Lec: 3 **Nematodes** **3rd class**

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Order: Strongylida

Family: Trichostrongyloidea

Morphological characteristic

1- Distinctive **copulatory bursa** on adult males.

2- Well-developed dorsal, ventral and lateral expansions of the surface cuticle at the posterior end, referred to as **lobes**.

3- Lobes supported by muscular **rays**.

3. Exception in the superfamily Metastrongyloidea where copulatory bursa is less well- developed.

4. Buccal area (same for males and females) at anterior end of the four different superfamilies of Strongylida.

Development of eggs and free living larvae

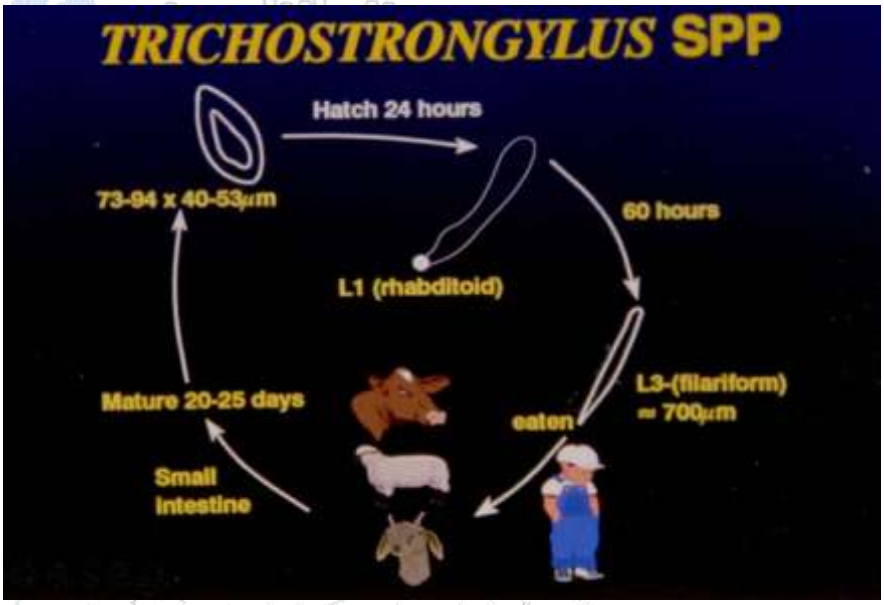
Eggs passed in feces hatch in 24 hours to **rhabditiform L1**. In successive molts, rhabitiform **L1 and L2**, which feed on micro-organisms, develop to **filariform infective L3**. L3 do not feed (they live on reserves) and are protected from adverse environments by a sheath, the retained cuticle of the L2. Some very resistant species, such as *Nematodirus,* also retain the L1 cuticle and egg membrane.

life cycle (in general)

The life cycle of Trichostrongyloedea is direct, and infection is through ingestion of L3.

Egg → L1 → L2 → L3 occurs on the ground. L1 and L2 feed on bacteria, and L3 is exsheathed and represents the infective stage. L3 cannot feed, but do contain a finite amount of stored food to provide energy for movement. Infection is via ingestion of the L3. L3 → L4 → adult, these stages generally occur in the stomach or small intestine.

Parasitic development initially occurs in gastric glands or intestinal crypts, species dependent. Adults are generally found on the mucosal surface, and the prepatent period is typically about 3 weeks.



Routes of infection

1- Ingestion with pasture herbage is the ONLY ROUTE OF INFECTION of importance for the superfamily Trichostrongyloidea genera (except Ollulanus) and for the superfamily Strongyloidea genera (except Syngamus and Stephanurus

*IT IS VERY IMPORTANT TO KNOW THAT INGESTION OF PASTURE HERBAGE IS THE ONLY ROUTE OF INFECTION FOR TRICHOSTRONGYLES AND STRONGYLES

2-Skin penetration or ingestion is used by the free-living infective larvae of genera in the superfamily Ancylostomoidea. Some species use a lactogenic (transmammary) route of infection as well, this is especially important in canine hookworm disease .

