

OTHER IMPORTANT NEMATODE PARASITES

Below are descriptions of other important nematode (roundworm) parasites - which can cause significant disease but less commonly than those, mentioned above.

SMALL INTESTINE

COOPERIA CURITICAE

Description: Small intestinal worm. 0.5 to 0.8 cm. Eggs are typical of the Trichostrongyloidea Superfamily.

Epidemiology: This parasite can also infect goats. The life cycle is typical with the larvae burrowing into the intestinal crypts and adults living on the surface. Prepatent period is 2 weeks. *Cooperia* becomes hypobiotic (arrested) in the late fall. Lambs/kids at pasture are most likely to develop heavy infections. Adults tend to remain immune but shed low numbers of eggs.

Clinical Signs: Signs are mild or absent unless infection is very heavy in which case poor appetite and growth are most notable.

Post Mortem: Parasite numbers may be high, even without severe signs of disease. The parasite causes villous atrophy of the intestine.

BUNOSTOMUM TRIGONOCEPHALUM

Description: "Hookworm". It is fairly large at 1 to 3 cm in size and the adults live in the small intestine. Eggs are typical of the Trichostrongyloidea Superfamily.

Epidemiology: This parasite can also infect goats. At this time, we have found no evidence of this parasite so it is likely not common in central Canada. The infective L3 can penetrate the skin where they migrate to the lungs and then migrate to the digestive tract. Ingested L3 do not migrate to the lungs. The prepatent period is 4 to 8 weeks. This parasite prefers more tropical climates.

Clinical Signs: Adult hookworms suck blood and a fairly low infection of 500 worms is associated with anaemia, hypoproteinemia (bottle jaw), weight loss and death.

Post Mortem: The carcass is pale. The intestinal lining is reddened and edematous. The worms may be seen attached to the intestinal mucosa or in the lumen of the intestine.

STRONGYLOIDES PAPILLOSUS

Description: "Threadworm". Very slender worms < 1 cm in length. The eggs are larvated and are about 50% as large as typical trichostrongyle eggs.

Epidemiology: It is very commonly found in diagnostic samples. This parasite can infect other ruminants. Only the females have a parasitic stage and both females and males are free-living. The females can produce eggs by parthenogenesis (asexual reproduction). To build up significant infestations in the environment, the conditions must remain warm and moist as the larval stages are all susceptible to environmental conditions. L3 infective larvae can be ingested, penetrate skin or infect lambs through ewe's milk. The pre-patent period is 8 to 14 days. While eggs are commonly seen on faecal egg counts, disease is typically uncommon and not severe.

Clinical Signs: In very high levels of infection, young lambs/kids (2 weeks of age) may exhibit signs of diarrhea and reduced gains. High faecal egg counts are not always indicative of a clinically significant infection.

Post Mortem: The adult parasite can cause inflammation of the intestine and villous atrophy.

CHABERTIA OVINA

Description: “Large-mouthed bowel worm”. The adults are 1.5 to 2 cm in length and found in the colon.

Epidemiology: This parasite also infects goats. Most sheep/goats have a low level of infection but as little as 300 adult worms can cause disease. The adults ingest chunks of mucosal tissue and cause loss of blood and protein. The L₃ can over-winter on pasture and the L₄ can become hypobiotic and re-emerge in the spring. However, severe disease is unusual in temperate climates.

Clinical Signs: Severe infections cause diarrhea with blood and mucus, sometimes with visible worms. The lamb/kid develops anaemia, hypoproteinemia and weight loss.

Post Mortem: The young larvae are in the caecum and the adults in the colon. The damaged mucosa is evident along with visible worms.

OESOPHAGOSTOMUM COLUMBIANUM

Description: “Nodular worm”. The adult worms are slightly > 1 cm in length and found in the large intestine. Eggs are typical of the Trichostrongyloidea Superfamily.

Epidemiology: This parasite also infects goats and wild ruminants. It is considered an important parasite in tropical and sub-tropical countries but is found worldwide. The L₃ penetrate the mucosa of the small or large intestine and form nodules where they develop to the L₄ stage. They may remain in the nodules for up to 1 year. When the L₄ emerge, considerable damage may be caused to the intestinal wall. The pre-patent period is 45 days.

