

## Lecture # 18: Superfamily Strongyloidea: The Strongyles of equids.

### Objective:

1. List the morphological features shared by genera of the superfamily Strongyloidea.
2. Describe the larval migrations of *Strongylus vulgaris* in horses and relate this to the pathogenesis of *S. vulgaris* induced disease.
3. Outline general control measures for horse strongyles.
4. Compare the clinical signs associated with *Strongylus vulgaris* infection to those associated with small strongyle infections.

### Outline:

- I. Superfamily Strongyloidea: Distinctive morphology. **Contains the most important helminth parasites of horses; i.e. strongyles in large intestine.** Also includes the nodular worms of ruminants and pigs, and kidney worm of pigs.
  - A. Large, thick bodies.
  - B. Large buccal cavity, usually thick walled (capsule).
    1. **leaf crown** - projections at opening of the buccal cavity
    2. teeth, if present, are at the posterior part of the buccal cavity. Teeth serve to lacerate the mucosa.
  - C. Copulatory bursa is well developed, spicules are long and thin.
- II. Life cycle of the superfamily Strongyloidea.
  - A. For the genera of veterinary importance the life cycle outside the host is the same as for the trichostrongylid nematodes. DIRECT life-cycle with free-living, microbivorous first and second larval stages, and INFECTIVE third stage larva. Remember that development is facilitated by warm and humid conditions; at the third larval stage, the parasites are very resistant and survive cold and dry conditions as long as stored energy reserves in infective L3 larvae are not depleted by activity at warm temperatures.

Exceptions to the above life cycle: *Stephanurus* (kidney worm of pigs) and *Syngamus* (tracheal or 'gape'-worm of birds) where L3 can use earthworms as transport hosts.

- B. Host species: Most are found as adults in the **large intestine of equids** i.e. large strongyles (e.g. *Strongylus* sp.) and small strongyles (many different species of cyathostomes).

Also found in ruminants (e.g. *Oesophagostomum* in large intestine of cattle, sheep, goats), swine (e.g. *Stephanurus*, the kidney worm of pigs; *Oesophagostomum* in pigs), and in avian species (e.g. *Syngamus*)

### C. Horses:

Three large strongyles of the genus *Strongylus* are important: *Strongylus vulgaris*, *S. equinus*, and *S. edentatus*, which are found as adults in the cecum and colon.

The L3 larvae can migrate through the gut into surrounding abdominal organs and tissues and cause damage, especially in young animals.

Larvae of cyathostomes (small strongyles) do not migrate beyond the gut wall. However, cyathostome larvae can arrest in the gut wall and reactivate at later times to cause colitis and severe diarrhea.

