



Superfamily Trichostrongyloidea :

- Are small hair like worms in the bursate group.
- The buccal capsule is absent or very small and is devoid of leaf-crowns and usually bears no teeth.
- The male bursa is well developed with large lateral lobes and a small dorsal lobe and two spicules .
- Adults parasitic in the alimentary canal of sheep, cattle, equines and birds. Genera of important include : Ostertagia, Marshallagia, Haemonchus, Trichostrongylus, Cooperia, Nematodirus, and Dictyocaulus (only parasitic in the lung).

Hypobiosis (arrested larval development)

This phenomenon may be defined as temporary cessation in development of a nematode .

The accumulation of arrested larvae often with the onset of cold autumn , winter or very dry condition , the maturation of these larvae coincides with the return of environmental condition .

The stage at which larvae become arrested varies between species of nematode (L3 in Trichostrongylus , L4 in Ostertagia and Haemonchus), in mature adult in Dictyocaulus .

Ostertagia :

This genus is the major cause of parasitic gastritis in ruminants in temperate areas of the world.

Host : Ruminants

Site : abomasum

Species :

Ostertagia ostertagi in cattle , O. circumcincta in sheep and goats, O. trifurcata in sheep and goats

Distribution :

Worldwide

Identification :

-The adults are slender reddish-brown worms up to 1cm long, occurring on the surface of the abomasal mucosa and are only visible on close inspection.

-The larval stages occur in the gastric glands and can only be seen microscopically following processing of the gastric mucosa, species differentiation is based on the structure of the spicules which usually have three distal branches.

Bovine Ostertagiosis:

Ostertagia ostertagi : (brown stomach worms)

O. ostertagi is perhaps the most common cause of parasitic gastritis in cattle. The disease, often simply known as ostertagiosis. Is characterized by weight loss and diarrhea and affected young cattle during first grazing season.



Life cycle :

O. ostertagi has a direct life cycle. The eggs passed in the feces under optimal conditions developed within fecal pad to the infective third stage (L3) within two weeks. When moist conditions the L3 migrate from the feces on to the herbage ingestion L3, and exsheaths in the rumen and lumen of abomasal glands. Two parasitic moults occur before the L5 emerges from the gland around 18 days after infection to become sexually mature on the mucosal surface. The entire parasitic life cycle usually takes three weeks, but under certain circumstances many of the ingested L3, become arrested in development at the early fourth larval stage (EL4) for periods of up to six months.

Pathogenesis :

- The presence of *O. ostertagi* in the abomasum in sufficient numbers gives rise to extensive pathological and biochemical changes - These changes are maximal when the parasites are emerging from the gastric glands this is usually about 18 days after infection, but it may be delayed for several months when arrested larval development occurs.
- the parietal cells, which produce hydrochloric acid, are replaced by rapidly dividing undifferentiated, non-acid-secreting cells.
- reduction in the acidity of abomasal fluid, increase PH, loss of bacteriostatic, hypoalbuminemia, due to elevated plasma pepsinogen
- Macroscopically the lesion is a raised nodule with a visible central orifice, in heavy infections, these nodules coalesce to produce an effect reminiscent of morocco leather.
- The abomasal folds very edematous, hyperemic and some times necrosis and sloughing of the mucosal surface occurs and enlargement of local lymph nodes as a reduction in the acidity of the abomasal fluid.

Clinical signs :

Bovine ostertagiosis is known to occur in two clinical forms as follows :

- * Type I disease is usually seen in calves grazed intensively during their first grazing season, as the result of larvae ingested 3-4 weeks previously.
 - persistent watery diarrhea and has a characteristic bright green color.
 - coats dull and the hind quarters heavily soiled with feces
 - morbidity is usually high 75% but mortality is rare provided treatment is instituted within 2-3 days.
- * Type II disease occurs in yearlings usually in late winter or spring following their first grazing season and results from the maturation of larvae ingested during the previous autumn and subsequently arrested in their development at the early fourth larval stage.
 - diarrhea is often intermittent and anorexia and thirst are usually present
 - coat dull.
 - hypoalbuminaemia is more marked
 - moderate anemia
 - submandibular oedema.
 - mortality is very high unless early treatment with an anthelmintic effective against both arrested and developing larval stages is instituted



Diagnosis :

1- clinical signs

2- The season .

3- The grazing history

4- Fecal egg counts. these usually more than 1000 eggs per gram (epg) and are a useful aid to diagnosis.

5- Plasma pepsinogen levels.

6- Post-mortem examination if this is available the appearance of the abomasal mucosa is characteristic there is a putrid smell from the abomasal contents due to the accumulation of bacteria and the high pH. The adult worms, reddish in color and 1.0cm in length can be seen on close inspection of the mucosal surface.

Treatment :

treatment at the standard dosage rates with any of the modern benzimidazoles (albendazole, fenbendazole or oxfendazole), the pro-benzimidazoles (febantelnetohimin and thiophanate) levamisole or the avermectins / milbemycins eg: ivermectin. All of these drugs are effective against developing larvae (arrested or hypobiotic larvae) and adult stages.

Control :

Ostertagiosis has been prevented by :

1-routinely treating young cattle with anthelmintic over the period when pasture larval levels are increasing.

2-grazing calves on new grass leys.

3-resting pasture or grazing it which another host, such as sheep, which are not susceptible to *O. ostertagi*, until most of the existing L3, on the pasture have died.

Ovine Ostertagiosis :

In sheep *O.circumcincta* and *O. trifurcata* are responsible for outbreaks of clinical ostertagiosis, particularly in lambs.

Life cycle :

Both the free-living and parasitic phases of the life cycle are similar to those of the bovine species.

Pathogenesis :

In clinical infections, this is similar to the situation in cattle and the same lesions are present at necropsy in subclinical infection

Clinical signs :

The most frequent clinical sign is a marked loss of weight. Diarrhea is intermittent and although stained hindquarters are common, the fluid feces which characteristic bovine ostertagiosis are less frequently seen.

Diagnosis :

This is based on clinical signs, seasonality of infection and fecal egg counts and, if possible, post – mortem examination, when the characteristic lesions can be seen in the abomasum. Plasma pepsinogen levels are above the normal .

Treatment :

Ovine ostertagiosis responds well to treatment with any of benzimidazole or probenzimidazole, levamisole , which in sheep is effective against arrested larvae , or the avermectins / milbemycina.



