

## Overview of the Order Strongylida

### Objectives:

- 1) Describe the general structure and function of the copulatory bursa of adult males of the Order Strongylida (“the bursate worms”).
- 2) Compare buccal area morphology of (a) Trichostrongyloidea, (b) Strongyloidea, (c) Ancylostomatoidea, and (d) Metastrongyloidea.
- 3) Describe the life cycle stages outside of the host for the four superfamilies listed below:
  - (a) **Trichostrongyloidea** (includes the following eight genera: *Trichostrongylus*, *Ostertagia*, *Haemonchus*, *Cooperia*, *Nematodirus* [variation in life cycle], *Dictyocaulus*, *Hyostrongylus*, *Ollulanus* [variation in life cycle]);
  - (b) **Strongyloidea** (includes the following five genera: *Strongylus*, *Triodontophorus*, cyathostomes [many different small strongyles], *Oesophagostomum*, *Stephanurus* [variation in life cycle]).
  - (c) **Ancylostomatoidea** (includes the following three genera: *Ancylostoma*, *Uncinaria*, *Bunostomum*);
  - (d) **Metastrongyloidea** (includes the following seven genera: *Metastrongylus*, *Protostrongylus*, *Muellerius*, *Parelaphostrongylus*, *Crenosoma*, *Aelurostrongylus* and *Filaroides* [variation in life cycle]).
- 4) Describe the pathogenesis and etiology of disease associated with *Ostertagia* in cattle, *Haemonchus* in sheep.
- 5) Outline the general control measures for trichostrongyles (*Trichostrongylus*, *Ostertagia*, *Haemonchus* and *Cooperia*) in cattle.
- 6) Outline the general control measures for trichostrongyles in sheep.
- 7) Describe the pathogenesis and etiology of pneumonia caused by *Dictyocaulus*.

### Outline:

- I. General morphology of the **Order Strongylida (bursate worms)**: distinctive **copulatory bursa** on adult males.
  - A. Structure
    1. Well-developed dorsal, ventral and lateral expansions of the surface cuticle at the posterior end, referred to as **lobes**.
    2. **Lobes** supported by muscular **rays**.
    3. Exception in the superfamily Metastrongyloidea where copulatory bursa is less well-developed.
  - B. Function
    1. To grasp the female worm at the vulvar site.
    2. To facilitate male spicule insertion and movement of sperm from the male cloaca to the female vulva.
- II. Buccal area (same for males and females) at anterior end of the four different superfamilies of Strongylida.
  - A. Trichostrongyloidea (most of these are found in the abomasum of small intestine of ruminants) - very small **buccal cavity** or absent.
  - B. Strongyloidea (most of these are found in the large intestine of horses) - very large **buccal cavity** with a well-developed **buccal capsule** that has a **leaf crown** at the opening and **teeth** at the base where it opens into the esophagus.
  - C. Ancylostomoidea (these are the “hookworms” found in the small intestine of a variety of mammals) - very large **buccal cavity** bent dorsally, **buccal capsule** made up of pointed **teeth** or smooth edged **cutting plates** at the anterior opening.
  - D. Metastrongyloidea (most of these are found in the lungs, others in vascular or nervous systems of mammals) - usually lacks a buccal cavity..

- III. Comparison of free-living larval stages of the four superfamilies.
- A. Egg morphology - adult females of all species in this Order (with minor exceptions noted below) lay ellipsoid-shaped eggs with smooth shells containing a morula stage embryo = “strongylid” or strongyle-type” eggs. Eggs of the superfamily Metastrongyloidea contain a larva when passed in feces or have already hatched inside the host so that first stage larvae are found in feces.
  - B. First stage larva hatches from the egg (exception - *Nematodirus* retains larva through two moults in egg) and begins feeding on microbes in manure pat or soil (exception - *Dictyocaulus* larval stages apparently do not feed). First stage larvae of the genera of Metastrongyloidea infect snails or slugs or earthworms to develop to infective L3.
  - C. Moulting twice yields infective third (L3) stage larvae. **Process requires moisture, oxygen and temperatures above 50°F.** Third stage larvae retain cuticle from the previous stage; this **sheath** adds a protective layer against desiccation.
  - D. Routes of infection.
    1. **Ingestion with pasture herbage** is the **ONLY ROUTE OF INFECTION** of importance for the superfamily **Trichostrongyloidea** genera (except *Ollulanus*) and for the superfamily **Strongyloidea** genera (except *Syngamus* and *Stephanurus*).

