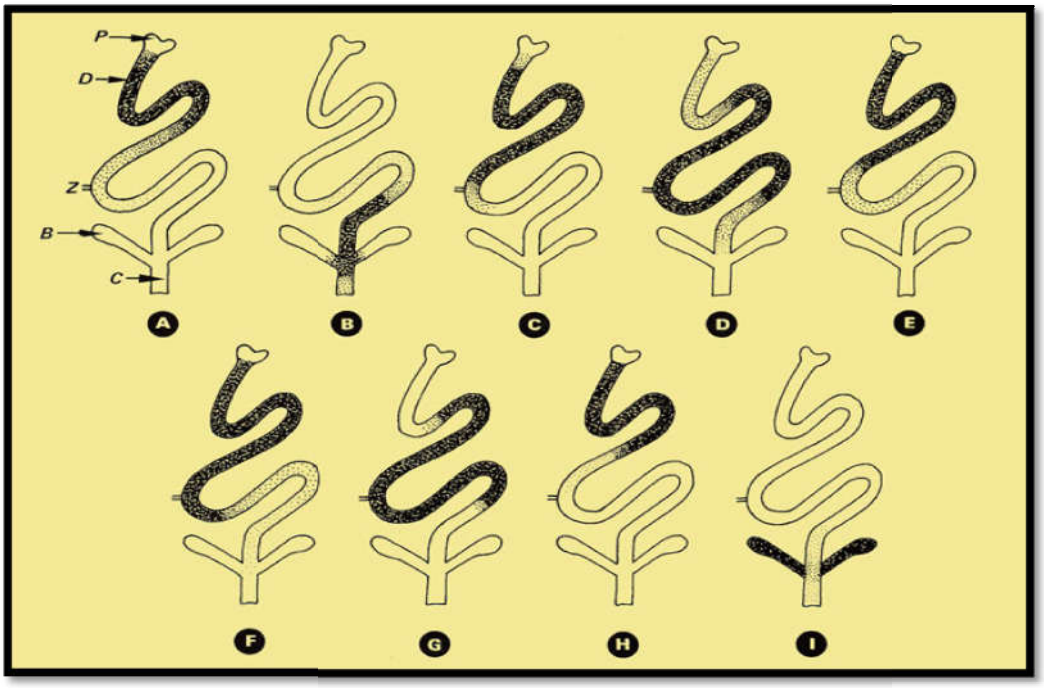
Ex:(*Eimeria* spp. in chicken):

Intermediate and final host and life cycle:

The hosts Mammals, birds, reptiles and fish, and the life cycle of all *Eimeria* spp. involves two or more generations of an asexual development known as schizogony, followed by a sexual phase termed gametogony which results in the formation of oocysts. Oocysts are excreted in the faeces and become infective in the litter only after sporulation during which sporozoites are produced.

Geographical Distribution: Worldwide; especially common in large breeding and egg production units.

Site of Infection: *Eimeria* are not only very host specific (e.g. the species of chickens cannot be transmitted to ducks or geese), but also develop exclusively in cells of peculiar intestinal regions:



Diagrammatic representation of the infection sites of the different *Eimeria* species of the domestic chickens according to various authors.

(a) ***E. acervulina*; (b*) E. brunetti*; (c) *E. hagani*; (d) *E. maxima*; (e) *E. mivati*; (f) *E. mitis*; (g) *E. necatrix*; (h) *E. praecox*; (i) *E. tenella*.**

B caecum; C colon; D duodenum; P pylorus; Z jejunum (shortened)

Diagnostic feature for parasite: (*E. tenella*):

-Unsporulated oocysts with very resistant oocyst wall.

-A sporulated oocyst contain 4sporocysts each with 2 sporozoites (8sporozoites are released from the oocyst.

-The sporozoites becomes **a schizont**: look like a round cell full of merozoites. Schizont begin as small basophilic rounded cells (mother merits) located intracellular within host cells.

-Mature schizonts appear as membrane-bound clusters of small basophilic bodies (similar to bunches of grapes).

- **The merozoites**: look like small banana shaped objects

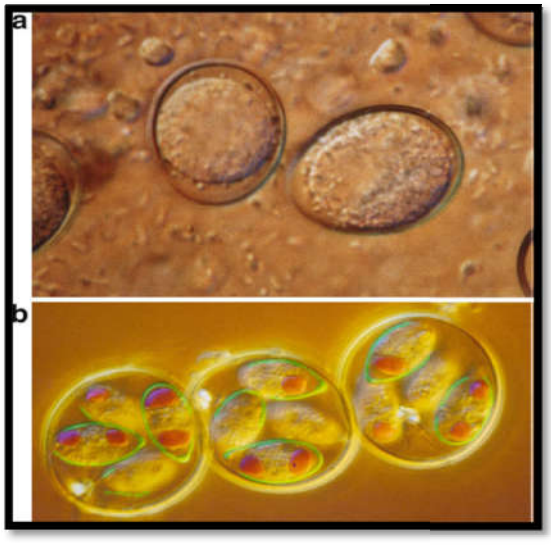
- The merozoites develops into **Gamonts** either a macrogametocytes or a microgametocyte. microgamonts (♂) apparent as multinucleate basophilic stages ultimately shedding small bi flagellated microgamete's; and macrogamonts (♀) evident as uni nucleated eosinophilia cells with a single ovoid nucleus

Diagnoses:

Coccidiosis is characterized by the occurrence of masses of unpopulated oocysts in the feces, which can be diagnosed microscopically with the help of flotation or by concentration methods. In cases of bloody and fluid feces, the use of concentration methods is mostly not needed, since then masses of oocysts occur. The exact species diagnosis can be obtained by special laboratories, but is mostly not needed, even if several parasitic species of parasites are obviously present, since the available medication covers practically all species. At postmortem in the acute phase, the caecum are distended by blood following erosion of the mucosa. Large second generation schizonts and free merozoites can be detected in smears from the caecum mucosa. Caecal cores are composed of necrotic debris, gametocytes and oocysts may be found during the recovery period of the host. Acute deaths without the presence of oocysts may occur.

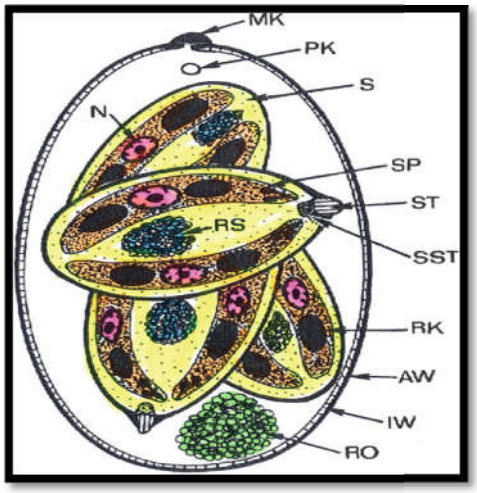
Clinical signs: Acute coccidiosis is characterized by severe diarrhea as based on several types of enteritis (catarrhalic up to hemorrhagic ones). The feces are a watery fluid, mixed with flakes of slime, contain blood and appear often greenish. The destruction of the epithelia and/or other organs induces considerable pathophysiological disturbances (e.g. increase of the acidity of the intestinal contents; loss of plasma proteins, blood and vitamins; loss of carbohydrates; dysfunction of the kidneys; hypothermia before death). In addition, secondary bacterial infections many increase these effects.

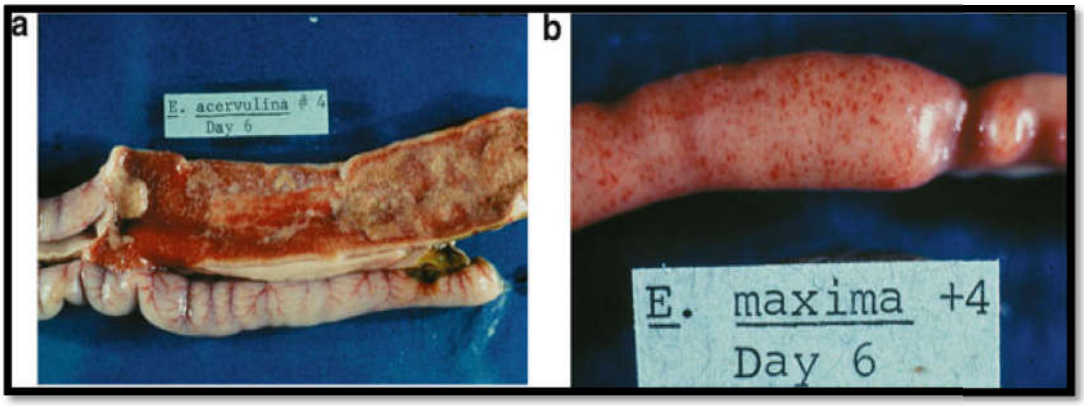
Pathogenicity: The disease is usually chronic, with birds showing poor weight gains but little mortality. Clinical disease occurs about 3 days following the ingestion of large numbers of oocysts. The first stage meronts of this species develop deep in the glands. The second stage meronts are unusual in that the epithelial cells in which they develop leave the mucosa and migrate into the lamina properia and sub mucosa. When these meronts mature and rupture, about 72 hours after ingestion of oocysts, hemorrhage occurs, the mucosal surface is largely detached and clinical signs become apparent.



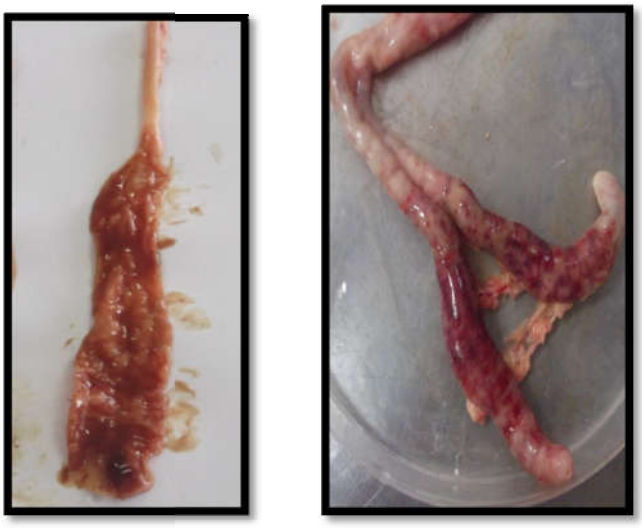
Light micrographs
(a)Unpopulated oocysts
(b) Sporulated oocysts of the genus *Eimeria*. Inside the sporulated oocysts, each 4sporocysts are visible.

Diagrammatic representation of an oocyst of an *Eimeria* species.
AW Outer oocyst wall;
IW Inner oocyst wall;
MK Cover of micropyle;
N Nucleus;
PK Polar body;
RK Refractile body;
RO Residual body of oocyst;
RS Residual body of sporocyst;
S sporocyst;
SP sporozoite;
SST Substieda body;
ST Stieda body.





Macrophotos of intestines of chickens parasitized by *E. acervulina* (a) and *E. maxima* (b)



Macro photo of *Eimeria tenella*-infected caeca of a domestic chicken showing large hemorrhages.

***Eimeria tenella* Sporulated oocysts**

***E. tenella* Sporozoites**

Cryptosporidium parvum

Kingdom: Protozoa

Phylum: Apicomplexa

Class: Conoidasida

Order: Eucoccidiorida

Family: Cryptosporidiidae

Genus: *Cryptosporidium parvum*

Name of Disease: Cryptosporidiosis.

Intermediate and final host and life cycle:

As soon as an oocyst has been ingested by its host and reached the small intestine, the four sporozoites are released from the oocyst and become attached at the surface of an intestinal cell. Each sporozoite grows up to a spherical schizont, which forms an inner vacuole into which the developing merozoites protrude after repeated nuclear divisions. The schizogonic reproduction may be repeated several times so that the parasites may cover practically most of the intestinal cells.

Geographical Distribution: Worldwide.

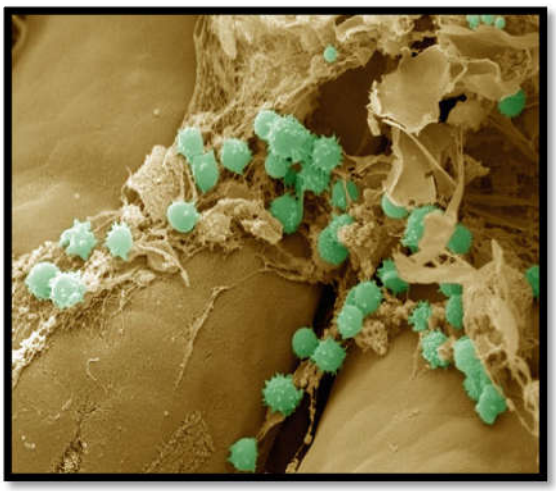
Site of Infection: Small and large intestine.

Diagnostic feature for parasite: Mature oocysts is ovoidal or spheroidal, 5.0 by 4.5 µm (range 4.6–5.4 by 3.8–4.7 µm) and have a length/width ratio of 1.19.

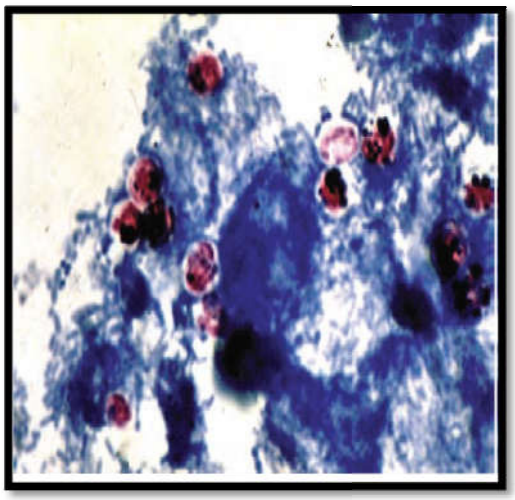
Diagnoses: Oocysts may be demonstrated in Ziehl-Neelsen'scarbol-fuchsin stained smears of diarrhea faces.

Clinical signs: Infection with Cryptosporidium is generally asymptomatic but may cause acute diarrhea in neonatal animals or more chronic diarrhea in young immunosuppressed animals or in animals with intercurrent and debilitating diseases such as distemper in dogs or feline leukemia virus (FeLV)/feline immunodeficiency virus (FIV) in cats.

Pathogenicity: *Cryptosporidium parvum* is pathogenic for calves, sheep, pig, rat and man. Bovine cryptosporidiosis is a zoonosis. Diarrhea due to *Cryptosporidium parvum* alone is often mild and self-limiting, although the severity may be related to the general strength of the calf and the density of the pathogen in the environment and the intensity of the exposure to the organism. Combination of the infection with rota- and/or coronavirus are common and result in persistent diarrhea, emaciation and death.



Scanning electron micrograph of *Cryptosporidium* stages on damaged or even absent intestinal epithelium



Oocysts of *C. parvum*
(Ziel-Neelsen Stain ˣ 1000)

Toxoplasma gondii

Kingdom: Protozoa

Phylum: Apicomplexa

Class: Conoidasida

Order: Eucoccidiorida

Family: Sarcocystiidae

Genus: *Toxoplasma gondii*

Name of Disease: Toxoplasmosis.

Intermediate and final host and life cycle:

Cats and other members of the Felidae are definitive hosts. Many mammals including all domestic animals act as intermediate hosts. Infection is acquired orally by ingestion of sporulated oocysts from faeces of the final host.

Geographical Distribution: Worldwide.

Site of Infection: Tachyzoites are found developing in vacuoles in many cell types, for example fibroblasts, hepatocytes, reticular cells and myocardial cells. In any one cell, there may be 8–16 organisms, each measuring 6.0–8.0 μm. Tissue cysts, measuring up to 100 μm in diameter, are found mainly in the muscle, liver, lung and brain and may contain several thousand lancet□shaped bradyzoites.

Diagnostic feature for parasite:

-Oocyst: Each sporulated oocyst contains two sporocysts and each sporocyst contains four sporozoites.

-The tachyzoite: Is often crescent shaped with a pointed anterior(conoidal) end and around posterior end. The nucleus is usually situated toward the central area of the cell. It is active and motile.

-**Tissue cysts:** In the brain are often spheroidal where as intramuscular cysts are elongated. Although tissue cysts may develop in visceral organs, including the lungs, liver, and kidneys, they are more prevalent in the neural and muscular tissues, including the brain, eyes and skeletal and cardiac muscles. It encloses hundreds of crescent-shaped bradyzoites.

-Bradyzoites: Differ structurally only slightly from tachyzoites. They have a nucleus situated toward the posterior end, where as the nucleus in tachyzoites is more centrally located. Bradyzoites are slenderer than are tachyzoites and bradyzoites is shorter than tachyzoites.

-Microgamonts: Mature male gamonts (microgamonts) are ovoid to ellipsoidal in shape. The anterior end is a pointed structure. Microgametes use their flagella to swim to and penetrate and fertilize mature macrogametes to form zygotes.

Diagnosis:

(A) Final hosts cats:

Demonstration of the typical unpopulated oocysts in fresh feces or sporulated ones in stools, which have been situated for several days outside of the body.

(B) Intermediate hosts:

Microscopical demonstration of cysts in muscle probes of infected animals. In addition, a broad spectrum of serological tests (complement fixation, hemagglutination or indirect immunofluorecent antibody tests) but the most convincing diagnosis is the isolation of the parasite by inoculation of suspect material into mice. Impression smears of peritoneal fluids or tissues stained with Giemsa, or tissue sections of brain, liver, spleen, lungs and Lymph nodes may reveal the organisms by direct microscopic examination give indications; however, it remains often doubtful whether an infection is acute or old.

Clinical signs:

(a) Cats:

In the case of a low-grade infection, no clinical symptoms occur. In cases of high-grade infections, cats show phases of diarrhea, which stop often without treatment and may introduce a rather solid immunity. Old cats or immunosuppressed cats, however, may also become severely ill, showing an acute toxoplasmosis which is due to the infection of numerous macrophages and different tissues (e.g. muscles or legs and heart). In addition, the brain of such cats may become infected. Thus, such cats may even die from an own generalized toxoplasmosis, but it is much more common in infected intermediate hosts. In these cases, mostly hepatitis, myocarditis,

pneumonia and encephalitis are diagnosed. In cases of prenatal infections, often abortions of cat puppies may occur, too.

(b) Dogs:

Dogs are apparently bad hosts for *T. gondii,* since mainly only young dogs show severe symptoms of disease. However, toxoplasmosis might be combined with infections such as pneumonia or diarrheas. Also, cases of ataxia have been reported in heavily infected dogs.

(c) Cattle:

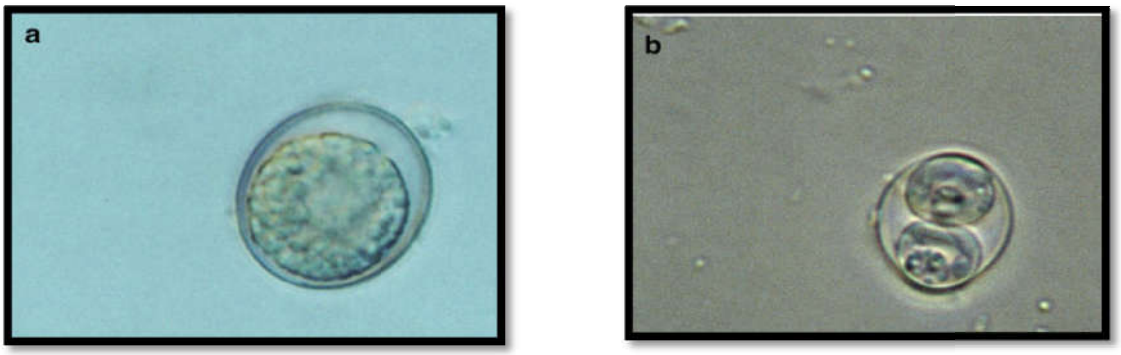
These animals—although being infected—show in general no or only low-grade infections.

(d) Sheep and goats:

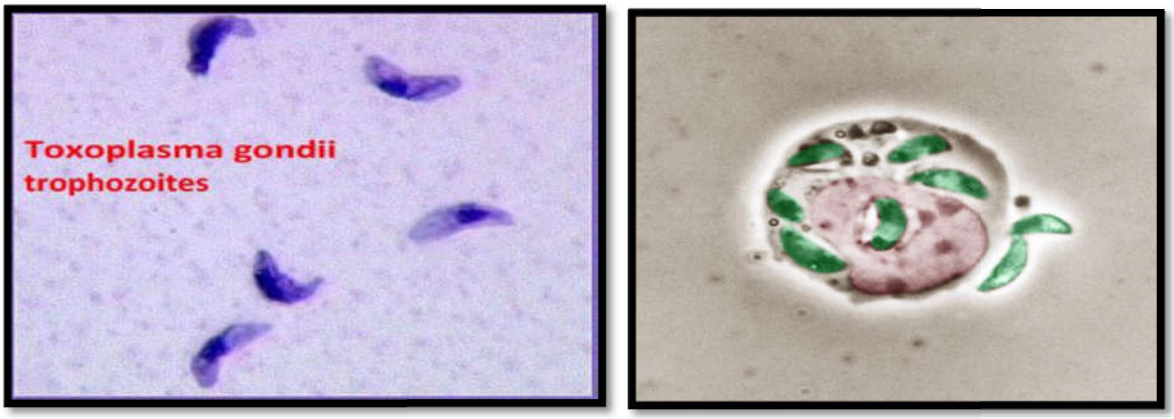
These hosts are highly susceptible and reach seroprevalence rates of up to 100 %. Pregnant animals show often cases of abortus. Low infections introduce in general few and low-grade symptoms of disease.

Pathogenicity:

In heavy infections, the multiplying tachyzoites may produce areas of necrosis in vital organs such as the myocardium, lungs, liver and brain. Examination of the brain may reveal focal microgliosis. The lesions often have a small central focus of necrosis that might be mineralized. Focal leucomalacia in cerebral white matter, due to anoxia arising from placental pathology, is often present.



***Toxoplasma gondii*: Light microscopic representations of (a) an unpopulated oocyst and (b) a sporulated oocyst**



Toxoplasma gondii: Light microscopic representation of a macrophage being infected by ***T. gondii* tachyzoites**

Sarcocystis spp.

Kingdom: Protozoa

Phylum: Apicomplexa

Class: Conoidasida

Order: Eucoccidiorida

Family: Sarcocystiidae

Genus: *Sarcocystis*

Name of Disease: Sarcocystiasis

Intermediate and final host and life cycle:

(1) Parasites of this genus occur in carnivores (definitive hosts) and herbivores (intermediate hosts).If an intermediate host has ingested oocysts or single sporocysts, schizonts are developed within 6 days, inside endothelial cells of the liver, brain, etc. However, these schizonts are not found inside a parasitophorous vacuole, but they are situated immediately inside the Cytoplasm motile sporozoites hatch from the ingested sporocysts inside the intestine of the intermediate host, i.e., swine.

(2) Two generations of schizonts are formed (5–6 and 12–17 days after infection) inside endothelial cells of blood vessels, giving rise to 60–100 merozoites by endopolygeny.

(3) Free motile merozoites; first-generation merozoites enter other endothelial cells and for schizonts, whereas merozoites of the second generation induce formation of tissue cysts.

(4) Cyst formation inside typical cells (muscle fibers, brain cells); within these cysts, the parasites are reproduced by repeated endodyogeny leading to thousands of cyst merozoites which are situated inside chamber-like hollows.

(5) When the final host man has eaten cyst containing raw or insufficiently cooked meat, the cyst merozoites are set free and enter cells of the lamina propria.

(6) Formation of female (macrogametes) via gamonts within 14 h after infection.

(7) Fusion of gametes.

(8) Formation of the oocyst wall around the zygote.

(9–11) Formation of 2sporocysts (containing 4 sporozoites each) inside the host cells. The smooth oocyst wall often becomes disrupted. Thus, fully sporulated oocysts are found in the feces.

Geographical Distribution: Worldwide.

Site of Infection: In cattle, the meronts found in the endothelial cells are quite small, measuring 2–8 μm in diameter. In contrast, the bradyzoite cysts can be very large and visible to the naked eye as whitish streaks running in the direction of the muscle fibers. They have been reported as reaching several centimeters in length, but more commonly they range from 0.5 to 5.0 mm. The cyst wall is thin and

smooth and has a small number of flattened protrusions0.3–0.6 μm long, without fibrils. Sarcocystis are nearly always in skeletal muscle or esophageal muscle.

Diagnostic feature for parasite:

Oocyst: containing two sporocysts each one contains 4 sporozoites. The oocysts produced in the intestine of the carnivore definitive host will sporulated immediately (in the host's intestine) and the fragile oocyst wall will usually rupture (again while still in the host), thus the diagnostic stage is the sporocysts (containing 4 sporozoites).

Sporocysts: It is oval shaped; each sporocyst contains four banana-shaped sporozoites. Mature sporocysts are infective stage to other susceptible hosts.

Sporozoites: morphology is very similar to that for bradyzoites.

Sarcocysts or Miescher's tube: This is spindle shaped structure with thick striated wall (are present in the bovine skeletal muscles), In the middle of the Miescher's tube there are large numbers of merozoites called bradyzoites, which are fusiform, elongated &cylindrical .The tube is divided into many compartment by septa. While in the periphery, there is usually a rounded fully developed cells called metrocystes.

Bradyzoites: have distinct anterior and posterior ends.

Gamonts: macrogamonts are rounded and microgamonts are elongated or slender with bi-flagellated

Diagnosis: Generally, not possible with the naked eye. Demonstration of muscle cysts in histological sections or following artificial digestion of infected muscles. Acute cysticercosis in swine may be diagnosed by Giemsa-stained blood smears taken from the surface of organs at necropsy or by stained histological sections. Serological techniques may be employed to discover infections (important is the presence of cysts).

Clinical signs in the intermediate host:

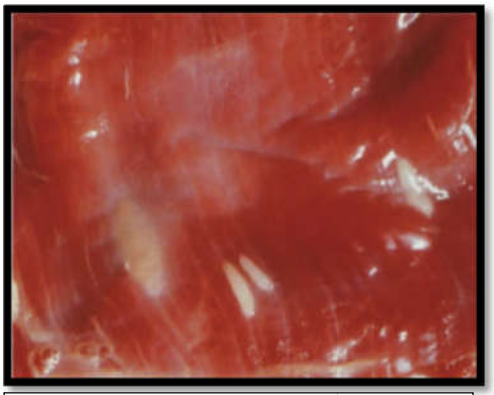
-During schizogony, the intermediate hosts may show severe symptoms of disease and may even die in case they are infected with a very virulent species.

- Anemia (watery anemia), fever and anorexia, diarrheas, wasting, increase saliva, tremor muscle, hair fall and death.

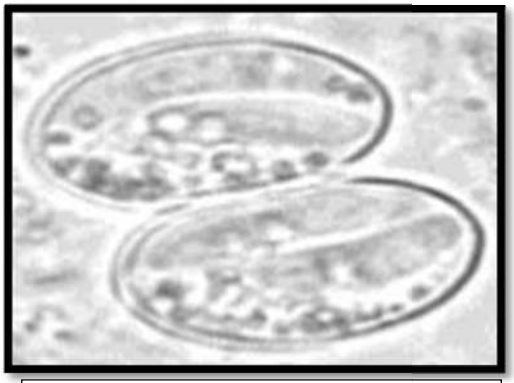
Clinical signs in the final host: As soon as the final host (carnivores, omnivores) have ingested raw, cyst containing meat, the cyst merozoites are set free in the host's intestine. Then they enter the cells of the lamina propria. In the case that the number of such merozoites is large, this infection may induce severe diarrhea as, leading to a large loss of water and a collapse of the infected animal.

Pathogenicity: Infection in the final host is normally non□pathogenic, although mild diarrhea has occasionally been reported. The principal pathogenic effect is attributable to the second stage of merogony in the vascular endothelium. Heavy experimental infections of calves with *S. bovis canis* have resulted in mortality 1 month later, with necropsy showing petechial hemorrhages in almost

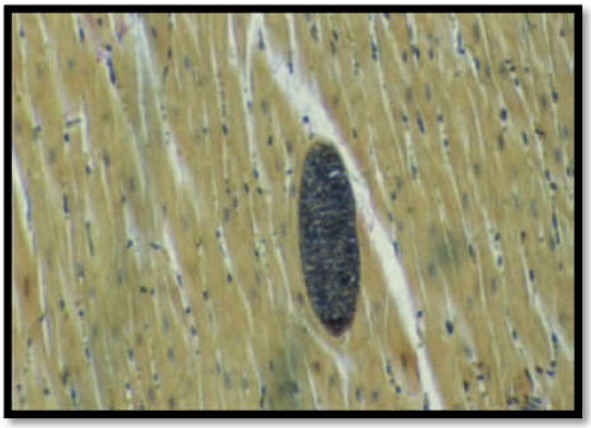
every organ including the heart, together with generalized lymphadenopathy. Experimental infection of adult cows has resulted in abortion. A naturally occurring chronic disease of cattle, Dalmeny disease, has been recognized in Canada, the USA and Britain. This is characterized by emaciation, submandibular oedema, recumbence and exophthalmia; at postmortem examination, numerous meronts are found in endothelial cells, and developing sarcocysts in areas of degenerative myositis.



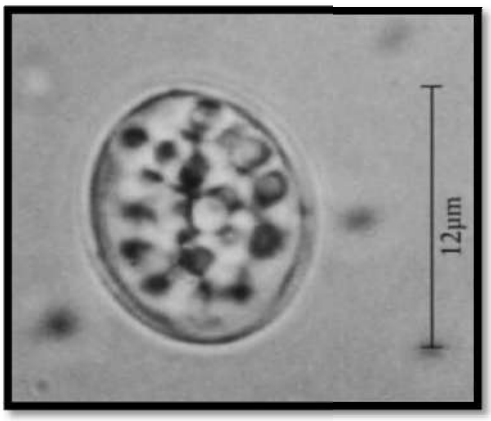
Cysts of Sarcocystis sui canis in diaphragm



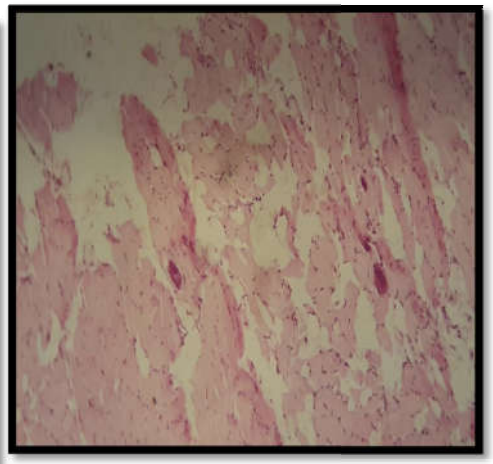
Oocysts of Sarcocysts spp.



Cysts of *Sarcocystis* sp. in the musculature



Sporocyst of *S. sui canis;* excreted by dog



Cysts of *Sarcocystis* sp.

Babesia spp.

Kingdom: Protista

Subkingdom: protozoa

Phylum: Apicomplexa

Class: Aconoidasida

Order: Piroplasmida

Family: Babesiidae

Genus: Babesia spp.

More than 100 species have been reported, only a few have been identified as causing human infections

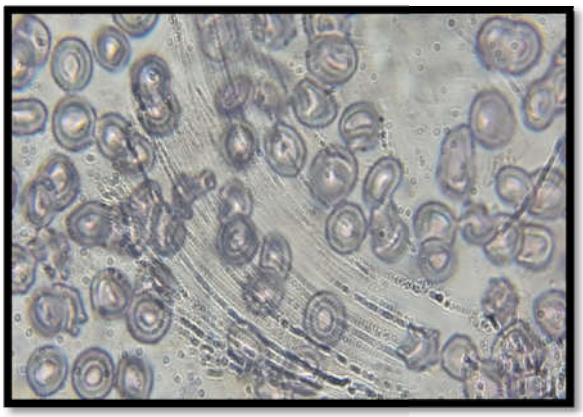
Babesia peircei…..Birds

B. bovis……Cattle

B. motasi…Sheep

B. equi……Horses

B. microti … Human



Babesia spp.

Vector host: Ixodid ticks(Hard ticks)

Vertebrate host: Domestic animals, including cattle, sheep, goats, horses, swine, dogs, and cats, as well as numerous wild animals and human.

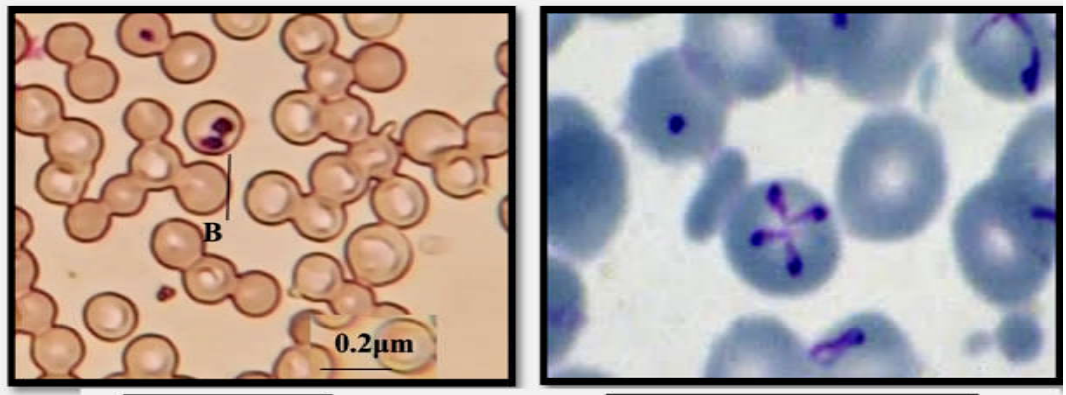
Geographical Distribution: Worldwide, infect wild and domestic vertebrate animals, and occasionally human.

Site of infection: Inside the R.B.Cs. (reproduce by binary fission).

Diagnostic feature for parasite: Tetrad forms (the so-called Maltese cross formation), although not common, are unique to *Babesia*.

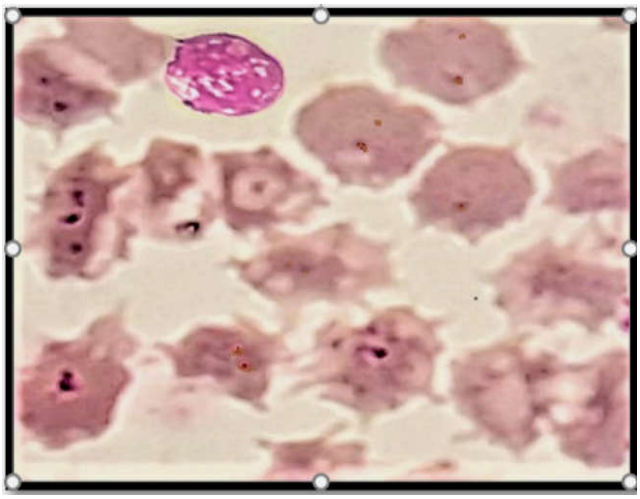
Diagnoses: Microscopy examination of a blood smear, Serological assays and Molecular assays.

Morphology: Intra erythrocytic stages appear singly as small round, ovoid or elongate trophozoites (2-4µm), in pairs as pyriform shaped merozoites, or in tetrads as cruciform merozoites.



B. bigemina | *B. duncani* (tetrad form)

Pathogenicity: As the parasite infects red blood cells of the body, erythrocyte lysis occurs, which including hemolytic anemia, jaundice, anorexia, hemoglobinemia, hemoglobinuria, as well as renal failure, fever and generalized myalgia, dark urine, and may result in death.



Erythrocytic lysis with babesiosis

Life cycle:

1- *Babesia* sporozoites are injected into the bloodstream of a vertebrate host with minute amounts of saliva, during the blood meal of an infected tick.

2- After invading erythrocytes, they differentiate into trophozoites, which divide asexually (merogony) into two or sometimes four merozoites.

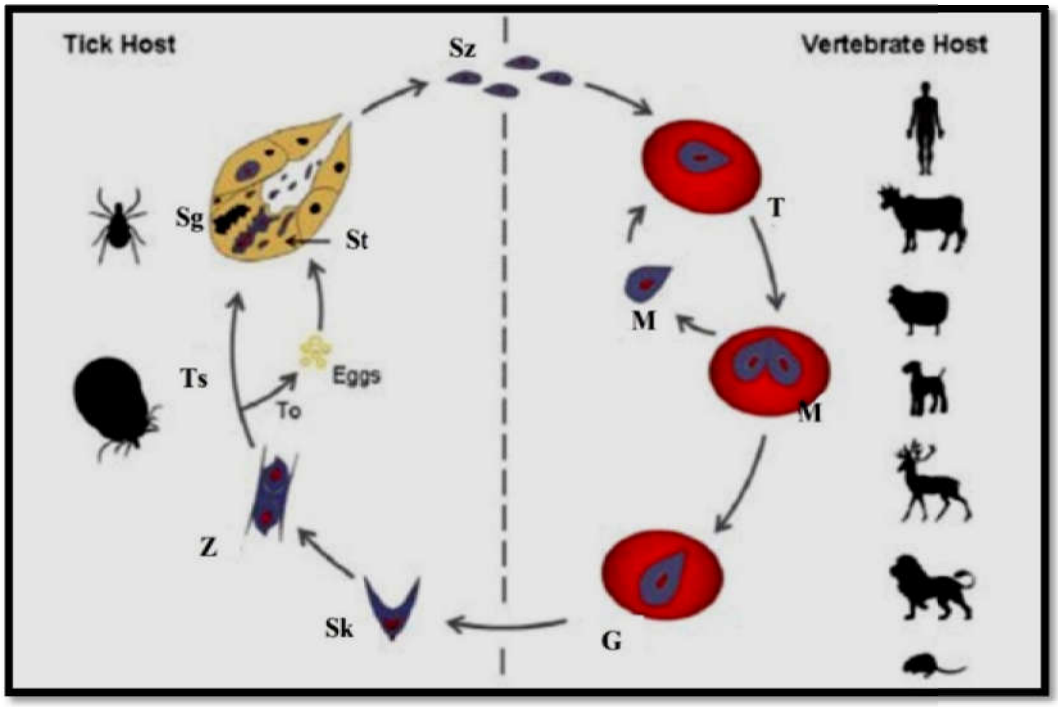
3- Merozoites exit the erythrocytes and invade new ones, continuing the replicative cycle in the host.

4- A few merozoites stop division and transform into gamonts or pregametocytes.

5- Gamogony and sporogony take place in the tick. When gamonts are taken up by a tick feeding on an infected host, they differentiate in the gut into gametes, also known as ray bodies or Strahlenkörper, that fuse forming a diploid zygote (gamogony).

6- Zygotes undergo meiosis giving rise to motile haploid kinetes, which multiply by sporogony and access the hemolymph, invading and continuing their replication in several tick organs, including the salivary glands.

7- A final cycle of differentiation and multiplication takes place, in which kinetes transform into sporozoites that will infect a vertebrate host after the tick has molded into the next stage, i.e., larvae to nymph or nymph to adult (transtadial transmission).



Generic life-cycle of *Babesia* spp.

Theileria spp.

Kingdom: Protista

Subkingdom: Protozoa

Phylum: Apicomplexa

Class: Aconoidasida

Order: Piroplasmida

Family: Theileriidae

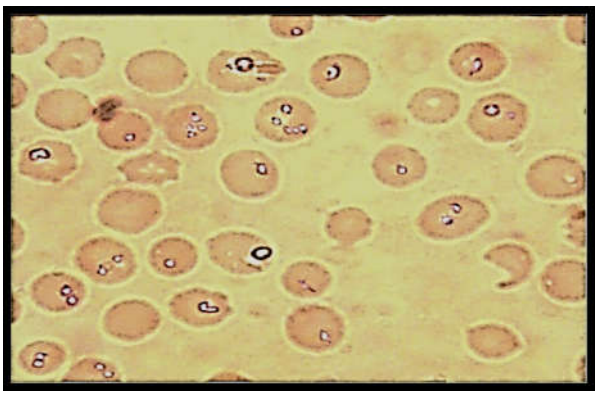
Species: *Theileria* spp.

Theileria annulata…..Bovine (Tropical theileriosis)

T. parva……..Bovine(East Coast fever)

T. uilenbergi …… Sheep and goats

T. equi…….Horses



Theileria spp.

Vector host: Ixodid ticks (Hard ticks)

Vertebrate host: Domestic and wild ungulates

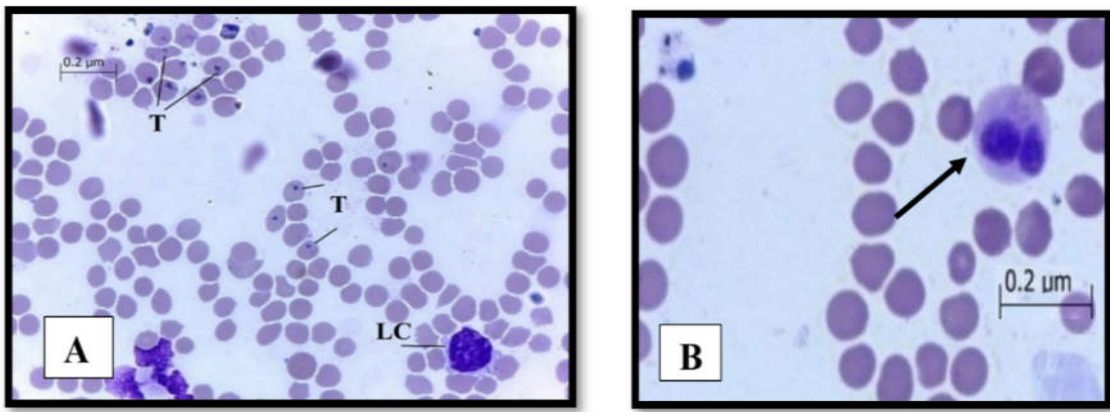
Site of infection: Inside WBC and RBC (obligate intracellular).

Geographical Distribution: Limited to geographic distribution of appropriate tick vectors in tropical and subtropical regions of the world.

Diagnostic feature for parasite: **1-** Presence of schizonts in infected leukocytes, **2-**The intra-erythrocytic piroplasm stages small and rod-shaped or oval with (0.6 – 1.25 µm) in diameter.**3-**Swollen superficial lymph nodes**.**

Diagnoses: Microscopy examination of a blood smear, Serological assays and Molecular assays.

Morphology: The piroplasms in the red blood cells form same to be spot or smudge looks like round, oval, or ring rod shaped with (0.6 – 1.25 µm) in diameter. Also, in the WBCs there are large schizonts, commonly known as **Koch's blue bodies**, appear a few days after onset of symptoms. One with large chromatin granules gives (8-16 Macromerozoites) in lymphocytes, while the microschizont appear later in the lymphocytes with small chromatin granules gives (50-120 Micromerozoites).



Infected blood cells with *T. annulata*(T), LC: Lymphocyte cell (A), Koch's blue body (black arrow) in lymphocyte (B), (1000X) Giemsa stain

Clinical signs: Range from in apparent or mild to severe and fatal. Fever (41 C° or higher), lymph nodes swelling becomes pronounced and generalized, diarrhea, lateral recumbence anemia, weakness, anorexia, the animal rapidly loses condition, lacrimation and nasal discharge, pulmonary exudate pours from the nostrils.

Pathogenicity: Lymphocytes are killed by invading T*heileria* and later in disease **lymphopoeisis** is reduced and prevented. Hemorrhage and necrosis of the liver, spleen, and intestine, swelling and nodule formation of other organs, severe anemia, wasting, jaundice, and anterior shoulder and posterior bone lymphadenopathy. Just before death, a sharp decrease in body temperature is usual, and pulmonary exudate pours from the nostrils. Death usually occurs 18–24 days after infection.

Life cycle:

1- *Theileria* are transmitted via the species of tick vectors.

2- Sporozoites enter mononuclear cells of the host and develop into trophozoites andmultinucleate schizonts by asexual reproduction. This process stimulates proliferation of the host cells, allowing further multiplication of the parasite.

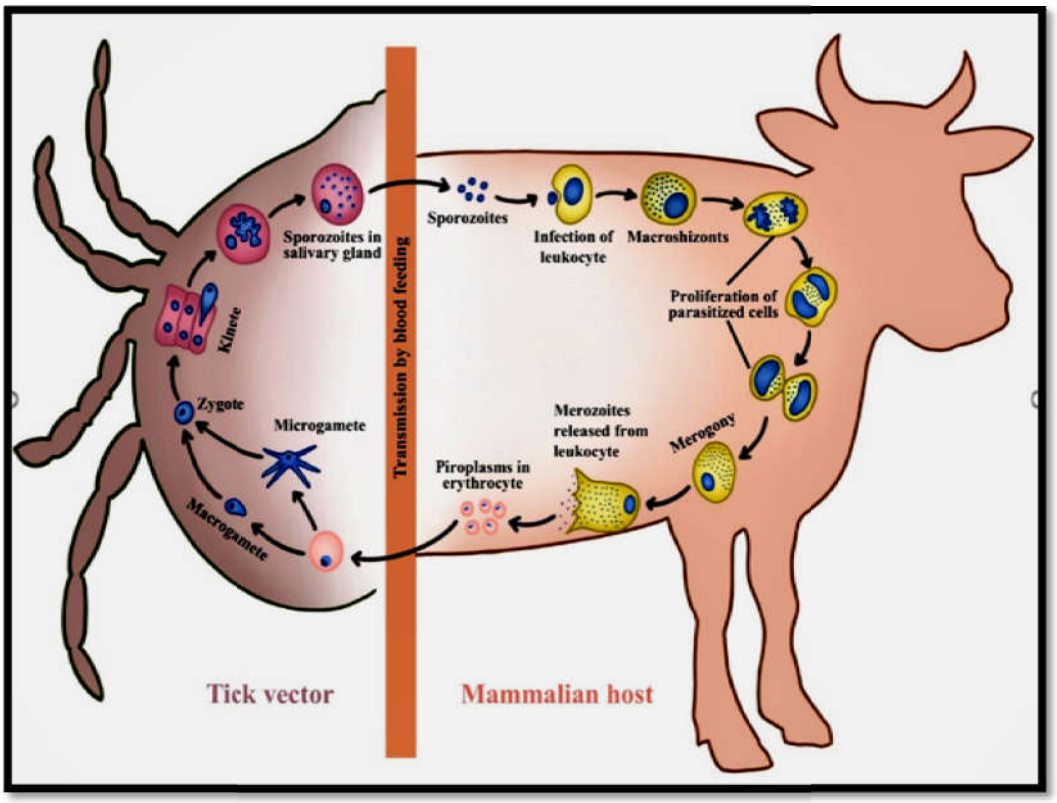
3- The local lymph nodes are first infected.

4- Schizonts then disseminate through the lymphoid tissues before differentiating into merozoites.

5- The merozoites enter the erythrocytes and form piroplasms which are infective to ticks and capable of sexual reproduction.

6- Sexual reproduction occurs within the nymph and larval stages of the tick and the final infective stage is present within

the salivary glands and is transmitted to mammalian hosts when blood feeding.



Life cycle of *Theileria annulata*

Leishmania spp.

Kingdom: Protista

Subkingdom: Protozoa

Phylum: Sarcomastigophora

Class: Zoomastigophora

Order: Kinetoplastida

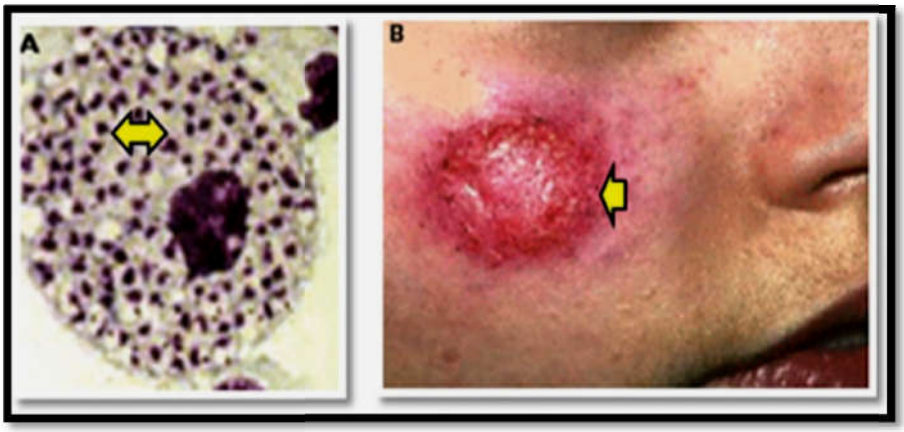
Family: Trypanosomatidae

Genus: *Leishmania* spp.

Species: *L. tropica* Cutaneous Leishmaniasis

L. donovani visceral Leishmaniasis

L. braziliensis Mucocutanous Leishmaniasis



A- Macrophage filled with a mastigotes of *L. donovani.*
B- Facial ulcerating nodule of cutaneous Leishmaniasis.

Vector host: Female phlebotomine sand flies.

Vertebrate host: Humans, dogs and rodents

Site of infection: The most common forms are cutaneous Leishmaniasis, which causes skin sores, and visceral Leishmaniasis, which affects several internal organs (usually spleen, liver, and bone marrow).

Geographical Distribution: Found in the tropics, subtropics, and southern regions. The ecologic settings range from rain forests to deserts. Leishmaniasis usually is more common in rural than in urban areas.

