

The order Strongylida comprises 4 superfamilies

Order: **STRONGYLIDA I.**

- **gastrointestinal worms**
 - **lung worms**
- **worms of other organs**

(ruminants, horses, pigs, carnivores, rabbits, poultry) – the main species according the hosts, morphology, life cycles, epidemiology, prevalence, pathogenesis and clinical signs, diagnosis, treatment and control.

prof. Alica Kačičová, DVM, PhD.

Order: **STRONGYLIDA**

Superfamily: **STRONGYLOIDEA**

ANCYLOSTOMATOIDEA

TRICHOSTRONGYLOIDEA

METASTRONGYLOIDEA

Superfamily: **STRONGYLOIDEA**

Family: **STRONGYLIDAE**

Subfamily: Strongylinae (*Bidentostomum*, *Craterostomum*,
Oesophagodontus, *Strongylus*,
Triodontophorus)

Cyathostominae (*Caballanema*, *Coronocylus*, *Cyathostomum*,
Cylicocyclus, *Cylicodontophorus*,
Cylicostephanurus,)

Gyalocephalinae (*Gyalocephalus*)

CHABERTIIDAE

Subfamily: Chabertiinae (*Chabertia*)
Oesophagostominae (*Oesophagostomum*)

SYNGAMIDAE

Subfamily: Syngaminae (*Cyathostoma*,
Mammomonogamus, *Syngamus*)
Stephanurinae (*Stephanurus*)

Family: **STRONGYLIDAE**

Strongylinae and Cyathostominae of horses

- Includes both **large** and **small strongyles**
- **Most common**
- Commonly referred to as **bloodworms**
Because of color after it ingests blood from host animal

Genus: **STRONGYLUS**

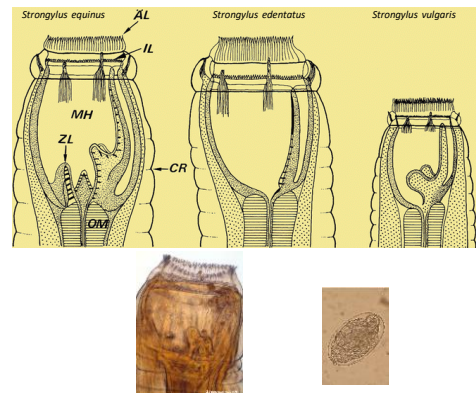
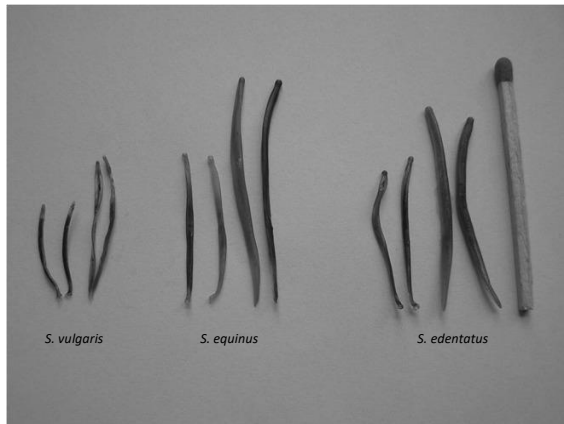
- Members of this genus live in the **large intestine** of horses and donkeys and, with *Triodontophorus*, are commonly known as **large strongyles**.
- **Site of infection:** Caecum and colon
- **Species:** *S. vulgaris*; *S. edentatus*; *S. equinus*
- post infection larvae – **parenteral migration in the host**

Genus: **TRIODONTOPHOROUS**

Members of this genus are **non migratory large strongyles** frequently occur in the large numbers in the colon and contributes to the deleterious effect of mixed strongyle infection.

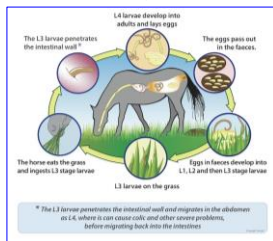
Species

- *T. serratus*
- *T. tenuicollis*
- *T. brevicauda*
- *T. minor*



LIFE CYCLE

➤ The adult parasites live in the caecum and colon (large intestine).



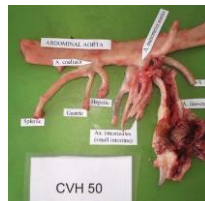
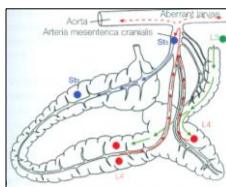
The life cycle of these worms involves the **migration of larvae through the blood vessels of the intestine and liver**, where they can cause **inflammation and obstruction of the blood vessels** resulting in damage to the intestine supplied by the damaged vessel.

Symptoms of infestation can include **colic, ill thrift, and diarrhoea**.



