

Lecture # 21 Order Enoplida: *Trichinella spiralis*, *Trichuris* and *Capillaria*.

And the non-nematode “thorny headed worm” *Macracanthorhynchus* of the Phylum Acanthocephala.

Objectives:

1. Draw a “stichosome esophagus” and a bipolar plugged egg.
2. Describe the life cycle of *Trichinella spiralis* and explain why pigs are a source of infection for humans.
3. List the control measures used to reduce risk of trichinosis for humans.
4. Describe the life cycle of *Trichuris sp.* and list reasons why it is difficult to treat and control this parasite.

Outline:

- I. Enoplida: This Order contains the genera of the superfamily Trichinelloidea and the genus *Dioctophyme*, which is in another superfamily and contains the largest parasitic nematode.
 - A. Trichinelloidea genera have a **stichosome esophagus** which is a capillary tube-like structure associated with a “stack” of gland cells (like a stack of donuts) called stichocytes.
 - B. Trichinelloidea genera as well as *Dioctophyme* have eggs with bipolar plugs.
- II. *Trichinella spiralis* and 5 additional species have encapsulated larva: whereas, *T. pseudospiralis* and two other species produce unencapsulated muscle larva: **ALL CAN INFECT MAMMALS. *T. pseudospiralis* also infects birds and two other unencapsulated species also infect reptiles.**
 - A. Morphology: Adults found in the small intestine are tiny, 1.4 to 4 mm long and have a stichosome esophagus typical of the genera of the superfamily Trichinelloidea.
 - B. Life cycle: Adult males and females found in the small intestine, but males live only long enough to copulate with females. Fertilized females penetrate into the mucosa through Lieber Kuhn's glands and produce larvae for a few weeks that enter lymphatic ducts and then blood to be distributed to muscles. L₁ larvae in muscle tissue stimulates fibrous capsule formation (*T. spiralis* and 5 other species) or do not, in the case of *T. pseudospiralis* and 2 related species. Muscle cell metabolism is redirected to support larval needs for growth; larvae can remain viable (infective) for many years, but do not develop further until they are ingested with muscle tissue by an appropriate vertebrate host. Larvae are liberated from muscle by digestion and adults develop quickly (less than 2 days) in the small intestine. Unlike most of the other nematodes studied so far, those of the Order Enolipa (including *Trichinella* and *Trichuris*) have L1 that are infective.
 - C. Pathogenesis: Enteritis due to adult worm development is transient; the primary disease problem is associated with larvae invading muscle tissue causing myositis and even paralysis of muscles in heavy infections. Paralysis of respiratory muscles can lead to death.
 - D. Clinical signs and Diagnosis: Clinical disease is mostly a concern for humans, and is rarely diagnosed in pigs or other animals (although it may occur); **trichinosis in man is a major public health concern**. A history of eating meat that was not completely cooked, high antibody levels against *Trichinella* antigen, eosinophilia, muscle pain, facial edema are classic for trichinosis.
 - E. Control and Treatment: The control strategy in the U.S.A. is to advise people to cook pork (145°F) thoroughly, in contrast to the control strategy in Europe that requires all pork be inspected for *Trichinella* larvae in muscle. The U.S.A. had much higher rate of trichinosis in humans than Germany. Also, requiring garbage fed to swine to be cooked has reduced *Trichinella* in swine, but other routes of infection exist, such as rodents eaten by pigs. No treatment of pigs for trichinosis is feasible. Albendazole or fenbendazole (50 mg/kg for several days) and pyrantel (50 mg/kg 2x/day for 2 weeks) are used to treat human and the rarely diagnosed pet infection.

III. *Trichuris sp.*: found in the large intestine, and cecum. *Trichuris vulpis* is specific for the dog. *T. campanula* (rare) is specific for the cat. *T. ovis* is specific for sheep and goats. *T. discolor* is specific for cattle. *T. suis* is specific for swine. *T. trichiura* in primates including humans.

- A. Morphology: Adults are 50 to 80 mm long. The anterior 2/3 of the body is thin (thread-like), whereas the posterior 1/3 is thick. **Eggs are double operculated, bipolar, typical of all genera in this superfamily.** The esophagus is composed of the characteristic stacked stichocytes.
- B. Life cycle: Adult females in the large intestine produce eggs that require at least 3 weeks to become infective. **Eggs containing L1 are infective and very resistant and long-lived outside the host.** When eggs are ingested L1 hatch in the small intestine and penetrate the mucosa for 8 to 10 days before returning to the intestinal lumen and migrating to the cecum and colon. Actively feeding larval stages develop to adults in the cecum and colon. **The prepatent time is 3 months in dogs** but as short as 6 weeks in swine.
- C. Pathogenesis: swine and dogs demonstrate the most severe lesions. These lesions include inflammation of the large intestinal mucosa, hemorrhagic foci and eosinophil infiltration.
- D. Clinical signs and Diagnosis: Swine recently turned out on pasture (3 to 4 weeks) and dogs on dirt lots or runs are most often affected. Swine show a severe dysentery that is intermitently bloody, anorexia, anemia weight loss or failure to gain weight. Dogs show intermittent diarrhea and anorexia. Fecal exam may show characteristic bipolar eggs, but **immature adults and larvae can cause severe disease** and adults are sporadic egg producers.
- E. Treatment and Control: Several drugs are effective against the mature worm only, these include dichlorvos, fenbendazole, mebendazole, glycothiarsol and milbemycin. **These drugs do not kill larval stages or immature adults during the first 2 months of infection in dogs.** Oxfenbendazol or fenbendazole may be effective against larvae at high doses or with prolonged administration. Pyrantel or hygromycin B are used in swine as a continuous feed additive that kills larvae hatching from eggs. **Therefore, it is imperative to treat and repeat treatment monthly, then recheck in 3 months because of the long prepatent development time of larvae.** Sanitation is very important because eggs are long-lived in soil. Milbemycin can be dosed as the heartworm preventative Interceptor every 3 weeks instead of 4 weeks.

