Ostertagia ostertagi



Nematodes : Strongylids : Trichostrongyles

GastroIntestinal Nematodes 1

Take Homes

Ostertagia ostertagi

- Brown Stomach Worm: is the primary GIN of Cattle. Most economically important GIN of Cattle
- Ostertagiasis is a DZ of 1st & 2nd year calves. Older cattle, via Acquired Immunity, show little to no pathology.
- Is a Pasture-borne Nematode (ingest L3s while grazing)
- Pathology is due to L4's in the Gastric Glands of the abomasum.
- The dysfunction of the gastric (abomasal) mucosa leading to increased pH; the interruption of digestion, causing a negative nitrogen balance and a systemic increase in protein catabolism.
- Clinical Signs: Anorexia, Weight-loss, Stunted Growth, Profuse & Persistent Watery Diarrhea, Hypoproteinemia with bottle-jaw & edema
- On Necropsy: Abomasal mucosa has appearance of Moroccan Leather (Prominent NAVLE question)
- Understand the differences b/w Type I & Type II Ostertagiasis
 - Type I L3s trickle in during early grazing season, L4s don't arrest, DZ gradually increases, 1st grazing season young calves, High Morbidity, Low Mortality, deworm with adulticide.
 - TYPE II L3s acquired in late grazing season, L4s arrest (hypobiotic) throughout non-grazing season, beginning of 2nd grazing season sudden eruption of large numbers of reactivated L4's do much damage, High Mortality, deworm with larvicide near end of 1st grazing season to eliminate population of arrested L4s.
 - Understand grazing seasons per region: South (think Arizona), hot, dry in Summer → no grass growth in Summer. North (think Iowa) frigid winters→ no grass growth in Winter. NC is Northern. (Understand how inter-regional transport of livestock may confound regional Type II presentation of Ostertagiasis.)
- Diagnostics: FEC, MOO test, pepsinogen levels in blood, abomasal-centesis, necropsy.
- Pasture management practices (Good v/s Bad)

Ostertagia ostertagi General & Life Cycle

General

- A primary GastroIntestinal Nematode (GIN)
- Brown Stomach Worm
 - Infect the abomasum
- Most economically important helminth of Cattle
- Ostertagiasis is a disease of calves and young cattle
 - Mainly during 1st and 2nd Grazing season





[•] Ostertagiasis in Cattle: a Review -- https://journals.sagepub.com/doi/epdf/10.1177/104063878900100225

Pathophysiology of Ostertagiasis -- https://www.sciencedirect.com/science/article/pii/030440179390055F

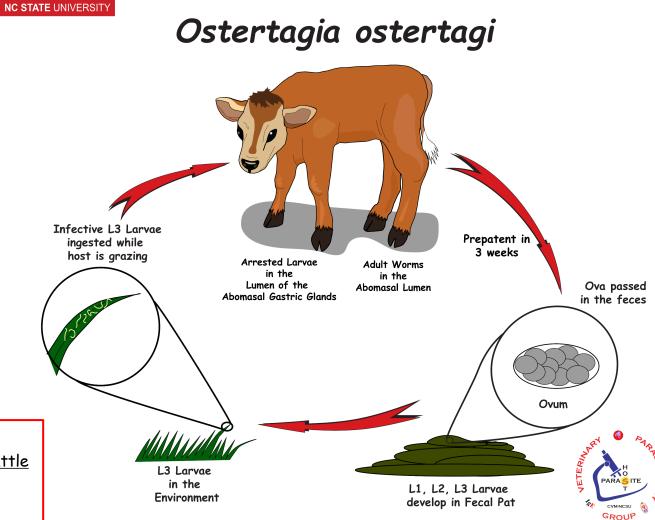
Ostertagia ostertagi General & Life Cycle

Life Cycle

- Typical Pasture-borne Nematode Life Cycle
 - Ingestion of L3 on-pasture while grazing
- Adult worms in abomasum
- PPP 3 weeks
- Low fecundity (produces a low number of offspring)
 - FEC = 100 epg \rightarrow concern
 - FEC = 1000 epg \rightarrow dangerous
- L3s
 - Relatively hardy in cold weather -> some L3s over-winter on pasture.
 - Keep Larval Storms in mind
- L4s
 - Arrest and develop in the lumen of gastric glands
 - Don't penetrate into the mucosa like some others.
- Ostertagia "Over-winters" via:
 - 1. Some L3s on pasture.
 2. Mostly arrested L4s in the host

Immunity

- Acquired Immunity is an important advantage for cattle
 - develops at approx. 2 years of age
 - mature cows do not commonly show clinical signs



Ostertagia ostertagi Pathology & Clinical Signs

Brief Description of Pathology

- Main Pathology Due to growth and development of L4s in the lumen of the Gastric Glands
- Ostertagiasis results from the dysfunction of the gastric (abomasal) mucosa leading to the interruption of digestion, as well as various other pathologies, involving a cascade of hormonal and physiological events.
- Ultimately, these <u>changes result in negative nitrogen balance</u>, <u>leads to systemic increase in protein catabolism</u>, resulting in symptoms of protein deficiencies (weight loss, stunted growth, poor feed conversion, poor meat and/or milk production)