Clinical Signs: Severe infections are typified by dark, green diarrhea; milder infections by intermittent diarrhea and poor growth.

Post Mortem: The nodules are found in the lower intestine and can be up to 2 cm in diameter. The inflammation associated with rupture of the nodules can cause adhesions and even perforation resulting in peritonitis.

TRICHURIS OVIS

Description: “Whipworm”. The adults are very long worms (4 to 8 cm) with a thick posterior and very slender anterior which is usually buried in the mucosa of the large intestine. Eggs are oval with a transparent plug at either end.

Epidemiology: This parasite also infects goats and occasionally other ruminants. The L₁ remains in the egg in the environment and is the infective stage. Eggs can remain viable in the environment for up to 4 years. Once ingested, the caps on the eggs are digested, releasing the larva which then penetrates the lining of the distal small intestine and large intestine. The pre-patent period is 7 to 10 weeks. Infections are quite common in central Canada but significant disease is not.

Clinical Signs: Most infections are light and there are few clinical signs. The parasite causes a mild colitis.

Post Mortem: The adult parasite is easily seen. The lining of the colon is haemorrhagic around where the worm is imbedded.

LUNG

DICTYOCAULUS FILARIA

Description: “Lungworm”. The adult worms live in the large and smaller airways (bronchi) of the lungs and are quite large (5 to 10 cm). While described as being found worldwide, infections are sporadic and uncommon in Canada.

Epidemiology: This parasite also infects goats and deer. The females lay eggs in the airways, which are coughed up and swallowed. The eggs hatch and the L₁ larvae are passed in the faeces where they moult to L₃. A routine faecal examination may miss them because the eggs have hatched - a special test (Baermann technique) is needed to diagnose an infection. Larvae can over-winter on pasture but most pasture contamination comes from infected sheep and goats in the same grazing season.

Clinical Signs: Coughing (with mucus) and ill-thrift are the most common signs. Secondary pneumonia can exacerbate the signs.

Post Mortem: The lungs are wet and red and the bronchi, and potentially trachea, are filled with worms.

MUELLERIUS CAPILLARIS

Description: “Nodular lungworm”. Although 1 to 3 cm in length, these worms are hard to see because they are located in the lung tissue.

Epidemiology: This parasite also infects goats and deer. The life cycle requires an intermediate host, in this case snails and slugs. The L₁ larvae are coughed up and swallowed, then passed in the faeces. They penetrate the foot of the mollusk, mature and reside there for 2 to 3 weeks until eaten by a sheep or goat. The mollusc is digested releasing the L₃ larva which penetrates the digestive tract and travels through the blood and lymphatic system to the lungs. The prepatent period is 6 to 10 weeks. The adult worm can live for several years, with the result that infection can be cumulative in sheep and therefore the lung damage can as well.

Clinical Signs: This parasite is not considered to be very pathogenic in sheep, but goats appear to express disease more. Coughing, dyspnea and pneumonia can be seen. Radiographs reveal increased density (white) dorsally in the lung suggesting disease due to *Muellerius* and / or Caprine Arthritis Encephalitis.

Post Mortem: The dorsal surface of the lung has nodules from very small up to 2 centimetres in diameter. The nodules are hard (lead shot) and contain one to several worms. When the worm dies, the nodules become necrotic. More extensive areas of lung consolidation occur in goats.

NERVOUS SYSTEM

PARELAPHOSTRONGYLUS TENUIS

Description: “deer meningeal worm”, “moose sickness”. These are long, slender worms up to 9 cm in length.

Epidemiology: The final host is the white-tailed deer, but many other species of ruminants can be infected - in particular, sheep, goats, moose and new world camelids. The intermediate hosts are snails and slugs. Normally, when deer ingest an infected snail or slug, the worm migrates up the spinal nerves from the gut - eventually reaching the brain and spinal cord. Eggs are laid in the small blood vessels and make their way to the lungs where they hatch, and larvae are coughed up, swallowed and passed in the faeces. It is difficult to control this disease as the parasite is common in white-tailed deer, and the snails and slugs are ubiquitous. Preventing the sharing of pastures is very difficult in Ontario and Quebec where white-tailed deer are very common.