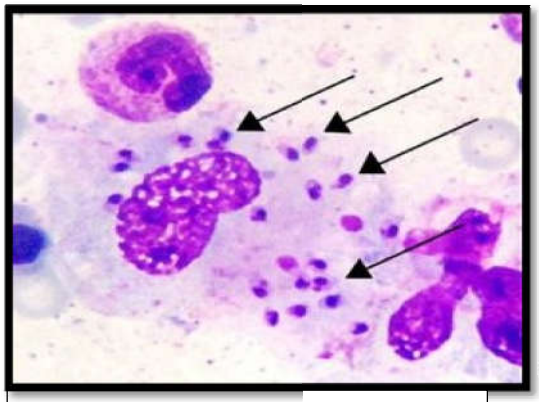
Diagnoses:

(**1**) Demonstration of parasite in tissues by light microscopic examination of the stained specimen, (**2**) Detection of parasite DNA in tissue samples, and (**3**) Immunodiagnosis by detection of parasite.

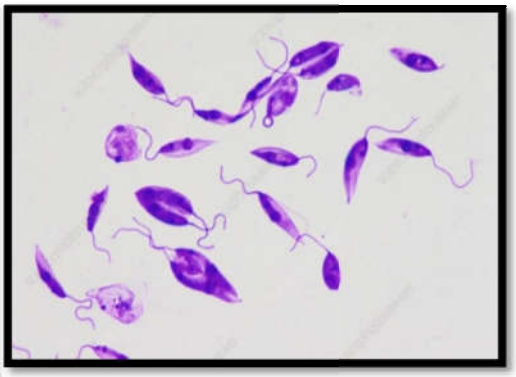
Morphology: Two developmental stages are formed:

1- A **mastigotes** are small spherical non-flagellated cells ranging from 2-4µm in diameter. The nucleus and kinetoplast are surrounded by small ring of vacuolated cytoplasm.

2- **Promastigotes** are thin elongate cells with an anterior kinetoplast and an emergent free flagellum. They are generally lance-like in shape and range in size from 5-14µm in length by 1.5-3.5µm in width.



***Leishmania* a mastigotes phagocytosed by macrophages (1000×)**

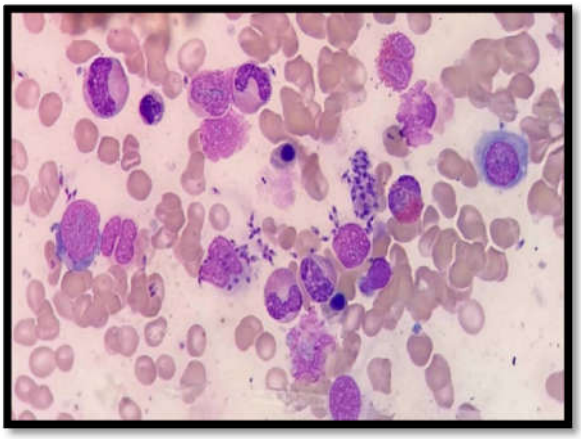


Promastigotes of *L. donovani*

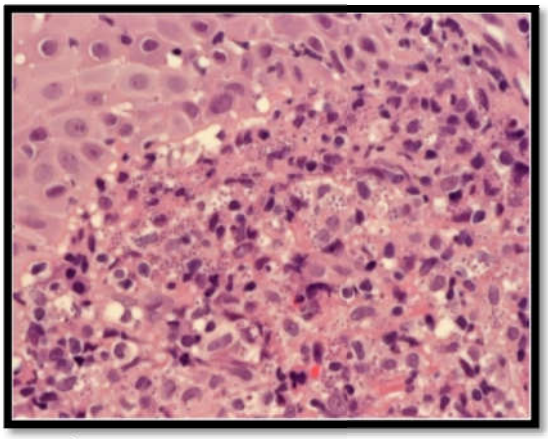
Clinical signs and Pathogenicity: Include three cases: **Cutaneous Leishmaniasis**: Appear as bump at the site of a sand fly bite, which may slowly enlarge and often becomes an open sore. The sores are usually painless and cause no other symptoms unless a secondary bacterial infection. The sores eventually heal on their own after several months but may persist for more than a year. They leave permanent scars similar to those due to burns.

Mucosal Leishmaniasis: Affects the mucous membranes of the nose and mouth, causing sores and destroying tissue. The parasites spread from the skin through the lymph and blood vessels to the mucous membranes. Mucosal Leishmaniasis can develop while the skin sore is present or months to years after the sore heals.

Visceral Leishmaniasis (kala-azar): Affects the internal organs, particularly the bone marrow, lymph nodes, liver, and spleen. People may have irregular bouts of fever. They may lose weight, have diarrhea. The liver, spleen, and sometimes lymph nodes enlarge. The number of blood cells decreases, causing anemia. Without treatment, visceral Leishmaniasis can result in death.



Visceral Leishmaniasis



Mucosal Leishmaniasis

Life cycle:

1- Leishmaniasis is transmitted by the bite of female phlebotomine sand flies.

2- The sand flies inject the infective stage, promastigotes, during blood meals.

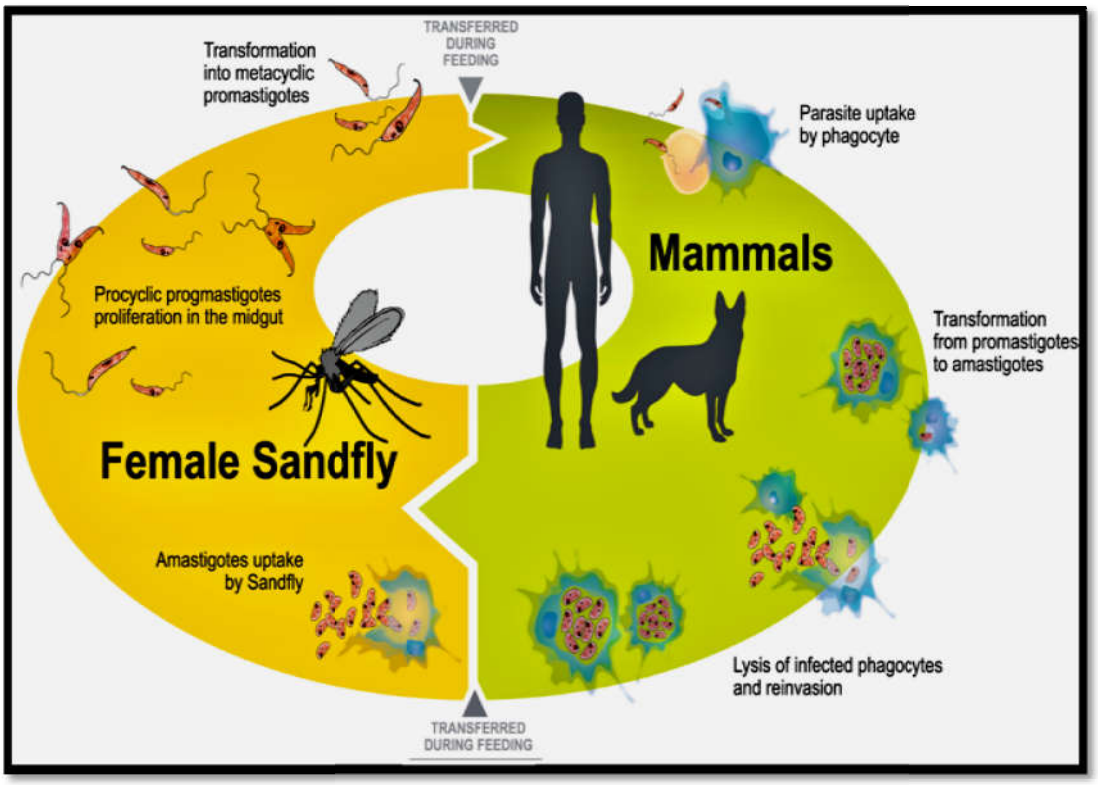
3- Promastigotes that reach the puncture wound are phagocytized by macrophages and transform into a mastigotes.

4- A mastigotes multiply in infected cells and affect different tissues, depending in part on the *Leishmania* species.

5- This originates the clinical manifestations of Leishmaniasis.

6- Sand flies become infected during blood meals on an infected host when they ingest macrophages infected with a mastigotes.

7- In the sand fly's mid gut, the parasites differentiate into promastigotes, which multiply and migrate to the proboscis.



Life cycle of Leishmaniasis

Trypanosoma spp.

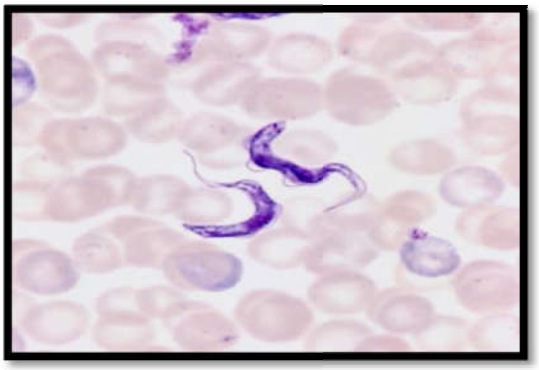
Kingdom: Protista

Subkingdom: Protozoa

Phylum: Sarcomastigophora

Class: Zoomastigophora

Order: Kinetoplastida

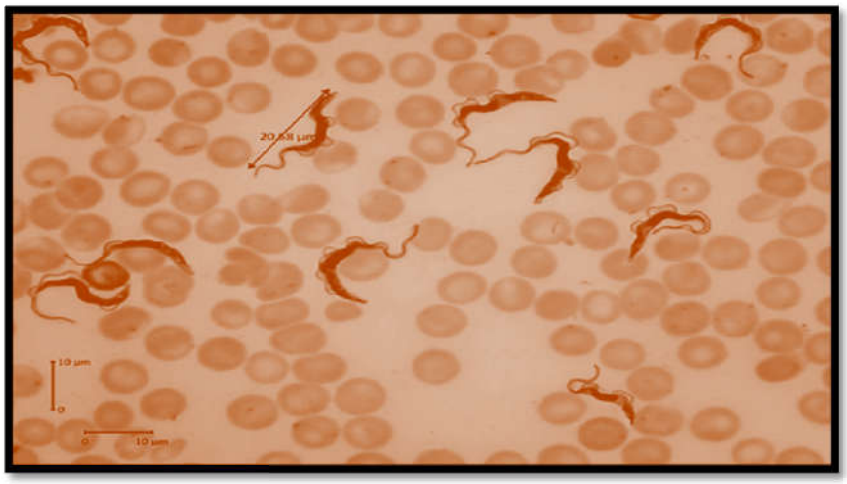


***Trypanosoma* spp.**

Family: Trypanosomatidae

Genus: Trypanosoma spp.

Species: *Trypanosoma evansi* Causes surra in animals, *T. brucei Human and animals*, *T. equiperdum* Causes venereal disease in horse and donkey



Trypanosoma evansi

Vector host: Tsetse fly, both the male and female of *Glossina* genus.

Vertebrate host: Human (sleeping sickness), Animals (in cattle the disease is called *Nagana*)

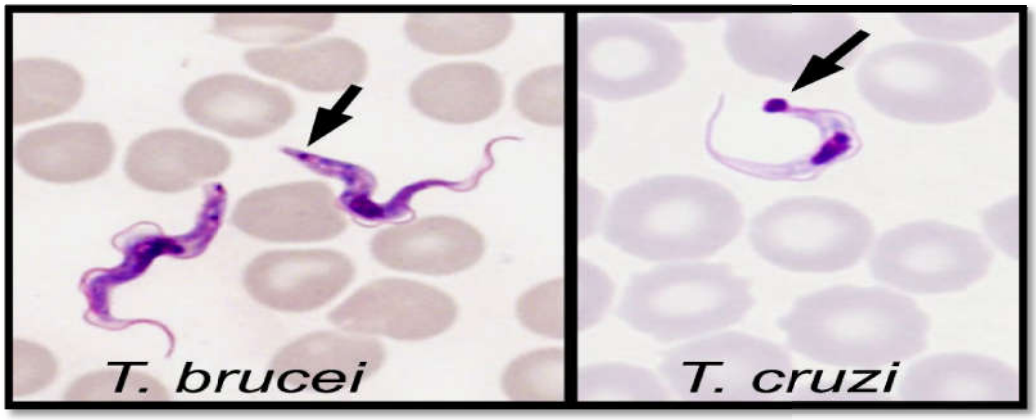
Site of infection: The bloodstream or an extracellular environment in the vertebrate host. The intestine in an invertebrate host.

Geographical Distribution: It is the most economically important livestock disease, mainly distributed through the Africa, the Americas and Asia. The disease develops in areas ranging from a single village to an entire region.

Diagnostic feature of parasite: Incubation period is generally 8–20 days. *T. congolense* usually becomes apparent in 4–24 days, *T. vivax* in 4–40 days, and *T. brucei brucei* highly variable. Infections with more virulent isolates have a shorter incubation period.

Diagnoses: (**1**) Microscopic examination

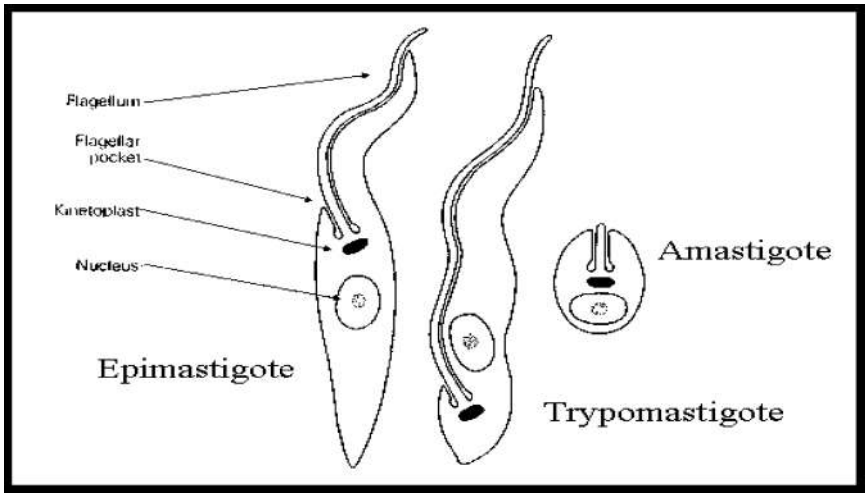
(**2**) Serologic testing, and (**3**) Genomic detection.



Trypanosoma *brucei* and *T.* cruzi

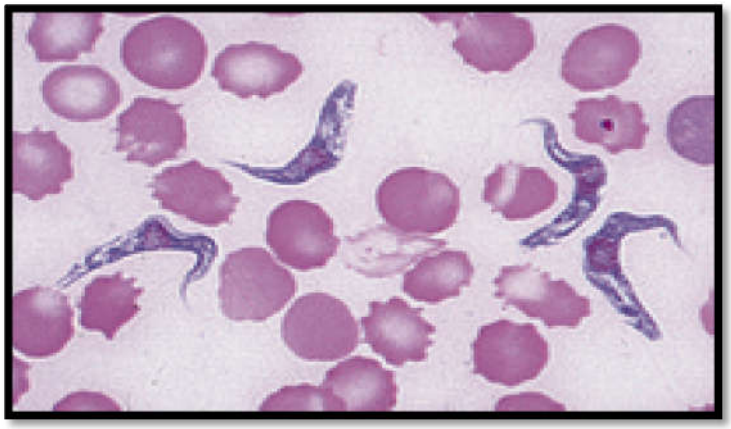
Morphology: The trypanosomes undergo a series of morphological changes during their transition from invertebrates to vertebrates:

- **Trypomastigote** is identified by their flagella that is attached to the body by an undulating membrane and the kinetoplast lies in the posterior portion of the body. This form of trypanosomes is found in the vertebrate host but is developed in the invertebrate host.
- **A mastigote** is another form of *Trypanosoma* that is identified by no visible external flagella or cilia. It is the intracellular form that is found during the replication phase of the organism.



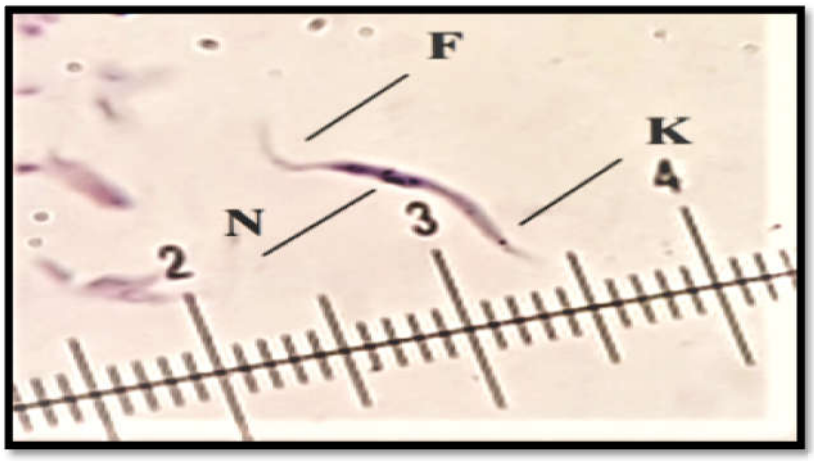
Trypomastigote and A mastigote

Clinical signs: Disease is classically acute or chronic, and is affected by poor nutrition, concurrent diseases, and other stressors. Trypanosomiasis in cattle is usually chronic – some may slowly recover but usually relapse when stressed. The most important clinical sign is non regenerative anemia, intermittent fever, oedema, enlarged lymph nodes, abortion, decreased fertility, loss of appetite, early death in acute forms, emaciation and eventual death in chronic forms often after digestive and/or nervous signs.



T. equiperdum

Pathogenicity: Infected tsetse inoculate metacyclic trypanosomes into the skin of animals, where the trypanosomes reside for a few days and cause localized inflammation (chancres). They enter the lymph and lymph nodes, then the bloodstream, where they divide rapidly by binary fission. Some species of trypanosomes attach to endothelial cells and localize in capillaries and small blood vessels, while others can invade tissues and cause tissue damage in several organs.



***Trypanosoma* sp.: (F) free flagellum, (N) nucleus, and (K) kinetoplast. Giemsa stain (400x)**

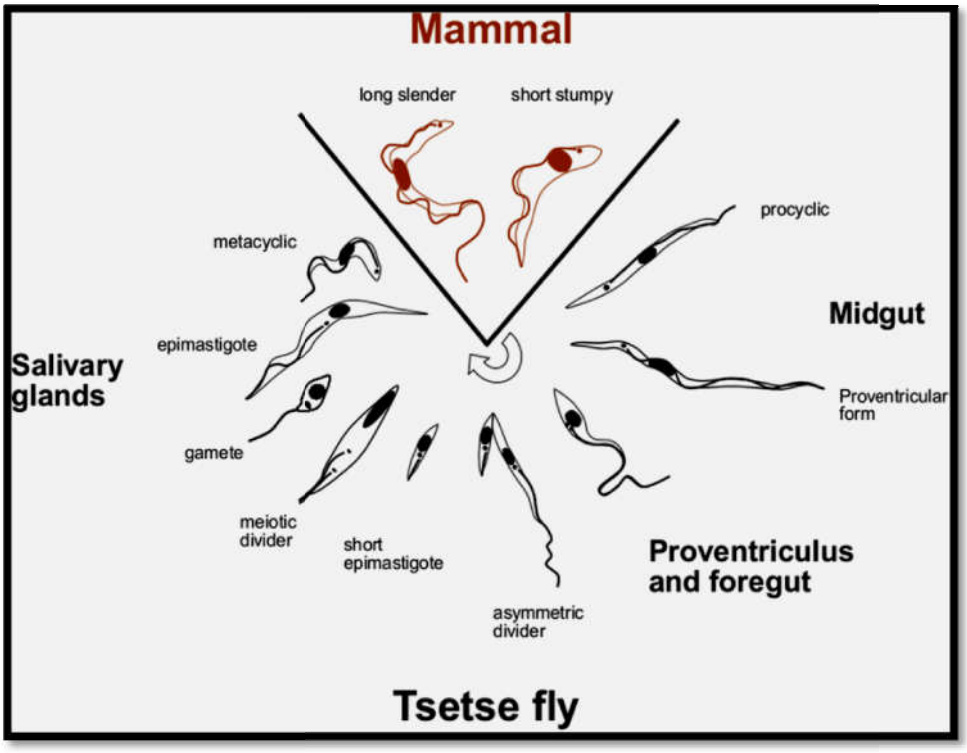
Life cycle: *Trypanosoma* exist in two different types and that have different types of life cycles – Stercorarian and Salivarian species.

1- The Stercoraria *Trypanosoma* species first infects a triatomine kissing bug during a blood meal and develops in the posterior gut of the insect. It is then released in the faeces followed by deposition of the skin of the host. The *Trypanosoma* then penetrates in the body of the host and causes infection.

2- The Salivarian species develops in the anterior gut of tsetse fly and is transmitted to the host by insect feed.

Trypanosoma brucei: It causes African Trypanosomiasis, commonly known as Sleeping sickness whereas in animals it causes animal Trypanosomiasis or Nagana disease. The insect vector Tsetse fly picks up the protozoa during a blood meal and then undergoes various morphological changes as it transitions from insects to mammals. Common symptoms of this disease include itchiness, headache and joint pains which slowly advances into numbness, poor coordination and trouble sleeping.

Trypanosoma cruzi: They are parasitic euglenoids that feed on blood and lymph. It causes Chagas disease in humans, Brucellosis in cattle, covering sickness and Surra in horses. The vector for transmission of this disease is kissing bugs that infect the host by a simple insect bite. Early symptoms of the disease include swollen lymph nodes, headaches and swelling at the site of the bite which advances into chronic infections.



Life cycle of *Trypanosoma* spp.

Trichomonas spp.

Kingdom: Protista

Subkingdom: Protozoa

Phylum: Sarcomastigophora

Class: Zoomastigophora

Order: Trichomonadida

Family: Trichomonadidae

Genus: Trichomonas spp.

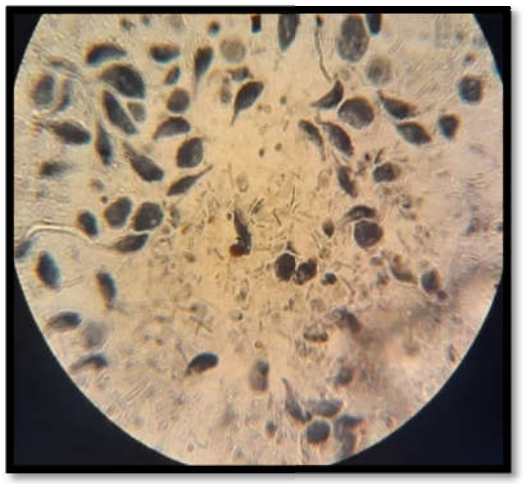
Species: *T. vaginalis, T. tenax, T. hominis* (Human)

Tritrichomonas foetus (Bovine)

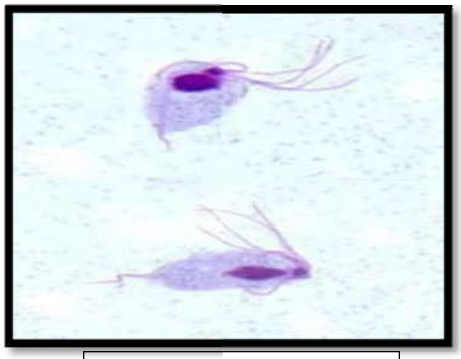
T. gallinae(Gastrointestinal tract in birds)

T. enteris (Caecum, colon of Ox, zebu)

T. equi (Caecum, colon of horse)



***Tritrichomonas* spp.**



T. gallinae

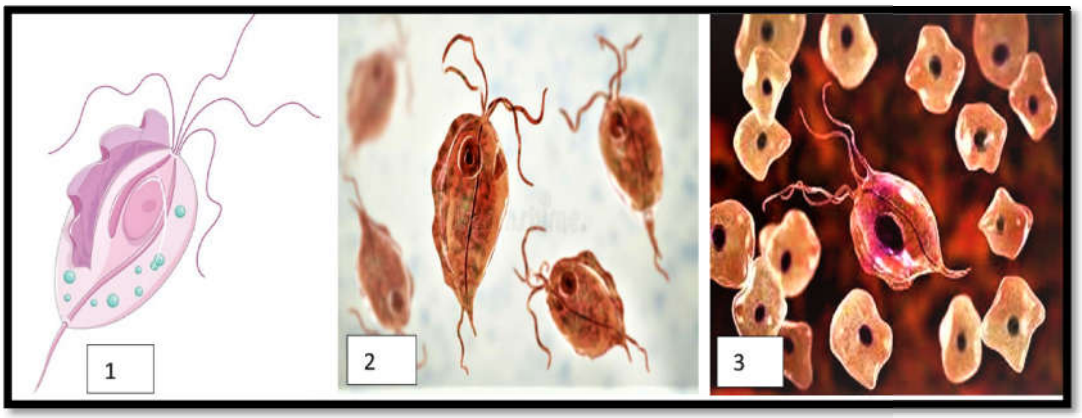
Host: Human and animals (**Direct life cycle**)

Site of infection: It can be found in the urogenital tract , oral cavity (in human), in the digestive tract and respiratory tract.

Geographical Distribution: Trichomonads are worldwide, its common parasites of many vertebrate and invertebrate species.

Diagnostic feature of parasite: Signs of early abortion, repeated returns to service and irregular estrous cycles, Cows may show outward signs of infection, namely a sticky, white vaginal discharge, which may occur for up to two months after the initial infection. The disease results in abortion of the embryo, often within 10 days of conception.

Diagnoses:1-Microscopic observation,**2-** Broth culture technique,**3-** DNAtechnology,**4-** Immunoblot studies,**5-** Antimicrobial susceptibility testing,**6-**Routine examination for Vaginitis (pH evaluation with pH paper).

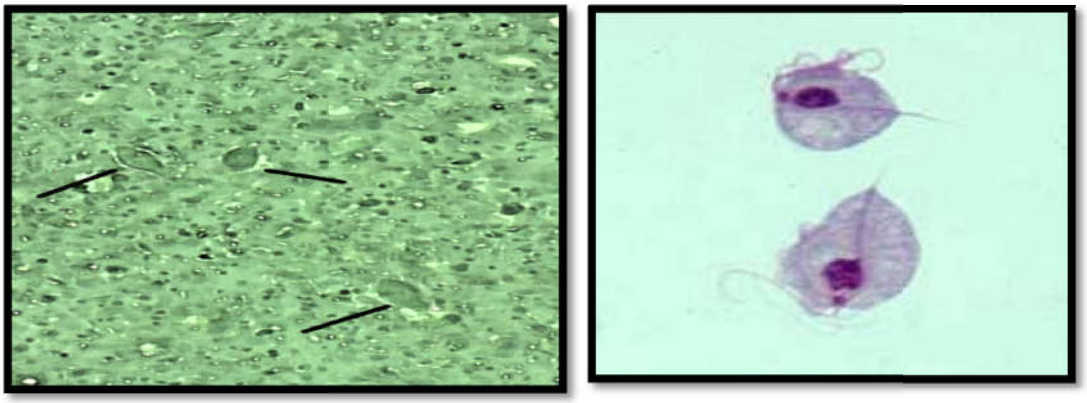


1- *T. vaginalis*, 2- *T. hominis*, 3- *T. tenax*

Morphology: *Trichomonas* consists of only one trophozoitic form and has a simple lifestyle. Usually bear flagella and have a conspicuous pelta-axostyle complex, and the recurrent flagella are often associated with a lamellar undulating membrane underlain by a striated costal fiber. The number of free flagella characterizes each genus of the family. Thus, the genus *Tritrichomonas* is characterized by having three free flagella, whereas the genera *Tetratrichomonas* and *Pentatrichomonas* possess four and five flagella, respectively.

Among the various species of trichomonads thus far identified, only a number of them are regarded as pathogens.

***Tritrichomonas foetus*:** The parasite is 5-25 µm in size and is spindle shaped with four flagella, which are whip like projections, and an undulating or wavy membrane. Three of the flagella are found on the anterior end and approximately the same length as the body of the parasite. The fourth is on the posterior end. Their movement is jerky and in a forward direction.



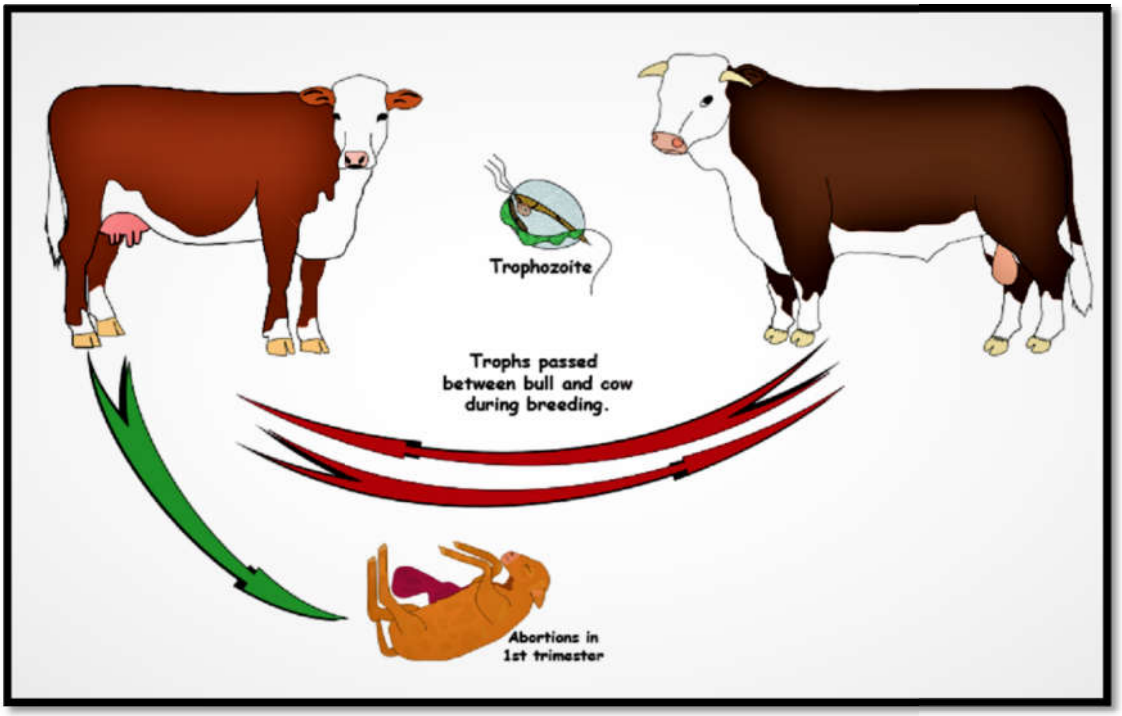
Tritrichomonas foetus

Clinical signs: Are vary from mild vaginitis or endometritis, to acute inflammation of the entire reproductive tract. Infection during pregnancy leads to early embryonal death, abortion and, sporadically, pyometra. Cows may show outward signs of infection, namely a sticky, white vaginal discharge, which may occur for up to two months after the initial infection. The disease results in abortion of the embryo, often within ten days of conception. No clinical signs are present in infected bulls

Pathogenicity: *T. foetus* is an obligate a protozoan parasite that has been traditionally identified as a cause of reproductive tract disease in cattle and gastrointestinal tract infection in cats.

Moreover, *T. foetus* is also well known as a commensal of the nasal cavity, intestines, and stomach in swine.

Life cycle: Bovine tritrichomonosis, a sexually transmitted disease in extensively managed herds throughout many geographic regions worldwide. *T. foetus* is a venereal disease in cattle causing early embryonic death and abortion. The protozoa reside on the surface and in the lumen of the female reproductive tract and in the crypt of penile epithelial cells. Transmission is achieved from infected individuals during mating and direct contact. *T. foetus* has no known cyst stage, although carrier cows occur very rarely and the pathogenesis of the carrier state is unknown. Therefore, trophozoites replicate simply by binary fission. Under natural conditions, *T. foetus* is transmitted directly from an infected animal to a healthy animal, almost exclusively through natural mating, or mechanically during the practice of artificial insemination or vaginal examination.



***Tritrichomonas foetus* life cycle**

Plasmodium spp.

Kingdom: Protista

SubKingdom: Protozoa

Phylum: Apicomplexa

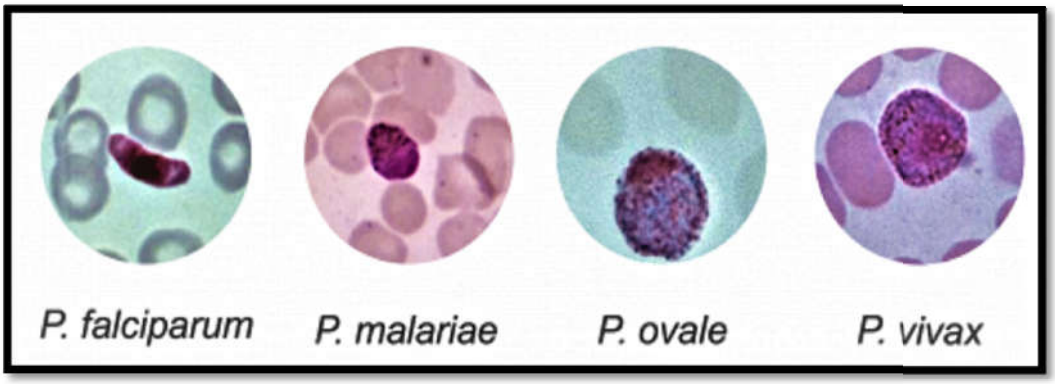
Class: Aconoidasida

Order: Haemospororida

Family: Plasmodiidae

Genus: Plasmodium spp.

P. falciparum*, *P vivax*, *P malariae*, and *P ovale (Human and animals), ***P. knowlesi****(non-human primates)*



Shape of the gametocytes according the species of *Plasmodium*

Vector host: Insects such Culex, Anopheles, Aedes spp. as invertebrate host.

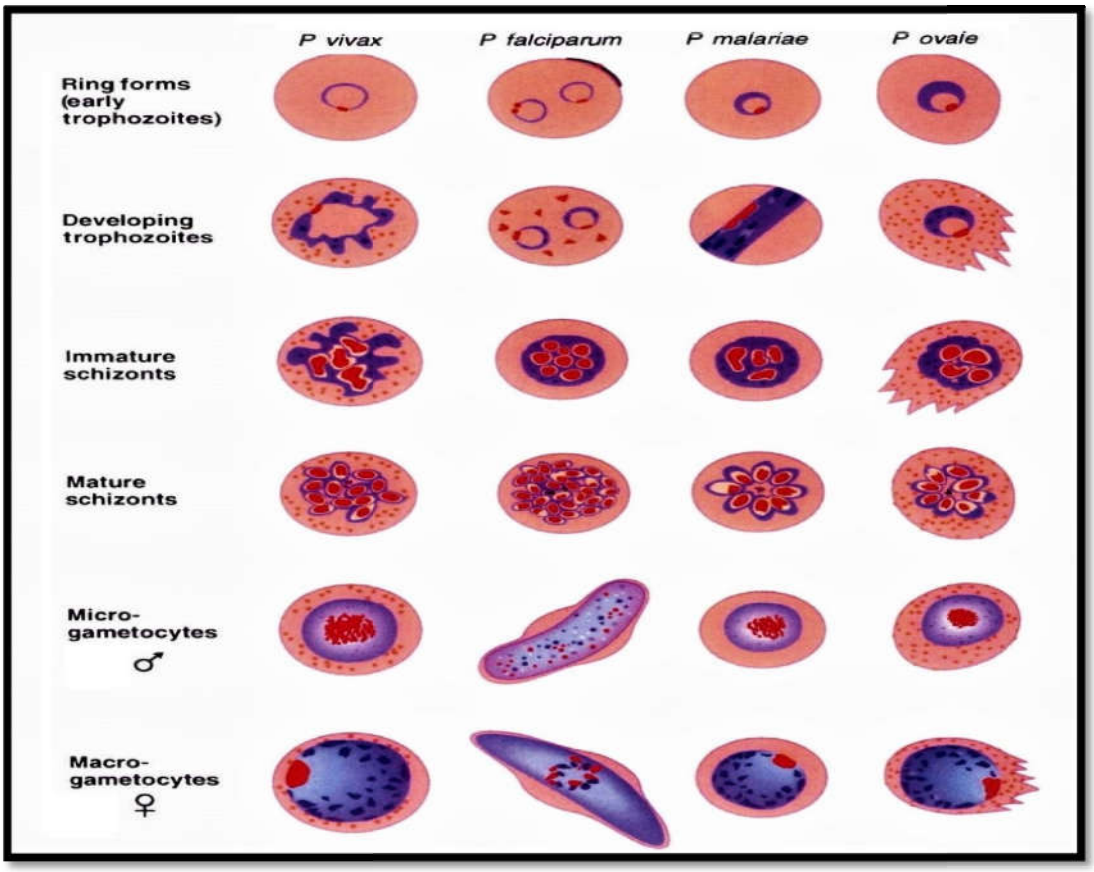
Vertebrate host: Human and other mammals, reptiles and birds.

Site of infection: Liver and red blood cells.

Geographical Distribution: Malaria is distributed worldwide throughout the tropics and subtropics.

Diagnoses:1- Microscopic examination (thick and thin blood smear), **2-**Polymerase chain reaction (PCR), **3-** Complete blood count (CBC), **4-** A blood glucose test.

Diagnostic feature and Morphology: The morphological characteristics (size, shape and appearance) of blood stages are characteristic for each *Plasmodium* spp.



Four different malaria parasite species and five their live-stage

Malarial parasites form four developmental stages in vertebrate host: **Hepaticschizonts→ Intraerythrocytic Trophozoites→ Schizonts** and **→Gamonts,** and three developmental stages in mosquitoes (**Ookinetes**, **Oocysts** and **Sporozoites**).

Early trophozoite (ring form): One red nucleus on the ring-like light blue cytoplasm; single infection in a cell. Infected RBC like normal RBCs.

Late trophozoite: It is irregular shape like amoeboid form with pseudopodia; within cytoplasm, brown pigment granules (malarial pigment-haemozoin) appear. Infected RBCs are pale in color, and have schuffner's dots in it (fine red granules).

Immature schizont: Oval in shape, nucleus divided into 2-4 or more, malarial pigment begins to concentrate in a mass.

Mature schizont: Nucleus divided into 12-24; and cytoplasm also divided, each nucleus surrounded by a portion of cytoplasm to form merozoites, malarial pigment clumped.

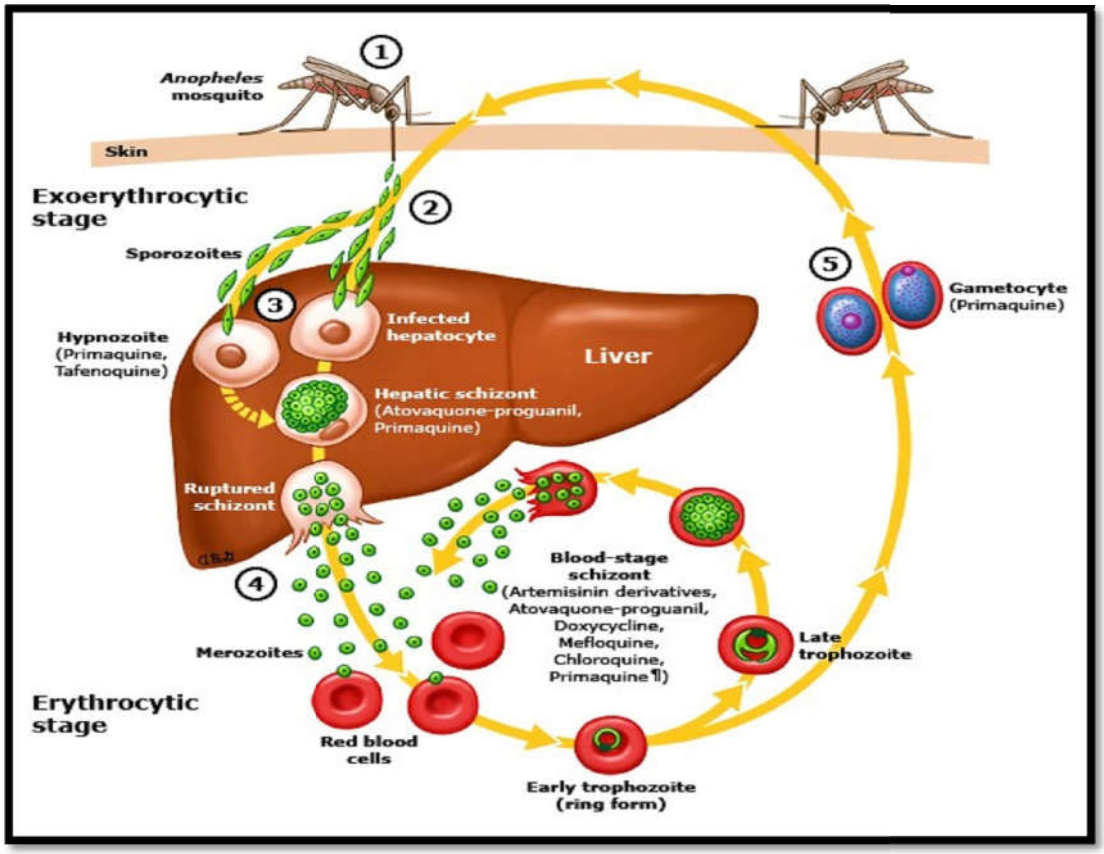
Male gametocyte (Microgametocyte): Oval in shape; one loose nucleus in center of it; malarial pigments diffuse.

Female gametocyte (Macrogametocytes): Oval in shape; one compact nucleus not in center of it.

Clinical signs and Pathogenicity: All the clinical symptoms associated with malaria are caused by the asexual erythrocytic or blood stage parasites. The attacks occur every second day with the "**Tertian**" parasites (*P. falciparum*, *P. vivax*, and *P. ovale*) and every third day with the "**Quatrain**" parasite (*P. malariae*)as uncomplicated

malaria. Fever, chills, sweats, headaches, nausea and vomiting, body aches, general malaise. While the manifestations of severe malaria include the following: Cerebral malaria, with abnormal behavior, impairment of consciousness, seizures, coma, or other neurologic abnormalities which is associated with high mortality, severe anemia, acute respiratory distress syndrome (ARDS), low blood pressure, acute kidney injury.

Life cycle:



Life cycle of *Plasmodium* spp.

(1) *Plasmodium*-infected *Anopheles* mosquito bites a human and transmits sporozoites into the bloodstream. **(2)** Sporozoites migrate through the blood to the liver where they

invade hepatocytes and divide to form multinucleated schizonts (pre-erythrocytic stage).

(3) Hypnozoites are a quiescent stage in the liver that exist only in the setting of *P. vivax* and *P. ovale* infection. This liver stage does not cause clinical symptoms, but with reactivation and release into the circulation, late-onset or relapsed disease can occur up to many months after initial infection.

(4) The schizonts rupture and release merozoites into the circulation where they invade red blood cells. Within red cells, merozoites mature from ring forms to trophozoites to multinucleated schizonts (erythrocytic stage).

(5) Some merozoites differentiate into male or female gametocytes. These cells are ingested by the *Anopheles* mosquito and mature in the mid gut, where sporozoites develop and migrate to the salivary glands of the mosquito. The mosquito completes the cycle of transmission by biting another host.

PLATYHELMINTHES

CHARACTERISTICS OF PLATYHELMINTHES

Platyhelminthes have the following important characteristics:

1. They are triploblastic, a coelomate, and bilaterally symmetrical.

2. They may be free-living or parasites.

3. The body has a soft covering with or without cilia.

4. Their body is dorsoventrally flattened without any segments and appears like a leaf.

5. They are devoid of the anus and circulatory system but have a mouth.

6. They respire by simple diffusion through the body surface.

7. They have an organ level of organization.

8. They do not have a digestive tract.

9. The space between the body wall and organs is filled with connective tissue parenchyma which helps in transporting the food material.

10. They are hermaphrodites, i.e., both male and female organs are present in the same body.

11. They reproduce sexually by fusion of gametes and asexually by regeneration by fission and regeneration. Fertilization is internal.

12. The life cycle is complicated with one or more larval stages.

13. They possess the quality of regeneration.

14. The flame cells help in excretion and osmoregulation.

15. The nervous system comprises the brain and two longitudinal nerve cords arranged in a ladder like fashion.

Fasciola hepatica

Kingdom: Animalia

Phylum: Platyhelminthes

Order: Plagiorchiida

Family: Fasciolidae

Genus: *Fasciola*

Species*: F. hepatica*

Adult *Fasciola hepatica* specimen

Name of Diseases : A disease known as Fascioliasis in mammals that are its final host. Known as liver flukes.

Intermediate And Final Host And Life Cycle: *Fasciola hepatica* are mainly parasites of domestic and wild ruminants (most commonly, sheep, cattle, and goats; also, camels, sardines, and buffaloes). Infection occasionally occurs in anomalous and non-

ruminant herbivores, including horses, lagomorphs, macropods, and rodents. Detection of *Fasciola spp.* Eggs in the feces of carnivores likely represent a pseudo-passage after consumption of contaminated liver.. Snail intermediate hosts of *Fasciola spp.* It is in the family Lymnaeidae, especially species in the genera *Lymnaea*, *Galba, Fossaria*, and *Pseudosuccinea*.

Life cycle :

1) Unembroynated eggs are passed in feces.

2) Eggs become embryonated in water.

3) Miracidia hatch from eggs, seek out snail intermediate host.

4) Miracidia penetrate snail intermediate host.

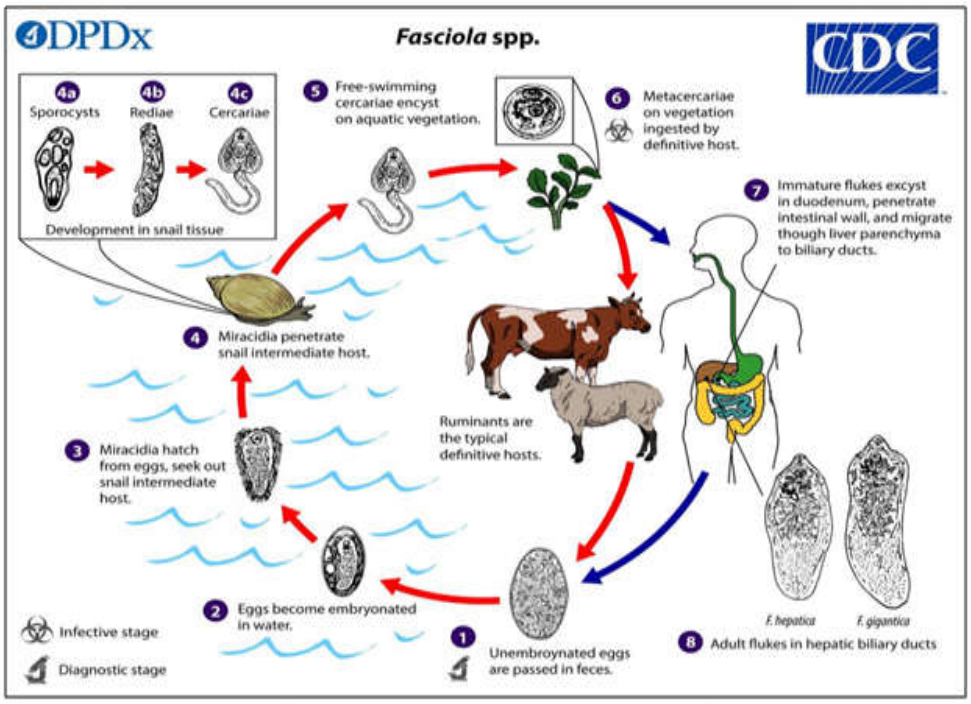
Development in snail tissue : Sporocysts , Rediae , Cercaria

5) Free-swimming cercaria encyst on aquatic vegetation.

6) Metacercaria on vegetation ingested by definitive host.

7) Immature flukes excyst in duodenum, penetrate intestinal wall, and migrate though liver parenchyma to biliary ducts.

8) Adult flukes in hepatic biliary ducts



life cycle of *fascipla hepatica*

Geographical Distribution: *Fasciola hepatica* is found on all inhabited continents, in more than 70 countries, mainly where sheep or cattle are raised. and Latin America (eg, Bolivia and Peru), the Caribbean, Asia, Africa and, rarely, Australia

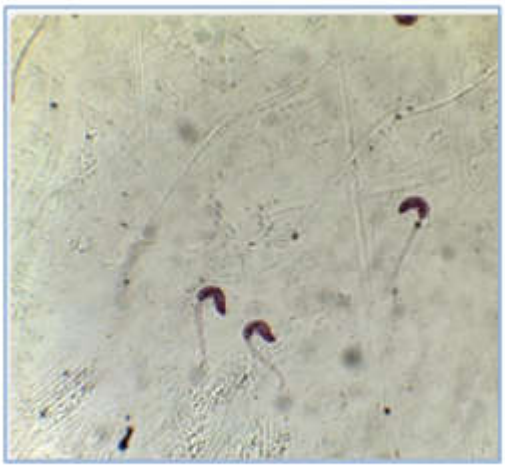
Site Infection: The liver in particular, the main and the digestive system in general.