3-Ingestion of infected intermediate hosts (snail, slug or earthworm) or paratenic hosts containing infective larvae is used by the genera of Metastrongyloidea .

Trichostrongylus sp.

Trichostrongylus axei in the stomach or abomasum of primarily ruminants and horses; Trichostrongylus colubriformis in the small intestine of ruminants .

Main properties

Adults are less than 7 mm long, little or no buccal cavity, produces very few eggs .

Pathogenesis

Clinical disease of watery diarrhea requires more than 100,000 adult worms ,which is many more than required to produce disease with the other trichostrongyles. Causes plaques of eroded epithelium in stomach or duodenum.

Generally not seen as the major cause of disease in ruminants since pasture conditions and poor management allowing such burdens of Trichostrongylus will have permitted much more severe disease due to Ostertagia ,Haemonchus or Cooperia. *T. axei* will infect horses and is more pathogenic in this host, such that it is advised not to co-graze horses with sheep or goats. 3 week prepatent time .

Ostertagia ostertagi

most important helminth parasite of cattle - sheep and goats. Common name is the **brown stomach worm .**

Main properties

About 7 to 14 mm long, brown in color, broad shallow buccal cavity, female has vulvar flap and produces less than 100 eggs/day/worm .

Pathogenesis

infective L3 larvae ingested from pasture, cast off retained sheaths and enter gastric glands of the abomasum where they develop to L4 before emerging in the lumen of the abomasum. Alternatively, L4 arrest in gastric glands (become hypobiotic). Active L4 and adults cause dedifferentiation of chief and parietal cells in the gastric glands which leads to loss of acid production and increase in abomasal pH. Mucosal cells form hyperplastic nodules at infected gland opening, which gives a moroccan leather appearance to mucosal surface of the abomasum. Systemically, there is increased catabolism of protein, increased loss of nitrogen in urine and loss of protein into the gut lumen resulting in a negative nitrogen balance and symptoms of protein deficiency. 3 week prepatent time .

Clinical signs

Anorexia and diarrhea, decreased weight gain or weight loss, evidence of protein deficiency in matrix osteoporsis of bone (resulting in poor growth) and hypoproteinemia. Diagnosis based on fecal egg counts if high, low egg counts do not necessarily mean low worm burden (may have arrested L4), and based on response to treatment .

Haemonchus contortus

most important helminth parasite of sheep and goats in USA & Haemonchus placei - cattle. Common name is the **barber pole worm .**

Main properties

It is 10 to 30 mm long, males have asymmetrical dorsal lobe of copulatory bursa , female ovaries twist around red (blood-filled) intestine giving barber pole appearance .

Pathogenesis

prepatent L5 and adult worms are voracious blood feeders that can cause blood loss anemia and hypoproteinemia as early as one week after large intake of L3 from heavily contaminated pasture. 3 weeks prepatent time .

Clinical signs

black or tarry feces (not diarrhea), pale mucus membranes - high fecal egg count when adults are fully mature. Females are prolific egg producers - thousands of eggs/day/worm. Diagnosis based on signs of anemia, usually (but not always) egg count and response to treatment .

Cooperia sp.

C. punctate and C. pectinata are pathogenic for cattle, whereas ***C. oncophora*** infects cattle, sheep and goats but is rarely pathogenic. ***Cooperia curticei*** infects sheep .

Main properties

It is 5 to 10 mm long, cuticle at anterior end is inflated and has transverse striations .

Pathogenesis

Adults lie deep in the mucosa of the proximal small intestine. *Cooperia punctata* and *C. pectinata* are associated with enteritis, catarrhal inflammation. Some areas of the world are seeing ivermectin resistant *Cooperia*. *C. oncophora* that becomes resistant also associated with more clinical disease. 3 weeks prepatent time .