Haemonchus:

(Stomach worms, Wire worms, Barber pole worms)

Species : Haemonchus contortus (H.Placei), H.Similis

Hosts: sheep , goats and cattle

Site: Abomasum

Distribution : Worldwide.

Identification:

Gross: The adults are easily identified because of their specific location in the abomasum and their large size (2.0-3.0 cm) in fresh specimens. The white ovaries winding spirally around the blood-filled intestine produce a " barber's pole" appearance.

Microscopic:

The male has an asymmetrical dorsal lobe and barbed spicules. The female usually has a valval flap in both sexes there are cervical papillae and a tiny lancet inside buccal capsule.

Life cycle :

This is direct and the preparasitic phase is typically trichostrongyloid. The females are prolific egg layers.

The eggs hatch → to L1 on the pasture and develop → L3 in as short a period as five days but development may be delayed for weeks or months under cool conditions → ingestion(L3) and exsheathment in the rumen the larvae moult twice in close apposition in gastric glands then develop the piercing lancet which enables them to obtain blood from the mucosal vessels adults they move freely on the surface of the mucosa. The prepatent period is (2-3) weeks in sheep and four weeks in cattle.

Ovine Haemonchosis :

Pathogenesis :

*Acute Haemonchosis

-anemia due to the blood-sucking habits of the worms each worm removes about 0.05ml of blood per day by ingestion and seepage from the lesions so that a sheep with 5000 H.contortus may lose about 250 ml daily.becomes apparent about two weeks after infection

-progressive and dramatic fall in the packed red cell volume loss of iron and protein

-At necropsy, between 2000 and 20000 worms may be present on the abomasal mucosa which shows numerous small haemorrhagic lesions, the abomasal contents are fluid and dark brown due to the presence of altered blood.

- The carcass is pale and oedematous and the red bone marrow has expanded from the epiphyses into the medullary cavity.

*Hyper acute haemonchosis

Less commonly, in heavier infections of up to 30.000 worms apparently healthy sheep may die suddenly from severe haemorrhagic gastritis this is termed.

*Chronic haemonchosis :

is negligible and develops during a prolonged dry season when reinfection but the pasture becomes deficient in nutrients.



Clinical signs :

- In hyperacute cases, sheep die suddenly from haemorrhagic gastritis.
- In Acute haemonchosis is characterized anemia, variable degrees of odems, submandibular form and ascites are most easily recognized. Lethargy, dark colored feces and falling wool diarrhea is not generally a feature.
- Chronic haemonchosis is associated by or with progressive weight loss and weakness, neither sever anaemia.

Epidemiology :

Is best considered separately depending on whether it occurs in tropical and subtropical or intemperate areas. Haemonchosis is a primarily a disease of sheep in warm climates, since high humidity is also essential for larval development and survival.

The frequency and severity of outbreaks of disease is largely dependent on the rainfall in any particular area.

Diagnosis :

- Acute Haemonchosis
- * history and clinical signs.
- * fecal worm egg count.
- * Necropsy finding

- Hyper acute haemonchosis

death may have occurred and only the abomasum may show changes

- Chronic haemonchosis

Is more difficult because of the concurrent presence of poor nutrition

Treatment

benzimidazoles, Levamisole, avermectin /milbemycin or salicylanilide.

Control :

Depending on the:

- duration and number of periods in the year when rainfall and temperature permit high pasture levels of *H. contortus* larvae to develop at such times it may be necessary
- use an anthelmintic at intervals of 2-4 weeks depending on the degree of challenge. Sheep should also be treated at least once at the start of prolonged rain to remove persisting hypobiotic larvae whose development could pose a future treat

Bovine Haemonchosis :

is similar in most respects to haemonchosis in sheep and is important in the tropic and subtropics during seasonal rains when sever outbreaks may occur. However the disease has also been recorded at the end of a long dry season due to the maturation of hypobiotic larvae.

Unlike haemonchosis is sheep grazing cattle over two years old are relatively immune at though this may be broken, down by drought conditions which lead to poor nutrition and heavy challenge from congregation of animals around watering points. Treatment and control are similar to that described for *H. contortus* in sheep.

The self-Cure phenomenon:



In areas of endemic haemonchosis it has often been observed that after the advent of a period of heavy rain the fecal worm egg counts of sheep infected with *H. contortus* drop sharply to near zero levels due to the expulsion of the major part of the adult worm burden.

This event is commonly termed the self-cure phenomenon, and has been reproduced experimentally by superimposing an infection of *H. contortus* larvae on an existing adult infection in the abomasum the expulsion of the consequence of an immediate-type hypersensitivity reaction to antigens derived from the developing larvae, it is thought that a similar mechanism operates in the naturally occurring self-cure.

Trichostrongylus :

Hosts :

Ruminants, horses. Pigs, rabbits and fowl.

Species :

T. axei: abomasum of ruminants and stomach of horses and pigs

T. colubriformis ruminants.

T. vitrines

T. capricola sheep and goats

T. tenuis small intestine and caeca of game birds.

T. axei (hair worm)

Distribution : Worldwide

Gross :

The adults are small and hair-like usually less than 7mm long and difficult of with the naked eye.

Microscopic :

The worms have no obvious buccal capsule. The spicules are thick and unbranched and in the case of *T. axei* are also unequal in length in the female the tail is bluntly tapered and there is no vulval flap. In *T. axei* the eggs are arranged pole to pole longitudinally.

Life cycle :

This is direct and the parasitic phase is typically Trichostrongyloids except exsheathment of L3 of intestinal species occurs in the abomasum. Under optimal conditions, development from the egg to infective stage occurs in 1-2 weeks, the parasitic phase is no-migratory and the prepatent period in ruminants 2-3 weeks. In the horse, *T. axei* has a prepatent period of 25 days.

Pathogenesis :

Following ingestion, the L3 of the intestinal species penetrate between the epithelial glands of the mucosa with formation of tunnels beneath the epithelium, after infection, there is considerable haemorrhage, oedema and plasma proteins are lost into the lumen of the gut. Grossly, there is an enteritis, particularly in duodenum,, the villi become distorted and flattened reducing the area available for absorption of nutrients and fluids. parasites are congregated within a small area, erosion of the mucosal surface is apparent. In heavy infections diarrhea occurs, and this, together with the loss of plasma protein into the lumen of intestine, leads to weight loss. A reduced deposition of protein,



calcium and phosphorus has also been recorded. In the case of *T. axei* the changes induced in the gastric mucosa are similar to those of *Ostertagia* with an alteration in pH and an increased permeability of the mucosa.

