Small Strongyles

Nematodes : Strongylids : Strongyles

Small Strongyles

Take Homes

Small Strongyles

- Currently, most common and important helminth of Horses
- Is a Pasture-borne Nematode (ingest L3s while grazing)
- Adult worms in the lumen of the large intestine and cecum
- Pathology is due to encysted or reactivated L4's in the walls of the large intestine and cecum.
- Two primary forms of Pathology
  - Granulomatous colitis: severe immunological response to encysted L4s
  - Larval Cyathostominosis: Sudden eruption of large numbers of reactivated L4s from the gut wall causes intense mucosal damage and inflammatory RXN.
    - Sometimes initiated by the post-treatment loss of Premunition.
- Clinical Signs:
  - Presenting: Persistent diarrhea, weight loss, unthriftiness
  - Clues: young grazing horse, ventral edema, adult worms on feces/ palpation glove
- FEC:
  - Doesn't correspond to pathology as pathology is prior to PPP.
  - For Control: Helps identify individuals with high contamination potential (>500 epg); Supports refugia efforts
  - FECRT: Monitor for dewormer resistance
- Control:
  - Various deworming strategies & pasture management
  - Horses >3 years
    - Focus on small strongyles.
    - Target high contaminators: >500 epg
    - Target L4s with larvicide at end of grazing season
    - FECRT every 3 years

Small Strongyles
General

General

- aka Cyathostomins; Cyathostomes; small red-worms
- Over 40 species of small strongyles of horses
  - (ex. *Cyathostomum spp., Cylicocyclus spp., Cylicostephanus spp.*, etc.)
- Most common & important parasites of horses
- Cyathostominosis affects horses at any age.

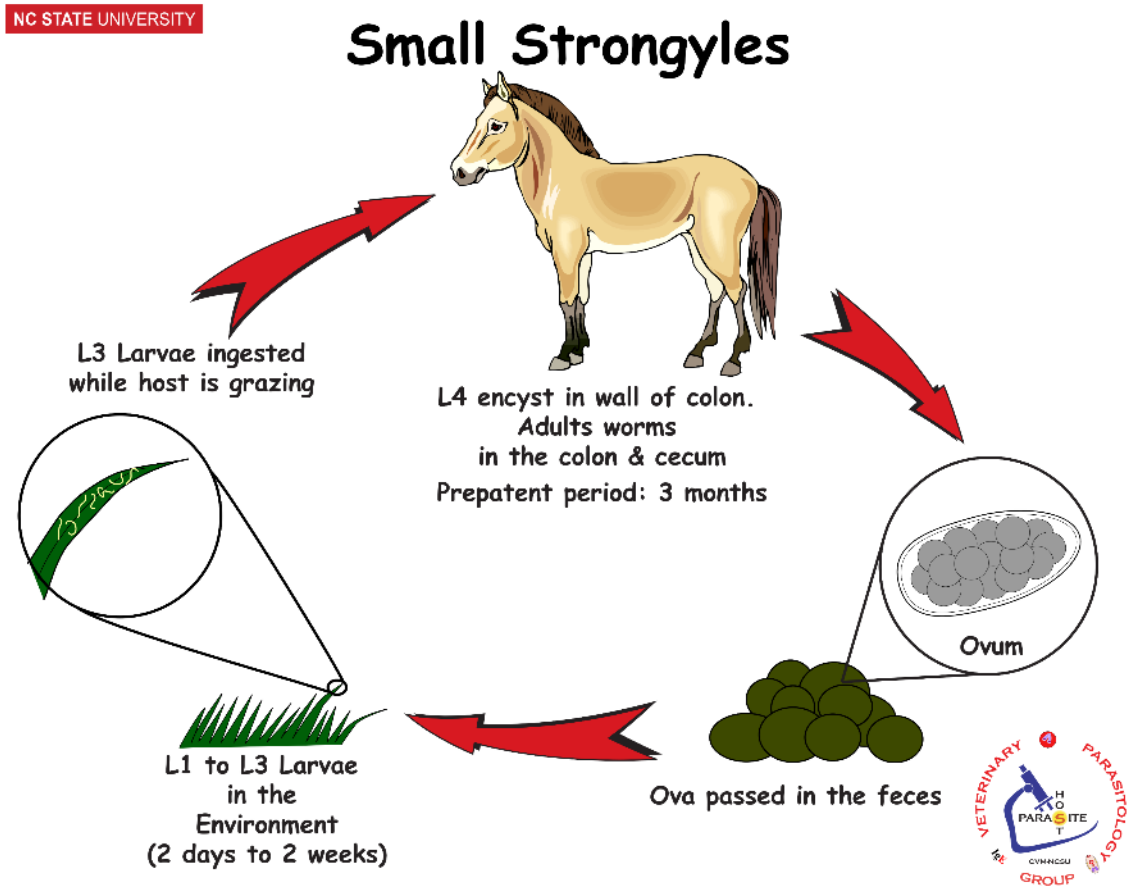
www.ivis.org/library/clinical-companion-of-donkey-2nd-edition/parasitology

Small Strongyles

Life Cycle

- Typical Pasture-borne Nematode Life Cycle
  - Ingestion of L3s on-pasture while grazing
- PPP: 2 – 3 months (w/o arresting)
- LC specifics
  - Adult worms in lumen of cecum / large intestine
  - L3s Migrate to the mucosa of the large intestine & cecum
    - 2 options:
      - Do not arrest; but develop directly through L4 stage to adults in lumen.

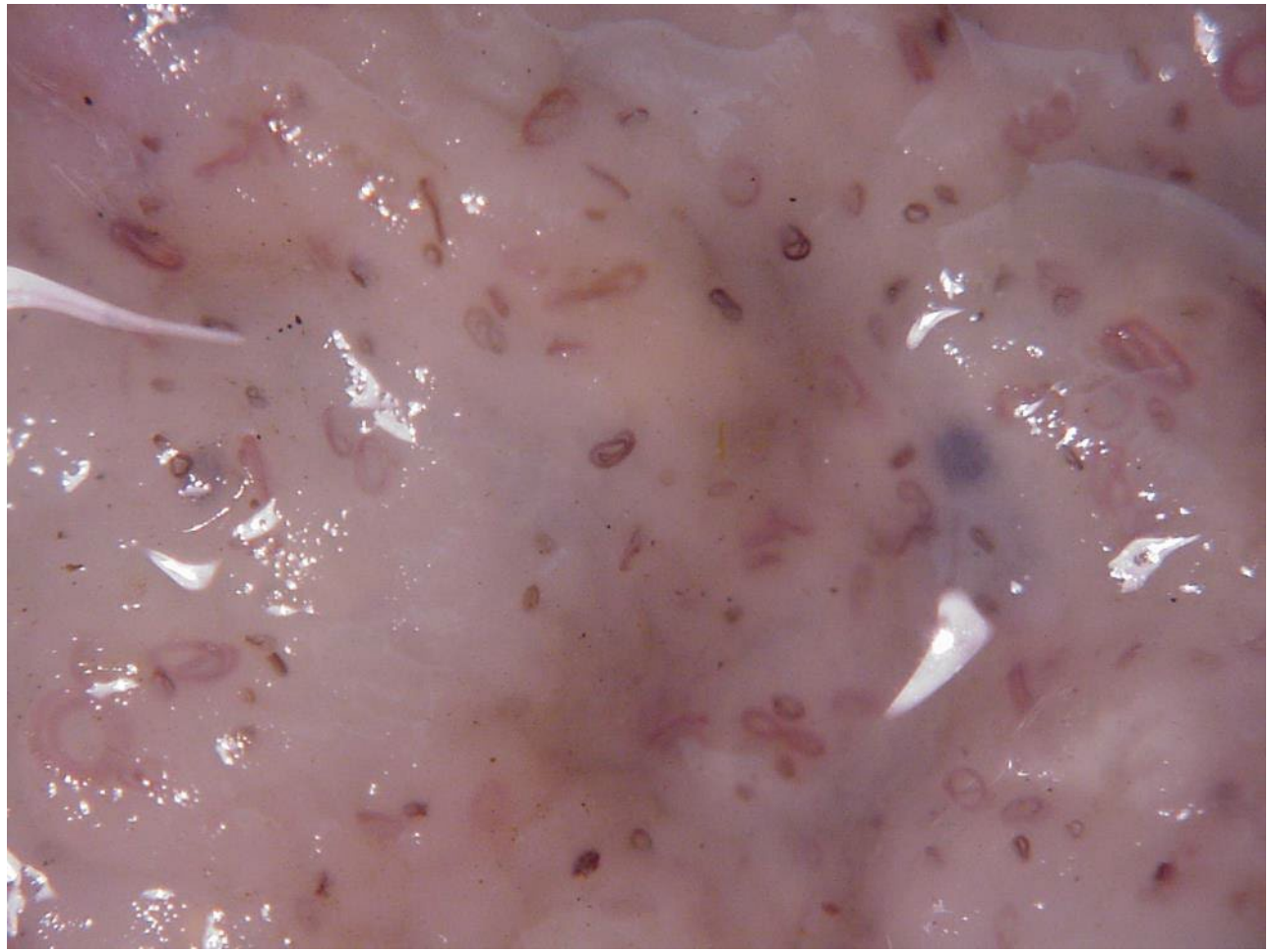
      OR
      - Arrest and Encyst in the wall of the gut and develops to L4s
  - Encysted L4s
    - Becomes arrested (aka hypobiotic) for an undetermined period
    - Various triggers reactivate the L4s to excyst
    - Enter the lumen of the gut and develop to adult worms