- Young Animal
- Profuse & Persistent Watery Diarrhea
- Hypoproteinemia bottle jaw & edema
- Rough Hair Coat
- Anorexia (reduction in food intake)
- Weight loss
- Stunted growth
- Weakness





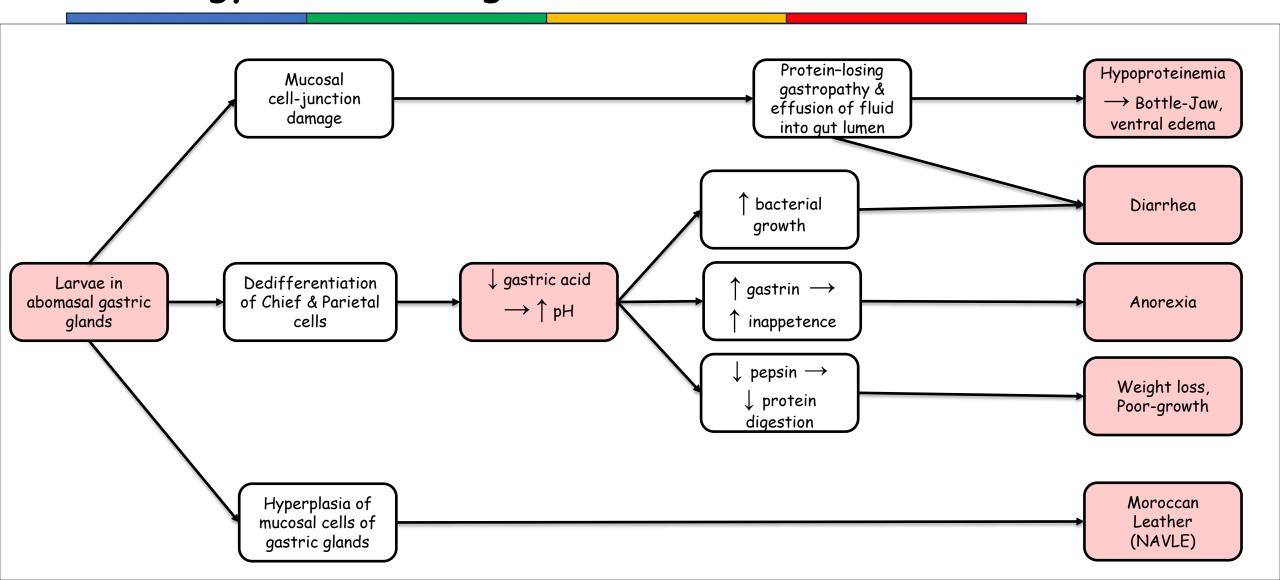
Ostertagia ostertagi Pathogenesis

More detailed Pathogenesis

- The pathogenesis of Ostertagiasis involves cellular changes of gastric gland cells caused by Ostertagia larvae and adults.
 - L3 larvae that are ingested from pasture enter the gastric glands & develop to L4
 - L4 larvae cause the parietal and chief cells lining the gastric glands to dedifferentiate and stop producing acids.
 - Increases gastric pH from 2 to 6. [> 4.5 and digestion stops]
 - increased gastrin => inappetence => anorexia
 - allows overgrowth of bacteria => increased diarrhea
 - As mucosal cells proliferate, nodules are formed giving the abomasal lining the appearance of "Morocco leather". {pathognomonic, often on boards}
 - Neighboring non-parasitized gastric glands are also induced to dedifferentiate; exacerbating the pathology.
 - Developing L4s and emerging young adults further damage the mucosa and induce a severe inflammatory reaction.
 - The mucosa becomes leaky, causing the loss of protein and fluid into the gut lumen.
 - Persistent Diarrhea
 - Hypoproteinemia => edema (bottle jaw)
 - Increased protein catabolism leads to weight loss and poor muscle / bone growth.
 - Looks similar to Johne's DZ (Mycobacterium avium paratuberculosis) [aka Paratuberculosis] {reportable}

Ostertagia ostertagi Pathology & Clinical Signs

Things to know for this course



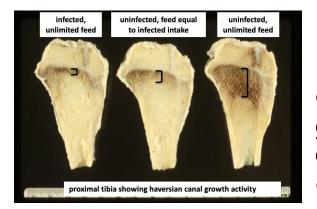
Ostertagia ostertagi Clinical Signs



Poor Body Condition, Rough Hair Coat Stunted Growth, Anorexia



Hypoproteinemia, Bottle-Jaw



Calves show stunted growth due to anorexia and protein catabolism.



Diarrhea

FYI

Practice the 5-Point Check

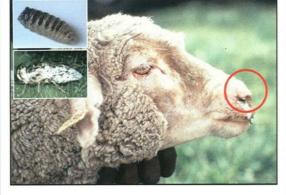




https://extension.okstate.edu/factsheets/body-condition-scoring-of-cows.html

Score cards for 5√





Spines	individually clearly felt, sharp, obvious	Form a smooth line with deep undulations	Only slightly detectable undulations	Only detectable with firm pressure	Not detectab
Transverse processes	Fingers easily pass underneath	Smooth round edges	Well covered. Have to push firmly to get fingers underneath	Cannot be felt at all	
Muscle	Very little. Concave	Concove	Not concave. Not convex	Maximally developed. Convex	
Fat layer	No	Very thin	Moderate	Thick	Very thick to form a dip along top n
Spino venedia sone see over the lon					6
Condition score	1	2	3	4	5

This scheme may be used in goats, but half a score is added to the score, since goats

preferentially store fat intro-abdominally and not over the lower back.