WHAT ARE THE IMPORTANT CONCERNS YOU WANT YOUR CLIENT TO KNOW ABOUT TREATMENT AND CONTROL OF *TRICHURIS* INFECTIONS IN DOGS?

IV. *Capillaria sp.* (recent genus name change for several of these species): over 300 species have been described in mammals, birds and fish. All are found as adults in **epithelial surfaces** and the **site in the host is often specific** for the particular *Capillaria* species.

- A. General morphology: The body type is capillary-like and the stichosome esophagus is long. Males have a spicule sheath even when there is no spicule. Females produce eggs with bipolar plugs.
 - B. *Capillaria sp.* of dogs and cats.
 - 1. *Capillaria bohmi* (or *Eucoleus bohmi*) and *Capillaria (Eucoleus) aerophilus* are found as adult worms in **nasal sinuses (bohmi), bronchi and trachea (aerophilus) of canids and felids.**
 - a. life cycle - egg containing L1 and L1 in earthworm paratenic host are infective. L1 hatch in the intestine and migrate to the lungs, then up the bronchi in the epithelium.
 - b. pathogenesis - dogs and cats show a slight cough, whereas fox may have severe bronchitis.
 - c. diagnosis – **differential with allergic rhinitis.**
- IMPORTANT: corticosteroids will exacerbate nematode infection.
Fox may be a reservoir host for infections to pets in urban as well as rural environments.

2. *Capillaria plica* (or *Pearsonema plica*) is found as adult worms in the **urinary bladder** of cat and dog, whereas *Capillaria feliscati* is found in the urinary bladder of cats.
- life cycle - indirect, using earthworms as intermediate hosts and an unknown paratenic host is suspected.
 - pathogenesis - possible cause of cystitis but usually asymptomatic when eggs are detected in urine sediment.
- C. *Capillaria bovis* and *C. brevipes*: adults are found in the **small intestine** of ruminants. They probably use a direct life cycle and cause no pathological lesions.
- V. Control and Treatment of capillariasis.
- Treatments using levamisole, fenbendazole or ivermectin (0.2 mg/kg) have been reported to be successful.
 - Control involves management to prevent access to paratenic hosts or sanitation to minimize egg contamination similar to that used for whipworms. Pyrantel as a feed additive has been used to kill hatching L1 in birds.
- VI. Urbanized raccoon as source of helminth infection.
- Macracanthorhynchus ingens* – dogs eating beetles where raccoons are common.
 - Heterobilharzia americana* – blood fluke of raccoons easily infects dogs. Water contact, skin penetration by cercariae (see Trematode section, Schistosomatidae).
 - Dracunculus insignis* – ingestion of water containing infected intermediate host.
 - Physaloptera* – stomach worm.
 - Baylisascaris procyonis – severe visceral larval migrans from ingestion of infective eggs.
- VII. *Macracanthorhynchus hirudinaceus*: found in the small intestine of swine.
- M. ingens*** from reservoir host raccoons to dogs. The common name is thorny-headed worm.
- Morphology: Adults are typical of worms in the Phylum Acanthocephala (this is not a nematode, not a cestode, not a trematode). They have a flattened body insitu before fixation, no digestive tract, a retractable spiny attachment organ at anterior end. This species is pale red in color, 100 to 350 mm long and 4 to 10 mm thick. The cuticle is transversely wrinkled and females produce brown eggs with a pitted surface that contain an acanthor larva.
 - Life cycle: Adult male and female worms in small intestine mate to produce eggs that are passed in feces. When these very resistant eggs are ingested by dung beetles and other bugs (millipeds) an acanthor larva is released that encysts in the bug. Pigs and dogs ingest the bug or its grub containing the larva and an adult worm is present in the intestine in 2 to 3 months.
 - Pathogenesis: Penetration of the worm proboscis deep into intestinal mucosa results in inflammation and granuloma at the site; sometimes there is perforation of the intestinal wall.
 - Clinical signs and Diagnosis: If there is deep penetration, the host may suffer diarrhea, acute pain and anorexia. The easily recognized eggs passed in feces are diagnostic.
 - Treatment and Control: Prevent access to dung beetles and their grubs. No effective drug treatment is FDA approved but 0.2 mg/kg ivermectin for seven days has been reported to clear infections.