LECTURERS IN VETERINARY PARASITOLOGY

INTRODUCTION, NEMATODA AND SYSTIMIC PLATYHELMINTHES

THIRD YEAR STAGE

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CHAPTER TWO

NEMATODA

Nematoda Are free- living or parasitic ,un segmented worm ,usually cylindrical and elongate in shape, with **General characteristics**

1- un segmented worm

2- usually cylindrical and elongate in shape

3- An alimentary canal is present and consisted of

- mouth: which has thick cuticular walls and may contain special tooth-like structures

- Pharynx: which is usually cylindrical and surrounded by muscular tissue

- oesophagus: it is variation of structure that are used for the classification of species , it has wall contains three oesophageal glands.

- The intestine is simple tube ,it leads into the rectum into cloaca in male and anus in female

4-Tapering at the extremities

5- The cuticle which forms the covering is usually provided with circular annulations(not visible to the naked eye)

6- The body (cutical)may be smooth or have longitudinal striation

7- The cuticula is relatively thick in nematodes and is continuous with the cuticular lining of the buccal cavity , the oesophagus ,the rectum and the distal portion of the genital ducts.

8- cuticula may form special adhesive structures like;

Hooks or cephalic collar, cervical alae, bursa, papillae, lips, buccal capsule

9- The body layers consisting of an(outer layer is cortical ;matrix layer,fiber layer)

10- They are well supplied with nerve fibers &glandular structure

11- The part of the body behind the anal or cloacal opening is called the tail .

12- The excretory system ,which is also osmoregulatory,

13- It is a separated sex.

FAMILY: ASCARODIDAE

General Characteristics

- Mostly large nematodes

-Mouth surrounded by three lips.

-No buccal capsule.

-Oesophagus usually lacks posterior bulb.

-Intestine may have caeca .

- Tail of female blunt, of male frequently coiled.

- Two spicules in the male.

-Life cycle may be direct or indirect.

Parascaris equorum

General Characteristics

-Occurs in the small intestine

-The host :equines including the zebra and perhaps also cattle.

-The males are 15-28cm long and the females up to 50cm by 8mm.

-It is a rigid ,stout worms with a large head.

- Having three main lips .

-The male tail with small lateral alae .

-There are two double and three single pairs of postcloacal papillae .

-The spicules are about 2-2.5mm long.

-The vulva is situated at the end of the first quarter of the body.

- Eggs are subglobular with thick.

Life Cycle

Fertilized female may lay 2ooooo eggs peer day, so, the eggs in feces of the host:

- develop to the infective stage(second stage larva in egg) in 10 days or longer depending on the temperature.

-the eggs are very resistant to adverse condition ,like drying or freezing and to chemicals and may remain viable for as long as five years. But sandy soil with direct sunlight kill them in few weak.

-The larva has two ecdyses occur before eggs hatch to third stage larva.

-The infection takes place through ingestion of the eggs with food or water or from the soiled skin of the mother in the case sucking .

-The ingested eggs hatch in the intestine .

-The larvae burrow into the wall of the gut.

-The larvae passed into the liver through the peritoneal cavity or by hepatoportal blood stream.

-The larvae arrive in the liver in 24 hours after infection.

-From the liver a larva carried by blood through the heart to the lungs.

-The larvae are recognizable as third stage larvae between the 4-5 days after infection.

-There is one moult in lung and liver.

-Larvae break out of alveolar capillary in to the alveolus and pass through the alveolar detach the small bronchioles and then gradually ascend the bronchial tree after than into large bronchi and trachea.

-Larvae migrate to the pharynx when they are swallowed and 3ed stage larvae arrived in the intestine through 7-8 after infection.

-There are two moult occurred in the intestine it reach to adult worms.

-Worms reach maturity 80-83 days after infection (prepatent period).

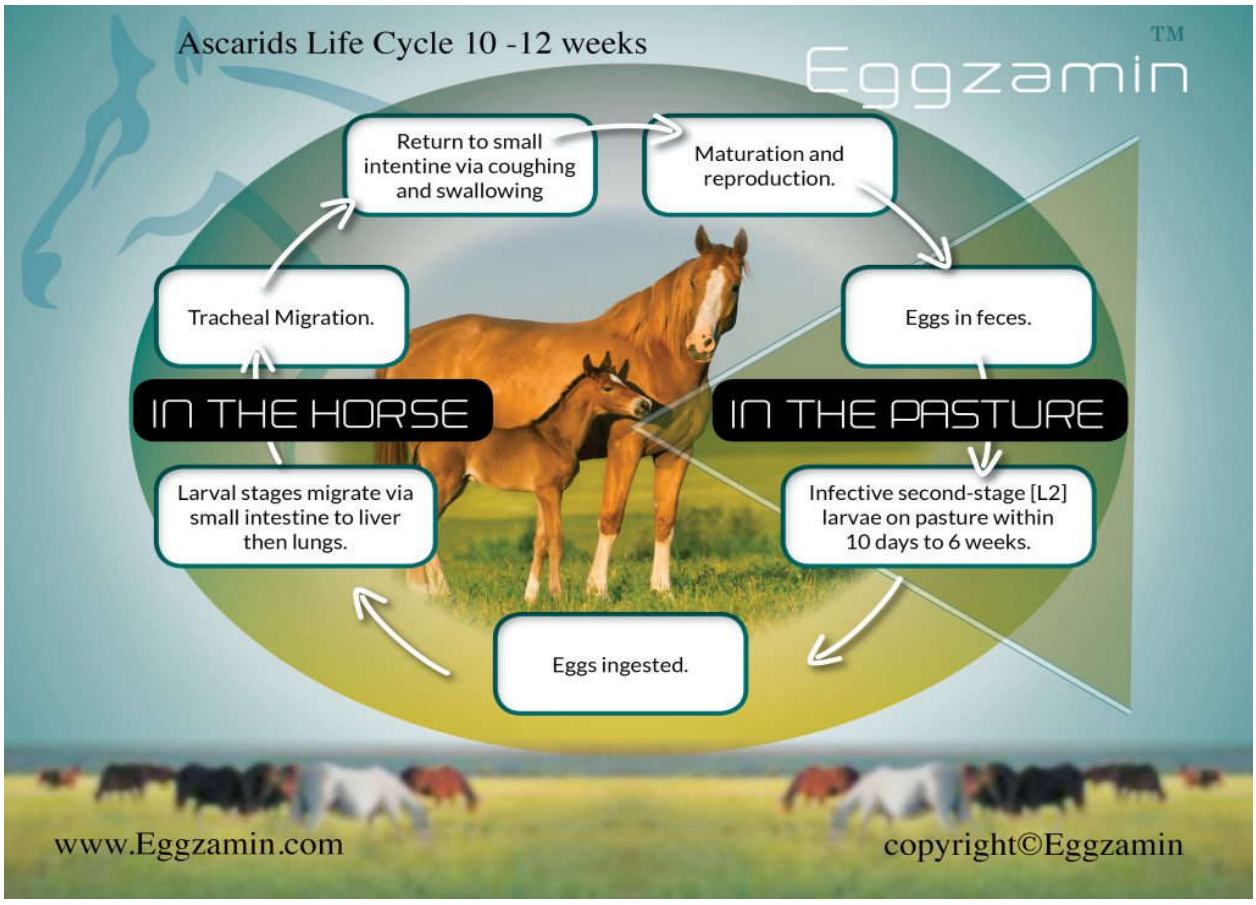


Figure (1): Ascaridia Life Cycle

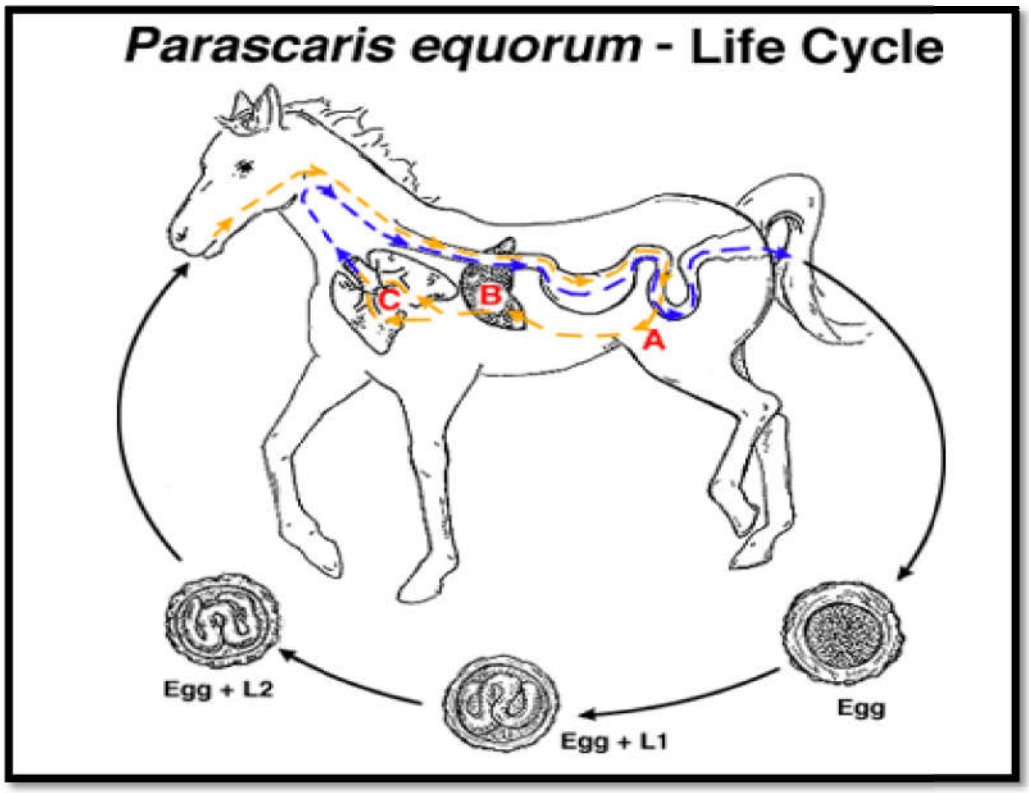


Figure (2): Life Cycle of *Parascaris equorum*

Pathogenicity and Pathogenesis

Pathogenicity in Horses:

1-catarrhal enteritis

2-intestine obstruction

3-a depression of albumin synthesis

4- hepatic and pulmonary damage caused by larval migration.

5-circulating eosinophilia.

Clinical Signs

Foal three to nine months of age especially suffer from this parasite .

1-diarrhoea which may be foetid in odour and pale in colour

2-general malais

3- the coat is harsh

4-animals may become pot-bellied

5- coughing

Diagnosis

- clinical signs

-fecal exam.

-serological exam.

Treatment

Anthelmintic used in horses are effective against adult ascaris:

Rx, thiabendazaol, mebendazaol, fenbendazaol and haloxon

Toxocara canis

General Characteristics

-Occurs in the small intestine inside their final host dog and fox.

-The males are 10cm long and the females up to18cm.

- large cervical alae .

-It having three main lips .

-The male tail has a terminal narrow appendage and caudal alae .

-The female genital organs extend interiorly and posteriorly to the vulvar

-The spicules are 0.75-0.95mm long.

-The eggs are subglobular with thick ,final pitted shells and measure about 90 by 75Mm.