No faecal soiling at all No indication for treatment / action Very slight soiling on edge of tail / on each side No treatment / action needed Slight soiling on edge of tail and on each side Usually no treatment / action Moderate soiling of tail and wool Dag formation Consider treatment / action Severe soiling extending far into the wool Severe dag formation Treatment / crutching recommended Very severe, watery diarrhea extending to the crutching essential Copyright® University of Pretoria

DAG SCORECARD

Ostertagia ostertagi Necropsy



Abomasum at Necropsy Pathognomonic





Slippers of Moroccan Leather (aka goatskin)





Ostertagiasis ecology Seasons & Triggers

Ostertagiasis Seasons

- When is the grass not growing?
 - Deep South (dry & arid in Summer) → grazing season is Fall, Winter, Spring
 - North (cold Winter) → grazing season is Spring, Summer, Fall
 - NC → Northern season
 - always consult extension agent in your area.

Triggers

- When to arrest?
 - L1s, L2s, L3s that are on-pasture
 - detect increasing adverse conditions on the pasture (usually temperature)
 - The eminent harsh condition will not support next generation on pasture.
 - So, L4s arrest and don't become egg-producing adults until better conditions.
- When to reactivate and resume development?
 - Arrested L4's in the gastric glands get various signals
 - Timing mostly genetically predetermined ("internal clock")
 - But also influenced by host's stressors
 - Parturition
 - Poor nutrition
 - Concurrent infection
 - Poor Host immune response



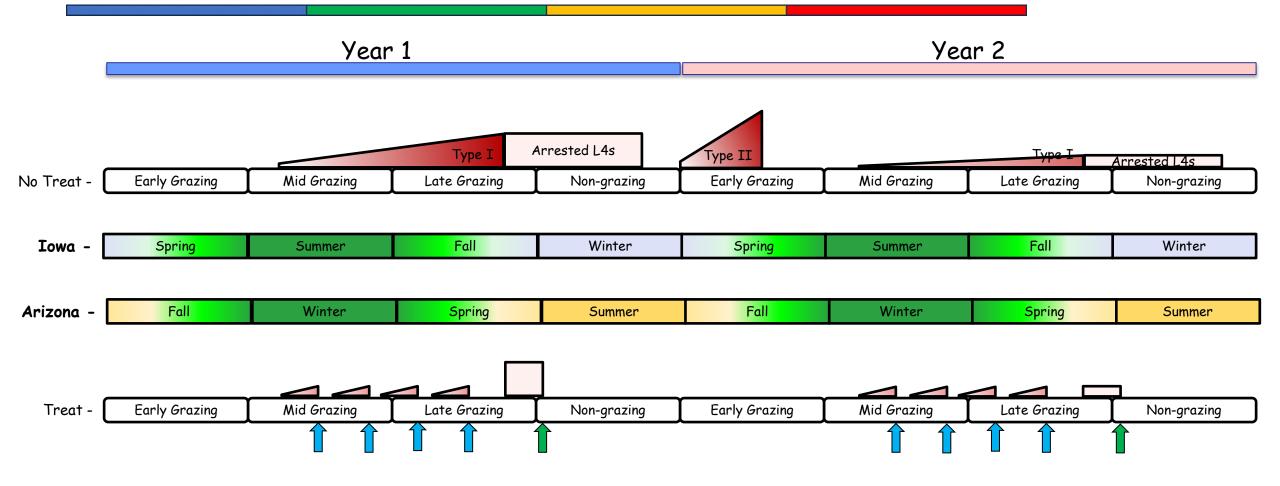
Ostertagia ostertagi Ostertagiasis: Season & Region

Larvicide (Arrested L4)

dewormer

dewormer





Types of Ostertagiasis

Type I Ostertagiasis



L4s don't arrest; develop directly to adult worms.

- Primarily young calves in 1st grazing season
- L3s ingested early to mid grazing of 1st grazing season
- Pathology occurs mid to late of 1st grazing season

Pathology by Region & Season

- Cool Region (Iowa): Summer & Fall of 1st grazing season
- Arid Region (Arizona): Winter & Spring of 1st grazing season

High Morbidity, Low Mortality

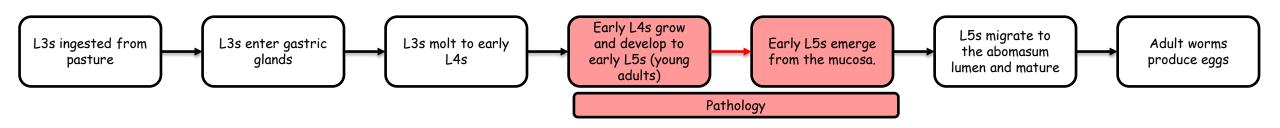
■ L3s "trickle-in" while grazing \rightarrow slow, progressive pathology.

Treatment

- Young 1st season calves
- Early in grazing season
- Target adult worms to prevent pasture contamination
- Treat & Move

OR

 Treat & Repeat treatment in 2 to 3 weeks (PPP), if left on contaminated pasture.