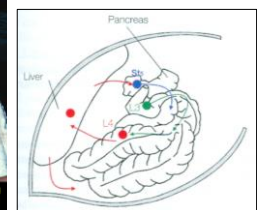
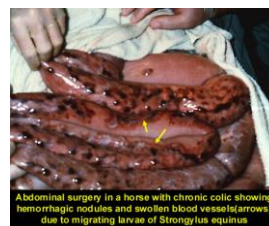
Strongylus vulgaris

- The L3 penetrate the intestinal mucosa and **moult to L4 in the submucosa**.
- These then **enter small arteries** and migrate on the endothelium to their predilection site in the **cranial mesenteric artery** and its main branches.
- After a period of several months the larvae **moult to L5 and return to the intestinal wall** via the arterial lumina.
- **Nodules** are formed around the larvae **mainly in the wall of caecum and colon**.
- The **Prepatent period is 6-7 months**.



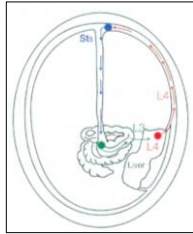
Strongylus equinus

- Exsheathed L3s **invade the wall of the small intestine, cecum and colon** and become **encapsulated in nodules** in which they moult to L4s by 12-14 days after infection.
- L4s leave their nodules and cross the peritoneal cavity to the **liver**.
- Finally, they begin to **migrate back to the large intestine** by leaving the liver and **crossing the abdominal cavity directly or by first passing through the pancreas** and then the abdominal cavity.
- **Prepatent period is approximately 9 months**.



Strongylus edentatus

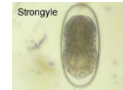
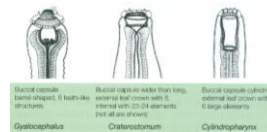
- L4 migrate to root of mesentery, some going to liver and lungs and then to wall of caecum and right ventral colon
- **Immature adults** then appear in lumen of these portions of the large intestine
- **Prepatent period - 11 Months**

**Subfamily: Cyathostominae - small Strongyles**

Genera: *Cyathostomum*, *Caballonema*, *Coronocylus*, *Cylicocylus*, ...

These parasites are present in the **large intestine** of horses.

Their effects on the host range from poor performance to clinical signs of **severe enteritis**.

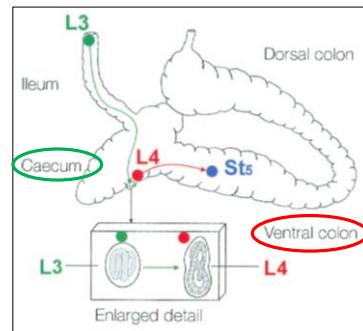
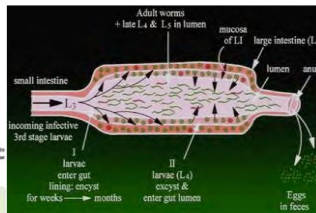
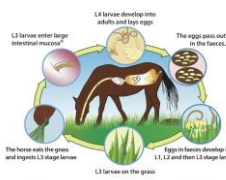
**LIFE CYCLE (Stages)**

Free living stages same as large strongyles.

L3 infective; exsheath in stomach, small intestine but **do not migrate - only forms nodules**

L3 enter walls of caecum, colon; develop and moult; return to gut lumen as early L5

Prepatent period - 4 to six weeks up to 3 to 4 Months.



Internal development

PATHOGENESIS

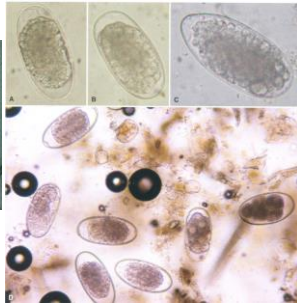
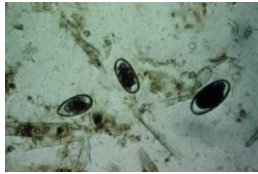
- **Granulomatous colitis**; masses of cyathostomin larvae embedded in mucosa = **bright red L4**
- May be large numbers of L4, early L5 in faeces with watery diarrhoea



Encysted larvae

Diagnosis of Strongyles

Fecal flotation
Necropsy



Treatment

- Benzimidazoles - BZ resistance shown by several spp; Moxidectin (best to date) approved for horses Ivermectin or other avermectins
- Pyrantel salt - probably some resistance

CHEMICAL CLASS	COMPOUND	DOSAGE	LARGE STRONGYLES		CYATHOSTOMINS	
			ADULTS	MIGRATING LARVAE	ADULTS AND LUMINAL LARVAE	ENCYSTED LARVAE
Benzimidazoles	Fenbendazole	5 mg/kg	Yes	Yes*	Yes	Yes [†]
	Oxfendazole	10 mg/kg	Yes	No	Yes	No
	Oxibendazole	10 mg/kg	Yes	No	Yes	No
Heterocyclic lactones	Piperazine	88 mg/kg	No	No	Yes	No
	Ivermectin	0.2 mg/kg	Yes	Yes	Yes	No
Tetrahydropyrimidines	Moxidectin	0.4 mg/kg	Yes	Yes	Yes	Yes [‡]
	Pyrantel pamoate	6.6 mg/kg	Yes [§]	No	Yes	No
	Pyrantel tartrate*	2.64 mg/kg/day	Yes	No	Yes	No

*Requires a regimen of 10 mg/kg daily for 5 consecutive days.