Life Cycle

The life cycle of *T. canis* is complex and according to the age of the host may involve

1- prenatal(transuterine) in female but in male is somatic migrants.

2-by colostral (lactogenic) transmission .

3-direct transmission.

4-by paratenic host.

-The female may lay as 2oooo eggs in day.

The eggs in the feces of the host-

- develop to the infective stage(second stage larva in egg) in 10-15 days or longer depending on the temperature.

-The eggs are very resistant to adverse condition ,like drying or freezing and to chemicals and may remain viable for as long as five years. But sandy soil with direct sunlight kill them in few weak.

-The larva has two ecdyses occur before eggs hatch to third stage larva.

-The infection takes place through ingestion of the eggs with food or water or from the soiled skin of the mother in the case sucking .

-The ingested eggs hatch in the dudenum .

-The larvae burrow into the wall of the gut.

-The larvae passed into the liver through the lymph stream to mesenteric nodes and then by hepatoportal blood stream.

-The larvae arrive in the liver in 2 days after infection.

-From the liver a larva carried by blood through the heart to the lungs.

-The larvae are recognizable as third stage larvae between the 4-5 days after infection.

-There is one moult in lung and liver.

-Larvae break out of alveolar capillary in to the alveolus and pass through the alveolar duct tch o the small bronchioles and then gradually ascend the bronchial tree after than into large bronchi and trachea.

-Larvae migrate to the pharynx when they are swallowed and 3ed stage larvae arrived in the intestine through 7-8 after infection.

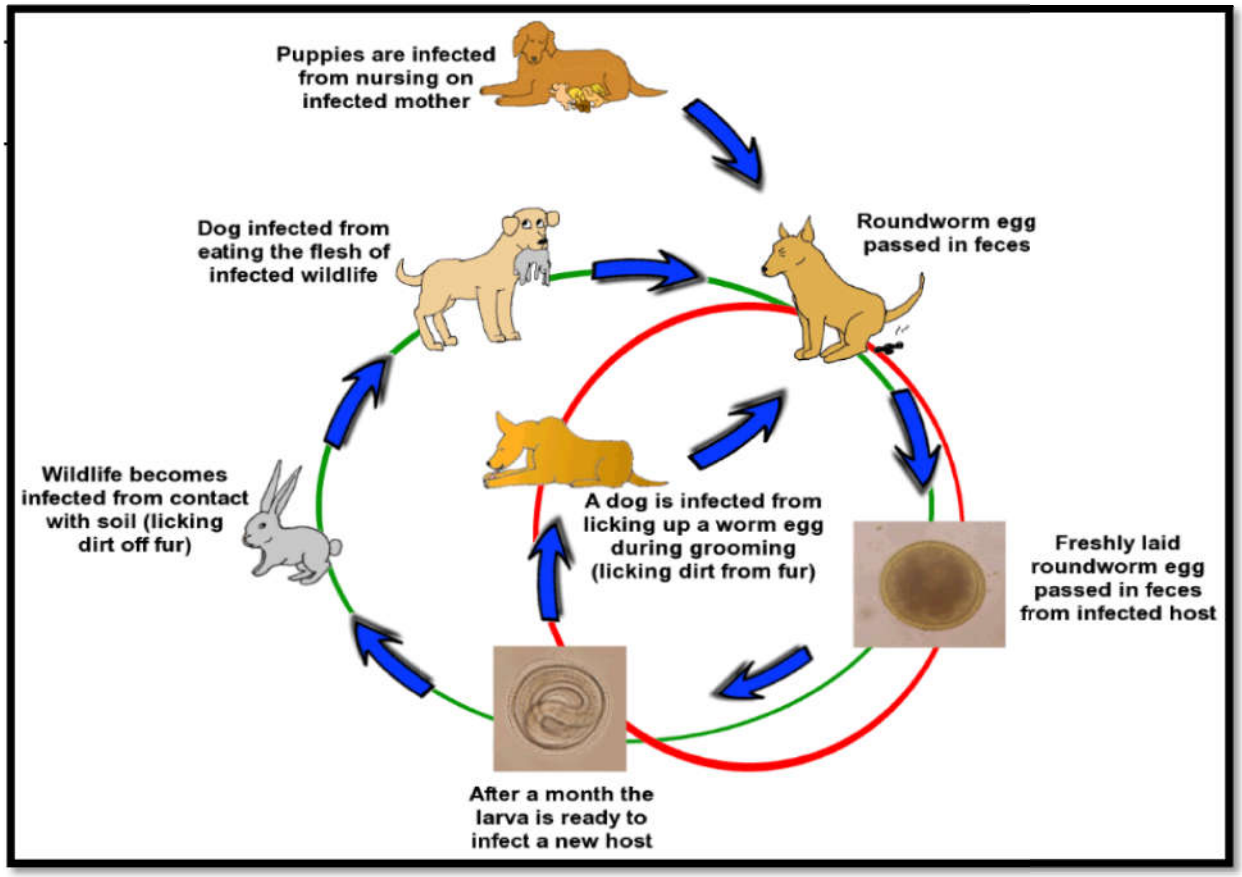


Figure (3): Life Cycle of Toxocara canis in Dogs and herbivorous animals

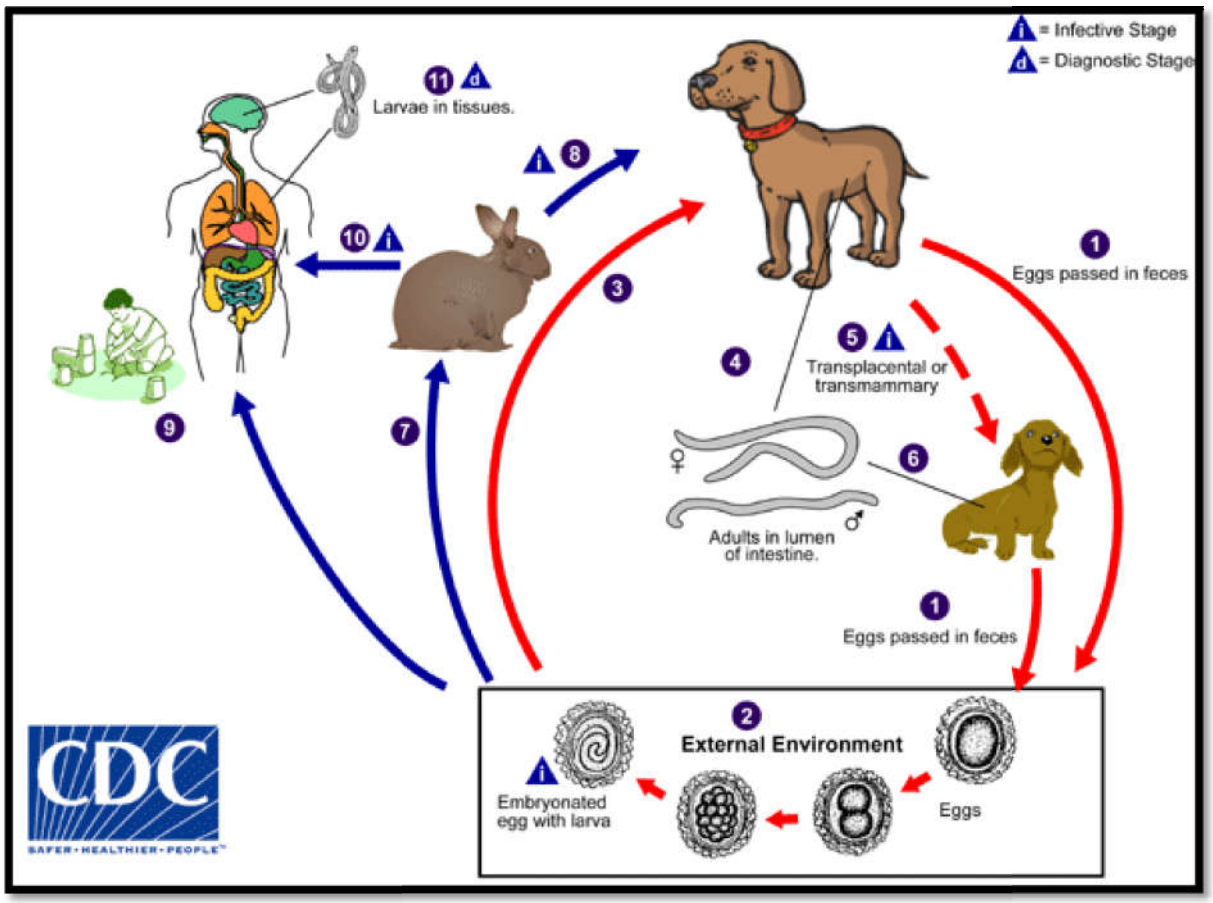


Figure (4): Life Cycle of Toxocara canis in Dogs and Human as Paratenic Host

Pathogenicity and Pathogenesis

-occur in puppies.

1-catarrhal enteritis

2-intestine obstruction

3-a depression of albumin synthesis

4- hepatic and pulmonary damage caused by larval migration.

5-circulating eosinophilia.

6-pnemonia

Clinical Signs

1-diarrhoea which may be foetid in colour and pale in colour

2-general malais

3- the coat is harsh

4-animals may become pot-bellied

5- coughing

6-vomiting

7-death frequently occur 2-3 weeks after birth.

Diagnosis

- clinical signs

-faecal exam.

-serological exam.

Treatment

Anthelmintic used in dog are susceptible adult ascaris .Larval stages in the tissues are much less susceptible .

Rx, salt of piprazine (dihydrochloride and citrate), thenium and Ivermctine

Visceral Larva Migrans

This condition of children is mainly caused by the larvae of *T. canis* ,though the larval stages of *T. leonina ,T. cati, Capillaria hepatica* (of rodents),it is characterized by

1-chronic granulomatus (usually eosinophilic)lesion in liver, lung, brain ,eye .

2-enlarged liver

3- hepatomegaly

4-pulmanary infiltration

5-loss of weight and appetite

6-cough

7- circular eosinophilia

Ascaridia galli

General Characteristics

-Occurs in the small intestine

-The host :fowl, guinea fowl turkey, goose and various wild birds in most parts of the world.

-The males are 50-76mm long and the females 72-116mm.

-It having three large lips .

-The male tail has small alae and bears ten pairs of papillae .

-the oesophagus has no posterior bulb.

-The spicules are sub –equal, about 1-2.4mm long.

-The eggs are subglobular with thick.

Life Cycle

-The female may lay eggs.

The eggs in the feces of the host-

- develop to the infective stage(second stage larva in egg) in 10 days or longer depending on the temperature.

-the eggs are very resistant to adverse condition ,like drying or freezing and to chemicals and may remain viable for as long as three months. But dry ,hot weather killed the eggs.

-The larva has two ecdyses occur before eggs hatch to third stage larva.

-The infection takes place through ingestion of the eggs with food or water sucking

-The ingested eggs hatch in the intestine .

-The larvae burrow into the wall of the gut after eight days.

-The larvae passed into the intestine mucosa from the eighth to the seventeenth days.

-The larvae reenter the lumen and reach maturity in six to eight weeks according to the age of the host may involve .

-The larvae are recognizable as third stage larvae between the 4days after infection , but L4 at 14-15days.