Types of Ostertagiasis

Type II Ostertagiasis



L4s arrest; reactivate later.

- Primarily older calves (yearling calves) in 2nd grazing season
- L3s ingested late grazing of 1st grazing season
- Pathology occurs early 2nd grazing season

Pathology by Region & Season

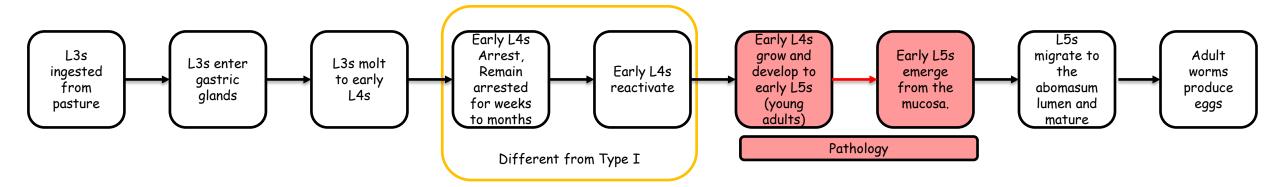
- Cool Region (Iowa): Spring of 2nd grazing season
- Arid Region (Arizona): Fall of 2nd grazing season
- Special Note: Southern Calves moved to Northern feedlots in the fall have the potential for Type II

Low Morbidity, High Mortality

- Mass of arrested L4s reactivate, grow, & re-emerge simultaneously
- Sudden acute pathology

Treatment

- Older 1st season calves
- Late in grazing season
- Target Arrested L4s to prevent Type II that occurs early in the 2nd grazing season



Ostertagia ostertagi Case

Ostertagiasis at the Feedlot

A group of 1st year calves were grazed from September to May in Arizona.

The calves were dewormed in mid-December.

In May, they were shipped to a feedlot in South Dakota.

At the feedlot, in October, the calves came down with a bad case of Ostertagiasis.

- Which Type of Ostertagiasis was targeted with the December dewormer?
- Which Type of Ostertagiasis was causing pathology in October at the feedlot?
 Explain your answer.
 - Which Type of Ostertagiasis is normally seen in South Dakota in October?
 - Did the calves become infected at the feedlot?
 - When did these calves become infected?



Ostertagia ostertagi Diagnostics

Diagnostics

- Fecal Egg Count (Wisconsin or Double Centrifugation)
 - Assess eggs present for possible treatment of sub-clinical cases and reduce pasture contamination.
- MOO Test (Milk Ostertagia ostertagi ELISA): Assess antibodies v/s Ostertagia present in bulk milk sample.
- Serum Pepsinogen levels increase
- Abomassal-centesis: Check for increased pH
- Deworm & observe
- Necropsy: Moroccan Leather





Ostertagia ostertagi Treatment & Resistance

Dewormer options (Beef and Dairy)

Ivermectin, Moxidectin, Doramectin, Fenbendazole, Eprinomectin

Type 1 (adulticide)

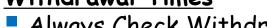
Most Common: IVOMEC injection or pour-on

Type 2 (includes a larvicide)

- Beef: IVOMEC injection or pour-on
- Dairy: EPRINEX pour-on is the most appropriate and has zero milk withdrawal time

Withdrawal Times

Always Check Withdrawal times!





Dewormer Resistance

For Ostertagia: No Resistance issues reported in Cattle.









Ostertagia ostertagi Control: Pasture Management

Maintain Healthy Pastures

- Avoid Overgrazing and/or Overstocking
 - Continuous Grazing
 - Not so Good
 - Rotational Grazing
 - Planned Intensive Grazing

Parasite Avoidance Specific to Cattle

- Avoid infective L3's
 - Rotational Grazing
 - Naïve calves on fresh pasture before older calves
 - BAD! If older calves first; will seed pasture to infect naïve calves. BAD!
 - Don't use same pasture for calves every year, as some L3s overwinter on pasture..
 - Co-grazing (vacuum cleaners)
 - Intraspecific (Age-related immunity)
 - Don't mix young calves and older <u>calves</u>
 - Cow / Calf operations: Immune Cows act as vacuum cleaners to decrease
 L3s available for the calves at their side.

Parasite Avoidance

- Genetically Resistant / Resilient Host Breeds
- Avoid Overgrazing and/or Overstocking
 - Deters Excessive Pasture Contamination
- Include Plant Browse with anthelmintic properties
- Avoid infective L3's
 - Rotational Grazing
 - Run-away from L3's
 - prior to ova → L3 development
 - Planned Intensive Grazing
 - Avoid vertically migrating L3's
 - Graze forage to 4 inches then move
 - Compost Feces
 - Take Hay off contaminated pastures
 - Rest pastures until L3 die-off
 - Co-grazing (vacuum cleaners)
 - Interspecific (Host specificity)
 - Intraspecific (Age-related immunity)
 - Avoid Larval Storms