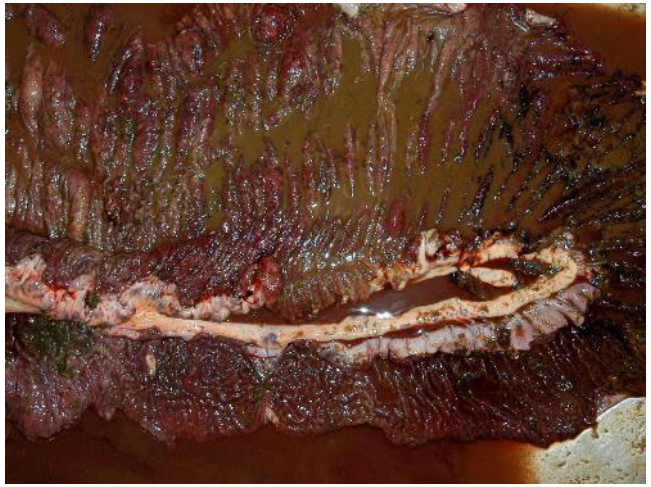
Small Strongyles

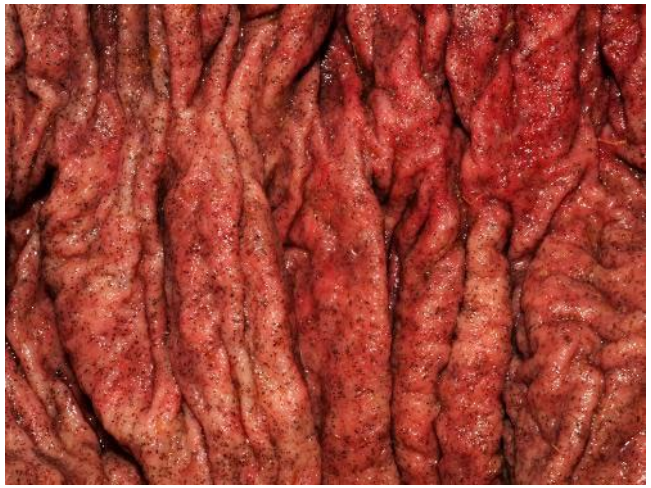
Pathology

- **L4's are the cause of pathology**
  - ➔ Diagnosis: may be low FEC during pathology



Small Strongyles





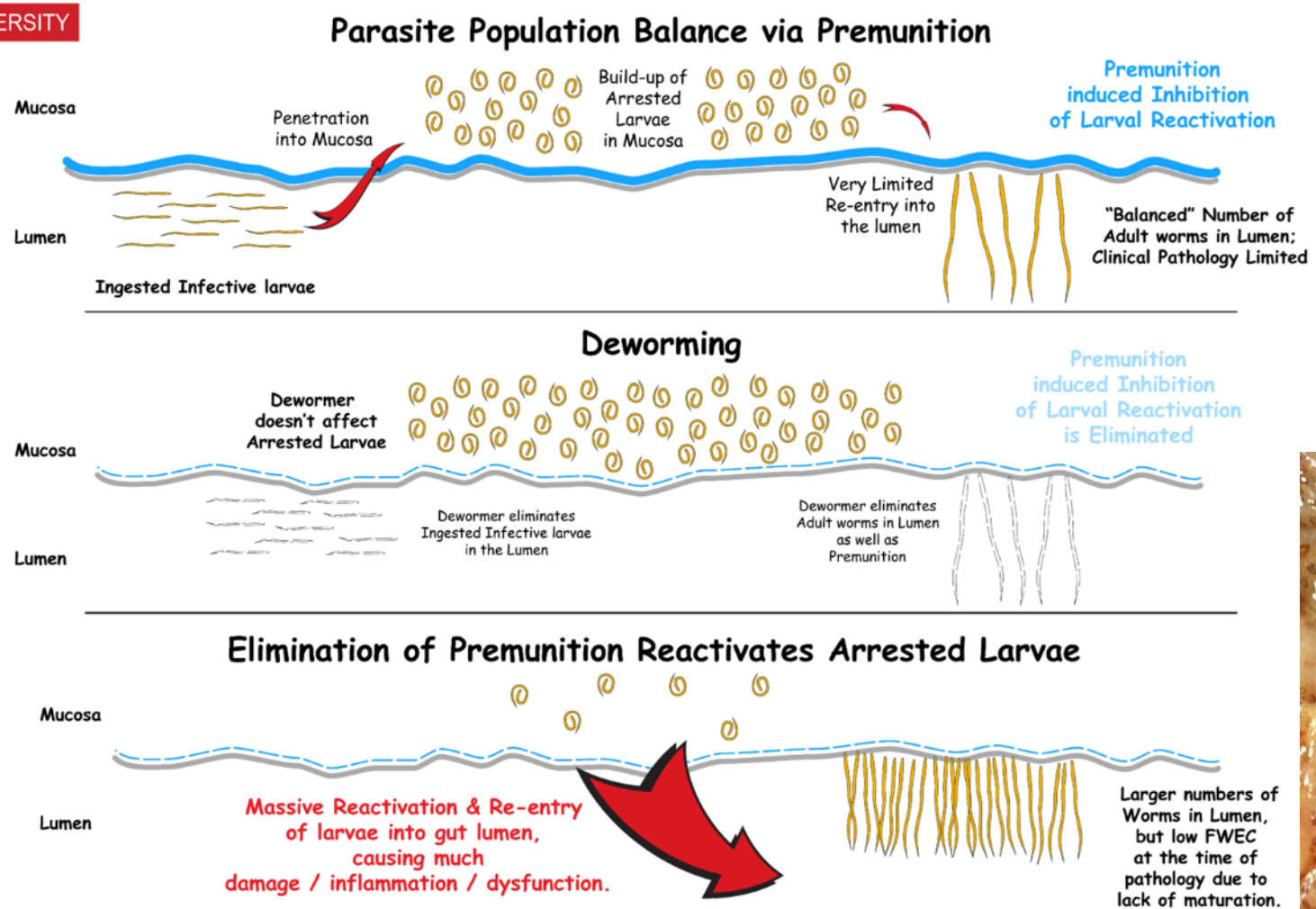
Forms of pathology

- **Granulomatous colitis:** Severe immunological response to a large population of encysted larvae within the mucosa.
- **Larval Cyathostominosis:** "Clinical disease … results from the sudden eruption of large numbers of small strongyle larvae encysted in the lining of the large intestine into the intestinal lumen. Severe damage to the intestinal mucosa ensues, which significantly compromises the function of the intestinal lining and allows major loss of body fluid and serum proteins."[1] The resulting inflammatory reaction and damage to the mucosa becomes extreme and potentially fatal. Larval Cyathostominosis causes severe diarrhea, colic, and mortality rates as high as 50%.
  - Can occur post-deworming by eliminating the premunition protection. (Elimination of adult worms often triggers the reactivation of encysted L4s)
    - ➔ Treatment: Include anti-inflammatories when deworming clinical horses

[1] Lyons, ET; JH Drudge; SC Tolliver. 2000. LARVAL CYATHOSTOMIASIS. VETERINARY CLINICS OF NORTH AMERICA: EQUINE PRACTICE. 16: 501-513.

