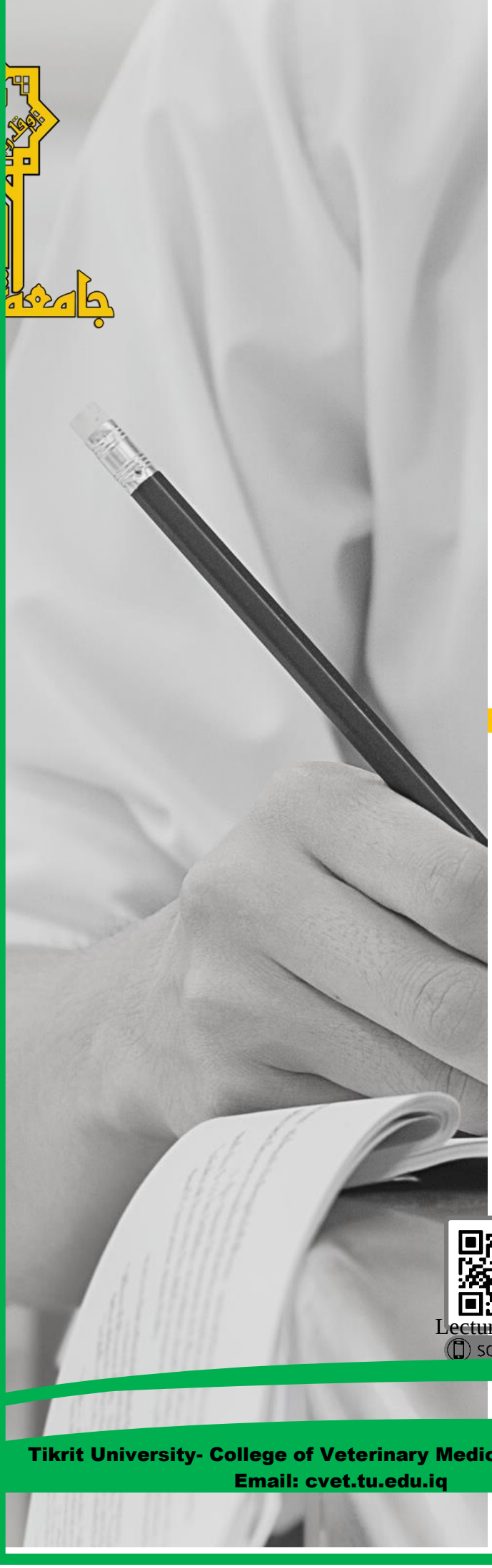

Tikrit University
College of Veterinary Medicine

Nematoda

Subject name: Parasites

Subject year:2024

Lecturer name: Dr.Omaima I.

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Lecturers link

SCAN ME

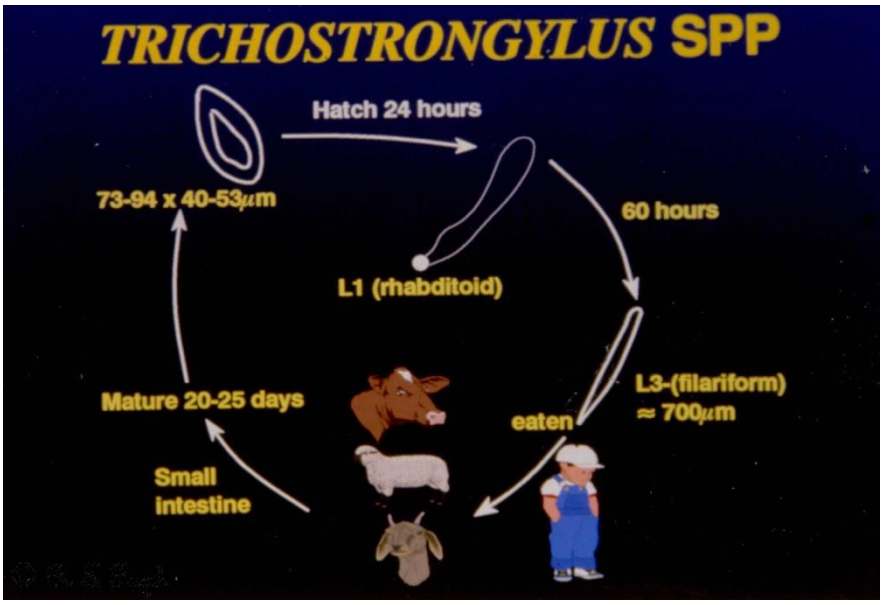
Family: Trichostrongyloidea

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.



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 .

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 .

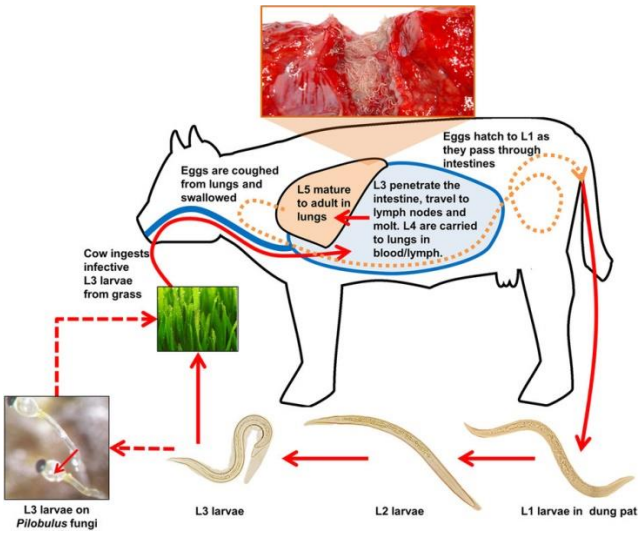
Life Cycle

*__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.