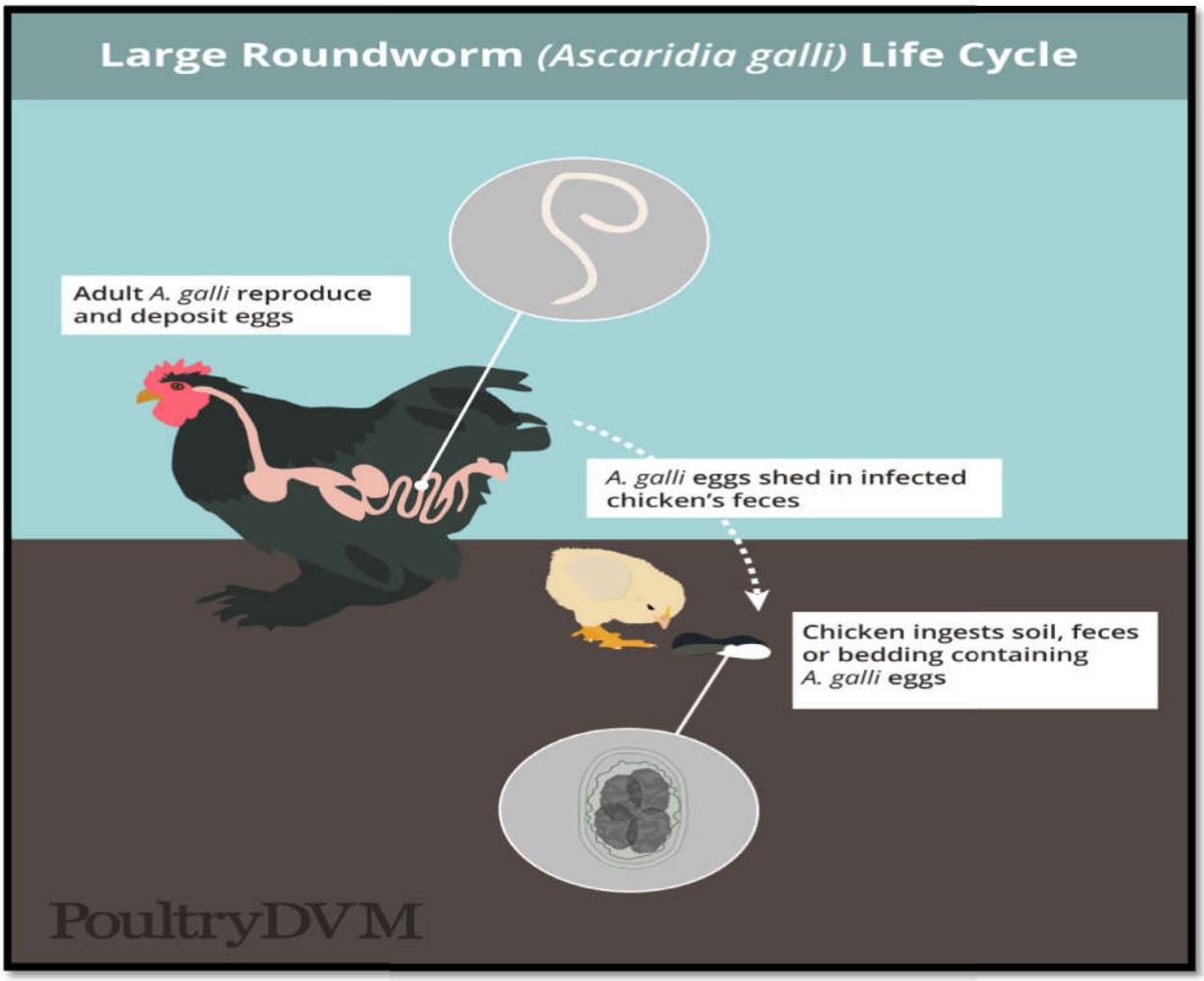


Figure (5): Life Cycle of *Ascaridia galli*

Pathogenicity and Pathogenesis

-young birds are more susceptible to infection than adult birds chickens over three month of age are more resistant to infection ,marked lesion may be produced when large numbers of parasites.

1- enteritis

2-intestine obstruction

3-they cause haemorrhage

4-pnemonia

Clinical Signs

1-diarrhoea

2-the birds become unthrifty

3- markedly emaciated

4-generally weak and egg production is decreased

Diagnosis

- clinical signs

-faecal exam.

-serological exam.

Treatment

Rx, salt of piprazine (dihydrochloride and citrate) and Albendazoal

FAMILY: HETERAKIDEA

General Characteristics

-Medium sized to small worms.

-it having three lips around the mouth.

-a small buccal cavity and pharynx.

-Lateral alae extending down the body.

-Oesophagus in three parts (a short pharynx , a cylindrical,middle par and bulbous posterior part.

-A pre-anal sucker at the tail end of male which has a chitinous rim ,with many anal papillae

Hetrakis gallinarum

General Characteristics

-Occurs in the caeca.

-The host :fowl, guinea fowl turkey, goose and various wild birds in most parts of the world.

-The males are 7-13mm long and the females 10-15mm.

-It having three large lips .

-The male tail is provided with large alae

-per-cloacal sucker and twelve pairs of papillae.

-there are large lateral alae extending some distance down the side of the body .

-the oesophagus has a strong posterior bulb.

-The spicules are unequal, the right being slender and 2mmlong,while the left has broad alae and measure about0.65-0.7mm long.

-the vulva opens directly behind the middle of the body.

-The eggs have thick ,smooth shell ,they measure 65-80×35-46Mm and are un segmented when laid.

Life Cycle

-The female lay eggs.

-The eggs in the feces of the host

- develop to the infective stage(second stage larva in egg)in the open in 14 days or longer depending on the temperature.

-the eggs are very resistant to adverse condition , and may remain viable soil for months. But dry ,hot weather killed the eggs.

-The infection takes place through ingestion of the eggs with food or water sucking.

-The ingested eggs hatch in the intestine after 1 or 2 hours .

-second stage larvae spend two to five days in the glandular epithelium before continuing their development in the lumen.

-They molt to the third stage on the sixth day after infection ,L4 on the tenth day and L5 on the fifteenth day .

-The prepatent period is 24- 30 day.

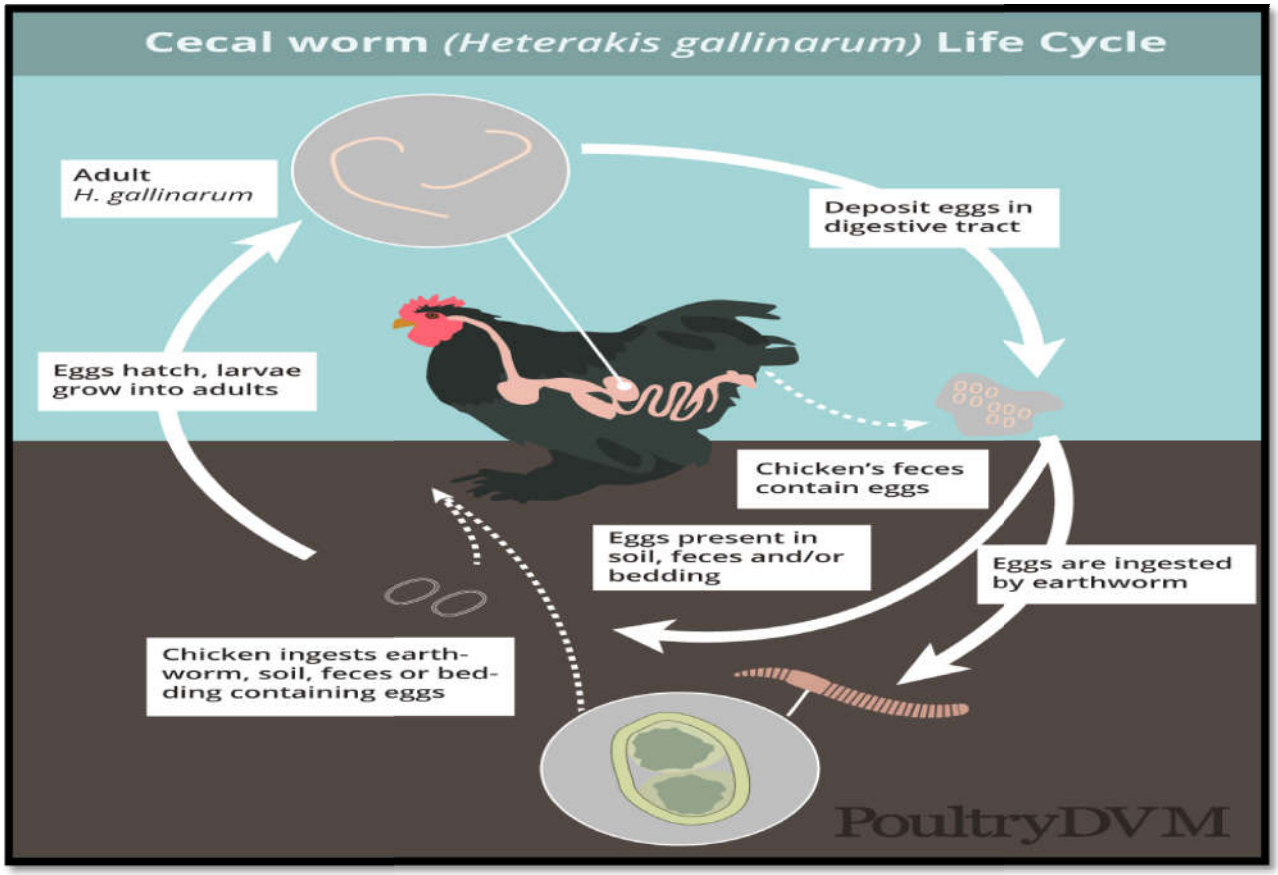


Figure (6): Life Cycle of *Heterakis gallinarum*

Pathogenicity and Pathogenesis

The heavy infection may there be thickening of the caecal mucosa

1- nodular typhlitis.

2-number of petechial haemorrhages on the surface of intestine

3-pnemonia

Clinical Signs

1-diarrhoea

2-wasting

3- markedly emaciated

Diagnosis and Treatment

- clinical signs

-faecal exam.

-serological exam.

And treatment with: Rx, salt of piprazine (dihydrochloride and citrate) and Albendazoal

FAMILY: SUBULURIDEA

Subulura brampti

General Characteristics

-Occurs in the caeca.

-The host :fowl, guinea fowl ,turkey and wild related birds in Africa ,north and south America and Asia .

-The males are 6.9-10mm long and the females 9-17.5mm.

-It having the small buccal capsule has three teeth at its base .

-The male tail is provided with large lateral alae and is curved ventral.

-the per-cloacal sucker is an elongate slit, surrounded by radiating muscle fibers.

-there are lateral alae .

-the oesophagus has a small swelling posteriorly ,followed by a deep constriction and then a spherical bulb.

-There are ten pairs of small caudal papillae .

-The spicules are equal 1.3-1.5mm long.

-the vulva is situated just anterior to the middle of the body.

-The eggs have thick ,smooth shell ,they measure 52-64by 41-49Mm and are fully developed embryo when laid.