Clinical signs :

The principal clinical signs in heavy are :

- weight loss and diarrhea.
- in appetence and poor growth rates.
- sometimes accompanied by soft feces, are the common signs.

Epidemiology :

The embryonated eggs and infective L3 of *Trichostrongylus* have a high capacity for survival under adverse conditions whether these are extreme cold or desiccation, larval numbers increase in pasture in summer and autumn giving rise to clinical problems during this season, the seasonal occurrence being similar to that of *ostertagia* spp. In contrast to other trichostrongyles hyobiosis occurs at the L3, stage although their role in outbreaks of disease has not been established.

Diagnosis :

This is based on :

- 1-clinical signs
- 2- seasonal occurrence of disease
- 3-postmortem examination.
- 4-Faecal egg counts are a useful aid to diagnosis although fecal cultures are necessary for generic identification of larvae.

Treatment and control :

Depending on the host, this is as described for bovine ostertagiosis, parasitic gastroenteritis in sheep and strongylosis in the horse.

T. tenuis infection :

In game birds, heavy infections produce an acute and fatal haemorrhagic typhlitis. Lighter infections result in a chronic syndrome characterized by anemia and emaciation. On game farms, therapy with levamisole in the drinking water has proved useful



Cooperia :

Hosts : Ruminants.
Site : small intestine.
Species :
C.punctate
C.pectinata

Distribution : Worldwide

Identification :

Gross :

In size cooperia are similar to ostertagia

life cycle :

This is direct and typical of the superfamily. The life cycle of Cooperia are similar requirement to Haemonchus. The prepatent period varies from (15-18 days)

pathogenesis

Cooperia are generally considered to be mild pathogens in calves and lambs respectively although in some studies they have been associated with in appetite and poor weight gains.

Clinical signs :

- Loss of appetite, - Poor weight gains.
- In C.punctate, C.pectinata sever diarrhea sever weight loss, and submandibular oedema.

Epidemiology :

This is similar to that of Ostertagia in temperate areas and similar to that of Haemonchus in subtropical areas.

Control and Treatment :

Treatment and control similar to those applied in bovine Ostertagiosis.

Nematodirus

Is a special importance as a parasite of lambs

Hosts : Ruminants

Site : small intestine

Species :

Nematodirus battus sheep, calves

N. filicolis sheep and goats

N. spthiger sheep and goats, occasionally cattle

Distribution : Worldwide

Nematodirus battus

Gross :

The adult are slender worms about 2cm long. Twisted worms produces an appearance similar to that of cotton wool.



Microscopic :

N. battus is characterized by having only one set of parallel rays in each bursal lobe while the female worm has a long pointed tail and the large egg is brownish with parallel sides.

Life cycle :

the main difference in the life cycle of *N. battus* compared with other parasitic worm is that development to an infective larvae take place within the egg

the egg → L1 develop in the egg → L2 develop in the egg → L3 infective stage (they migrate in to herbage and wait to eating by sheep → L4 and mature develop in to adult.

Pathogenesis :

Following ingestion of large numbers of L3 there is distribution of the intestinal mucosa particularly in the ileum. On the mucosal surface, development through L4 to L5 is complete and this consider with sever damage to the villi and erosion of the mucosa leading to the villous atrophy, exchange fluids and nutrients is grossly reduced, diarrhea, dehydrated, enteritis.

Clinical signs :

In severe infections diarrhea is the most prominent clinical signs dehydration, thirsty.

Epidemiology:

The two most important features of the epidemiology of *Nematodirus* :

- 1- The capacity of the free living stages (Larvae) ,the egg containing the L3 to survive on pasture , some for up to two years.
- 2- The critical hatching requirement of most eggs.

Diagnosis :

- Grazing history
- Clinical signs
- Post-mortem examination
- egg fecal count.

Treatment:

- Levamisole , - Avermectin , - benzimidazole

Control :

The disease can be controlled by avoiding the grazing of successive lamb crops on the same pasture. Control can be achieved by anthelmintic prophylaxis.

Family : Dictyocaulidae

Genus : Dictyocaulus (lung worms)

This genus living in the bronchi of cattle, sheep , horses and donkeys, is the major cause of parasitic bronchitis in these host.

Hosts : Ruminants, horses and donkeys.



Species :

Dictyocaulus viviparous in cattle

D. filaria in sheep and Goats

D. arnfieldi in donkeys and horses.

Distribution : Worldwide

Identification :

The adults are slender thread-like worms up to 0.8 cm in length , their location in the trachea and bronchi and their size are diagnostic.

D. viviparus :

Is the cause of parasitic bronchitis in cattle, also known verminous pneumonia of dictyocaulosis. The disease is characterized by bronchitis and pneumonia and typically affects young cattle during their first grazing season. The disease is prevalent in temperate areas with high rainfall.

Life cycle :

Eggs hatch in the lung L1 swallowed pass through the feces L2 L3 Ingestion penetrate intestinal wall Lymph vessels moult to L4 lung (male and female in the bronchi of the host)

The prepatent period is around 3-4 weeks.

Pathogenesis :

This may divided into 4 phases.

1- Penetration phase : 1-7 days during this period the larvae are making their way to the lungs and pulmonary lesions are not yet apparent.

2- Prepatent phase : 8-25 days this phase starts with appearance of the larvae with in the alveoli and bronchi where they cause alveolitis and bronchitis.

The end of this phase characterized by mucus containing immature lung worms in the air ways.

Heavily infected animals whose lungs contain several thousand developing worms may die from day 15 due to respiratory failure sever interstitial emphysema and pulmonary oedema.

3- Patent phase : 26-60 days this is associated with 2 main lesions

first a parasitic bronchitis characterized by the presence of hundreds or even thousands of adult worms in the frothy white mucus in the lumina of the bronchi.

secondly the presence of dark red collapsed areas around infected bronchi this is parasitic pneumonia caused by the aspiration of egg and Larvae

4- Post patent phase : 60-61 days in untreated calves, this is normally the recovery phase after the adult lung worms have been expelled. The lesion is interstitial emphysema , pulmonary oedema, gaseous exchanges at the alveolar surface it is thought to be due to the dissolution and aspiration of dead worm material into the alveoli.