Clinical Signs: The infection in white-tails is usually sub-clinical, but in non-target ruminants, severe

neurological signs may occur. This is because of aberrant (misdirected) migration in the non-target host. The worms migrate down trunk nerves or into the brain causing inflammation. The animal becomes disorientated and may develop severe pruritus (itching) along the trunk nerves. Moose may wander into towns, staggering. Sheep and goats may develop paralysis - particularly in the hind-end, circling, blindness, weight loss and death. Treatment often involves long term use of anthelmintics as well as anti-inflammatory drugs.

Post Mortem: Presence of the worm in the brain or spinal canal. In Ontario, most aberrant infections are seen in alpacas and llamas but may not be recognized in sheep or goats without a post-mortem diagnosis.



NON-NEMATODE INTERNAL PARASITES

There are many other types of internal parasites that cause significant disease in sheep in central Canada. Below includes descriptions of three types: protozoa, which includes coccidia (*Eimeria*) and *Cryptosporidium*; cestodes, both adult tapeworms and larval (intermediate stage) tapeworms; and liver flukes. The lifecycles are described under each parasite.

PROTOZOA

CRYPTOSPORIDIUM PARVUM

Description: “**Crypto**”. A microscopic protozoal parasite of the small intestine.

Epidemiology. The parasite also infects cattle, goats, horses, deer and humans. It is zoonotic and may cause severe gastrointestinal disease in people. This is a coccidial organism with a similar life cycle to *Eimeriaspp* but it takes only a few days to complete the life cycle in animals and produce oocysts (eggs). The oocysts are infective in fresh faeces, are very resistant to environmental factors, and build-up quickly in lamb-rearing areas.

Clinical Signs: Diarrhea in lambs/kids, usually greater than 1 week of age up to 3-4 weeks. Affected lambs/kids can become very unthrifty because of villous atrophy, malabsorption, and maldigestion. They can become severely dehydrated, depressed and cachexic, and in some outbreaks this parasite can be a significant cause of lamb/kid death. There is no effective treatment available in Canada. It also infects humans so great care should be used when handling sick lambs.

Post Mortem: Lesions are mild and usually only seen in the ileum.

COCCIDIOSIS CAUSED BY EIMERIA SPECIES

Coccidiosis – because it is a very important parasitic disease, will be covered in a separate section below.

TAPEWORMS (CESTODES)

MONIEZIA EXPANSA

Description: “**Sheep tapeworm**”. The adult is found in the small intestine. It is white and comprised of segments (egg packets) 1 to 1.5 cm wide, and a scolex (head) which is anchored to the intestinal wall. It can be quite long with many segments. The eggs are triangular shaped and easily identified on faecal examination. Each egg contains one embryonic tapeworm.

Epidemiology: This parasite also infects goats. The life cycle requires an intermediate host (as do all

tapeworms), and in this case, the intermediate host is a free-living forage mite. The eggs are passed in the faeces of the sheep and goat and the forage mite ingests them. The eggs then hatch and the larvae migrate to the body cavity of the mite where they develop into a cysticercoid (a tapeworm head in a solid structure). When the mites are ingested by sheep, they develop into adults. Ingestion to egg production in sheep takes about 6 weeks. Interestingly, the adult tapeworms do not live long - approximately 3 months. Infection is usually worse in summer months but the cysticercoids can overwinter in the mites

Clinical Signs: This tapeworm is generally believed not to cause significant disease in sheep and goats. However, a severe infection can be associated with diarrhea and unthriftiness, and occasionally the volume of parasites in the gut is associated with intestinal blockage and may be a risk factor for *Clostridium perfringens* Type D infection (pulpy kidney, also called enterotoxaemia).

Post Mortem: The parasite is easily seen but, unless very numerous, is not associated with any pathology. However, because lambs often pass large segments - producers see large white “worms” in the faeces and assume that the lamb is suffering from their presence.

LIVER FLUKES (TREMATODES)

FASCIOLA HEPATICA

Description: “liver fluke”. This is a parasite of the liver and bile ducts and is leaf-shaped and flat, about 2.5 to 3.5 cm in length as adults. The egg is large and has an operculum at one end.