Diagnostic features for parasites :

Eggs of *F. hepatica* are oval, measure approximately 140 µ by 75 µ, and have a thin, smooth shell and an operculum (lid) at one end. When passed, each egg contains a clump of cells. It is fairly difficult to distinguish the eggs of *F. hepatica* from those of *F. magna*

egg of *fasciola hepatica*

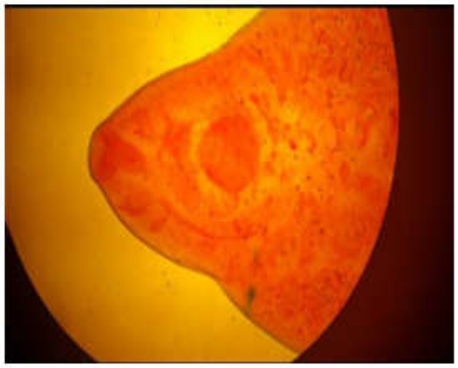
Cercaria This larval stage (cercaria) has the ability to move freely through the water for an approximate period of time of about 10 hours. At the end of these, they lose their tail and generally adhere to the aquatic plants, turning into metacercaria . The latter constitutes the infective form of the definitive host (mammals)



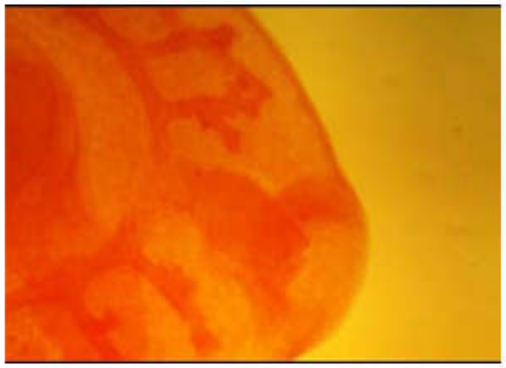
Cercariae of *F.hepatica*

Adult: *F. hepatica* like leaf shape measure up to approximately 30 mm by 13 mm and have distinct "shoulders" immediately behind the cone-shaped anterior part of the body. The tegument of the fluke is covered with sharp spines. The oral and ventral suckers, and many of the internal structures, particularly elements of the alimentary and reproductive systems, can easily be seen microscopically in fixed,

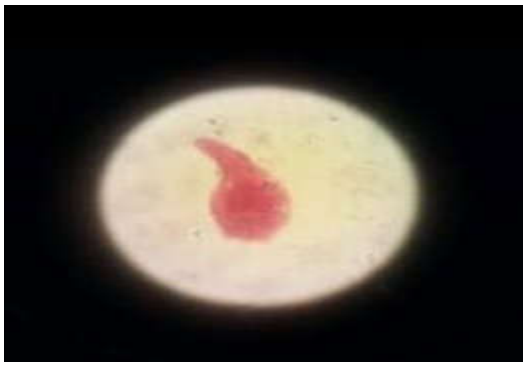
stained specimens. It is hermaphrodite (male and female reproductive organs are present on the same parasite)



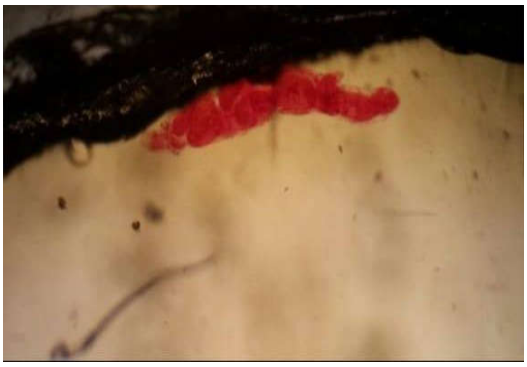
Fasciola hepatica posterior end



Fasciola hepatica anterior end



Fasciola hepatica miracidia



Fasciola hepatica redia

Diagnoses: The infection typically is diagnosed by examining stool (fecal) specimens under a microscope. The diagnosis is confirmed if *Fasciola* eggs are seen. More than one specimen may need to be examined to find the parasite. Certain types of blood tests also may be helpful for diagnosing *Fasciola* infection.

Clinical signs:

In the animal : Acute and sub acute disease is more common in sheep and camels and is often fatal. Traumatic hepatitis occurs when immature flukes migrate through liver tissue before entering and

remaining in the bile ducts. Anemia, submandibular edema, weight loss, and decreased milk production can occur.

Pathogenicity: Although low levels of infection may not be an apparent problem, in endemic areas of the world *F. hepatica* is a major cause of economic loss to cattle producers, both beef and dairy, and to the sheep industry. In cattle the disease is usually chronic – associated with adult flukes in the biliary system and the resulting hepatic fibrosis - and causes production losses as well as clinical disease. In sheep, liver fluke disease can be either acute – associated with the damage caused by the immature flukes migrating through the liver parenchyma – or chronic. Acute *F. hepatica* infection may pre-dispose sheep to clostridial infection, particularly "black disease" *Clostridium novyi*.

Fasciola gigantica

Kingdom: Animalia

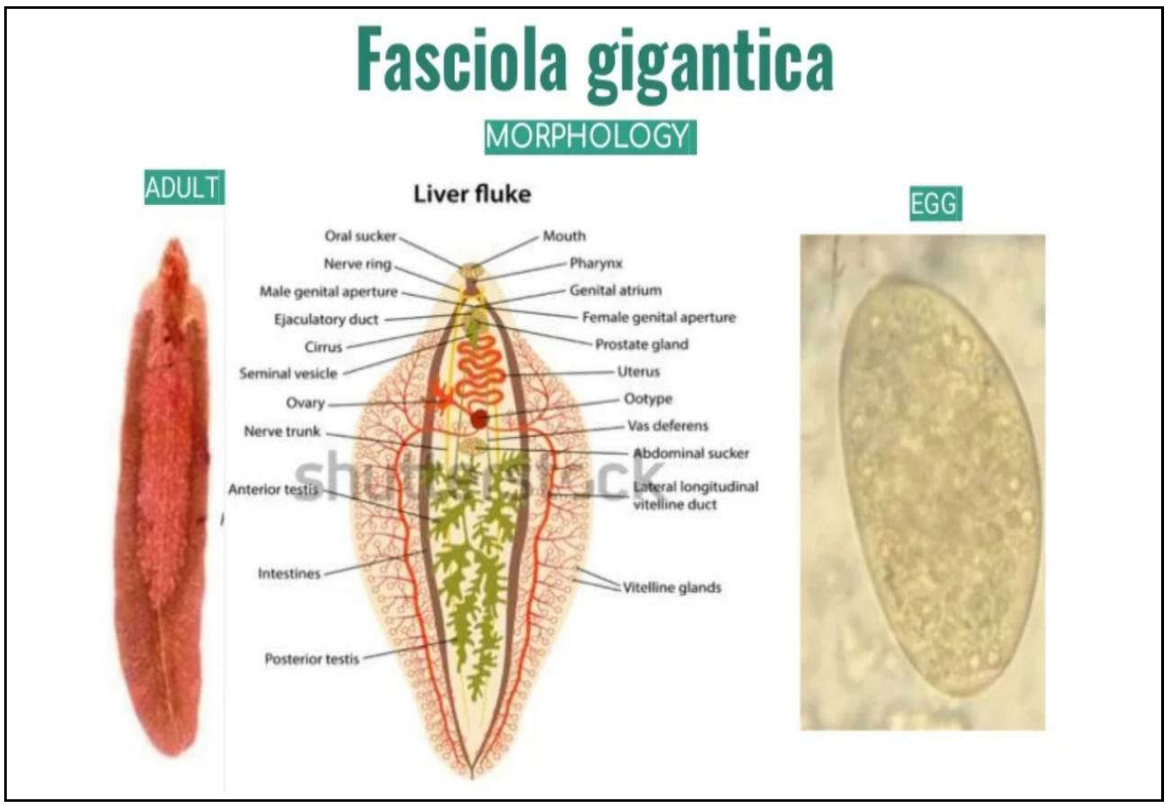
Phylum: Platyhelminthes

Order: Plagiorchiida

Family: Fasciolidae

Genus: *Fasciola*

Species: *F. gigantica*



Fasciola gigantica

Also known as the "common liver fluke" and "giant liver fluke," *Fasciola gigantica*, is one of two *Fasciola* trematoda species to infect the livers of both animals and humans.

Name of Diseases : Fascioliasis is one of the world wide parasitic disease (common in ruminants)sheep, goat, cattle, buffaloes, camels swine, horses, donkeys and rabbits.

Intermediate And Final Host And Life Cycle :

Intermediate Hosts: Egyptian freshwater snails (*Radix natalensis*), European ear snail (*Radix auricularia*), in Iraq (*Lymena arcularia*), while, the final hosts was wet environments. African buffalo, Domestic cattle , Domestic goats, Domestic pigs, sheep , Wild boar and Humans.

Life Cycle :

1-The habitat of *F. gigantica* changes with the stage of its life cycle, which is approximately 17 weeks.

2- Unembroynated eggs are passed in faeces and embryonated in freshwater over the course of approximately 2 weeks; subsequently, they mature and release miracidia.

3- Miracidia seek out snails, which serve as the intermediate host, and undergo several developmental stages (sporocyst → rediae → cercaria) before departing from the snail and become free-swimming cercaria.

4- Humans and animals become infected by ingesting the infective *Fasciola* larvae (metacercaria) through contaminated vegetation or water; the duodenum is the primary location where metacercaria penetrate through the intestinal wall into the peritoneal cavity of the definitive host.

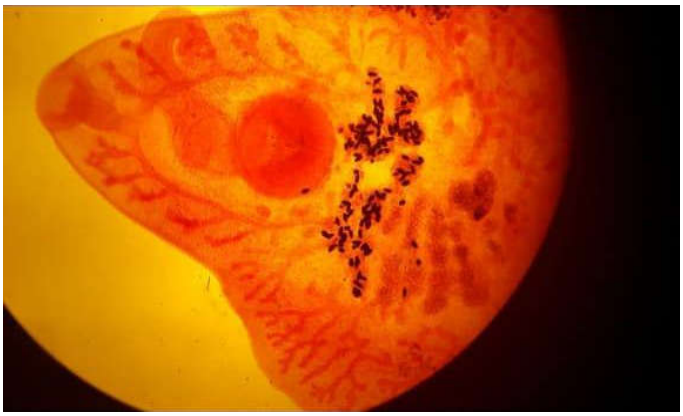
5- After approximately 7-8 weeks, metacercaria migrate through the parenchyma of the liver into the biliary ducts, where maturation into adult flukes occurs (occurs over approximately 3 to 4 months).

6- Adult flukes can survive in their definitive host for several years.

Geographical Distribution: Metacercaria are found in the waters of tropical and subtropical locations of the Middle East, Africa, Europe, South and Southeast Asia, and Hawaii of the United States.

Site infection: The liver in particular, the main and the digestive system in general.

Diagnostic features for parasites: *Fasciola gigantica* very rarely infects humans, the life cycle, transmission, morphology, clinical representation and treatment of the *F. gigantica* trematodes are very similar to those of *Fasciola hepatica*. An adult can grow to 75 mm in length. With the use of a scanning electron microscope the surface of *F. gigantica* appears very rough due to abundant microscopic spines and surface folding. Spikes range from 30 μm to 58 μm and have serrated edges with anywhere from 16 to 20 sharp points. *Fasciola gigantica* also has three different types of surface papillae which are used as sensory receptors. The eggs of *F. gigantica* can reach sizes of 0.2 mm in length.



Fasciola gigantica anterior end

Diagnoses: The infection typically is diagnosed by examining stool (fecal) specimens under a microscope. The diagnosis is confirmed if *Fasciola* eggs are seen. More than one specimen may need to be examined to find the parasite. Certain types of blood tests also may be helpful for diagnosing *Fasciola* infection.

Clinical signs: Acute symptoms include fever, skin rashes, and browning of the hair coat. Chronic symptoms include anemia, jaundice, and continued browning of the hair coat.

Pathogenicity :

- Pale or citric mucous membranes, subcutaneous tissues, abdominal fat, omenta.
- Hepatitis developing into hepatic necrosis and fibrosis.
- Swollen liver with a pale, orange-brown colour.
- Cholangitis leading to fibrosis and calcification of bile ducts.
- Enlarged, dark, friable spleen.

Dicrocoelium spp.

Kingdom: Animalia

Phylum: Platyhelminthes

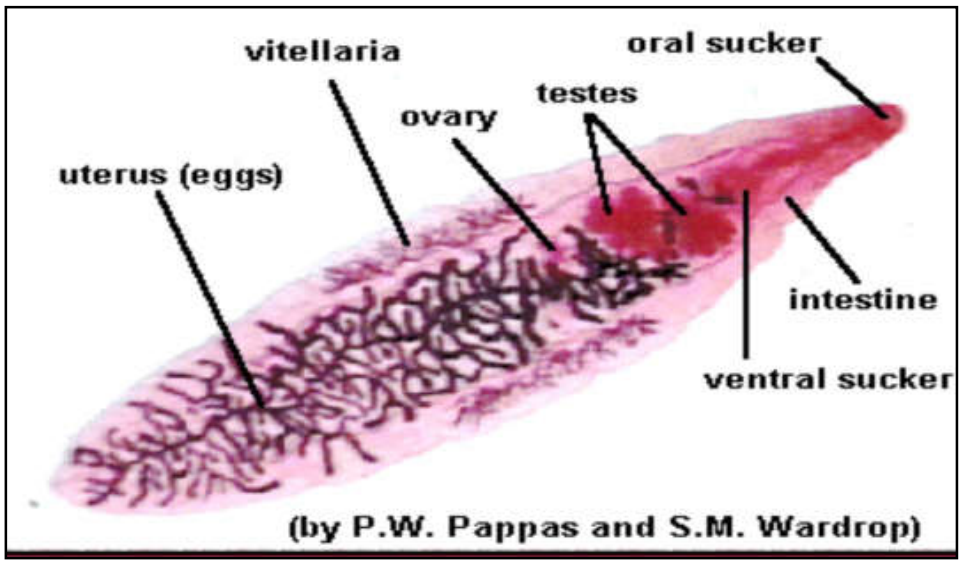
Class : Trematoda

Order: Plagiorchiida

Family: Dicrocoeliidae

Genus: *Dicrocoelium*

Species : *D. Dendriticum , D . Hopes.*



Dicrocoelium dendriticum

Name of Diseases : Dicrocoeliosis commonly known as the 'lancet fluke' or 'small liver fluke'. *D. dendriticum* is a small lancelet trematoda parasite that commonly infects the biliary tract of animals (mainly ruminants) and humans.

Intermediate And Final Host And Life Cycle: First intermediate hosts include a wide variety of air-breathing land snail species, such as those in the genera Cochlicopa (Cionella), Helix, Xerolenta (Helicella), and Zebrina. Many different types of ants can serve as second intermediate hosts, especially members of the genus Formica. The Ruminants, especially cattle and sheep, are the normal definitive hosts of *Dicrocoelium spp*. Apart from humans, aberrant infections have been identified in various non-human primate species and domestic dogs.

Life Cycle :

1- Embryonated eggs containing miracidia are shed in feces of definitive hosts, which are typically ruminants.

2- The eggs are then ingested by the first intermediate host (snail). When the miracidia hatch , they migrate through the gut wall and settle into the adjacent vascular connective tissue, where they become mother sporocysts . The sporocysts migrate to the digestive gland where they give rise to several daughter sporocysts. Inside each daughter sporocyst, cercaria are produced.

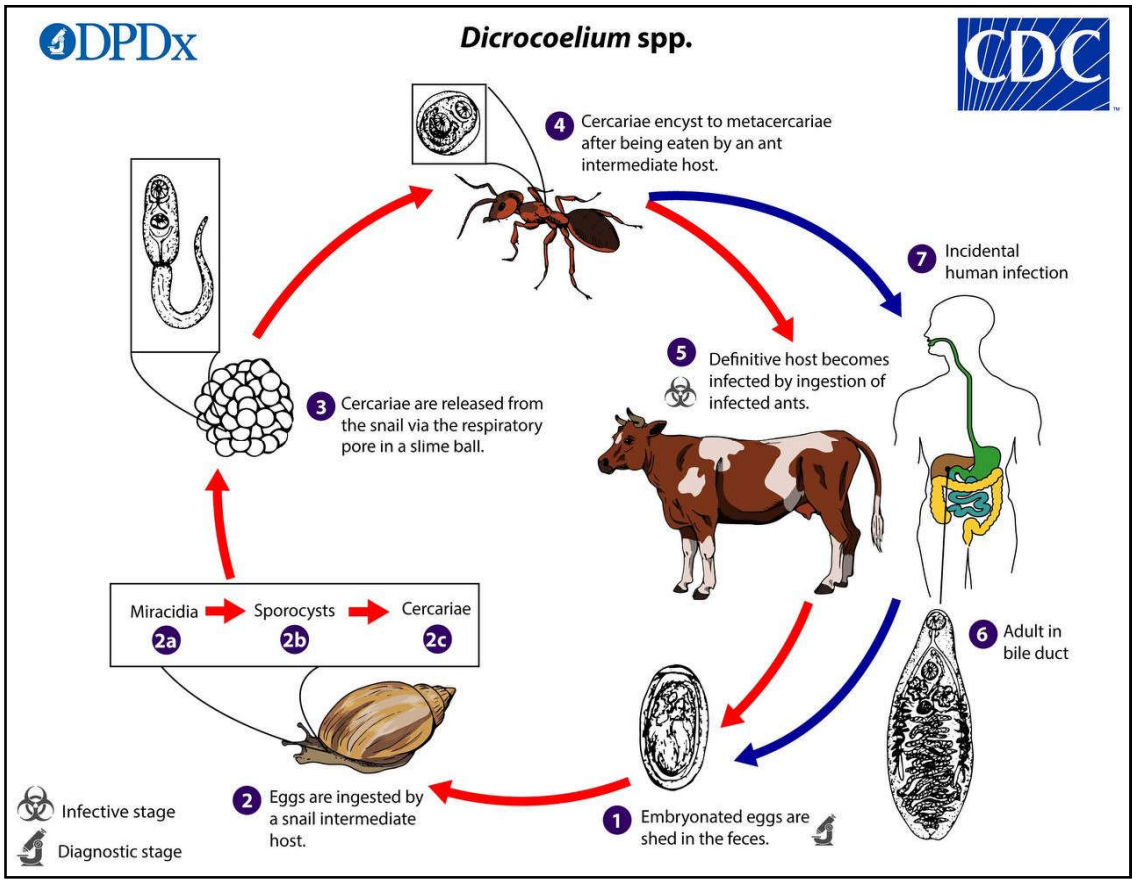
3- Cercaria migrate to the respiration chamber where they are shed in slime ball from the snail

4- After a slime ball is ingested by the second intermediate host (ant), the cercaria become free in the intestine and migrate to the hemocoel where they become metacercaria.

5-When the infected ant is eaten by a suitable definitive host.

6- the metacercaria excyst in the small intestine. The worms migrate to the bile duct where they mature into adults.

7- Humans can serve as definitive hosts after ingesting infected ants (e.g. on contaminated food items).



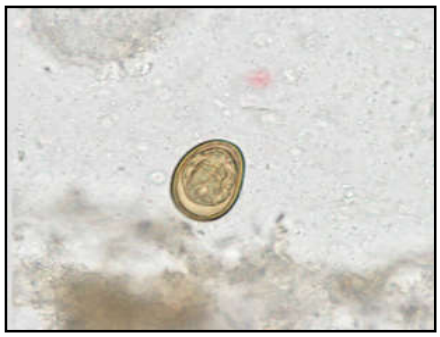
Life cycle *Dicrocoelium dendriticum*

Geographical Distribution: These parasites are believed to be endemic or potentially endemic (naturally occurring) in at least 30 countries. *Dicrocoelium dendriticum* is found throughout Europe, the Middle East, Asia (China, Japan, Vietnam), Africa, North and South America and Australia.

Site Infection: That usually affects the gallbladder and bile ducts of herbivores.

Diagnostic features for parasites :

Eggs of *Dicrocoelium dendriticum* are operculated and measure 35-45 µm long by 20-30 µm wide. The eggs are thick-shelled and usually dark brown in color. Eggs are fully embryonated when shed in feces.



egg of *D .Dendriticum*

Adults of *Dicrocoelium spp*. 5 to 15 mm long and 1.5 to 2.5 mm wide are flattened and taper at both the anterior and posterior ends. The paired testes lie just behind the anteriorly-located ventral sucker (acetabulum). The ovary is small and located behind the testes. Adults reside in the bile ducts of the definitive host.

adult *Dicrocoelium dendriticum*

Diagnose: Traditionally, involves the identification of *Dicrocoelium dendriticum* eggs in the faeces of human or other animal. However, in humans, eggs in the stool may be a result of

ingesting raw infected animal liver and may not in fact indicate Dicrocoeliosis. Therefore, examining bile or duodenal fluid for eggs is a more accurate diagnostic technique in combination with a liver-free diet.

Clinical Signs: Most infected animals show no signs; Severe infections may cause Anemia, Edema, Abdominal discomfort, Weight loss.

Pathogenicity: In more intensive infections, signs may include generalized /abdominal discomfort. Occasional animals that harbor these flukes have subcutaneous masses/enlargements. Decreased liver function in sheep (due to damage from the flukes) can cause toxemia in pregnant ewes and mastitis in lactating ewes when combined with other risk factors. At necropsy a heavily infested animal may have liver abscesses and thickened bile ducts.

Schistosoma spp.

Kingdom: Animalia

Phylum: Platyhelminthes

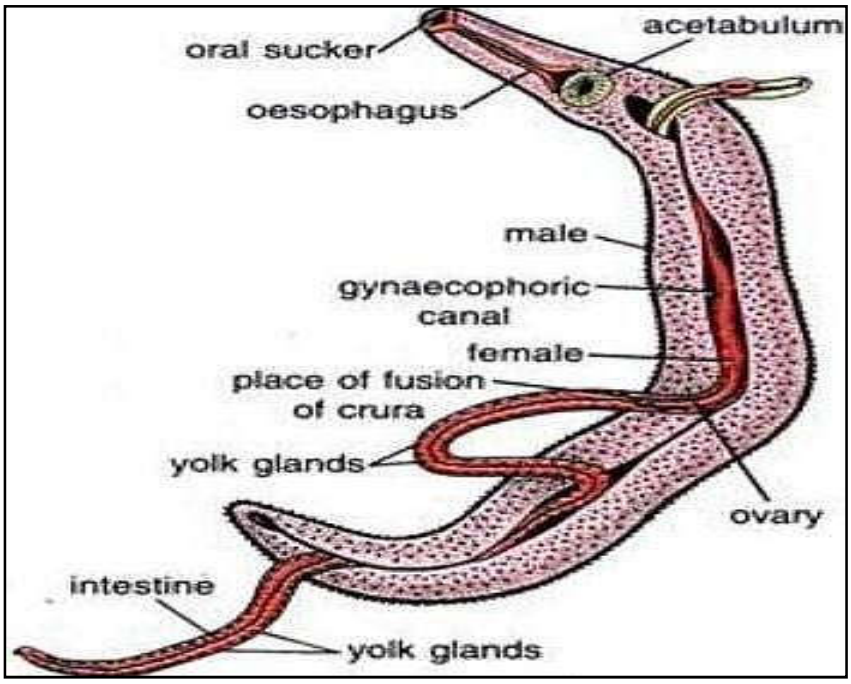
Order: Diplostomida

Family: Schistosomatidae

Subfamily: Schistosomatinae

Genus: *Schistosoma*

Species : *S . haematobium , S . Japonicum , S. Mansoni*



Schistosoma japonicum

Name of Diseases: A genus of trematodes commonly known as blood flukes' Urinary Schistosomiasis , Intestinal Schistosomiasis (Bilharzia)

Intermediate And Final Host And Life Cycle:

Intermediate host

Schistosoma haematobium→ *Bulinus truncatus*

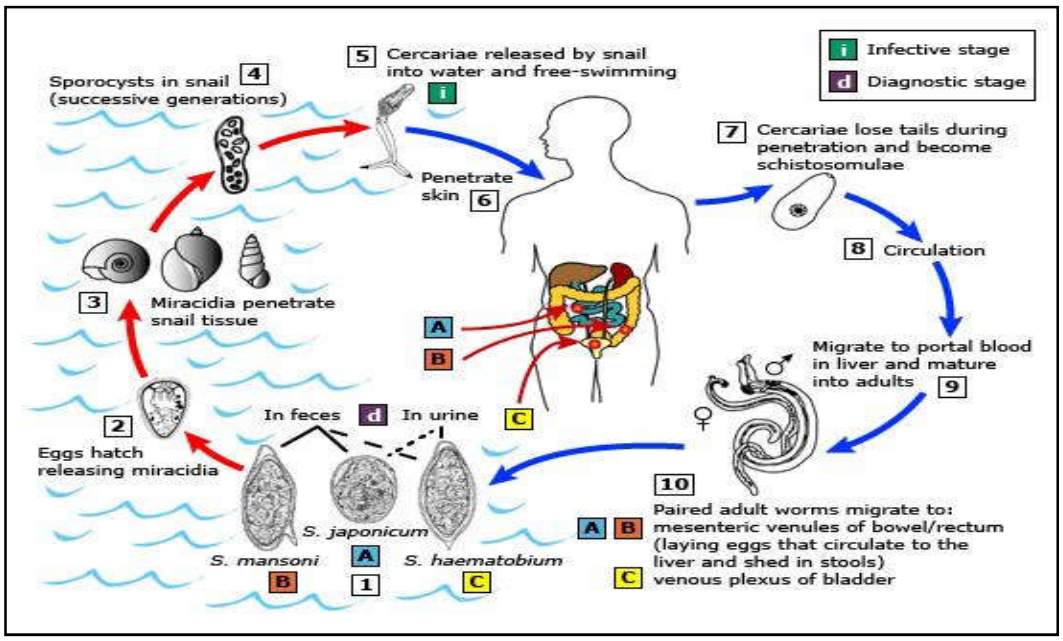
Schistosoma mansoni→ *Biomphlaria alexandria*

S. japonicum→ *Bulinus* spp. and *Lymena* spp.

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

Life Cycle : (1) elimination from the host as eggs in feces or urine (diagnostic stage), (2) hatching of miracidia, (3) infection of species-specific aqueous snail intermediate hosts, (4) proliferation of sporocysts within snails, (5) release of cercaria into water (infective stage), (6) infection of host by skin penetration, (7) development into Schistosomulum, (8) circulation, (9) maturation within portal vasculature, and (10) migration of paired adult worms to target organs. Elimination of *Schistosoma* eggs in either feces or urine depends on whether the adults reside in the mesenteric venues of the bowel/rectum (primarily (A) *Schistosoma japonicum* and (B)

Schistosoma mansoni) or in the venous plexus of the bladder (primarily (C) *Schistosoma haematobium*), respectively.



life cycle of *Schistosoma* spp.

Geographical Distribution:

Schistosoma haematobium - Africa,

Schistosoma mansoni - Africa and America

Schistosoma japonicum - Far East Asia,

Some parts of Africa Epidemiology Approximately 250 million people are infected with Schistosomes and 600 million are at risk.

Site Infection:

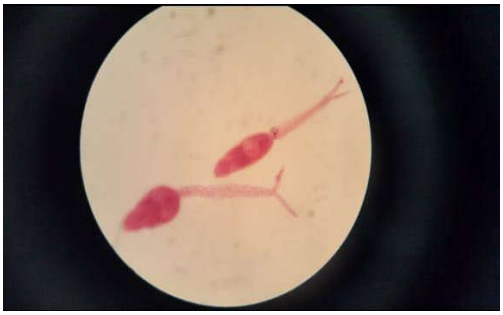
S. japonicum - Superior mesenteric veins draining the small intestine

S. Mansoni - Superior mesenteric veins draining the large intestine

S. Haematobium - Venous plexus of bladder, it can also be found in the rectal venues

Diagnostic features for parasites:

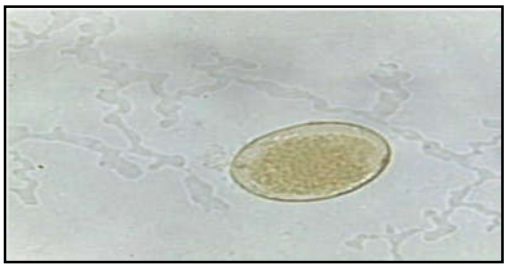
cercaria Man is infected by fork tailed cercaria in fresh water by skin penetration. The cercaria travel through the venous circulation to the heart, lungs and portal circulation. They mature and reach: the mesenteric veins (*S. japonicum and S. mansoni*). The bladder vessels (*S. haematobium*) where they live and ovulate for the duration of the host's life, Schistosoma cercaria (forked tail). Found in fresh water, Penetrate the skin of human upon contact with water containing it.



Cercaria of *Schistosoma* spp.

Schistosoma japonicum

Eggs: The ova of *S. japonicum* are 55-85µm by 40-60 µm. They are large, round and non operculate and have a transparent shell with a minute lateral spine or knob that may be inconspicuous and difficult to see. The eggs of *S. japonicum* frequently have faecal debris

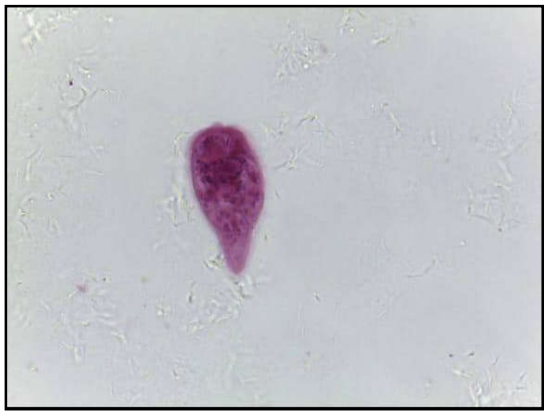


egg of *Schistosoma japonicum*

adhering to the shell that can obscure them.

Miricidium: The miricidium is an ovoid, ciliated, free swimming organism with a functionless digestive tract.

Adult Worms: The worms are yellow or yellow brown in colour. The male measures 12mm by 0.5mm and female measures 20mm by 0.4mm. Both sexes have a strong sucker around the mouth and the tegument of the worms is coated with tiny spines, ridges and sensory organs.



Schistosoma japonicum miracidia

Schistosoma japonicum couple

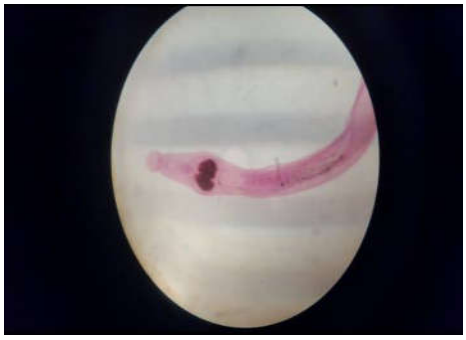
S. haematobium

The eggs of *Schistosoma haematobium* are large (110-170 µm long by 40-70 µm wide) and bear a conspicuous terminal spine. Eggs contain a mature miricidium when shed in urine.

The adult worms are 1–2 cm long with a cylindrical body that features two terminal suckers, a complex tegument, a blind digestive tract, and reproductive organs. The male's body forms a groove or gynecophoric channel, in which it holds the longer and thinner female. As permanently embraced couples, the Schistosomes live

within the perivesical (*S. haematobium*) or mesenteric (other species) venous plexus. Schistosomes feed on blood particles through anaerobic glycolysis.

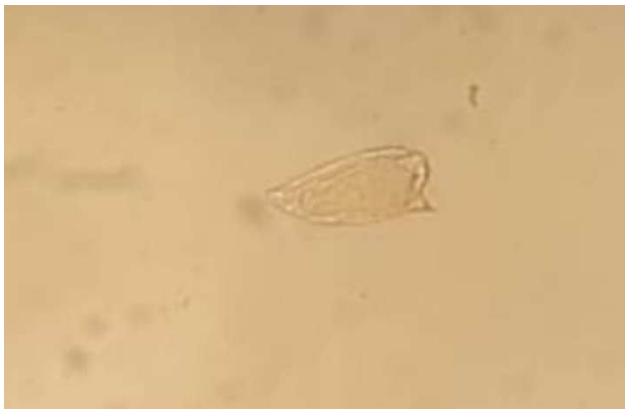
Schistosoma haematobium female and male



Schistosoma haematobium male

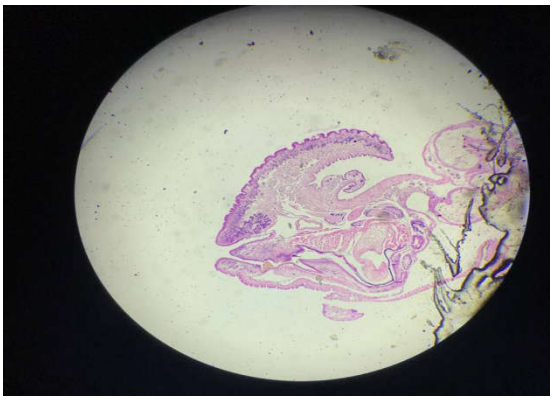
Schistosoma mansoni

The eggs of *Schistosoma mansoni* are large (114 to 180 µm long by 45-70 µm wide) and have a characteristic shape, with a prominent lateral spine near the posterior end. The anterior end is tapered and slightly curved. When the eggs are excreted in stool, they contain a mature miricidium.



Schistosoma mansoni egg

Adults of *Schistosoma mansoni* unlike the flukes, adult *Schistosomes* have the sexes separate, with the female residing in a gynecophoral canal within the male. Male worms are robust, tuberculate and measure 6-12 mm in length. Females are longer (7-17 mm in length) and slender. Adult *S. mansoni* reside in the venous plexuses of the colon and lower ileum and in the portal system of the liver of their host. Sexes are separate (they are diocious).



Schistosoma mansoni sporocyts

Diagnose: Stool, urine samples , Bile stained eggs in stool , ***Schistosoma haematobium*** - Terminal spine, ***Schistosoma mansoni*** - Lateral spine, ***Schistosoma japonicum*** - Lateral knob

clinical signs: Hepato splenomegaly, ascites, muscle atrophy, anemia, diarrhea hemorrhage from the gastrointestinal tract

Pathogenicity :*S. mansoni* and *S. japonicum* includes: Katayama fever, periportal fibrosis, portal hypertension, and embolic egg granulomas in brain or spinal cord.

S. haematobium includes: Haematuria, calcification, squamous cell carcinoma, and occasional embolic egg granulomas in brain or spinal cord.

Clonorchis spp.

Kingdom: Animalia

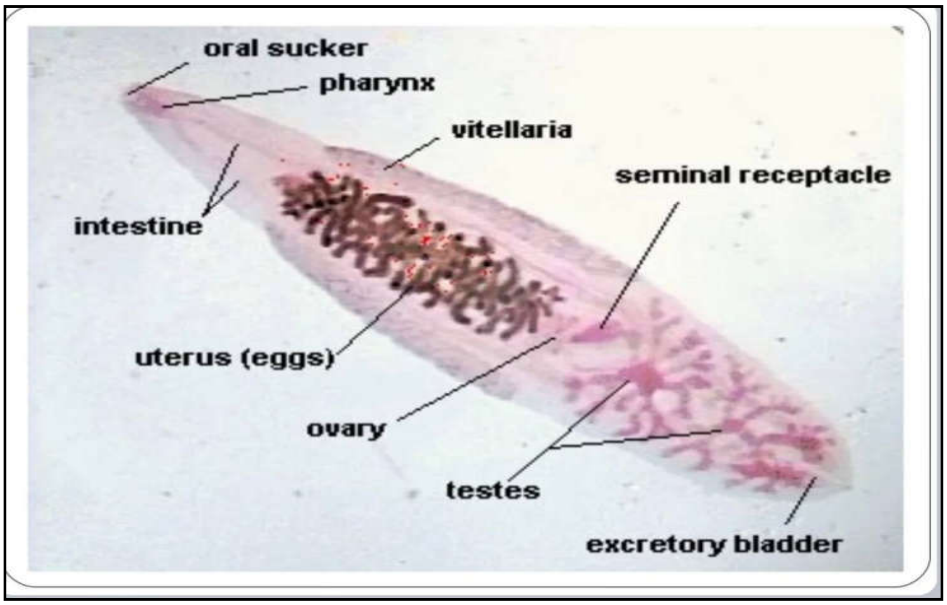
Phylum: Platyhelminthes

Order: Plagiorchiida

Family: Opisthorchiidae

Genus: *Clonorchis*

Species: *Clonorchis sinensis*



Clonorchis sinensis adult fluke

Name of Diseases: cause disease Clonorchisasis.

Intermediate And Final Host And Life Cycle: The Intermediate Hosts was first Snails: *Parafossarulusmanchouricus*, and second Fish : 12 species of fish are capable of passing the

infection to human. The final or Definitive was mammals Humans are the principal definitive host when eating fishes, but dogs and other fish-eating canines act as reservoir hosts.

Life Cycle :

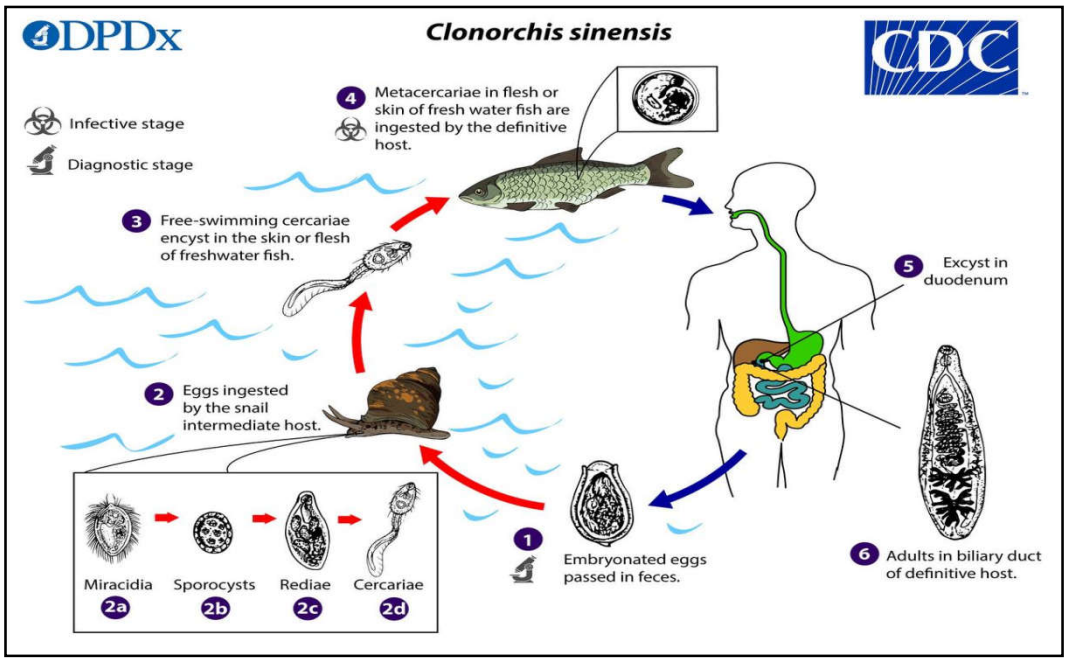
1-The eggs produced by the worms pass through the bile duct and are excreted in the stool.

2- If the eggs reach a body of fresh water (small ponds, streams and rivers, submerged rice fields, and reservoirs), they are ingested by the snails, which act as the primary intermediate host.

3- Asexual reproduction in snails results in the release of thousands of cercaria daily one to two months after the snail has been infected. Free-swimming cercaria penetrate the tissues of freshwater fish, transforming into fully infectious metacercaria under the skin of the fish or in the muscle after 21 days.

4-Other animals that feed fish Other animals that feed fish become infected by eating raw or undercooked whole freshwater fish (salted, pickled or smoked) that contain metacercaria.

5- After ingestion, metacercaria is excreted into the bile duct through an ampulla it takes maturity to puberty



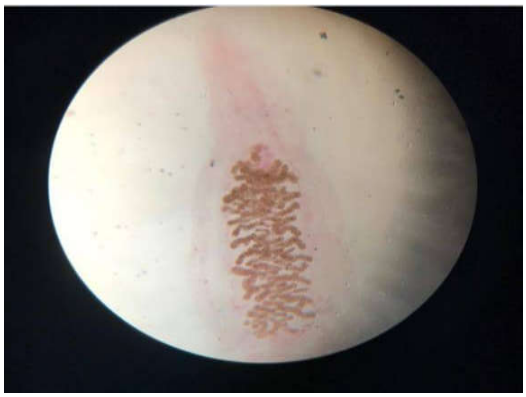
life cycle of *Clonorchis sinensis*

Geographical Distribution: This parasite infects humans and mammals by eating fish in the Far East countries such as China, Japan, Korea and Vietnam.

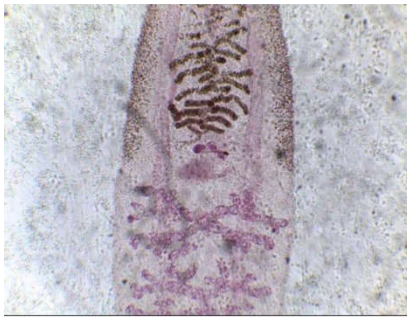
Site Infection: The adult worms live in the terminal branches of the bile ducts.

Diagnostic features for parasites: Adult Its shape is flat, elongated, and soft to the touch (without thorns), with a bright gray color, and when it is yellow, it turns golden and contains two mouthfuls and another abdominal one located at the end of the first five of the body. The digestive system consists of a spherical pharynx that leads to two wide intestinal rings and ends with a closed end near the back of the body. The male reproductive system consists of two lobed testicles, one ahead of the other and located in the back of the body. The female reproductive system consists of a slightly lobed and small ovary located in the last third of the body. As for the uterus, it

is slightly curved, located in the middle of the body, and opens into the common genital opening. Adult worm, elongated worm, with the size 10-15×3-5 mm

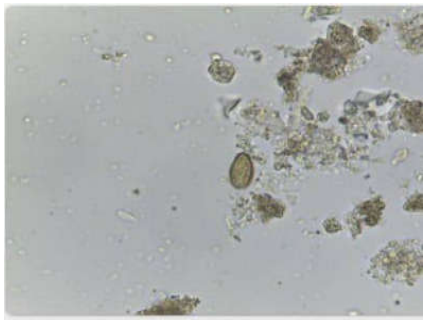


adult of *Clonorchis sinesis*



Clonorchis sinensis

The eggs Small, 26-30 x 15-17 um are oval in shape with a thick brownish-yellow shell. We also notice two edges of the egg from the top and a small scar from the bottom, which is a diagnostic characteristic.



egg of *Clonorchis sinensis*

Diagnoses: Depends on the presence of eggs of these worms when direct microscopic examination of stool smears or using concentration methods Examination of the contents of the duodenum ten and yellow

Clinical Signs: Gastrointestinal symptoms, Hepatomegaly, neurasthenia, Cholangitis, cholecystitis.

Pathogenicity: Enlargement of the liver, necrosis and cirrhosis of its visceral cells Thickening of the bile ducts and sometimes causing perforation of the walls of these ducts Eggs collected in the bile sac become a center for the formation of gallstones.

Paragonimus spp.

Kingdom: Animalia

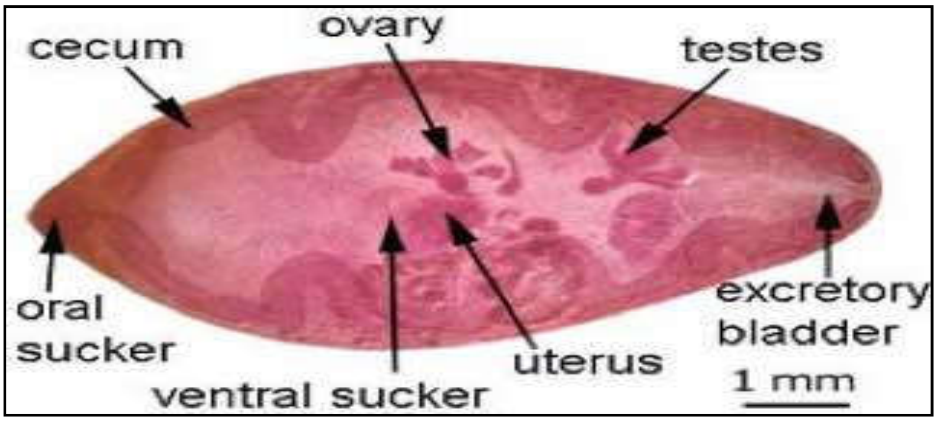
Phylum: Platyhelminthes

Order: Plagiorchiida

Family: Paragonimidae

Genus: *Paragonimus*

Species: *P. westermanii , P. kelicotti*



Paragonimus westermanii

Also known as the Oriental lung fluke (the lung distome) pinkish-brown colour and bean shaped trematodes

Name of Diseases: Paragonomiasis

Intermediate And Final Host And Life Cycle:

Intermediate Host: First intermediate host: A fresh-water snail of the genus *Melania* spp., while, second intermediate host: A fresh-water crayfish or a crab.

Definitive Hosts: Man and Domestic animals (usually host in Asia are the tiger & leopard)

Life Cycle :

1. In the human host, Unembroynated eggs pass out of the body in sputum, or they are swallowed and passed with stool.

2. In the external environment, eggs become embryonated, and miracidia hatch.

3. Miracidia seek a snail (first intermediate host), and penetrate its soft tissues.

4. Inside the snail, miracidia develop into sporocysts, then rediae, and then many cercaria, which emerge from the snail.

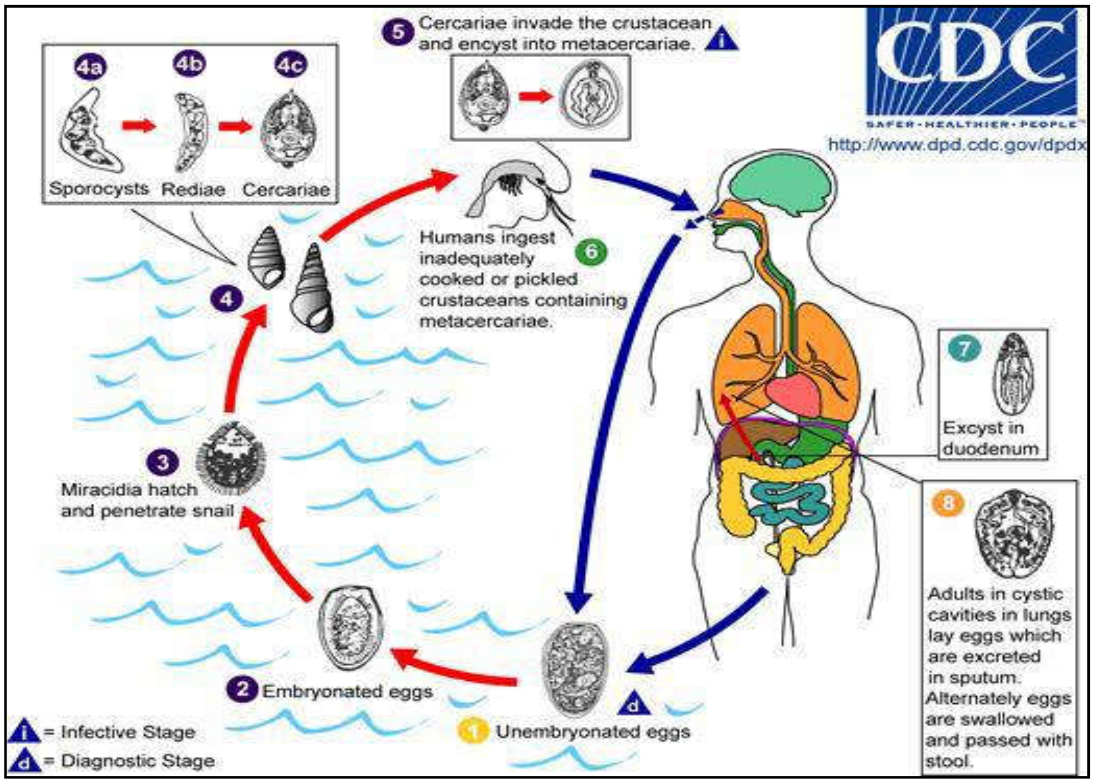
5. Cercaria invade a crustacean such as a crab or crayfish (2nd intermediate host), where they encyst and become metacercaria (the infective stage for mammalian hosts).

6. Humans are infected with *P. westermanii* by eating inadequately cooked or pickled freshwater crab or crayfish that contain metacercaria.

7. The metacercaria excyst in the duodenum.

8. They then penetrate the intestinal wall and move into the peritoneal cavity, then through the abdominal wall and diaphragm

into the lungs; there, they become encapsulated and develop into adults, which produce eggs. The eggs exit the body in sputum that is coughed up and spit out or swallowed and passed in stool. The worms can also reach other organs and tissues, but in such sites, the life cycle cannot be completed because the eggs cannot exit the body.