[†]Label claims for efficacy against early third-stage (EL₁), late third-stage (LL₁), and fourth-stage (L₄) cyathostomin larvae.

[‡]Label claims for efficacy against late third-stage (LL₁) and fourth-stage (L₄) cyathostomin larvae.

[§]Efficacy against Strongyle resistance is less than 90%.

[¶]When fed daily, aids in the control of certain nematode parasites.

Control Measures

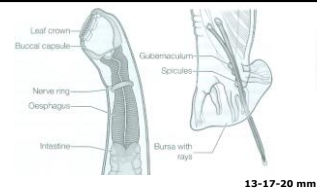
- Clean bedding in stalls
- Not running young foals with older, more immune horses
- Rotation onto "clean" pastures
- Strategic, regular treatment programs
- Alternate dewormer types annually (one type each year, using the same type each time a dewormer is administered in one year)
- Pasture contamination due to large number of parasites and eggs produced makes management difficult - it is practically impossible to remove all parasites from a horse's gut.

Superfamily: **STRONGYLOIDEA**

Family: **CHABERTIIDAE**

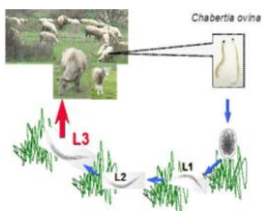
Subfamily: **Chabertiinae (Chabertia)**
Oesophagostominae (Oesophagostomum)

Gastrointestinal worms of ruminants and pigs



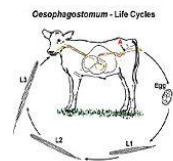
Chabertia ovina / CHABERTIOSIS

- catarrhal - nodular enteritis colitis (column wall - edematous, bloodied, thickened with haemorrhages)
- small and caecum - larval stages
- large intestine - adult worms most pathogenic - L5 and adult worms - drawing intestinal mucosa into the oral capsule (already with 300 worms)!
- diarrhea with blood
- anemia, weight



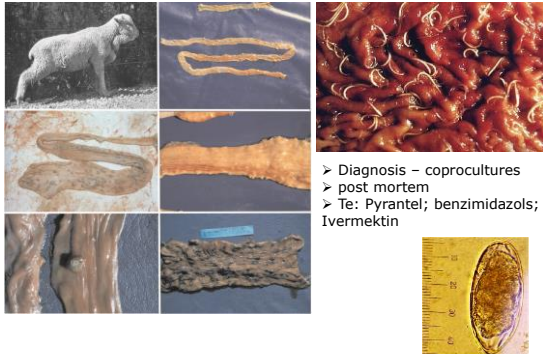
OESOPHAGOSTOMOSIS OF RUMINANTS

- **Oesophagostomum columbianum**
- **Oe. venulosum**
- **Oe. asperum**
- **Oe. multifoliatum**
- **Oe. radiatum**



- nodules in caecum (adult worms)
- larval stages: from pyloric to rectum
- direct development (prepatent period: about 45 days)
- severe enteritis (also *Oe. venulosum*, which does not form nodules)
- local inflammatory reaction - encapsulation
- impairment of normal intestinal motility and absorption
- thickening of the intestinal wall
- intensive mucus production

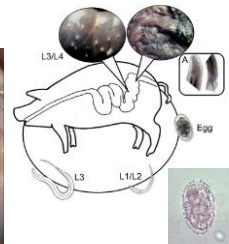


***Oesophagostomum columbianum* - ovce**

- Diagnosis – coprocultures
- post mortem
- Te: Pyrantel; benzimidazols; Ivermektin

**OESOPHAGOSTOMOSIS OF PIGS**

- *Oesophagostomum dentatum*
- *Oe. brevispiculum*
- *Oe. quadrispinulatum*



- prepatent period: 43-55 days;
- resistant larvae – survive in the environment for a year or more;
- Ivermectin, benzimidazoles

Superfamily: STRONGYLOIDEAFamily: **SYNGAMIDAE**

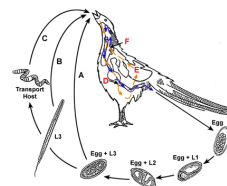
Subfamily:

Syngaminae (*Cyatostoma*,
Mammomonogamus, *Syngamus*)
Stephanurinae (*Stephanurus*)

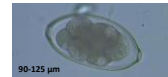
- respiratory tract of poultry
- kidney of pigs

***Syngamus trachea* / SYNGAMOSIS**

- 7-20 mm;
- forked worms; a male of 2-6 mm
- direct life cycle



- Prepatent period: 12-17-20 days
- Adult worms survive 23-147 days in chickens, 48-224 days in turkeys, 98 days in guinea fowl.