Small Strongyles

NC STATE UNIVERSITY

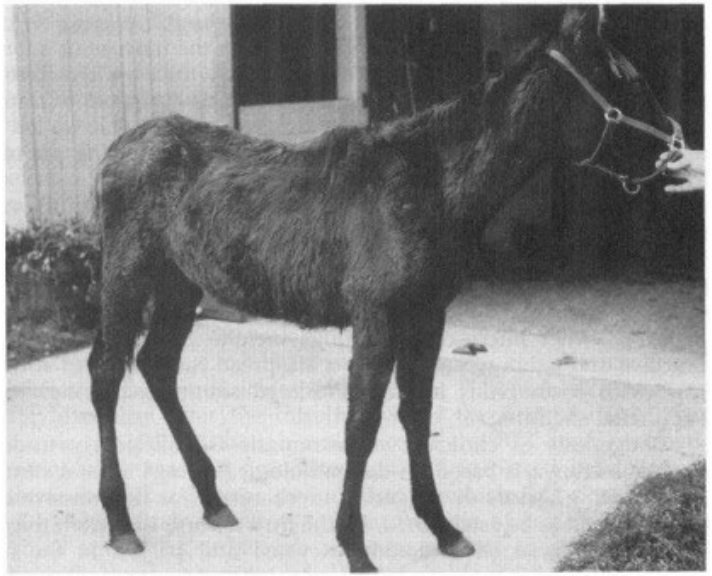


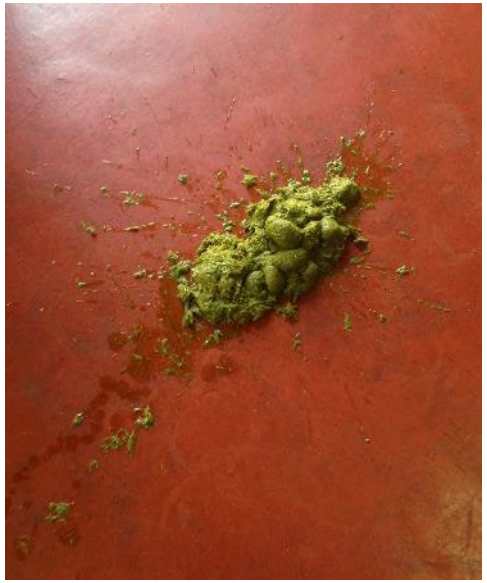
- **The Loss of Premunition is a serious concern when treating foals for Small Strongyles**
  - Severe Larval cyathostominosis can occur post-deworming by eliminating the premunition protection. (Elimination of adult worms often triggers the reactivation of encysted L4s)
    - ➔ Treatment: Include anti-inflammatories when deworming clinical horses

Small Strongyles
Clinical Signs, Diagnosis

Clinical signs

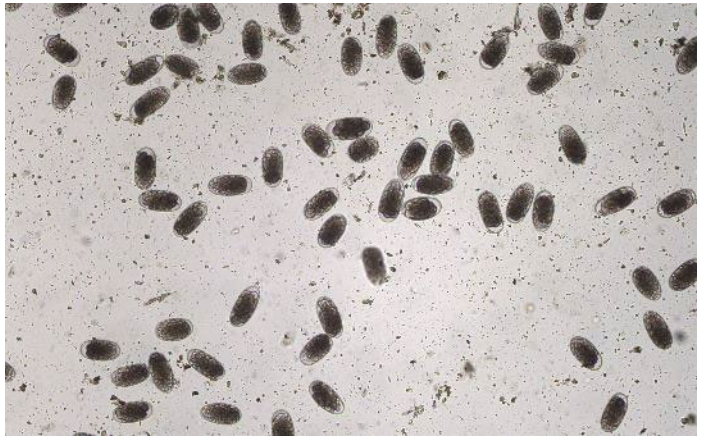
- persistent diarrhea → dehydration → death
- hypoproteinemia → ventral edema
- Weight loss, poor body condition, unthriftiness
- Other clues:
  - Young Horses on Pasture
  - End of Grazing Season
  - Poor deworming efforts / resistance problem
  - Adult worms on feces or palpation glove





Diagnostics

- McMasters
  - May confirm small strongyle issue
  - Identifies pasture contaminators
    - Low contaminators: 0-200 epg
    - Moderate contaminators: 200-500 epg
    - High Contaminators: >500 epg
  - Current suggested cut off: FEC =/> 500 epg
    - To maintain refugia



Small Strongyles
Control

Strategic Deworming

- PPP interval deworming – BAD!
- Fecal Egg Counts (FEC) => test & treat
- Event Deworming
  - Beginning of Grazing Season
    - Pre-season contamination potential (reset FEC to "zero")
    - And Target "Spring Rise"
  - End of Grazing Season
    - Maximum accumulated infection
  - Target Arrested L4s
    - Moxidectin, Fenbendazole [Power-Pack]

Dewormer resistance

- Resistance is an important and increasing issue.
  - Current resistance against fenbendazole and pyrantel.

Avoid Infective L3s

- Rotational Grazing
  - Run-away from L3s
  - Rest pastures until L3s die-off
- Don't co-graze naïve young with older horses
- Compost Feces
- Take hay off contaminated pasture

AAEP Guidelines for Mature Horses (> 3 years old)

- Focus on control of small strongyles.
  - FEC to target horses with a high strongyle contamination potential. (>500 epg)
  - Focus anthelmintic treatments during seasons of peak transmission.
    - usually spring and fall
  - Include a treatment effective against encysted L4s at a time when the mucosal burden is at its peak. (larvicidal dewormer)
    - Typically, this occurs at the end of the grazing season
      - Northern (including NC): Treat in Fall
      - Deep South: Treat in spring
- Evaluate the efficacy of the dewormers at least every three years using the Fecal Egg Count Reduction Test (FECRT).
  - To check efficacy of dewormer → anthelminthic resistance

https://aaep.org/resource/internal-parasite-control-guidelines/

Large Strongyles

Nematodes : Strongylids : Strongyles

Large Strongyles

Take Homes

Strongylus vulgaris

- Historically, the most important/pathogenic helminth of Horses
- Is a Pasture-borne Nematode (ingest L3s while grazing)
- Adult worms in the lumen of the large intestine and cecum.
- L4s constantly migrate & do not encyst and arrest.
- Pathology is due to migrations of the L4s in the intima of the arterial system of the LI & Cecum.
- 4 months of L4 migrations within the intima of the mesenteric arterial system cause inflammation, endarteritis, thrombus formation, leading to thromboembolisms, intestinal infarctions, acute peritonitis, and death
- Clinical Signs & Diagnostics
  - Signs of intense colic
  - Presenting: Colic w/ appearance of peritonitis
  - Clues: Enlarged CMA on rectal palpation, hyperemic mucous membranes, fever
  - Diagnostics: Abdominocentesis confirms peritonitis
  - Confirmation: Exploratory laparotomy confirms NSII
- FEC doesn't correspond to pathology as pathology prior PPP. Large Strongyles have low fecundity v/s small strongyles
- Treatment:
  - Mild colic: larvicidal deworming therapy may be possibly.
  - Intense Colic: Laparotomy to assess infarctions and prognosis. Intestinal Resection if prognosis is good. Deworm after recovery from surgery
- Control:
  - Quarantine protocols to prevent establishment on a farm
  - Basic pasture management
  - Base-line deworming 1-2X/year to eliminate *S. vulgaris* from farm
    - (6-month PPP -> can't reach adulthood to contaminate pastures; also, don't arrest -> all stages susceptible to deworming all the time)
- Issue: Re-emerging in Europe

Large Strongyles

General

- Also known as blood worms, palisade worms, sclerostomes, or red worms.
- Three Species of Large Strongyles
  - *Strongylus vulgaris, Strongylus equinus, Strongylus edentatus*
  - The adult worms are in lumen of the colon & cecum,
    - Are "mild" blood-feeders but not to the point of anemia.