### III. *Strongylus vulgaris*, *S. equinus* and *S. edentatus*

- A. Morphology: buccal capsule with teeth characteristic for each species: *S. vulgaris* has two rounded teeth; *S. equinus* has three pointed teeth; *S. edentatus* has no teeth. Adult worm sizes are also different: *S. vulgaris*, 15 to 25 mm; *S. equinus* and *S. edentatus*, 25 to 45 mm.
- B. Life cycle: internal migrations within the horse are different for each species.
  1. *S. vulgaris* larvae enter the submucosa of the large intestine 1-7 days after infection, moult to L<sub>4</sub> migrate into the arterial walls (8 to 14 days post infection), and most work their way up into the wall of the cranial mesenteric artery, some enter the aorta and cause pathological changes. Thrombi (+/- larvae) may occur from 2 to 8 weeks post infection. At 2 to 4 months post infection, most larvae in the cranial mesenteric artery return via the bloodstream to the colon and cecum, where they survive as mature adults. Prepatent time is 6 months.
  2. *S. equinus* L<sub>4</sub>s leave the cecum and colon, and migrate to the liver. After about 2 months, they enter the pancreas or abdominal cavity. Adults re-enter the intestine about 4-5 months after initial infection. Prepatent time is 9 months.
  3. *S. edentatus* L<sub>3</sub>s burrow out of the large intestine, and reach the liver via the portal veins. The L<sub>4</sub> wander in the liver for about 2 months and then work their way back through the peritoneal cavity to the colon and cecum where they survive as adult worms. Prepatent time is 11 months.
- C. Pathogenesis: Since the larval migration of each species is different, the pathologies are also different. Only *S. vulgaris* will be considered here due to the severity of lesions associated with these larvae. Pathology and clinical signs include:
  1. Acute Disease (less than 14 days) - fever, depression, colic, diarrhea, or constipation. Death may occur due to obstruction of the arteries and infarction of the bowel. Seen shortly after turnout on a heavily contaminated pasture.
  2. Lesions associated with larvae within the cranial mesenteric artery:
    - a. intestinal distress or colic due to ischemia of bowel wall.
    - b. sudden death due to rupture of aneurism close to the posterior aorta. Can sometimes rectally palpate aneurism/fibrosis of the cranial mesenteric artery.
    - c. blood profile: eosinophilia; hypergammaglobulin; hypoalbuminemia in serum during the migratory phase of the larvae.
- D. Diagnosis: Fecal egg counts indicate presence of the adult worms, but there may be extended prepatent times with no detectable eggs in the feces. Clinical signs can be quickly relieved with larvicidal anthelmintics (which kill L<sub>4</sub>s; see below).
- E. Treatment and Control: See Section V below.

WHY HAS THE PREVALENCE OF LARGE STRONGYLE INFECTIONS RAPIDLY DECLINED WITH USE OF MACROCYCLIC LACTONE ANTHELMINTICS AND BENZIMADAZOLES?

IV. Small strongyles (e.g. *Cyathostomum*, *Cylicocyclus*, *Cylicodontophorus*). Includes **more than 40 species in horses; also in elephants, pigs, marsupials, turtles.**

- A. Morphology: all have distinct buccal cavities which can be used in identification. Most adult cyathostomes are less than 15 mm long i.e. smaller than the 'large' strongyles.
- B. Life cycle: similar to *Strongylus sp.* except larvae do not migrate beyond intestinal wall; L4 larvae can arrest within gut wall and reactivate at later times. Minimum prepatent time about 2.5 - 3 months.
- C. Pathology: granulomatous colitis with numerous larvae embedded in mucosa.
  - 1. Adult worms are not major blood suckers - protein loss in feces (hypoalbuminemia).
  - 2. Emerging arrested larval stages can cause severe enteritis and diarrhea. Some develop to become adult worms but many are swept out in the manure.
- D. Clinical signs and diagnosis: anemia, persistent diarrhea, hypoalbuminemia, poor body condition. A spring rise (emergence) of arrested L<sub>4</sub> is typically seen even when horses are on pasture year-round; this co-incides with foaling season but occurs in both sexes. Egg counts may not necessarily reflect the true worm burden -- pathology is largely due to the emergence of arrested larvae. Emergence can be stimulated by anthelmintic treatment.  
Cyathostomes are very fecund and produce >75% of strongyle-type eggs in the feces of naturally infected horses. WHICH NEMATODES PRODUCE STRONGYLE-TYPE EGGS?
- E. Control: older horses (2-4 year olds) are a major source of pasture contamination, carrying the infection from one grazing season to the next. **Small strongyles have developed resistance to several drugs including benzimidazoles and pyrantel;** (at present, there is no evidence of drug resistance in the large strongyles.). Steroid therapy will reduce inflammation. Most anthelmintics are not highly effective against these arrested larvae, but new treatment regimens with fenbendazole and moxidectin appear to have some efficacy.

