

Nematode Appendix

(Supplemental Material that will NOT be on Exams)

Lecture #19 Order Rhabditida: genera *Rhabditis*, *Halicephalobus*.

***Rhabditis*:** *Rhabditis strongyloides* - free-living soil nematode. Other species of *Rhabditis* infect conjunctival sac or hair follicule of rodents.

- A. Life Cycle: *Rhabditis strongyloides* replicates in manure, larval stages can penetrate the epidermis on soiled cattle, swine and dogs. Association with wet straw bedding.
- B. Clinical Signs: Dermatitis may look like mite induced mange but find rhabditiform larvae (less than 600 um long) on skin scraping.

Halicephalobus (synonym = *Micronema*)

- A. Life Cycle: Adult female with rhabditiform esophagus has been found in various tissues of horses. Normally free-living in soil and manure.
- B. Clinical Signs: swelling of tissues; meningitis.

Lectures #20 & #21: Overview of the Order Strongylida: Superfamily Trichostrongyloidea

***Nematodirus* sp.:** *N. helveticus* in cattle, *N. spathiger* in cattle and sheep, *N. battus* in sheep where it can cause severe disease in sheep due to “larval storms” in certain restricted localities (may be spreading).

- A. Morphology: 10 to 23 mm long, inflated cuticle at anterior end: males have long, thin spicules; females have a spine at the tip of the posterior end and the uterus will contain very large “football shaped” eggs.
- B. Life cycle: Variation from usual trichostrongyle free-living larval development in that L1, L2 and L3 remain in the egg. L3 hatches from the egg. Adult female produces very few eggs but larvae in eggs are well protected and survive better than free-living L1 and L2. The timing of L3 hatches and appearance on pasture of infective L3 is coordinated in *N. battus* by freezing followed by a certain number of warm days. In some localities this leads to “larval storms” that can cause severe disease in grazing sheep.
- C. Pathogenesis: Larvae and adults are found in the proximal small intestine usually associated with slight enteritis and villus atrophy similar to *Trichostrongylus colubriformis*.
- D. Clinical signs: usually in sheep infected with *N. battus* and presents with severe watery diarrhea and dehydration.

***Ollulanus tricuspis*:** infects stomach of pigs and cats.

- A. Morphology: tiny, less than 1 mm long, anterior end rolled up, female has several sharp pointed structures at the posterior end; males have well developed copulatory bursa.
- B. Life cycle variation: females produce larvae not eggs (viviparous), larvae complete development in the stomach to adult stage without leaving the host. New infections established when vomit is ingested by young animals.

Hyoststrongylus rubidus - stomach of pigs.

- A. Morphology: thin, reddish color, 5-10 mm long, transversely striated cuticle, males have well developed bursa, strongyle-type ova.
- B. Life cycle: Adult males and females in lumen of stomach, attached and sucking blood from gastric mucosa, eggs passed in feces require 7 days to hatch and develop to L₃ infective larval stage **on pasture or dirt** lot, route of infection is ingestion; L₃ enter gastric glands like *Ostertagia* in ruminants, moult twice, and emerge to lumen of stomach as adults. Prepatent time is 17-19 days.
- C. Pathogenesis: Similar to *Ostertagia* in ruminants, larval stages and young adults in gastric glands cause decreased acid and pepsinogen production and mycosal nodules, emergence from glands and blood-feeding adults cause gastritis, ulceration, and anemia often with catarrhal inflammation and thick mucus.
- D. Diagnosis: Ova in feces similar to *Oesophagostomum*; clinical signs of gastritis include vomiting (blood tinged). Also diarrhea and thirst.
- E. Treatment and Control: Dichlorvos effective against adults, fenbendazole and other new benzimidazoles as well as ivermectin effective against adults and larval stages. Remove pigs from pasture or dirt lots.

Lecture # 22: Superfamily Strongyloidea: The Strongyles of equids.