Clinical signs :

* Mildly affected animals cough intermittently, particularly when exercised.

* Moderately affected animals have frequent coughing at rest tachypnea,

* severely affected animals show severe dyspnea , month breathing with the head and neck outstretched, deep harsh cough, salivation, anorexia and sometimes time death in 24-48 hours.



Epidemiology :

Generally only calves in their first grazing season are clinically affected, where the disease is endemic older animals have a strong acquired immunity. In endemic areas infection may persist from year to year in two ways :

- 1- Over wintered larvae L3 may survive on pasture from autumn until late spring in sufficient numbers to initiate infection.
- 2- Carrier animals : small numbers of adult worms can survive in the bronchi of infected animals, particularly yearlings until the next grazing season.

Diagnosis :

- Clinical signs .
- history of grazing
- Larvae are found only in the feces of patent cases by Baermann method
- eggs may be found in the sputum or discharge

Treatment : Benzimidazoles, levamisol, avermectin.

Control :

- The best method of preventing parasitic bronchitis is to immunize all young calves with lung worm vaccine.
- Control of parasitic bronchitis in first year grazing calves has been achieved by the use of prophylactic anthelmintic treatment.

Dictyocaulus arnfieldi :

The parasite of donkeys and horses

Life cycle :

The life cycle is not fully known. But is considered to be similar to that of bovine lung worm. *D. viviparus* except in the following respects. The adult worms are most often found in the small bronchi and their eggs containing the first stage larvae hatch soon after passed in the feces. The prepatent period is between 2-4 months.

Pathogenesis :

The characteristic lesion is similar in both horses and donkeys and in some differences from bovine parasitic bronchitis.

Clinical signs :

- are rarely seen in donkeys.
- hyperpnea and harsh lung sounds may be detected.
- In older horses infections become patent but are often associated with persistent coughing and increased respiratory rate.

Epidemiology :

Most commonly this occurs when donkeys are grazed and companion with horses, donkeys acquire infection as foals and tend to remain infected.

Diagnosis :

- modified Baermann technique is used for larvae detection from feces
- clinical signs may be suggestive of *D. arnfieldi* infection.

Treatment :

Levamisol, febendazole or mebendazole.



Control :

- Ideally , horses and donkeys should not be grazed together.
- Treat the donkeys preferably in the spring with a suitable anthelmintic.

Dictyocaulus filaria :

- The most important lung worm of sheep and goats.
- Is commonly associated with a chronic syndrome of coughing and unthriftiness which usually affects lambs and kids.

Life cycle :

Similar to that of *D. viviparus* except that the prepatent period is five weeks.

Pathogenesis :

Similar to that of *D. viviparus* infection.

Clinical signs :

Coughing and unthriftiness, dyspnea , nasal discharge are also present, diarrhea or anemia due to concurrent gastrointestinal trichostrongylosis or Fasciolosis.

Epidemiology :

the ewe as a carrier are significant factors in the persistence of infection on pasture from year to year. In ewes it seems likely that the parasites are present largely as hypobiotic larvae in the lung during each winter and mature in the spring.

Diagnosis :

Similar to that of *D. viviparus* infection.

Treatment and control :

Similar to that of *D. viviparus* infection



Super family Spiruroidea :

Family : Spiruridae

Genus : Habronema

Hosts : Horses and Donkeys

Intermediate hosts : Muscid flies.

Sites : Stomach

Species : Habronema muscae , H. microstoma ,H.megastoma

Distribution : world wide.

Identification :

- Slender white worms 1-2.5 cm long.

Life cycle :

Eggs or L1 are passed in the faces the L1 are ingested by larval stages of muscid flies including Musca, stomoxys and Haematobia which are often present in faces development to L3 the L3 pass from its mouth parts and put on to the nostrils ,lips, wounds of the horse . when swallowed, development to adult takes place in the glandular area of the stomach in approximately two months.

Pathogenesis :

The adults in the stomach may cause:

- gastritis .

-granulomatous lesion in wounds

- conjunctivitis with nodule are thickening and ulceration of the eyelids associated with invasion of eyes.

Clinical signs :

-non healing reddish cutaneous granuloma

-later the lesion may become more fibrous

-conjunctivitis with nodular ulcer .

Epidemiology :

The seasonality of cutaneous lesion is related to the activity of fly vectors.

Diagnosis :

- clinical signs.

- the Gastric infection is not easily diagnosed, because few eggs and larvae are passed.

-larvae or worms may be gastric lavage through stomach tube

Treatment and control :

Ivermectin, radiation therapy and surgery have been used in more chronic cases, prevent injuries and to control fly populations.

Family : Thelezidea

Genus: Thelezia

Hosts : Cattle ,birds, other domestic animals and occasionally man.



Intermediate hosts :

Musca domestica .

Site : Ocular region especially the conjunctival sac and lachrymal duct.

Identification :

Small thin white worms 1-2 cm long a mouth capsule is present and the cuticle has prominent striations at the anterior end.

Life cycle :

Indirect , the L1 passed by the female worm in the lachrymal secretion → ingested L1 by the fly
development L1 to L3 occurs in the fly → L3 migrate to the mouth parts of the fly → then
transferred to the final host when the fly feeds development in the eye

Pathogenesis :

Lesions are caused by the serrated cuticle of the worm and most damage results from movement by the active young adults causing lachrymation, followed by conjunctivitis in heavy infections the cornea may become cloudy and ulcerated.

Clinical signs :

Lachrymation, conjunctivitis and photophobia. Flies are usually clustered around the eye because of the excessive secretion , in sever the whole cornea can be opaque.

Epidemiology :

Thelazia infections occur seasonally and are linked to periods of maximum fly activity the parasite can survive in the eye for several years.

Diagnosis :

This is based on observation of the parasites in the conjunctival sac, it may be necessary to instill a few drops of local anesthetic to facilitate manipulation of third eyelid.

Treatment and control :

Based on the:

- manual removal of the worms under a local anesthetic.
- administering an effective anthelminitic such as levamisole or an avermectin



Super family Filarioidea :

Family : Filarioidea

Genus: Parafilaria

P. bovicolla :

Hosts : cattle and buffalo.

Intermediate hosts : Muscid flies.

Site : subcutaneous and intramuscular connective tissue.

Distribution : Africa, Asia , Europe and Sweden.