Ostertagia & other GINs

Parasite (Host)	Transmission	Pathology	Clinical Signs	Diagnostics	Treatment & Control	Notes
Haemonchus contortus (Sheep & Goats) Abomasum	Ingest L3s on pasture	Anemia	Pale Mucous Membranes, Bottle Jaw, Tarry Feces, Lethargy	FAMACHA, FEC, FECRT	Deworm @ 1st sign Based on FAMACHA & FEC Good Deworming practices, Good Pasture Management	Hyperacute, Acute, Chronic Most Important for S. Ruminants Resistance a huge issue
Ostertagia ostertagi (Cattle) Abomasum	Ingest L3s on pasture	Gastric gland damage, Increase pH, Digestion Stops, Protein catabolism	Watery Diarrhea, Bottle Jaw, Poor Body Condition, Anorexia	FEC, Blood Pepsinogens, Abomassal-centesis (increase pH)	Deworm & Supportive care Good Deworming practices, Good Pasture Management	Ostertagiasis Type I & Type II in different regions & different seasons Moroccan Leather Most Important for Cattle
Small Strongyles (Horse) Cecum & colon	Ingest L3s on pasture	Granulomatous colitis, Larval Cyathostomiasis	Diarrhea, Colic, Ventral edema, Poor Body Condition	FEC, FECRT	Deworm & Supportive care Good Deworming practices, Good Pasture Management	Most Important for Horses
Trichostrongylus colubriformis (Sheep & goats) Small Intestine	Ingest L3s on pasture	Enteritis	Dark green watery Diarrhea, Dags, Dingle-berries, Poor Body Condition	FEC, FECRT	Deworm & Supportive care Good Deworming practices, Good Pasture Management	Fly Strike

Minor GINs

Trichostrongylus, Nematodirus, Cooperia



Nematodes: Strongylids: Trichostrongyles

GastroIntestinal Nematodes 2

Take Homes

Trichostrongylus axei (stomach hair worm)

- A Minor GIN in the ruminant abomasum and horse stomach
- Most pathology in Small Ruminants
- A Pasture-borne Nematode (ingest L3s while grazing), L3s overwinter well, (L4's don't arrest).
- Pathology is due to adult worm activity, Gastritis
- Pathology mostly in the spring, diarrhea.
- Usually not a problem for horses, if one does have a problem then don't co-graze horses with small ruminants

Trichostrongylus colubriformis (Bankrupt worm)

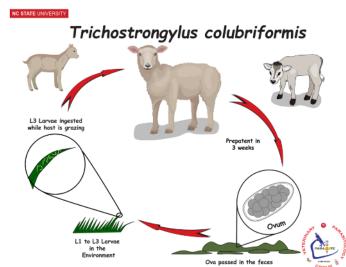
- A Minor GIN in the ruminant Small Intestine
- Most pathology in Small Ruminants
- A Pasture-borne Nematode (ingest L3s while grazing), L3s overwinter well, (L4's don't arrest).
- Pathology is due to adult worm activity, Enteritis
- Clinical Signs: Protracted dark watery diarrhea, (Black Scours), Dingleberries & Dags, Fly Strike, anorexia, stunted growth
- Salvage deworming or Prevention by deworming young early in grazing season.

Trichostrongylus axei & Trichostrongylus colubriformis

Trichostrongylosis

- DZ name for T. axei or T. colubriformis
 - More often a disease of sheep & goats
 - Pathogenic only with heavy burdens (>100,000 worms).
 - Stress, malnutrition contribute to DZ
- Secondary DZ causing GIN
 - v/s Primary GIN (Haemonchus, Ostertagia, Small Strongyles)
 - During mixed infections, Trichostrongylus spp. are overshadowed by primary GIN species.
 - But Trichostrongylus spp. may confound diagnostics
 - Strongyle-type eggs look the same as the ova of other GINs





<u>Pathology</u>

Due activity of adult worms

<u>Life Cycle</u>

- Typical Pasture-borne Nematode Life Cycle
 - Ingestion of L3 on-pasture while grazing
- PPP 3 weeks
- Low fecundity
 - Rarely exceed 5,000 epg
- L3s over-winter well on pasture but die-off in Summer, but not before re-contamination of pasture by Fall.
 - Most pathology in early spring
- L4s only migrate below the mucosal surface and do not arrest.
- Trichostrongylus "Over-winters" via:
 - L3s on pasture, only.

Trichostrongylus axei



<u>Trichostrongylus axei</u>

- Stomach Hair worm
- Abomasum: Sheep, goats, cattle
- Stomach: horses, rabbits, humans

Ruminants

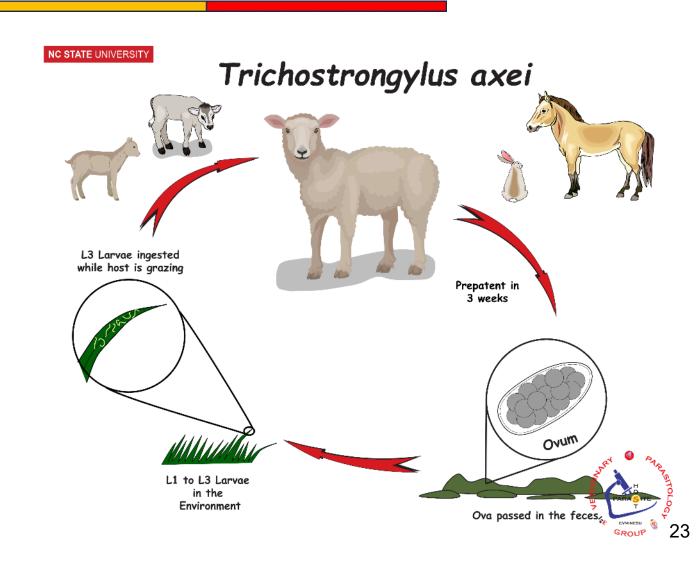
- Gastritis
 - Erosions/damage to the abomasal mucosa