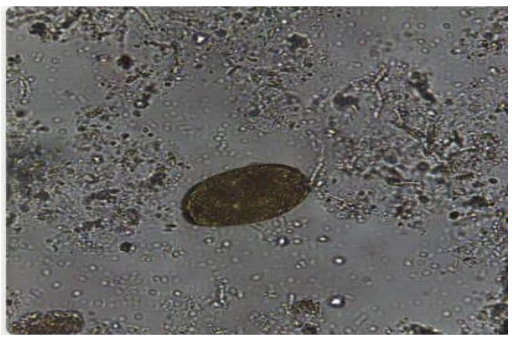
Life cycle *of Paragonimus westermanii*

Geographical Distribution: It is endemic in China, Korea, Japan, the Philippines, and Taiwan Japan, Korea, Formosa, China, Manchuria, the Philippine Islands and India Infection is also found in parts of tropical West Africa, from the Congo and Nigeria, especially from Southern Cameron Rare in the US.

Site Infection: adult worm live in respiratory tract (lung).

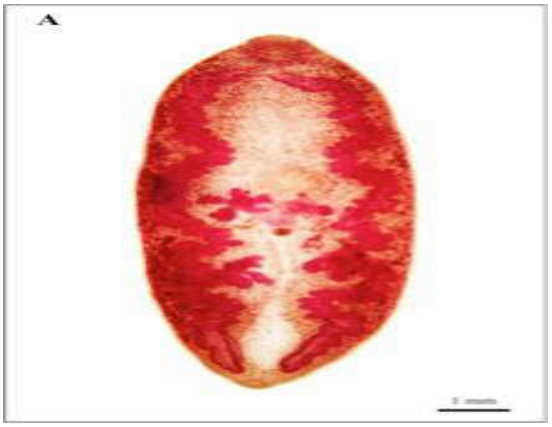
Diagnostic features for parasites:

Egg Golden brown in colour, oval in shape flat end opercula 80-120 µm by 50- 60µm in size , Contain an un-segmented ovum surrounded by yolk cell.



egg of *Paragonimus westermanii*

Adult (looks like a coffee bean), size: - 10 mm long, 5 mm wide and 4 mm thick. Adult worms live in the lungs, usually in pairs in cystic spaces communicating with the bronchi. Its front end is slightly wider than the rear end. The abdominal lollipop is located near the middle of the body. , The life span of an adult worm is about 6 to 7 years • The excretory vesicle is large and extends from the posterior end to the anterior region, dividing the body into two equal halves.



adult *Paragonimus westermanii*

Diagnoses: The infection is usually diagnosed by identification of *Paragonimus* eggs in sputum. The eggs are sometimes found in stool samples (coughed-up eggs are swallowed). A tissue biopsy is sometimes performed to look for eggs in a tissue specimen.

Clinical Signs: Fever, Diarrhea, Abdominal pain, Itching and hives, Cough , Shortness of breath

Pathogenicity: Once the parasite is in the lung or another organ, the worm stimulates an inflammatory response that eventually coats tissue. If worms enter the CSF of the spinal cord it can result in partial or total paralysis. There have also been fatal cases of Paragonomiasis by infection of the heart. Cerebral cases result in cerebral cysticercosis (condition in which fluid-filled cysts surrounding the worm are present)

CESTODA

CHARACTERISTICS OF THECESTODA

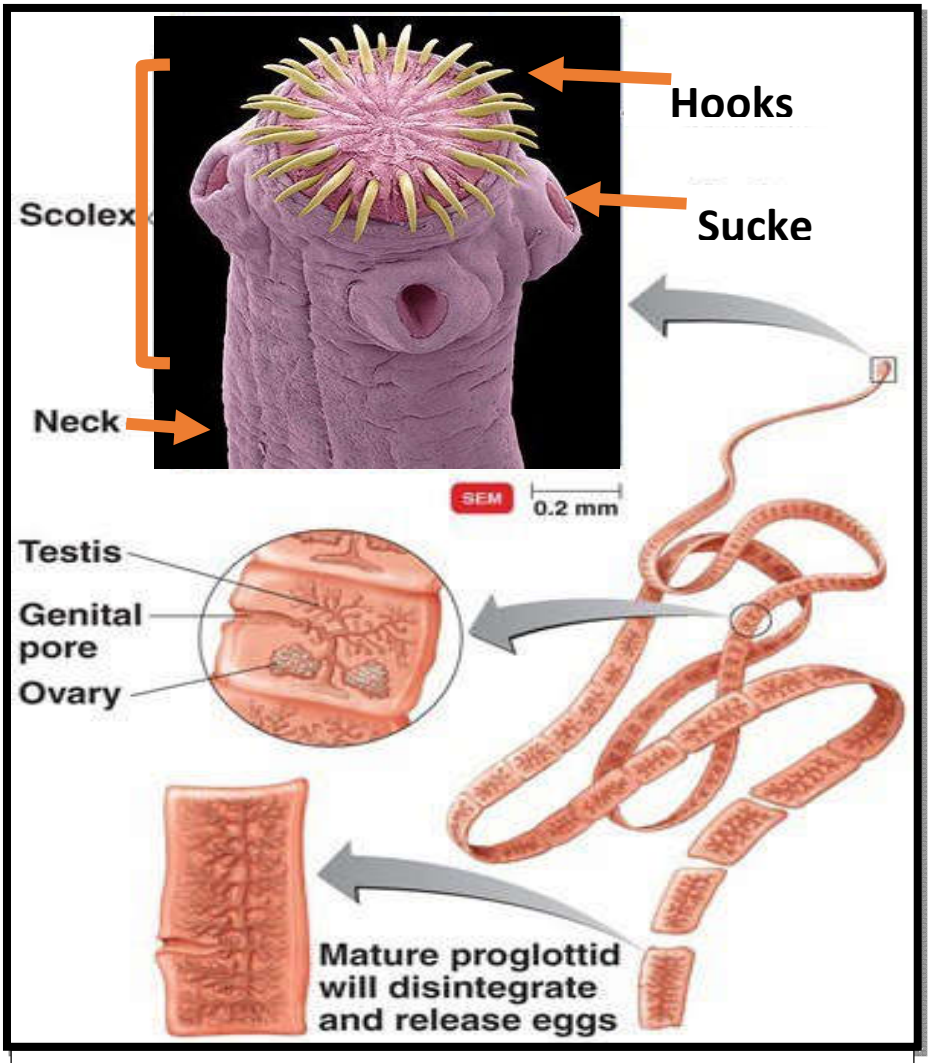
1- Majority are long, segmented and tape-like are called tapeworms.
2- Dorso-ventrally flattened.
3- Size varies from a few mm to several meters.
4- Adult worms are found in the intestinal canal of man and animals.
5- "head" or scolex is provided with suckers and sometimes with hooks as organs of attachment.
6- There are 3 regions in an adult worm:
    - Head: scolex
    - Neck
    - Strobila (body or trunk): Consist of a series of segment called proglottids.
7- Sexes are not separate.
8- Body cavity is absent.
9- Alimentary canal is absent.
10- Excretory and nervous systems are present.
11- Reproductive system is present and complete in each segment.
    - According to maturity of reproductive organs, three types of segments of the strobila can be recognized:
        - Immature: male and female organs are not differentiated(after neck).
        - Mature: male and female organs have become differentiated (male organs appear first).
        - Gravid: uteri are filled with eggs.

Order Cyclophyllidea Characteristics:

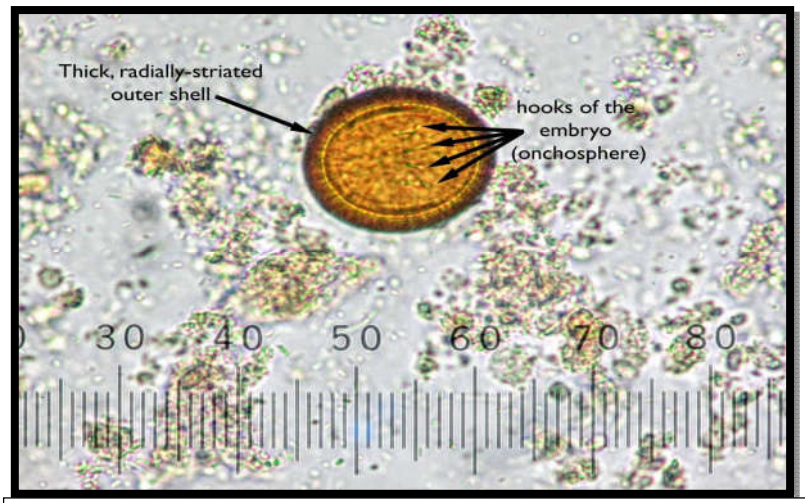
1- Large or small worms consisting of chains of segments.
2- Scolex is quadrate with four cup-like round suckers.
3- An apical rostellum with hooklets may be present.
4- Common genital pore is marginal (on lateral side of segment).
5- No uterine opening for the exit of eggs from the gravid segments.
6- Eggs only escape from the rupture or disintegration of segments.
7- Eggs are not operculated.
8- Oncosphere is never a ciliated embryo.
9- Larval development proceeds in one intermediate host.

Order Pseudophyllidea Characteristics:

1- Large worms consisting of a long chain of segments.
2- "head" has two slit-like sucking grooves called botheria instead of suckers.
3- Genital pores are on the ventral surface of the segment and are not marginal.
4- Uterus opens to the exterior surface through which eggs come out.
5- Eggs are operculated.
6- Larval development proceeds in two intermediate hosts:
    - First larval stage is called procercoid
    - Second larval stage is called plerocercoid.



Typical Diagram of *Taenia* spp. modified from



***Taenia* spp.: Eggs measure 40–60, µm 35–43**

Taenia saginata

Kingdom: Animalia

Phylum: Platyhelminthes

Class: Cestoda

Order: Cyclophyllidea

Family: Taeniidae

Genus: *Taenia*

Species: *T. saginata*

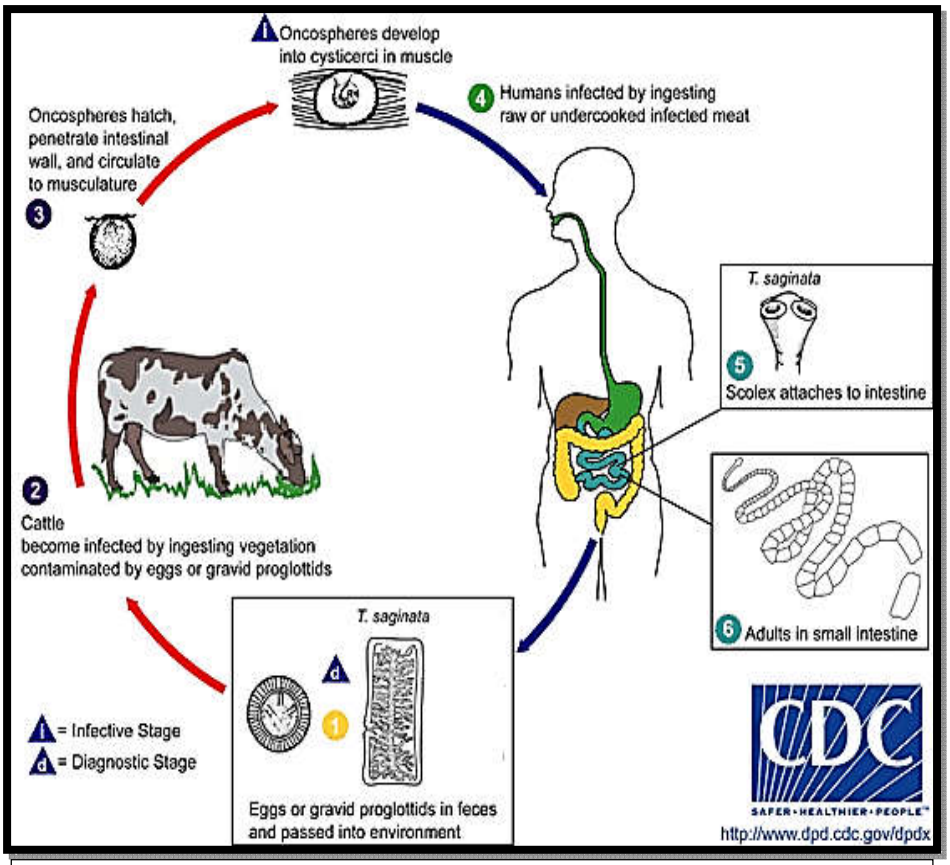
Name of Disease:

- Cysticercosis in cattle: is a tissue infestation caused by larval stage (*Cysticercus bovis*) of the *T. saginata* tapeworm.
- Taeniasis in humans: it is intestinal a parasitic infestation in humans caused by a zoonotic tapeworm.

Intermediate and final host and life cycle:

- Intermediate host: Cattle.
- Final host: human.

Humans are generally infected as a result of eating raw or undercooked beef which contains the infective larvae, called *Cysticercus bovis*. As hermaphrodites, thus, reproduction is by self-fertilization. From humans (final host) embryonated eggs, called oncospheres, are released with faeces and are transmitted to cattle through contaminated fodder. Oncospheres develop inside muscle, liver, and lungs of cattle (Intermediate host).



The life cycle of *Taenia saginata*, the beef tapeworm.

Geographical Distribution: worldwide.

Site of Infection:

- Cattle: in striated musculature with larval stage as *Cysticercus bovis*.
- Human: in small intestinal.

Diagnostic feature for parasite:

The eggs: typical taeniid type ova with a embryonated with six hooklets inside.

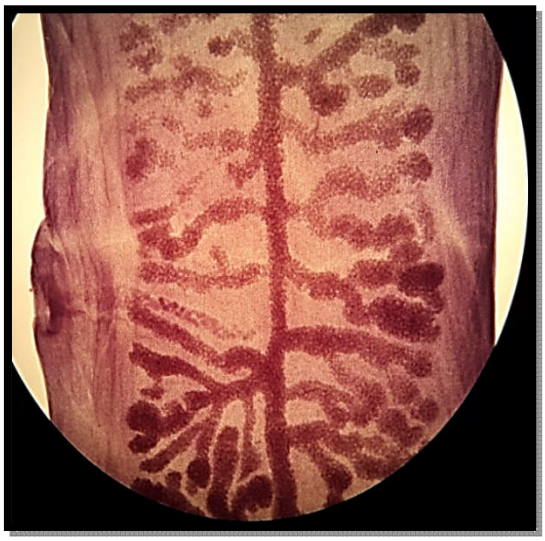
Adult: *T saginata* is the largest of species in the genus *Taenia*. An adult worm is normally 4 to 10 m in length, but can become very large; specimens over 22 m long are reported.

- The gravid proglottid, the uterus contains up to 15 side branches filled with eggs.

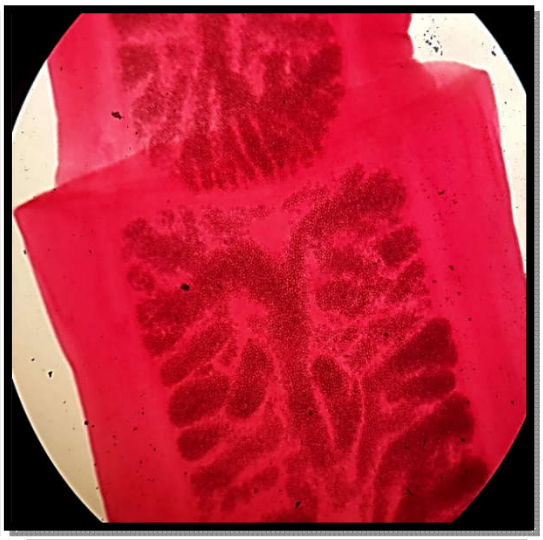
- The scolex has four suckers, but they have no hooks, Lack of hooks.

- *T. saginata*: inside of each mature proglottid is complete male and female reproductive systems, including the uterus, ovary, genital pore, testes, and vitelline gland.

Cysticercus is a single invaginated scolex in a large, fluid-filled cyst, cavity.



Taenia saginata (graved



Taenia saginata (graved

Diagnoses:

The basic diagnosis is done from a faecal examination sample. Feces are examined to find parasite eggs. When found the proglottids in feces identification often requires histological observation of the uterine branches. The uteri of *T. saginata* stem branches from the center to 12 to 20 branches, the branches are less in number and thicker.

-PCR (Polymerase Chine Reactions) detection.

Clinical signs:

- Clinically asymptomatic of Cysticercosis in cattle (intermediate hosts).
- Asymptomatic of Taeniasis in humans, but heavy infection often results in weight loss, abdominal pain, diarrhea, headaches and loss of appetite. Intestinal obstruction in humans.

Pathogenicity:

The larval stage for this tapeworm is a *Cysticercus bovis*. These infective are found in (skeletal and cardiac muscles) of cattle. If heavy infection in the muscle tissue, they can interfere with muscle function, produce pain, and myositis.

Adult: *T. saginata* is a zoonotic tapeworm, can also lead to produce antigens that can cause an allergic reaction in the individual.

Taenia solium

Kingdom: Animalia

Phylum: Platyhelminthes

Class: Cestoda

Order: Cyclophyllidea

Family: Taeniidae

Genus: *Taenia*

Species: *T. solium*

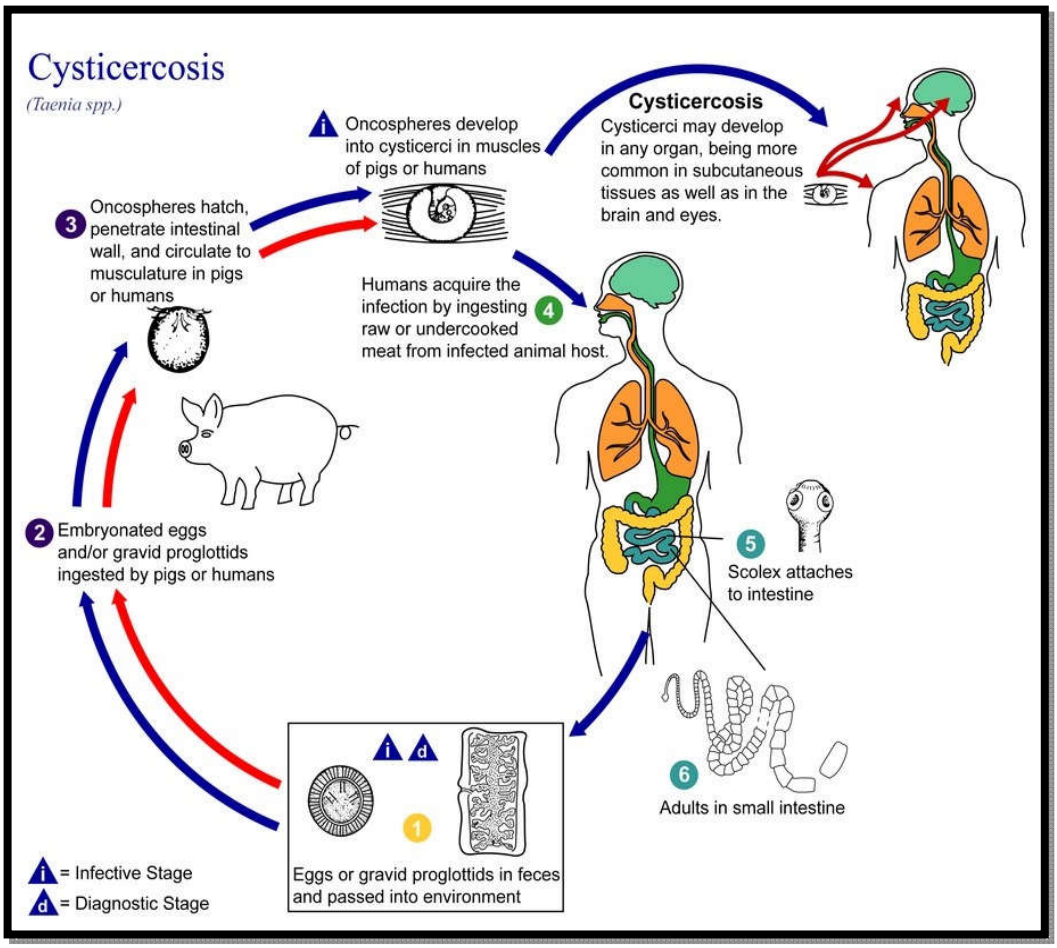
Name of Disease:

- Cysticercosis is an infection caused by the larvae *Cysticercus cellulosa* of the parasite *T. solium*, this infection occurs after a person swallow's tapeworm eggs. These larval cysts infect tissues such as muscle and brain, or other tissue, in pigs.
- Taeniasis in humans.

Intermediate and final host and life cycle:

- Intermediate host: pigs
- Final host: human

It is transmitted to pigs through human feces that contain the parasite eggs (infective stage) and contaminate their fodder. Pigs ingest the eggs, which develop into larvae, then into oncospheres, and ultimately into infective tapeworm cysts, called *Cysticercus*. Humans acquire the cysts through consumption of uncooked or under-cooked pigs and the cysts grow into an adult worm in the small intestine.



Life cycle of *T. solium* red arrows indicate pig; blue arrows human.

Geographical Distribution:

It is found throughout the world and is most common in countries where pigs are eaten.

Site of Infection:

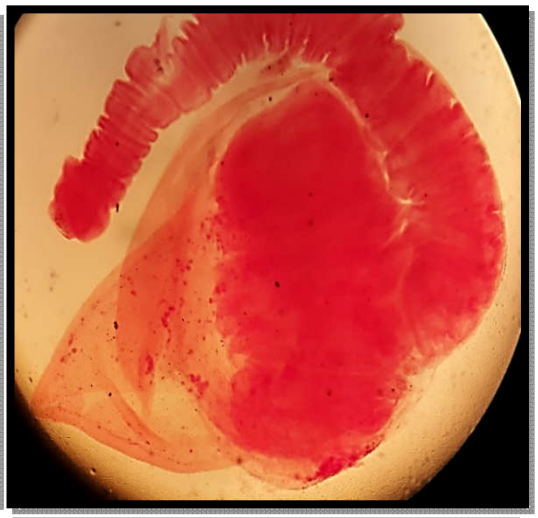
- Pigs: in striated muscular with larval stage as *Cysticercus cellulosa*.
- Human: in small intestinal with adult worm.

Diagnostic feature for parasite:

The eggs: typical taeniid type ova with a embryonated with six hooklets inside.

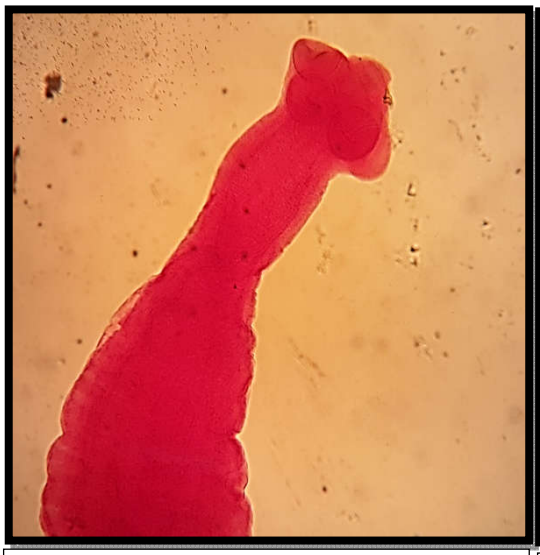
The larval stage: this a cysticercus, or bladder worm, known as *Cysticercus cellulosa,* the cysticercus can develop within their muscles and within nervous tissue such as the brain, eye, and spinal cord. Cystic cavity contains the larval form: scolex is single and invaginated; contains a rostellum, 4 suckers and 22 - 23 hooklets.

Taenia solium (*Cysticercus cellulosa*)

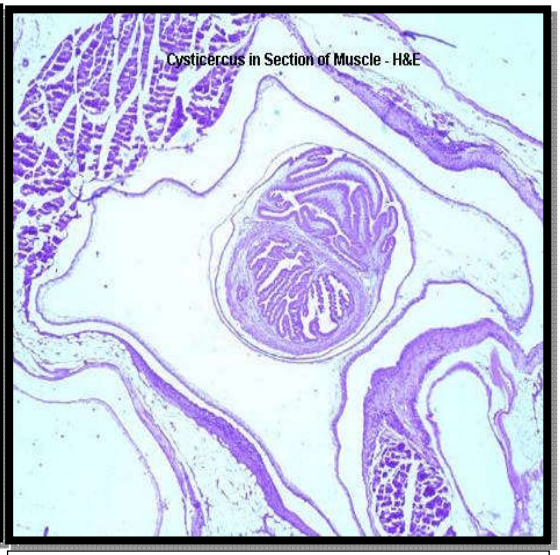


Taenia solium (*Cysticercus cellulosa*)

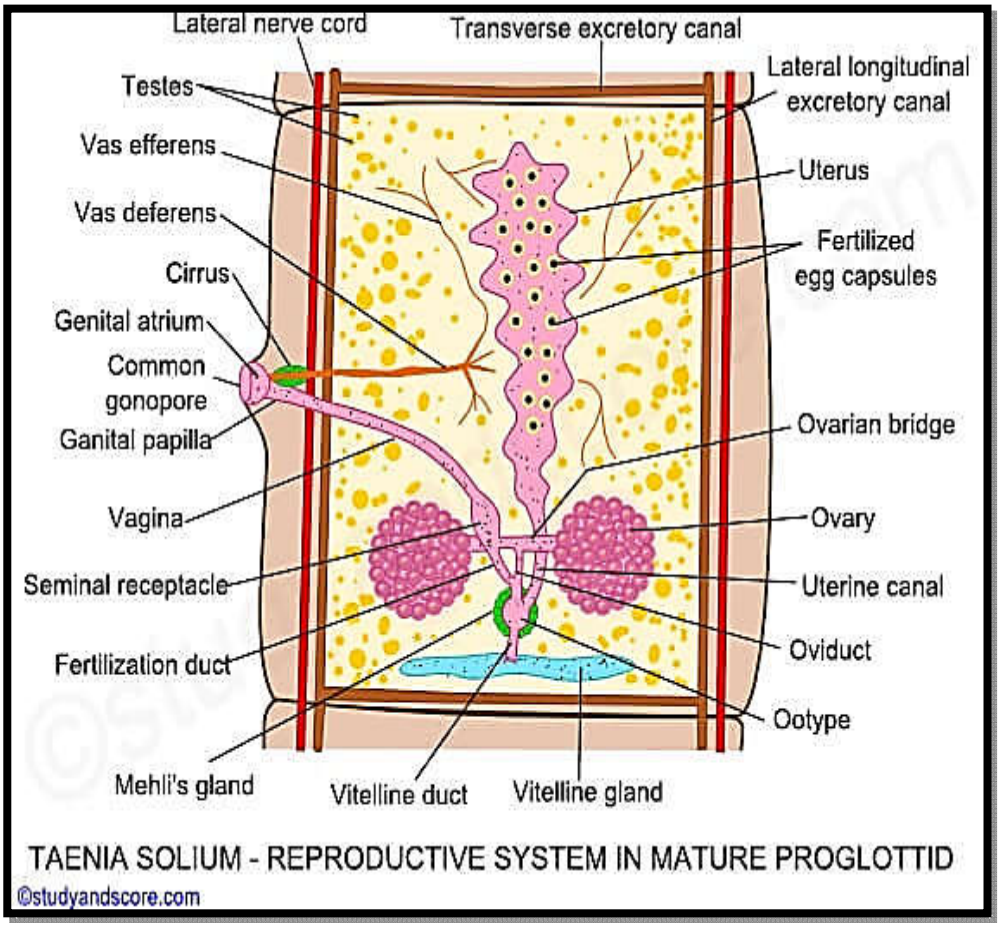
The adult worm: flat, ribbon-like body which is white and measures 2 to 3 meters long or more. Its tiny scolex, contains 4 suckers and an armed rostellum with a double row of hooks that attach to the wall of the small intestine. The adults can be identified by their characteristic lateral branches of the uterus; it is consisted 7 to 16 branches within each gravid proglottid.



Taenia solium(scolex)



Cysticercus in section of muscle



Diagnoses:

The diagnosis of Cysticercosis: Radiographic view of (*Cysticercus cellulosa*) with in human muscle, or pigs muscle, Neuro cysticercosis usually by X ray.

Faecal examination for human: diagnosed by microscopy of eggs in faeces, the ova can be found on fecal flotation.
Faecal tapeworm antigen detection: Using ELISA, PCR increases the sensitivity of the diagnosis.

Clinical signs:

Intermediate: usually with no symptoms, the other form called Cysticercosis is due to eating food, or drinking water, contaminated with faeces from someone infected by the adult worms, thus ingesting the tapeworm eggs, instead of the cysts. The eggs go on to develop cysts primarily in the muscles.
Final host: In large numbers, the adult worms can cause intestinal obstruction.

Pathogenicity:

There are two forms of human infection. One is called taeniasis, and is due to eating under-cooked pork that contains the cysts and results in adult worms in the intestines, cause intestinal obstruction in the human host if present in sufficient numbers.

It is also important to note that if humans ingest the eggs of *T. solium,* the cysticercus can develop within their muscles and within nervous tissue such as the brain, eye, and spinal cord, when cysts are found in the brain, the condition is called neuro cysticercosis.

Taenia hydatigena (Thin-necked bladder worm)

Kingdom: Animalia

Phylum: Platyhelminthes

Class: Cestoda

Order: Cyclophyllidea

Family: Taeniidae

Genus: *Taenia*

Species: *T. hydatigena*

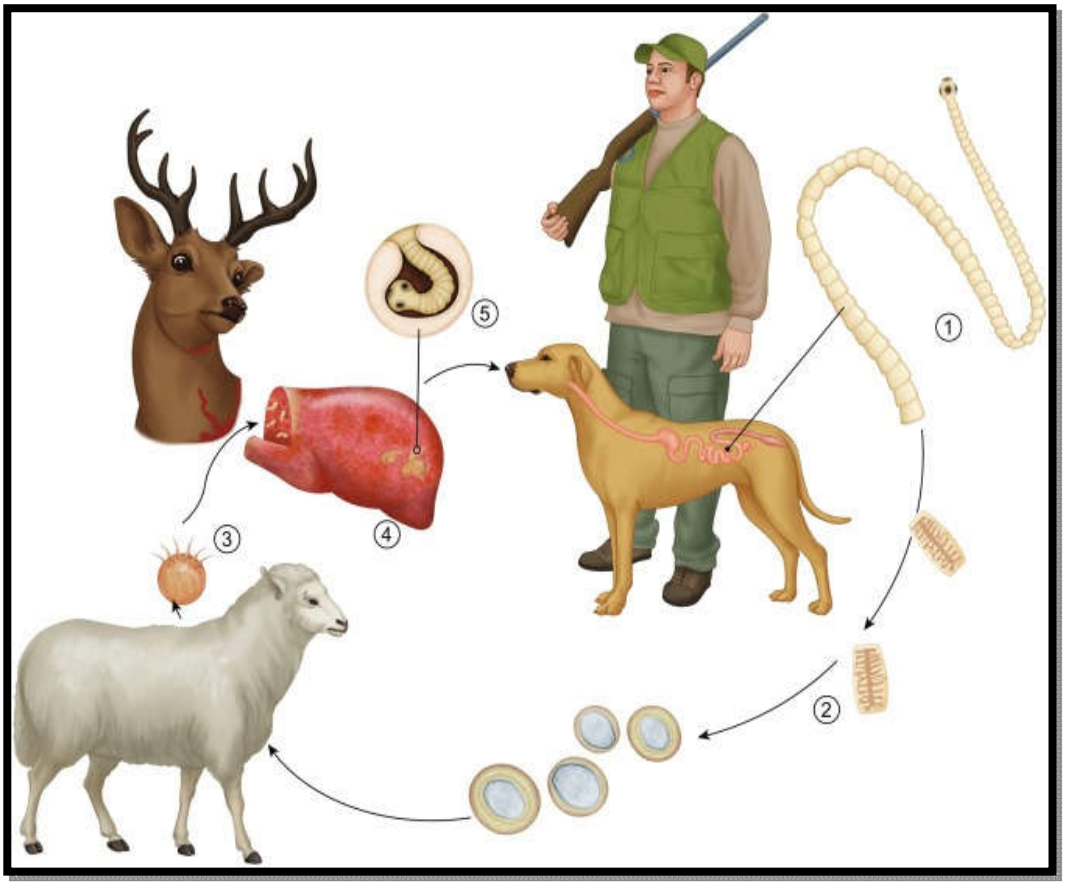
Name of Disease:

- Cysticercosis: a parasitic tissue infestation caused by larval cysts (*Cysticercus tenuicollis*) of the tapeworm *Taenia hydatigena* and infect the omentum or other abdominal organs, in cattle, sheep, goats.
- Taeniasis in Dogs.

Intermediate and final host and life cycle:

- Intermediate host: Cattle, sheep, goats
- Final host: Dogs

Adult *Taenia* tapeworms (1) live attached to the canine small intestine. Mature proglottids (egg-containing) are shed from the caudal part of the worm and exit the dog in its feces; (2) the proglottids disintegrate in the environment and the eggs are released. The intermediate host ingests eggs with the fodder; (3) the oncosphere larva is freed from the egg in the intermediate host and penetrates its organs; (4) fluctuating *C. tenuicollis* metacestodes are formed typically in the omentum or liver. They are infective for the definitive host; and (5) the dog gets the infection by eating, e.g., metacestode-containing abattoir waste. The prepatent period is about 7 weeks.



Life cycle of *T. hydatigena*

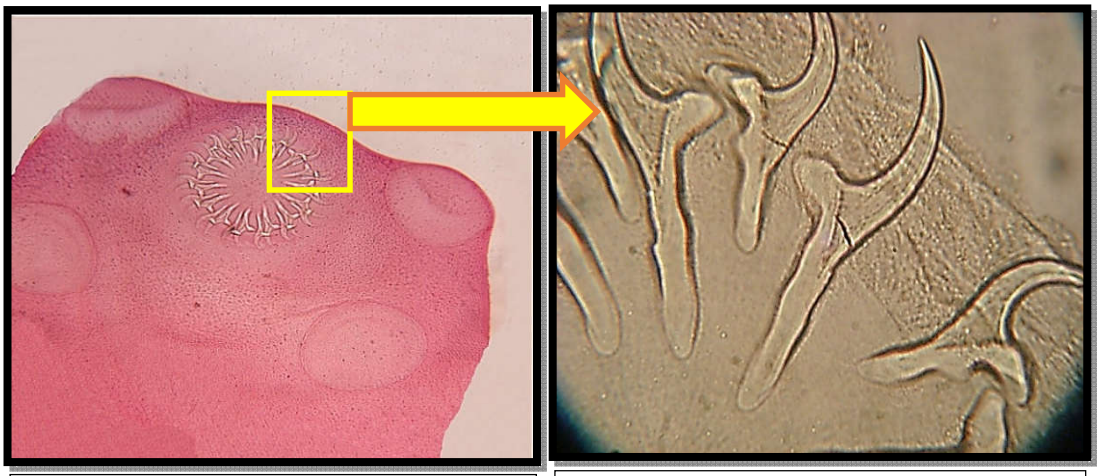
Geographical Distribution: Worldwide.

Site of Infection:

- Cattle: in the omentum or other abdominal organs as larval stage (*Cysticercus tenuicollis*) which is usually attached.
- Dogs: found in the small intestine as adult tapeworm *T. hydatigena*.

Diagnostic feature for parasite:

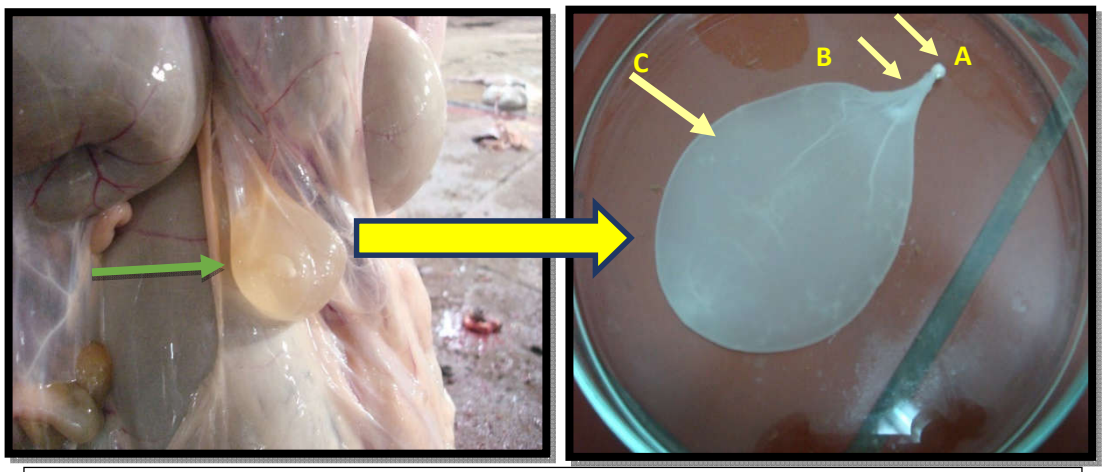
Adult: *T hydatigena* is a large tapeworm measuring up to 5 m in length. The adult worm has an armed rostellum, scolex is large and has two rows of 26 and 46 rostellum hooks. The proglottids have a single, lateral genital pore, measure 12 by 6 mm and the uterus have 5–10 lateral branches.



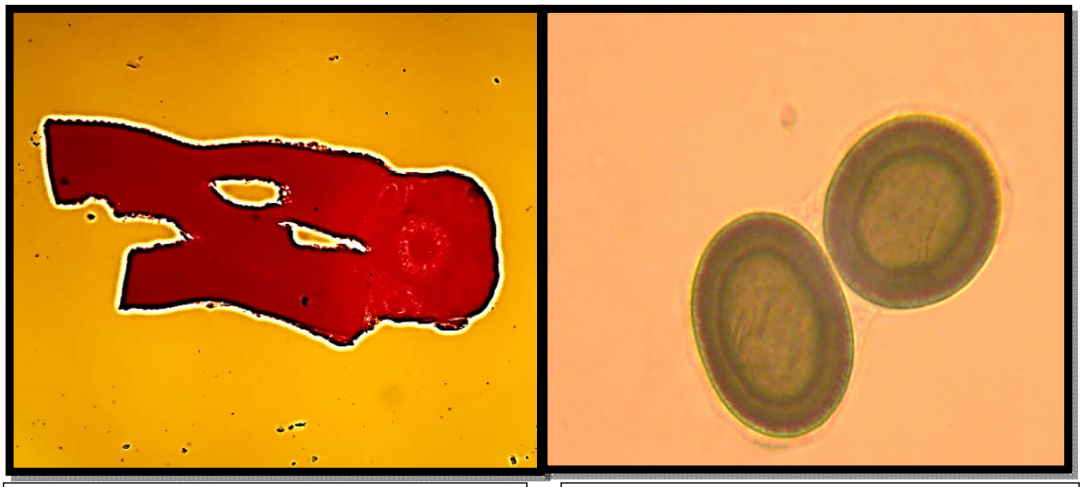
Taenia hydatigena(scolex) 40X | *Taenia hydatigena*(hooks) 400X

Larval stage: The fluid-filled bladder semi-transparent *Cysticercus tenuicollis* may be up to 5–7 cm in size and contains a watery fluid and invaginated scolex with a long neck, cysticercus that is ingested by the dog, lead to one adult tapeworm will develop in the small intestine.

Eggs: are typical taeniid type ova with a striated embryo pore surrounding an oncosphere with six hooklets inside.



***Cysticercus tenuicollis* (Larval Stage) from Sluagtered Animales**



***Taenia hydatigena* (scolex)10X**	***Taenia hydatigena* (egg) 400X**

Diagnoses:

Larval stage *Cysticercus tenuicollis*: Post mortem examination of the intermediate host will reveal the cysticercus stage in the abdominal cavity of the ruminant intermediate host.

Adult tapeworm *Taenia hydatigena*: Diagnosis is made by finding the taeniid ova on standard fecal flotation.

Clinical signs:

Intermediate host: no symptoms, the larval stage is fluid-filled bladder called *Cysticercus tenuicollis,* which is usually attached to the greater omentum or other abdominal organs of the ruminant intermediate host.

Final host: Infections in the definitive host (dogs) are generally subclinical; however, the passage of segments from the rectum may induce anal pruritis. In large numbers, the adults can cause obstruction of the intestinal tract.

Pathogenicity:

- considered nonpathogenic to the intermediate host, but in large numbers lead to pathogenicity including destruction of cells or organs.
- The adults can cause obstruction of the intestinal tract of dogs.

Taenia pisiformis

Kingdom: Animalia

Phylum: Platyhelminthes

Class: Cestoda

Order: Cyclophyllidea

Family: Taeniidae

Genus: *Taenia*

Species: *T. pisiformis*

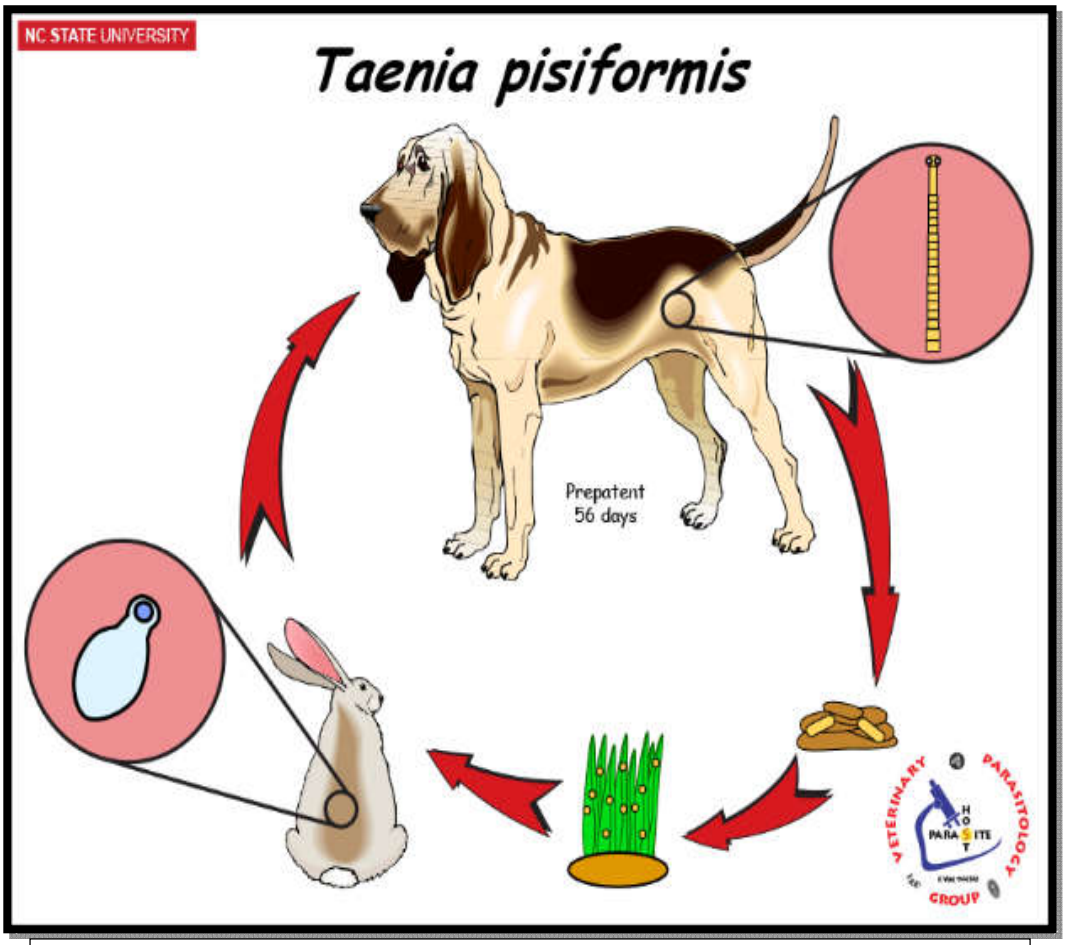
Name of Disease:

- Cysticercosis: a parasitic tissue infestation caused by larval cysts (*cysticercus pisiformis*) of the tapeworm *T. pisiformis* and infect the peritoneal cavity in rabbits
- Taeniasis in dogs and cats.

Intermediate and final host and life cycle:

- Intermediate host: rabbits.
- Final host: dogs, cats and other carnivores such as foxes.

Eggs are introduced into the environment through infected canine feces. The eggs are then ingested by a rabbit. Once inside the rabbit's gut the larva phase will then penetrate into the intestinal wall until they reach the blood stream. When the reaches the liver, the larva transforms into a cysticercus form. This cysticercus will stay in the liver for about two to four weeks, then move to the peritoneal cavity where it will wait for the definitive host to eat the rabbit. The definitive hosts are dogs. Once ingested the cysticercus finds its way into the intestine and attaches to the intestinal wall with hooks and suckers. After the worm has time to develop and grow in size, the gravid proglottids is released.



The life cycle of *T. pisiformis*

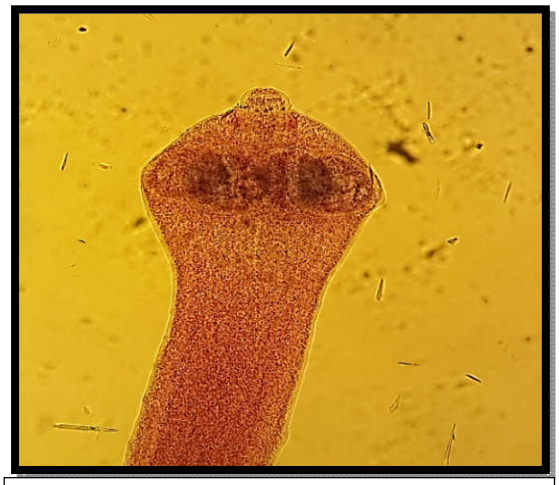
Geographical Distribution: worldwide

Site of Infection:

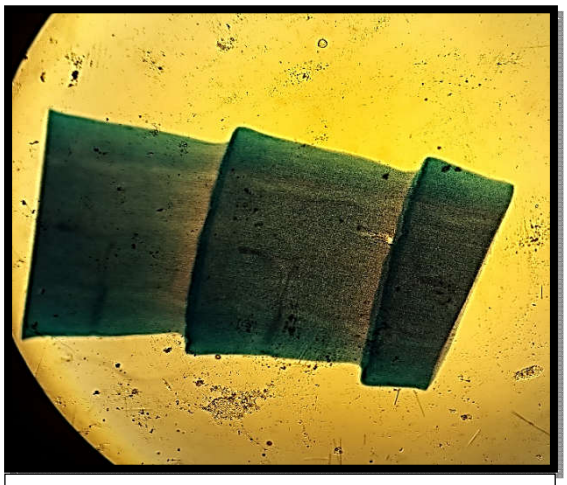
- rabbits: found in the peritoneal cavity as metacestode (the larval stage) known as *Cysticercus pisiformis.*
- dogs: found in intestinal as adult worm.

Diagnostic feature for parasite:

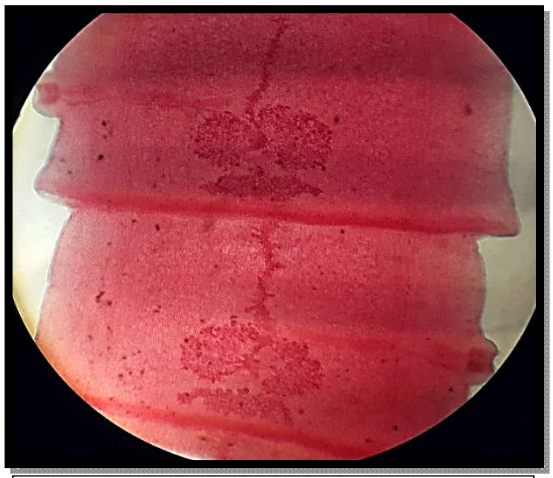
The adult tapeworm can measure up to 2 m in length. It has a large scolex with narrow strobila and the rostellum has 34–48 hooks in two rows. Gravid segments have a uterus with 8–14 lateral branches on either side. The cysticercus is a small pea-like transparent cyst and usually occurs in bunches.



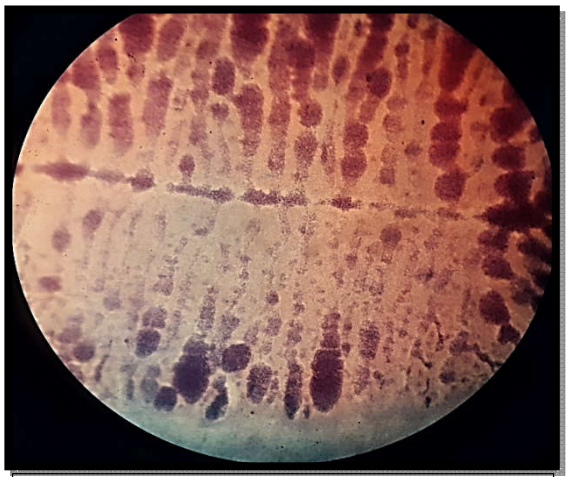
Taenia pisiformis (scolex)



Taenia pisiformis (immature)



Taenia pisiformis (mature)



Taenia pisiformis (graved)

Diagnoses

Intermediate host: The diagnose can only be seen when a post mortem is done to the rabbit. For the intermediate host there will be between two and 20 pea-sized cysts found inside the liver.

Final host: This can be found using a fecal float on a sample that can easily be done by a vet, there will be gravid proglottids with striated eggs seen in the feces.