Identification :

Slender white worms 3-6 cm in length anteriorly, small embryonated eggs are laid on the skin surface where they hatch to release the microfilariae

Life cycle :

Eggs or larvae (L1) present in exudates from bleeding points in the skin surface ingested by flies develop to L3 in flies → L3 deposit in lachrymal secretions or skin wounds in other cattle when infected flies feed → L3 migrate and develop to the adult stage under the skin in 5-7 months.

Pathogenesis :

The adults of this genus live under the skin, they produce inflammatory lesions and, during egg laying, haemorrhagic exudates or "bleeding point" on the skin surface.

Clinical signs

The signs of parafilariosis are pathognomonic. Active bleeding lesions are seen most commonly in warm weather to coincide with the presence of the fly intermediate host.

Epidemiology :

Area previously free from infection Parafilaria infection may be introduced by the importation of cattle from endemic areas, spread will depend on presence of specific fly vectors.

Diagnosis :

- clinical signs.
- Small embryonated eggs or microfilariae may be found on examination of exudate from bleeding points.
- Sero-diagnosis using ELISA technique has been developed.

Treatment : Ivermectin or nitroxylin



Family : Onchocercidae

Genus: Onchocerca

Species: O. armilata vector: unknown
 O. gutturosa vector: simulium
 O. gibsoni vector: culicoides

Hosts : cattle

Intermediate hosts : unknown

Sites : in the skin of hump, neck, dewlap and aorta .

Identification :

The slender worms range from 2-6 cm in length and lie tightly coiled in tissue nodules, in active lesions the presence of worms is readily established on section of these nodules.

Life cycle :

The life cycle of Onchocerca Spp. is typically filaroid, with the exception that the microfilariae occur in the tissue spaces of the skin, rather than in the peripheral blood stream.

Diagnosis :

- Depends on the finding of microfilariae in skin biopsy samples.
- The piece of the skin is placed in warm saline and teased to allow emergence of the microfilariae, and is then incubated for six hours or more, the microfilariae are readily recognized by their sinuous movements in a centrifuged sample of the saline.

Treatment :

Ivermectin , local treatment with synthetic pyrethroids

Control :

The use of microfilaricides will reduce the numbers of infected flies.

Family : Setaridae

Genus: Setaria

Hosts : ruminants and equines.

Intermediate hosts : Many species of mosquito.

Site : usually the peritoneal surface and free in the peritoneal cavity less commonly the pleural cavity and following erratic migration to the CNS.

Species : Setaria digitata in cattle and wild ruminants
 Setaria equine in horses and donkeys.

Distribution : worldwide

Identification :

Long slender worms, up to 12 cm in length the site and gross appearance are sufficient for generic identification.

Life cycle :

Indirect , The microfilariae in the blood stream are taken up by mosquitoes in which development to L3 takes about 12 days, the prepatent period is 8-10 months.



Pathogenesis :

The worms in their normal site are harmless and are only discovered at necropsy. *S. digitata* may have an erratic migration in sheep and goats and enter the spinal canal causing cerebrospinal nematodosis "lumbar paralysis" which is irreversible and often fatal.

Clinical signs :

There are no clinical signs when the worms are in their normal site, but when nervous tissue is involved there is locomotor disturbance, usually of the hind limbs.

Epidemiology :

Their prevalence is higher in warmer countries. Where there is seasonal activity of the mosquito vectors.

Diagnosis :

- Finding of the microfilariae in routine blood smears in adult (living animal).
- In cases of cerebrospinal nematodosis, diagnosis is only possible by microscopic examination of the spinal cord.

Treatment :

There is no treatment for setarial paralysis. Ivermectin has been reported to be effective against adult *S. equine*.

Control : depend on control of the mosquito vectors.



Super family Trichuroidea :

- Are found in a wide variety of domestic animals.
- There are 3 genera of interest.
- 1- Trichuris is found in the caecum and colon of mammals.
- 2- Capillaria is most commonly present in the alimentary or respiratory tract of mammals of birds. Both lay eggs with plugs at both poles.
- 3- Trichinella are found in the small intestine of mammals and produce larvae which immediately invade the tissue of the same host.

Family : Trichuridae

Genus : Trichuris (whip worms)

- The adults are usually found in the caecum.
- The common species are: Trichuris ovis □ sheep and goat
Trichuris globulosa □ cattle.

Identification :

Gross : The adults are 4-6cm long with a thick posterior end tapering rapidly to a long filamentous anterior end which is characteristically embedded in the mucosa because of their appearance the members of this genus are often called the "whip worms".

Microscopic :

The male tail is coiled and possesses a single spicule in a sheath; the female tail is merely curved. The characteristic eggs are lemon (or barrel) shaped with a transparent plug at both ends in the faces these eggs appear yellow or brown in color.

Life cycle :

The infective stages is the L1 with in the egg which develops in one or two months of being passed in the faces depending on the temperature. Survive for several years , after ingestion , the plugs are digested and the released L1 penetrate the glands of the caecal mucosa, subsequently all four moults occur within these glands, the adults emerging to lie on the mucosal the prepatent period ranges from 6-12 weeks depending on the species.

Pathogenesis :

Most infections are light and a symptom a tic occasionally when large numbers of worms are present they cause a diphtheritic inflammation of the caecal mucosa. This results from the sub-epithelial location and continuous movement of the anterior end to the whip worm as it searches for blood and fluid.

Clinical significance :

The clinical signs of this genus especially in ruminants is negligible although isolated our breaks have been recorded.

Epidemiology :

The most important feature is the longevity of the eggs which after three of four years may still survive as a reservoir of infection.



Diagnosis :

- Diagnosis may depend on finding numbers of trichuris eggs in the feces.
- Clinical signs may occur during the prepatent period.

Treatment : Benzimidazoles, avermectins, levamisole.

Control : Prophylaxis is rarely necessary. Particularly in ruminants.

Family : Capillaridae

Genus : Capillaria

- Very thin hair-like worms are not readily visible to the naked eye in unprepared gut contents.
- There are many species in mammals and birds.

Identification :

- These are very fine filamentous worms between 1-5cm long.
- The males have a long single spicule and often possess a primitive bursa like structure.
- The females contain eggs which resemble those of Trichuris in possessing bipolar plugs. But which are more barrel – shaped and colorless.

Capillaria in Birds :

Capillaria obsignata : is present in the upper small intestine of chickens, turkeys and pigeons and has direct life cycle.