Rabbits

- Gastritis
 - Erosions/damage to the stomach mucosae

Horses

- Not considered a primary parasitic pathogen of horses
- Has become very rare in equine operations
- Catarrhal Gastritis has been suggested
 - Possible Erosions & Ulcers
 - Possible weight loss



Trichostrongylus colubriformis



Trichostrongylus colubriformis

- Bankrupt worm
- Small intestine: Sheep, goats, cattle

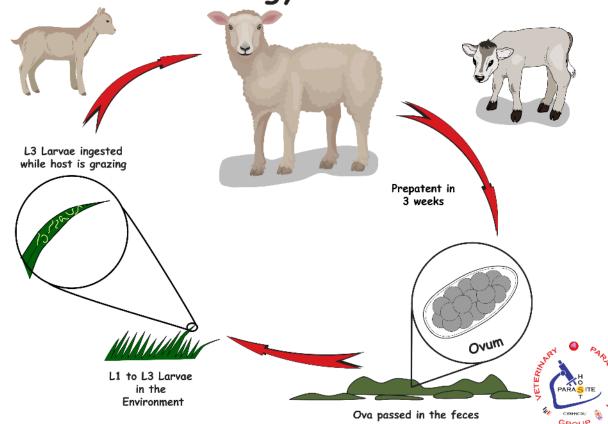
Enteritis

- Damage to the Intestinal mucosa
 - Loss of protein into gut lumen → hypoproteinemia
- Villus atrophy
 - Decreased brush border enzyme activity → decreased digestion
 - Decreased absorption
- Protracted watery diarrhea
 - Weakness, weight loss & wasting → "bankrupt worm"
 - "Fly Strike" complications
- Anorexia → decreased food intake
 - Decrease bone growth → stunting
 - Decreased growth rate → decreased production → "bankrupt worm"



"upper small intestines that looked like they had been scrubbed with a wire brush" ---Paul Nilon of Nilon Farm Health, Tasmania for wormboss.com.au NC STATE UNIVERSITY

Trichostrongylus colubriformis



Trichostrongylus colubriformis

<u>Diagnostics</u>

- Primarily a combination of:
 - Clinical Signs
 - Lack of Deworming
 - Farm has history of Trichostrongylosis
- FEC
 - not reliable for Trichostrongylus spp. only infections
 - Rarely exceed 5,000 epg
 - Overshadowed by Haemonchus ova.
- Larval Speciation
 - Coproculture + Baermann isolation
 - Specialty labs; cumbersome / delayed results

Clinical Signs

- Diarrhea
 - Watery, protracted, dark green
 - Black Scours
 - Feces soiling may extend to hocks
 - Dingleberries (pea size) & Dags (egg-size) hanging from fleece or hair
 - Attracts blow flies → maggots "Fly Strike"
 - Crutching: Refers to the removal of wool from around the tail and between the rear legs of a sheep to prevent "Fly Strike".



Strongyle-type ova



black scours, dingleberries, dags



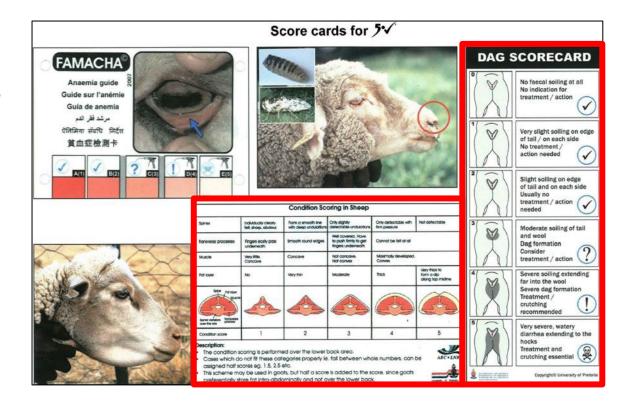
Crutching

Trichostrongylus spp.

Five-Point Check (small ruminants)

- 1. Eye FAMACHA n/a (Haemonchus)
- 2. Back Body Condition Score
 - May reveal weight loss / wasting
- 3. Tail Dag Score Most appropriate for diarrhea-causing GIN's, including Trichostrongylus spp.
 - Protracted watery dark-green diarrhea extending to hocks. (black scours)
 - Dingleberies / Dags present consider deworming
- 4. Nose nasal discharge n/a (Oestrus)
- 5. Jaw bottle Jaw n/a (Haemonchus / Ostertagia)





Trichostrongylosis

Deworming & Pasture Management

Tactical Deworming

- Salvage deworming
 - @ clinical signs
 - Trichostrongylosis protracted dark green diarrhea & wasting in Small Ruminants

Strategic Deworming

- Scheduled Deworming of small ruminants specifically targeting Trichostrongylosis
 - Most important issues
 - <u>L3's</u> of Trichostrongylus spp. overwinter well on pasture & will infect flock early in the grazing season.
 - Deworm young early in grazing season

Parasite Avoidance Specific to Horses

- Horses usually don't get T. axei unless co-grazed with sheep or goats.
 - If T. axei is a problem \rightarrow Don't co-graze ruminants with horses.