Triodontophorus sp.: large intestine of equids.

- A. Morphology: robust, red worm, 10 to 25 mm long.
- B. Life cycle: similar to *Strongylus sp.* except larvae do not migrate beyond the wall of the large intestine. Prepatent time is 2 to 3 months.
- C. Pathogenesis: active blood feeders, cluster in groups, can cause ulceration of bowel mucosa.
- D. Clinical signs: diarrhea and anemia.

Stephanurus dentatus (kidney worm of pigs)

- A. Morphology: 20 to 45 mm long, stout body, buccal cavity is cup-shaped and has a thick-walled capsule with teeth at the base; male copulatory bursa is small. Strongyle-type eggs are found in urine.
- B. Life cycle: Adult worms found in cysts in or near kidney with communication channels to ureters, thus eggs passed in urine. Infective larvae (L₃) develop in the environment (as for trichostrongyles).
Infection of the definitive host occurs by ingestion of L₃ (direct or via earthworms which act as facultative intermediate host) or by skin penetration. L₃ develop into L₄ which migrate to the liver, break through after 4-9 months of wandering and encyst in the area around the kidneys or ureters where they mature and begin producing eggs. Prepatent time is 9 - 16 months.
- C. Pathogenesis: Migrating larvae cause cirrhosis and abscesses in the liver; aberrant migration of larvae may cause other lesions including spinal cord lesions and fibrous scarring of loin muscle (carcass downgrade). Adults are in green pus-filled cysts.
- D. Clinical signs and Diagnosis: Skin penetration by the infective larvae can cause subcutaneous nodules and stiffness in legs. Liver lesions result in anorexia and emaciation. Eggs in urine are diagnostic but may not always be present; often diagnosed postmortem.
- E. Treatment and Control: Generally associated with swine on pasture or dirt lots because infective L₃ larvae do not survive on dry surfaces such as concrete slabs. Parasites can be eliminated with a program of breeding only gilts, where after one litter of piglets, the young sows are sold (prior to time when mature worms can shed eggs). Levamisole and fenbendazole are effective against adult worms.

Superfamily Metastrongyloidea

***Protostrongylus* and *Muellerius*:** found in the lungs of sheep and goats.

- A. Morphology: Slender, red. *Protostrongylus* is 15 to 35 mm long, male has a short bursa; *Muellerius* is 10 to 25 mm long, male lacks a copulatory bursa, posterior end is coiled.
- B. Life cycle: *Protostrongylus* adults are found in the bronchioles, *Muellerius* adults in alveoli and parenchyma; both produce larvated eggs (containing L1) that is passed in feces; both require snails as intermediate hosts. When infected snails are ingested by sheep or goats, the L3 migrate to the lungs via the lymphatics. Prepatent time is 30 to 37 days.
- C. Pathogenesis: *Protostrongylus* - young animals most susceptible; localized lobular pneumonia. *Muellerius* -- older animals infected, causes subpleural grayish nodules.
- D. Diagnosis: L₁ in feces have "S" shaped tail tip. *Muellerius* also has spines. Distinguish both from *Dictyocaulus* L₁. Use the Baermann funnel technique to isolate larvae from feces.
- E. Treatment and Control: fenbendazole, ivermectin; remove snails and slugs.

***Parelaphostrongylus tenuis*:** adult found in the meninges of white-tailed deer. Aberrant infections

- cause neurological disease in sheep, goat, elk, moose. "Menigial worm of deer".
- A. Life cycle: Adult worms in spinal cord and brain release eggs which hatch. L₁s traverse the lungs and exit in feces; infect snail intermediate host. Grazing deer (including sheep or goats) are infected by ingesting infected snails. Prepatent time is 3 months, but larvae can reach the central nervous system in 10 days.
 - B. Clinical signs: neurological signs/paralysis in sheep, goats, and moose. Infection is NOT patent in aberrant species; i.e. cannot be diagnosed from a fecal exam. No clinical signs in deer.