Capillaria caudinflata: is also found in the small intestine of chickens and turkeys, the egg of this species requires to be ingested by an earth worm in which it hatches, the final host being infected by ingestion of the earth worm.

Capillaria contorta : occurs in the esophagus and crop of the chicken, turkey, duck and wild birds. Earth worm intermediate host also essential. The prepatent period of these three avian species is 3-4 weeks.

Pathogenesis and clinical significance :

Like Trichuris the anterior ends of the parasite are buried in the mucosa and in heavy infection cause diphtheritic inflammation leading to in appetite and emaciation diarrhea , mortality in cases may be high.

Epidemiology :

Young birds are most susceptible to Capillaria infections, while adults may serve as carriers, C. obsignata is perhaps most important since, having a direct life cycle. Its occur indoors in birds kept on deep litter.

Diagnosis :

- Because of the non-specific nature of clinical signs and the fact that, in heavy infections, these may appear before Capillaria eggs are present in the feces.
- Diagnosis depends on necropsy and care full examination of the esophagus, Crop or intestine for the presence of worms, this may be carried out by microscopic examination of mucosal scrapings

Treatment :

Levamisole in the drinking water is highly effective.



Family : Trichinellidae

Genus: Trichinella

Trichinella spiralis

Is a nematode with a very wide host range and the cause of an important world wide zoonosis.

Hosts : Most mammals, from the zoonotic aspect.

Sites :

The adults occur in the small intestine and their larvae in the striated muscles, the diaphragmatic, intercostal and masseter muscles are considered to be predilection sites.

Distribution : worldwide

Identification :

Because of their short lifespan, the adult worms are rarely found in natural infections , the male is about 1.0mm long , the oesophagus is at least one third of the total body length and the tail has two small cloacal flaps. But no spicule the female 3mm long and the uterus contain developing larvae. *Trichinella* infection is most easily identified by the presence of coiled larvae in striated muscle.

Life cycle :

Ingestion cystic larvae which are found in muscle → 1st two moults are completed within 26 hours and fourth moults occur less than two days in small intestine of host adult worm copulation male die and female produce egg L1 thoracic duct lymph upper left vena cava blood striated muscles surrounded by capsule muscle calcification larvae have no development until eating .

Pathogenesis and clinical signs :

Infection in domestic animals is invariably light, and clinical signs do not occur.

Epidemiology :

Depends on two factors first, animals may become infected from a wide variety of sources , predation and cannibalism being perhaps the most common other feeding on carrion, the second factor is the wide host range of the parasite.



Class :Trematoda

The class Trematoda falls into two main subclasses

1- Monogenea, which have a direct life cycle, found mainly as external parasite of fish

2-Digenea, which require an intermediate host. which found in vertebrates and are of considerable veterinary importance.

The adult digenetic trematodes, commonly called flukes

occur primarily in the bile ducts, alimentary tract and vascular system.

Most flukes are flattened dorsoventrally. have a blind alimentary tract. suckers for attachment ,and are hermaphrodite.

The eggs pass out of the final host usually in faeces or urine, the larval stages develop in a molluscan intermediate host for a few species,

second intermediate host is involved, but the molluscan is essential for all members of the group

There are many families in the class Trematoda, and those which include parasites of major veterinary

importance are the Fasciolidae, Dicrocoelidae

Paramphistomatidae and Schistosomatidae. of lesser importance are the Troglotrematidae- and

Opisthorchiidae.

Subclass Digena

STRUCTURE AND FUNCTION OF DIGENETIC TREMATODES

The adult possesses two suckers for attachment.

The oral sucker at the anterior and surrounds the mouth and the ventral,

The body surface is a tegument which is absorptive and is often covered with spines. The muscles lie immediately below the tegument.

There is no body cavity and the organs are packed in a parenchyma

The digestive system is simple, the oral opening leading into a pharynx, oesophagus and a pair of

branched intestinal caeca which end blindly. Undigested material is presumably regurgitated.

The excretory system consists of a large number of ciliated flame cells, which impel waste metabolic products along a system of tubules which ultimately join and open to the exterior.

The nervous system is simple consisting of a pair of longitudinal trunks connecting anteriorly with two ganglia

The trematodes are usually hermaphrodite and both cross-and self-fertilization may occur.

The male reproductive system consists of a pair of testes each

leading into a vas deferens; these join to enter the cirrus sac

containing a seminal vesicle and the cirrus, a primitive penis which terminates at the common genital opening.

The female system has a single ovary leading into an oviduct which is expanded distally to form ootype , there the ovum acquires a yolk from the secretion of the vitelline glands and ultimately a shell.

The egg passes along the uterus, the shell becomes hardened and toughened and is finally extruded through the genital opening adjacent to the ventral sucker



The mature egg is usually yellow because of the tanned protein shell and most species have an operculum

Food, generally blood or tissue debris is ingested and passed into the caeca where it is digested and absorbed.

Metabolism appears to be primarily anaerobic

THE LIFE CYCLE OF DIGENETIC Trematodes

The essential point of the life cycle is that whereas one nematode egg can develop into only one adult one trematode egg may eventually develop into hundreds of adults.

This is due to the phenomenon of paedogenesis in the molluscan intermediate host,

The adult flukes are always oviparous and lay eggs with an operculum or lid at one pole.

In the egg the embryo develops into a pyriform (pear-shaped), ciliated larva called a miracidium.

Under the stimulus of light, the miracidium releases an enzyme which attacks the proteinaceous cement holding the operculum in place

The miracidium, propelled through the water by its cilia, does not feed and must, for its further development, find a suitable snail within a few hours. It is believed to use chemotactic responses to 'home' on the snail and, on contact, it adheres by suction to the snail and penetrates its soft tissues aided by a cytolytic enzyme.

The entire process of penetration takes about 30 minutes after which the cilia are lost and the miracidium develops into an elongated sac,

the sporocyst, containing a number of germinal cells these cells develop into rediae

which migrate to the hepato-pancreas of the snail;

rediae are also larval forms possessing an oral sucker, some flame cells and a simple gut. from the germinal cells of the rediae arise the final stages.

the cercariae, although if environmental conditions for the snail are unsuitable, a second or daughter generation of rediae is often Produced instead . the cercariae in essence young flukes with long tail , emerge actively from the snail , usually in considerable numbers .

the cercariae swim for some time, utilizing even a film of water

, and within an hour or so attach themselves to vegetation, shed their tails and encyst this stage is called a metacercaria

Encysted metacercariae have great potential for survival extending to months.