Clinical Signs:

When looking for signs of infection in the intermediate host or definitive host, the signs are not very externally seen.

- Rabbits' infestation is usually asymptomatic, in severe infections can cause severe hepatitis or death.
- Dogs are asymptomatic, when heavy infections may cause non-specific abdominal symptoms such as diarrhea or constipation and abdominal pain.

Pathogenicity:

In rabbits look weak or ill, the major illness seen is signs of liver failure. In very few cases the cysts will migrate to the lungs or brain; these cases can cause breathing complication; the rabbit will have sudden death.

For dogs, in highly infected cases the dog will experience blockage in the intestines. In all cases the proglottids will be seen in the feces.

Moniezia spp.

Kingdom: Animalia

Phylum: Platyhelminthes

Class: Cestoda

Order: Cyclophyllidea

Family: Anoplocephalidae

Genus: *Moniezia*

Species *M. expansa.* *M. benedni*

Moniezia spp. is commonly known as sheep tapeworm or double-pored. It is a large tapeworm inhabiting the small intestines of ruminants such as sheep, goats and cattle.

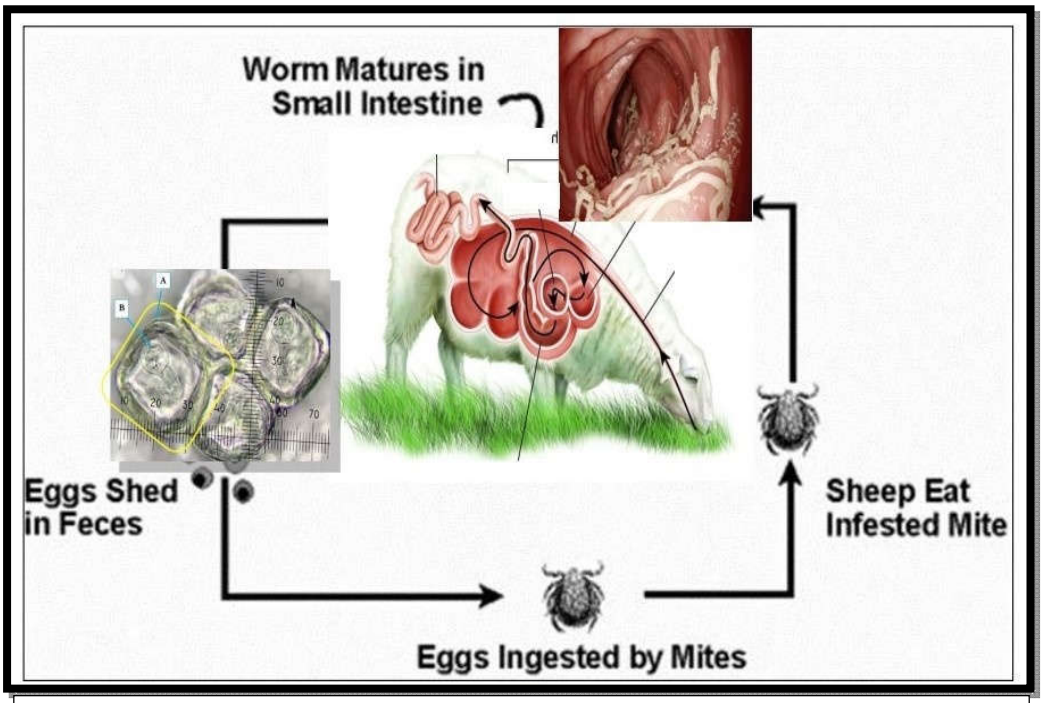
Name of Disease:

- Monieziasis: Infection of ruminants with tapeworms of the genus *Moniezia*.

Intermediate and final host and life cycle:

- Intermediate host: Oribatid mites.
- Final host: Ruminants.

Tapeworm eggs are shed in faeces segments from the host. Ruminants are infected following ingestion of the intermediate host (free-living pasture mites) containing (cysticercoids)the tapeworm larvae.



life cycle of *Moniezia* spp.

Geographical Distribution:

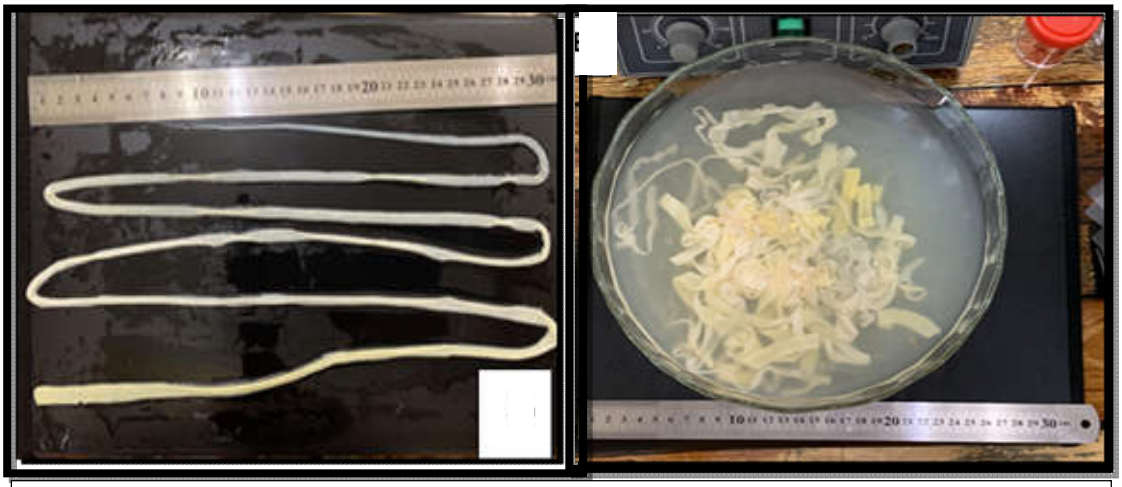
widely distributed between agricultural ruminant animals of worldwide.

Site of Infection:

- Adult *Moniezia* spp infestation in the small intestine of ruminants.

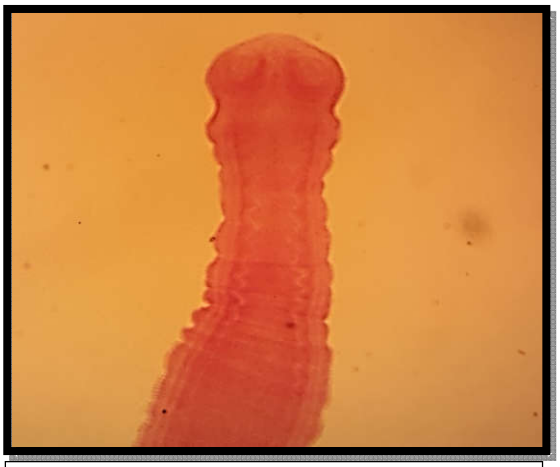
Diagnostic feature for parasite:

Adult: *Moniezia* species (e.g., *M. expansa*): a typical cestode is characterized the adult worm it is whitish in color, up to 10 m long.

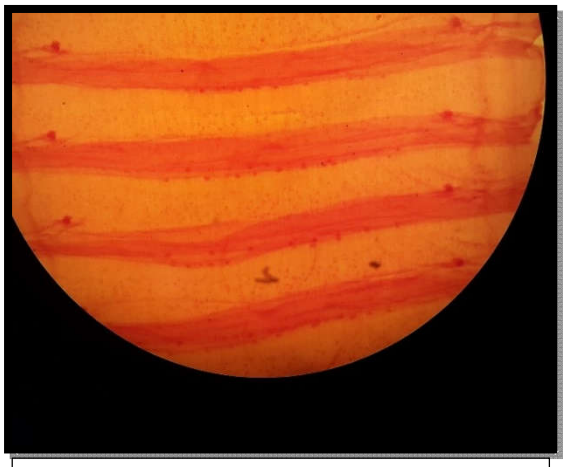


Moniezia spp. sample, geometric ruler appeared in extremely long

unarmed scolex, scolex with four suckers, without hooks. the proglottids contain two sets of sexual organs, which are located at both lateral sides of each proglottid, and associated genital pores.

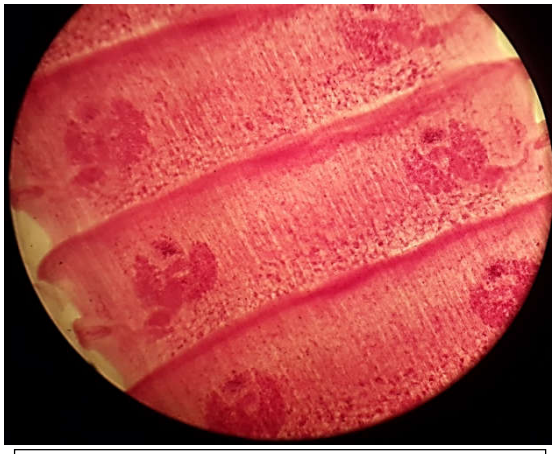


Moniezia expansa (scolex) 4X

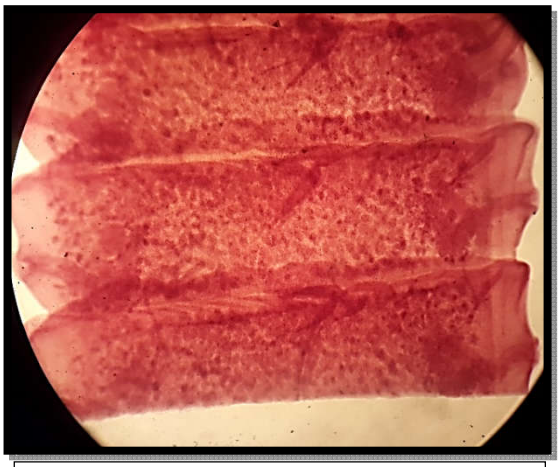


Moniezia expansa (immature)4X

Moniezia species are large tapeworms, they can grow to 6 m long and Individual proglottids are very short and wide up to 1.6 cm at the widest margins. These tapeworms produce eggs with a characteristic square or triangular shape. The eggs of both species possess a pyriform (pear-shaped) apparatus.

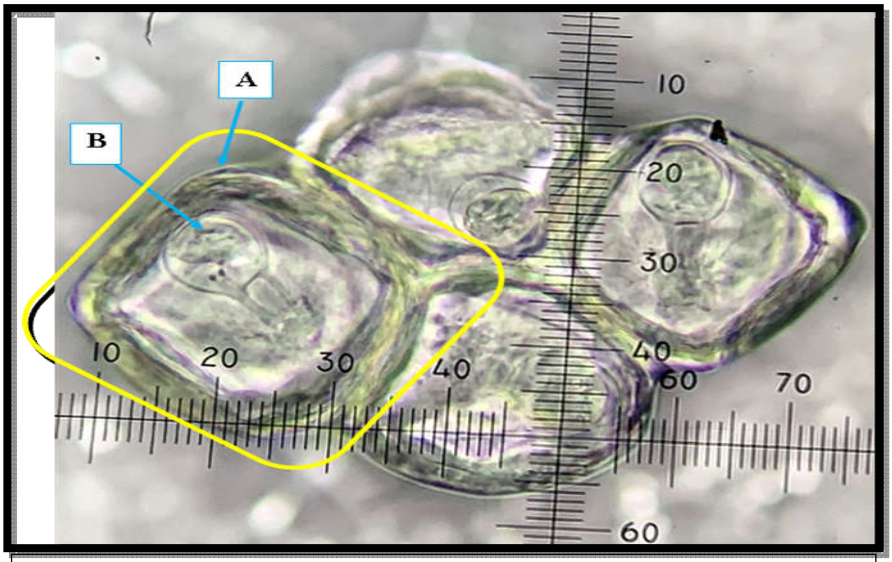


Moniezia expansa (mature)4X

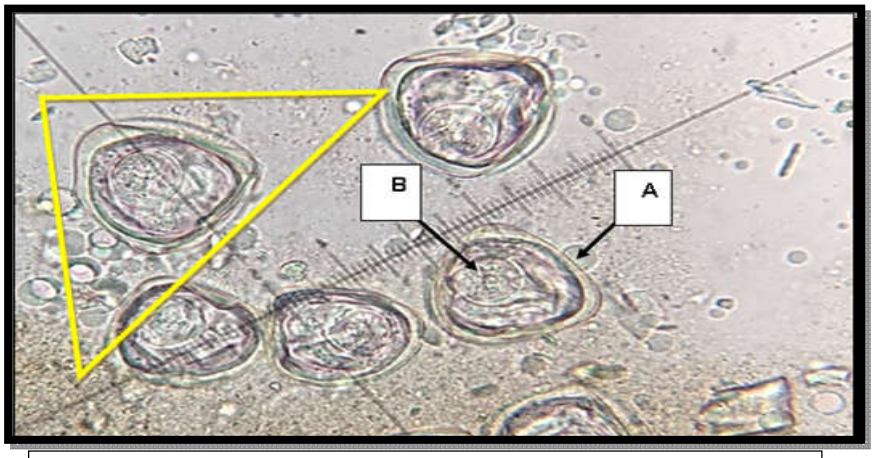


Moniezia expansa (graved)4X

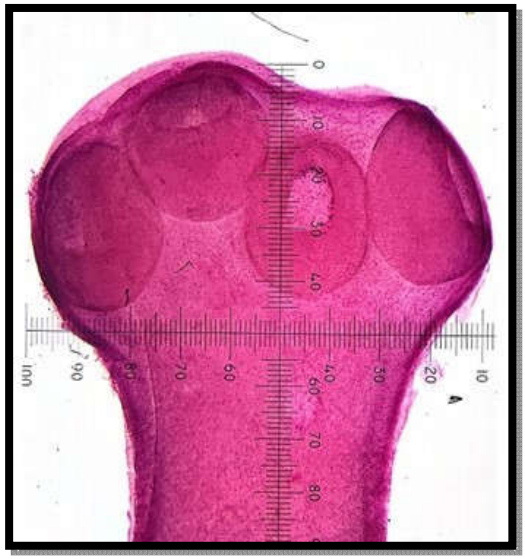
Two species are common among ruminants: *M. benedni* in cattle and *M expansa* in cattle, sheep, and goats. The eggs of both species can be easily differentiated using standard fecal flotation procedures. The eggs of *M. benedni* are square or cuboidal in shape and approximately 75 µm in diameter. The eggs of *M. expansa* are triangular or pyramidal in shape and 56 to 67 µmin diameter.



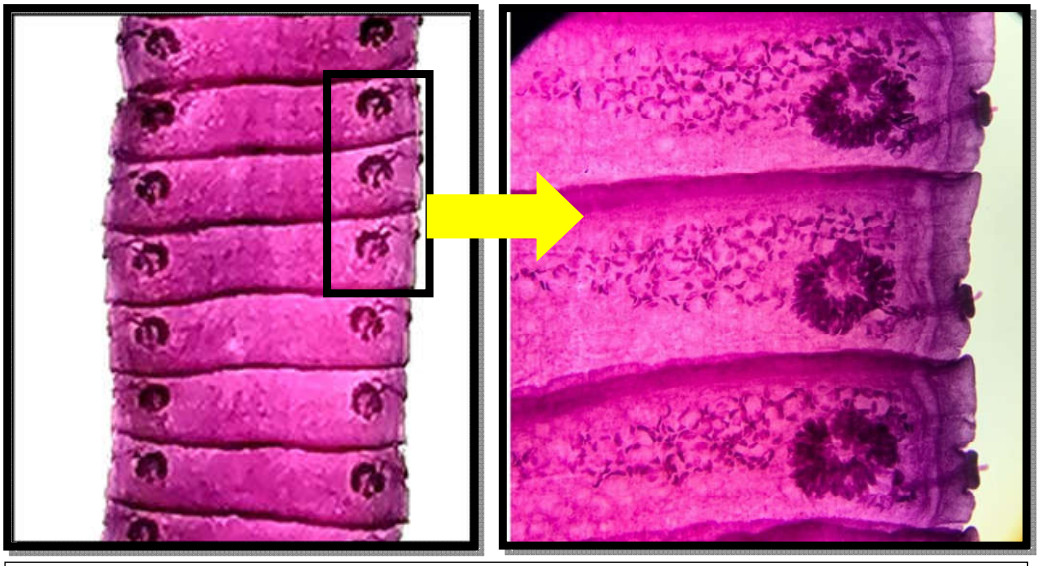
Eggs of *Moniezia benedni* **A:** Thick shell Sub-Cuboidal in shape



Eggs Mo*niezia expansa*, triangular shape **A:**Shell thick **B:**Pyriform apparatus(100X)



Moniezia spp.(scolex)10X



Developed genital structures of mature, broad proglottids, *M. benedni*. The ring shows shaped of ovaries, testes in the central field.4X

Diagnoses

Diagnosis is done by analysis feces sample in which eggs can be detected by fecal flotation tests size: 65–75µm in diameter, or often observation of the gravid proglottids in feces and anus.

Clinical Signs:

M. expansa infections are generally harmless and asymptomatic. However heavy infection may cause intestinal obstruction, diarrhea and weight loss.

Pathogenicity

In the cases of *Moniezia* infections only show severe symptoms of disease, which appear starting with weakness, colic, anemia, loss of hair, intestinal blocking. Very young animals may even die, that heavy infection may cause reduced growth in young animals.

Echinococcus spp.

Kingdom: Animalia

Phylum: Platyhelminthes

Class: Cestoda

Order: Cyclophyllidea

Family: Taeniidae

Genus: *Echinococcus*

Species: *E. granulosus*

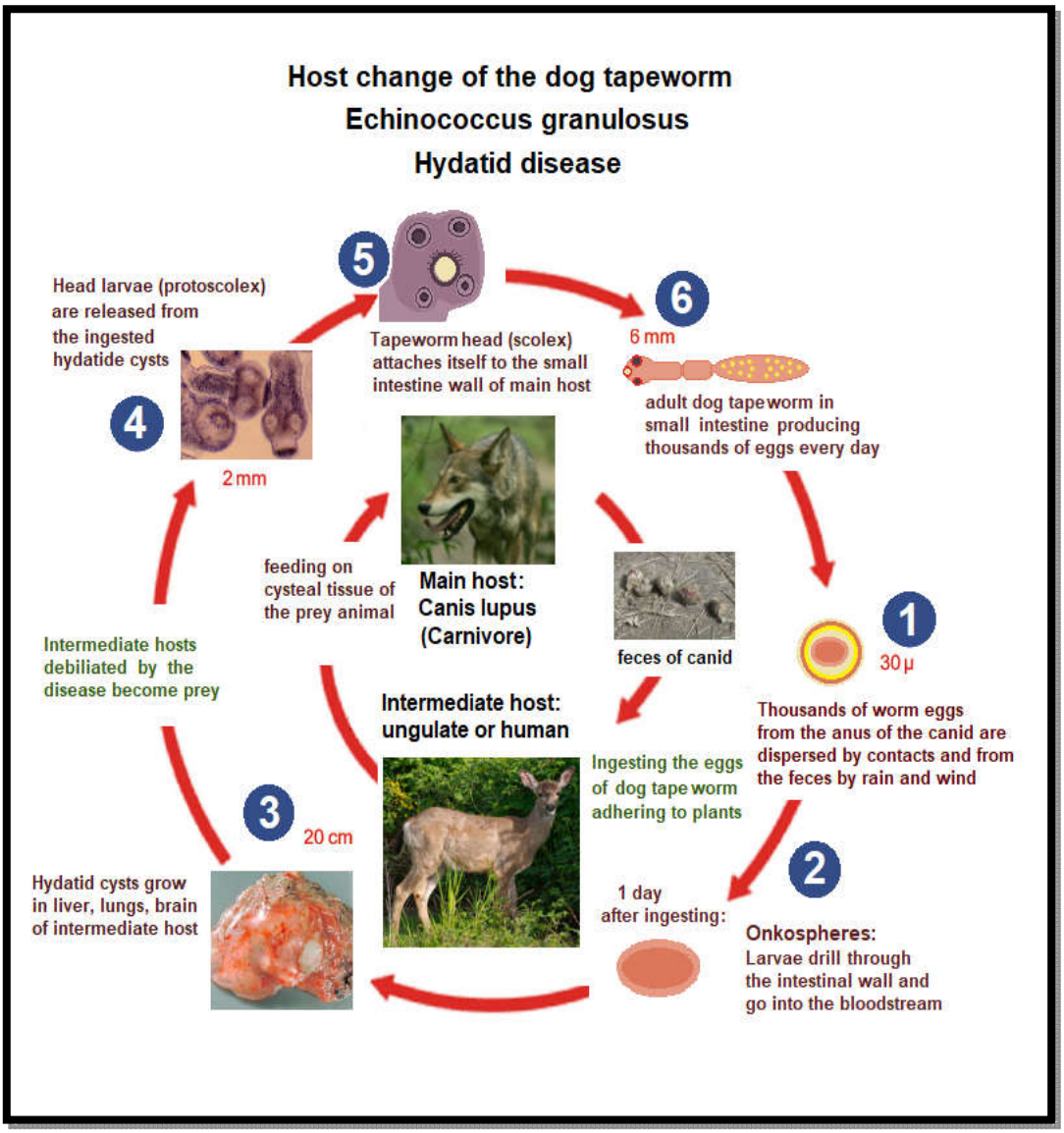
Name of Disease:

- Hydatid disease or Cystic Echinococcus: is caused by infestation of (intermediate hosts) sheep, cattle, goats, pigs, cattle, other herbivore and humans, with the larval stage of *E. granulosus,* a tapeworm found in dogs (definitive host).
- **Echinococcosis:** in dogs.

Intermediate and final host and life cycle:

- Intermediate: livestock and humans.
- Final host: dogs

Carnivores acquire infections through the ingestion of metacestodes (hydatid) in the tissues of prey animals. Prey animals become infected with the metacestode through the ingestion of eggs passed in carnivore feces.



life cycle of *Echinococcus. granulosus*

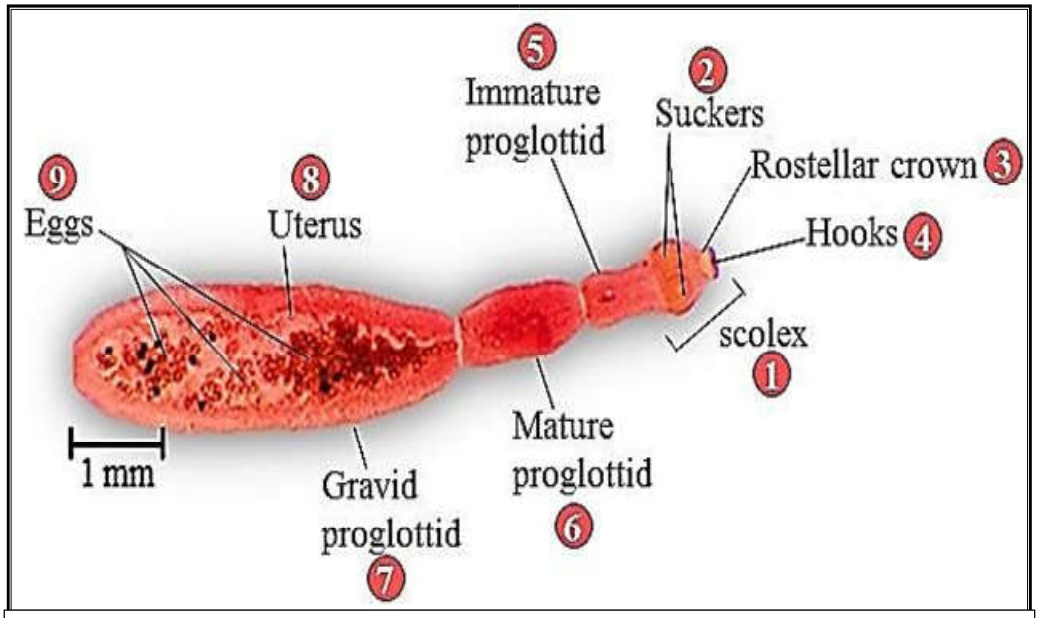
Geographical Distribution: worldwide.

Site of Infection: Cattle and human: abdominal cavity.

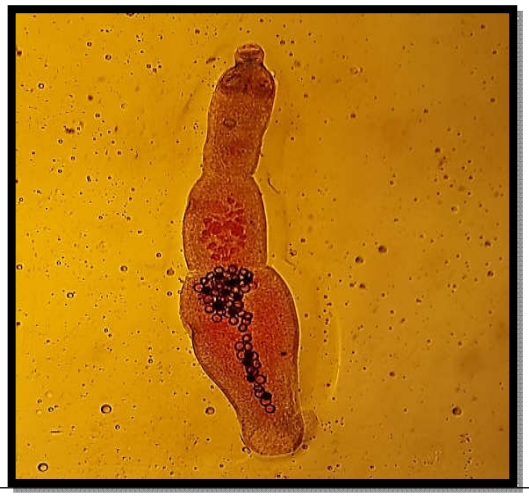
- Dog: Small intestine.

Diagnostic feature for parasite:

The adult tapeworm ranges in length from 3 mm to 6 mm and has three proglottids (segments) an immature proglottid, mature proglottid and a gravid proglottid. *E. granulosus* has four suckers on its scolex (head), and also has a rostellum with hooks.

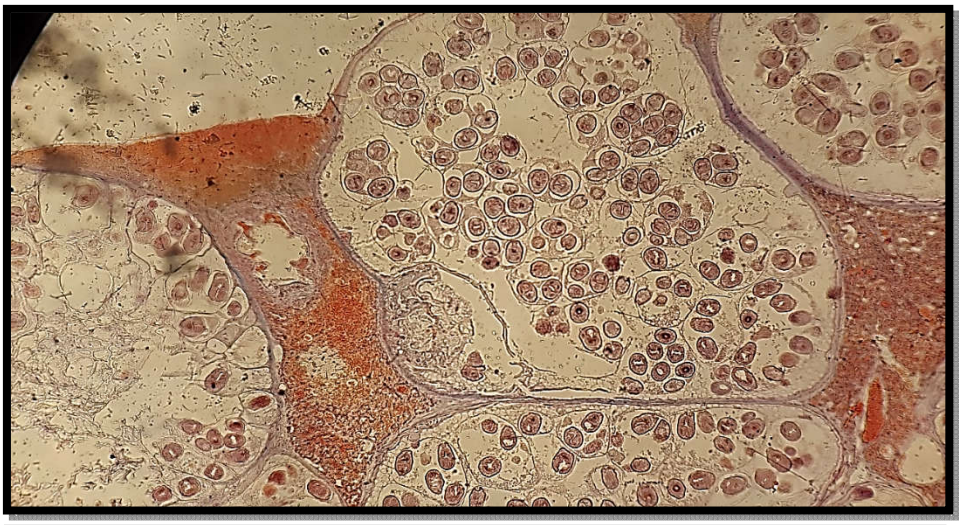


***Echinococcus granulosus* as typical diagram (adult worm)**

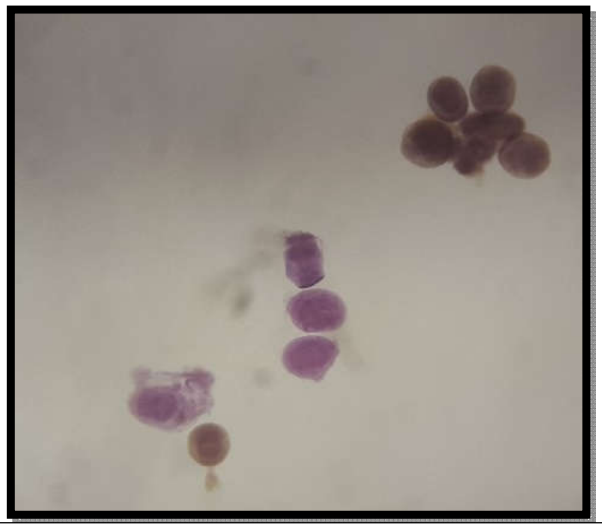


Echinococcus granulosus (adult)40X. under light microscope.

The hydatid cyst of *E. granulosus* has a thick cyst wall with a thin germinal membrane located just inside it. Brood capsules containing protoscolices. from this germinal membrane. When a dog ingests the hydatid cyst containing the brood capsules and protoscolices, each protoscolex develops into an adult tapeworm.



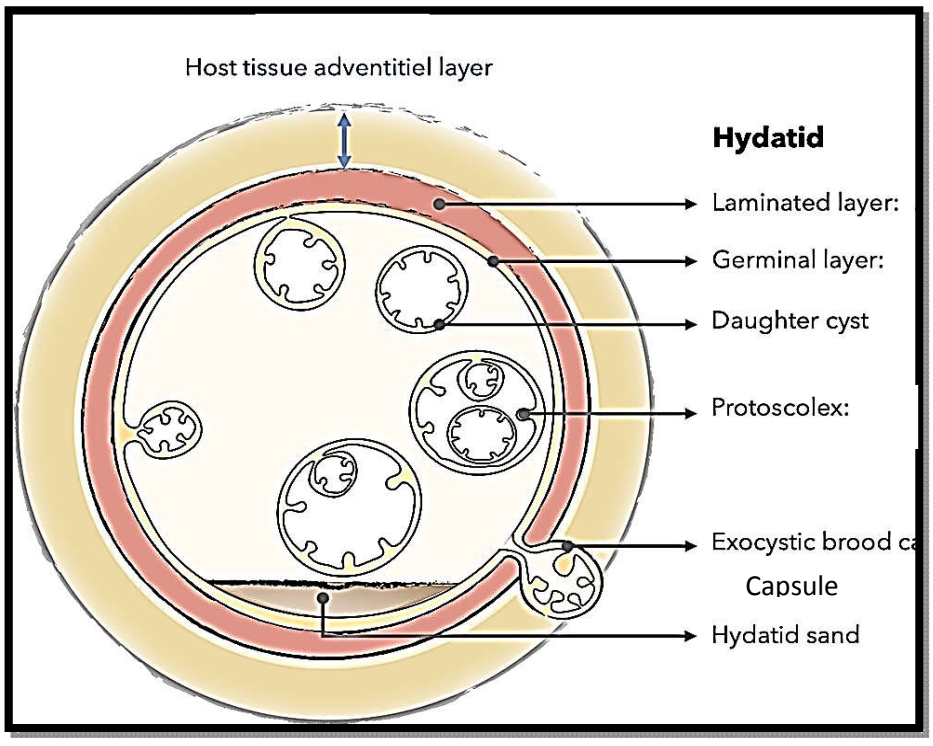
Histopathological Section in the Hydatid cyst wall and scolices.40X



Echinococcus granulosus (hydatid sand)40X

Hydatid laminated layer following:

1- Laminated layer: A cellular, barrier against host immunological response.
2- Germinal layer: Produces several biological elements of the metacestode (cystic fluid, brood capsules and protoscolex).
3- Daughter cyst.
4- Protoscolex: Budding of the membrane of the brood capsules of the hydatid cyst.
5- Exocystic brood capsule: Brood capsules may become embedded in the laminae of the laminated layer and progressively be pushed outwards.
6- Hydatid sand.



Anatomical structure of

Diagnoses

The intermediate host:

1.Immunologic tests: the intradermal test (or Kasoni test), immune electrophoresis(IEP), indirect fluorescent antibody (IFA) tests. ELISA and enzyme immunoassays (EIA) are sensitive tests for detecting the antibodies in the infestation animal's serum.

2. X-ray: it is useful in the cysts of the lung, bones, liver capsule.

Definitive host:

Diagnosis in the dog may be: Laboratory diagnosis, faecal flotation, and post mortem examination of the small intestine, or detection by Polymerase chain reaction (PCR) is also used to identify the parasite from DNA isolated from eggs or adult worm.

Clinical Signs:

The intermediate host:

The infections remain silent for years before the enlarging cysts cause symptoms in the affected organs. The signs depend primarily on the size of the cyst which depends on the cyst age, the infection can result in abdominal pain, a mass in the hepatic area, pulmonary involvement can produce chest pain and cough.

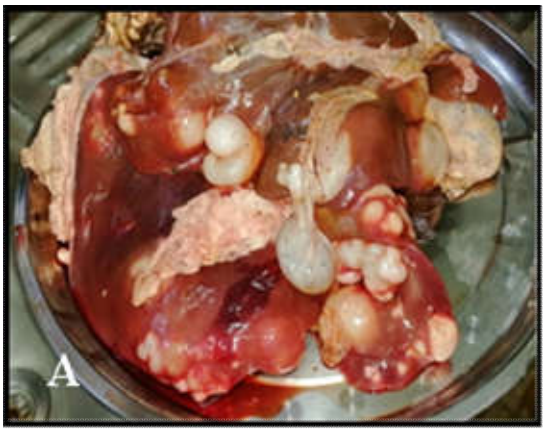
Definitive host: Infections in the definitive host dog are subclinical. *Echinococcus spp*. are important due to zoonotic disease.

Pathogenicity:

In cases developed disease and occurs rupture of a hydatid cyst into other organs (brain, bone, heart) can also be involved, Hydatid cysts of the brain

produces increasing symptomatic evidence of an intracranial tumor. Human and livestock infection with hydatid cysts can cause serious disease and death.

Important signs: Coughing, enlargements of the infected organs depend on the size, age and position of the cyst, and also necrosis in the tissue of the organ. In the liver, the cyst may cause an obstructive jaundice because of the bile duct obstruction.



Hydatid cyst in slaughtered liver (sheep)

Hydatid cyst in slaughtered lung (sheep)

Diphylobothirum spp.

Kingdom: Animalia

Phylum: Platyhelminthes

Class: Cestoda

Order: Diphyllobothriidea

Family: Diphyllobothriidea

Genus: *Diphylobothirum*

Species: *D. latum*

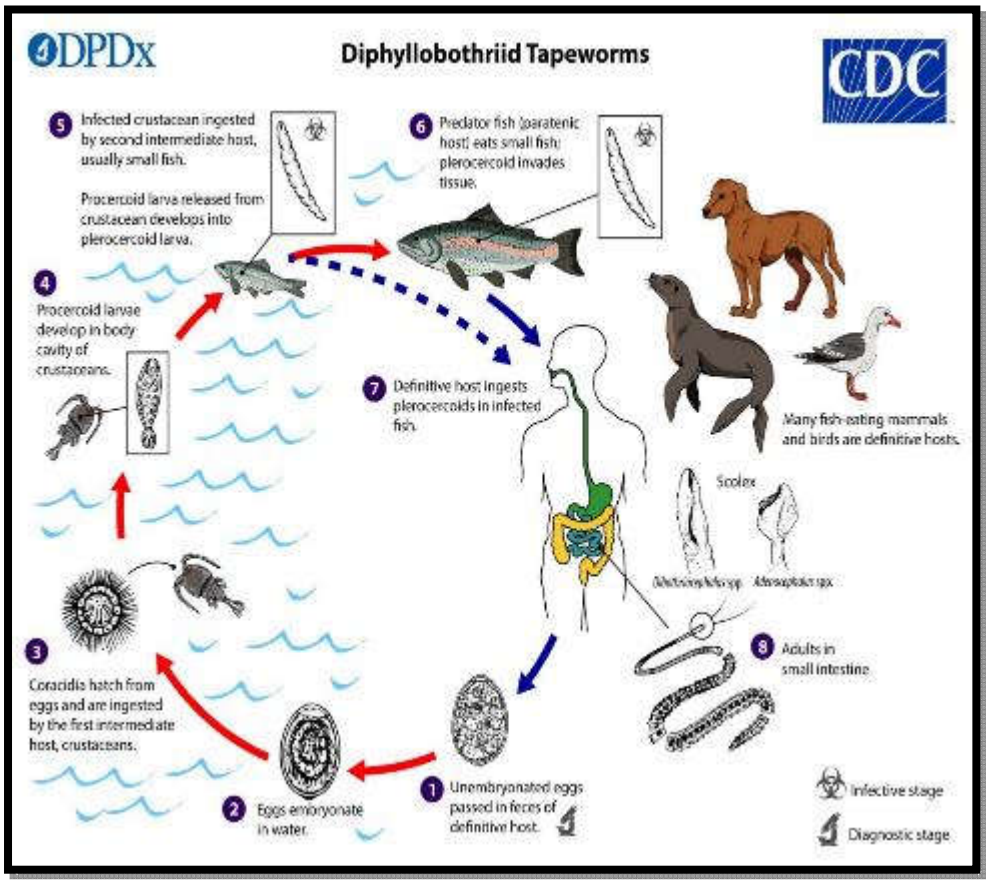
Name of Disease:

Diphylobothriasis: is caused by infestation of (definitive host) humans, with the adult worm of *D. latum*, by ingestion of fish contain larval stage for *Diphylobothirum* larvae (plerocercoid), that are raw, undercooked, or unfrozen.

Intermediate and final host and life cycle:

- First Intermediate host: crustaceans.
- Second intermediate hosts: fish.
- Final host: human.

Consumption of infected second intermediate hosts by larger predatory fish, which serve as paratenic hosts; the parasites thereupon migrate into the musculature awaiting consumption by definitive hosts in which adult tapeworms will then finally develop in the small intestine to release up to a million mature eggs per day per parasite.



life cycle *Diphylobothirum latum*

Geographical Distribution:

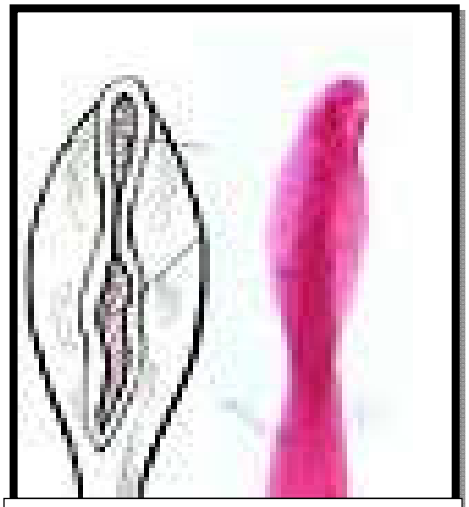
Diphylobothriasis occurs in areas with human consumption of raw or undercooked freshwater fish, like sushi in Japan. Such areas are found in Europe, North America, Asia, Uganda.

Site of Infection:

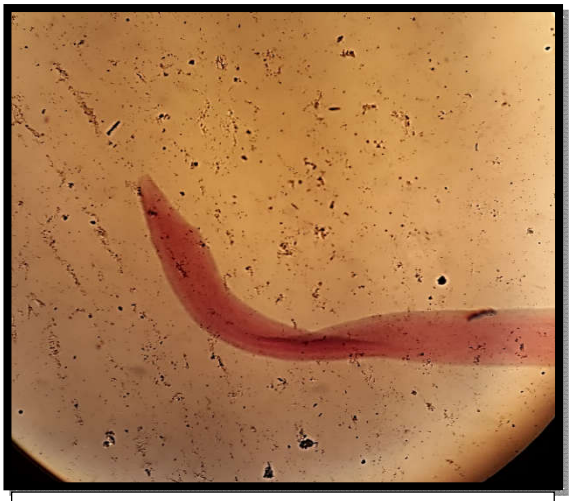
- **Fish**: found in the musculature of the fish (second intermediate host) which infestation with *Diphylobothirum* larvae, the larval stage (plerocercoid).
- **Human**: in the small intestine and jejunum with adult worm.

Diagnostic feature for parasite:

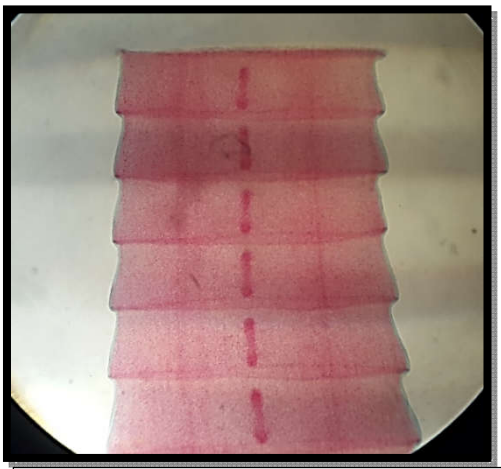
Diphylobothirum species this tapeworm can be 2 to 12 m in length. Almonds – shaped (scolex) equipped with attachment grooves on the dorsal and ventral surfaces, instead of four suckers on its anterior end, this organ called botheria.



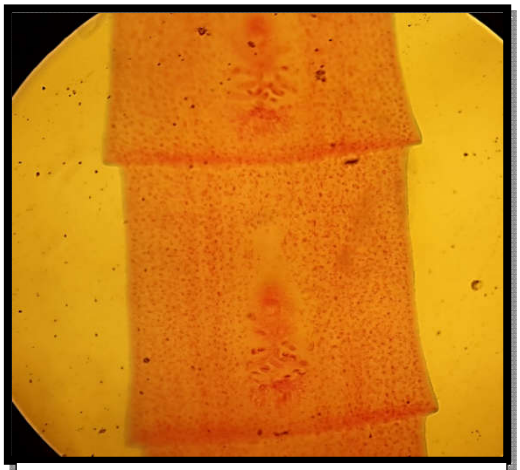
(Scolex, botheria)



Diphylobothirum (scolex)



Diphylobothirum (immature)

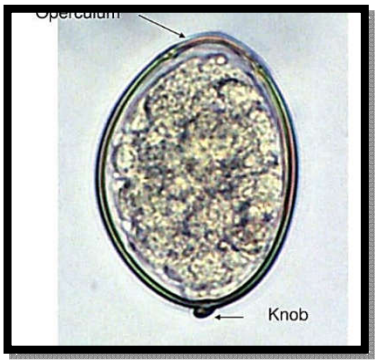


Diphylobothirum (mature)

Each proglottid of this tapeworm has a centrally located, and an associated uterine pore through which the eggs are released.

The egg of *Diphylobothirum* species also resembles that of a fluke (digenetic trematoda). The egg is oval and possesses a distinct operculum at one end of the pole of the shell. The eggs are light brown, averaging 67 to 71 µm× 40 to 51 µm, and tend to be rounded on one end, the operculum is present on the end opposite the rounded pole. The eggs are Unembroynated when passed in the feces.

Mature segment (rosette-shaped



Diphylobothirum

Diagnoses

Diagnosis is usually made by Faecal examination which identifying proglottid segments, or characteristic eggs in the faeces.

Clinical Signs:

Most infections are asymptomatic. Infections may be long-lasting, persisting for many years or decades (up to 25 years) if untreated.

Symptoms (when present) are generally mild. Manifestations may include abdominal pain and discomfort, diarrhea, vomiting, constipation, weight loss, and fatigue.

Pathogenicity:

D. latum competes with the host for vitamin B12 absorption, absorbing some 80% of dietary intake and causing deficiency vitamin B12 with pernicious anemia.

Gastrointestinal obstructions rarely, massive infections may result in intestinal obstruction.

Dipylidium caninum

Kingdom: Animalia

Phylum: Platyhelminthes

Class: Cestoda

Order: Cyclophyllidea

Family: Dipylidiidae

Genus: *Dipylidium*

Species: *D. caninum*

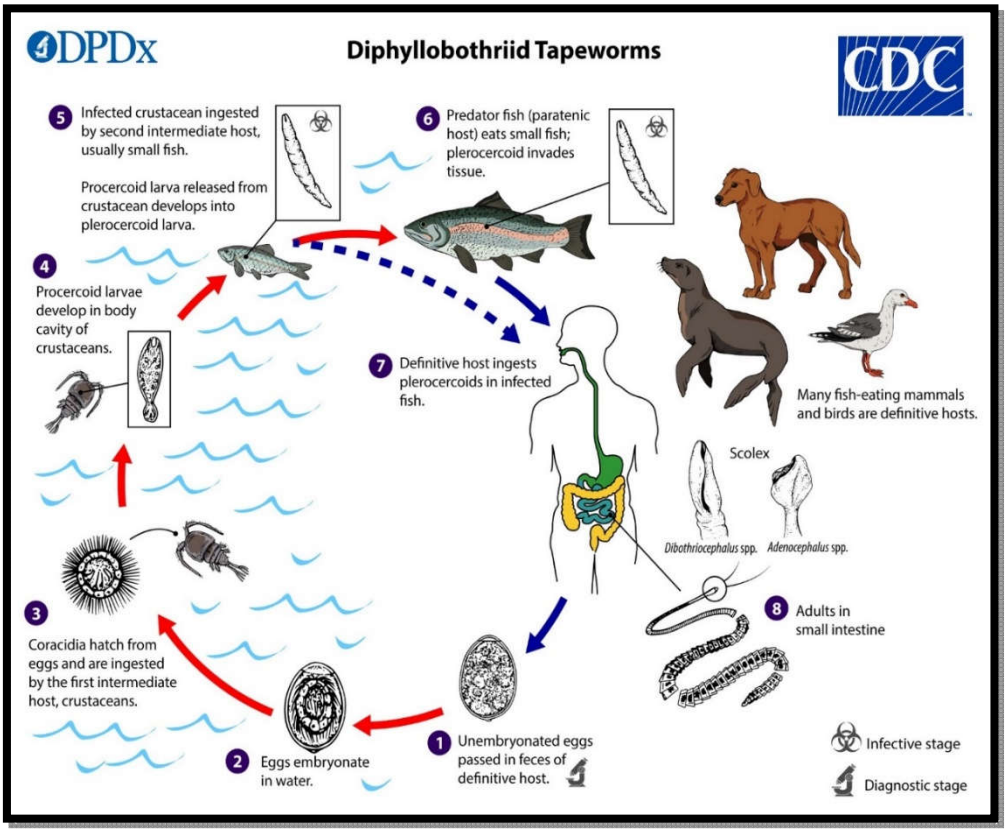
Name of Disease:

Diplydiasis: is disease caused by a tapeworm infestation of dogs, cats and humans caused by tapeworm species *D caninum*.

Intermediate and final host and life cycle:

- Intermediate host: fleas.
- Final host: Dogs, cats, and in rare cases young children.

The first stage in the life cycle is when the gravid proglottids are either passed out through faecal matter. The gravid proglottids once out of the definitive host release eggs. Then, an intermediate host (the larval stage of a flea or chewing louse) will ingest an egg, which develops into a cysticercoid larva, which must be ingested in a flea by the dog. Humans can also become infected by *D. caninum* by accidentally ingesting an infected flea. In the small intestine of the definitive host, the larva develops into an adult tapeworm.



life cycle *Dipylidium caninum*

Geographical Distribution:

Worldwide; this tapeworm is common among pet dogs and cats; human infection is rare.

Site of Infection:

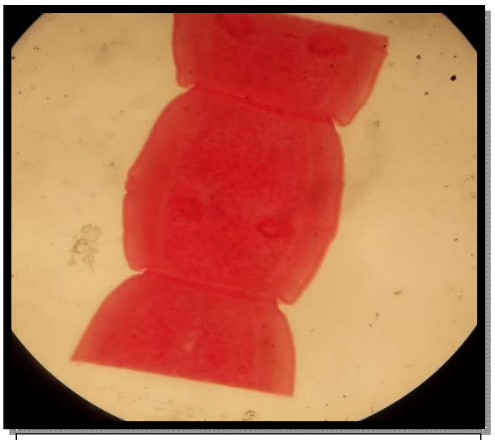
- Fleas and lice: in the body cavity with the larval stages (cysticercoid larva).
- Dogs and cats, human: in small intestinal with adult worm.

Diagnostic feature for parasite:

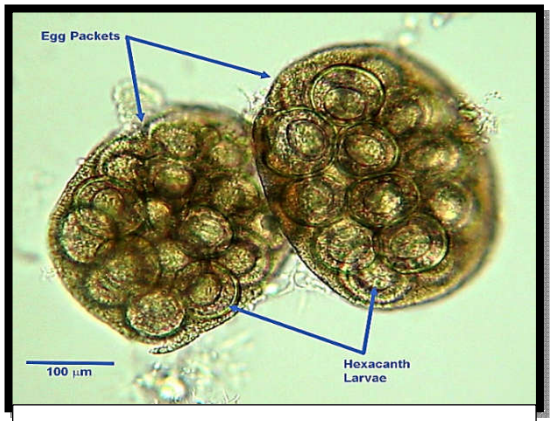
Dipylidium caninum, also called the flea tapeworm, double-pored tapeworm, or cucumber tapeworm (in reference to the shape of its cucumber-seed-like proglottids), may reach lengths of 20–50 cm. The scolex has a width possesses 4 suckers and a rostrum with 3–4 crowns of hooks, retractable rostellum with

four rows of hooks. In each proglottids, two sets of male and female sexual organs are formed This can be only hardly recognized in terminal proglottids, the released gravid proglottids appear similar to cucumber seeds, egg packages each containing 8–30 eggs which measure 34–40 µm in diameter.