Maintain Healthy Pastures

- Avoid Overgrazing and/or Overstocking
 - Continuous Grazing
 - Not so Good
 - Rotational Grazing
 - Planned Intensive Grazing

Parasite Avoidance

- Genetically Resistant / Resilient Host Breeds
- Avoid Overgrazing and/or Overstocking
 - Deters Excessive Pasture Contamination
- Include Plant Browse with anthelmintic properties
- Avoid infective L3's
 - Rotational Grazing
 - Run-away from L3's
 - prior to ova → L3 development
 - Planned Intensive Grazing
 - Avoid vertically migrating L3's
 - Graze forage to 4 inches then move
 - Compost Feces
 - Take Hay off contaminated pastures
 - Rest pastures until L3 die-off
 - Co-grazing (vacuum cleaners)
 - Interspecific (Host specificity)
 - Intraspecific (Age-related immunity)
 - Avoid Larval Storms

GastroIntestinal Nematodes 2

Take Homes

Nematodirus spp. (Thread-necked Intestinal Worm)

- A Minor GIN in the ruminant Small Intestine
- Most pathology in Lambs & Kids
- A Pasture-borne Nematode (ingest L3s while grazing), L3s overwinter within the egg, (L4's don't arrest).
- Massive Larval Storms: over-wintered eggs hatch simultaneously in the spring. Pathology in spring.
- Pathology is due to adult worm activity, Acute Enteritis
- Clinical Signs: Sudden onset of Profuse diarrhea, unthriftiness, dehydration, death
- Control: Lamb & Kid in pastures not graze during previous season, deworm early in grazing season

Cooperia spp. (Small Intestinal Worm)

- A Minor GIN in the ruminant Small Intestine
- A Pasture-borne Nematode (ingest L3s while grazing).
- Prominent pathogen in Cow-Calf operations
- Pathology is due to adult worm activity, Enteritis
- Clinical Signs: Diarrhea, Anorexia, Emaciation
- Calf has dramatic decrease in weight gain
- Some resistance to Macrocyclic lactones

Nematodirus spp. Thread-necked Intestinal Worm

General

- Small Intestine of Ruminants
 - DZ of lambs & kids, less so for calves
- Secondary DZ causing GIN
- Mainly UK, New Zealand, Australia
- Sporadic in USA

Life Cycle

- Typical Pasture-borne LC
- PPP: 15 days
- Low fecundity
- L3 larvae over-winter in the egg, hatch and ir hosts in spring. But die-off in Summer, but not before re-contamination of pasture by Fall.
 - Most pathology in early spring
- L4s only migrate below the mucosal surface and do not arrest.
- Nematodirus spp. "Over-winters" via:
 - Ova on pasture only.

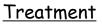
Pathology

- Mainly due to adult activity
- Acute Enteritis
- Extreme Diarrhea
 - Severe & Debilitating
 - Leads to death via rapid dehydration
 - High mortality (20%)

Diagnostics

- Eggs in feces are very distinct
 - Very Large eggs
 - but DZ often prior to PPP
- Clinical Signs: Diarrhea
- Pasture History
 - Lambing in same pasture that was grazed the previous year.
- DZ History
 - Is Spring diarrhea a regular occurrence?
- Necropsy: dehydrated carcass & enteritis, 10,000+ worms

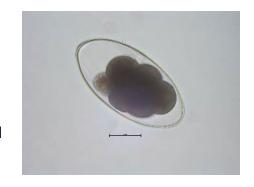




 Most anthelminthics are effective



Nematodirus spp. Thread-necked Intestinal Worm



Special Ecology & Epidemiology

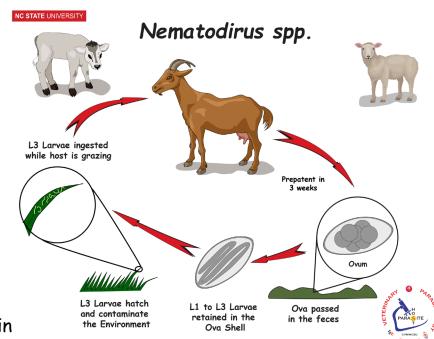
- In-Egg development
 - L1 to L3 development within eggs
 - Eggs overwinter better than exposed larvae
 - But larvae can't feed, so energy stores can't be created for longer life.
 - Consequences:
 - Massive build up of eggs on pasture through out a whole year.
- L3 Hatching requirements
 - Eggs must be chilled (overwinter) before hatching during following warm season (spring).
 - Consequences:
 - All eggs from previous year hatch simultaneously in Spring > Massive Larval Storms
- Adult ruminants (including cattle) do not develop acquired-immunity
 - Consequences:
 - Adult ruminants are contributing to pasture contamination even if asymptomatic
- After Hatching, L3 survive for only 1 month on Pasture
 - Benefit:
 - Pastures cleanup quickly during that grazing season.
 (if Nematodirus is your only concern)