- winter season in paratenic hosts (earthworms, beetles, flies, snails, ...)
- disease of young birds (pheasants, turkeys, mustard, chickens, ...)
- petechial and hemorrhagic bleeds;
- mucosal inflammation;
- creation of knots;
- bronchopneumonia – migrating larvae;
- pulmonary edema;
- dg: eggs

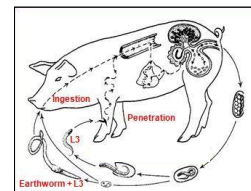
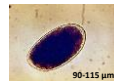
Te: benzimidazols, Ivermectin



- For aquatic poultry: *Cyathostoma bronchiale*
- male and female – live separately;
- already 2-3 worms can cause death by asphyxiation

***Stephanurus dentatus* / STEPHANUROSIS**

- 30-45 mm; 20-30 mm
- cysts in the kidneys
- inflammation of the abdominal and thoracic cavities



Prepatent period: 6-19 months
Adult worms live cca 2 years

- percutaneous infection - nodules in the skin, swelling and enlargement of regional lymphatic nodules;
- migrating larvae - abscesses and liver cirrhosis;
- adult worms - kidney cysts;

Dg: eggs in urine
Te: levamisole, ivermectin, benzimidazols



Superfamily: TRICHOSTRONGYLOIDEA

Family: Amidostomatidae (*Amidostomum*, ...)

Trichostrongylidae (*Cooperia*, *Graphidium*, *Haemonchus*, *Hyostrongylus*, *Marshallagia*, *Ostertagia*, *Teladorsagia*, *Trichostrongylus*, ...)

Molineidae (*Nematodirus*, *Ollulanus*, ...)

Ornithostrongylidae (*Ornithostrongylus*)

Helligmonellidae (*Nippostrongylus*, *Heligmosomoides*)

Dictyocaulidae (*Dictyocaulus*)

Genus: *Trichostrongylus*

Trichostrongylus spp. are widely distributed throughout the world.

- *Trichostrongylus* species are the smallest members of the family **Trichostrongylidae**.
- They are thin and with a length of 7 mm or less are difficult to see without a microscope

Epizootiology

- *Trichostrongylus* species are not usually primary pathogens in temperate regions of the world.
- Their role is usually contributory to parasitic gastroenteritis in which *Ostertagia* or *Haemonchus* are the primary pathogens in ruminants.
- However, in warmer subtropical areas *Trichostrongylus* species are important pathogens in grazing ruminants and counts of 10,000 or more worms per animal are not unusual in clinical outbreaks.
- **Hypobiosis** occurs at the L3 stage and is an important controlling feature of life cycles in temperate areas of the world.
- **Survival of preparasitic stages**
- Eggs and infective L3s are able to survive both heat and cold. In temperate zones of both hemispheres enough L3s may survive winter and produce clinical outbreaks of trichostrongylosis in grazing ruminants in early spring.
- The ability of *T. axei* to cross-infect between horses and ruminants may lead to amplification of *T. axei* infections in horses when mixed grazing of pastures with horses and ruminants is used as a parasite control measure.

Species of *Trichostrongylidae* and *Nematodirus*, autochthonous in Europe.

Predilection site of adults	Species (prepatent period in days of selected species)	Length of females (mm) ²	Prevalence ³		
			Cattle	Sheep	Goat
Abomasum	<i>Haemonchus contortus</i> (18-19)	20-30	(+)	+++	+++
	<i>Ostertagia ostertagi</i> (18-21)	8-9	+++	(-)	(-)
	<i>Ostertagia circumcincta</i> (17)	8-11	+++	(-)	(-)
	<i>Teladorsagia circumcincta</i> (16)	12-13	(+)	+++	+++
	<i>Trichostrongylus axei</i> (20)	9-11	(-)	+++	+++
	<i>Trichostrongylus colubriformis</i> (15)	5-6	+++	+++	+++
	<i>Trichostrongylus vitreus</i> (18-19)	5-6	(-)	+++	+++
	<i>Trichostrongylus capricornis</i>	5-7	(-)	+	+
	<i>Trichostrongylus longicaudatus</i>	5-7	+	+	+
	<i>Cooperia curatiae</i> (14)	5-6	+	+++	++
Small intestine	<i>Cooperia oncophora</i> (17)	8-12	+++	+	+
	<i>Cooperia zumbadoi</i> ⁴	7-9	+++	+	+
	<i>Cooperia pacchalis</i>	7-9	+	+	+
	<i>Cooperia punctata</i> (14)	8-2	+++	(-)	(-)
	<i>Nematodirus helvetianus</i> (21-28)	15-24	+++	(-)	(-)
	<i>Nematodirus filicollis</i> (21)	12-20	(-)	+++	++
	<i>Nematodirus battus</i> (15-28)	18-15	+	+++	++
	<i>Nematodirus sphegiger</i> (15-21)	12-20	(+)	++	++

¹ At necropsy, specimens of the species typically inhabiting the abomasum may be found in anterior parts of the small intestine, and those rarely the species from the small intestine in the abomasum.

