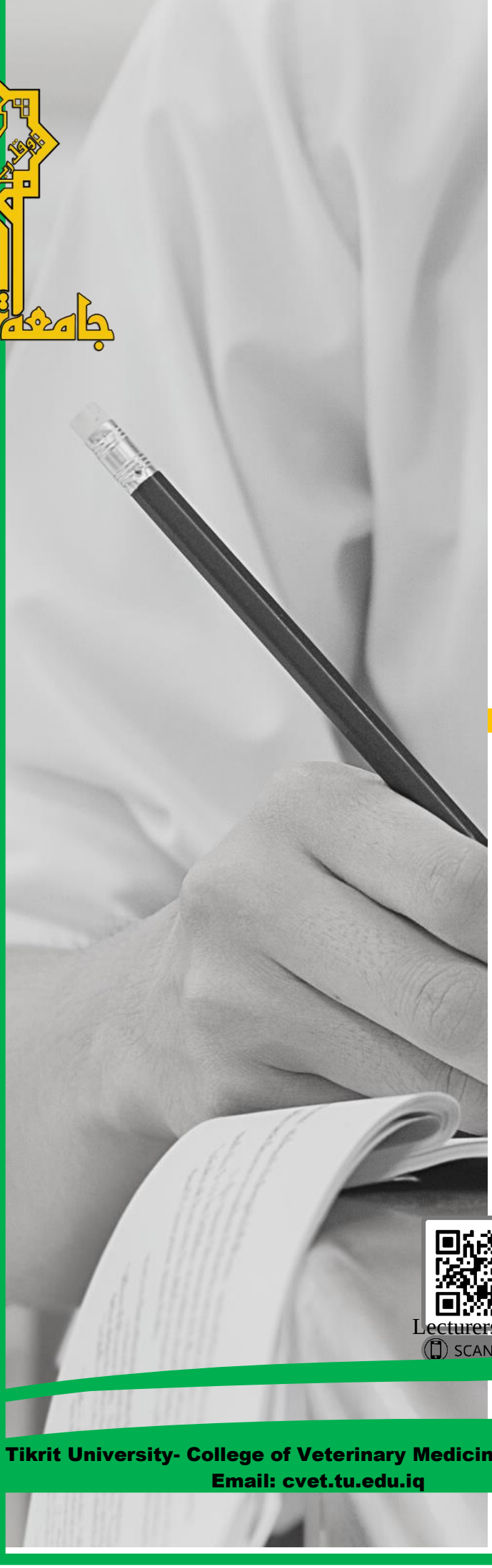

Tikrit University
College of Veterinary Medicin

Nematoda

Subject name: Parasites

Subject year:2024

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Lecturers link

SCAN ME

Family: Strongylidae

Strongylus vulgaris

Main properties

These worm are stout-bodied. They range in length from 2 centimeters (*S. vulgaris*) to 5 centimetres (*S. equinus*). Fresh specimens are reddish-brown due to the ingestion of blood from the host

Life cycle

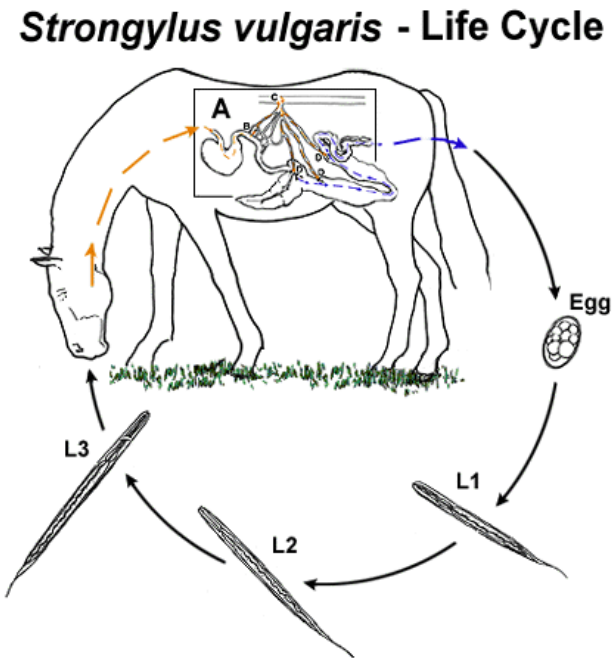
*The *S. vulgaris* **eggs** are shed in the faeces. Under optimal conditions of high humidity and temperatures over 10 degrees, the eggs will transform into **rhabditiform L1** larvae within a few days.

*They then transform into **L2**, and subsequently **L3** without leaving the parasite envelope.

*L3 are ingested by the host when feeding on pasture. L3 enter the small intestine, where they shed their envelope, and then begin migration across the mucosal surface, where they transform into **L4.**

*L4 reach the arterioles of the intestine. Around 2 weeks after the eggs were first ingested they reach the colic and caecal arteries, and then finally the cranial mesenteric artery.