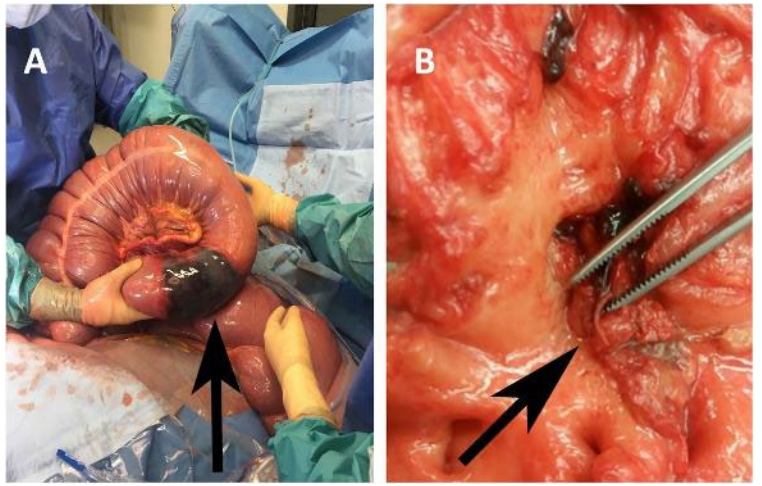
Strongylus vulgaris

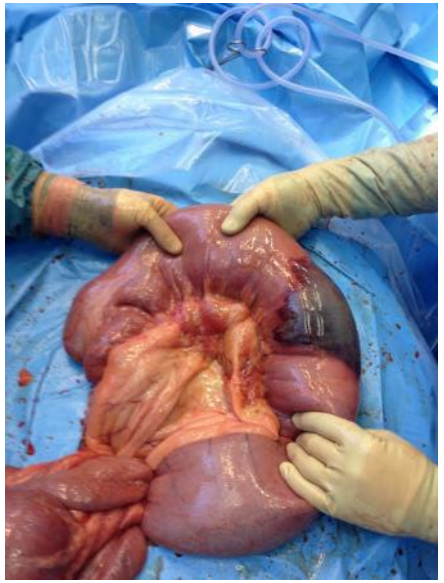
Strongylus equinus

Strongylus edentatus

Strongylus vulgaris

- Historically (pre-ivermectin), was the most important helminth of horses.
  - "Equine Bloodworm" had a prevalence of 80 – 100%,
  - But very low prevalence now.
  - In the past, Verminous thrombi & emboli may have accounted for most of the fatal and non-fatal equine colic cases.
  - Cases often present as Acute Peritonitis / Colic.

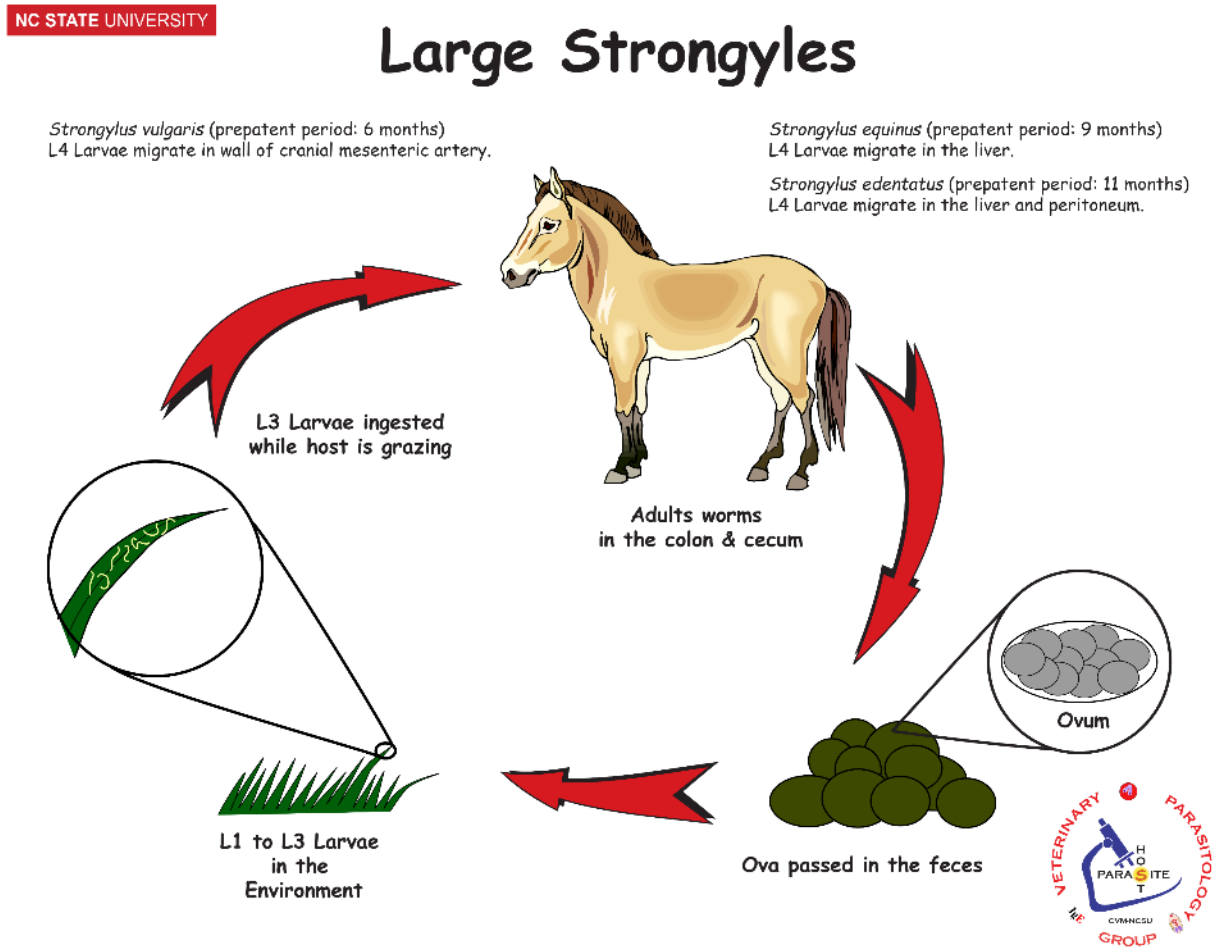




Strongylus vulgaris

Life Cycle

- Typical Pasture-borne Nematode Life Cycle
  - Ingestion of L3 on-pasture while grazing
- PPP: 6 months
- LC specifics
  - Adult worms in lumen of cecum / colon
  - Eggs passed in the feces
  - L1 → infective L3 on pasture
  - L3s ingested & migrate
    - Penetrate the submucosa of the cecum and colon
    - Molt to L4s



Strongylus vulgaris

Fibrin tracks in the aorta due to migrating larvae of Strongylus vulgaris

LC specifics continued

- L4s migrations
  - Penetrate into the intima of arterioles
  - Migrate within the arterial intima (b/w endothelium & smooth muscles) and up larger & larger vessels
    - Causes "threadlike elevations" of the arterial intima
  - Eventually to the cranial mesenteric artery (CMA)
    - Sites of predilection (CMA & ileo-cecal-colic artery)
    - Migrate, develop, & enlarge for about 4 months
      - **Period of greatest Pathology**
    - (a few may reach the aorta & may cause aortic aneurysm)
  - Molt to L5s (immature adults), and migrate out of the arterial intima into the blood stream
- L5s, within the blood stream, are swept back down the arterial tree to smaller vessels.
  - In the walls of cecum and colon, L5s occlude arterioles and form abscesses
  - Abscesses rupture releasing L5s into the gut lumen.
  - L5s become sexually mature adults and produce eggs.

Strongylus vulgaris
Pathology

In general:

- Pathology is caused by migrating L4s
  - L4s do not arrest
- The high pathogenicity of *S. vulgaris* is mainly due to the L4s migrating for about **4 months**, in the mesenteric arterial system.
  - Causing arterial damage, inflammation, and thrombus formation.
  - Potentially causes fatal thromboembolism, intestinal infarction, and acute life-threatening peritonitis.