***Crenosoma vulpis*:** found in the bronchi and trachea of dogs, raccoons, wolves and fox.

- A. Morphology: 5 to 15 mm long with overlapping circular folds in the cephalic cuticle.
- B. Life cycle: Adults in the bronchi produce eggs that hatch to L1; larvae are passed in feces and become L3 in snails/slugs which are ingested by fox, or less commonly, dogs.
- C. Pathogenesis: Bronchitis with/without nasal discharge; severe disease in fox raised for fur.
- D. Diagnosis: L1 in feces and nasal discharge; use Baermann technique to isolate larvae.
- E. Treatment and Control: Treat with levamisole or fenbendazole; prevent access to snails/slugs.

***Filaroides osleri* (or *Oslerus osleri*) and *F. hirathi*:** both are found in the dog.

- A. Morphology: 5 to 15 mm long, males lack copulatory bursa.
- B. Life cycle: Direct, no intermediate hosts. Adult worms in lung. Eggs hatch to release infective L1 which are ingested by pups from saliva or feces of bitch; larvae migrate and mature in lungs of pup. Prepatent time ~10 weeks for *F. osleri*, 5 weeks for *F. hirathi*.
- C. Pathogenesis: *F. osleri* adults are in submucosal nodules (~1 cm diameter) in trachea or bronchi -- block air flow, most severe in 6 to 12-month old dogs. *F. hirathi* adults in lung parenchyma cause minimal pathology, but immunosuppressed dogs can die from *F. hirathi* -- due to hyperinfection resulting from autoinfection.
- D. Diagnosis: Bronchoscopy and/or radiography show nodules of *F. osleri*; L1 are hard to find in sputum (collect night or early morning samples). Potential problem in dog colonies.
- E. Treatment and Control: *F. osleri* is difficult to treat successfully - albendazole or fenbendazole. *F. hirathi* can be cleared with albendazole. Hand-rear the pups if infections in bitches can not be eliminated. Very difficult to control in kennels.

Lecture # 23: Superfamily Ancylostomatoidea (hookworms)

Uncinaria stenocephala: found in the small intestine of dogs and cats; less common hookworm.

- A. Morphology: 0.5-1.2 cm long; paired chitinous plates (not teeth) in buccal capsule. Eggs are slightly longer than those of *A. caninum*, being ~65-80 by 40-50 um.
- B. Life cycle: Infection is by ingestion of infective larvae. Prepatent period of ~ 16 days. Occurs in temperate and northern geographic regions. No transmammary transmission.
- C. Pathogenesis: relatively non-pathogenic.

Bunostomum: in cattle, sheep and goats.

- A. Morphology: Adults are 1 to 3 cm long, dorsally bent anterior end, large buccal capsule with chitinous plates.
- B. Life cycle: Infection by ingestion or skin penetration. No transmammary transmission.
- C. Pathogenesis: Blood loss anemia; lower leg pruritus (stamping and licking), diarrhea. D. Diagnosis: If diarrhea, suspect nematode infection but not *Haemonchus* (no diarrhea). Culture feces for L3 larvae; it is the smallest sheathed larva. Feedlot or wet pasture areas facilitate transmission (same as *Strongyloides*) -- an important observation in your history.
- E. Treatment and Control: Most broad spectrum anthelmintics are effective; drain wet areas

Lecture # 24: Order Oxyurida & Order Ascaridida

Toxascaris leonina: found in the small intestine of dogs and cats.