-----

IT IS VERY IMPORTANT TO KNOW THAT INGESTION OF PASTURE HERBAGE IS THE ONLY ROUTE OF INFECTION FOR TRICHOSTRONGYLES AND STRONGYLES WHEN ADVISING ABOUT RUMINANT AND EQUINE HERD HEALTH MANAGEMENT, RESPECTIVELY. WHY? (choose all correct answers)

- a. it can save you client money in anthelmintic costs.
  - b. it warns against using certain hay crops.
  - c. it helps evaluate risk of infection related to climate and weather.
  - d. it helps in planning management strategies for minimizing infection.
- 

2. **Skin penetration or ingestion** is used by the free-living infective larvae of genera in the superfamily **Ancylostomoidea**. Some species use a lactogenic (transmammary) route of infection as well, this is especially important in canine hookworm disease (see below).
3. **Ingestion of infected intermediate hosts** (snail, slug or earthworm) or paratenic hosts containing infective larvae is used by the genera of **Metastrongyloidea**.

## SUPERFAMILY TRICHOSTRONGYLOIDEA

- I. *Trichostrongylus* sp.: *Trichostrongylus axei* in the stomach or abomasum of primarily ruminants and horses; *Trichostrongylus colubriformis* in the small intestine of ruminants.
  - A. Morphology: Adults are less than 7 mm long, little or no buccal cavity, produces very few eggs.
  - B. Pathogenesis: Clinical disease of watery diarrhea requires more than 100,000 adult worms, which is many more than required to produce disease with the other trichostrongyles. Causes plaques of eroded epithelium in stomach or duodenum. Generally not seen as the major cause of disease in ruminants since pasture conditions and poor management allowing such burdens of *Trichostrongylus* will have permitted much more severe disease due to *Ostertagia*, *Haemonchus* or *Cooperia*. *T. axei* will infect horses and is more pathogenic causing catarrhal gastritis in this host, such that it is advised **not** to co-graze horses with sheep or goats. 3 week prepatent time.
  
- II. *Ostertagia ostertagi* - most important helminth parasite of cattle in USA (llamas to lesser degree). *Teladorsagia circumcincta* - sheep and goats. Common name is the brown stomach worm.
  - A. Morphology: 7 to 14 mm long, brown in color, broad shallow buccal cavity, female has vulvar flap and produces less than 100 eggs/day/worm.
  - B. 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.
  - C. Clinical signs: **Anorexia** and **diarrhea**, decreased weight gain or weight loss, evidence of protein deficiency in matrix osteoporosis 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. First year grazing calves require prolonged exposure to develop immunity.
  
- III. *Haemonchus contortus* - most important helminth parasite of sheep and goats in USA (camelids to lesser degree). *Haemonchus placei* - cattle. Common name is the barber pole worm.
  - A. Morphology: 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.
  - B. 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.
  - C. Clinical signs: black or tarry feces (not diarrhea), pale mucus membranes - FAMACHA, 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. Sheep and goats fail to develop solid immunity at any age.

- IV. *Cooperia* sp.: *C. punctata* and *C. pectinata* are pathogenic for cattle, whereas *C. oncophora* infects cattle, sheep and goats but is rarely pathogenic. *Cooperia curticei* infects sheep.
- A. Morphology: 5 to 10 mm long, cuticle at anterior end is inflated and has transverse striations.
  - B. Pathogenesis: adults lie deep in the mucosa of the **proximal small intestine**. *Cooperia punctata* and *C. pectinata* are associated with enteritis, catarrhal inflammation. Some areas of the world are seeing ivermectin resistant *Cooperia*. *C. oncophora* that becomes resistant also associated with more clinical disease. 3 weeks prepatent time.
  - C. Clinical signs: anorexia and diarrhea that is very similar to *Ostertagia* but not usually a sole cause of clinical disease. Subclinical disease recognized as weight gain after deworming. But recent reports of primarily *Cooperia* infections in cattle in northern Florida and other locations indicate that *Cooperia* infection can be a primary cause of disease. Antemortem, fecal culture to obtain L3 is required to determine genus and species of Trichostrongyloidea. Postmortem, examine duodenal mucosal scrapings under magnification for adults.
- V. Ecology of transmission of trichostrongyle infection in ruminants is entirely based on pasture grazing.
- A. Adult worm population determines degree of pasture contamination.
    1. Source of adult worms is infective larvae ingested from pasture by a susceptible host.
      - a. infective larvae require warmth, air and moisture to **develop** from eggs; L3 requires warmth and moisture for movement on grass that is necessary for being ingested during grazing = **transmission**; the best time of the year for **development** and **transmission** on pasture is in the summer and early fall in cool climates (North in USA) and in the winter and early spring in semi-tropical climates having hot/dry summers (South in USA).
      - b. in the case of *Ostertagia* when ingested L3 become L4 that **do not arrest** and progress directly to become adults in large numbers. This is called **Type I Ostertagiasis**.
      - c. infective larvae differ by genera in ability to survive cold on pasture – *Nematodirus*, *Cooperia* and *Ostertagia* overwinter well.
    2. Source of adult worms can also be arrested L4 stage larvae in gastric glands of the abomasum
      - a. L3 ingested in Fall of the year when temperatures are declining in cool temperate regions, or L3 ingested in Spring of the year preceding dry season in warm temperate to semi-tropical regions, tend to arrest as L4.
      - b. L3 ingested when abomasum is already populated by adult worms tend to arrest as L4.
      - c. L4 become activated to develop to adults in early spring in the North, whereas in the South they become activated to develop to adults in the early fall. When this leads to large numbers of adult *Ostertagia* it is called **Type II Ostertagiasis**.
      - d. removing an existing adult worm population allows L4 to become a new adult worm population. **It is important to know if anthelmintic you are using is active against L4.**
      - e. onset of lactation stimulates arrested L4 to become adult worms. **Periparturient haemonchosis is very important in ewes of all ages. Periparturient ostertagiasis is important in cows usually at first calving only.**
  - B. Host susceptibility to infection determines host role in pasture contamination.
    1. **Individual variation in susceptibility to infection. 15 – 20% of herd carry most of the adult worm burden and produce ~80% of eggs.** This can be the basis for selective deworming in livestock and provide refugia worm population to prevent drug resistance.
    2. Age
      - a. cattle that are more than 2 years old generally **do not** carry large numbers of adult worms. The value of deworming adult cattle must be weighed carefully.
      - b. sheep often fail to maintain resistance to infection even as adult animals. **It is important to deworm ewes at lambing and adults on heavily contaminated pastures. Lambs are always at risk on pasture.**