Life cycle

The intermediate hosts are various beetle of the *Blaps* and the cockroach *Blatella germanica.*

FAMILY: OXYURIDEA

General Characteristics

- mostly medium –sized or small nematodes

-Mouth surrounded by three inconspicuous lips.

-no buccal capsule.

-oesophagus has a well-developed bulb.

-intestine may have caeca .

- tail of female tapering, the valva is situated near the anterior end of the body

-the female are usually much larger than the males and have long

- two spicules in the male and has bears a number of large papillae around the cloacal opening.

-The eggs are usually flattened on one side and development takes place without the need for an intermediate host.

Oxyuris equi

General Characteristics

-Occurs in the large intestine .

-The host :equines in all parts of the world.

-The males is 9-12mm long and the females up to150mm.

-the oesophagus is narrow at the middle and the bulb is nt distinctly marked off.

-The male has one pin-shape specula which is 120-150Mm long and the tail bears two pairs of papillae.

-The young female are almost white in colour.

-the mature female have a slatey-grey or brownish colour and narrow tail which may be more than three times as long as the rest of the body.

-The eggs are elongate, slightly flattened on one side ,provided with a plug at one pole ,and measure about 90 by 42 Mm.

Life Cycle

-The males and young females inhabit the caecum and large colon .

-After fertilization the mature females wander down to the rectum and crawl out through the anal opining .

-Eggs are laid in clusters on the skin in the perineal region .

-development of the egg is rapid ,reaching the infective stage in three to five days.

- the infective stage may be reached on the perineal region.(the egg falls off to the ground)

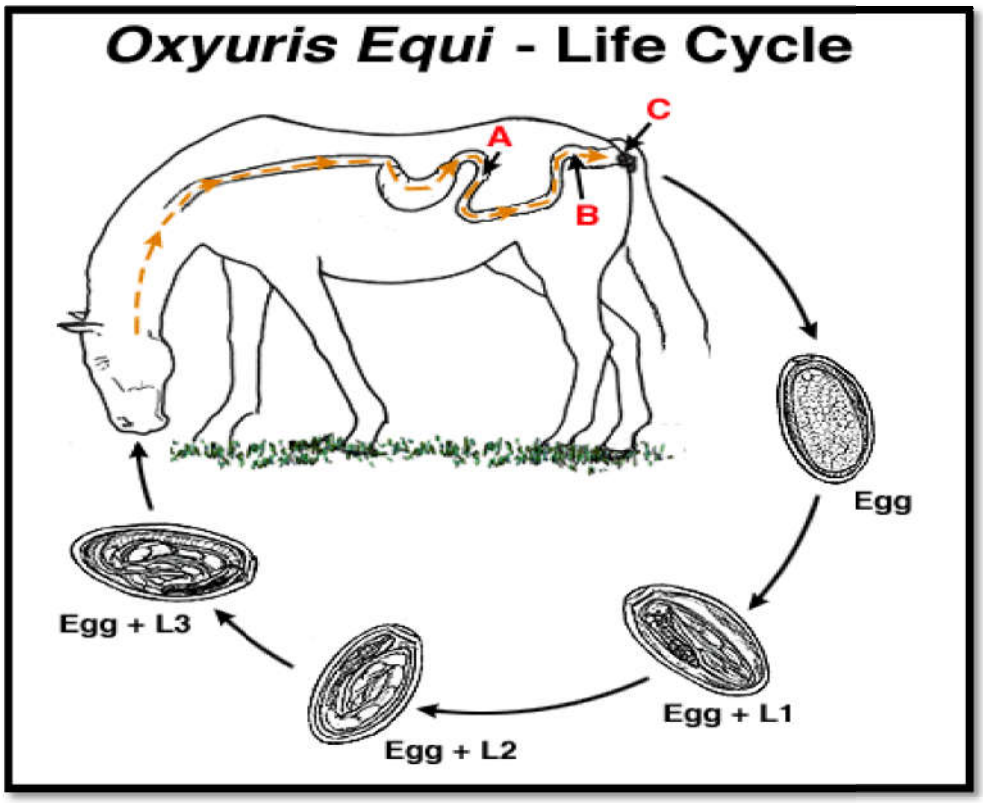
-eggs probably survive for several weeks in moist .

-Infection by ingestion of the infective stage eggs on fodder and bedding.

-infective larvae are laberated in the small intestine and L3 are found in the mucosal crypts of the ventral colon and caecum.

-L4 produce about 8 -10 days after infection .

-p.p is 5 months.



Figure(7): Life Cycle of *Oxyris equi*

Pathogenicity and Pathogenesis

1- the fourth –stage larvae feeds on the intestinal mucosa of the host.

2-the chief feature of oxyuriasis in equines is the anal pruritus produced by the egg-laying females.

Clinical Signs

1-The irritation caused by the anal pruritus produces restlessness and improper feeding

2-lose of condition

3-dull coat.

4-The animal rubs the base of its tail against any suitable object, the tail to acquire an un groomed ,rat tailed, appearance.

Diagnosis

- clinical signs

-faecal exam.

-rectum exam.

Treatment

Rx, mebendazole, Albendazoal and fenbendazol.

Control

-Good hygiene

-Bedding should be removed frequently.

-A clean supply of water should be available.-

Strongylus sp.

General Characteristics

1- There is a globoid buccal capsule and dorsal gutter.

2-there is leaf crown or corona radiate(external and internal round the mouth opening.

3-teeth may be present in the depth of the buccal capsule .

4-The male bursa is strongly developed and has typical rays.

5-the life cycle is direct .

Strongylus

There are three important genus

1-*S. equinus*

General Characteristics

-Occurs in the caecum and colon

-the host ;equines including the zebra.

-the fresh worms are dark grey in color and sometimes the red color of the blood in the gut can be seen .

- The male is 26-35mm long and the female 37-47mm.

-the head without neck .

-the buccal capsule is oval with leaf crown .

-at the base of buccal capsule there is a large dorsal tooth with a bifid tip and two smaller sub ventral teeth.

- the dorsal gutter is found.

-the male has two simple ,slender spicules .

-the vulva in the posterior extremity.

-the eggs are oval thin-shelled ,segmented when laid, and measure 70-85 by 40-47

2-*S. edentatus*

General Characteristics

-Occurs in the large intestine of equines as final host.

- The male is 23-28mm long and the female 33-44mm.

-the head is somewhat wider than the following portion of the body.

-the buccal capsule is oval with leaf crown .

-at the base of buccal capsule there is no teeth

3-*S. valgaris*

General Characteristics

-Occurs in the large intestine of equines as final host.

- The male is 14-16mm long and the female 20-24mm.

-this worms is smaller than above species .

-the buccal capsule is oval and contains two ear-shaped dorsal teeth at the base
- with leaf crown .

Life cycle

1-*S. equinus*

-Exsheathed infective larvae penetrate the mucosa of the caecum and colon

-enter the subserosa where they cause the formation of nodules .

-eleven day after infection L4 occur in the peritoneal cavity and then to liver in 4-8 weeks

-Between 2-4month after infection larvae leave the liver via the hepatic ligaments and pass via the pancreas to the peritoneal cavity .

-The moult L5 occurs about 118 days after infection and back to the caecum and colon.

-The prepatent period is about 260 days.

2-S.edentatus

-Exsheathed infective larvae penetrate the mucosa of the intestine to liver via the portal system.

-11-12 day after infection L4 are produced.

-in nine weeks after infection larvae leave the liver and pass between the hepatic ligaments and the peritoneal layers.

-The moult L4 and L5 are found in

-the larvae migrate between the layer of the mesocolon to the wall of the caecum and colon after than to the lumen.

-P.P is 300-320 days.

3-*S.valgaris*

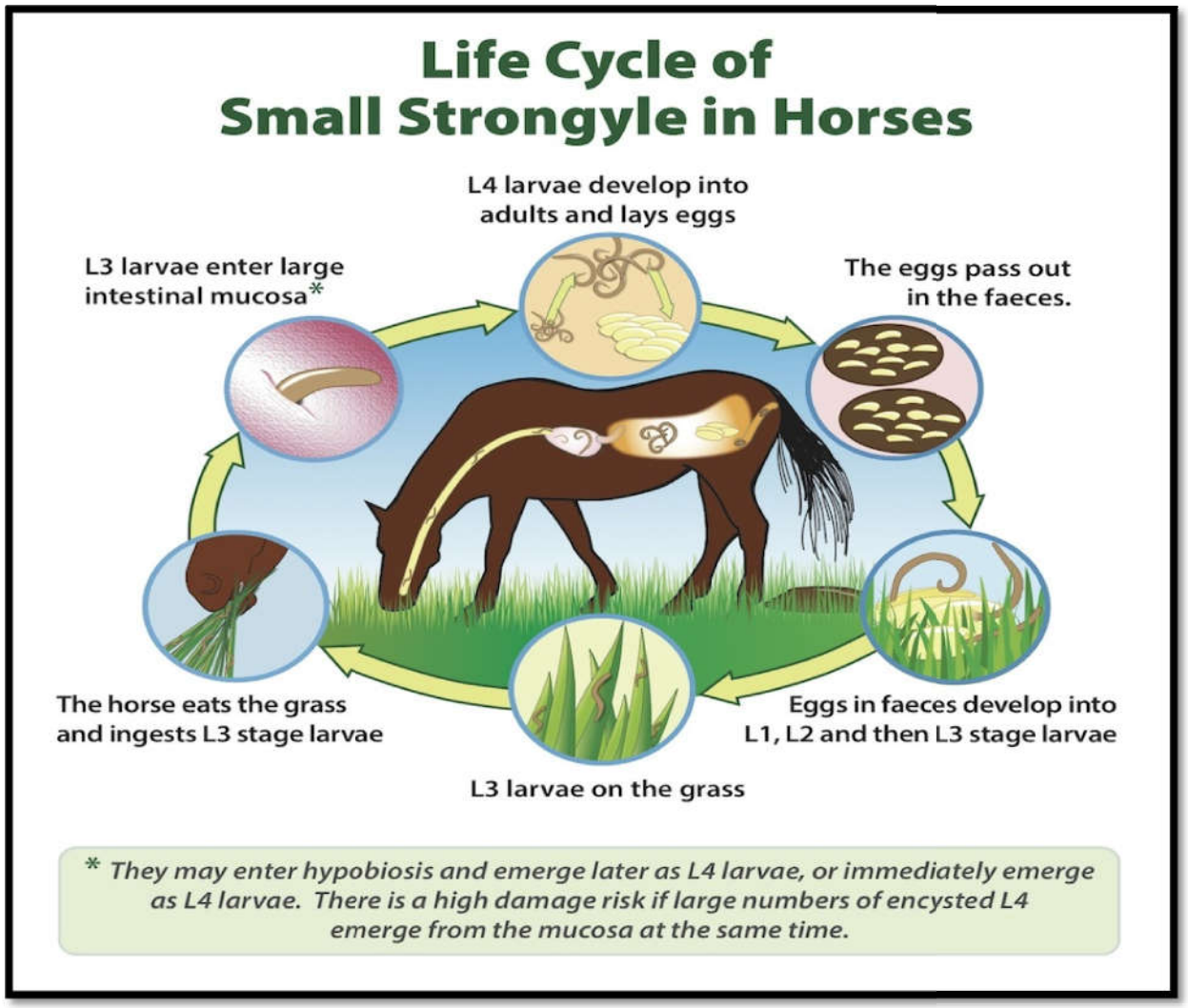
-Exsheathed infective larvae penetrate the mucosa of the intestine wall in 8 days after infection .