Dipylidium caninum



Dipylidium caninum



***Dipylidium caninum* (Egg)**

Diagnoses

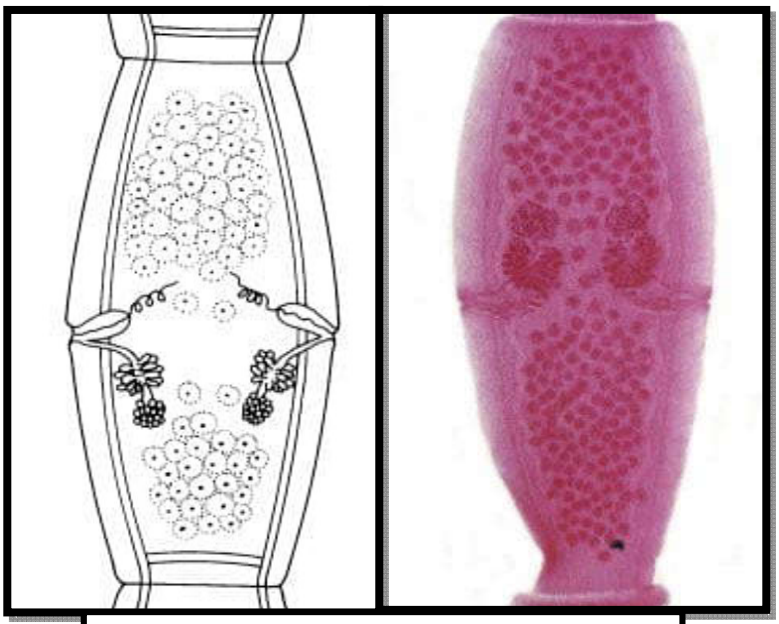
This tapeworm is typically diagnosed in one of two ways. The egg packets can be found on fecal flotation. The gravid proglottids may also be seen on bedding, or in the hair coat.

Clinical Signs:

Accompanied by slight gastrointestinal disturbances, as this is the region where the worms inhabit. Most heavy infections may be identified in an infected individual: mild diarrhea, abdominal colic, rectal itching, and pain due to emerging proglottids through the anal cavity.

Pathogenicity:

Tapeworm infection usually does not cause pathology in the dog or cat, and behavior may reflect the presence of anal discomfort and itching, or pruritis.



***Dipylidium caninum* (Mature**

Raillietina spp.

Kingdom: Animalia

Phylum: Platyhelminthes

Class: Cestoda

Order: Cyclophyllidea

Family: Davaineidae

Genus: *Raillietina*.

Species: *R. tetragona*, *R. echinobothrida*, *R. cesticillus*

parasitic tapeworm belonging to the class Cestoda, it is a helminthes of the small intestine of pigeon, chicken, turkey, guinea fowl.

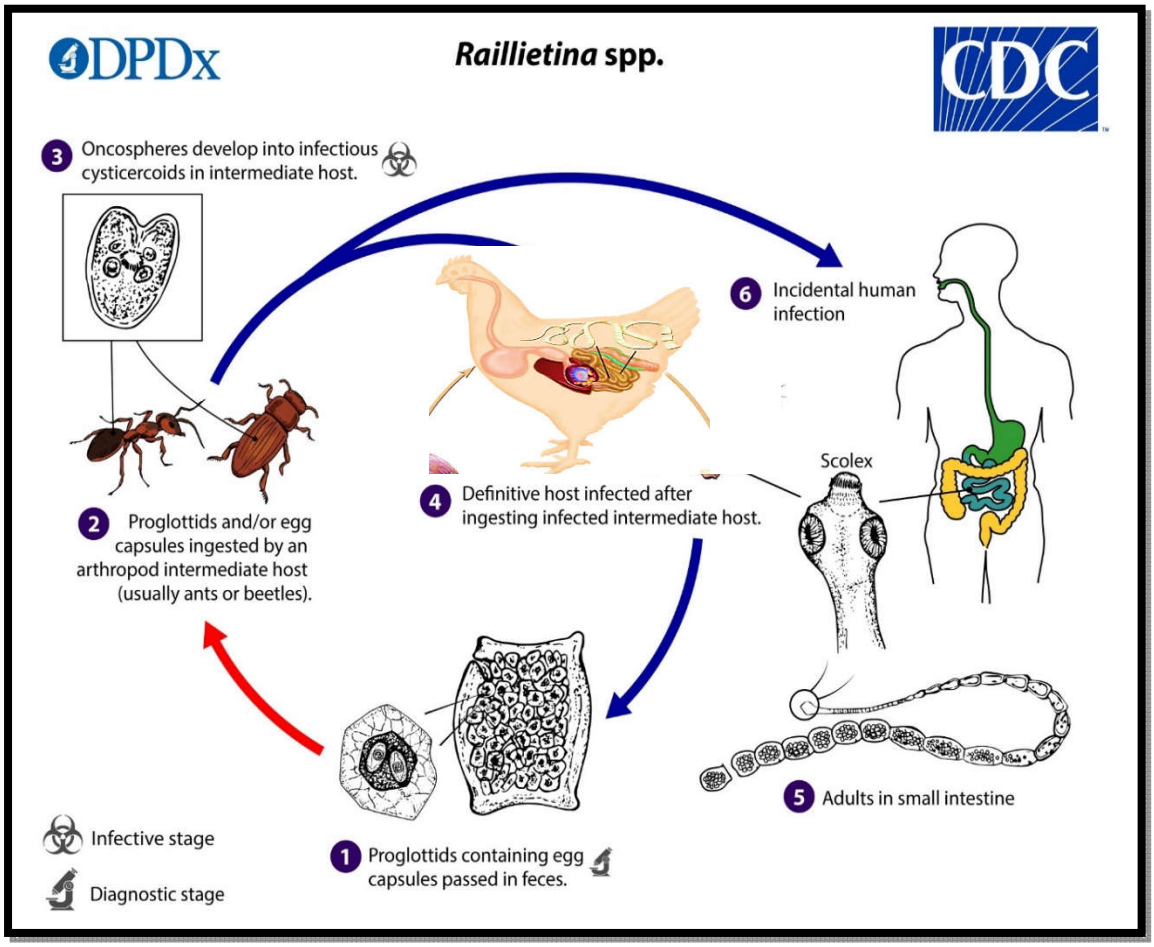
Name of Disease:

- Raillietiniasis: is an intestinal infestation of bird caused by parasites *Raillietina* spp. (tape worm).

Intermediate and final host and life cycle:

- Intermediate host: insects, such as ants and beetles.
- Final host: birds.

Raillietina require two different hosts for a complete life cycle. The definitive hosts are mostly birds. The intermediate hosts are insects, such as ants and beetles. Mature eggs are released from the avian host through faeces. Eggs develop into larval forms (cysticercoids), which are ingested by ants. A cysticercoid stage, which is the ultimate infective form. When the insect with infective larvae is ingested by birds, the cysticercoid is released in host, then a mature tapeworm develops.



Life cycle of *Raillietina* spp.

Geographical Distribution: Worldwide.

Site of Infection:

- Small intestine of a wide variety of domestic and wild birds.

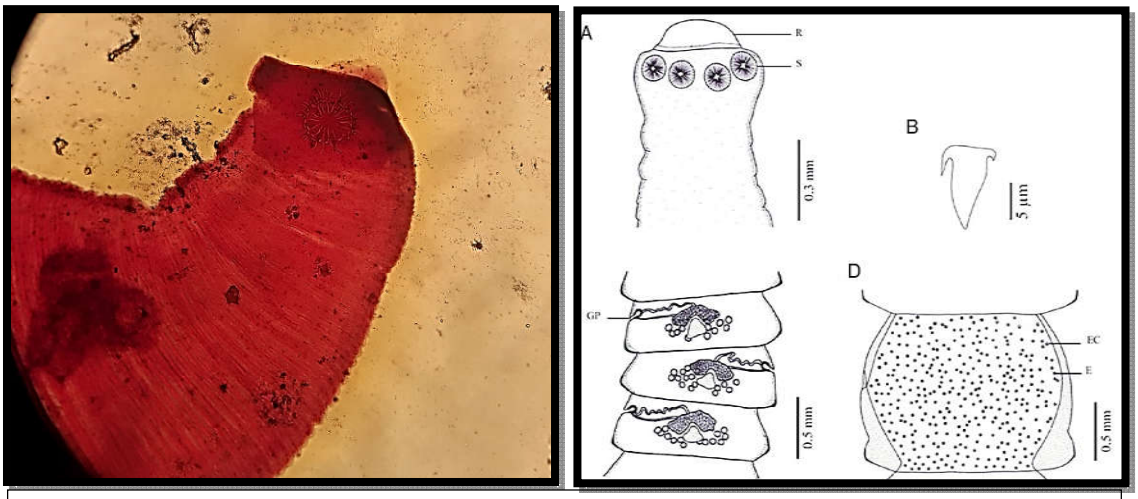
Diagnostic feature for parasite:

A -Scolex bearing the rostellum (R) surrounded by 4 unarmed round suckers (S).

B- Large hammer-shaped hook.

C- Mature proglottid showing irregularly alternating opening of the genital pore (GP).

D- Gravid proglottid showing a single egg (E) per egg capsule (EC).



Adult *Raillietina* spp.

Diagnoses

Infection is directly diagnosed by identifying proglottids in the faeces, detection of eggs by fecal examination, or adult worms in the intestine post mortem of bird.

Clinical Signs: Tapeworm infestations are asymptomatic.

Pathogenicity: The level of the clinical pathogenicity is characteristic of each species:

- *R. cesticillus* is quite harmless in terms of symptoms.

- *R. echinobothrida* is highly pathogenic cause severe enteritis and death in domestic poultry, and causes nodular tapeworm disease under heavy infection, loss of meat and egg productions.

Hyminolips spp.

Kingdom: Animalia

Phylum: Platyhelminthes

Class: Cestoda

Order: Cyclophyllidea

Family: Hymenolepididae

Genus: *Hyminolips*

Species: *H. nana*, *H. dimenuta*

Name of Disease:

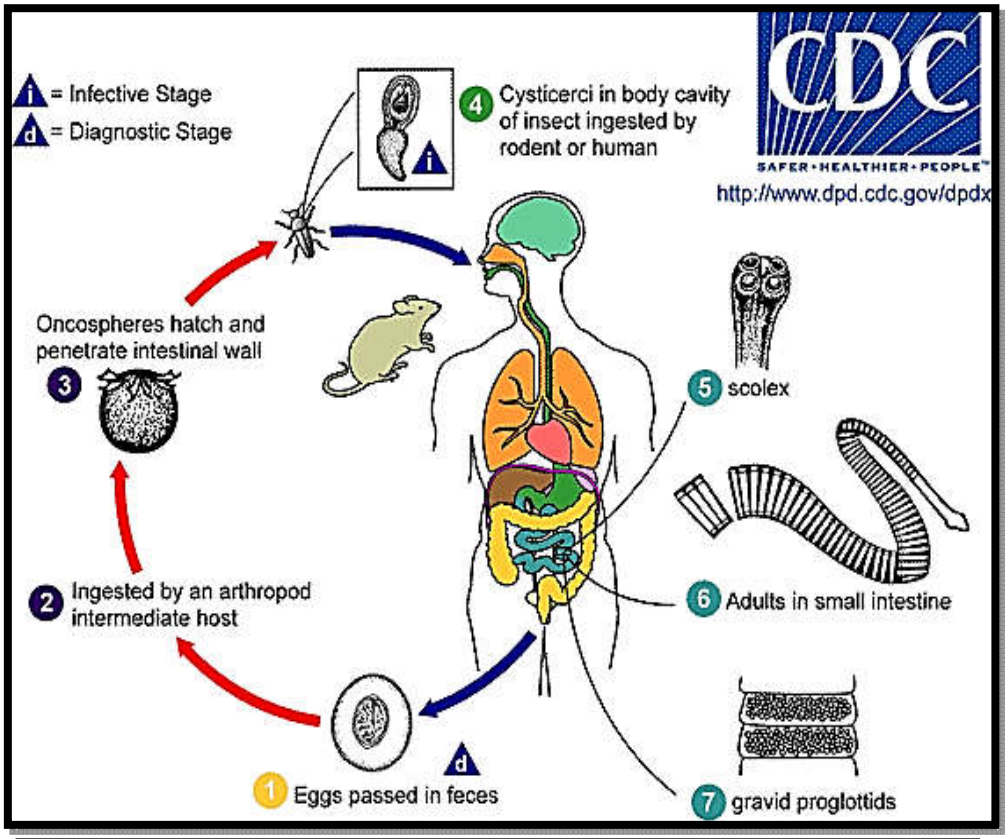
Hyminolepiosis: is infestation by species of tapeworm: *Hyminolips nana* or *H. dimenuta,* a type of intestinal worm or helminthes infecting humans, especially children and rate.

Intermediate and final host and life cycle:

Intermediate host: insects, fleas and beetles (arthropods).

Final host: humans, rate.

The life cycle of *H. nana* can be completed via a direct cycle (Without an intermediate host) or indirect cycle (with an intermediate host), the most common intermediate hosts for *H. nana* are arthropods. When an egg is ingested by the definitive host, it hatches and releases the oncosphere (hexacanth) which penetrates the villi of the small intestine and develops into a cysticercoid, they can gradually develop into adults. Autoinfection may be result if adults produce of eggs remain in the intestine. *H. nana* infections are much more common in humans than *H. dimenuta* infections in rate.



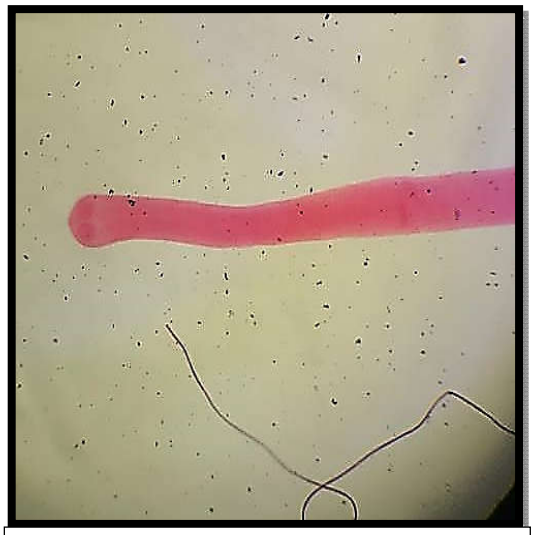
The life cycle of *Hyminolips spp.*

Geographical Distribution: worldwide.

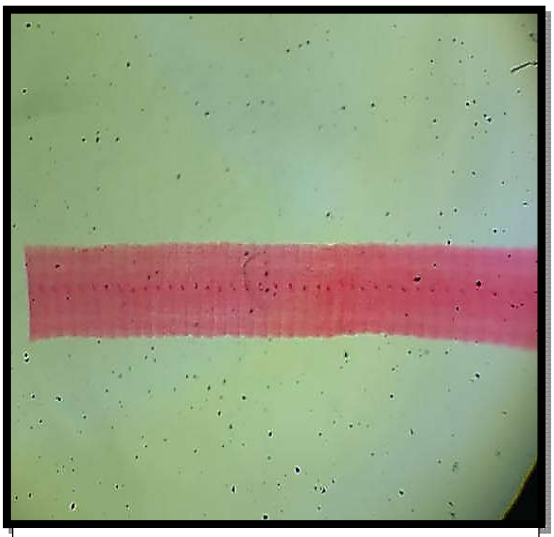
Site of Infection: in the small intestine humans and rats.

Diagnostic feature for parasite:

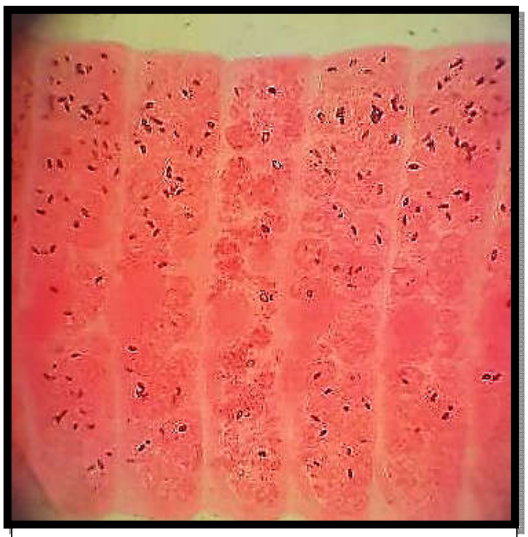
H. nana is a tapeworm; a small species, 40 mm long and 1 mm wide, it consists of scolex (head) It's in a rhomboidal shape, the neck is long and slender, the segments are wider than long. Genital pores are situated on the same side, each segment contains three testes and a single ovary. May be distinguished *H. nana* by it having a small scolex bearing four suckers with short rostellum which is armed with a single row of hooklets, however *H. dimenuta* has 4 suckers and a retractable rostellum that does not bears have hooks, unarmed rostellum.



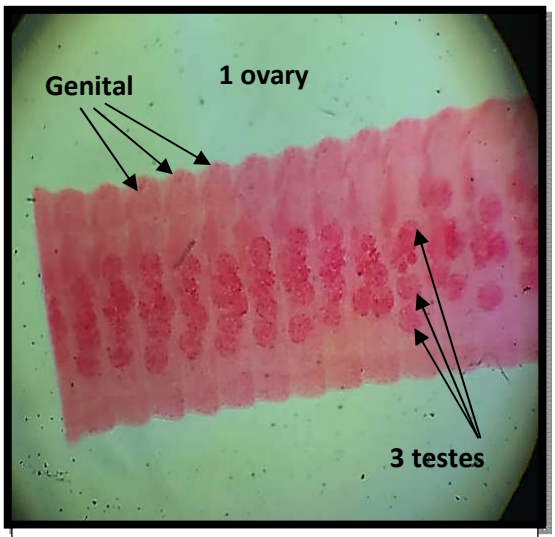
Head, long neck of *Hyminolips spp.*



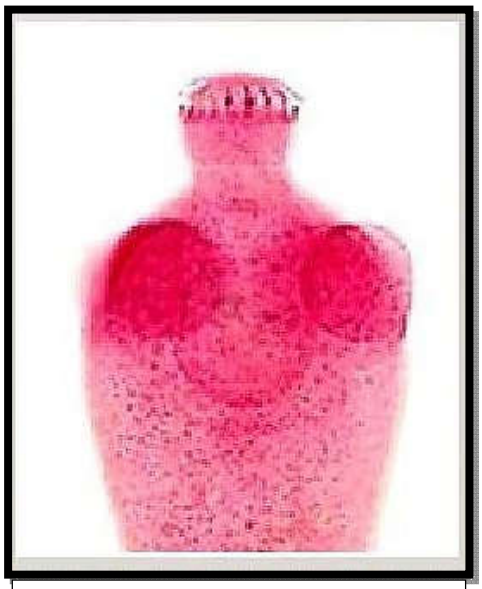
Immature of *Hyminolips spp.*



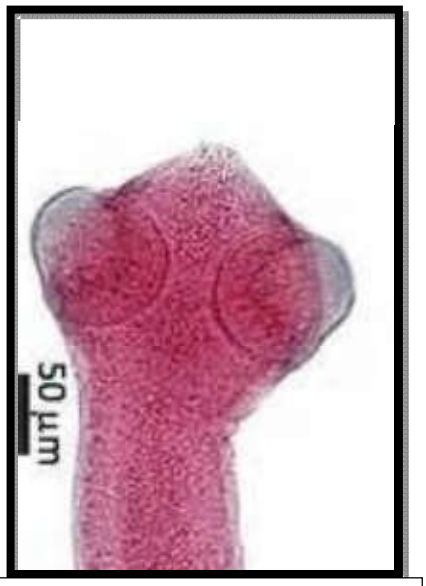
Gravid of *Hyminolips spp.*



Mature of *Hyminolips spp.*

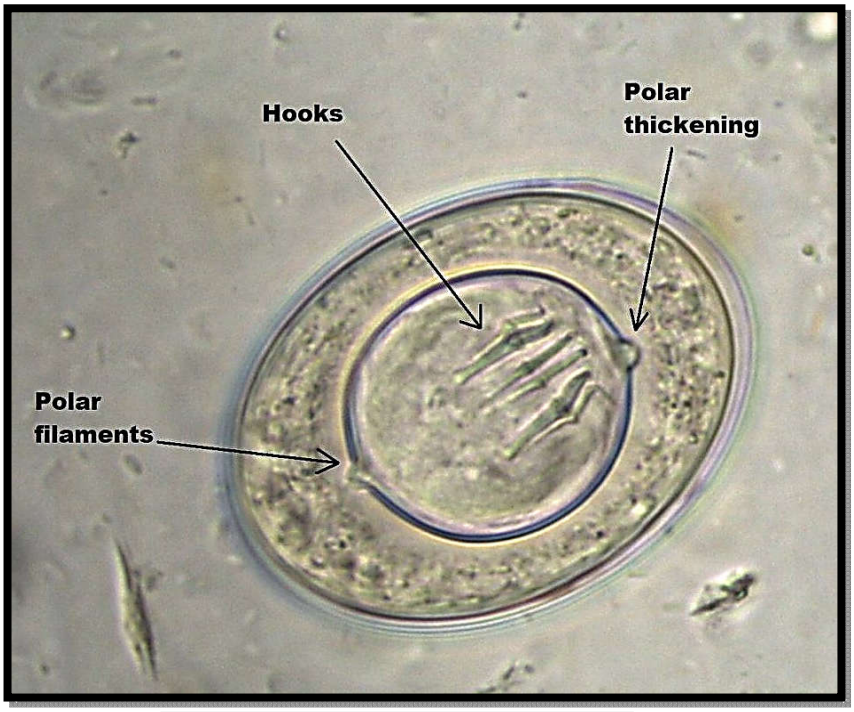


H. nana, armed rostellum



H. dimenuta, unarmed

Eggs of *Hyminolips* spp. are oval, a size range of 30 to 50 µm. On the inner membrane are two polar thickening, hooks.



Eggs of *Hyminolips* spp

Diagnoses

Hyminolips spp. can be diagnosed by the presence of eggs in routine fecal flotation or adult worms in the small intestine.

Clinical Signs:

Hyminolips spp. infections are most often asymptomatic. Heavy infections can cause weakness, headaches, anorexia.

Pathogenicity:

- Light infections: asymptomatic.
- Heavy infections: toxemia, significant intestinal inflammation, diarrhea, abdominal pain, for young.
- Children: head-ache, itchy bottom, difficulty sleeping.

Nematohelminthes

Nematoda consisting of about 12,000 known species, and many more predicted species of worms (commonly known as roundworms or threadworms). Nematodes live in the soil and other terrestrial habitats as well as in freshwater and marine environments. Many are damaging parasites of plants and animals, including humans.

General Characteristics

1- Nematodes or roundworms are very important group of helminthes which infect animals and humans.

2- The nematodes have both direct and indirect life cycle affecting wide range of animals, almost free living/ parasitic and unsegmented, cylindrical in shape.

3- Nematodes cannot be stained for identification

4- Some of the nematodes have on their cuticle specialized adhesive structure such as hooks in *Tetrameres* sp. Cephalic vesicle in *Oesophagostomum* sp.

5- Some of the nematodes have lateral flat cuticular expansion called as (Alae).

6- If the alae is situated in the cervical region it is called as cervical alae and in posterior region as caudal alae.

7- Alimentary canal is present.

8- They have a body cavity or coelom. But it is not true coelom. (Pseudo coelom).

9- The extremities are tapering except the females of *Tetrameres* spp. which become almost spherical after copulation.

10- The sexes are separate the body is covered with cuticle. The cuticle is provided with circular annulation or it may be smooth or it may have longitudinal striation.

11- The cuticle is relatively thick in nature and may extend into the buccal capsule, esophagus, rectum and distal portion of genital duct.

12- Sometimes the cuticle forms a special adhesive structure for Eg. Hooks in *Tetrameres* spp. – male

13- Simple or complicated cuticular thickening in *Gongylonema* spp.

14- Cervical collar or cephalic collar in *Physaloptera* spp.

15- Cephalic vesicle in *Oesophagostomum* spp.

Ascaris spp.

Kingdom: Animelia

Phylum: Nematohelminthes

Order: Ascaridida

Family: Ascarididae

Genus*: Ascaris lumbricoides*

Name of Disease: Ascardiosis

Intermediate and final host and Life Cycle:

A common "round worm of man", is the largest of the intestinal nematodes parasitizing humans and the most common worm found in human. The life cycle consist a single female lays about two hundred eggs per day. Eggs are passed in the faeces of host and develop to infective stage in about 10 days or above depending upon the environmental temperature. The eggs are resistant to adverse conditions like drying, freezing and chemicals and remain viable for 5 years. The infection of host by ingestion of embryonated eggs containing L_2 larva. Ingested egg hatch in the intestine and liberate L_2. The liberated L_2 penetrate the intestinal wall reach the peritoneal cavity and then to liver. Majority of L_2 reach the liver via hepatoportal system. From the liver, the larvae carried to the right atrium via superior vena cava by blood and then to lungs. In the lungs larvae are arrested in the capillaries where the L_2 moult to L_3. These L_3 break out of the alveolar capillaries and pass through alveolar duct and small bronchioles. Then the larvae gradually ascend

the bronchial tree and reach the trachea. From the trachea the larvae migrate to pharynx and mouth and finally swallowed to reach the intestine. This migration is called as, *"Tracheal migration"*. Large number of L_3 reach intestine in about 7 to 8 days after infection. In the intestine L_3 moult to L_4 in about 14 to 21 days and L_4 to L_5 in about 21 to 29 days. The worms reach maturity in about 50 to 55 days. Eggs appear in faeces in about 62 days. In P. equorum, the worm reach maturity in 80 to 83 days.

Geographical Distribution: Worldwide distribution, An estimated 1 billion people are infected with *A. lumbricoides* worldwide. While infection occurs throughout most of the world, *A. lumbricoides* infection is most common in sub-Saharan Africa, the Americas, China, and east Asia. *Ascaris lumbricoides* eggs are extremely resistant to strong chemicals, desiccation, and low temperatures. The eggs can remain viable in the soil for several months or even years.

Site of Infection: Intestine

Diagnostic feature for parasite and Morphology:

1- Large nematode, buccal capsule is absent.

2- Mouth is surrounded by 3 lips.

3- Esophagus is devoid of posterior bulb.

Ascaris lumbricoides is characterized by its great size. The three lips are seen at the anterior end. The margin of each lip is lined with minute teeth which are not visible at this magnification. Males are 2–4 mm (0.08–0.2 in) in diameter and 15–31 cm (5.9–12 in) long. The

male's posterior end is curved ventrally and has a bluntly pointed tail. Females are 3–6 mm (0.1–0.2 in) wide and 20–49 cm (7.9–19 in) long. The vulva is located in the anterior end and accounts for about one-third of its body length. Uteri may contain up to 27 million eggs at a time, with 200,000 being laid per day.

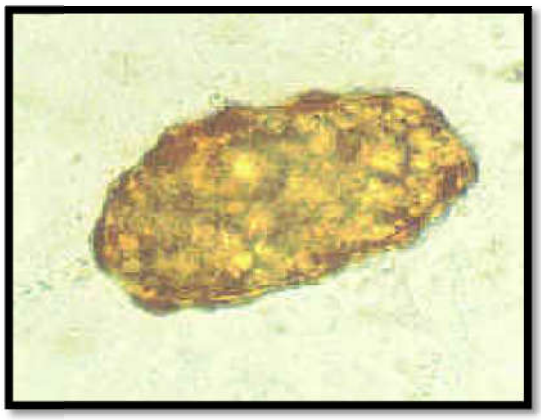
Egg: There are three kinds of the eggs; fertilized eggs, unfertilized eggs and decorticated eggs. We usually describe an egg in 5 aspects: size, color, shape, shell and content.

1. Fertilized eggs: broad oval in shape, brown in color, an average size 60× 45µm. The shell is thicker and consists of ascaroside, chitinous layer, fertilizing membrane and mammillated albuminous coat stained brown by bile. The content is a fertilized ovum. There is a new-moon(crescent) shaped clear space at the each end inside the shell.

2. Unfertilized egg: Longer and slender than a fertilized egg. The chitinous layer and albuminous coat are thinner than those of the fertilized eggs without ascaroside and fertilizing membrane. The content is made of many refractable granules various in size.

3. Decorticated eggs: Both fertilized and unfertilized eggs sometimes may lack their outer albuminous coats and are colorless.

A fertilized *Ascaris* egg, still at the unicellular stage, as they are when passed in stool. Eggs are this stage when passed in the when passed in stool. Eggs are normally at this stage when passed in the stool.



The chitinous layer and albuminous coat are thinner than those of the fertilized eggs without ascaroside and fertilizing membrane. The content is made of many refractable granules various in size.

Diagnosis: The diagnosis is established by finding characteristic eggs in the feces. Eggs are elliptical in shape, measure 30 by 50 pm, and have a rough, wavy, albuminous coat over their shell. They are highly resistant and may remain viable up to 6 years. The pulmonary phase may be diagnosed by finding larvae and eosinophils in sputum. Occasionally, patients pass adult worms from the rectum and the nose following migration through the nares in febrile patients the mouth in vomits.

Clinical signs: Most infections are asymptomatic, although nonspecific gastrointestinal tract symptoms may occur in some patients. Pulmonary involvement . During the larval migratory phase, an acute transient pneumonitis (Loffler syndrome) associated with fever, cough and wheezing (hypersensitivity) and marked eosinophilia may occur. Heavy worm loads can lead Acute intestinal

obstruction may develop in patients with heavy infections. Children are more prone to this complication because of the smaller diameters of the intestinal lumen and heavy worm burden. To obstruction of appendix. *Ascaris lumbricoides* has been found in the appendiceal lumen in patients with acute appendicitis, but a causal relationship is uncertain. The adult worms can be stimulated to migrate by stressful conditions (eg, fever, illness, or anesthesia) and by some antihelmintic drugs. Worm migration can cause peritonitis, secondary to intestinal wall penetration Common bile duct obstruction resulting in acute obstructive jaundice.

Pathogenicity: *Ascaris lumbricoides* is the largest of the nematode parasites that colonize humans; females can grow to 49 cm (19 inches).The name "lumbricoides" alludes to its resemblance to earth worms (*Lumbricus* sp.). The parasite is acquired by ingesting its eggs. *Ascaris* can cause intestinal obstruction and pancreatico biliary symptoms. Treatment is albendazole.

Types of Ascaris

A. suum

Pathogenesis of *A. suum*

Young pigs are commonly affected. Heavy infection in neonatal pigs causing pneumonia, cough and exudate in the lungs. These symptoms similar to that of symptoms occurs in "thumps" or piglet anemia. Varying degrees of fibrosis occurs in liver, the fibrosis is localized in the form of "Milk spot". Petechieal hemorrhage occurs in lungs. Desquamation of alveolar epithelium, edema of lungs and eosinophilic

infiltration is seen. L4 feed on caecal mucosa while adult worms feed on intestinal contents. The chief clinical feature of oxyuriasis is ‘anal pruritus” produced by egg laying females.

Clinical signs: Restlessness, improper feeding and dull coat, animal rubs its base of the tail against any suitable hard object causing hair to break off and the tail to acquire an ungroomed ‘rat-tailed appearance’.

P. equorum

Characters and Pathogenesis: The host of this parasite was small intestine of horses, with separated sex and Females longer than males. The head with white or creamy colour. The mouth part with three large main lips and three small lips at the middle, while, the tail of male with lateral alae. The vulva located in the end of first quarter of length of body. The eggs semi spherical and it has external pitted rough sheath.

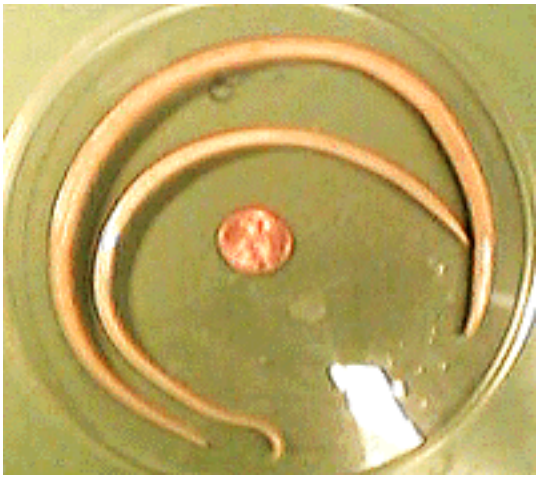
Pathogenesis and clinical signs of *P. equorum*: Foals 3 to 9 months of age are commonly affected. Heavy infection causes coughing and circulating eosinophilia. Adult worm causes catarrhal enteritis and foetid diarrhea. General malaise, debility and pot belly may occur. Hair Coat becomes very rough, sometimes the adult worms enter into the bile duct causing jaundice and also the adult worm enter into the intestinal wall causing generalized or localized peritonitis.

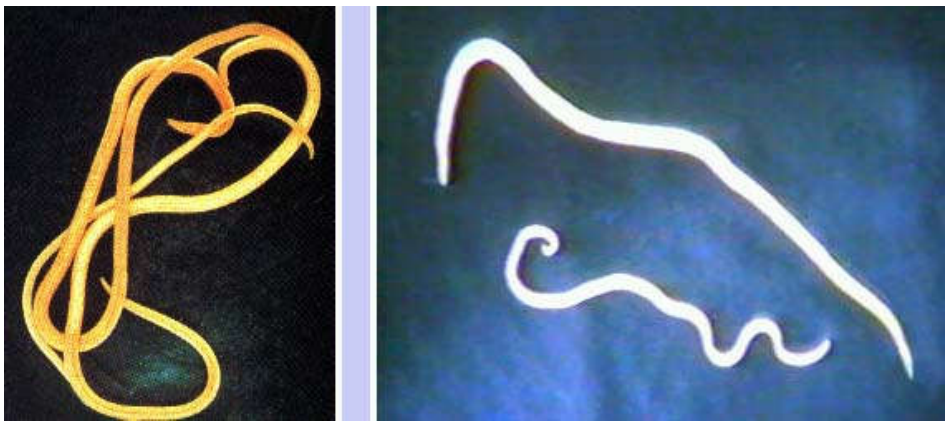
Diagnosis: Demonstration of eggs in faeces. Demonstration of larvae in sputum especially for *A. suum*. Based on the clinical signs. During meat inspection, the encysted larvae seen as a

white spot can be visible to naked eye. For routine examination in meat industry small piece of muscle pressed between the slide, then viewed under trichino scope. Digest the muscle piece in acid pepsin then put in the Baermann's apparatus for larval separation. Also, there are among the most important parasites that occur in dogs and cats.

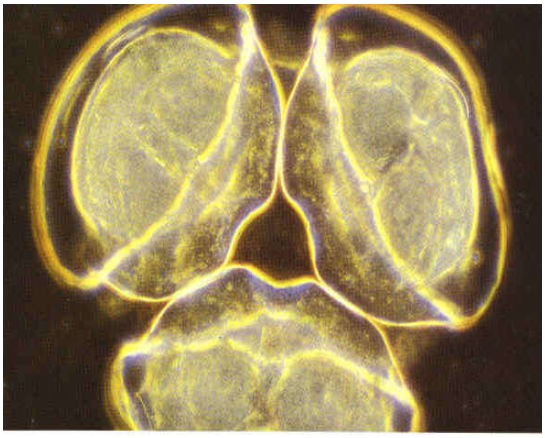
There are 3 species of **Order Ascaroidea** of most concern:

1. *Toxocara canis*
2. *Toxocara cati*
3. *Toxascaris leonine*

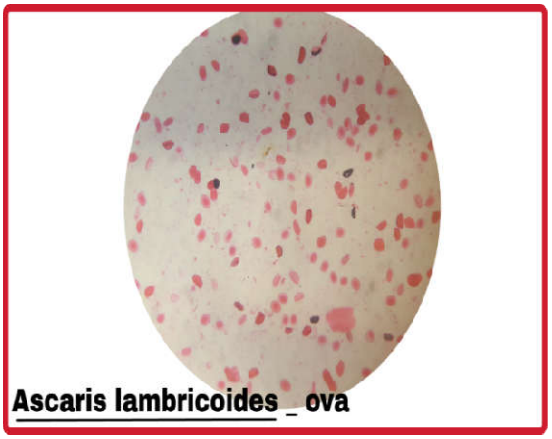


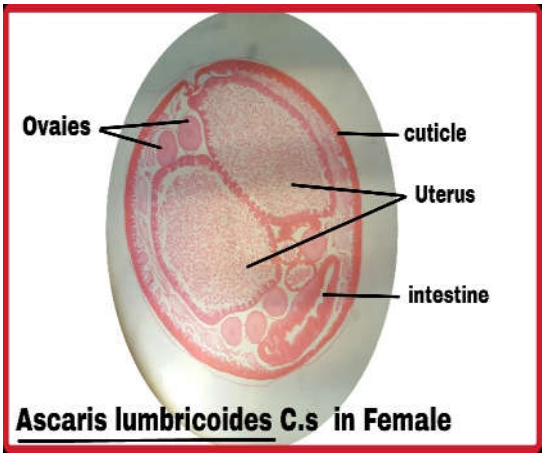


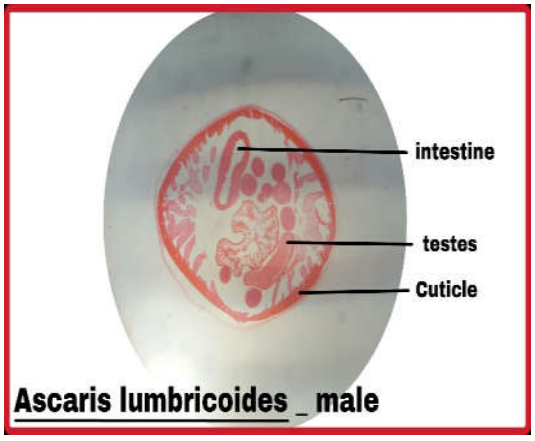
Adult worm of *A. lumbricoides*

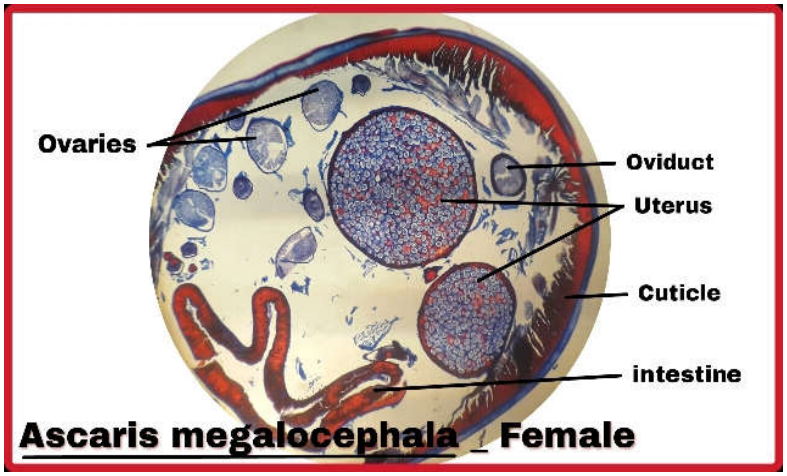


The lips of *A. lumbricoides*









Toxocara canis

Kingdom: Animelia

Phylum: Nematohelminthes

Order: Ascaridida

Family: Ascarididae

Genus*: Toxocara canis, Toxocara cati, Toxascaris leonine*

Name of Disease: Toxocariasis (dog round worms)

Intermediate and final host: No intermediate host, while final host was dogs, cats and other carnivorous.

Accidental Host: Humans and other mammals, Children more susceptible than adults.

Geographical Distribution: Worldwide especially on stray dogs and cats

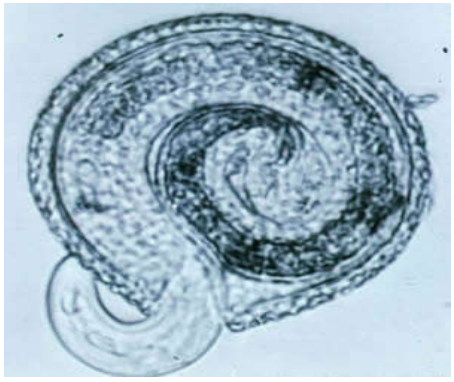
Site of Infection: Occurs in the small intestine of final hosts.

Diagnostic feature for parasite and Morphology: Having 3 main lips, with large cervical alae. The male tail has a terminal narrow appendage(finger project) and caudal alae. The female genital organs extend anteriorly and posteriorly to the vulva. Eggs are spherical with thick, fine pitted shells.

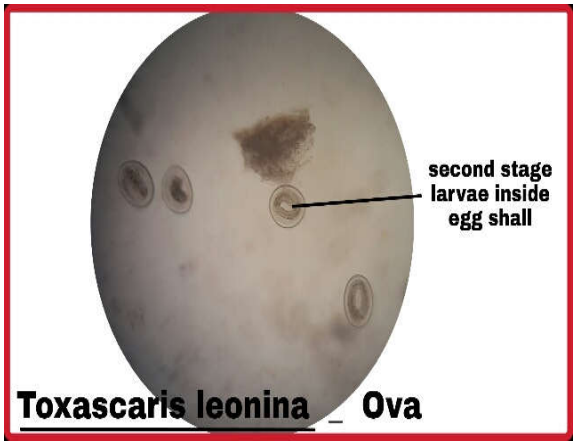
Egg: Measured 85µm x 75µm, have thick brown shell

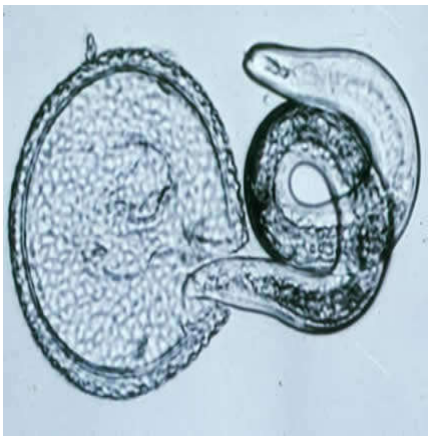
Toxascaris leonine

The persist habitant in small intestine of dogs and fox, females longer than males, present large cervical alae anterior end curved to the posterior end, tail of male has narrow terminal appendix and caudal alae, present two specula. The eggs are sub globular, it has smooth external sheath.



Second stage larvae inside egg shall of *Toxocara canis*

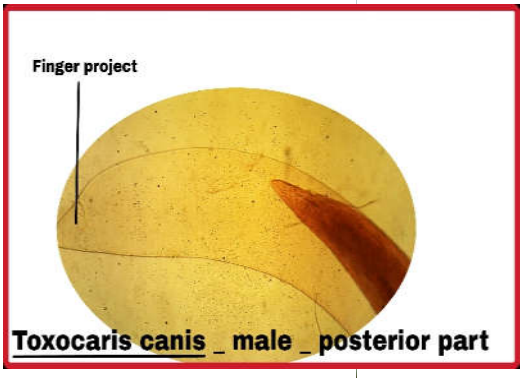


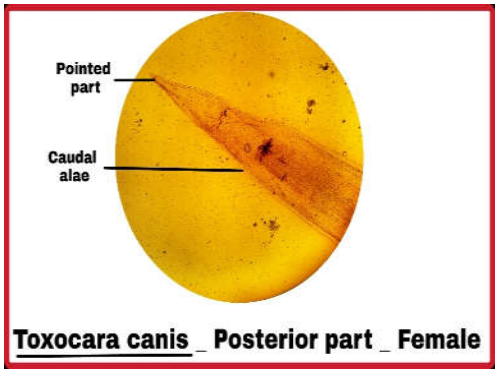


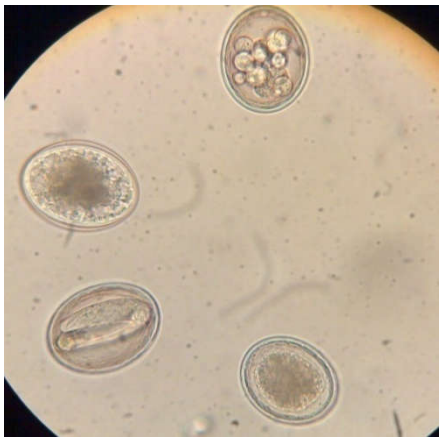
Larvae inside egg shall Larvae outside egg shall

-Adults

Female; 5 to 18 cm long, while, adult male; 4 to 10 cm long.



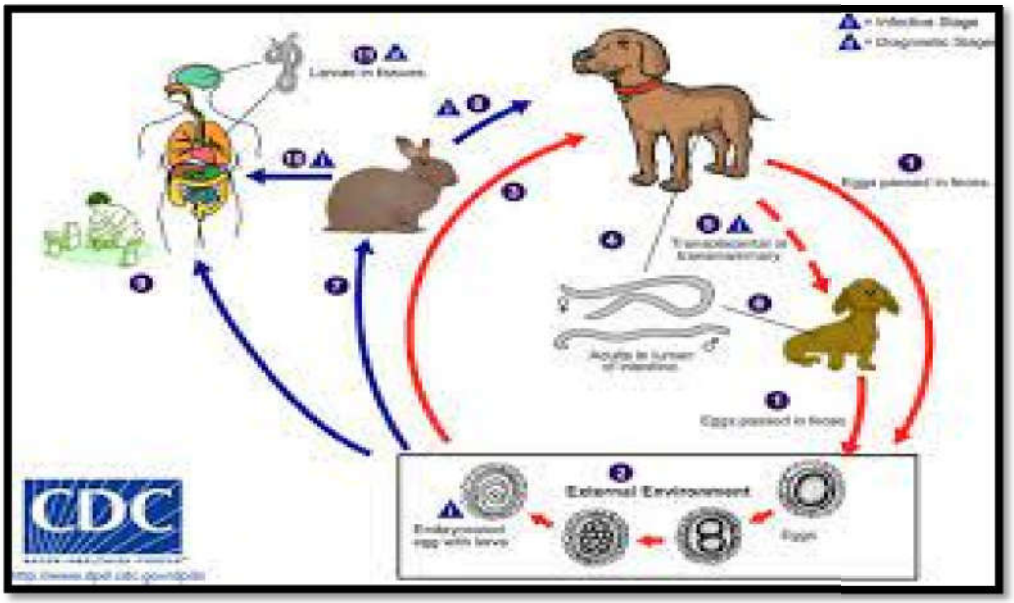




Anterior with three lips of *Toxocara canis* Embryonated ova of *Toxascaris leonine*

Life Cycle: The *Toxocara canis* life cycle normally involves dogs; humans are infected only accidentally. Un embryonated eggs are passed in feces of dogs (the definitive host). In the environment, eggs embryonated and become infective. After eggs are ingested by a dog, they hatch and release larvae, which penetrate the intestinal wall. In younger dogs, the larvae migrate through the lungs and bronchial tree; larvae are coughed up, swallowed, and returned to the small intestine, where they

mature. Adult female worms deposit eggs in the small intestine. Although older dogs may be infected in the same way, larvae are more likely to encyst in tissues. Encysted stages are reactivated in female dogs during late pregnancy and infect the puppies via the transplacental or transmammary route. As a result, adult worms become established in the small intestine of the puppies (a major source of environmental contamination). *T. canis* can also be transmitted through ingestion of transport hosts. Eggs ingested by small mammals (eg, rabbits) hatch into larvae, which penetrate the intestinal wall and migrate into various tissues where they encyst. The life cycle is completed when dogs eat these hosts, and the larvae develop into egg-laying adult worms in the small intestine. Humans are accidental hosts who become infected by ingesting infective eggs in contaminated soil or infected transport hosts. After ingestion, the eggs hatch into larvae. The larvae penetrate the intestinal wall and are circulated to various tissues (eg, liver, heart, lungs, brain, muscle, eyes).



Ascaridia galli

Kingdom: Animelia

Phylum: Nematohelminthes

Class: Chromadorea

Order: Ascaridida

Family: Ascarididae

Genus*: Ascaridia galli*

Name of Disease: Ascardiosis

Intermediate and final host: Fowl, guinea fowl, turkey , goose and various wild birds.

Geographical Distribution: Worldwide distribution.