3. Premunition or concomitant immunity
  - a. existing adult worm populations inhibit new infections and L4 becoming adults.
  - b. removal of adult worms often requires repeated treatments due to repopulation by L4 becoming adults if not using an anthelmintic that kills L4 as well as adults (eg. Levamisole is an adulticide only, most newer drugs kill trichostrongyle L<sub>4</sub> and adults).

## VI. Control

- A. Cattle – treatment is primarily of calves/long yearlings less than 1.5 years old.
  1. Treatment when there is a high risk of disease due to large number of infective larvae on pasture is called “tactical treatment”.
    - a. Treat and move
    - b. Treat and repeat in 2 -3 weeks to prevent patentcy (3 weeks). Longer between treatments if anthelmintic has residual effect (macrolides – ivermectin, etc.)
  2. Treatment to prevent build-up of infective larvae on pasture from egg-producing adult worms (adults can be developing from arrested L4 or recently ingested L3) is called “strategic treatment”.
    - a. Treat calves and long-yearlings in spring in cool temperate regions (including NC) 3 - 6 weeks after turn-out and repeat in 3 weeks. This makes these hosts “death traps” or “vacuum cleaners” for the few L3 that survived over-winter and will be ingested during spring grazing. If these few L3 are removed from pasture without infecting calves and generating adult egg producers, then pasture contamination will be minimized during summer grazing.
    - b. Treat calves on pasture in fall when dry season ends in South.
    - c. Treat calves with adult and L4 killing anthelmintic in late spring to early summer at start of dry season in South if they are to be grazed in fall. This will remove arrested L4 thereby preventing development of egg-laying adults which would contaminate wet fall and winter pastures with L3 at the end of the dry summer.
    - d. Treat calves with adult and L4 killing anthelmintic in the late fall at start of cold winter season in North if they are to be grazed in the spring. This will remove arrested L4, thereby preventing development of egg-laying adults which would contaminate lush spring pastures with L3 at the end of the cold winter.
  3. Treat first calf heifers at start of lactation. Older cows usually do not need to be treated.
- B. Sheep and Goats – must include treatment of adult sheep and goats.
  1. FAMACHA: selective deworming based on clinical signs of anemia. **Purpose is to slow drug resistance development by preserving “refugia” populations of drug susceptible *Haemonchus*.**
- C. Anthelmintics: Macrocytic lactones (macrolides) such as ivermectin, moxidectin, doramectin, eprinomectin all kill adult and L4 stage forms of the trichostrongyles (but not L4 of strongyles of horses). Benzimidazoles such as albendazole and fenbendazole kill adult and L4 forms of trichostrongyles (but not L4 of strongyles of horses). Formulations include oral, injectable, pour-on and sustained release bolus for different anthelmintics.
- D. In cool temperate regions, such as North Carolina and further north, do fecal egg counts to assess strategic deworming program in late summer or early fall when adult worm populations would be expected to be highest.

- VII. *Dictyocaulus* sp. (lung worm): *D. viviparus* - cattle, *D. filaria* - sheep and goats, *D. arnfieldi* - equine. (host species specific)
- A. Morphology: 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.
  - B. 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 in 5 days. 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.
  - C. Clinical 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.
  - D. Control: Vaccination with attenuated L3 larvae is used in Europe (not available in USA due to limited demand). Most broad spectrum anthelmintics are effective in treatment. Avoid wet, low lying pastures. Do not graze horses with donkeys. Donkeys often support patent infections whereas horses do not have patent infections but they can suffer pathological lesions. *Dictyocaulus* does NOT require an intermediate host. Infective larvae are free on pasture like trichostrongyles. The infective larvae of *Dictyocaulus* are very susceptible to desiccation, which limits their geographic distribution. Treat at first signs of respiratory disease.