V. General treatment program to control strongyle infections in horses.

- A. Pasture management to prevent L<sub>3</sub> development - remove feces, keep grass short, avoid putting foals on pasture with yearlings.
- B. With high density grazing and/or young (1-3 year old) susceptible horses, anthelmintic treatment may be required every 2 months; but should be used judiciously, at strategic times, to delay the onset of drug resistance and for cost-effectiveness. Modify deworming frequency based on stocking density, climactic conditions, management strategies, etc. Under less intense conditions of infection, treat less frequently.  
For example, 2 horses on a 10 acre lot may only require 2 dewormings per year. In cool temperate regions, with evidence of winter arrested L<sub>4</sub>, springtime deworming should be considered.
- C. Use larvicidal drugs when coming off pasture and when returning to pasture to eliminate migrating or encysted larval stages. Use 2 consecutive daily treatments with oxfenbendazole OR 5 daily doses of fenbendazole OR a single dose moxidectin. Note: each of these drugs are good adulticides but have varying efficacy against encysted arrested L<sub>4</sub> stages. Many of the new-generation drugs have some limited efficacy in eliminating the encysted small strongyle larvae before they can emerge to cause clinical signs.

WHY ARE THE LARGE STRONGYLE L<sub>4</sub> KILLED EASILY WITH MOST ANTHELMINTICS BUT SMALL STRONGYLE L<sub>4</sub> ARE NOT?

- D. Monitor fecal egg counts to judge need and effectiveness. Best practices tending to this approach: **Selective Deworming based on fecal egg counts.** Considers individual horse susceptibility to infection and provides refugia to minimized drug resistance development in the small strongyles. Only 15 - 20% of horses in a herd produce 80% of ova shed on pasture.

WHY IS TREATMENT AT MANY STABLES DONE EVERY 2 MONTHS?

## Lecture # 18 continued Superfamily Strongyloidea (Contd.)

*Oesophagostomum* in cattle and pigs

*Syngamus* in turkeys

### Superfamily Metastrongyloidea (lungworms)

#### Objectives:

1. Describe the pathogenesis of nodular worm disease in ruminants.
2. Describe the life cycles of the genera *Metastrongylus* and *Aelurostrongylus* and state how these life cycles are representative of the superfamily Metastrongyloidea.

#### Outline:

##### Superfamily Strongyloidea (Contd.)

- I. *Oesophagostomum* sp.: adult worms are found in the large intestine. *O. radiatum* in cattle; *O. columbianum* and *O. venulosum* in sheep and goats; *O. dentatum* and *O. brevicaudum* in pigs. The common name is **nodular worm** because larvae become encapsulated (reactive inflammation) in the gut wall of previously sensitized hosts.
  - A. Morphology: 15 to 20 mm long, stout body, narrow anterior end relative to the rest of the body. Buccal cavity is shallow. Just posterior to the buccal cavity, the cuticle forms a distinctive fold with a ventral groove. --- Eggs: morphology not distinguishable from other strongyle or trichostrongyle eggs.
  - B. Life cycle: Prepatent time 1 to 2 months.
    1. Ruminants: free-living larval stages on pasture, the same as for trichostrongyles and strongyles; infective L3 larvae are ingested while grazing.  
Pigs: sows also acquire the infection while feeding. Egg output typically rises 6 to 7 weeks after farrowing, which increases the risk of infection to piglets.
    2. L3 larvae are encapsulated in submucosal cysts throughout the intestine. L4 may then emerge several days post infection or remain in the cysts for several months (arrest). Emerging larvae continue development to become adults in the lumen of the intestine.
  - C. Pathogenesis and Clinical signs: Usually associated with larvae and less to adult worms.
    1. Due to a reactive inflammation, nodules form around the L3 larvae, hence the name "nodular worm." Adhesions and pus-filled cysts may result.
    2. Watery, dark, fetid diarrhea; quick loss of body condition; weakness. Triggered by acute inflammatory response associated with nodule formation or with emergence of L4.

#### WOULD THIS MANIFEST AS A PATENT INFECTION?

3. Post mortem reveals a "pimple gut" due to the pus-filled, caseous nodules on the serosa.
  4. Older nodules become calcified and are not the cause of a current acute parasitic enteritis.
- D. Treatment and Control:
1. Anthelmintic treatment is effective during a clinical outbreak, but needs to be repeated due to repopulation of the gut by L4 emerging from the nodules.
  2. To control infection, treat repeatedly. Remember that arrested L4 are resistant to many drugs. Pyrantel as a feed additive may prevent nodule formation by killing the in-coming infective L3 before they can enter the gut wall.
  3. Pasture management - L1 larvae are very susceptible to desiccation; keep animals away from wet areas.