Clinical signs

anorexia and diarrhea that is very similar to *Ostertagia* but not usually a cause of clinical disease.

Diagnosis Of intestinal Trichostrongyloidea:

Diagnosis is based on detection in faeces (methods of Fulleborn, Kato) or duodenal contents of helminth eggs. The value of this study increases in the study sample after standing in the heat for 1-3 days. Immunodiagnostics not been developed.

Treatment

There are the following treatments of trichostrongyloidea : Vermox , Combantrin and Thiabendazol

Dictyocaulus sp. (lung worm)

D. viviparus - cattle, *D. filaria* - sheep and goats, *D. arnfieldi* equine. (host species specific)

Main properties

30 to 80 mm long, thin, buccal cavity is small, male copulatory bursa is smaller than others in this superfamily and spicules are stout, females lay eggs containing L1 that hatch while in the host .

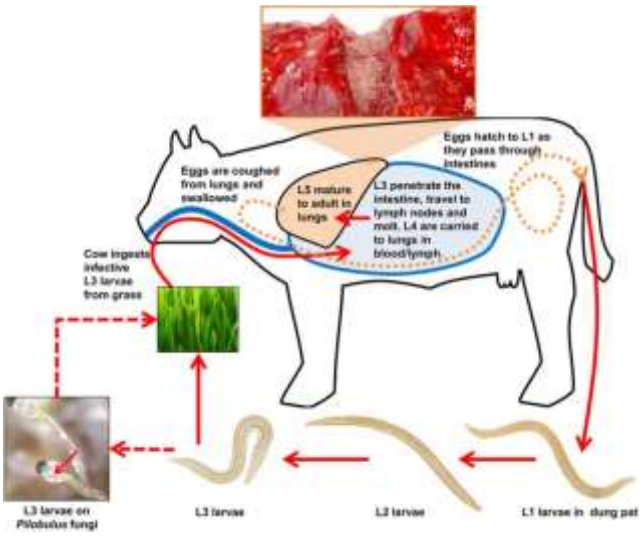
Life Cycle

*__The adult__ worms live in the bronchi of the lungs. **Eggs** are laid in the lungs and may hatch there or in the intestine (after being coughed up and swallowed).

*__First stage larva__ emerge from the cow in the feces. The larvae reach the ensheathed infectious **third-stage (L3)** by about 4 days.

*On pasture, **the L3** migrate up grass blades and be eaten by the cow. The L3 exsheath in the small intestine, penetrate the bowel wall and molt to **L4** in the mesenteric lymph nodes.

*They then migrate to the <u>lungs via the thoracic duct</u>. They reach the lungs about 2 weeks after they were ingested. They molt to **the adult** stage at about 15 days of infection and begin laying eggs around 22 days post-infection (PI).



Pathogenesis

Adult worms reside in the primary and secondary bronchi. Infective larvae ingested from pasture, penetrate gut wall and migrate by lymph ducts and mesenteric lymph nodes to the thoracic duct from which they are carried by venous blood to the lungs (5 days migration time to reach lungs). L4 moult to L5 and adults begin laying eggs about 3 to 4 weeks after infection. Eosinophil and leukocyte containing exudate fills bronchi blocking air flow where worms are located. 3 to 4 week prepatent time .

Clincial signs

Young animals affected most often during first grazing season, often confused with bacterial pneumonia. They show rapid breathing and coughing beginning one week after placed on contaminated pasture; L1 larvae are found in fecal floats or by Baermann examination. Dark brown food granules are visible inside the larvae .

Diagnosis

Definitive diagnosis can be gained by performing a Baerman technique on a faecal sample to identify larvae. Post mortem examination can also be diagnostic; recovery of worms from lungs by the "Inderbitzen" or lung perfusion technique. Worms are flushed out of lungs by pumping water through pulmonary arteries. Water and worms passed out of trachea collected over sieve.

Treatment

If the animal is clinically affected, treatment with anthelmintic such as ivermectin can be used.