Epidemiology: The definitive hosts are sheep, goats, cattle, horses and deer. The intermediate host is an amphibious snail of the *Lymnaea* genus. The snails prefer wet, low lying land and so the disease is associated with such pastures. Fasciola has been reported in Quebec, but not in Ontario (yet). It is also reported in upper New York State. The adult fluke lays eggs in the bile ducts of the liver, which are shed in the faeces. The eggs hatch under warm conditions and produce a miracidium which must penetrate a suitable snail within 3 hrs of hatching. It then divides and develops into as many as 600 cercariae. These cercariae are shed from the snail, and attach themselves to blades of grass where they encyst as metacercariae and are more resistant to the environment. The sheep or goats consume the metacercariae while grazing, which then migrate through the intestinal wall to the liver. The young flukes wander through the liver for about 2 months before moving into the bile ducts where they mature to egg laying adults. The adult flukes may survive for years in the animal. The prepatent period is 10 to 12 weeks.

Clinical Signs: The disease may be acute, sub-acute or chronic depending on the number of metacercariae ingested and the stage of the disease. If several thousand infect the liver, the damage can be so severe that bleeding and secondary clostridial infections of the liver may occur. Subacute disease is associated with ingestion of smaller numbers of metacercariae (500 to 1500) and disease is evident about 6 to 10 weeks later (late fall, early winter) with bile duct inflammation as well as damage to the liver. The animal has severe anaemia and hypoproteinemia (bottle jaw) and if untreated, will die within 1 to 2 weeks. The chronic form is the most common and is seen in late winter to early spring, 4 to 5 months after ingesting 200 to 500 metacercariae. Again, anaemia and hypoproteinemia are the main presenting signs - but in this case, the fluke eggs can be demonstrated in faeces. Sheep and goats do not develop immunity so any age can be affected. Diagnosis can be aided by blood tests that detect evidence of severe liver damage.

Post Mortem: The liver is enlarged and may be haemorrhagic in the acute form, and scarred and pale in the chronic form. The flukes can be seen in the liver and bile ducts.

Treatment: Regular anthelmintics (fenbendazole, ivermectin) are not effective against flukes. Albendazole double dose is effective against adult stages only. Triclabendazole (Fasinex), which is effective against the immature stages as well as the adults, is available only with an emergency drug release.

FASCIOLOIDES MAGNA

Description: “**large American liver fluke**”. This is a very large fluke - up to 10 cm (3”) in length.

Epidemiology: This parasite is usually found in deer and moose, but sheep and goats can be affected. The intermediate host is a freshwater snail. The fluke migrates through the liver and causes haemorrhage. Sometimes the flukes are found in the lungs or free in the abdomen. They do not produce eggs in the faeces. This infection is common in deer in the northern Great Lakes region and eastern Manitoba. Sheep flocks and goat herds in these regions often become infected. Since the snail prefers wet, stagnant boggy pastures, it is best to prevent grazing of these kinds of lands.

Clinical Signs: Sudden death in sheep and goats grazing wetlands that infected deer also graze. Several outbreaks that involve animal death have been reported in Manitoba and north-western Ontario.

Post Mortem: Haemorrhage of the liver. To detect the flukes, it is necessary to slice the liver up thoroughly as they may be anywhere in the parenchyma; they do not enter the bile ducts and no eggs are shed into the faeces.

DICROCOELIUM DENDRITICUM

Description: “**small lanceolate fluke**”. This fluke is very small < 1 cm and pointed in appearance.

Epidemiology: The final hosts are sheep, goats, cattle, deer and rabbits. The intermediate hosts are firstly a land snail which, like other flukes, produces the cercariae - but then these are ingested by ants. The resulting metacercariae infect the brain, causing the ants to act very crazily and to climb to the top of blades of grass, where they are more likely to be eaten by grazing ruminants. The flukes are very long-lived and infections can be cumulative in bile ducts. The eggs are fluke-type and can be identified in the faeces. This fluke has been reported in southern Ontario. Because of the sylvatic cycle in wildlife, and the fact that land snails are the intermediate host, it may be difficult to avoid infection if present in a geographic location.

Clinical Signs: It is not considered to be very pathogenic to sheep or goats but heavy infections may cause ill thrift. One case has been reported of mortality associated with copper toxicity, presumably triggered by liver damage from the parasite.

Post Mortem: There is no parenchymal migration with this fluke. The damage in severe infections is due to bile duct scarring and secondary cirrhosis. The livers are condemned.