² For ease of entry, only the body length of females is listed; males are a little shorter.

³ Approximate prevalence: +++ >70%, ++ >50%, + >20-50%, (-) <10%, (±) sporadic, empty table: no data.

⁴ Prevalence are related to wide variation; data predominantly from AE, DE and SE.

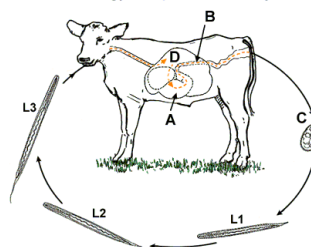
⁵ Syn. *Shastanaga*.

⁶ Formerly allocated to the genus *Ostertagia*. *T. microscopus* and *Ostertagia phrynos*, both occurring in sheep and goats, are morphologically very similar and described either as distinct species or as synonyms.

⁷ Alternative spelling: *C. zumbadoi*.

Life cycle

Trichostrongylus species - Life Cycles



Pathogenesis

- Inflammatory reactions of the abomasum (abomasitis)
- And small intestine (enteritis)

Clinical signs

- **Light infections** are usually asymptomatic but may contribute to poor appetites, diminished growth rates and soft faeces
- **Heavy infections** (10,000+ worms) will produce a diarrhoea that is serious and can be debilitating, especially in ruminants. The term "black scours" is sometimes used to describe the diarrhoea because it is often watery and dark green (almost black) in colour.

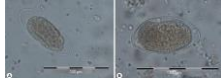
Data on the parasitic development of some trichostrongylid and *Nematodirus* species

Species (host animal)	Days p.i.	Development and prepatency M: moulting, L3: parasitic L3, L4: 4 th stage larva, St. 6: preadult 9 th stage	Stage of hypobiosis
<i>Haemonchus contortus</i> (sheep)	1-8	L3 → abomasum, crypts, some in lumen of gastric glands M → to L4, return to mucosa surface M → to St. 5, further development	L4
	9-11	prepatency	
	18	prepatency	
	18	prepatency	
<i>Ostertagia ostertagi</i> (cattle)	1-8	L3 → abomasum, lumen of gastric glands (mostly in fundus region) M → to L4	L4
	8-15	M → to St. 5 in lumen of gastric glands	
	>15	emergence from gastric glands to mucosa surface, further development	
	18	prepatency	
<i>Trichostrongylus colubriformis</i> (sheep)	1-4	L3 → small intestine → M to L4	L3
	5-10	M to St. 5	
	15	prepatency	
	15	prepatency	
<i>Cooperia curatiae</i> (sheep)	1-2	L3 → lumen of anterior small intestine	L4
	3-4	migration of L3 into crypts, M → to L4	
	5	most of L4 free in intestinal lumen	
	9-10	M → to St. 5, further development	
<i>Nematodirus battus</i> (sheep)	14	prepatency	
	14	prepatency	
	15-5	L3 → small intestine, M → to L4	?
	12	nearly all parasites in St. 5	
	14	prepatency	
	14	prepatency	

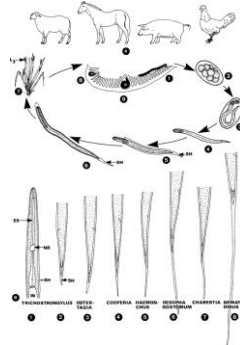
¹ Parasitic L3 = infectious larva after eclosion.

Diagnosis

- A diagnosis of trichostrongylosis is made on the basis of a combination of factors
- Clinical signs
- Seasonal occurrence of disease
- Necropsy findings
- Presence of Strongyle-type eggs in the feces of infected animals
- Fecal cultures to identify *Trichostrongylus* L3s
- *Trichostrongylus* eggs are "strongyle-type" and, as such, are not distinguishable from other g.i. nematodes that produce similar eggs.

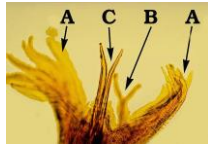


Light microscope view of *Trichostrongylus colubriformis* (A) and *Trichostrongylus axei* (B) eggs



Haemonchus

- The copulatory bursa (A) of the male is distinct because it has an asymmetrical dorsal lobe with
- a Y-shaped dorsal ray (B), which is sometimes confused with the spicules (C).



Genus: Haemonchus

- *Haemonchus* species are the largest of the nematodes found in the abomasum of ruminants.
- They range from 10-30mm in length and are reddish when fresh because they are blood suckers.
- They achieve this by using a tiny lancet in their small buccal capsule. Because they feed on blood, the female has a striking appearance, looking like a barber pole because the white ovaries wind around the red blood filled intestine.



Nematode species	Host species	Predilection site
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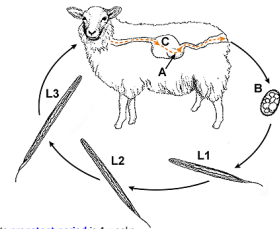
Haemonchus contortus
Haemonchus placei

sheep and goats
cattle

Abomasum
Abomasum

Haemonchus - Life Cycle

The predilection site is the abomasum of sheep and goats. "strongyle-type" eggs which pass to the external environment in host faeces.