<u>Clinical Signs</u>

- Lamb, Kid, Calf
- Dz in Late spring
- Sudden onset with rapid progression
 - Unthriftiness
 - Profuse diarrhea
 - Marked dehydration
 - Deaths begin 2-3 days after 1st clinical signs

Control

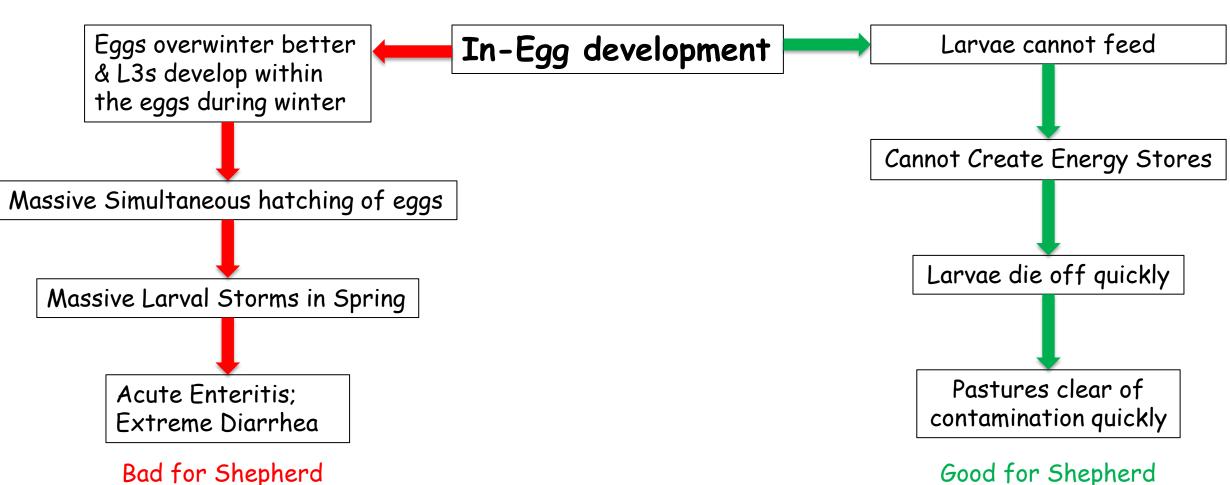
- Lambing & Kidding in different locations each year
 - Don't lamb in same pasture that was grazed previous season



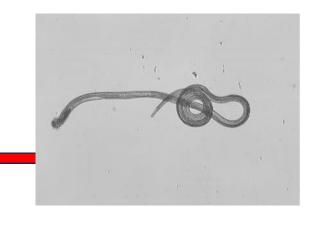
Nematodirus spp.

Thread-necked Intestinal Worm

Special Ecology & Epidemiology



Cooperia spp. Small Intestinal Worm



General

- Duodenum of ruminants
- Secondary DZ causing GIN

<u>Life Cycle</u>

- Typical Pasture-borne Nematode Life Cycle
 - Ingestion of L3 on-pasture while grazing
- PPP: 12-15 days
- Low fecundity

Treatment

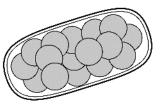
- Some Cooperia species are showing resistance to macrocyclic lactones (Ivermectin)
 - Use benzimidazoles (Fenbendazole).

<u>Pathology</u>

- Due to Adult activity
- Enteritis: Catarrhal inflammation, mucosal congestion
- Diarrhea, Anorexia, Emaciation

Cooperia oncophora

- Prominent pathogen of Cow-Calf operations
- In calf, cause dramatic decrease in daily weight gain



<u>Diagnostics</u>

- FEC (but includes all strongyle-type ova)
- Speciation via fecal culture & Baermann isolation

Resistance Issue FYI only

- When developing Ivermectin treatment regime
 - Cooperia was the rate-limiting nematode
 - So, why resistance in Cooperia?

Minor GINs

Parasite (Host)	Transmissi on	Pathology	Clinical Signs	Diagnostics	Treatment & Control	Notes
Trichostrongylus axei (Sheep & Goats - Abomasum) (Horse - stomach)	Ingest L3s on pasture	Sheep & goats - gastritis Horses - rare gastritis in horses	Spring diarrhea	(overshadowed by Haemonchus)(overshadowed by small strongyles)	Good Deworming practices, Good Pasture Management	If endemic on a farm, then don't co-graze small ruminants & horses
Nematodirus spp. (Cattle, Sheep & Goats) Small Intestine	Ingest L3s on pasture	Acute enteritis	Sudden onset of profuse diarrhea, unthrifty, dehydration, death. DZ in Spring	Distinct eggs on FEC or Fecal Float	Deworm & Supportive care Good Deworming practices, Good Pasture Management Don't Lamb & Kid in same pasture that was grazed in the Fall.	Lambs & Kids show DZ Eggs over-winter and hatch simultaneously in Spring (Larval Storms due to eggs hatching simultaneously)
Cooperia spp. (Cattle, Sheep & Goats) Small Intestine	Ingest L3s on pasture	Enteritis	Calves Diarrhea, Anorexia, Emaciation Primarily Cow-Calf Operation	FEC or Fecal Float	Deworm & Supportive care Good Deworming practices, Good Pasture Management	Calves show dramatic decrease in weight gain Prominent Pathogen of Cow-Calf operations Some resistance to Macrocyclic Lactones reported.