Severity of Pathology

- Generally, Proportional to # of L3 ingested.
  - Few L3s => few occlusions => chronic intermittent colic but recovery
  - Many L3s => many occlusions => fatal infarctions

Experimental Infections:

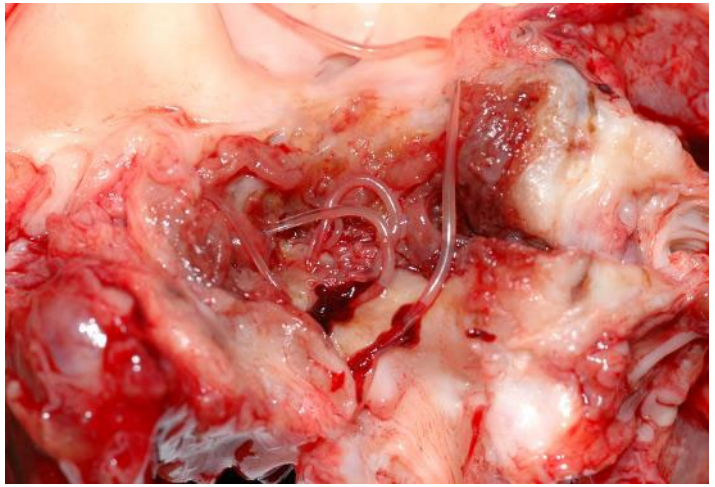
- L3 dose of approx. 750
  - Non-Fatal
  - Colic, intermittent (beginning 2 weeks PI)

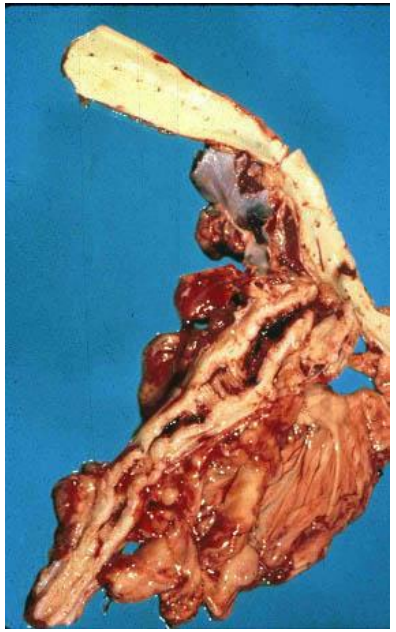
Experimental Infections:

- L3 dose of approx. 1000+
  - Fatal within 3 weeks PI
  - Intense colic
  - Severe pathology due to infarctions / ischemia of the gut wall.
    - Intestinal necrosis => perforated intestine => fatal peritonitis

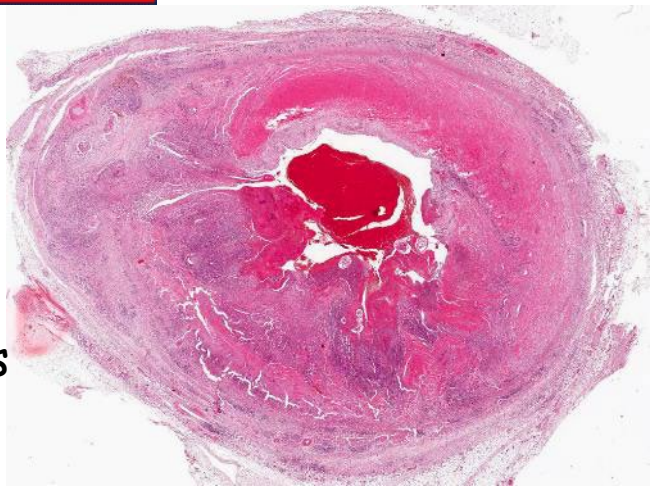
Strongylus vulgaris
Pathology

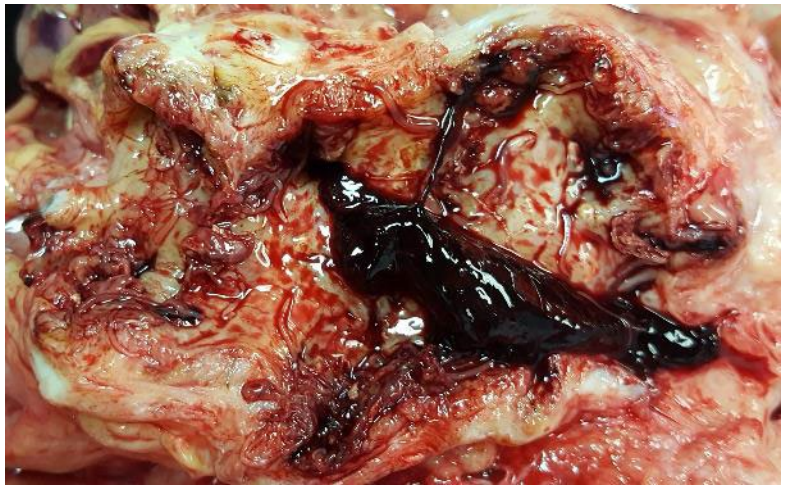
Endarteritis, with worms and stenosis of vessel





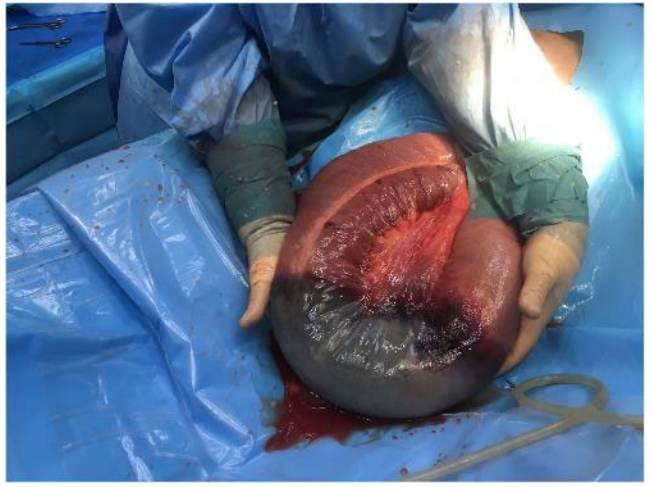
Thrombus occluding vessel & Immune Infiltrates





Thrombus & Worms

Fibrosis & Stenosis of Cranial Mesenteric Artery

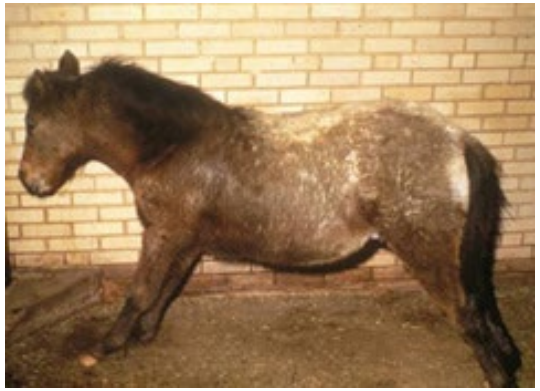


Infarction & Necrosis

Strongylus vulgaris
Diagnostics

General Diagnostics

- *Strongylus vulgaris* presents to the clinic as peritonitis
- Diagnostic Clues:
  - Signs of colic
  - rectal palpation of Cranial Mesenteric Artery (CMA)
  - fever, hyperemic mucous membranes
  - Poor deworming history, etc.
- Confirmatory diagnosis:
  - Nonstrangulating Intestinal Infarction on Exploratory Laparotomy (or post-mortem necropsy)