The prepatent period is 2-3 weeks in sheep.
The cattle species, *H. placei*, has a similar life cycle but its prepatent period is 4 weeks.

Pathogenesis

- Haemonchosis is characterized by a hemorrhagic anemia attributable to blood loss via the blood-sucking activities of worms in the abomasum.
- Fourth stage larvae as well as adults are blood suckers which means that blood losses, sufficient to cause a clinically obvious anemia, may occur before an infection is patent.
- The actual mechanism of blood sucking involves the worm attaching to the mucosa and extruding its oral lancet to slit capillaries in the abomasal mucosa. They ingest blood flowing from these slit capillaries.
- Worms also secrete an anticoagulant into the bleeding lesion ensuring that these lesions will continue to bleed after the worm is replete and has moved away.

Trichostrongylosis in ruminants: selected pathophysiological features

Secretory disturbances of the abomasum (infections with <i>Ostertagia</i> , <i>Trichostrongylus</i> , <i>Haemonchus</i> and <i>T. axei</i>)
Reduced HCl secretion → elevation of pH → lower activation of pepsinogen to pepsin → reduced denaturation of proteins* → colonization of the abomasum with gram-positive and gram-negative bacteria → increased gastric secretion Etiology: secretory/secretory products of adult trichostrongylids inhibit HCl secretion of parietal cells (documented for <i>O. ostertagi</i> and <i>O. contortus</i>); possibly also long-term amines and prostaglandins, produced by infected hosts, could have an inhibitory effect; destruction of parietal cells, associated with massive inflammatory lesions of the mucosa, may contribute to reduced HCl secretion
Disturbed gastric secretion → hypergastrinemia → inhibited motility of the gastrointestinal system → "stalled food" Etiology: already at a slight reduction of HCl production, secretion of gastrin increases. Gastrin stimulates acid production of parietal cells and vagus-mediated secretion of chief cells; furthermore, gastrin can influence gastrointestinal motility, absorption and epithelial proliferation
Disturbed pepsinogen secretion → hypoparagastria Etiology: parasite products* and gastrin stimulate chief cells to increased secretion of pepsinogen. Pepsinogen is not activated in the abomasal lumen and passes through the mucosa to the blood circulation. Mucosa permeability is increased, probably due to the activity of mediators of inflammation. Inflammatory factors are upregulated in <i>O. ostertagi</i> infections of cattle
Blood loss (in haemonchosis) → Loss of haemoglobin and plasma protein into the gastrointestinal tract → anaemia, impairment of protein metabolism, energy production and other functions Etiology: blood losses due to blood feeding by L4, pre-adult and adult stages of <i>H. contortus</i> and taking of blood due to mucosal lesions. Blood loss per worm per day approximately 0.05 ml in haemonchosis of sheep with worm burdens of 2,000-2,000 per animal. Adult blood loss <0.5-1.0 ml
Protein loss (in abomasal and intestinal trichostrongylid infections) Leakage of serum proteins (especially albumin) into gastrointestinal tract, and increased gastrointestinal epithelial turnover → hypoalbuminemia → reduced oncotic pressure of blood → oedema formation in various tissues (edema of abomasum, subcutis, intermuscular spaces) → increased albumin synthesis in liver → later on mobilisation of protein from muscle tissue → emaciation, reduced utilization of protein for growth and production of meat, milk and wool → internalisation of protein for energy generation Etiology: leakage of plasma protein, mainly albumin, through damaged abomasal and intestinal mucosa; in spite of partial reabsorption of protein a net loss occurs, enhanced in heavy infections by increased loss of gastrointestinal epithelia * = consequences; evidence, but not yet confirmed

Clinical signs

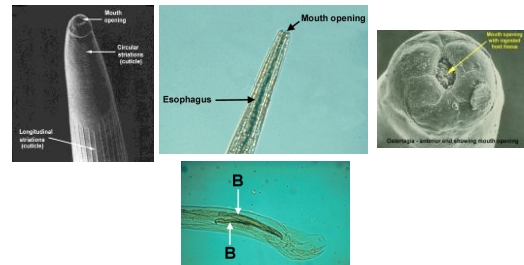
- In **hyperacute haemonchosis**, sudden deaths in a flock of previously healthy sheep are seen.
- This syndrome results from ingestion of **large numbers of infective larvae** by sheep grazing on a heavily contaminated pasture.
- In **acute haemonchosis**, grazing sheep develop a sudden onset anemia. In the absence of treatment, and if grazing continues, these animals will progressively worsen.
- The PCV drops initially, plateaus, then falls even further, signaling exhaustion of the erythropoietic system. Without treatment, death is the usual outcome



➤ ascites and submandibular edema - "bottle jaw"

Genus: *Ostertagia*

- small (~10mm) reddish brown worms found in the abomasum of ruminants.
- On a global basis they are the major cause of parasitic gastritis (**Ostertagiosis**) of ruminants in temperate climates.



