Superfamily STRONGYLOIDEA

- horses
  strongyles of large intestine
- ruminants
  nodular worms of large intestine
- swine
  kidney worms and nodular worms
- birds
  tracheal “gapeworms”
Morphology

- large, thick body
- large buccal cavity
- leaf crown
- teeth (+/-)
Male worm:
Copulatory bursa is well-developed
Horses:

**LARGE** and **SMALL** strongyles in large intestine
Large intestine mucosal surface
Superfamily Strongyloidea

• Life cycle
  – direct (strongyles of horses)
  – a few exceptions: earthworm as transport host
    • e.g. kidney worm of pigs
    • e.g. tracheal worm of birds
In horses:

- **Strongylus** species (large strongyles)
  - *S. vulgaris*
  - *S. edentatus*
  - *S. equinus*
- **Triodontophorus** species
- Cyathostomes (small strongyles)
Life-cycle of *Strongylus vulgaris*

- L3 ingested when horse is grazing on infected pasture (only route of infection). Just like trichostrongyles in ruminants.

- Larva penetrates intestinal wall (~1-7 days)
Life-cycle of *Strongylus vulgaris*

- migrates in vessel wall up to the cranial mesenteric artery or aorta taking 8-21 days

- L₄ break into blood stream and carried back to colon & cecum, develop to adult stage during 2-4 months

- total prepatent period ~6 months
Strongylus equinus

• 25 to 45 mm long
• Teeth: 3 pointed
• L3s migrate to & within liver ~ 2 months enter the pancreas or abdominal cavity, then return to intestine
• Prepatent period is ~ 9 months
**Strongylus edentatus**

- 25 to 45 mm long (like *S. equinus*)
- Teeth: None.
- L3s migrate to & within the liver, 2 months, then return via peritoneal cavity to the intestine
- Prepatent period is ~ 11 months
Prepatent periods of large strongyles

Large strongyles are easily controlled and not found often. Why?
Remember these strongyles do not arrest, no hypobiosis. Current drugs effective against all stages. Hint: how often would you need to treat to prevent patency?
Signs/Pathology: *Strongylus vulgaris*

Acute disease: (soon after turnout)
- fever, depression, colic, diarrhea/constipation
due to infarction/obstruction of arteries and inflammation from entering larvae

Chronic disease:
Episodes of colic, with/without sudden death, from thromboemboli and/or rupture of an aortal aneurism
ISCHEMIA

INFARCTION
Felt on rectal palpation: Hypertrophy of arterial cranial mesenteric aneurism. walls
Pathology

COLIC:
- blood flow disrupted
- thrombo-embolism
- mechanical damage to intestine
- altered motility
- allergic irritation
- innervation abnormalities
Pathology

Blood chemistry:
- eosinophilia
- hypergammaglobulinemia
- hypoalbuminemia, esp. with migratory larvae
Diagnosis

- Eggs in feces, but same type as small strongyles

......BUT IF..... clinical signs + fecal -

What’s your explanation?
Cyathostomes, the small strongyles

- >40 species in large intestine of horses
- also species specific to elephants, pigs, marsupials, turtles
- distinct buccal capsules
- 'smaller' than the large strongyles, ~15 mm
Cyathostomes

- Larvae do **NOT** migrate beyond the large intestine.
- L₄ can arrest in intestinal mucosa and reactivate at later times, especially when the horse is stressed or in spring time/foaling season.
- Prepatent period is ~2.5-3 months without arrest.
Cyathostomes

- granulomatous colitis from larvae in mucosa
- major loss of protein, **not** blood
- re-emerging larvae = enteritis + diarrhea

Poor body Condition!
Cyathostomes

• Control:
  – All ages of horses can be source of contamination
  – cyathostomes produce >75% of eggs in the feces
  – arrested larval forms are resistant to many drugs
  – drug resistant strains have evolved
  – steroid therapy in face of clinical signs
  – drugs: “drug dejour” combinations and reformulations, plus new compounds
Treatment & Control

- pasture management
  - remove manure; ‘drag & dry’
  - keep grass short
  - avoid mixing foals with yearlings (Why?)