Site of Infection: Small intestine

Diagnostic feature for parasite and Morphology:

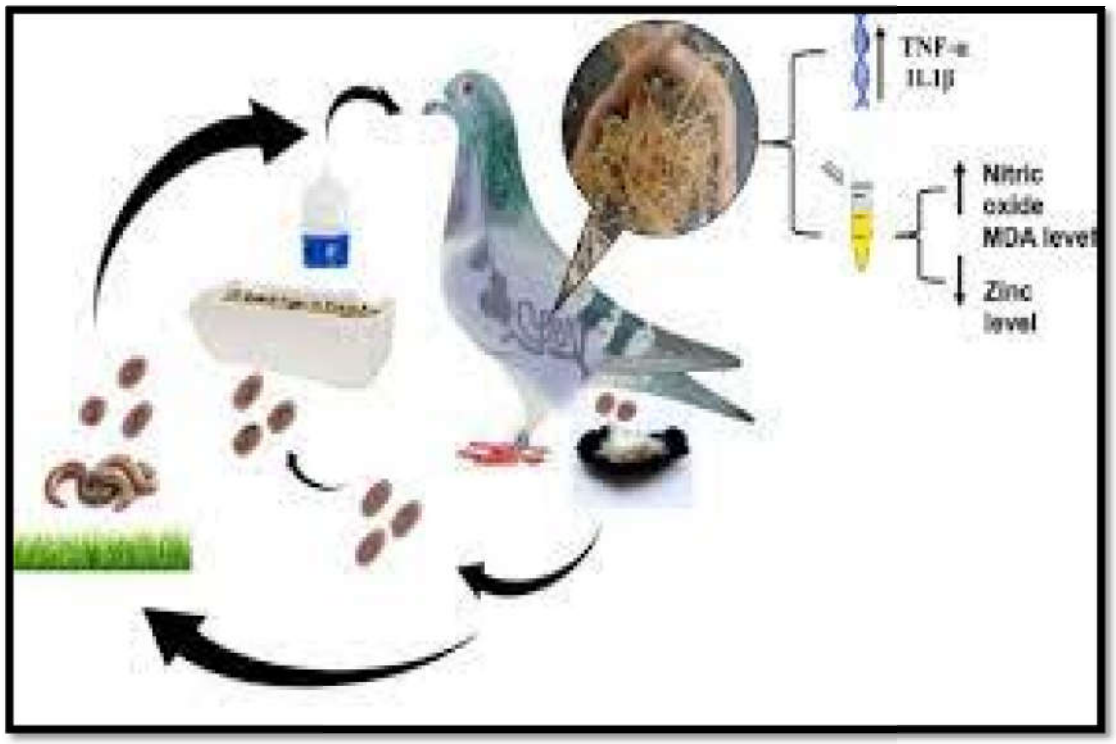
It is the largest nematode in birds, with females measuring 72 to 112 mm long. The body is semitransparent, creamy-white, and cylindrical. The anterior end is characterized by a prominent mouth, which is surrounded by three large, trilobed lips. The edges of the lips bear teeth-like denticles. The body is entirely covered with a thick proteinaceous structure called cuticle. The cuticle is striated transversely through the length of the body and cuticular alae are poorly developed. Two

conspicuous papillae are situated on the dorsal lip and one on each of the sub ventral lips. These papillae are the sensory organs of the nematode. *A. galli* is diocious with distinct sexual dimorphism. Females are considerably longer and more robust, with a vulva opening at the middle portion (roughly midway from anterior and posterior ends) of the body and anus at the posterior end of the body. The tail end of females is characteristically blunt and straight. Males are relatively shorter and smaller (measuring 50 to 76 mm long), with a distinct pointed and curved tail. Ten pairs of caudal papillae are found towards the tail region of the body, and they are arranged linearly in well-defined groups such as pre cloacal (three pairs), cloacal (one pair), post cloacal (one pair), and sub terminal (three pairs) papillae. Eggs found in the feces of infected birds are oval with smooth shells and measure 73–92 by 45–57 microns.

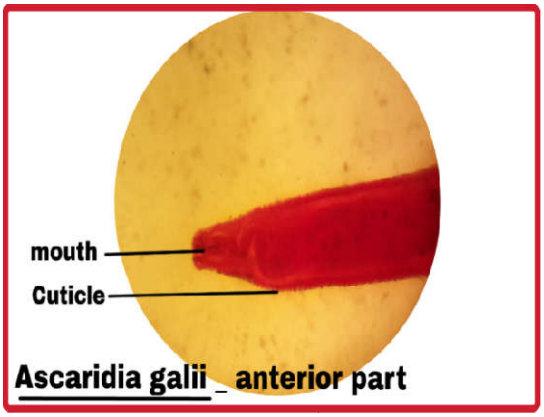
Life Cycle

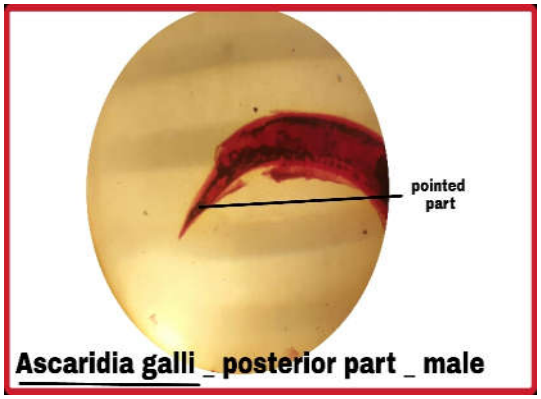
The life cycle of *A. galli* is direct in a single host, involving two principal populations, namely the sexually mature parasite in the gastrointestinal tract and the infective stage (L2). The eggs are oval in shape and have thick, albuminous shells that are highly resistant to desiccation and persist for a long time in the environment. Larvae do not hatch, but moult inside the eggs until they reach the L2 stage. This can take about two weeks, but the period depends on other factors such as the weather condition. The lifecycle is completed when the infective eggs are ingested by new hosts through contaminated water or feed. The eggs

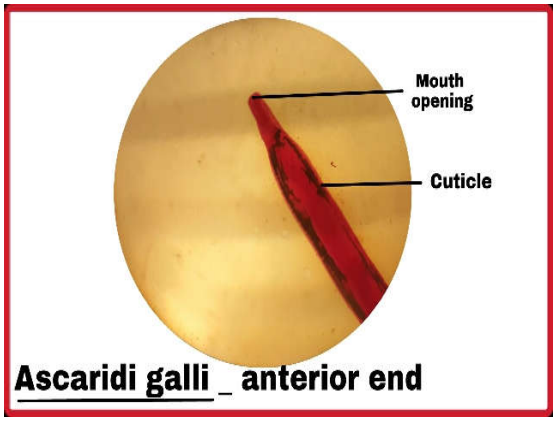
containing the L2-larvae-passive are mechanically transported to the duodenum, where they molt and become larvae stage 3 and then larvae stage 4. The infective eggs are ingested by a chicken; when it reaches the proventriculus, it hatches. Temperature, carbon dioxide levels, and pH are thought to be triggering factors that signal the larva to hatch from its egg. The larva then burrows into the mucosal lining of the small intestine, where it undergoes two additional molts. In this phase of their lifecycle, these worms cause the most damage to their hosts. They then re-enter the small intestine and develop into adults, where they live their lives out feeding on gut content and making a vast number of eggs that would then be excreted by a host and free to continue their lifecycle. If the animal is able to mount an immune response to the larvae, i.e. from pre-exposure, the larvae do not develop into adults, but hide in the mucosa of the small intestine. This is common for infection of older birds. Transport hosts such as earthworms are thought to play a role in transmission of *A. galli*, hence free range birds tend to have a higher risk of infection.



The nematode infects fowl of all ages, but the greatest degree of damage is often found in birds under 12 weeks of age. Heavy infection is the major cause of weight depression and reduced egg production in poultry husbandry. In severe infections, intestinal blockage can occur. Un thriftiness, drooping of the wings, bleaching of the head, and emaciation are seen. Infection also causes loss of blood, reduced blood sugar content, increased urates, shrunken thymus glands, retarded growth, and greatly increased mortality. In heavy infections, adult worms may move up the oviduct and be found in hens' eggs, and sometimes they are also found in the birds' feces.


mouth
Cuticle
Ascaridia galii _ anterior part


pointed part
Ascaridia galli _ posterior part _ male


Mouth opening
Cuticle
Ascaridi galli _ anterior end

Heterakis gallinarum

Phylum: Nematohelminthes

Class: Chromadorea

Order: Ascaridida

Family: Heterakidae

Genus*: Heterakis*

Species: *Heterakis gallinarum*

Name of Disease: Heterakiasis

Intermediate and final host: Fowl, guinea fowl, turkey , goose and various wild birds.

Geographical Distribution and Epidemiology: *H. gallinarum* is geographically distributed worldwide, commonly found in chickens, domesticated turkeys, and many other species of fowl, primarily of poultry. Their eggs are found to live for years in soil making it difficult to eliminate *H. gallinarum* from a domestic flock. Earthworms may ingest the eggs of *H. gallinarum* and contribute to the cause of infections in poultry. Although the eggs are themselves infective, they can develop further into a second infective larval stage. This development occurs around 27 °C and takes 2–4 weeks.

Site of Infection: lumen of cecum

Diagnostic feature for parasite and Morphology: *Heterakis gallinarum* is a nematode parasite that lives in the cecum of some galliform birds, particularly in ground feeders such as domestic chickens and turkeys. It causes infection that is mildly pathogenic. However, it often carries a protozoan parasite *Histomonas meleagridis* which causes of histomoniasis (blackhead disease). Transmission of *H. meleagridis* is through the *H. gallinarum* egg. *H. gallinarum* is about 1–2 cm in length with a sharply pointed tail and a pre anal sucker. The parasite is a diocious species with marked sexual dimorphism. Males are smaller and shorter, measuring around 9 mm in length, with a unique bent tail. Females are stouter and longer, measuring roughly 13 mm in length, with a straight tail end.

Life Cycle:

Direct lifecycle involving birds suchas chickens, turkeys, ducks, geese, grouse, guineafowl, partridges, pheasants,and quails as def initive hosts. Eggs of *H. gallinarum* are passed in feces by the host. At optimal temperature (22 °C), they become infective in 12–14 days and remain infective for years in soil. Upon ingestion by a host, the embryonated eggs hatch into second-stage juveniles in the gizzard or duodenum, and are passed to the cecum. Their development is completed in the lumen, but some may enter the mucosa and remain for years without further development. The prepatent time is 24–30 days. Earthworms and houseflies are considered paratenic hosts, as they can ingest the egg in feces and a juvenile may hatch in

tissues, which stays dormant until eaten by birds.

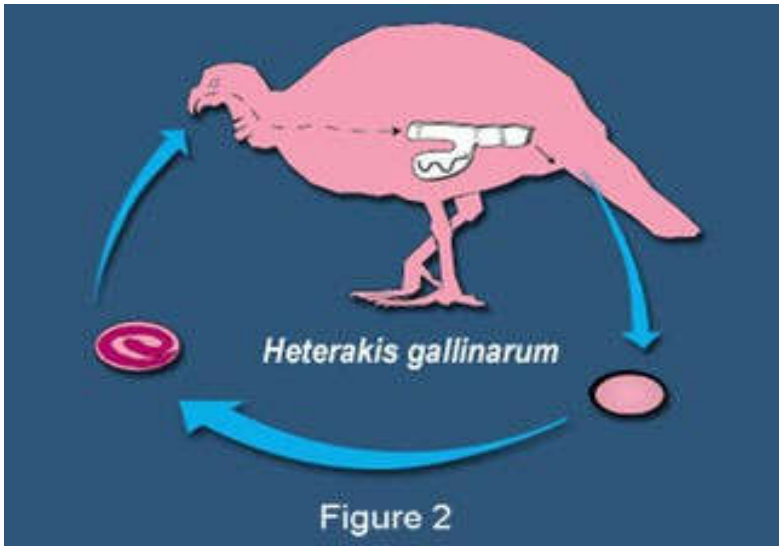
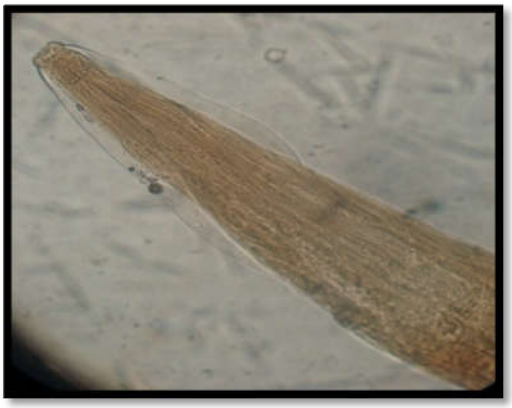


Figure 2

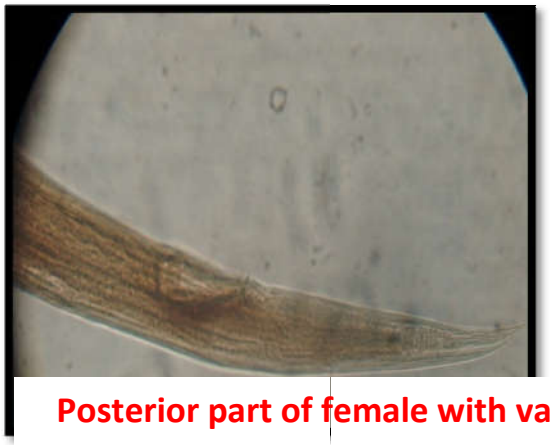
Pathogenicity: *Heterakis gallinarum* infection is itself is mildly pathogenic. However, *H. gallinarum* plays the role of carrier in the lifecycle of *Histomonas meleagridis*, the causal pathogen of enterohepatitis "blackhead" of turkeys. *H. meleagridis* stays viable while inside the egg of *H. gallinarum*. Heavy infection in pheasants indicated gross lesions characterized by congestion, thickening, petechial hemorrhages of the mucosa, in tissues caption, and nodules in the cecal wall. In addition under microscopy, chronic
diffuse typhlitis, haemosiderosis, granulomas with necrotic center in the submucosa and leiomyomas in the submucosa, muscular and serosa associated with immature *H. gallinarum* worms were observed.

Diagnosis: Primary infections are usually not apparent. Secondary infections are characterized by the formation of

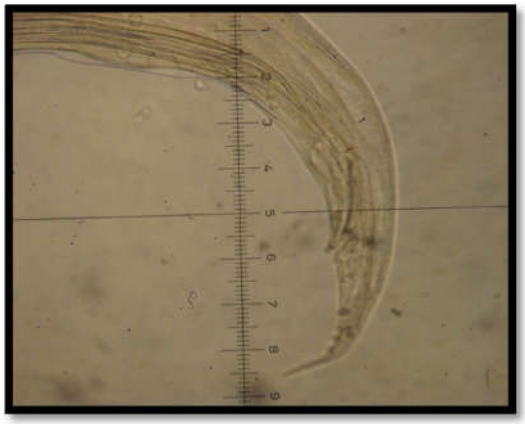
nodules in the cecum and the submucosa of the cecum. During heavy infections, intestinal walls may thicken and exhibit marked inflammation. In egg-laying hens, heavy infection significantly reduces egg production. Diagnosis commonly is through the presence of eggs in host feces.



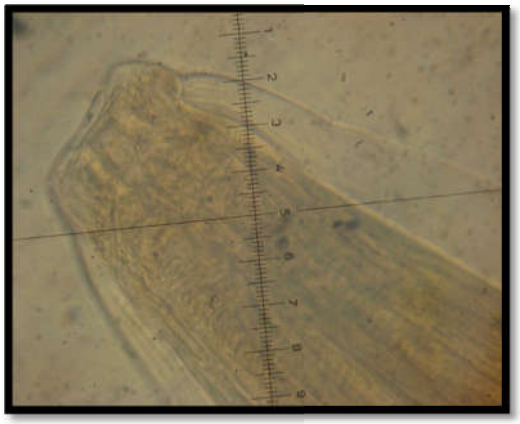
Anterior part of female



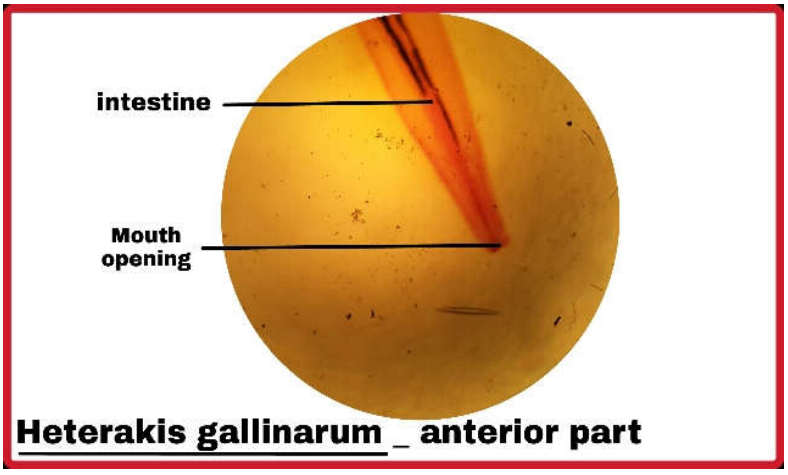
Posterior part of female with valve



Posterior part of male



Anterior part with lips



Entrobius vermicularis

Phylum: Nematohelminthes

Class: Secernentea

Order: Ascaridida

Family: Oxyuridae

Genus*: Entrobius*

Species: *Entrobius vermicularis*

Name of Disease: Entrobiasis

Intermediate and final host: humans are the only normal host of *vermicularis* other species infect goats, sheep, horses, rabbits, mice, rats and others. No intermediate host (direct life cycle)

Geographical Distribution and Epidemiology: Worldwide distribution, but more in temperate areas. Clothing and beading become infested quickly, also found in dust in school rooms and cafeterias. Most commonly infection occurs form soiled fingers or objects or eating soil (esophagi). Airborne eggs can also be inhaled and swallowed. For example 20-30% of elementary school children in the United States. Worldwide it is estimated that 500 million people are infected

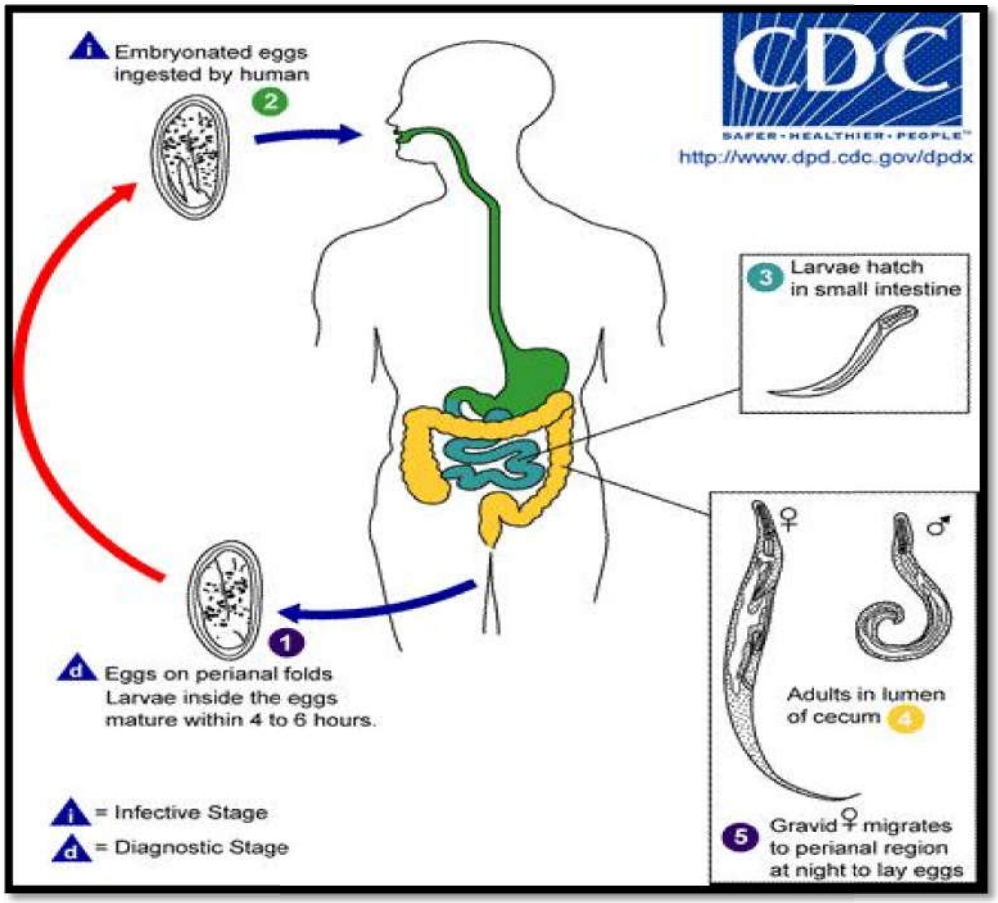
Site of Infection: Large intestine of hosts.

Diagnostic feature for parasite and Morphology:

Entrobius vermicularis is known as the human pin worm of temperate climate zones. Most common anywhere poor hygiene is practiced. Adult has three lips surrounding the mouth and a bulb on the posterior end of the esophagus. Males are 1 to 4 mm long and have the posterior ends strongly curved (button hook) and have a single Spicule which is 100 to 141 um long. While, Females measure 8 to 13 mm long and have the posterior end extended into a long, slender point. Each has two uteri that can contain thousands of eggs that are elongate-oval and flattened on one side, measuring 50 to 60 um by 20 to 30 um.

Life Cycle: The entire life cycle, from egg to adult, takes place in the human gastrointestinal tract of a single host, from about 2–4 weeks or about 4–8 weeks. *E. vermicularis* molts four times; the first two within the egg before hatching and two before becoming an adult worm. Although infection often occurs via ingestion of embryonated eggs by inadequate hand washing or nail biting, inhalation followed by swallowing of airborne eggs may occur rarely. The eggs hatch in the duodenum (i.e., first part of the small intestine). The emerging pinworm larvae grow rapidly to a size of 140 to 150 µm, and migrate through the small intestine towards the colon. During this migration, they moult twice and become adults. Females survive for 5 to 13 weeks, and males about 7 weeks. The male and female pinworms mate in the ileum (i.e., last part of the small intestine),where after the male pinworms usually die, and are passed out with stool. The gravid female pinworms settle in the ileum, caecum (i.e., beginning of the large intestine), appendix and ascending colon, where they attach themselves to the mucosa and ingest

colonic contents. Almost the entire body of a gravid female becomes filled with eggs. The estimations of the number of eggs in a gravid female pinworm range from about 11,000to 16,000. The egg-laying process begins about five weeks after initial ingestion of pinworm eggs by the human host. The gravid female pinworms migrate through the colon towards the rectum at a rate of 12 to 14 cm per hour. They emerge from the anus, and while moving on the skin near the anus, the female pinworms deposit eggs either through (1) contracting and expelling the eggs, (2) dying and then disintegrating, or (3) bodily rupture due to the host scratching the worm. After depositing the eggs, the female becomes opaque and dies. The female emerges from the anus to obtain the oxygen necessary for the maturation of the eggs.



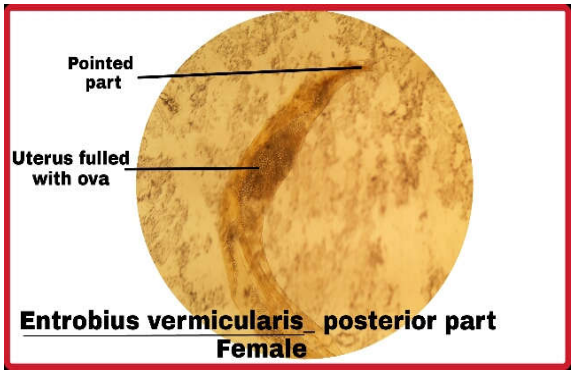
Entrobiasis is the medical condition of being infected with pinworms. The Pathogenicity depending on the phase as:

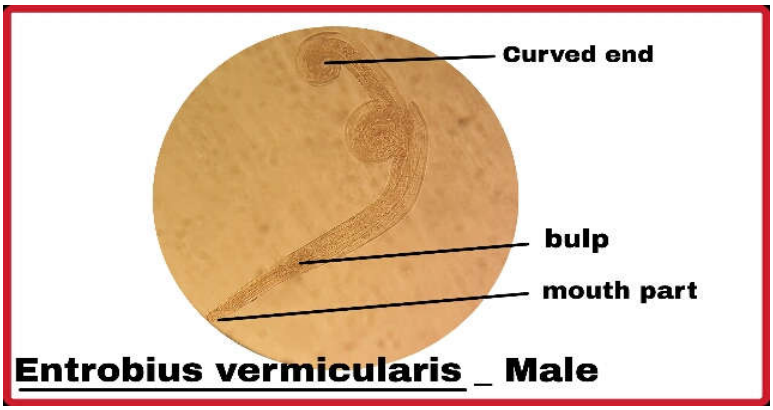
Asymptomatic other than itching of the anus, due to excretions of the female worm.

Children may experience loss of appetite, abdominal pain, insomnia and restlessness are the usual symptoms associated with pin worm infections

Vaginitis may occur in young girls with pin worm infections.

Diagnosis: often made clinically by observing the female worm (or many worms) in the peri-anal region, but can also be made using the "scotch-tape" test, or anile swab. A good hygiene, sanitation of clothing beading and environment





Order: Strongylida

General Characteristics

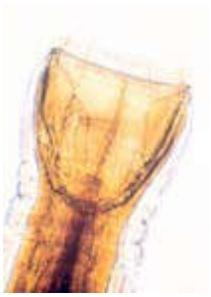
The posterior end of male is modified to be copulatory bursa which consist of dorsal, lateral and ventral expansion of the body cuticle (lobes) supported by muscular process called rays. The size of bursa lobe may be small or large than other or equal. Also the spicules of male tend to be long, thin, thick and flexible or may be even short or stout, the spicules are assist in mating.

The common Genera:

Oesophagostomum spp., Stephanurus spp., Strongylus spp. Syngamus

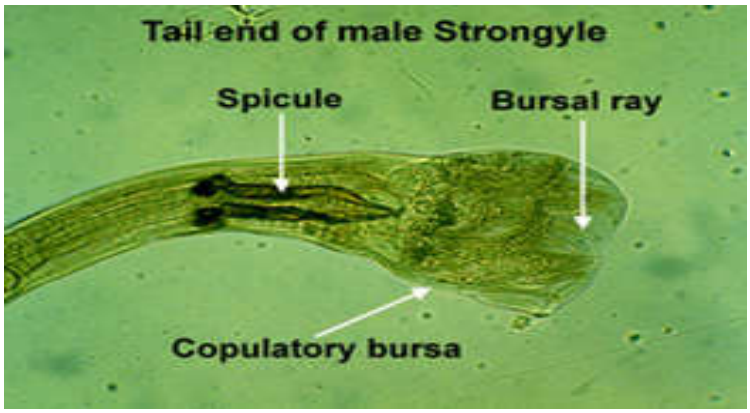
-Hosts: Horses, Dogs, Cats, Cow, Birds and different mammals.

2 teeth **3 teeth** **no teeth**



Strongylus vulgaris ***Strongylus equines*** ***Strongylus edentates***

Strogylidae copulatory bursa :



The posterior end of Male is modificate to be copulatory bursa which consist from dorsal , lateral and ventral expansion of the body cuticle (lobes) supported by muscular process called rays, The size bursa lobe may be small or large than other or equal. Also the spicules of male tend to be long , thin , thick and flexible or may be even shorter or stouter , the spicules are assist in mating which is supported by gubernaculum.

Strongyloides stercoralis

Phylum: Nematohelminthes

Class: Chromadorea

Order: Rhabditida

Family: Strongylidae

Genus: Strongyloides

Species: *Strongyloides stercoralis*

Name of Disease: A human pathogenic parasitic roundworm causing the disease Strongylodiasis.

Intermediate and final host: The genus Strongyloides contains 53 species, and *S. stercoralis* is the type species. *S. stercoralis* has been reported in other mammals, including cats and dogs. However, it seems that the species in dogs is typically not *S. stercoralis*, but the related species *S. canis*. Non-human primates are more commonly infected with *S. fuelleborni* and *S. cebus*, although *S. stercoralis* has been reported in captive primates. Other species of *Strongyloides* that are naturally parasitic in humans, but with restricted distributions, are *S. fuelleborni* in central Africa and *S. kellyi* in Papua New Guinea.

Geographical Distribution and Epidemiology: Worldwide

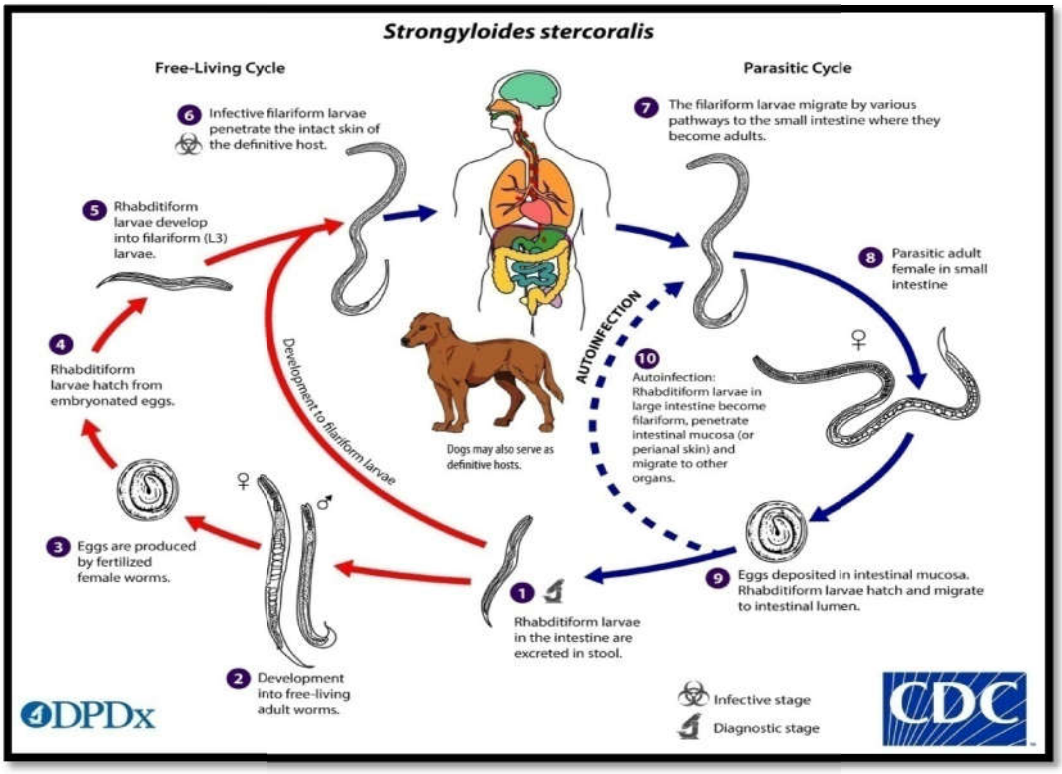
distribution.

Site of Infection: Small intestine

Diagnostic feature for parasite and Morphology: Whereas males grow to only about 0.9 mm (0.04 in) in length, females can grow from 2.0 to 2.5 mm (0.08 to 0.10 in). Both sexes also possess a tiny buccal capsule and cylindrical esophagus without a posterior bulb. In the free-living stage, the esophagi of both sexes are rhabditiform. Males can be distinguished from females by two structures:
the spicules and gubernaculum.The *Strongyloidesstercoralis* nem atode can parasitize humans. The adult parasitic stage lives in tunnels in the mucosa of the small intestine.

Life Cycle: The strongyloid's life cycle is heterogenic—it is more complex than that of most nematodes, with its alternation between free-living and parasitic cycles, and its potential for autoinfection and multiplication within the host. The parasitic cycle is homogonic, while the free-living cycle is heterogenic. The heterogenic life cycle is advantageous to the parasite because it allows reproduction in the absence of a host. In the free-living cycle, the rhabditiform larvae passed in the stool can either molt twice and become infective fillariform larvae (direct development) or molt four times and become free-living adult males and females that mate and produce eggs from which rhabditiform larvae hatch. In the direct development, first-stage larvae (L1) transform into infective larvae (IL) via three molts. The indirect route results first in the development of free-living

adults that mate; the female lays eggs, which hatch and then develop into IL. The direct route gives IL faster (three days) versus the indirect route (seven to 10 days). However, the indirect route results in an increase in the number of IL produced. Speed of development of IL is traded for increased numbers. The free-living males and females of *S. stercoralis* die after one generation; they do not persist in the soil. The latter, in turn, can either develop into a new generation of free-living adults or develop into infective fillariform larvae. The fillariform larvae penetrate the human host skin to initiate the parasitic cycle.



While *S. stercoralis* is attracted to chemicals such as carbon dioxide or sodium chloride, these chemicals are not specific. Larvae have been thought to locate their hosts via chemicals in

the skin, the predominant one being urocanic acid, a histidine metabolite on the uppermost layer of skin that is removed by sweat or the daily skin-shedding cycle. Urocanic acid concentrations can be up to five times greater in the foot than any other part of the human body. Some of them enter the superficial veins and are carried in the blood to the lungs, where they enter the alveoli. They are then coughed up and swallowed into the gut, where they parasitize the intestinal mucosa of the duodenum and jejunum. In the small intestine, they molt twice and become adult female worms. The females live threaded in the epithelium of the small intestine and, by parthenogenesis, produce eggs, which yield rhabditiform larvae. Only females will reach reproductive adulthood in the intestine. Female Strongyloides reproduce through parthenogenesis. The eggs hatch in the intestine and young larvae are then excreted in the feces. It takes about two weeks to reach egg development from the initial skin penetration. By this process, *S. stercoralis* can cause both respiratory and gastrointestinal symptoms. The worms also participate in autoinfection, in which the rhabditiform larvae become infective fillariform larvae, which can penetrate either the intestinal mucosa (internal autoinfection) or the skin of the peri anal area (external autoinfection); in either case, the fillariform larvae may follow the previously described route, being carried successively to the lungs, the bronchial tree, the pharynx, and the small intestine, where they mature into adults; or they may disseminate widely in the body. To date, occurrence of autoinfection in humans with helminthes infections is recognized only in *Strongyloides stercoralis* and Capillaria

philippinensis infections. In the case of *Strongyloides*, autoinfection may explain the possibility of persistent infections for many years in persons not having been in an endemic area and of hyper infections in immunodepressed individuals.

Diagnosis: Locating juvenile larvae, either rhabditiform or fillariform, in recent stool samples will confirm the presence of this parasite. Other techniques used include direct fecal smears, culturing fecal samples on agar plates, sero diagnosis through ELISA, and duodenal fumigation. Still, diagnosis can be difficult because of the day-to-day variation in juvenile parasite load.

Trichuris trichiura (Whip worm)

Phylum: Nematohelminthes

Class: Adenophorea

Order: Trichocephalida

Family: Trichuridae

Genus*: Trichuris*

Species: *Trichuris trichiura*

Name of Disease: causes a disease called trichuriasis

Intermediate and final host: human ,Equine, Ruminants, dogs

Reservoir: Humans are the main, but not the only reservoir for *T. trichiura*. Recent research verified by the application of molecular techniques (PCR) that dogs are a reservoir for *T. trichiura*, as well as *T. vulpis.*

Vector: Non-biting cyclorrhaphan flies (*Musca domestica*, *M. sorbens*, *Chrysomya rufifacies*, *C. bezziana*, *Lucina cuprina*, *Calliphoravicina* and *Wohlfarthiamagnifica*) have been found to carry *Trichuris trichiura*.

Transmission: Humans can become infected with the parasite due to ingestion of infective eggs by mouth contact with hands or food contaminated with egg-carrying soil. However, there have also been rare reported cases of transmission of *T. trichiura* by

sexual contact. Some major outbreaks have been traced to contaminated vegetables (due to presumed soil contamination).

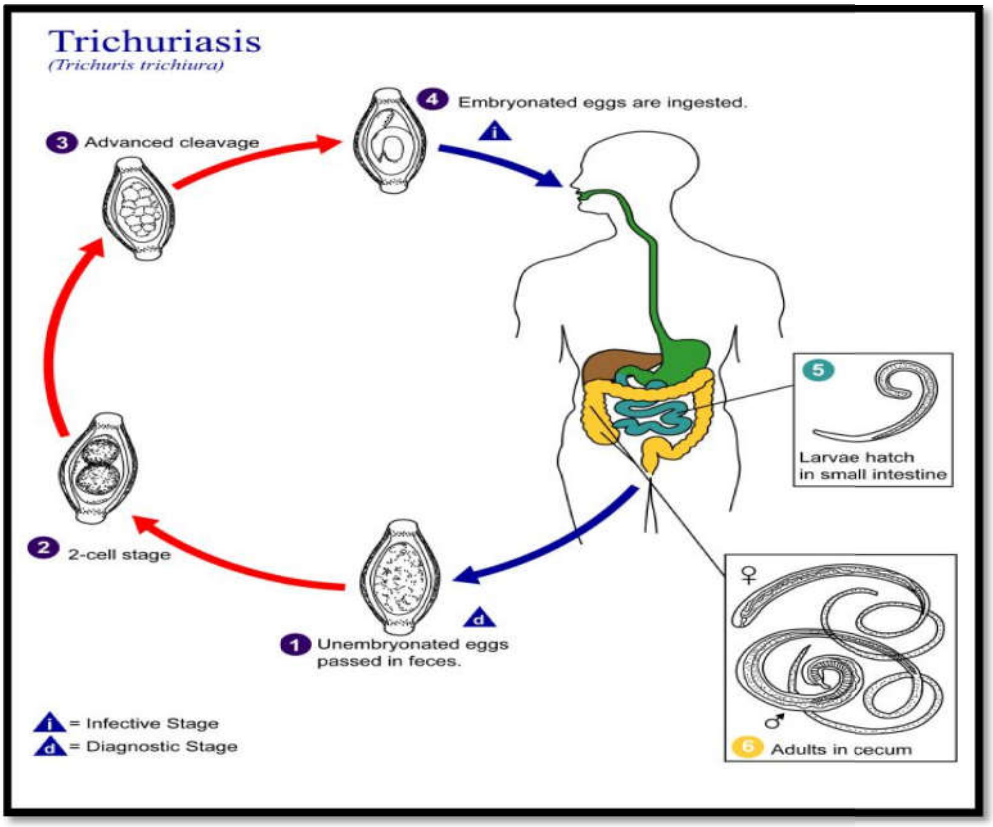
Sit of infection: Large intestine of their hosts

Geographical Distribution and Epidemiology: There is a worldwide distribution of *Trichuris trichiura*, with an estimated 1 billion human infections. However, it is chiefly tropical, especially in Asia and, to a lesser degree, in Africa and South America. Within the United States, infection is rare overall but may be common in the rural Southeast, where 2.2 million people are thought to be infected. Poor hygiene is associated with trichuriasis as well as the consumption of shaded moist soil, or food that may have been fecally contaminated. Children are especially vulnerable to infection due to their high exposure risk. Eggs are infective about 2–3 weeks after they are deposited in the soil under proper conditions of warmth and moisture, hence its tropical distribution.

Diagnostic feature for parasite and Morphology: The female with 35-50 mm in length, and consists of thin anterior three fifths and a thick posterior two fifths of the worm looks like a whip. With cellular esophagus occupying all the thin anterior part while the intestine and the single set of reproductive organs occur in the posterior thick part. The vulva situated at the junction of the thin & the thick parts. While, male measures 30- 45 mm in length, and consists of anterior thin part & posterior thick part, with single spicules inside a retractile spine, covered sheath. And eggs measures 1- 50-55 µm by 25 µm, barrel-shaped laid in one –cell stage embryo, also, it has a thin transparent inner

membrane and a golden- brown outer shell. Their characteristic eggs are barrel-shaped(lemon shape) have two polar plugs.

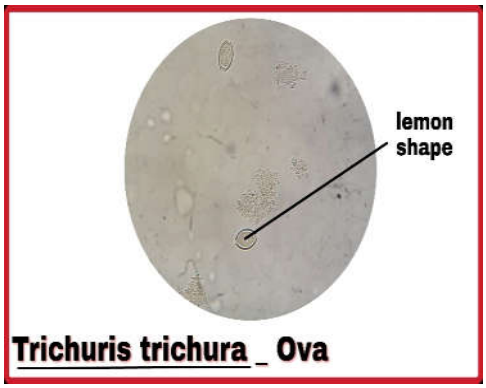
Life Cycle: The female *T. trichiura* produces 2,000–10,000 single-celled eggs per day. Eggs are deposited from human feces to soil where, after two to three weeks, they become embryonated and enter the "infective" stage. These embryonated infective eggs are ingested by hand-mouth or through for mites and hatch in the human small intestine, exploiting the intestinal microflora as a stimulus to hatching. This is the location of growth and molting. The infective larvae penetrate the villi and continue to develop in the small intestine. The young worms move to the caecum and penetrate the mucosa, and there they complete development as adult worms in the large intestine. The life cycle from the time of ingestion of eggs to the development of mature worms takes approximately three months. During this time, there may be limited signs of infection in stool samples, due to a lack of egg production and shedding. The female *T. trichiura* begin to lay eggs after three months of maturity. Worms commonly live for about one year, during which time females can lay up to 20,000 eggs per day.

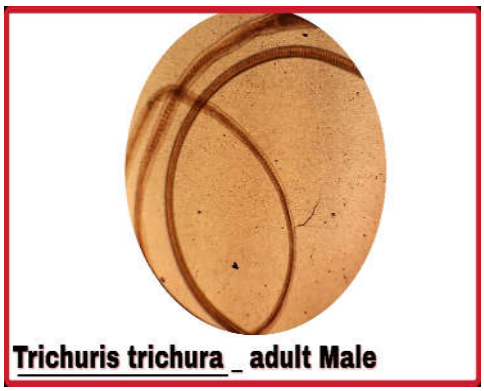


Pathogenicity: It can cause various symptoms and has been known to lead to death. *Trichuris trichiura* lives in the soil in its egg stage. Once it is ingested, it develops into the larvae stage, finally maturing into the adult stage in the intestines. Whipworm eggs are passed in the feces of infected persons, and if an infected person defecates outdoors or if untreated human feces is used as fertilizer, eggs are deposited on soil where they can mature into an infective stage. Ingestion of these eggs "can happen when hands or fingers that have contaminated dirt on them are put in the mouth or by consuming vegetables or fruits that have not been carefully cooked, washed or peeled." The eggs hatch in the small intestine, then move into the wall of the small intestine and develop. On reaching adulthood, the thinner end (the anterior of the worm) burrows into the large intestine, the thicker (posterior) end projecting into the lumen, where it mates with nearby worms.

The females can grow to 50 mm (2.0 in) long. *Trichuris trichiura* can cause the serious disease Trichuris dysentery syndrome, with chronic dysentery, anemia, rectal prolapsed, and poor growth.

Diagnosis: *T. trichiura* eggs are detected in stool examination. Eggs will appear barrel-shaped and un embryonated, having bipolar plugs and a smooth shell. Rectal prolapse can be diagnosed easily using defecating proctogram and is one of many methods for imaging the parasitic infection. Sigmoidoscopys show characteristic white bodies of adult worms hanging from inflamed mucosa (coconut cake rectum). Colonoscopy can directly diagnose trichuriasis by identification of the threadlike form of worms with an attenuated, whip-like end. Colonoscopy has been shown to be a useful diagnostic tool, especially in patients infected with only a few male worms and with no eggs presenting in the stool sample.





Trichinella spiralis

Phylum: Nematohelminthes

Class: Enoplea

Order: Trichocephalida

Family: Trichinellidae

Genus*: Trichinella*

Species: *Trichinella spiralis*

Name of Disease: causes a disease called Trichiniasis (Pork Tap worm).

Intermediate and final host: Definitive host: Dog, cats, pigs, rodents, rabbits, birds.

Intermediate host: Dog, cats, pigs, rodents, rabbits, birds.

Sit of infection: Small intestine of final hosts

Geographical Distribution and Epidemiology: Worldwide distribution especially human eating pork

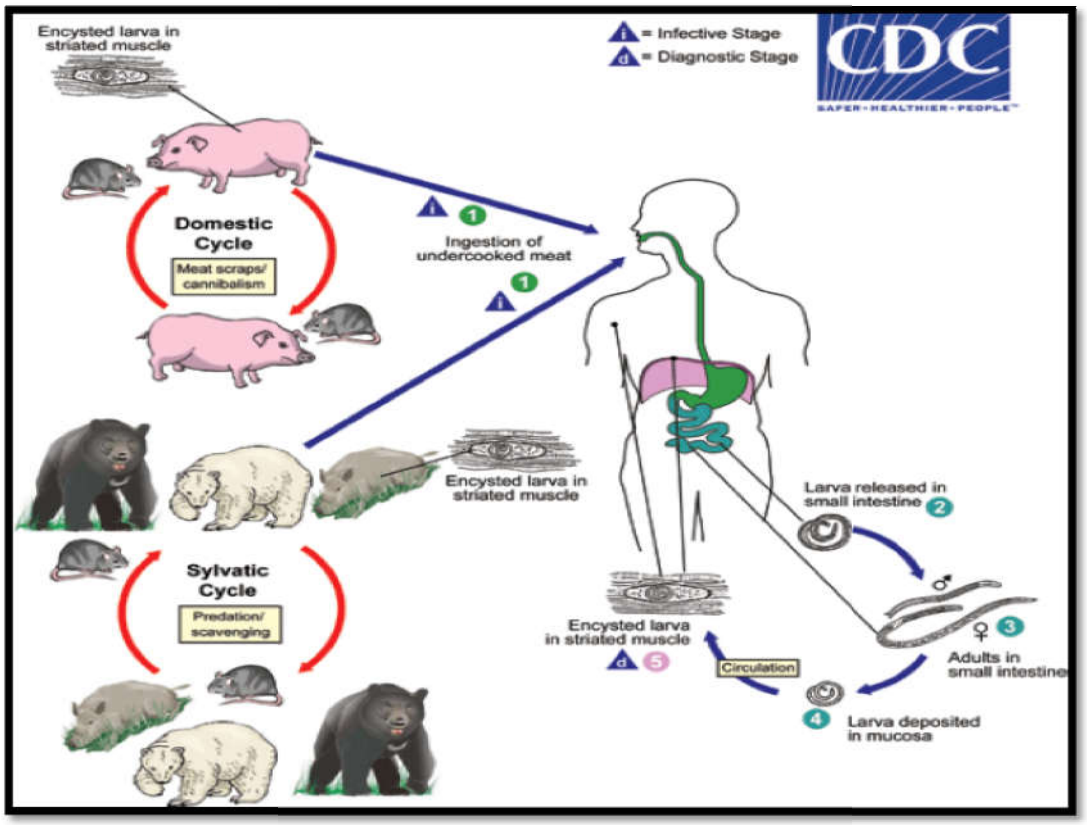
Diagnostic feature for parasite and Morphology: *Trichinella* species, the smallest nematode parasite of humans, has an unusual lifecycle, and are one of the most widespread and clinically important parasites in the world. The small adult worms mature in the small intestine of a definitive host, such as a pig. Each adult female produces batches of live larvae, which

bore through the intestinal wall, enters the blood (to feed on it) and lymphatic system, and are carried to striated muscle. Once in the muscle, they encyst, or become enclosed in a capsule. Humans can become infected by eating infected pork, horsemeat, or wild carnivores such as fox, cat, hyena or bear**.** 3-4 mm in length, with cellular esophagus, with one set of genitalia only; the ovary Situated at the posterior end, uterus proceeds to open in vulva at junction of anterior 5th with the rest of the body. At the posterior end, the uterus contains eggs gradually mature and then hatch before they emerge from the vulva (viviparous). While, male measures 1.5 mm in length, with cellular eesophagus. The cloacal opening is terminal and is guarded by a pair of conical papillae.

Encysted larvae: Larvae tend to lie in the longitudinal axis of the muscle fibers, each larvae is encysted singly or in heavy infections 2 or 3 larvae may encyst together. Larvae takes a spiral or figure 8 appearance. Cyst wall of fibrous tissue made up by reaction of the host.

Life Cycle: *Trichinella spiralis* can live the majority of its adult life in the intestines of humans. In lifecycle, *T. spiralis* adults invade the intestinal wall of a pig, and produce larvae that invade the pig's muscles. The larval forms are encapsulated as a small cystic structure within a muscle cell of the infected host. When another animal (perhaps a human) eats the infected meat, the larvae are released from the nurse cells in the meat (due to stomach pH), and migrate to the intestine, where they burrow into the intestinal mucosa, mature, and reproduce. Juveniles

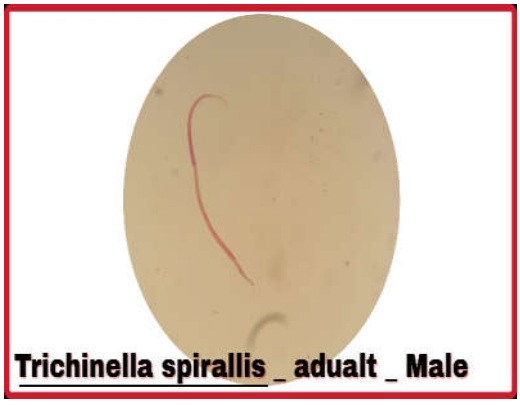
within nurse cells have an anaerobic or facultative anaerobic metabolism, but when they become activated, they adopt the aerobic metabolism characteristics of the adult. Female *Trichinella* worms live for about six weeks, and in that time can produce up to 1,500 larvae; when a spent female dies, she passes out of the host. The larvae gain access to the circulation and migrate around the body of the host, in search of a muscle cell in which to encyst. The migration and encystment of larvae can cause fever and pain, brought on by the host inflammatory response. In some cases, accidental migration to specific organ tissues can cause myocarditis and encephalitis that can result in death.