-8 day after infection L4 are produced and penetrate the intima of the submucosal arterioles and migrate in these vessels towards the cranial mesentricartery .

-They are to be found in the cranial mesentricartery from 14 days after infection onwards associated with thrombi and later aneurysms.

-The L4 are found in submucosa of the caecum and colon ,after then its moult to L5in 3 months and in lumen may be maturity

-The prepatent period is about 6-7months.



Figure(8): Life Cycle of *Strongyle equinus*

Pathogenicity and Pathogenesis

1- The adult worms from all species attach themselves to the mucosa of the large intestine and suck blood.

2-anaemia of the normochromic normocytic type.

3-small haemorrhagic ulcer in the large intestine.

4-larvae migrant caused primary lesions occurred in the wall of small and large intestine and .

5- Extansive irregular inflammatory lesion occur in the media of the affected arteries producing an endarteritis and the formation of thrombi ,the larval stages may be found embedded in the thrombus, this case called aneurysms or verminous arteritis. specially of (*S. valgaris)*.

6- haemorrhagic nodules

Clinical Signs

1-*S.equinus*

1-colic

2-anorexia

3-general malaise

2-*S.edentatus*

1-peritonitis

2-acute toxaemia

3-jaundice

4-fever

3-*S.valgaris*

1-colic

2- anaemia

3-acut verminous arteritis

4-diarrhoea

5-occasionally death

Diagnosis

- clinical signs

-faecal culture .

-serological exam.

Treatment

Rx, phenothiazine, piprazine salts, Albendazoal and thiabendazol.

Control

- routine use of Anthelmintic.

-pasture management

-kill the eggs and larvae by the heat of fermentation result from proper disposal of manure

Chabretia ovina

General Characteristics

-occur in the colon.

-the host; sheep ,goat ,cattle and number of the ruminants throughout the world.

-the males are 13-14mm long and the females 17-20mm long.

The anterior end is curved slightly ventral and large buccal capsule opens anteroventrally.

-there are leaf-crown and a shallow ventral cervical groove.

-the male bursa is well developed and the spicules are 1.3-1.7mm long.

-the eggs measure 90-105 by 50-55Mm

Life Cycle

-Infection occurs per os by larvae relatively long tail.

-L3 undergo an extensive histotropic phase in the wall of the small intestine prior to the third ecdyses 7-8 days after infection.

-up to 26 days reach the colon.

-L4 develop mainly in the lumen of the caecum.

-the fourth ecdyses occurs on average about 24 days after infection.

-P.P is 49 days.

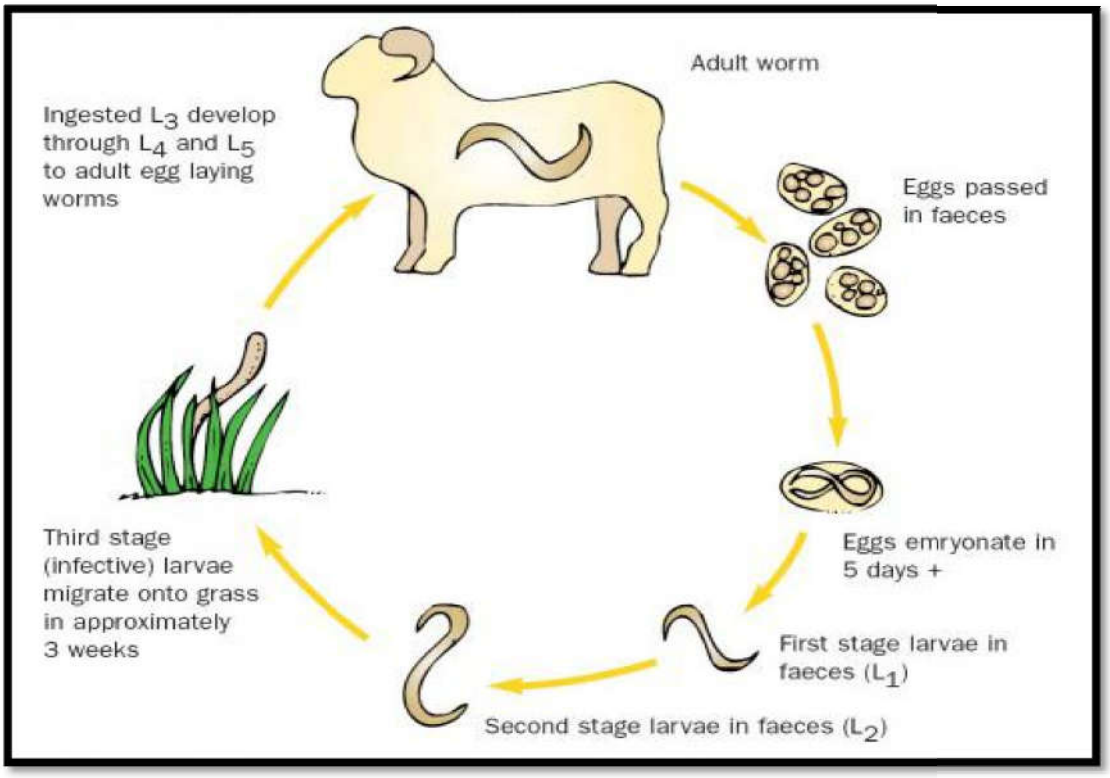


Figure (9): Life Cycle of *Chabretia ovina*

Pathogenicity and Pathogenesis

1-The granular layer which is digested by the secretion of the oesophageal glands of the worm.

2-worms suck blood.

3-an increased activity of goblet cells and infiltration with lymphocytes and eosinophils.

Clinical Signs

1-diarrhoea with much blood and mucus.

2-lose condition and become anaemic and die.

Diagnosis

- clinical signs

-faecal culture .

-serological exam.

Treatment

Rx and benzimidazoles.

FAMILY: ANCYLOSTOMATOIDEA

General Characteristics

- The buccal capsule bears on its ventral margin 1-4 pairs of teeth

-the dorsal gutter dose not project into the cavity of the buccal cavity to form a dorsal cone.

Ancylostoma caninum

General Characteristics

-occure in the small intestine

-the host; dog, fox , wolf and other wild carnivores .rarely in man.

-its cosmopolitan in distribution, being common in tropical and sub tropical zones in north Amerce ,Australia and Asia

-The male is 10-12mm long and the female 14-16mm long.

-The worms are fairly rigid and gray or reddish in colour, depending on the presence of the blood in the alimentary canal.

-The anterior end is bent dorsal and the oral aperture is the directed anterodorsally .

-The buccal capsule is deep

-There are bears three teeth on either side of the buccal capsule (in the depth of the capsule there is a pair of triangular dorsal teeth a pair of centrolateral teeth.

-The male bursa is well developed and the spicules are 0.8-0.95mm long.

- The vulva is situated near the junction of the second and last thirds the body .

The eggs measure 56-75 by 34-47Mm and contain about eight cells when passed in the faeces.

Life Cycle

Adult female produce 16000 eggs per day.

The pre-infection stages are none resistant to desiccation , so that they are found only in moist surrounding.

-The optimal temperature for development is between 23 and 30ºc.

A variety of developmental pathways may occur following infection.

1-oral infection

May be the larvae are placed in the mouth a preparation will penetrate the buccal and pharyngeal epithelia and undergo migration as skin penetration .

2- skin penetration

Leads to migration to the lungs and by tracheal migration to the intestine (somatic migration)

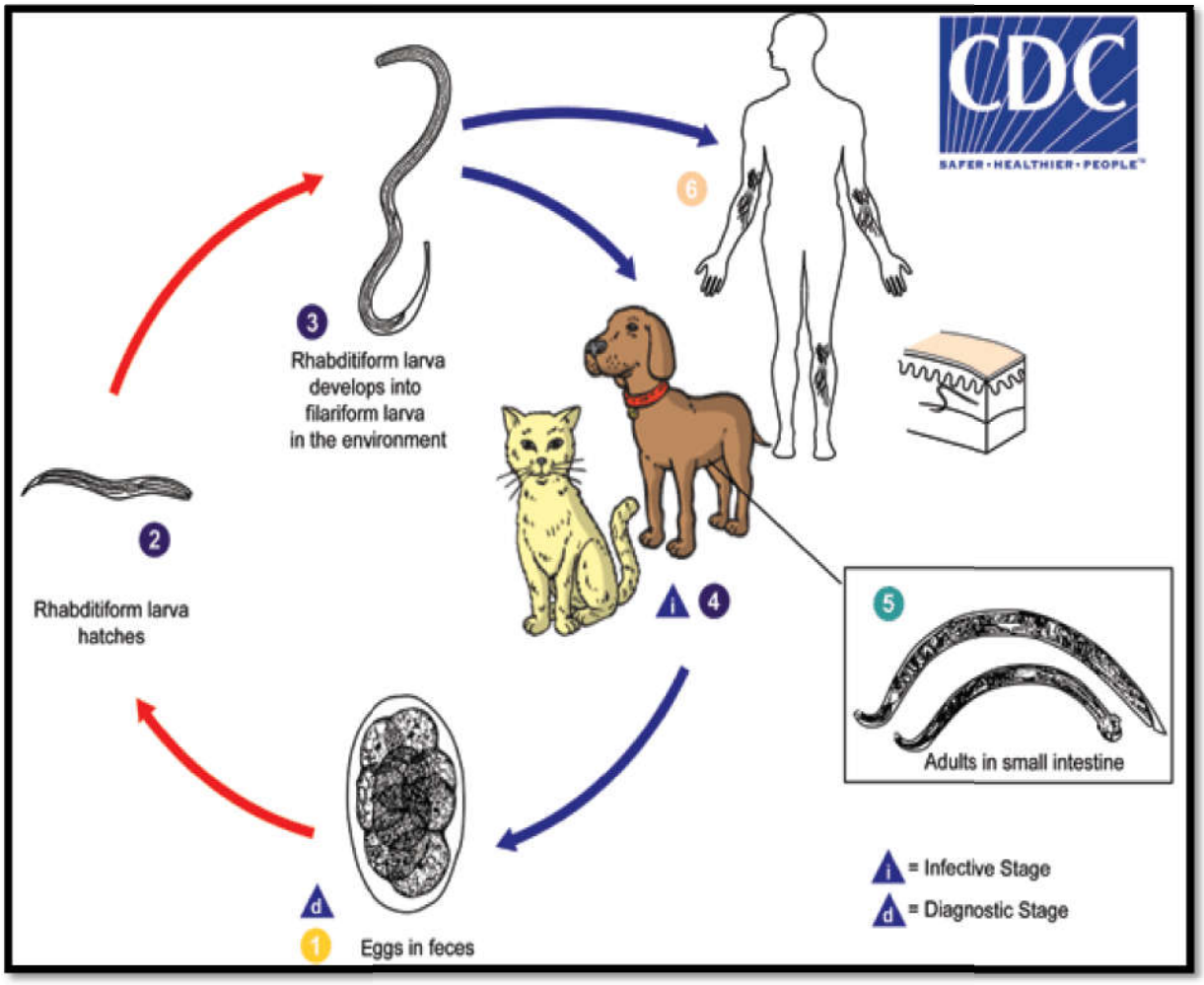
3-prenatal infection

4-colostral or lactogenic infection

- The larvae after enter the host reach blood vessels or lymphatic vessel and are carried by these via the venous system or thoracic duct to the heart and lungs

- larvae pass into the alveoli and migrate up the bronchioles ,bronchi and trachea and are then swallowed and mature in the small intestine .