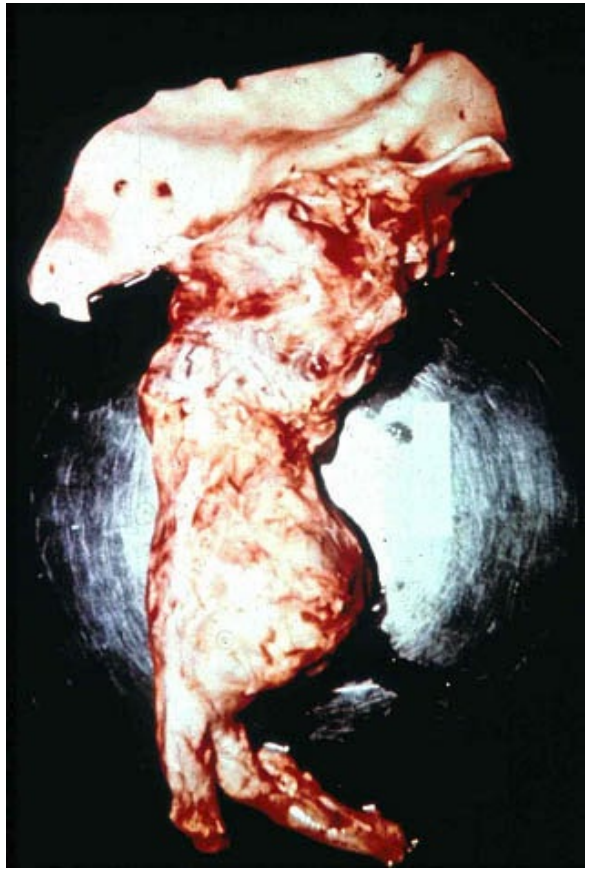


Laboratory findings:

- Abdominocentesis reveals a peritonitis with high numbers of white blood cells and increased protein and lactate content.
- FEC's have little clinical value

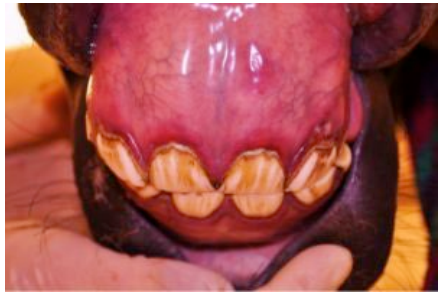
Initial phases manifest as colic.

Strongylus vulgaris
Clinical Signs

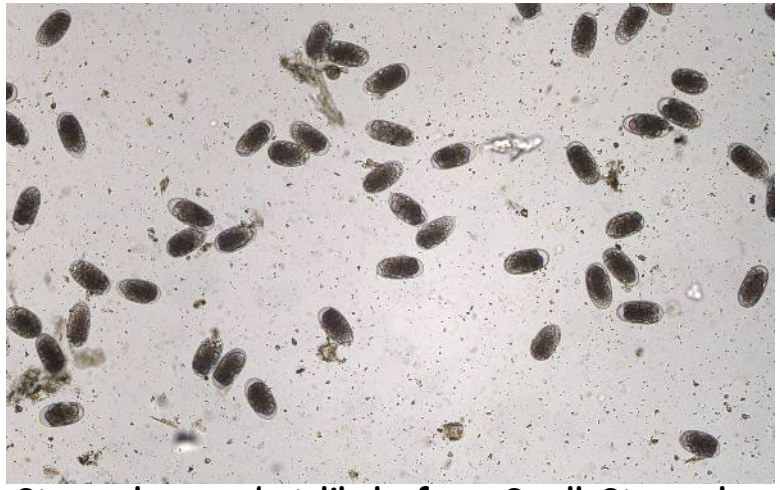


Enlarged Cranial Mesenteric Artery found on rectal palpation

Abdominocentesis reveals Peritonitis



Hyperemic mucous membranes



Strongyle eggs but likely from Small Strongyles

FEC have little clinical value

- Larvae pathogenic; not adult worms
- Larges strongyles show low fecundity
- 90% of strongyle ova are from small strongyles

Strongylus vulgaris
Treatment & Control

General Treatment
- Laparotomy to confirm infarction and determine prognosis
- Surgical correction if feasible
  - Resection of infarcted intestinal tissue
- Deworm with larvicide after patient is stabilized.

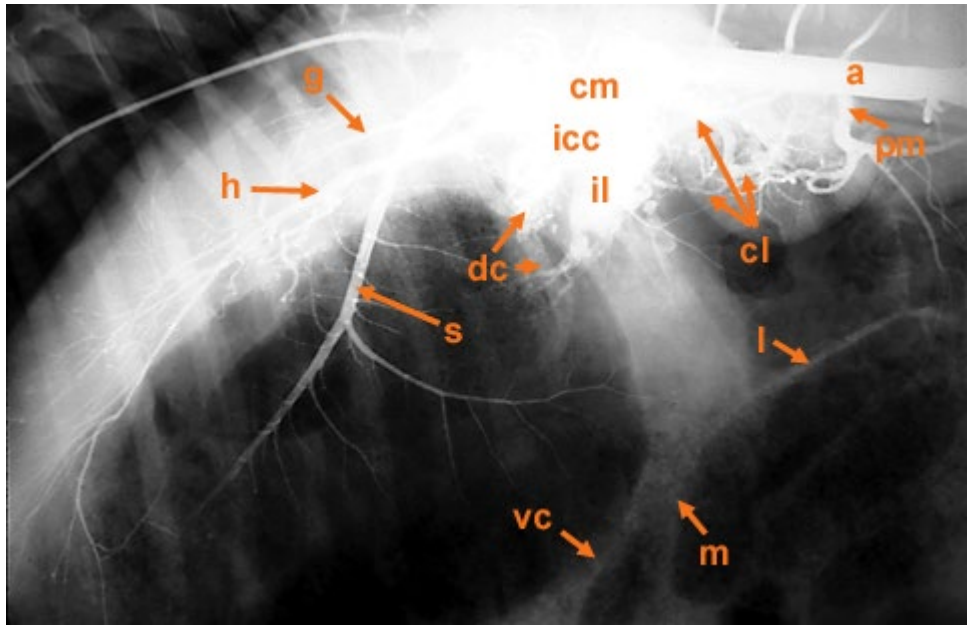
Treatment:
- When Laparotomy reveals intestinal infarction:
  - Determine extent of intestinal lesions
  - Determine feasibility of surgical correction
- If prognosis good, then surgical correction should proceed immediately.
  - Resection of infarcted intestinal tissue
- Prognosis:
  - Good to excellent for patients eligible for corrective surgery & <24 hrs after onset of clinical signs
  - Guarded if > 24 hours after onset of clinical signs
- Deworm with larvicide after patient is stabilized & peritonitis resolved.
  - Ivermectin, moxidectin, fenbendazole

Control
- General Pasture Management
- General Herd Management
- General Quarantine Protocols
- Base-line Deworming for horses
  - Per AAEP guidelines
  - Deworm minimum of 1 to 2 times per year.
  - **Large Strongyles are highly susceptible to deworming**
    - **PPP = 6 months**
      - **No time to mature and contaminate pastures b/w dewormings**
    - **Larvae never Arrest, always metabolically active.**
      - **Worms are always affected by dewormer**

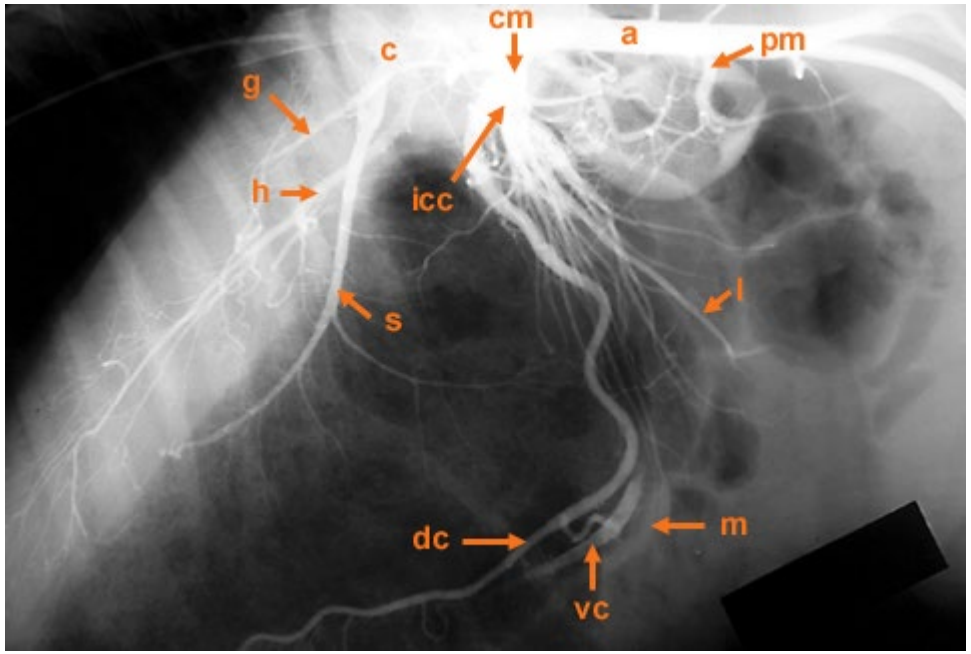
Anthelminthic resistance has not been reported for Large Strongyles, including *S. vulgaris*.