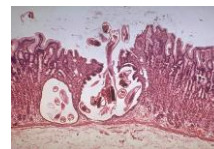
Nematode species	Host species	Predilection site
<i>Ostertagia ostertagi</i>	cattle	Abomasum
<i>Ostertagia circumcincta</i> (<i>Teladorsagia</i>)	sheep and goats	Abomasum
<i>Ostertagia trifurcata</i>	sheep and goats	Abomasum

Other, less common species include *O. lyrata*, and *O. kolchida* in cattle plus *O. leptospicularis* found in cattle, sheep and goats.

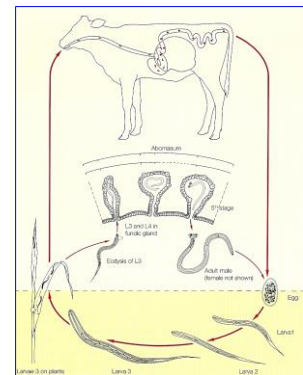
Ostertagia species are found throughout temperate and subtropical areas of the world.

The cattle species, *Ostertagia ostertagi* is particularly important in temperate areas wherever cattle are raised.

Life cycle



Larva emerges from the gastric glands



Epizootiology

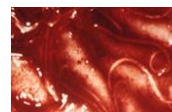
The epidemiological patterns of ostertagiosis have been well defined in most areas of the world. There are also four common features that influence these epidemiological patterns despite the wide range of climates and management conditions under which cattle are raised.

1. The influence of temperature and moisture on the survival and development of preparasitic stages of the life cycle.
2. The different ways in which pasture is used to raise dairy replacement heifers and beef calves.
3. The influence of seasonal arrested development (hypobiosis) on the parasitic phase of the life cycle.
4. The influence of the immune response on infection and disease.

Pathogenesis



Nodules in mucosa



Ostertagiosis type I and type II in cattle

	Ostertagiosis type I 'summer ostertagiosis'	Ostertagiosis type II 'winter ostertagiosis'
Season	July until end of grazing season	January until May
Animal group	young animals during their first grazing period	young animals after their first grazing period, rarely cattle after several grazing periods
Mortality	high, majority or all young animals of a herd more or less affected, older cattle usually without clinical signs	usually only a few animals affected
Morbidity	usually low, high in some years	low
Clinical signs?	mostly low	high
Prognosis	+	+
Diagnostics	+ (profuse, without blood)	+ (profuse, occasionally intermittent)
Weight loss	+	+ (rapid and marked)
Eosinophilia	+	+ (rare)
Subcutaneous oedema	+	+ (approximately 40% of clinical cases)
Fever	+	+
Anaemia	+	+ (in some cases)
Serum proteins	decreased	mostly decreased
Serum albumin	in severe cases decreased	mostly decreased
Pathology		
Egg detection in faeces	positive	positive or negative
Eos	low up to <1000	very low to high
Worm burden in abomasum	predominantly adults of <i>O. ostertagi</i> (often >40,000 specimens/animal)	in faeces infections up to >100,000 specimens with high proportion (50-80%) of hypobiotic and other immature stages
Pathology	abomasum: typical nodules, inflamed mucosa, oedema of abomasal folds, pH elevated in severe cases (2)	like in type I, usually less nodules, but marked oedema, pH often elevated (>3)
Therapy	broad spectrum anthelmintics effective	only certain broad spectrum anthelmintics effective against hypobiotic stages (► Table 19.2, p. 676)

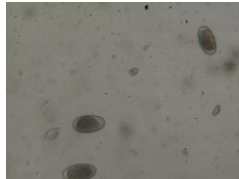
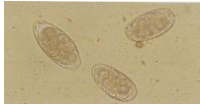
¹ Modified after Arnold et al. (1978) Bovine ostertagiosis. In: Chapman DM, Dennis J (eds) *Health and disease of cattle, sheep and horses in Europe*. Glasgow: Vet. Resour. Information Centre, pp. 11-22. ISBN 0-9502023-0-0.

+, usually present; -, usually absent.

Relevant, regularly, C. amoebae and some other species are involved in the infection, but detrimental effects caused by *O. ostertagi* predominate.

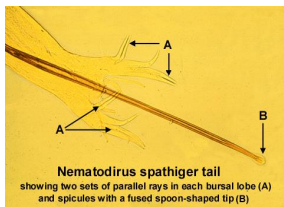
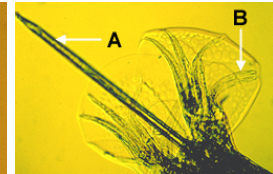
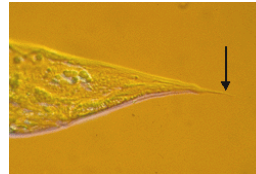
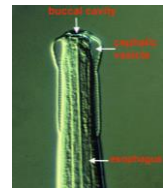
Diagnosis

- The presence of **strongyle type eggs** in the faeces of affected animals will merely confirm the presence of adult strongyle nematodes in the g.i. tract.
- In Type I disease, egg counts greater than 1,000 EPG are usually seen but counts are much more variable in Type II disease.