-P.P is 17 days.



Figure(10): Life Cycle of *Ancylostoma caninum*

Pathogenicity and Pathogenesis

1-Anaemia

2-worms suck blood due to poor source of iron in blood and milk .

3-an increased activity of goblet cells and infiltration with lymphocytes and eosinophils.

4-The animals are more able to cope with the blood lose, which could be at a rate of as much as one –quarter of the total circulating erythrocyte volume.

Clinical Signs

1-diarrhoea with much blood and mucus.

2-the dog lose condition and become anaemic and die.

3-oedema ,general weakness and emaciation

4- the coat becomes harsh and dry .

5- The chief clinical signs is anaemia.

Diagnosis

- clinical signs

-faecal culture .

-serological exam.

Treatment

Rx, Tetrachloroethylene, Bephenium compounds and Mebendazole.

Bunostomum spp

1-*B.trigonocephlum*

General Characteristics

-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 a pair of chitinus plates, near its base is a pair of small sub ventral lancets

-It has the dorsal gutter .

-the bursa is well developed and has an asymmetrical dorsal lobe.

-The spicules are slender , and 0.6-0.64mm long.

-The eggs measure 79-97by47-50Mm , the ends are bluntly rounded and the embryonic cells are darkly granulated.

Life Cycle

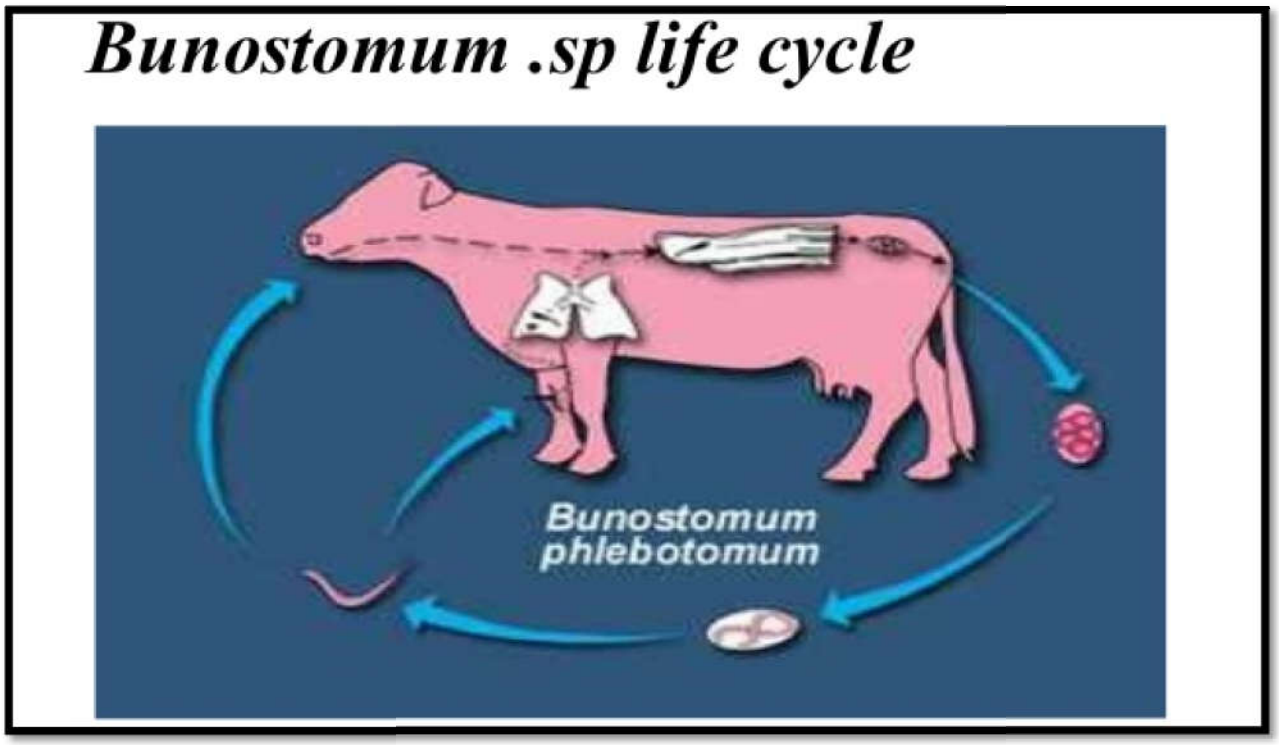
-Its direct

-Infection of the host occurs through the mouth or skin

-Followed skin penetration the larvae pass to the lungs where third ecdysis occurs.

-The fourth –stage larvae (which have buccal capsule), reach the intestine again after 11 days .

- the first eggs are passed 30-56 days after infection (P.P)



Figure(11): Life Cycle of *Bunostomum sp.*

Pathogenicity and Pathogenesis

1-Anaemia

2-worms suck blood .

3-there are changes in the blood picture ,hydraemia and oedema , which shows especially in the intermandibular region as a bottle-jaw

Clinical Signs

1-diarrhoea , the faeces may be dark in colour due to the presence of altered blood pigments .

2-the dog lose condition and become anaemic.

3-oedema ,general weakness and emaciation

4- death is frequently preceded by complete prostration.

5- The chief clinical signs is anemia.

Diagnosis

- clinical signs

-faecal culture .

-serological exam.

Treatment

Rx, Tetrachloroethylene, Bephenium compounds and Mebendazole.

Cutaneous Larva Migrans

This condition may be compared with V.L.M. It occurs in man 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.duoddenale*

-*B.phlebtomum*

-*Strongyloides spp*

-*Gnthstoma spp*

The severity of skin reaction is related to the degree of exposure to infection larvae .It lead to ;

1-papule formation

2-oedema

3-markedlly pruritic lesion

The larvae may be reach to the lungs and they have also been reported in opacities of the cornea.

Haemonchus contortus

Haemonchus represent the most economically important helminthes parasites in cattle, sheep, and goats occurs in nearly all subtropical and temperate areas of the world. Adult worms are attached to abomasal mucosa and feed on blood, which causes an anemia and eventually can lead to death, making *H. contortus* one of the most pathogenic nematodes of ruminants while another reason that makes *H. contortus* dangerous is its ability to rapidly develop resistance against antihelmintics and highly prolific, each capable of producing as many as 10,000 eggs per gram daily. The prevalence of *H. contortus* in sheep has been reported very high (25.1 to 92%) by many workers in Pakistan. In Middle Europe *H. contortus* is present in 50-75% of small ruminants . *Haemonchus contortus* is widely distributed in Iraq and can infected Awasse sheep . Although *H. contortus* can infect sheep, goats, cattle, and other ruminants, but its preferred host is sheep.

Description and Measurements

The male being shorter than the female and has an even reddish color, while the female, has white ovaries wind ,which turn to be the red intestine, giving the characteristic barber's pole appearance. In both sexes the anterior end had similar cuticular surface including cuticular transversal striae and cuticular ridges or synlophe. On the ventral side of the anterior end, the excretory pore is present. The

cervical papillae were spine-like and situated on the lateral sides of the anterior end. A small buccal cavity was present and carried a bent dorsal lancet.

In the male had elongated lobes supported by long ray, while the dorsal lobe was asymmetrically situated on the left hand side and supported by a Y–shaped dorsal ray and the surface of the dorsal lobe was covered by bursal bosses. Other important diagnostic feature was the barbed spicules. On the ventral aspect of the genital cone, the "O" or ventral papilla was observed. Vulva was located at the beginning of the posterior third of the female body and covered by an anterior flap which was frequently large and prominent, the surface of the vulvar flap were covered by cuticle with transversal striae .The anal pore was situated on the ventral side of the posterior end of the body.

Male: Body length 10-20(15) mm, maximum width 0.20-0.30(0.25) mm. Cervical papillae 208-402(305)µm length from anterior end. Esophagus length 1.4-1.63(1.5)mm. The spicules of the male measured 470-510(490)µm in the length. The distal tip of the spicules each had a single barb at unequal distance, the barb on the left spicule was (20.2-30)µm distance from the tip and the right spicules (40-40.2)µm.

Female: Body length 19-30(24.5) mm, maximum width 0.5-0.6(0.55) mm. The distance between cervical papillae from the anterior end is 406-560 (483) µm. Esophagus length 1.65-1.70 (1.67)mm. Tail 400.2-504(452.1)µm.

Life Cycle

In all genera the life cycle is direct(fig.2) without requiring an intermediate host. The eggs are passed in the feces and under suitable environmental conditions hatch, producing two successive non-parasitic larval stages and the 3rd infective larvae. Ingesting the 3rd larvae while grazing leads to infection in ruminants.The L3 of the trichostrongyle group penetrate the mucus membrane (in the case of *Haemonchus* and *Trichostrongylus*) or enter the gastric glands (in the case of *Ostertagia*). During the next few days the L3 moult to the 4th stage (L4) and remain in the mucous membrane or in the gastric gland, for about 10-14 days. Then they emerge and moult into the young adult stage L5. Most trichostrongylea mature and start egg production at about 3 weeks after infection.