Strongylus vulgaris
Post-treatment Recovery

Experimental Results from
Anthelmintic therapy (not surgery).



Pre-treatment:
1 month after infection with 500 *S. vulgaris* L3s.



Post-treatment:
1 month after larvicidal therapy

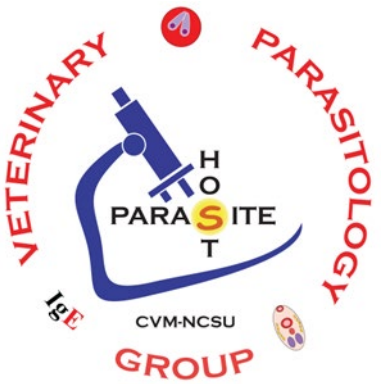
Strongylus vulgaris
Re-Emergence

Concern

- In Europe (Denmark & Sweden)
- Due to prescription-only legislation (i.e. no "over-the-counter" deworming)
  - Purpose of laws are to address small strongyle anthelminthic resistance due to dewormer over-use.
- Result: Now dewormers are under-used and *Strongylus vulgaris* has re-emerged.

- The emerging "deworm-based-on-FEC" problem
  - Small strongyles: If FEC = 100 epg
    - Then not deworming is not a problem
  - *Strongylus vulgaris*: If FEC = 100 epg
    - Then not deworming is a problem
      - (pathology & pasture contamination)

Oesophagostomum spp.



Nodular Worms

Take Homes

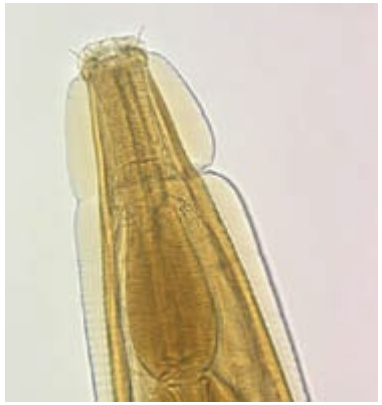
Oesophagostomum spp.

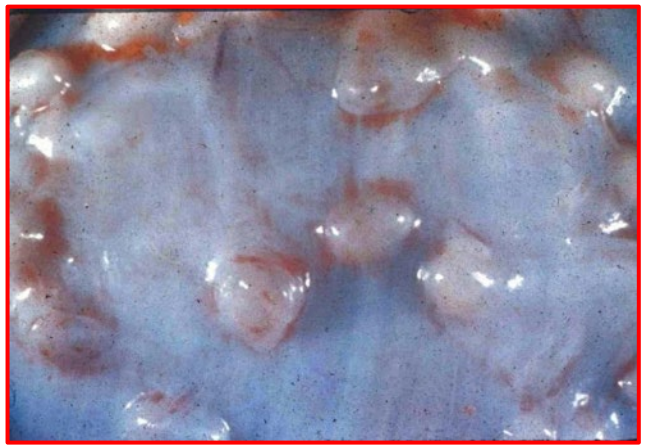
- Nodular worms of ruminants and swine
- Is a Pasture-borne Nematode (ingest L3s while grazing)
- Adult worms in the lumen of the large intestine and cecum
- Pathology is due to encysted L4s in the gut wall.
- Pathology:
  - Naïve Hosts: L4s don't encyst and mature to adults
  - Sensitized host: L4s encysted & host intense immune RXN + severe enteritis & potential intestinal motility issues
- Clinical Signs:
  - Presenting: Dark watery diarrhea w/ loss of body condition & weakness.
  - Abattoir reports condemnation of swine sausage casings (economic)
- FEC doesn't correspond to pathology, as pathology is due to encysted larvae.
- Necropsy:
  - Acute: larvae in pus-filled nodules in gut wall
  - Chronic: calcified larvae in caseous nodules
- Treatment - At time of clinical outbreak in the herd
  - Deworm and repeat with adulticide & larvicide
- Control:
  - Pasture management to avoid L3s. (especially wet areas)
  - Use in-feed dewormers to target incoming L3s

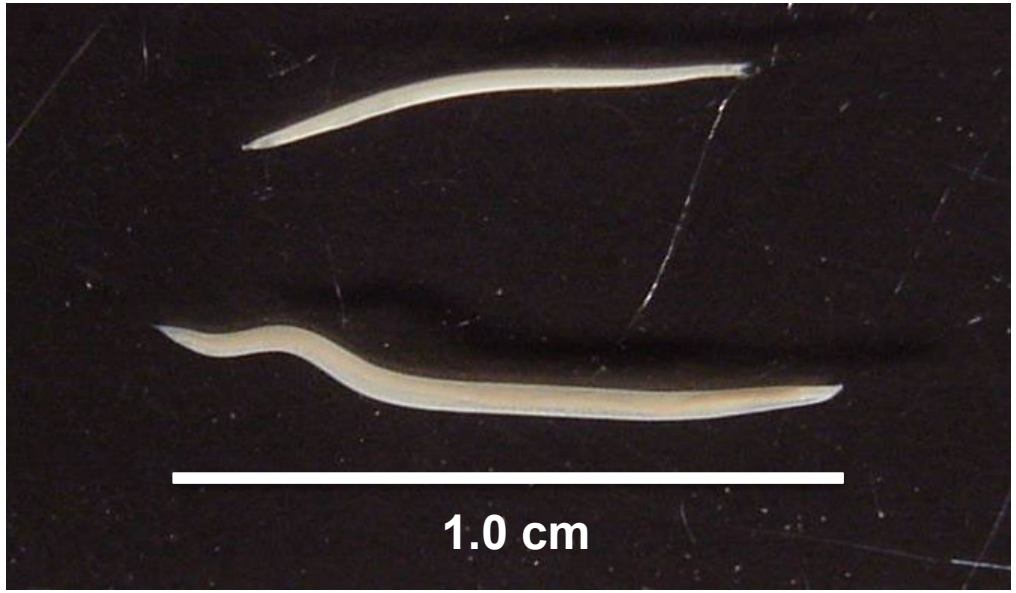
Oesophagostomum spp.
General

General

- Nodular Worm
- Parasites of Ruminants & Swine
  - Cattle: *Oesophagostomum radiatum*
  - Sheep & Goats: *O. columbianum* & *O. venulosum*
  - Pigs: *O. dentatum* & *O. brevicaudum*