Once ingested, the outer cyst wall is removed mechanically during mastication.

Rupture of the inner cyst occurs in the intestine and depends on a hatching mechanism, enzymatic in origin, triggered by a suitable oxidationreduction potential and a CO₂, system provided by the intestinal environment.

The emergent juvenile fluke then penetrates the intestine and migrates to the predilection site where it becomes adult after several weeks



Family :FASCIOLIDAE

These are large leaf-shaped flukes.

The anterior end is usually prolonged into the shape of a cone and the anterior sucker is located at the end of the cone

The ventral sucker is placed at the level of the 'shoulders of the fluke.

The internal organs are branched while the cuticle is covered in spines.

There are three important genera: Fasciola, and Fascioloides

Fasciola

The members of this genus are commonly known as liver flukes.

They are responsible for widespread morbidity and mortality in sheep and cattle characterized by weight loss, anemia and hypo proteinaemia

The two most important species are F, hepatica and F gigantica

Fasciola hepatica

Hosts

Most mammals: sheep and cattle are the most important

Intermediate hosts

Snails of the genus Lymnaea. The most common, L truncatula,.

Site

the adults are found in the bile ducts and the immature flukes in the liver parenchyma. Occasionally aberrant flukes become encapsulated in other organs, such as the lungs

Distribution

Worldwide

Identification

Gross

The young fluke at the time of entry into the liver is 1-2mm in length and lancet-like.

When it has become fully mature in the bile ducts it is leaf-shaped grey-brown in color and is around 3.5 cm in length and 1.0cm in width.

The anterior end is conical and marked off by distinct shoulders from the body

Microscopic

The tegument is covered with backwardly projecting spines.

An oral and ventral sucker may be readily seen.

The egg is oval, operculate, yellow and large (150um X 90um).

Life cycle

Eggs passed in the faeces of the mammalian host develop and hatch releasing motile ciliated miracidia.

This takes nine days at optimal temperatures of 22-26-°C and little development occurs below 10°C.



The liberated miracidium has a short life span and must locate a suitable snail within three hours if successful penetration of the later is to occur in infected snail development proceeds through sporocyst and radial stages to the final stages in the intermediate host .

the cercaria; these are shed from the snail as motile forms which attach themselves to firm surfaces, such as grass blades, and encyst there to form the infective metacercariae

It takes a minimum of 6-7 weeks for completion of development from miracidium to metacercaria, Metacercariae ingested by the final host excyst in the small intestine, migrate through the gut wall, cross the peritoneum and penetrate the liver capsule.

the young flukes tunnel through the parenchyma, then enter the small bile ducts where they migrate to the larger ducts and occasionally the gall bladder . the prepatent period is 10-12 weeks.

The minimal period for completion of one entire life cycle of *F.hepatica* is therefore 17-18 weeks.

The longevity of *F. hepatica* in untreated sheep may be years; in cattle it is usually less than one year

ECOLOGY OF LYMNAEA SPECIES IN TEMPERATE CLIMATES

Since *L. truncatula* is the most widespread and important species involved in the transmission of *F hepatica*,

L. truncatula is a small snail, the adults being about 1.0cm in length. The shell is usually dark brown and has a turreted appearance, being coiled in a series of spiral whorls.

EPIDEMIOLOGY

There are three main factors influencing the production of the large numbers of metacercariae necessary for outbreaks of fasciolosis.

1-Availability of suitable snail habitats:

L truncatula prefers wet mud to free water, and permanent habitats include the banks of ditches or streams and the edges of small ponds. Following heavy rainfall or flooding, temporary habitats may be provided by hoof marks, wheel ruts or rain ponds.

2-Temperature: A mean day night temperature of 10 C or above is necessary both for and for the development of *F. hepatica*

3-Moisture: The ideal moisture conditions for snail breeding and the development of *F. hepatica* within snails are provided when rainfall exceeds transpiration, and field saturation is attained.

Such conditions are also essential for the development of fluke eggs, for miracidia searching for snails and for dispersal of cercaria being shed from the snail.

PATHOGENESIS AND CLINICAL SIGNS

These vary according to the phase of parasitic development in the liver and the species of host involved.



Essentially the pathogenesis is two-fold:

the first phase Occurs during migration in the liver parenchyma and is associated with liver damage and hemorrhage.

The second occurs when the parasite is in the bile ducts, and results from the haematophagic activity of the adult flukes and from damage to the biliary mucosa by their cuticular spines

Ovine Fasciolosis

Fasciolosis may be acute, sub-acute or chronic.

"The acute disease occurs 2-6 weeks after the ingestion of large numbers of metacercariae, usually over 2000

. and is due to the severe haemorrhage which results when the young flukes. migrating in the liver parenchyma, rupture blood vessels. Damage to the liver parenchyma is also severe..

At necropsy the liver is enlarged, haemorrhagic and honeycombed with the tracts of migrating flukes

The surface covered with a fibrinous exudate

Subcapsular haemorrhagic are common and these may rupture so that a quantity of blood-stained fluid is often present in the abdominal cavity.

Outbreaks of acute fasciolosis are generally presented as sudden deaths during autumn and early winter subacute disease, metacercariae are ingested

over a longer period and while some have reached the bile ducts. where they cause a cholangitis, others are still migrating causing lesions less severe, but similar to those of the acute disease; thus the liver is enlarged with numerous necrotic or haemorrhagic tracts visible on the surface and in the substance.

clinical signs for 1-2 weeks prior to death; these include a rapid loss of condition, a marked pallor of the mucous membranes, and an enlarged and palpable liver. Submandibular or facial oedema and ascites may be present.

Chronic fasciolosis, which is seen mainly in late winter early spring, is the most common form of the disease. It occurs 4-5 months after the ingestion of moderate numbers, 200-500, of metacercariae.

The principal pathogenic effects are anaemia and hypoalbuminaemia and more than 0.5ml blood per fluke can be lost into the bile ducts each day.

Additional loss of plasma proteins occurs by leakage through the hyperplastic biliary mucosa

at necropsy the liver has an irregular outline and is pale and firm,

the liver pathology is characterized by hepatic fibrosis and hyperplastic cholangitis



clinically chronic fasciolosis is characterized by loss of condition and development of anemia and hypoalbuminaemia which can result emaciations paler of mucous membrane , submandibular odema and ascites

fasciola eggs can be demonstrated in the faces .