*L4 then transforms into the immature adult and returns to the L5 via the blood vessels. Here they form nodules on the wall of the caecum, and occasionally the colon, and are then released into the lumen.



Clinical signs & pathogenicity

Larvae irritate the walls of the small intestine and the arteries. Adult worms are **"plug feeders,"** meaning that they feed by ingesting plugs of mucosal tissue and capillaries.

Heavy feeding of this type produces intestinal damage, anemia, anorexia, depression, weight loss and dehydration. Each of the three species also produces other specific clinical effects. *S. equinus* and *S. edentatus* cause liver damage and peritonitis.

As noted, S. vulgaris larvae (the L3 stage) live within the lumen of the horse's colon, causing damage as they feed on the host's blood. However, the most serious damage is caused by the L4 larvae, which penetrate the network of mesenteric arteries and begin a 4-month cycle of migration through these blood vessels.

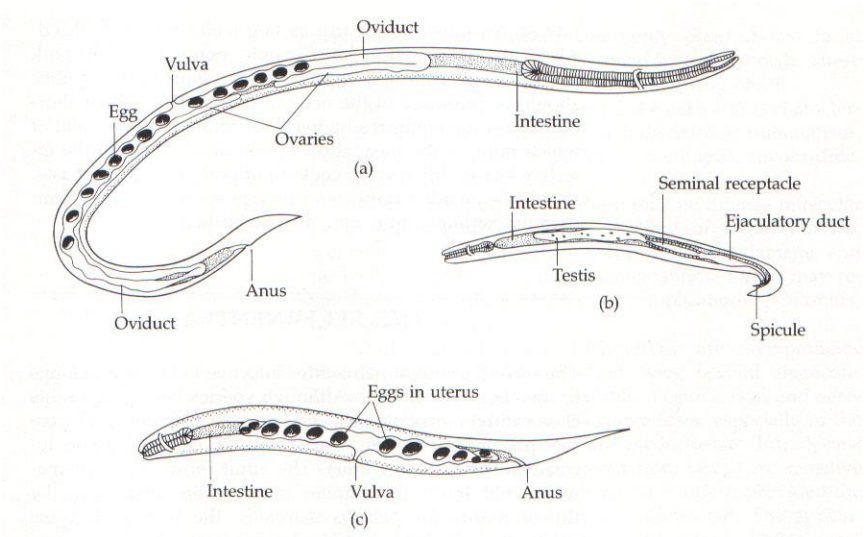
As they travel through the mesenteric arterial network, the larvae produce irritation of the blood vessel walls, inducing the formation of lesions. These arterial lesions can become severe enough to cause a thickening of

the arterial walls (verminous arteritis), a condition that may result in blood clotting and, subsequently, thrombosis. In addition, portions of these clots may break off, travel to, and lodge in other blood vessels, cutting off the blood supply in various other parts of the body. Should the blood supply in the intestines become impaired, the result is a serious condition called intestinal colic **(verminous colic)**.

Death can occur when this series of events progresses to include disintegration of intestinal tissue and the development of gangrene of the bowel. Weanlings and yearlings are partically susceptible to verminous colic.

Family: Strongyloididae

Strongyloides stercoralis



a) parasitic female , b) free-living male , c)free-living female

Life cycle

There are three stages in the life-cycle of Strongyloides stercoralis: free-living, parasitic, and autoinfection.

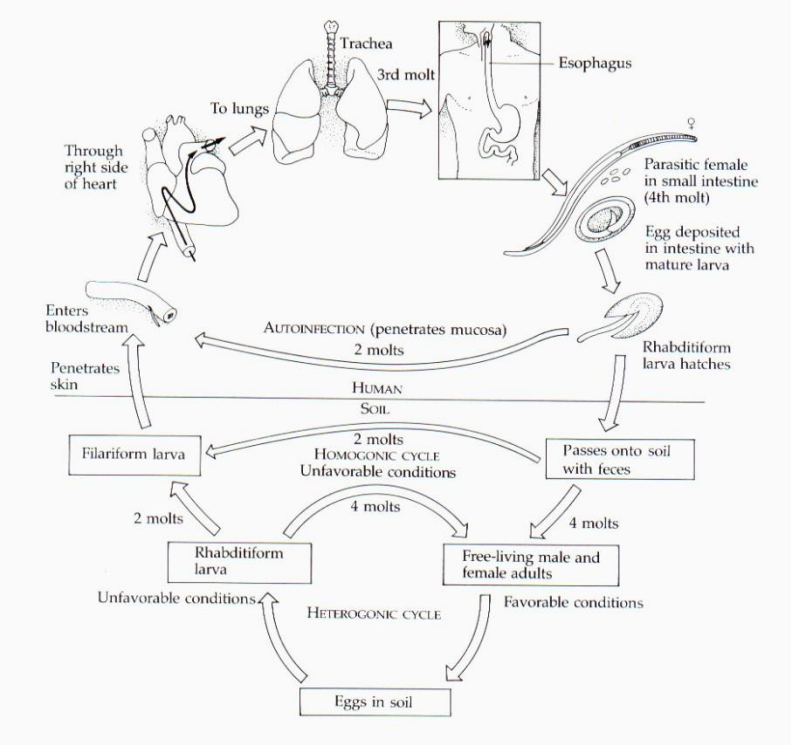
*After copulation, **the free-living female** accumulates eggs in her uterus that contain partially developed larvae. The eggs are laid in the soil where further development occurs.