- monitor fecal egg counts on individual horses as a basis for selective deworming
Control program against strongyles

• deworming schedule - strategic
  – during grazing season
  – with larvicidal drug at beginning and end of grazing period

• use a larvicidal drug q 6 months to kill emerging and arrested L4s
  e.g. fenbendazole? Ivermectin?, moxidectin

Any drug that kills metabolically active larvae will kill adults
Eggs per Gram (EPG)

- Strongyle-type eggs
  - Genus??
- Variability in results
- Large number = Large number of adult worms
- Interpretation of data
  - Horses >200 epg
  - Goats/sheep >500-1000 epg
  - Cattle >100-200 epg
EPG tells you the horse is……

- Infected with mature strongyle-type adult worms; probably has prepatent stages too
- Is contaminating the environment with eggs (which will become infective larvae)
• Need to treat; if recently treated, consider drug resistance or sources of infection (or need for repeat treatments)
• Control measures e.g. pasture management, deworming schedule, etc.
• Are other horses in stable infected?
“So, what’s a good deworming schedule for horses?!?!?”
Every 2-3 months?
The factors to consider are........
Some common dewormers FYI:

- Pyrantel
  - STRONGID-C (Pellets; tartarate salt; daily)
  - STRONGID-T (Paste; pamoate salt)
- Fenbendazole
  - PANACUR
- Ivermectin
  - EQVALAN paste/liquid, DURAMECTIN, ZIMECTIN (with praziquantel)
- Moxidectin
  - QUEST GEL
- Abamectin/Praziquantel
  - EQUIMAX
Superfamily Strongyloidea (cont)

- NODULAR worms of cattle, sheep, goats, swine - *Oesophagostomum*

**Adult worms:**
- in large intestine
- 15-20 mm long, stout
- cephalic collar at anterior end
**Oesophagostomum**

- L3s are ingested while grazing
- Larvae are within encapsulated cysts, *NODULES*
**Oesophagostomum**

- Pathogenesis: due mostly to larvae
  - adhesions, calcified cysts
  - Enteritis/diarrhea when L4s emerge i.e. acute inflammatory reaction
Oesophagostomum in PIGS

- Ingestion of infective larvae
- Transmission of infection to piglets - due to peak egg production by sow at ~6 weeks after farrowing
Syngamus trachea, ‘gapeworms’

- turkey, pheasants, fowl, captive emus in NC
- 2-20 mm long adults, in permanent copula
Syngamus trachea, ‘gapeworm’

- life-cycle is direct or via a transport host (earthworm, snail, slug)
- larvae migrate, enter the airways, mature in lungs; PPP ~3 weeks
- eggs are coughed up and swallowed
- bi-operculated egg in feces
Order Strongylida

Superfamilies:
- Trichostrongyloidea
- Strongyloidea
- Metastrongyloidea = infect lungs
- Ancylostomatoidea
Not enteric

- Metastrongyiles of respiratory system, but some affect the vascular and nervous systems.
- Life-cycle can be:
  - INDIRECT requiring snails, slugs, earthworms
  - Direct (*Filaroides* sp. not covered here)
Metastrongylus - PIGS

- adults worms are large, white, in bronchi

- larvated eggs are released from the female in the bronchi, coughed up, swallowed and passed out in feces.
**Aelurostrongylus abstrusus**

- Adults in lung parenchyma of CATS
- Occasionally seen in N.C.

Grayish subpleural nodules
**Aelurostrongylus abstrusus**

- Cat infected by eating
  - infected snail/slug
  - infected paratenic host e.g. bird
- Larvae migrate from stomach to lungs
- Prepatent period is 5-6 weeks
- L1 coughed up, swallowed, in feces
Pathogenesis:

Egg masses - small, grayish subpleural nodules

Adult worms - muscle hyperplasia, pulmonary hypertrophy
Adult worms in cross section

- Intestine
- Uterus
- Cuticle
Aelurostrongylus abstrusus

*Heavy burden can be fatal

Diagnosis:
- Respiratory signs
- Thoracic radiographs

![Image of lung tissue](image1)

![Thoracic radiograph](image2)

- Parenchymal densities
Aelurostrongylus abstrusus

Diagnosis:
- L1 larvae in feces or sputum
- Baermann or SEB direct smear
  Spine at posterior end
Aelurostrongylus abstrusus

Control:
fenbendazole, ivermectin
restrict access to paratenic hosts