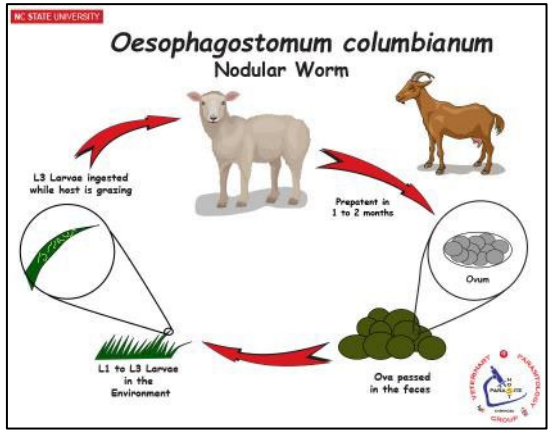


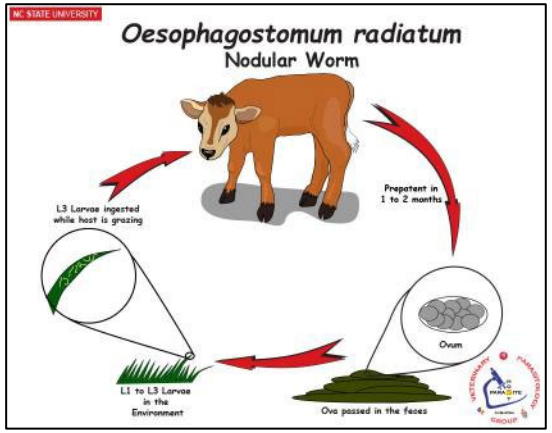


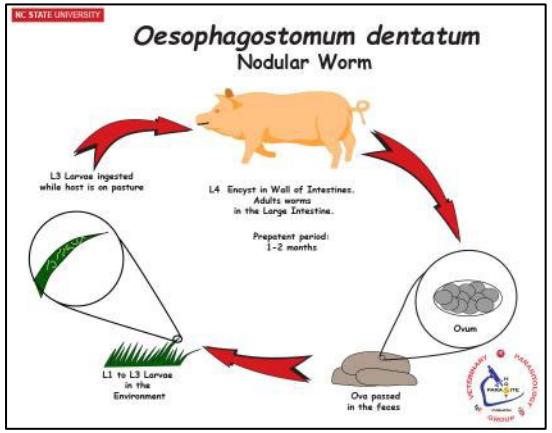
Oesophagostomum spp.
Life Cycle

Life Cycle

- Typical Pasture-borne Nematode Life Cycle
  - Ingestion of L3 on-pasture while grazing
- PPP: 1 – 2 months
- LC specifics
  - Adult worms in lumen of large intestine
  - Eggs passed in the feces
  - L1 → infective L3
  - L3s ingested
    - Migrates to the mucosa of the intestine
    - And develops to L4s
  - L4s in the gut wall
    - Non-sensitized host
      - L4s return to the lumen and develop into adults.
    - Sensitized host mounts an exuberant immune reaction
      - L4s are encysted and enteritis maybe intense
        - Eventually larvae are calcified.







Oesophagostomum spp.
Pathology, Diagnosis

Pathology

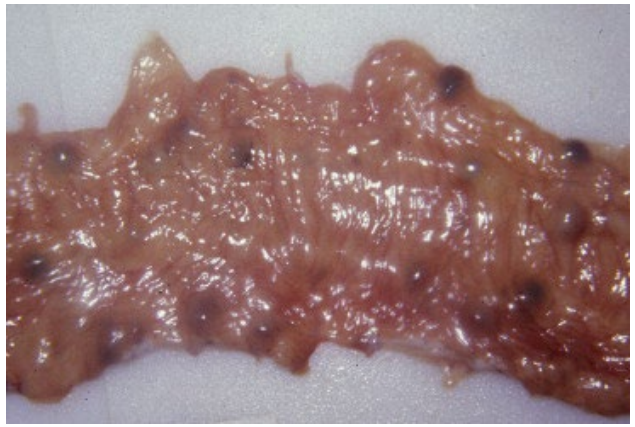
- Seen in adults and young animals older than the neonates.
- Encysted L4s in a sensitized host are the cause of pathology
  - ➔ Diagnosis: pathology during PPP ➔ FEC little value
- Acute inflammatory reaction & severe enteritis
  - Larvae in nodules
    - Pus-filled nodules
- Chronic cases
  - Larvae become calcified
    - Caseous nodules may affect intestinal motility
- Economic pathology in swine
  - Rejection of gut for use as sausage casings.

Clinical signs

- Watery, dark, fetid diarrhea
- Loss of body condition & weakness

Diagnostics

- Necropsy
  - Pus-filled or caseous nodules on serosa of gut.



Oesophagostomum spp.
Treatment & Control

Goat Small Intestine

DZ Treatment

- Deworming during clinical outbreak can be effective
- But include repeat treatment due to unaffected emerging larvae.

Control

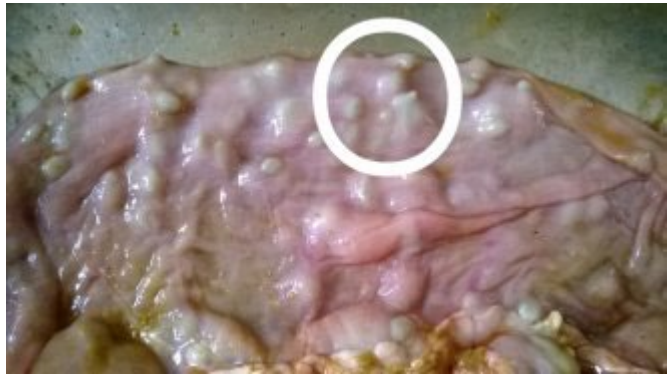
- In-feed dewormers target the incoming L3's prior to arresting in gut wall.

Avoid Infective L3's

- General Pasture management
- Infective L3's are very susceptible to desiccation.
  - Keep livestock away from wet areas.

Dewormer Resistance

- Swine only



Goat Large Intestine

http://tools.wormboss.com.au/sheep-goats/news/articles/worms-and-other-parasites/oesophagostomum-columbianuma-view-from-the-laboratory.php

Comparison Table

Nematode	LC	Pathology	Clinical Signs	FEC	Treatment	Control
Small Strongyles • Horses • Most Important	• Pasture-borne • Adults in colon/cecum • Encysted L4s in gut wall most pathogenic	• Granulomatous colitis – Severe immune RXN to encysted L4s • Larval Cyathostominosis – Acute Enteritis due to L4s bursting out of mucosa (loss of premunition concern)	• Presenting: Persistent diarrhea, weight loss, unthriftiness • Clues: young grazing horse, ventral edema, adult worms on feces/ palpation glove	• For Diagnostics: doesn't reflect pathology • For Control: Helps identify individuals with high contamination potential (>500 epg); Supports refugia efforts • FECRT: Monitor for dewormer resistance	Deworming • Target adult worms to decrease pasture contamination. • Target encysted L4s + anti-inflammatories • Resistance to many dewormers	• Various deworming strategies & pasture management Horses >3 years • focus on small strongyles. • Target high contaminators: >500 epg • Target L4s with larvicide at end of grazing season • FECRT every 3 years
Strongylus vulgaris • Horses • Historically: extremely Important • Now: re-emerging in Europe	• Pasture-borne • Adults in colon/cecum • Migrating L4s in mesenteric arteries most pathogenic (L4s do not encyst / arrest)	• L4s migrating for 4 months in mesenteric arteries • Cause endarteritis & thrombosis • May cause fatal thromboembolism, w/ intestinal infarction & acute peritonitis	• Presenting: Colic w/ appearance of peritonitis • Clues: Enlarged CMA on rectal palpation, hyperemic mucous membranes, • Diagnostics: Abdominocentesis confirms peritonitis • Confirmation: Exploratory laparotomy confirms NSII	• Does not reflect pathology	• Mild colic: deworming may provide recovery • Intense colic: Surgical correction & deworm after horse has stabilized • No dewormer resistance reported	• Quarantine protocols to prevent establishment on a farm • Base-line deworming 1-2X/year to eliminate S. vulgaris from farm • (6 month PPP – can't reach adulthood to contaminate pastures; don't arrest – all stages susceptible to deworming all the time)
***Oesophagostomum* spp.** • Nodular worm • Ruminants & Swine	• Pasture-borne • Adults in colon/cecum • Encysted L4s in gut wall most pathogenic	• Naïve Host: L4s straight to adults • Sensitized host: L4s encysted & host intense immune RXN + severe enteritis & potential intestinal motility issues	• Presenting: Dark watery diarrhea w/ loss of body condition & weakness. • Abattoir reports condemnation of swine sausage casings (economic)	• Does not reflect pathology Necropsy: • Acute: larvae in pus-filled nodules in gut wall • Chronic: calcified larvae in caseous nodules	Clinical outbreak: • Deworm and repeat with adulticide & larvicide	• Avoid L3s on pasture (especially wet pastures) • Use in-feed dewormers to target incoming L3s


VETERINARY
PARASITOLOGY
HOST
PARASITE
IgE
CVM-NCSU
GROUP