*The **rhabitiform larvae** either become free-living adults or they become infective **(filariform)** 3rd stage larvae. Only females can enter the next phase of development, the parastitic stage.

*To continue development, the filariform larvae must penetrate a host's skin or be ingested. If they enter the host through the skin, they must travel through host tissues in order to reach the final destination, the intestine.

*There, the females lay **parthenogenetic eggs** which give rise to **rhabitiform larvae.** These eventually leave the host via the feces and develop into **free-living adults** or metamorphose into **filariform larvae**.

*Another variant of the life cycle is the autoinfective phase. **Filariform** larvae, migrating to the exterior, can reinfect the host by penetrating the skin and traveling in the blood to the lungs.



Clinical Signs

Strongyloides can cause severe, life-threatening diarrhea (easily confused with *parvoviral enteritis*) and bronchopneumonia in young puppies and kittens. *Strongyloides tumefasciens* in cats, when symptomatic, causes a primarily large-bowel diarrhea. In addition a very pruritic (i.e. itchy) dermatitis may be noted especially when exposure to filth promotes free-form reproduction and accumulation of large numbers of skin-penetrating infective larvae. Alternatively, infection may be asymptomatic

Diagnosis

Diagnosis of mixed strongyle infection is based on demonstration of eggs in the feces. Specific diagnosis can be made by identifying the infective larvae after fecal culture.

Treatment

A number of anthelmintics, including the benzimidazoles, pyrantel, and ivermectin, are active against adult large strongyles. Large strongyle infections have been eliminated from closed herds with ivermectin treatment.

Family: Trichonematidae

Oesophagostomum sp. (nodular worm)

Oesophagostomum affects **cattle, sheep, goats** and other ruminants, as well as **pigs**. It also infects wildlife (deer, antelopes, camels, monkeys,

wild boars, etc.). It is found worldwide, but is more frequent in warm and humid climates in tropical and subtropical regions.

These worms are called "**nodular worms**" because they cause the appearance of characteristic **nodules** in the large intestine of their hosts.

There are several species of veterinary importance, e.g.:

- ***Oesophagostomum columbianum***. Found worldwide mainly in **sheep, goats** and wild ruminants.

Main properties

Adult *Oesophagostomum* worms are 15 to 20 mm long, whereby females are larger than males. The head of *Oesophagostomum* worms has a prominent cephalic vesicle. Characteristic for this species is that the ovaries are coiled around and along the gut. Males have two very long and slender **spicules** for attaching to the female during copulation.

The eggs are ovoid, have a thin shell, measure ~40-60x70-100 micrometers and contain several cells, depending on the species.

Life cycle

All *Oesophagostomum* species have a **direct life cycle**, i.e. there are no intermediate hosts involved.

*__Adult__ females lay eggs in the large intestine of the host that are shed with the feces.

*Once in the environment the eggs release the **L1-larvae** that complete development to infective L3-larvae in about 1 week, depending on temperature and humidity.

*Ingested larvae penetrate into the intestinal mucosa and form nodules. About a week later they abandon the nodules and migrate to the colon, where they complete development to adults and reproduce. A few larvae may cross the gut's wall and migrate to the liver across the abdominal cavity.

Pathogenicity and clinical signs

Oesophagostomum columbianum is very harmful for **cattle**, especially for stock younger than 2 years*:* massive infections can be fatal.

Infective larvae penetrate the intestinal wall and the host's organism reacts building nodules the size of a pea. This disturbs considerably the physiology of the gut, particularly the absorption of liquids, which causes diarrhea, but also the peristaltic movements. Digestion and defecation can be affected, and enteritis is possible. Deadly bacterial infections can happen if larvae migrating to the liver across the abdominal cavity, or if the nodules burst towards the abdominal cavity.

Diagnosis

Is confirmed through detection of characteristic eggs in the feces.

Treatment

Numerous broad spectrum anthelmintics are effective against adult worms and larvae, e.g. several benzimidazoles