- II. *Syngamus trachea* : These ‘gapeworms’ are found in the trachea of turkey, pheasant and guinea fowl. (*Cyathostoma sp.*, is a closely related species in water fowl and raptors. Also seen in emus in North Carolina.). Parasites of upper respiratory tract.
- A. Morphology: male 2-6 mm, female 5-20 mm long; wide mouth with 6-10 small teeth; adults in permanent copula.
  - B. Life cycle: direct or indirect (via paratenic hosts e.g. **earthworm**, snail, slug). Eggs (containing infective larva) or larvae are ingested, L<sub>3</sub>s penetrate the gastrointestinal wall and migrate via the bloodstream to the lungs. Larvae may reach the trachea by 7 days with patency at 17-20 days post-infection -- eggs are coughed up, swallowed and passed in feces. The larvated eggs can persist in the environment. Eggs have an operculum at both ends.
  - C. Pathology: respiratory distress, coughing, convulsive shaking of head, partial or complete obstruction of trachea.
  - D. Treatment and Control: Eliminate intermediate hosts, rotate the range area (as in pasture rotation for ruminants). Treat with ivermectin or benzimidazoles (in feed, for 7 days).

### SUPERFAMILY METASTRONGYLOIDEA

- III. Most are parasites of the respiratory system; some affect the vascular and nervous systems. **Life cycles are indirect**: Generally involves snails, slugs, or earthworms (*Metastrongylus* in the pig). *Filaroides sp.* do not require intermediate hosts; direct infection of definitive host.
- IV. *Metastrongylus sp.*: found in the primary and secondary bronchi of pigs. “Pig lung worm”.
- A. Morphology: Adults are white, large 15 to 60 mm long, with two trilobed lips at the anterior end (distinguish from ascarids); males have long thin spicules and a well developed copulatory bursa.
  - B. Life cycle: Adult worms in bronchi produce larvated eggs that are coughed-up, swallowed, and passed out in feces. Pigs are usually infected by ingesting infected, required intermediate host, earthworms (either intact or damaged earthworm tissue containing lungworm larvae). 24 days prepatent time.
  - C. Pathogenesis: possibly associated with reduced weight gain.
  - D. Clinical signs and Diagnosis: heavy infection may cause pigs to cough when stressed/“exercised”; larvated eggs found in fresh feces.
  - E. Treatment and Control: fenbendazole and ivermectin are effective; confinement rearing prevents access to earthworms.
- V. *Aelurostrongylus abstrusus*: found in the lung of cats; occasionally in North Carolina.
- A. Morphology: up to 10 mm long, males have a copulatory bursa.
  - B. Life cycle: Adults are in the lung parenchyma; eggs cause formation of small, grayish white subpleural nodules. L1 larvae are coughed up and swallowed to pass in feces. Larvae are eaten by snails and slugs and become infective in 2 to 5 weeks. Paratenic hosts (rodents, frogs and birds) may ingest the infected mollusks, paratenic host then ingested by a cat. Ingested larvae migrate from the stomach to the lungs. Prepatent period is 5-6 weeks.
  - C. Pathogenesis: Egg masses form nodules and cause lung pathology. Adult worms cause smooth muscle hyperplasia and pulmonary artery hypertrophy; heavy infections can be fatal.
  - D. Diagnosis and Clinical signs: Chronic cough, anorexia, wasting. Thoracic radiographs show parenchymal densities, signs of respiratory disease. L1 larvae in feces or sputum (360 um long), "S" shaped tail with spine. Baermann technique for detection of active larvae in feces.
  - E. Treatment and Control: prolonged fenbendazole (50mg/kg daily for 15 days), prevent access to paratenic hosts.