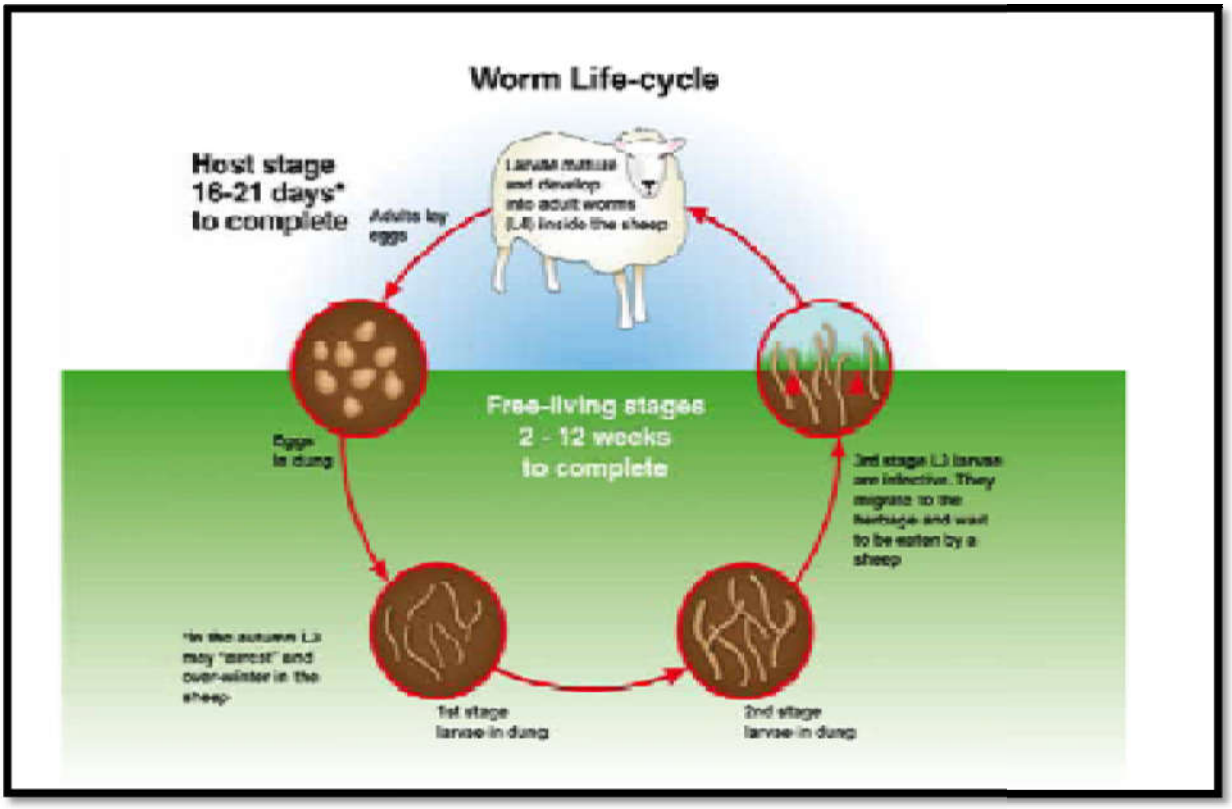


Figure (12): Life Cycle of *Haemonchus contortus*

Pathogenesis of *Haemonchus contortus*

The main pathogenic mechanisms of *H. contortus* are a direct lesion on the gastric mucosa and hematophagy. The effects of pathogenic mechanisms during intra-host parasite development and the subsequent response of infected ruminants provoke morpho-functional changes, particularly in the abomasum. Also, variations appear in some blood parameters, resulting in the appearance of both anemic and impaired digestion-absorption syndromes, adult parasites can ingest 0.05ml of blood/helminth/day causing notable blood loss with a reduction of packed cell volume (PCV). This parameter has, in fact, been used as a marker of parasite virulence and indirect estimation of parasite burden in Haemonchosis .

Many factors prevent such as protein losses from being replaced through feeding. Infected animals have lower food, due to anemiagastrin reduces food passage through the gastrointestinal tract bad digestion syndrome, caused by the increase of abomasal pH value, which prevents pepsin synthesis reduced amino acid and small peptides absorption. Increase in PH in the abomasum comes from a decrease in the production and excretion of hydrochloric acid (HCl) by the parietal cells of the gastric mucosa, generated by their loss and/or a decrease in their number. Lowered numbers of parietal cells is due to tissue lesion, cellular infiltration is also caused by the presence of the parasitic stages or their secretion-excretion products released into the medium and cellular replacement by immature nonfunctional cells.

It has been observed that the parasites release substances such as ammonium , which increases the pH around the parasites so as to avoid the action of gastric acids or pepsin on its cuticle, so there is an inhibition in the transformation of pepsinogen, produced in the principal cells of the gastric glands, to pepsin.

These pathogenic actions directly affect the health of the animals, they also indirectly affect ovine yield, although this loss may be unnoticed if the parasitism presents a sub clinical course. Thus, the nematode infection of the abomasum has a notable effect on live weight. This has also been observed in weight and carcass conversion, and this indirect effect is mainly due to a decrease in nutrient use. resulting from many factors, such as a lack of appetite and a decrease in the voluntary feed intake observed in the parasitized animal, irreversible loss of proteins in the gastric lumen by hematophagy, hemorrhagic gastritis and loss of plasma proteins through greater mucosa permeability and lowered digestibility .

The symptoms of haemonchosis include lack of appetite , lethargy, loss of weight, reduction in milk and wool production, presence of pale mucosa, edemas, diminution of PCV, hemoglobin, plasma proteins and increase in the number of circulating eosinophils in peripheral blood and serum pepsinogen and gastrin. The final stages of the disease may be accompanied by emaciation, and death may result. In the necropsy, the macroscopic lesions observed are: emaciation, pale mucosa, edemas in body cavities, degradation of the fat deposits , hypertrophy of local lymph nodes, edema of the abomasal mucosa with petechial hemorrhages, presence of nodules and the observation of adult parasites. In the microscopic evaluation, cellular infiltration, dilatation of the gastric glands, ulcers, edema, hemorrhage and an increase in the number of mastocytes and eosinophils were observed.

Diagnosis

Haemonchus contortus is probably the only nematode parasite of sheep and goats that can be accurately diagnosed without the aid of laboratory testing. Signs

of acute anemia are obvious. Past history and discounting other less common conditions causing anemia, will strongly suggest clinical Haemonchosis.

Laboratory testing

Faecal examination

Trichostrongyle infections can be diagnosed by examination of the feces for parasite eggs. Eggs can be found in the feces about 2 to 3 wk post-infection, Faecal egg counts and larval cultures, the tests most commonly employed for the in vivo diagnosis of gastrointestinal and pulmonary nematode infections in ruminants are faecal egg counts (FECs), preferably with speciation by way of larval culture and differentiation.

Apost-mortem examination and worm count

If gastrointestinal parasitism is suspected as the cause of an outbreak of disease in a flock, apost-mortem examination and worm count should be performed, preferably on two or three animals. It is not sufficient to attempt to visualise the number of worms in the abomasum or small intestine because, with the exception of *H contortus*, the worms are difficult to see and counts are impossible.

Serological tests

The usefulness of FEC as a measure of parasitism is further limited by our inability to reliably distinguish between the eggs of different species. Other methods like measurement of parasite-specific antibodies can be used as

supplementary diagnostic tools. Efforts to resolve this through detection of parasite antigen in faces, either directly through ELISA, or by amplifying ribosomal DNA using PCR have been partially successful, but are not yet in widespread use Differentiation of trichostrongylid eggs for all but a few easily recognizable species relies on fecal culture. The ratios of larvae so recovered may bear little relationship to the species composition of the worm population of origin.

Enzyme-linked immunosorbent assay (ELISA)

Dot-ELISA:

Polymerase Chain Reaction (PCR)

Prevention and Control Measures

Treatment involves use of anthelminthic drugs such as benzimidazoles, levamisole and ivermectin, among others. However, these drugs are becoming ineffective against many trichostrongylid parasites that affect sheep in several parts of the world. indicates that the disease can be controlled to an extent by providing better nutrition (especially protein supplementation to lambs), strategic drenching at times of heightened risk of infection, pasture management and perhaps biological control using fungi. Vaccination for improved immunity to *H. contortus* also appears to be promising but is not yet practical.

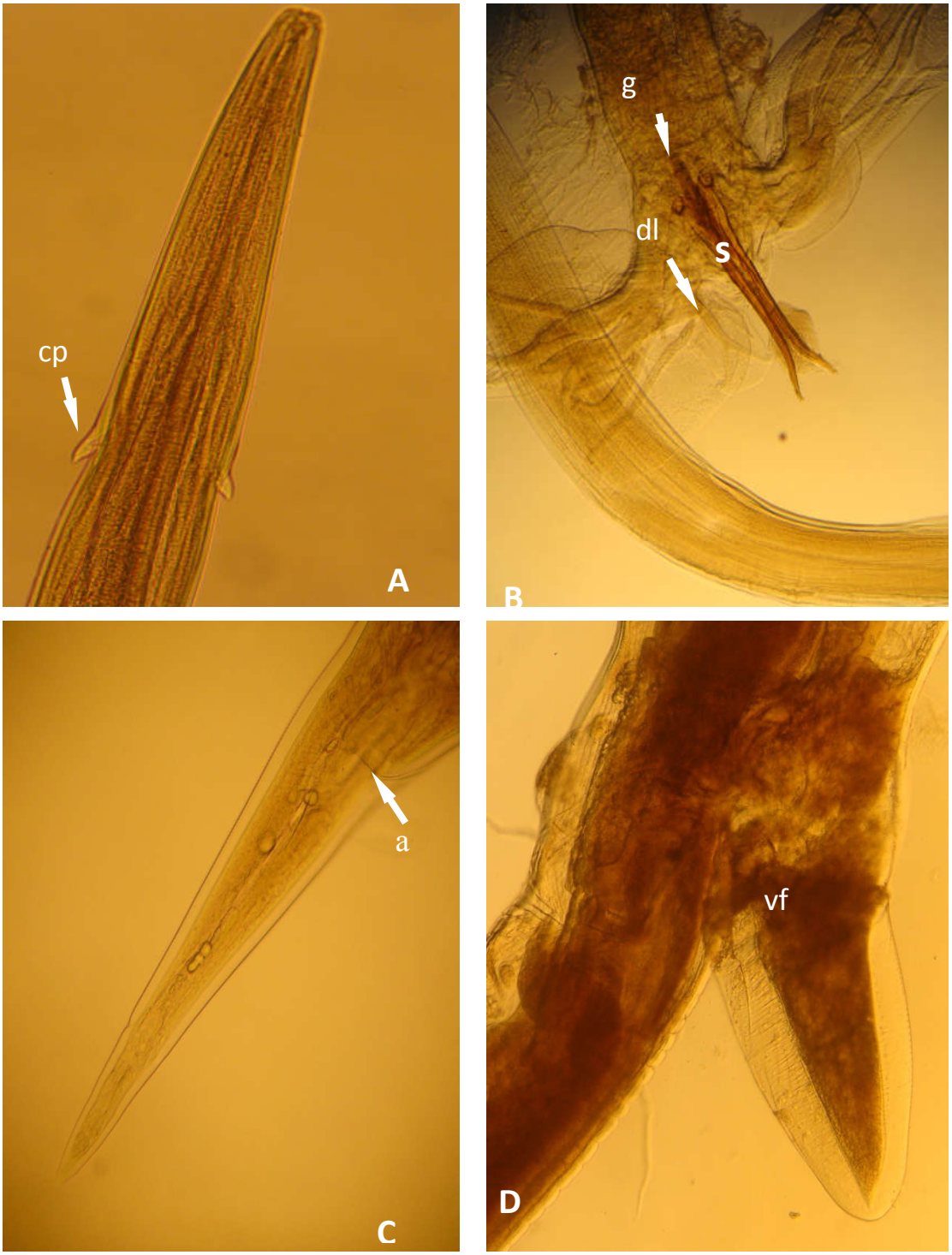


Figure (13): *Haemonchus contortus*, A- anterior extremity (cp : cervical papilla), B- posterior extremity of male (bursa with s: spicules ; dl: dorsal lobe; b:barb; t:tib & g :gubernaculums) C- posterior extremity of female(a:anus) D- vf: Vulva flap.((Linguiform)

Gastrointestinal nematodes (GINs):

Gastrointestinal nematodes of the order Strongylida are the most common cause of clinical Helminthosis, which is caused by the infection of digestive tract due to the presence and development of nematodes in the wall or the lumen of the abomasums, the small intestine, and /or large intestine.