- A. Morphology: Similar to *T. canis* except the adult male has a cone shaped posterior end whereas *T. canis* has a finger-like process at the posterior end. Eggs are oval and smooth with a translucent embryo (differentiate from *Toxocara* eggs on fecal exam).
- B. Life cycle: Infection is only by (1) direct ingestion of eggs, or (2) ingestion of an infected paratenic host (encysted larvae). Larvae hatch from the eggs or are released from tissues of the paratenic host, and develop directly into adult worms in the intestine. Prepatent period is ~ 8 to 10 weeks.
- C. Pathogenesis: Intestinal inflammation similar to *Toxocara* but less severe. Visceral larva migrans NOT reported with this species.
- D. Clinical signs: With heavy infections, signs can be similar to *Toxocara*. Infections are more common in mature dogs and cats.
- E. Treatment and Control: Same as for *Toxocara*.

Anisakis sp.: Adult worms are found in the intestinal lumen of marine mammals. Larval stages are found in the tissues of fish, crustaceans and squids. L3 larvae can be transmitted to man through uncooked or lightly salted fish, and migrate into the intestinal wall causing eosinophilic granulomas.

Lecture # 25: Genera of the Order Enoplida: *Trichinella spiralis*, *Trichuris sp.* and *Capillaria sp.*

***Capillaria putorii*:** adults are found in the small intestine of cats, but more commonly in bear and raccoon.

- a. life cycle - unknown, probably indirect.
- b. pathogenesis - none in the cat.

Capillaria sp. in birds. Most often found at various sites in the digestive system.

1. *Capillaria contorta* and *C. annulata*: adults are found in the crop and esophagus of turkeys, ducks and wild birds.
 - a. life cycle is indirect using the earthworm as a paratenic host or direct with a prepatent time in the bird of 1 to 2 months.
 - b. pathogenesis due to adult burrowing leaves inflammatory tracts in the mucosa of the crop which can be seen by stretching the crop and holding it up to light.
2. *Capillaria caudinflata* and *C. obsignata*: adults are found in the small intestine of chicken, turkeys and other birds including pigeons.
 - a. life cycle of *C. caudinflata* requires the earthworm, whereas *C. obsignata* eggs are directly infective.
 - b. pathogenesis is the result of burrowing in the small intestine mucosa causing a hemorrhagic and diphtheritic enteritis. Diarrhea and weight loss ensues. Worms can be seen microscopically in scrapings of the mucosa.
3. *Capillaria anatis* adults are found in the cecum of chickens and turkeys but are not usually pathogenic.

Lecture # 26: Overview and Details of the Order Spirurida

Thelazia sp.: adult worms and L₁ larvae are found in the conjunctiva and lacrimal duct of the eye. Cattle and sheep - *T. rhodesii*. Dogs, cats, sheep, man - *T. californiensis*.
Horses - *T. lacrymalis*.

- A. Morphology: These 10 to 20 mm long worms are easy to recognize based upon host site and the transverse striations present in the cuticle.
- B. Life cycle: Adults in the conjunctival spaces produce L₁ that infect various species of the fly genus *Musca* when flies are feeding on lacrimal secretions. L₃ develop in the fly and infect new hosts when flies feed near the eye.
- C. Pathogenesis and Clinical signs: Mechanical and/or inflammation associated with the adult worm leads to corneal ulceration. Severe lesions may cause blindness.
- D. Epidemiology and diagnosis: Close examination using topical anesthetic, which facilitates release of adults from the lacrimal duct, is used to find adults and larvae. There is a seasonality of appearance of adults and L₁ in conjunctiva of cattle that coincides with fly populations.
- E. Treatment: Levamisole has been used for cattle infections and fenbendazole at high dose for infections in horses. Ivermectin and other macrocyclic lactones are effective.

Spirocerca lupi: found in dogs, wild canids and wild felids (especially bobcats).