BOVINE FASCIOLOSIS

acute and subacute disease may occasionally occur especially in young calves, the chronic form of the disease is most important, as in sheep,

The pathogenesis is similar to that in sheep but has the added features of calcification of the bile ducts and enlargement of the gallbladder.

migration of the flukes is more common in cattle and encapsulated parasites are often seen in the lungs

on reinfection of adult cows migration to the foetus has been recorded resulting in prenatal infection.

In heavy infections, where anaemia and hypoalbuminaemia are severe.

submandibular oedema frequently occurs with smaller fluke burdens.

diarrhea is not a feature of bovine fasciolosis unless it is complicated by the presence of *Ostertagia* spp. Combined infection with these two parasites has been referred to as the fasciolosis /ostertagiosis complex.

Fasciola infections may cause a loss of production in milking cows during winter.

The main effects are a reduction in milk yield and quality,

DIAGNOSIS

1-Acute fascioliasis

Clinical signs (sudden death , blood and other fluids coming From the body opening

Necropsy finding (enlargement of liver , hemorrhage and fibrin exudate with young worms could be seen when squeeze the liver tissue

2-chronic fascioliasis

Clinical signs (loss of appetite , pallor mucous membrane , submandibular edema (bottle jaw))

Examination of fecal sample by sedimentation method to detect the eggs .

Necropsy finding : liver fibrosis , calcification in bile duct wall in bovine (pipe stem)

Serological test : detection of antibodies against components of flukes, the ELISA and the passive haemagglutination test being the most reliable



TREATMENT

1-Acute ovine fasciolosis:

Triclabendazole , rafoxanide , closantel and nitroxynil.

2-chronic ovine fasciolosis

Rafoxanide , nitroxynil , brotianide , closantel , oxyclozanide and triclebendazole, and can use albendazole and netobimin against adult flukes at increased dosage rate .:

Bovine fasciolosis

At present there is only one drug, namely triclabendazole, will remove the early stages. Apart from triclabendazole,

rafoxanide and nitroxynil and several others, such as clorsulon and niclofolan, used for subacute or chronic

albendazole is effective at an increased dosage rate

CONTROL

1 -Reduction of snail populations

2- Use of anthelmintics



Fasciola gigantica

Hosts

Ruminants

Intermediate hosts

Snails of the genus *Lymnaea auricularia*

Site

The adults are found in the bile ducts and the immature flukes in the liver parenchyma.

Distribution

Most countries . Does not occur in western Europe.

IDENTIFICATION

Gross

It is larger than *F. hepatica* and can reach 7.5cm in length.

The shape is more leaf-like, the conical anterior end is very short and the shoulders characteristic of *F. hepatica* are barely perceptible

Microscopic

The eggs are larger than those of *F. hepatica* ,measuring 190 X 100nm.

LIFE CYCLE

This is similar to *F. hepatica*, the main differences being in the time scale of the cycle. Most parasitic phases are longer and the prepatent period is 13-16 weeks.

PATHOGENESIS AND CLINICAL SIGNS

Similar to those of *F. hepatica*

. DIAGNOSIS AND TREATMENT

As for *F. hepatica*

CONTROL

The principles are the same as for the control of *F. hepatica* and are based on the routine use of anthelmintic together with measures to reduce populations of the snail intermediate host.

There is however, the important difference that the latter are water snails whose control depends on a different approach from that for the mud snail *L. truncatella*. Routine anthelmintic treatment of animals at seasons when heavy infections of adult flukes accumulate

Family :Dicrocoeliidae

These trematodes are small, lancet-like flukes occurring in the biliary and pancreatic ducts of vertebrates.

Miracidia are present in the eggs when they are passed in the faeces; there is no redial stage during development in the snail

Dicrocoelium

Hosts:

Sheep, cattle, deer and rabbits

Intermediate hosts:

Two are required:



Land snail of many genera
Brown ants of the genus formica
Site:
Bile ducts and gall bladder,
Distribution:
Worldwide.
Species:
Dicrocoelium dendriticum

IDENTIFICATION

Gross:

Is less than 1 cm long , lanceolate and semitransparent

Microscopic:

The gut is simple, consisting of two branches and resembles a tuning fork. Behind the ventral sucker testes lie in tandem with the ovary immediately posterior. There are no spines on the cuticle of fasciola).

The egg is small, , dark brown and operculate. usually with a flattened side contains a miracidium when passed in the faeces

LIFE CYCLE

The egg does not hatch until ingested by the first intermediate host, the land snail, in which two generations of sporocysts develop which then produce cercariae.

The latter are extruded in masses cemented together by slime. This phase of development takes at least three months.

The slime balls of cercariae are ingested by ants in which they develop to metacercariae mainly in the body cavity and occasionally the brain. The presence of a brain lesion in the ant, induced by metacercariae, impels the ant to climb up and remain on the tips of the herbage, thus increasing the chance of ingestion by the final host.

In the final host, the metacercariae hatch in the small intestine and the young flukes migrate up the main bile duct and thence to the smaller ducts in the liver. There is no parenchymal migration and the prepatent period is 10-12 weeks. The flukes are long lived and can survive in the final host for several years.

PATHOGENESIS

Although several thousand *D. dendriticum* are commonly found in the bile ducts, the livers are relatively normal; this is presumably due to the absence of a migratory phase . However, in heavier infections there is fibrosis of the smaller bile ducts and extensive cirrhosis can occur

Clinical signs

Anemia , edema and emaciations have been reported in severe cases



EPIDEMIOLOGY

There are two important features which differentiate the epidemiology of *Dicrocoelium* from that of *fasciola*

1-The intermediate hosts are independent of water and are evenly distributed on the terrain.

2-the egg can survive for months on dry pasture presenting a reservoir additional to that in the intermediate and final hosts.

DIAGNOSIS

based on fecal carination for eggs and necropsy findings.

TREATMENT

Nctobimin has been shown to be highly effective at a dose rate of 20mdkg. High doses of other anthelmintic required for effective removal of

Dicrorouliurn.The benzimiduole, albendazole. Give at 3 times is very effective , other drugs such as thiabendazole and fenbendazole are also effective but a very high dose rate

CONTROL

This is difficult because of the longevity of *D dendriticum*.

eggs, the wide distribution of the intermediate hosts and the number of reservoir hosts. Control depends almost entirely on regular anthelmintic treatment