Genus: *Nematodirus*

- These are slender, relatively long worms (up to 2.5 cm long). As the accompanying image shows, they are distinguished morphologically by an inflated cuticle around the anterior end which may also show **transverse striations**.
- Large numbers of these long slender worms are usually seen at necropsy, in the small intestine, as clumps resembling cotton wool.



Nematodirus spathiger tail showing two sets of parallel rays in each bursal lobe (A) and spicules with a fused spoon-shaped tip (B)



Nematodirus female showing blunt tail with attached spine

Nematode species	Host species	Predilection site
<i>Nematodirus battus</i>	sheep	small intestine
<i>Nematodirus spathiger</i>	sheep and goats	small intestine
<i>Nematodirus filicollis</i>	sheep and goats	small intestine
<i>Nematodirus helvetianus</i>	cattle	small intestine
<i>Nematodirus abnormalis</i>	sheep and goats	small intestine

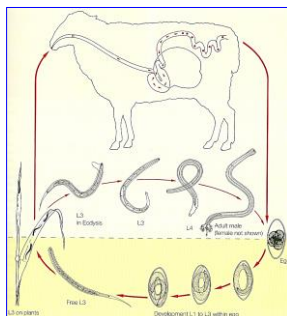
Life-cycle

Nematodirus is of special importance as a parasite of lambs in temperate regions.

Its hosts are ruminants, and its predilection site is the small intestine.

Nematodirus species are distributed worldwide, but more commonly in temperate zones.

Preparasitic phase of development occur within the egg.



Epizootiology

- The most important factor influencing transmission of *Nematodirus* species is the ability of L3s to **survive from year to year** enclosed inside their protective egg shells.
- Arrested development has been recorded in sheep (Canada, United States) and has been attributed to hypobiosis at the L4 stage as a mechanism for survival over winter.

Pathogenesis and clinical signs

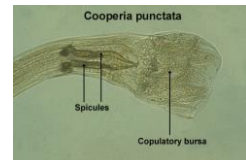
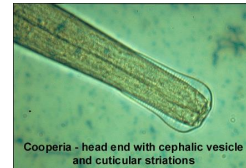
- Nematodirus is not usually a primary pathogen in ruminants. Its importance therefore is as an **additive** affect in mixed infections of nematodes causing **parasitic gastroenteritis**.
- Diarrhea followed rapidly by dehydration are the primary clinical signs. Adult ewes have developed a strong immunity to *Nematodirus* and are usually unaffected.

Diagnosis



Genus: Cooperia

Cooperia species are nematodes of the small intestine of ruminants. Species in domestic animals are usually 5-9 mm long and males have a prominent bursa in relation to their size.

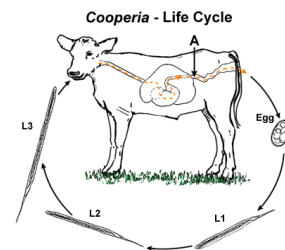


Nematode species	Host species	Predilection site
<i>Cooperia curticei</i>	sheep and goats	small intestine
<i>Cooperia oncophora</i>	cattle	small intestine
<i>Cooperia pectinata</i>	cattle	small intestine
<i>Cooperia punctata</i>	cattle	small intestine
<i>Cooperia surnabada</i>	cattle and sheep	small intestine

Life cycle

Following ingestion of infective L3s, the larvae reach the small intestine and develop into adults.

The **prepatent period** varies from 15 to 18 days.



Cooperia are generally considered to be mild pathogens. They contribute secondary effects to the primary pathogens;

A variety of clinical signs have been attributed to *Cooperia* species and these include: **diarrhea, weight loss, anorexia and poor weight gains**;

Eggs of family Trichostrongylidae



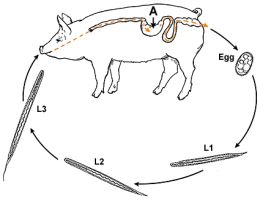
Genus: Hyostrongylus

The only species of importance in domestic animals is *Hyostrongylus rubidus*, commonly called the **"red stomach worm"** of pigs. It is a slender worm, 5-10 mm in length.



Life cycle

Hyoststrongylus rubidus - Life Cycle



➤ Pathogenic effects of *Hyoststrongylus* infections are similar to *Ostertagia* in cattle.

Therapy - trichostrongylidosis

- Benzimidazoles (albendazole, fenbendazole, mebendazole, oxfendazole)
- Imidazothiazoles (levamisole)
- Macrocyclic lactones (ivermectin, doramectin, eprinomectin, moxidectin)
- Amino-acetonitril derivatives (monepantel)
- Spiroindoles (derquantel)