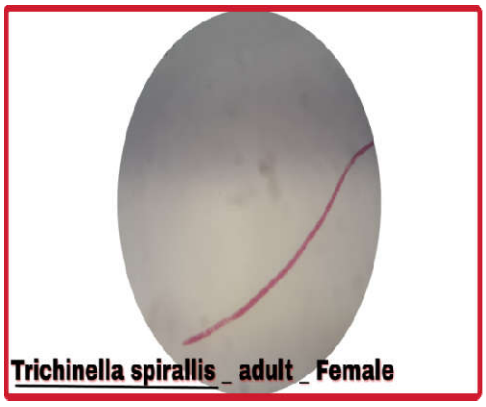


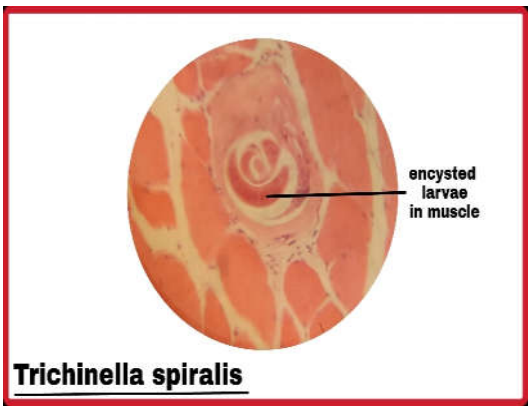
Pathogenicity: The migration of adult worms in the intestinal epithelium can cause traumatic damage to the host tissue, and the waste products they excrete can provoke an

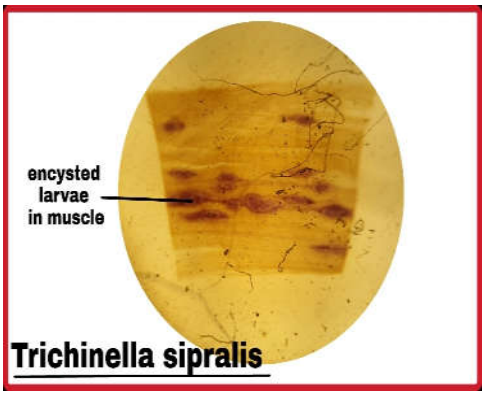
immunological reaction. The resulting inflammation can cause symptoms such as nausea, vomiting, sweating, and diarrhea. Five to seven days after the appearance of these symptoms, facial edema and fever may occur. Ten days following ingestion, intense muscular pain, difficulty breathing, weakening of pulse and blood pressure, heart damage, and various nervous disorders may occur, eventually leading to death due to heart failure, respiratory complications, or kidney malfunction, all due to larval migration. In pigs, infection is usually subclinical, but large worm burdens can be fatal in some cases

Diagnosis: Muscle biopsy may be used for trichinosis detection. Several immunodiagnostic tests are also available. Typically, patients are treated with either mebendazole or albendazole, but efficacy of such products is uncertain. Symptoms can be relieved by use of analgesics and corticosteroids.









Super family: Strongyloidea

Large buccal Intestinal tract capsule with cutting plates or teeth. Male bursate, while, female oviparous. Medium size, thick body

Sit of infection: intestine

The life cycle direct and the larvae on the ground ingested or penetrate skin. 2- Larvae may be sequestered in tissues of host.

Common Genera: *Ancylostoma spp., Uncinaria spp., Bunostomum spp., Necator americanum, Globocepha spp.*

Ancylostoma caninum

Phylum: Nematohelminthes

Class: Chromadorea

Order: Rhabditida

Family: **Ancylostomatidae**

Genus*: Ancylostoma*

Species: *Ancylostoma caninum*

Name of Disease: Ancylostomiasis (Hookworms).

Intermediate and final host: dog, fox , wolf and other wild carnivores .rarely in man.

Sit of infection: occur in the small intestine of final hosts

Geographical Distribution and Epidemiology: its cosmopolitan in distribution, being common in tropical and sub tropical zones in north Amerce ,Australia and Asia.

Diagnostic feature for parasite and Morphology: *Ancylostoma caninum* females are typically 14–16 mm (0.55–0.63 in) long and 0.5 mm (0.02 in) wide, while the males are smaller at 10–12 mm (0.39–0.47 in) in length and 0.36 mm (0.01 in) in width. Males have a copulatory bursa , which consists of spine-like spicules positioned on three muscular rays that grasp the female during mating. As with other nematodes, the sperm lack flagella. The copulatory bursa is a unique feature

of Strongylida members, thus making it a useful means for identifying members of this suborder; it is also used to distinguish members within the suborder due to differences in bursa appearance between species. The vulva of *A. caninum* females is located at the boundary of the second and final thirds of the body. The teeth of *A. caninum* are found in the buccal capsule and divided into three sets. Two ventral sets form a lower-jaw equivalent, while a further set projects from the dorsal side and loosely equates to an upper jaw.[2] Each ventral set has three points, with those furthest to the sides being the largest. While the ventral sets are prominent, the dorsal set is hidden deeper in the buccal capsule. Also, *A. caninum* bends its head end upward (dorsally), which has been noted to be a potential source of confusion when determining how the hookworm is oriented. If it has recently ingested blood, *A. caninum* is red in colour; if not, it appears grey. *A. caninum* has an alimentary canal made up of an esophagus, intestine, and rectum – the esophagus is highly muscular, reflecting its role in pulling intestinal mucosa into the body when it feeds. Esophageal and anal rings of *A. caninum* are the source of nerve fibers that extend throughout the body to innervate sensory organs, including amphids and phasmids. Eggs are laid by the females, typically when at the eight-cell stage. Eggs are 38–43 μm in width, with thin walls

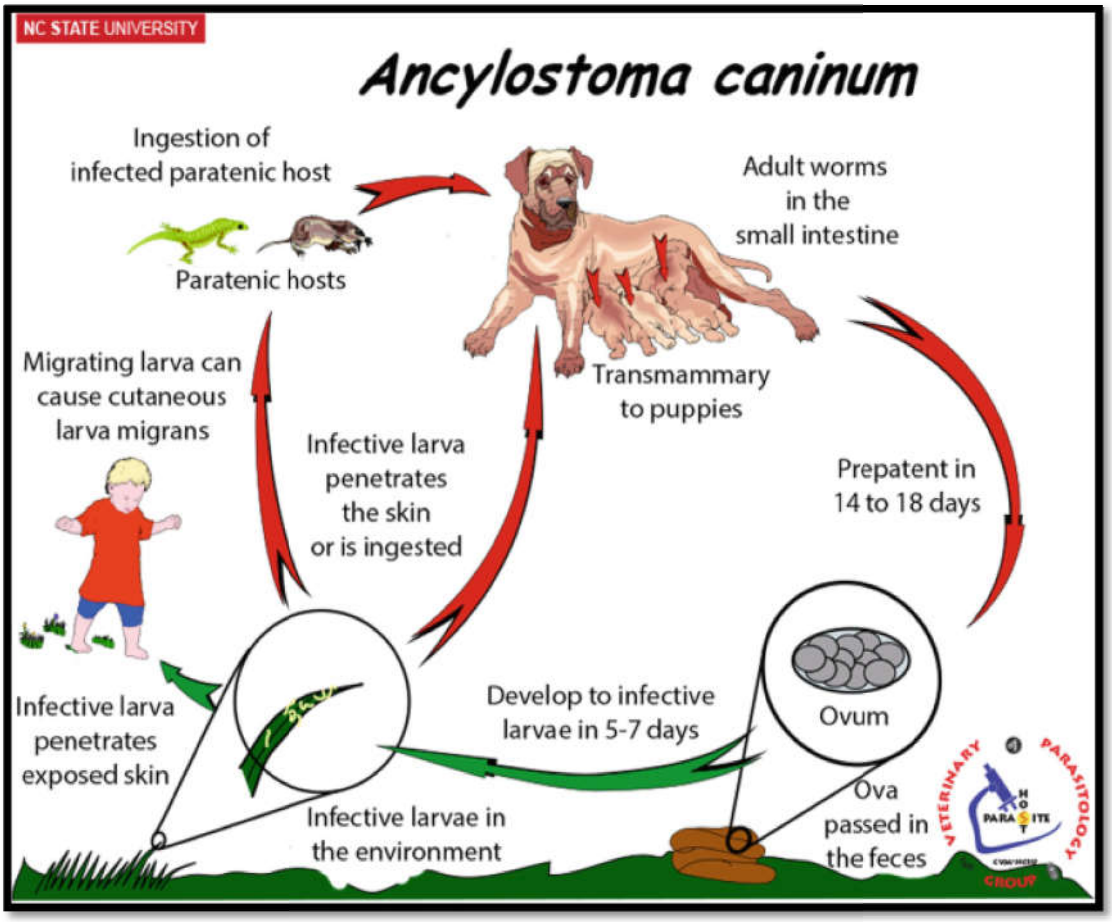
Life Cycle:

Transmission via the environment: Eggs are excreted from the host in the feces and typically hatch within a day on moist, warm

soil into larvae with a non-living cuticle layer. By 4–5 days, the larvae have moulted twice and are now able to infect a host. Migration occurs from the feces into the surrounding soil. Two routes of infection from the environment exist. The first route involves penetration of skin at hair follicles or sweat glands, especially between the footpads where contact with soil is frequent and the skin is thinner than otherwise. Secretion of a protease by *A. caninum* is thought to aid this process. The larvae then migrate through the dermis of the skin, enter the circulatory system and are carried to the lungs. *A. caninum* larvae exit the blood at the lungs, move from the alveoli up through the trachea and are swallowed to end up in the intestine. The second and more common route to the small intestine is by direct ingestion of *A. caninum* by the host, but the subsequent process is identical in either case. During this third stage of the larva, male or female reproductive organs become established. A third and final moulting occurs, resulting in the mature form of *A. caninum,* which then feeds on mucosa and blood of the small intestinal wall. The trigger of feeding is understood to be a receptor-mediated response; however, the detail of this process has yet to be established. Sexual reproduction also occurs in the intestine to produce a further round of eggs to complete the cycle. Females are thought to produce a pheromone which attracts males and are able to lay about 10,000 eggs per day.

Direct transmission: Direct transmission between hosts is also possible. Larvae having accessed through the skin may avoid exit via the lungs and remain in circulation for transport around the

body. At the uterine artery of a pregnant female, the larvae are able to cross the placenta to cause prenatal infection of foetus. Larvae of an infected foetus move to the liver until birth, when migration continues with movement to the intestine via the circulation and lungs as previously described. Alternatively, *A. caninum* larvae evading exit from the circulation at the lungs may instead be carried to the mammary glands and transmitted from the mother in her colostrum or milk to her pups; infection then proceeds in an identical manner as infection by ingestion from the environment. Infected mothers have been found to only rarely give prenatal transmission to pups, while the likelihood of causing transmission via the lactation route during nursing is much higher.



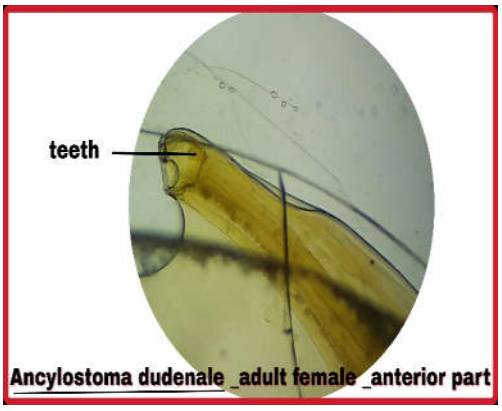
Pathogenicity:

Damage during migration to intestine: *A. caninum* larvae cause damage to the host at the point of entry through the skin, leaving a wound vulnerable to secondary infections. As the larvae migrate through the skin, an inflammatory response, dermatitis, is often stimulated, which can be exacerbated in hosts which have hypersensitive responses. Further damage is caused when the larvae leave the circulation and enter the lung, with the amount of damage dependent on the extent of the infection; pneumonia and coughing are common consequences.

Damage once in intestine: *A. caninum* attaches to and ingests the mucosal lining along with some consumption of blood; up to 0.1 ml in 24hrs. In a 24hr period *A. caninum* typically feeds from six sites. A group of anticoagulant proteins called *A. caninum* anticoagulant proteins, which inhibit a range of blood coagulation factors such as Xa, are used by *A. caninum* to help in the feeding process by preventing clotting and increasing blood loss. Blood losses peak just prior to egg production by the females because this is when their requirements for food are greatest; the amount that they are eating is also peaking, so maximal damage to the intestine is being caused. In inappropriate hosts such as humans, *A. caninum* is able to enter the skin, but cannot proceed into the circulation and on to the intestine; instead, the disease dermal larva migrans results, caused by movement of the nematode within the skin and which can persist for several months without intervention.

Diagnosis: Analysis of faeces is the definitive method by which

a suspected *A. caninum* infection is confirmed. The faeces are sampled and examined microscopically for the characteristic ovular, thin-shelled eggs of *A. caninum*. Absence of eggs in faeces does not rule out infection; a significant delay of at least 5 weeks exists between initial infection and excretion of eggs in the faeces (larvae must fully mature and reproduce before eggs can be laid). In fact, pups frequently die before passing of eggs in the faeces begins. Signs and symptoms expected to be observed are lethargy, weight loss, weakness, roughness of the hair coat, and pale mucous membranes indicative of anemia. Well-fed, older dogs with smaller infestations may present few or even none of these symptoms. Diarrhoea is rare, but stools are typically black due to the blood-derived hemoglobin present in them.



Bunostomum spp.

General characteristics

It is a hookworm which occur in the small intestine (ileum and jejunum). the host; sheep and goat, The males is 12-17mm long and the female 19-26mm.The anterior end is bent in a dorsal direction, so that the buccal capsule opens anterodorsally. There are pair of chitinous plates, near its base is a pair of small sub ventral lancets

Cutaneous larva migrans

This condition may be compared with V.L.M. It occurs in human and other hosts and is caused by the larvae of nematodes which inter the skin and migrate in it ,caused papules and inflamed tracks ,sometimes with thickening of the skin and pruritus. The nematodes whose larvae may cause it

-*A. caninum*, *A. braziliense*, *Uncinaria stenocephala*, *A. duodenal*, *B. phlebtomum*, *Strongyloides spp*., *Gnthstoma spp*

Super family: Trichostrongylidae

A small, slender digestive tract body, simple mouth, small buccal cavity, male bursate, while, female oviparous. With direct life cycle and larvae on the ground ingested, while, the site of Infection was a digestive tract.

Commone Genera

Trichostrongylus spp., Ostertagia spp., Nematodirus spp., Haemonchus spp., Obeliscoides spp.

Haemonchus contortus

Phylum: Nematohelminthes

Class: Chromadorea

Order: Rhabditida

Family: Trichostrongylidae

Genus*: Haemonchus*

Species: *Haemonchus contortus*

Name of Disease: Haemonchosis

Intermediate and final host: Herbivores animals

Sit of infection: Abomasum

Geographical Distribution and Epidemiology: Worldwide distribution between sheep and goats especially the younger in

rain seasons, infected sheep and goats, mainly during summer in warm, humid climates.

Diagnostic feature for parasite and Morphology:

The ova is yellowish in color. The egg is about 70–85 µm long by 44 µm wide, and the early stages of cleavage contain between 16 and 32 cells. The adult female is 18–30 mm. long and is easily recognized by its trademark "barber pole" coloration. The red and white appearance is because *H. contortus* is a blood feeder, and the white ovaries can be seen coiled around the blood-filled intestine. The male adult worm is much smaller at 10–20 mm. long, and displays the distinct feature of a well-developed copulatory bursa, containing an asymmetrical dorsal lobe and a Y-shaped dorsal ray.

Life Cycle:

The adult female worm can release between 5,000 and 10,000 eggs, which are passed out in the faeces. Eggs then develop in moist conditions in the faeces and continue to develop into the L1 (rhabditiform), and L2 juvenile stages by feeding on bacteria in the dung. The L1 stage usually occurs within four to six days under the optimal conditions of 24–29 °C (75–84 °F). The L2 rhabditform sheds its cuticle and then develops into the L3 filariform infective larvae. The L3 form has a protective cuticle, but under dry, hot conditions, survival is reduced. Sheep, goats, and other ruminants become infected when they graze and ingest the L3 infective larvae. The infective larvae pass through the first three stomach chambers to reach the abomasum. There, the L3

shed their cuticles and burrow into the internal layer of the abomasum, where they develop into L4, usually within 48 hours, or pre adult larvae. The L4 larvae then molt and develop into the L5 adult form. The male and female adults mate and live in the abomasum, where they feed on blood.

Pathogenicity: Clinical signs are largely due to blood loss. Sudden death may be the only observation in acute infection, while other common clinical signs include pallor, anemia, oedema, ill thrift, lethargy, and depression. The accumulation of fluid in the submandibular tissue, a phenomenon commonly called "bottle jaw", may be seen. Growth and production are significantly reduced.

Diagnosis: By fecal examination and clinical signs.

Arthropoda

General Characters of Arthropoda

1-The body is triploblastic, segmented and bilaterally symmetrical .

2-The body is divided into head ,thorax and abdomen .

3-Their body has jointed appendages which help in locomotion, three or four pair of legs , insects have one or two pair of wing .

4-The coelomic cavity is filled with blood.

5-They have an open circulatory system.

6-The head bears a pair of compound eyes.

7-The exoskeleton is made of chitin.

8-They unisexual and fertilization is either external or internal.

9-They have well- developed digestive system.

10-They have sensory organs like pair of antennae, hairs, simple and compound eyes and seta.

Ticks and Mites

Phylum: Arthropoda

Class: Arachinda

Subclass : Acari

Order :Parasitiformes

Sub Order: lxodida include ticks

Tick families : lxodidae (hard ticks)

Argasidae (soft ticks)

Order : Mesostigmata (free living predaceous and parasitic mites)

order : Prostigmata (chiggers , follicle mites)

order : A stigmata (house dust , storage &scabies mites)

Hard tick

General features :

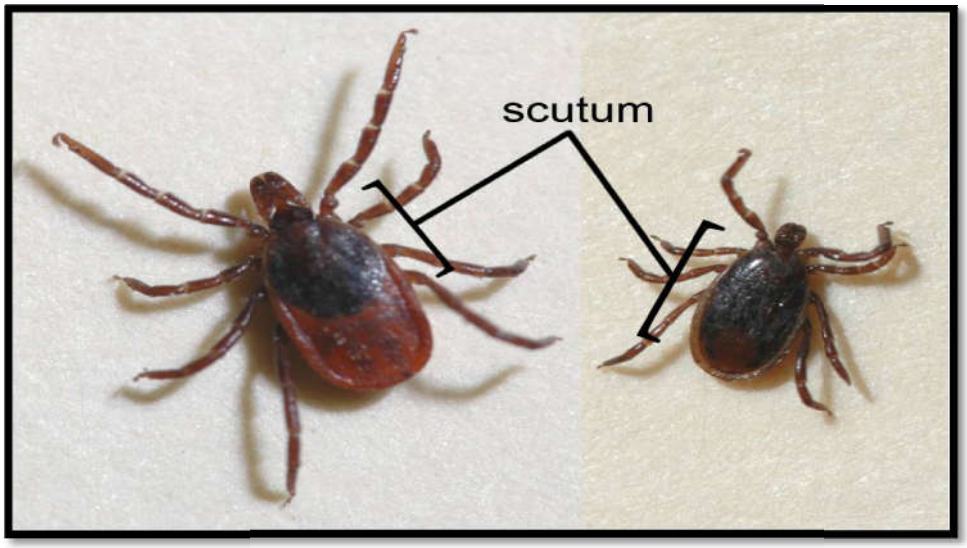
Have a hard upper surface called a shield that covers most of their body in male while in female have a relatively small scutum and its diagnostic features.

-Mouthparts (capitulum) are visible dorsally . They attach and feed for extended periods, using a different host in each life stage.

-Adult females are larger than males.

-Female produces as many as 6,000 to 7,000 eggs.

-Life cycle of Tick is incomplete.



Male and female of hard ticks

Name of Disease: Q fever, hemorrhagic fever, Rocky mountain Spotted fever.

Life cycle of Tick

Metamorphosis is incomplete (Egg-Larva. Nymph-Adult)

Egg: Female lays about 1000-8000 eggs all at one time and after that the female dies. The egg are brown in color and are deposited on the ground. After 2-3 weeks the eggs hatch into larva.

Larva: The tick larva possess 3 pairs of legs. The larva lies in wait of a suitable host and when host appears, attaches itself with it. The larval period may vary from 3-13 days.

Nymph: The nymph resembles the adult ticks having 4 pairs of legs.

Adult: The adult repeats the process of host finding, the engorged female drops off the host and hide away in a suitable location to digest the blood meal and lay her eggs.

Geographic Distribution: While ticks as a whole are worldwide in distribution, most species are restricted to various regions. All major biogeographic regions have tick species of public health importance.

Site of infection : External parasite found on the skin

Diseases that can be Transmitted by Ticks: Anaplasmosis is a disease caused by the bacterium *Anaplasma phagocytophilum* its transmitted to people by blacklegged ticks (deer ticks), the same ticks that transmit Lyme disease. Babesiosis is caused by *Babesia microti* its transmitted by the bite of infected by tick tick-borne . Rocky Mountain spotted fever This disease is transmitted by the American dog tick. viral encephalitis.

Diagnostic features of ticks: Have a hard upper surface called a shield that covers most of their body in male while in female have a relatively small scutum and its diagnostic features .

Differences between Hard and Soft Ticks

	Hard tick	Soft tick
Sectum	Covers the entire back in males. Only a small portion in front in females	Absent
Head	Situated at anterior end	Lies ventrally not seen from above.
Eggs	Several 100 or 1000 laid in one sitting	Laid in batches of 20-100 over a long period.
Nymph	One	Five
Habits	Cannot stand starvation, feed night and day	Stand starvation for years or more.
Diseases	Ticks typhus, viral encephalitis, hemorrhagic fever, tick paralysis and tularemia	Relapsing fever

Mite

Mites are very small sized acarines (body size ranges from 0.5-2.0 mm in length)

Name of the disease: Scabies (Mange)

Mange There are several types of mange that affect dogs, including: canine scabies(sarcoptic mange), ear mites, trombiculosis is a type of mange caused by the parasitic larval stage of mites of the family Trombiculidae (chiggers)

Life cycle

all mites include four stages : **egg, larva, nymph and adult.** However, there are typically a single larval stage and two nymphal stages.

Site of infection: Microscopic mites that invade the skin of otherwise healthy animals. The mites cause irritation of the skin, resulting in itching, hair loss, and inflammation. Most types of mange are highly contagious.

Distribution: Mite are worldwide in distribution.

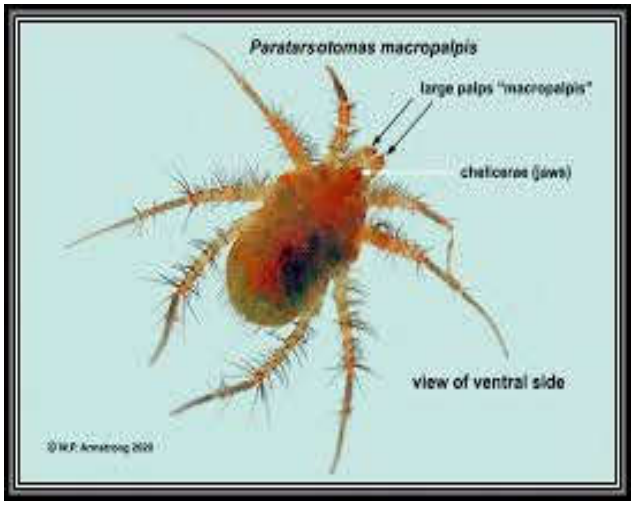
Diagnosis of mites: Is based on:

Studies comparing history, physical examination and signs.

Diagnosis is confirmed by careful examination of the affected areas.

Skin scrapings and looking at an animal's skin and hair samples with a microscope to identify the presence of mites

Some clinics might also use a blood test and to diagnose mange.



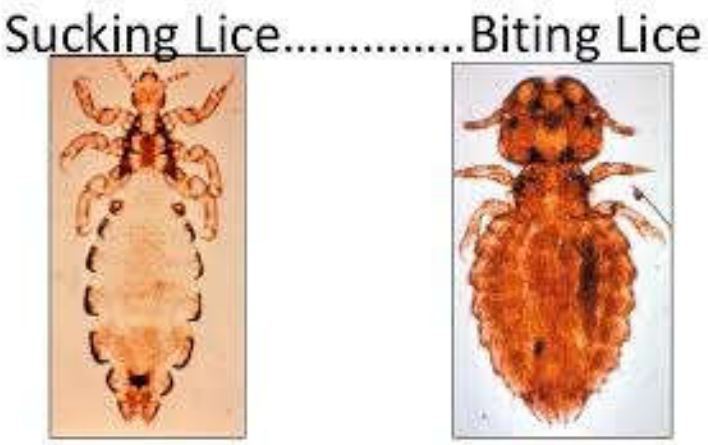
Lice

Lice are blood-feeding or skin/ hair / feather-chewing ectoparasites that are a menace to pets, livestock and humans. There are 3200 known species of lice that infect wild birds or animals but only a small percentage has any known medical or veterinary importance. lice are gradual metamorphosis.

There are two main groups lice:

Sucking lice consume a blood meal from their host and are more important in transmitting pathogens.

Chewing or biting lice ingest feathers, fur and skin and sometimes blood from their host.



Human Lice

Phylum : Arthropoda

Class : Insecta

Order : Anoplura

Family : Pediculidae

Genus : *Pediculidus humanus* and *phthirus*

Lice : pleural ….**louse :**single

Lice

are parasitic insects that feed on human blood and can infest the head, body, and pubic area.

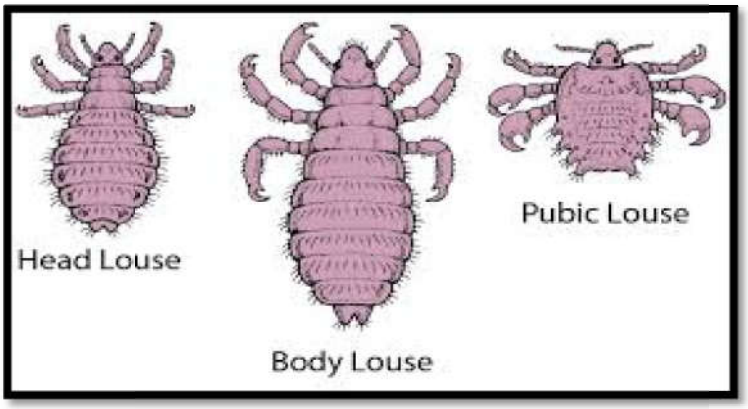
There are three types of lice that infest humans:

body louse *Pediculus humanus corporis*

head louse *Pediculus capitis*

pubic louse *Pthirus pubis*

lice that are found on the body are different from lice found on the head or on the pubic area. Body lice are only found on humans on the body.



Name of the disease: Pediculosis, Epidemic Typhus, Prison Fever

Intense itching (pediculosis) and rash caused by an allergic reaction to louse bites are common symptoms of body lice infestation.

Life Cycle: The life cycle of a louse occurs in three stages:

egg, also called a nit: The nit is a whitish-yellow speck that is about 1 millimeter in length. It attaches itself firmly to an individual hair strand close to the scalp.

Nymph : After 7 to 10 days, the nit hatches and becomes what is known as a **nymph**, or a young louse. Nymphs are usually between 1.1 and 1.3 millimeters, and tan or white in color. Nymphs mature into adult lice within about 9 to 12 days.

Mature adults: don't tend to grow larger than 2 millimeters, and female lice are larger than males. Mature lice live for about three to four weeks.

Geographical distribution of lice: Pediculosis worldwide in

distribution.

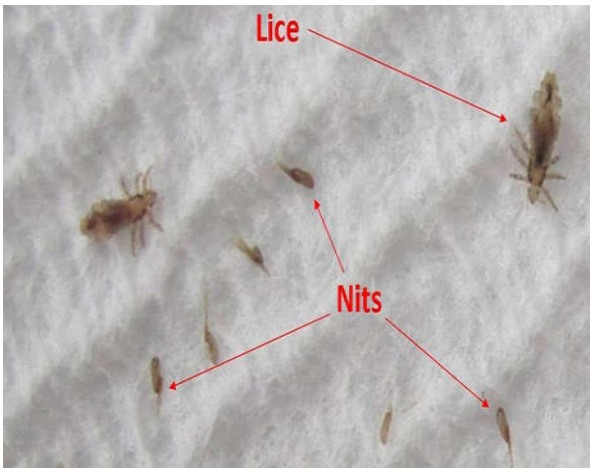
Diagnostic Features of Lice: An infestation of lice is typically diagnosed by looking at the hair , skin and clothing and observing eggs and crawling lice. The lice are about the size of a sesame seed and that diagnostic features They are big enough to see with the naked eye, The eggs (called nits) are usually found attached of the hair.

Clinical Signs: Common signs and symptoms of lice include:

Intense itching on the scalp, body or in the genital area.

The presence of lice on your scalp, body, clothing, or pubic or other body hair. Adult lice may be about the size of a sesame seed or slightly larger.

Lice eggs (nits) on hair shafts. Nits may be difficult to see because they're very tiny.



Cattle Lice

Name of the Disease:

1. Sucking lice that caused by *Linognathusvituli* feed on blood

2. Biting lice that caused by *Bovicola bovis* feed on dead skin cells, hair and oil secretions.

Life Cycle: Louse have five life cycle stages consisting of eggs, three nymphal stages and an adult. All life stages can be found on cattle , the complete life cycle from egg to adult takes 25-30 days. Females lay eggs at 24 to 36 hour intervals and the three nymphal stages take between 4 and 9 days each, depending on temperature. Each female commonly lays 30–35 eggs during a 4-6-week period. Adults can survive as long as 9 or 10 weeks.

Site of infection

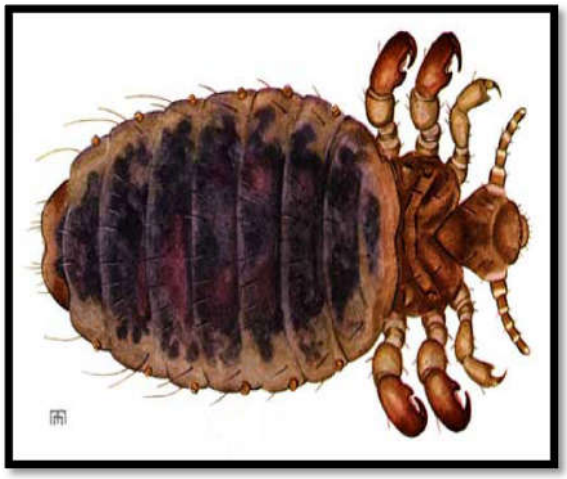
Sucking Lice: Feed on cattle blood and have fine stylets that they use to penetrate capillaries to feed. In large numbers these lice cause far greater production losses than biting lice.

Cattle Biting Lice: Usually most numerous on the head and in longer hair along the backline, shoulders, neck and rump. In heavy infestations they can be found on most parts of the body.

Diagnostic Features: The presence of lice can be confirmed by close examination of the hair and skin of visibly affected animals. Frequently, lice eggs are more readily seen than lice themselves. Eggs are white in color and are cemented to the shafts of hairs in clumps. Biting cattle lice are highly mobile (as they move when

the hair is parted). Sucking lice are grey or blue grey and have a pointed head which tends to remain fixed to the skin.

Clinical Signs: A large number of lice can be debilitating to an animal. They can cause anemia, dermatitis, allergic responses, hair loss and other disorders which can be costly to the livestock industry**.** Millions of lice per an animal can lead to restlessness, pruritus, anemia, low weight gain, low milk yield, dermatitis, hide or fleece damage, skin crusting or scabbing, and lameness. Sucking lice on cattle can cause serious damage to the host and severe anemia, abortions, or even death at times**.**



Chewing lice: Chewing lice are permanent ectoparasites primarily of bird species, and they feed on feathers and skin scales. These lice can be harmful to both domestic and wild hosts

Name of Common Parasite: *Menopon gallinae* which is yellowish color flat louse about 2mm long.

Life Cycle: Gradual metamorphosis which take place entirely on

the host about 3-5 weeks

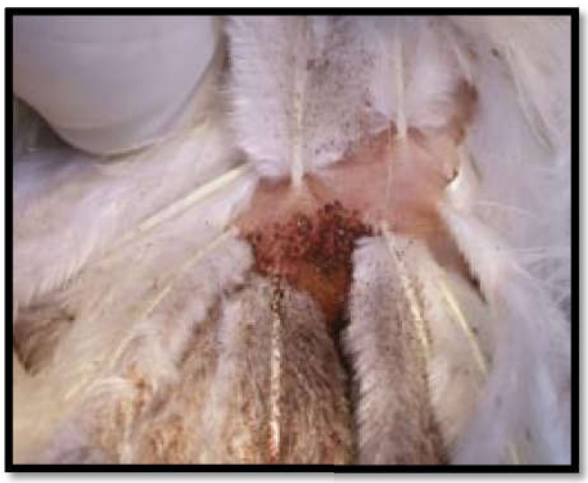
Egg commonly called nit are attached to the feathers of the host. The young lice developed through several stage of nymph which they look small, pale adult. Adult and nymph are transmitted from host to the another by contact.

Geographic distribution : Worldwide in distribution.

Site of Infection: Chewing lice live on birds feathers and feed on skin secretions, dried blood, feathers and skin debris. Although they are not blood suckers.

Diagnostic Features: The veterinarian will diagnose a parasite through physical examination, and found of the parasite. The clinical signs will most likely be evident; the presence of the lice on the wing, ragged feathers, and the constant itching of bird will be experiencing may point to the rapid diagnosis.

Clinical Signs: Symptoms of infestation include itching, loss of appetite, and lowered egg production in fowl , chewing lice can cause serious discomfort. . Infested animals are usually treated with a dust or dip.



Osteridae (warble flies , bot flies)

Phylum: Arthropoda

Class : Insecta

Order : Diptera

Family : Oestridae

Subfamily : Hypodermatinae

Genus : *Hypoderma*

Name of the Diseases: Myiasis the diseases that caused by larvae

Other Name Include: Heel flies, bomb flies and gad flies, while their larvae are often called (cattle grubs or wolves)

Site of Infection: larvae are internal parasite of mammals and can infect human, act as a final host nasal cavity of sheep and horse under skin of cattle(*Hypoderma bovis*) and rodents, sheep nasal bot fly (*Oestrus ovis*). Living larvae deposited into nostrils , attack to sinus membrane and feed on mucous Sneezed out and pupate in soil.

Geographic Distribution: Warble flies has a worldwide distribution, although many European countries have eradicated the fly through treatment programs

Life Cycle: The eggs of these flies are slender in shape, white in color and about 1 mm long. The larvae are light to dark brown in color**.** Larvae (L3) are involved with Mayasis, Larvae are white, spiny, barrel-shaped and have black mouth parts ,The larvae are light to dark brown in color. The adults morphologically look like bumble bees , Large, stout, with large inflated head and with reddish yellow hairs at the posterior extremity, adults non-functional mouthparts and short-lived and rarely seen.

Diagnostic Features: The presence of Hypoderma larvae may be detected by palpation of the warbles on the animals' backs (clinical parasitological examination) during the spring and summer months or by examination of subcutaneous tissues post mortem examination for examined internal organs**.** Some ELISA tests have been used in many countries for the serological diagnosis of the disease. Anti-Hypoderma antibodies persist in infected cattle for 3–4 months after the L3s fall from their backs to the ground and antibody levels peak.

Clinical Signs

Soft nodular swelling on the subcutis

Larvae embedded in skin

Various inflammation and may be necrosis in skin and tissues

In severe cases, paraplegia resulting from toxin release and pressure on the spinal cord (*H. bovis*) can occur.

Myiasis

Myiasis is the disease caused by The invasion of organs and tissues of humans or other vertebrate animals with dipterous larvae, which for at least a period feed upon the living or dead tissue or, in the case of intestinal myiasis, on the host's ingested food.

Type of Myiasis

There are three main families of flies that cause myiasis:

- Oestridae (obligate parasites)

- Calliphoridae (obligate or facultative)

- Sarcophagidae (obligate or facultative

Life Cycle

Eggs : Females lay 10-400 eggs on wounds, meat, scabs and sores, etc

Larvae : 3rd instar is formed in 2-3 days and is maggot-shaped, Mature larvae wriggle out after 4-12 days, drop to ground. Pupate in soil.

Adults: emerge after 7-12 days.

Geographic Distribution: In the worldwide especially in tropical and subtropical regions.

Types of Myiasis in Humans

- Cutaneous: include (Furuncular, Creeping and Wound)

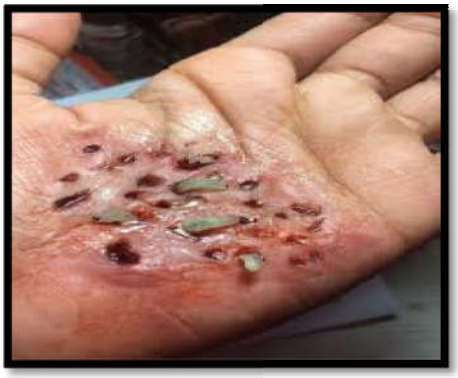
- Nasopharyngeal

- Opthamo myiasis

- Oral

- Intestinal

- Cerebral



Diagnostic Features: As the same in the warble fly

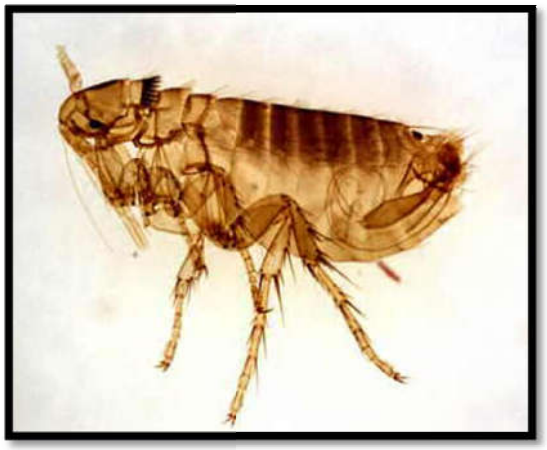
Clinical Signs:

Effects on Livestock

lesions that cause huge economic losses worldwide (in the hundreds of millions annually). Cause stress to the livestock and reduction in livestock health. High infection levels, or migration of larvae into important regions can lead to death.

Effects on Humans: Most commonly infected by obligate and facultative fly species. Humans may become infected if they spend extended periods of time around livestock • Depending on the species of fly and the type of infection, the effects can range from minor to severe and potentially fatal . Secondary infections are common.

Flea



Phylum: Arthropoda

Class: Insecta

Order: Siphonaptera

Family: Pulicidae

Genus: *Ctenocephalides*

Species :*Ctenocephalides canis*

Name of Disease: Plague

Common name : Dog Flea

Fleas are flightless insects, which often parasitize animals such as rodents, birds, wild mammals and companion animals. As a result of their ability to infect a vast variety of species fleas have been known to be carriers of multiple pathogens and diseases to your including:

Flea allergy dermatitis when a flea bites, it injects saliva into your pet's skin. This may trigger an allergic reaction. *Ctenocephalides canis* can act as intermediate hosts for parasitic worms including the double-pored tapeworm, *Dipylidium caninum*. Anemia Fleas can take in 15 times their own weight in blood. Dogs that lose too much blood may develop anemia,

Geographic Distribution: The dog flea is an important ectoparasite of both wild and domestic animal and human

around the world.

Life Cycle:

Eggs: A blood meal is required for a mated adult female flea to produce and lay eggs A single female flea can produce hundreds of eggs in her lifetime , Flea eggs range from 0.1 to 0.5 mm in length, are pearl-white in color, oval shaped, smooth, and non-sticky.

Larvae: *Ctenocephalides canis* larvae are white, elongate, contain numerous setae, and lack legs and eyes , it takes 10 days for larvae to progress through three larval instars and develop into pupae.

Pupae : are off-white in color, eventually becoming dark brown as they mature. Adults will emerge from pupae after seven days.

Adults: Adult dog fleas are small (2.0 - 3.25 mm), wingless, bilaterally compressed, and heavily chitinized . Members of the genus flea have genital and pronotal combs, large black eyes and 5-segmented labial palps.

***Ctenocephalides canis* larva and head part**

Diagnosis of Dog Flea: Flea infestations and flea allergy dermatitis are definitively diagnosed by:

Observing the fleas on the host's skin such as Pruritis, alopecia, excoriations, and other signs of trauma can be observed in affected animals.

Diagnosis is often based on clinical signs, especially face and ears are the main areas for clinical signs.

History, and lesion distribution. Sometimes the presence of flea excrement (“flea dirt”) on the dog's skin can support a presumptive diagnosis.

Although fleas are tiny (between one and three mm.) they are difficult to see , so if you look carefully you should be able to see them.

Clinical Signs: The sign of fleas for many pet owners is:

The dog itching, scratching or chewing at themselves, leading to hair loss and red or irritated skin. Visible fleas or flea dirt (flea faeces) on your dog. Although many dogs are very itchy and uncomfortable, some dogs may show no signs at all and fleas can be difficult to see.

Human Flea

Phylum: Arthropoda

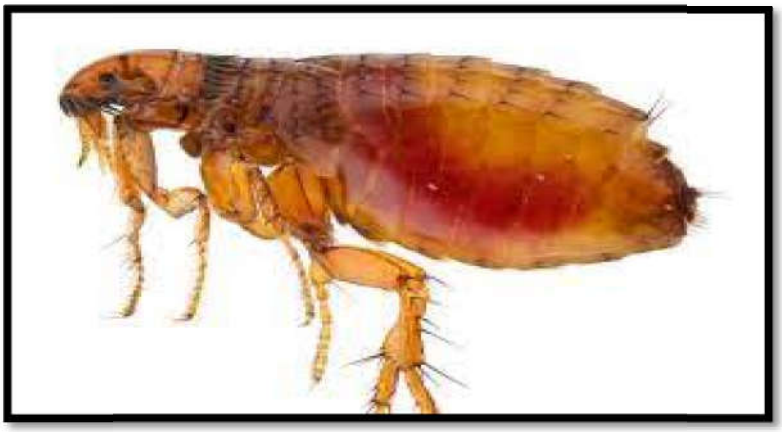
Class: Insecta

Order: Siphonaptera

Family: Pulicidae

Genus: *Pulex*

Species : *Pulex irritans*



Name of Disease: Plague, (Black Death)

General Character: The human Fleas are small, wingless insects with a tough cuticle bearing many bristles and frequently combs (ctenidia) of broad, flattened spines. The adult flea varies from about 0.1 to 0.32 cm in length and feeds exclusively on the blood of mammals (including humans), rodents and birds.

Site of Infection: Fleas are ectoparasites that live on the skin of the host.

Geographic Distribution: it is widely distributed with some—

such as the rat flea and the mouse flea—having been carried all over the world by humans. Native species of fleas are found in polar, temperate, and tropical regions.

Life Cycle: The four life stages are (complete metamorphosis) :

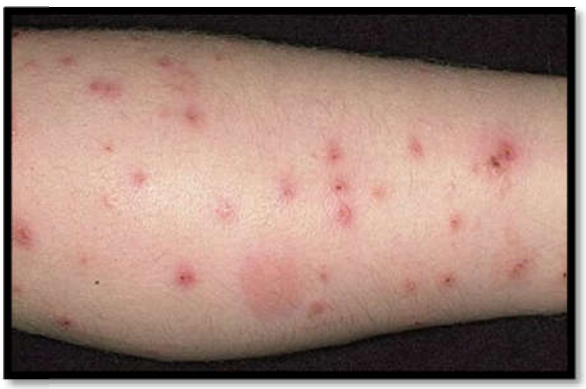
The egg : Pearly white oval eggs are deposited on the body of, or in the nest or habitat of, the host animal.

The larva : is small and legless and feeds on organic debris, such as dried excrement, dried bits of skin, dead mites, or dried blood found in the host's nest.

Pupa : the larva spins a silk cocoon that includes debris from the nest and enters the pupal stage. The pupa emerges as an adult some days or months later.

Clinical Signs: Flea bites are easily infected, as scratching can introduce nasty bacteria into the open sores. Infected flea bites become painful, red, and swollen. Fleas, particularly *Xenopsyllacheopis,* are thought to be the principal carriers *Yersinia pestis* bacteria cause the disease of humans as plague, rats and mice are the sources of infection

Diagnosis : The same as in dog fleas diagnosis



Mosquito

Kingdom : Animalia

Phylum : arthropoda

Class : Insecta

Order :Diptera

Suborder :Nematocera

Family :Culicidae

Genus*: Anopheles, Culex*

Name of Disease: Malaria, Dungue Fever

Diagnostic Feature: Mosquitoes are a family of small, midges-like flies ,female of most species are ectoparasites, whose tube-like mouthparts (called proboscis) forming piercing sucking part adults have two big compound eye, pair of antennae ,pair of wing and all species are complete metamorphosis insects therefore grow though an egg, larva, pupa to adult stage ,(larva and pupa are aquatic), the adults are free flying .

Adult:

Adult *Anopheles* species have slender bodies with three sections: head, thorax and abdomen. The head is pears sensory organs (eyes and antennae)*Anopheles* mosquitoes can be distinguished from other mosquitoes by the palps, which are as long as the proboscis, some species of Anopheles regarded as a vector of

malaria disease .

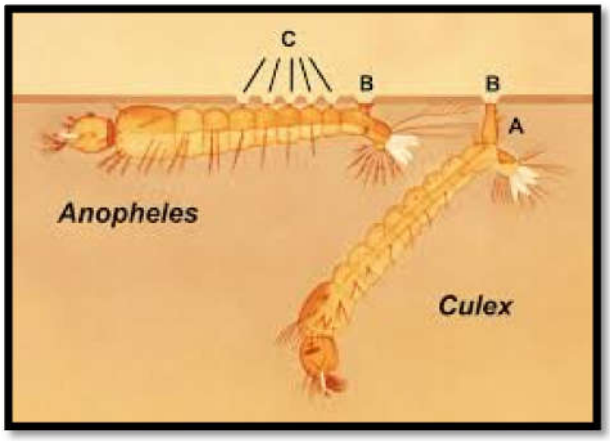
Larva :

The mosquito larva has a well-developed head with mouth brushes used for feeding, a large thorax and a nine-segment abdomen. It has no legs. In contrast to other mosquitoes, the *Anopheles* larva lacks a respiratory siphon, so it positions itself so that its body is parallel to the surface of the water.

Pupa :

The pupa is comma-shaped when viewed from the side. The head and thorax are merged into a cephalo thorax with the abdomen curving around underneath.

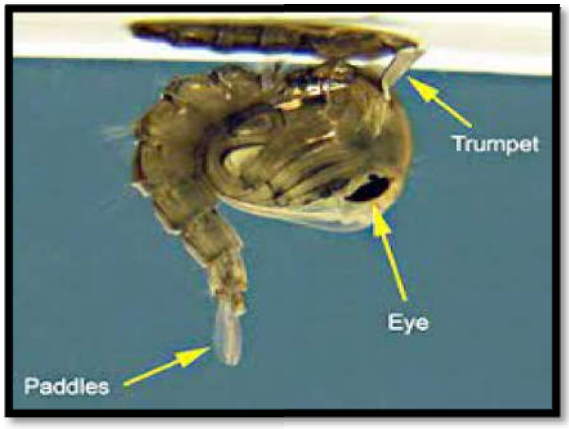
Adult female Male and female



Larvae

egg

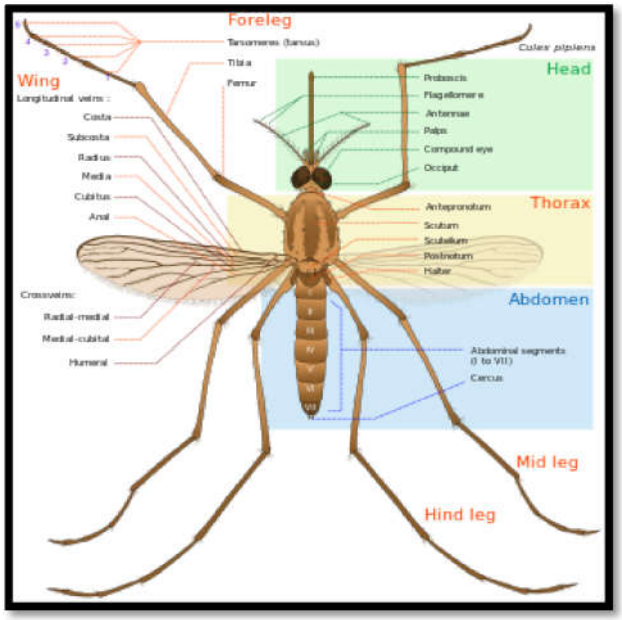
Pupa



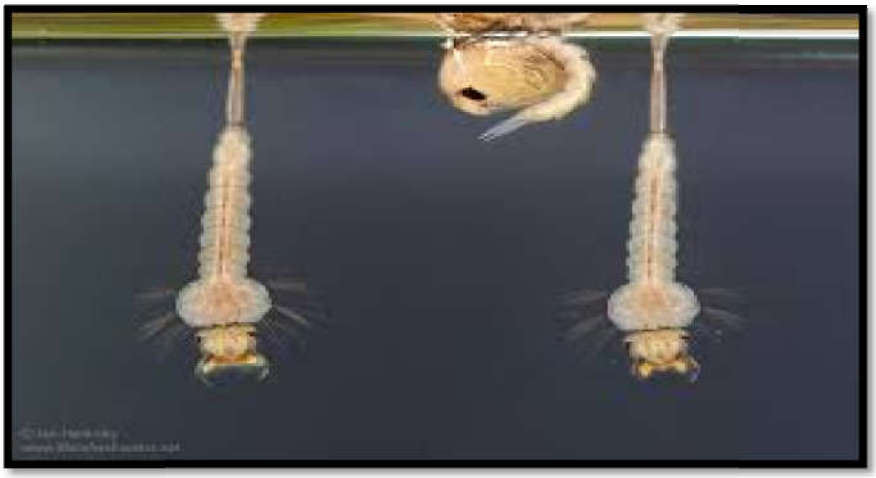
Larva and pupa of anopheles

Culex spp.

The genus of mosquitoes, several species of which serve as vectors of one or more important diseases of birds, humans and other animals. West Nile virus, filariasis, Japanese encephalitis, St. Louis encephalitis, and avian malaria.



egg



Larvae and Pupa

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