The gastrointestinal parasites are very common in sheep due to their grazing and watering habits. stated that the *H. contortus*, *Telodorsagia circumcincta, Trichostrongylus colubriformis,* and *Trichostrongylus axei. Cooperia* spp. They are also called the trichostrongyles of sheep. Prevalence of helminthes has been reported ranging from 0.72 to 84.1 % in domestic animals from various parts of the world. The trichostrongyloid nematodes are small, often hair like worms in the bursate group, which are especially common and pathogenic in grazing ruminants. The abomasums and small intestine are the usual locations in ruminants. Structurally they have few cuticular appendages and the buccal capsule is vestigial. The males have a well-developed bursa and two spicules, the configuration of which is used for species identification. referred that the morphmetrics of spicules, vulva flap and cervical papillae are reliable tools for species identification on individual worms. In all genera the life cycle is direct without requiring an intermediate host. The eggs are passed in the feces and under suitable environmental conditions hatch, producing two successive non-parasitic larval stages and the 3rd infective larvae. Ingesting the 3rd larvae while grazing leads to infection in ruminants. The L3 of the trichostrongyle group penetrate the mucus membrane (in the case of *Haemonchus* and *Trichostrongylus*) or enter the gastric glands (in the case of *Ostertagia*). During the next few days the L3 moult to the 4th stage (L4) and remain in the mucous membrane or in the gastric gland, for about

10-14 days. Then they emerge and moult into the young adult stage L5. Most trichostrongylea mature and start egg production at about 3 weeks after infection . Infections with GINs can cause trichostrongylidosis in small ruminants .The transmission, incidence and intensity of infections are determined by several environmental, host and parasite dependent influences and phenomena; the most dominant of which are, meteorological factors, methods of husbandry and systems of livestock production; host age, nutrition and acquired immunity; larval hypobiosis and concurrent infections. The infections occur mostly as mixed infections of different GIN species. Emaciation, persistent diarrhea and weight loss are usually the main symptoms. Villous atrophy results in impaired digestion and malabsorption of nutrients (Perry and Randolph, 1999; Al-Bayati and Arsalan.,2009).This leads to decreased live-weight gain, fiber and milk production and reproductive performance of small ruminants and therefore has a seriously impact on animal health and productivity. Hence, GIN parasitism represents the greatest economic constraint of small ruminant production.

Ostertagia spp.

A genus of worms in the family Trichostrongylidae. They are found in the abomasum, rarely the intestine, of ruminants, are thin , brown and called brown stomach worms. This genus is the major cause of parasitic gastritis in ruminants in all over the world. The three major species were *Ostertagia ostertagi, O.circumcincta,O.trifurcata*.Other, less common species *Teladorsagia circumcincta* and *T. trifurcata* are parasites of the stomach and are reported to be present in >75% of small ruminants in Germany.

Ostertagia circumcincta

Description and Measurements

The worm is small, slender, and brown in colour. In both sexes the anterior end had similar cuticular surfaces inflated, transversal striated, the body has cuticular ridges or synlophe and no transversal striae. Small cervical papillae set more posterior.

Male: Body length 8-8.5(8.25)mm, maximum width 0.15- 0.22(0.185) mm. Cervical papillae 380-402(392.5)µm length from anterior end. Esophagus length 0.76-0.85 mm , the spicules of the male measured 310-320(315) µm in the length. The male bursa has later and dorsal lobes and an accessory bursal membrane situated interiorly on the dorsal side.

Female: Body length 10.4-12(11.2)mm, maximum width 0.18-0.20(0. 19)mm. Cervical papillae 370- 420(392)µm length from anterior end. Esophagus 1.65-1.70(1.67)mm. Tail 280- 305 (292.1)µm.

Figure (14): *Ostertagia circumcincta* A-Anterior extremity (cp:cervical papilla) B-Posterior extremity of male, bursa (dl: dorsal lobe ;rs: right spicule ; ls: lift spicule) C- vf :Vulva flap) D-- posterior extremity of female (a:anus)

Trichostrongylus spp.

The hairworm, or bankrupt or black scour worm which an economically important. That inhabit the small intestine, in some cases the infected stomach, of a variety of herbivorous animals and gallinaceous birds. They burrow into the mucosa and suck blood; in large numbers they do serious damage, especially to young hosts. In Middle Europe several species of the *Trichostrongylus* genera occur and of these *T. colubriformis* and *T. vitrinus* are the most important ones in small ruminants with a prevalence of 50-75%.

The adults of both species live in the stomach of ruminants. *T. axei* is present in about 25-50% of the small ruminants and parasitizes in the small intestine. *T. axei* is the smallest member of the genus and parasitizes the simple stomach of ruminants or abomasums of a wide range of hosts including ruminants and horses where as the other species of trichostrongyles are parasites of the small intestine of ruminants and display a higher order of host specificity, and species of *Trichostrongylus* can be found throughout regions that are subtropical, tropical, and temperate. The adults are small and hair like, slender, pale reddish-brown worms without a specially developed head end and no buccal capsule. A most useful generic character is the excretory pore is usually situated in a distinct conspicuous ventral notch in the esophageal region. In males the bursa has long lateral lobes; the ventroventral ray of the male bursa is separated widely from the others and is conspicuously thinner than the lateroventral, which runs parallel with the lateral rays. The dorsal ray is slender and cleft near its tip in to two branches, which have short digitations. The spicules are usually pointed, short, stout, ridged or twisted and pigmented brown.

Male: Body 4.9-5.5(5.2)mm length, maximum width 0.10-0.22(0.16) mm. Esophagus length 0.64- 0.66(0.65)mm. The spicules of the male measured 0.135-0.152(0.143)µm in the length.

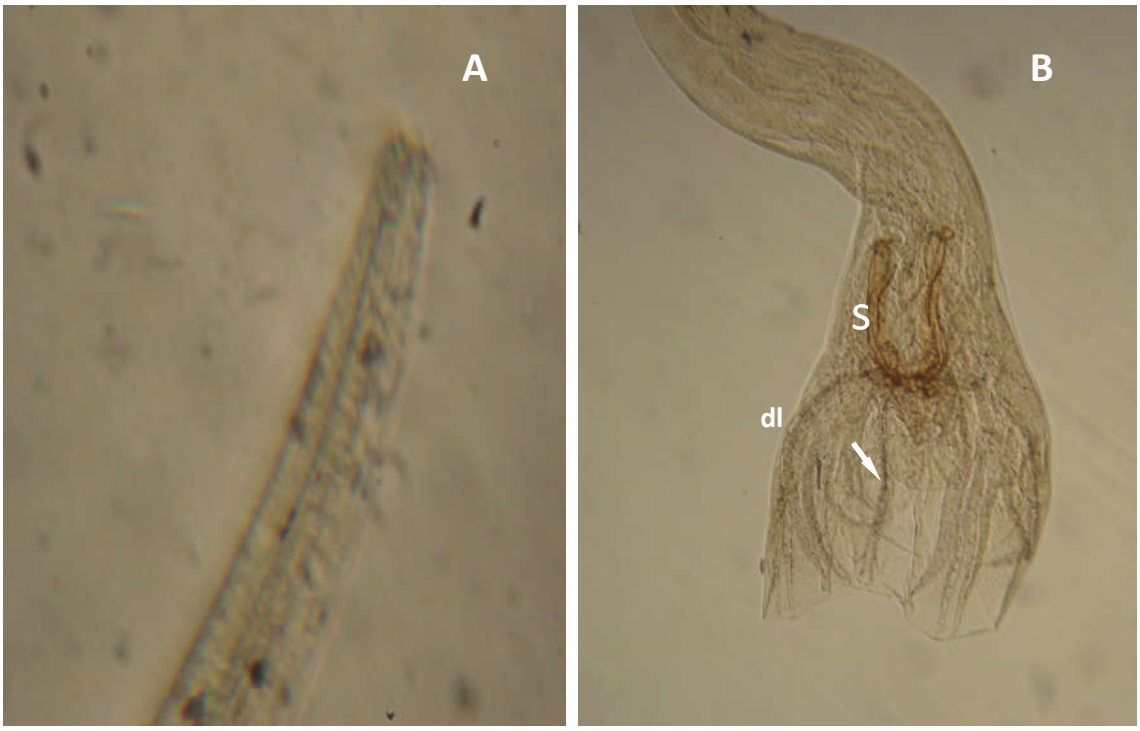


Figure (15): *Trichostrongylus* sp. A-anterior extremity B-posterior extremity of male,(bursa , dl: dorsal lobe ;s: sipcules) .

FAMILY: DICTYOCALIDAE

General Characteristics

-Number of this family occur in respiratory passages of the lung.

-Bursa is well formed and bursal rays well developed .

-Spicules are short and reticulated.

-The life cycle is direct.

-the only genus of importance is *Dictyocaulus*.

Dictyocaulus filaria

General Characteristics

-Occurs in the bronchi of sheep, goat and some wild ruminants.

-It has a worldwide distribution and causes serious losses in countries in eastern Europe and India.

-The male is 3-8 cm and the female 5-10cm long.

-The worms have the milk-white colour and the intestine shows as a dark line.

-There are very small lips and a very small, shallow buccal capsule .

-In the male bursa the medio –and posterolateral rays are fused together except at the tips.

-The spicules are stout , dark –brown, boot –shaped and 0.4-o.64mm long

-The vulva situated not far behind the middle of the body.

-The eggs measure 112-138 by 69-90µm and contain fully formed larvae when laid.

.

Life Cycle

- The eggs may hatch in the lungs to L1st which are coughed up and swallowed ,its passed through the alimentary tract of the host.

- some eggs may be expelled in the nasal discharge or sputum.

-L1st passed in the faeces is 0.55-0.58mm long and can be easily recognized by the presence of the small cuticular knob at the anterior extremity and numerous brownish food granules in the intestine cells .

-the free stage do not feed, but exist on food granules.

-after 1 or 2 days the larvae reaches the second stage , but does not cast the old cuticle so that its enclosed in to sheaths.

1- the first for protection.

2- the second for nutrition .

- after 6-7 days the larvae reaches the infective stage.

-infection of the host occurs per os.

- larvae penetrate into the intestine wall within three days and pass through the lymph vessels to the mesenteric lymph glands.

-the third ecdysis occurs in about four days A.I.

-in the fourth stage male and female can be distinguished .

-the worm reach to the lung through the lymph and blood vessels.

-they are arrested in the capillaries and break through into the air passages .

-development to maturity in the bronchi of the host takes about four weeks .

Pathogenesis

1- Catarrhal parasitic bronchitis.

2- the inflammatory process spreads to the surrounding peribronchial tissues and the exudates frequently back into the bronchioles and alveoli , causing atelectasis and catarrh, or pneumonia

3- secondary bacterial infection may lead to more extensive areas of pneumonia.

Clinical Signs

1- The disease may occur at all ages and is usually chronic.

2- coughing

3-dyspnoea

4-the respiration is more rapid

5- abnormal lungs sounds

Diagnosis

1- faecal exam

2-sputum exam

3-nasal discharge exam

4-serologycal tests

Prevention and Treatment

1- prophylaxis

- the animals must be removed from infected ground ,placed on dry pasture

2- A vaccine consisting of irradiation attenuated infective larvae.

3- treat by

- Levamezole

-tetramezole

-ivermectine

Epidemiology

- The larvae require moisture for development and at a temperature of 27°c .

- Infective larvae may survive over winter to following March ,April and May.

- The faecal fungal spirited the infective larvae with its sporocysts