- A. Morphology: Adults are 30 to 80 mm long and pink-red in color. Host site in the wall of the aorta or esophagus makes identification easy.
- B. Life cycle: Granulomatous cysts in the wall of the esophagus/aorta or stomach contain “nests” of adults, and the small oval eggs containing L₁ are passed into the digestive tract lumen by channels in the cyst wall. The larvated eggs are ingested by coprophagous beetles which are the intermediate host. Rodent and bird paratenic hosts are likely sources of infection to carnivores. L₃ when ingested by canids or wild felids migrate from the stomach by the gastroepiploic artery into the wall of the anterior aorta and esophagus where they remain in cysts that communicate with the esophagus. The prepatent time is 5-6 months.
- C. Pathogenesis: The granuloma in the aorta and/or esophagus may become a tumor that leads to hypertrophic pulmonary osteoarthropathy.
- D. Diagnosis: Small thick-shelled eggs, containing larvae, are few in number or absent from feces. More prominent clinical signs of esophageal constriction include vomiting of undigested food and radiographic opacity in the esophagus. Aortic rupture can result in sudden death.
- E. Treatment and Control: Disophenol is effective in killing adults, and avoidance of paratenic hosts as sources of infection is important for control.

Ascarops sp. and Physocephalus sp.: found in the stomach of domestic and feral pigs.

- A. Morphology: Both of these nematodes are 10-22 mm long and red in color. The pharynx has spiral cuticular thickenings that are apparent in cleared specimens. The males do not have well-defined copulatory bursa, which allows differentiation from *Hyostrogylus*. Eggs are small, thick shelled and contain larvae when passed in feces. Not necessary to differentiate these two genera.
- B. Life cycle: Adult males and females are found on or with anterior ends embedded in the stomach mucosal surface. Eggs passed in feces are eaten by the required intermediate host, dung beetles, and infective larvae develop in beetles in 4 weeks. Ingestion of the beetle releases larvae to penetrate gastric mucosa and develop to adults in pigs. The prepatent time is 6 weeks.
- C. Pathogenesis: Gastritis is especially apparent in the fundus region showing reddening and edema of mucosa, as well as ulceration.
- D. Clinical signs and diagnosis: Pigs show emaciation, marked thirst, anorexia. Eggs can be present in feces but they are very few in number.
- E. Treatment and Control: Dichlorvos and benzimidazoles such as oxbendazole are effective. To control prevent access to dung beetles.

Stephanofilaria: found in the dermis of cattle.

- A. Morphology: This is a tiny, thin worm 2 to 8 mm long. It is found in the abdominal dermis.
- B. Life cycle: Adult worms produce microfilariae that accumulate in the ventral abdominal dermis and are ingested by its intermediate host, the horn fly *Hematobia*. The microfilaria develops to the L3 infective larva in the fly and enters a new host through lesions in the skin created by the fly when it feeds.
- C. Diagnosis and clinical signs: Cattle with this infection show crusty skin, papules going to ulcers and hyperkeratosis. Microfilariae can be seen leaving skin biopsies allowed to incubate in warm saline.

Setaria: found in the **peritoneal cavity** of ruminants and horses (nonpathogenic); Adults are up to 50mm long. Microfilariae are found in the blood and mosquitoes are the intermediate host. Prepatent time is 7 to 10 month

Elaeophora schneideri: adults are found in the **carotid artery** of mule deer, elk and sheep in southwestern states mountain pastures above 6000 ft. elevation.

- A. Morphology: Adults are typical of filarid worms being thin and lacking distinctive posterior or anterior end characteristics. They are 60 to 120 mm long.
- B. Life cycle: Adults in the carotid artery produce microfilariae that are ingested by the intermediate host *Tabanus* (horsefly). L1 develops to L3 in 2 weeks in the horsefly. When the fly feeds on a new host adults appear in the carotid and become patent in 18 weeks.
- C. Pathogenesis and clinical signs: Adults in carotid (also iliac and mesenteric) arteries produce microfilariae that stimulate an inflammatory response resulting in pruritus and dermatitis on the head, face, and hoof coronary band of sheep and elk. As with the lack of response to *Dirofilaria immitis* microfilariae by dogs, deer show little or no pathology.
- D. Treatment: Piperazine in large doses has been reported usefull in early work, but ivermectin may be effective.