

INTRODUCTION

The intent of this handbook is to inform producers, veterinarians and extension personnel regarding some of the basics of controlling internal parasites that affect sheep and goats in Canada. It is not meant to replace the unique relationship between the veterinarian and the producer and his / her flock or herd, but only to augment the information available to them.

This handbook was written primarily for those working with sheep and goat farms in central Canada, i.e. Ontario and Quebec where recent research carried out by several research institutions has helped us to understand the epidemiology of internal parasites in this region. However, this information should also prove useful to those living in other parts of Canada - particularly those with a similar temperate climate. This handbook will always be a work in progress as we better understand parasitism of sheep and goats in this country. We hope that you will find it of use in developing internal parasite control programs for sheep flocks and goat herds.



IMPORTANT GASTROINTESTINAL NEMATODE PARASITES

This section contains a description of the most commonly found and important gastrointestinal nematode (GIN) parasites of sheep and goats found in Canada. Information on other important but less common nematode parasites of the gastrointestinal tract, lung and nervous system, as well as other internal and external parasites are to be found towards the end of the handbook. Unless indicated, the life cycles of the GIN are similar and described in Figure 1. In central Canada, the most common and the most pathogenic (disease causing) nematode parasites of sheep and goats are *Teladorsagia circumcincta*, *Trichostrongylus* spp and *Haemonchus contortus*. *Nematodirus battus* which is also highly pathogenic has been reported in sheep in Canada, in particular Nova Scotia - but, while *Nematodirus* spp infection is common in central Canada, most infections seem to be with the less pathogenic species.

HAEMONCHUS CONTORTUS

WHAT DOES IT LOOK LIKE?

Also called the “**Barber pole worm**”, “**Blood worm**”, or “**Wire worm**” The worms are large (1.5 to 3.0 cm), easily visible to the naked eye and the female oviduct is visible as a white stripe around the red blood-filled intestine, giving a barber-pole appearance. It is found in the abomasum. Eggs are typical of the Trichostrongyloidea Superfamily.

Trichostrongyle type egg



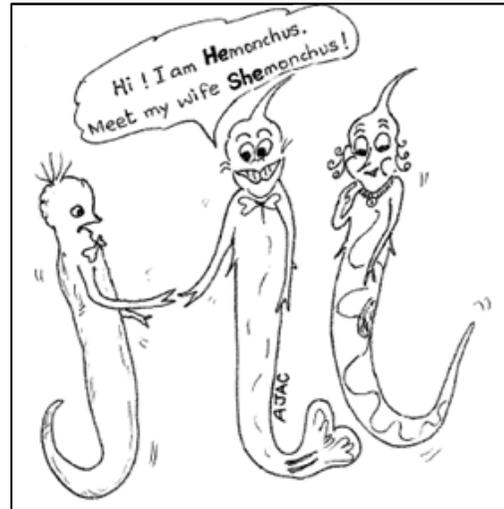
HOW DOES IT LIVE?

This parasite of sheep and goats also infects llamas, deer and occasionally cattle. It does not survive well overwinter on pasture in our Canadian climate. The form that overwinters outside the animal on pasture is the 3rd stage larvae (L3) and although some may be found in the spring in the pasture grasses, passed in the faeces of sheep the previous grazing season; they are weak and are not very successful at establishing infection in an animal.

However it is a very prolific egg producer - each female worm can produce several thousand eggs every day - enabling it to rapidly increase the pasture contamination to severe levels by mid-July. Pasture contamination comes from two sources: infected lambs are the worst, but early contamination comes from ewes where the parasite has overwintered in the ewe and starts to produce large numbers of eggs in the spring at lambing.

The eggs can develop to the infective L₃ stage in as little as 5 days (see Figure 1) but require fairly warm temperatures to do so. Egg development may be delayed up to 2 months if the weather is cool. The L₃ larvae can survive for months on pasture under moist conditions.

Severe disease outbreaks usually occur mid-July to August in lambs as well as adults on pasture but the exact time depends on the air temperature (i.e. prefers > 25° C) and moisture. So outbreaks occur earlier if the summer is hot and wet, and later in cool summers. Hot and dry conditions are not favourable to survival of L₃ on pasture. Finally, many of the ingested L₃ larvae become “arrested” in the abomasum, starting in early fall and do not complete development until the following spring around lambing-time (periparturient).



DISEASE IN THE SHEEP / GOAT

The adult worm has a lancet mouth piece which enables it to pierce the mucosal surface of the abomasum. It then feeds on the blood that seeps from the blood vessels. One worm can result in the loss of 0.05 ml blood per day from ingestion and seepage from the wounds they make. A load of 5,000 worms will cause a loss of 250 ml (1 cup) daily. A 25 kg lamb or kid (55 lb) only has 2000 ml of blood in total. However, clinical disease can occur with a load as few as 500 worms. In the course of a few weeks, infected young-stock will become severely anaemic from this blood loss. The bone marrow tries to compensate by increasing red blood cell production, but it is often a losing battle. The animal may drop dead on pasture with very severe infections (e.g. 30,000 worms). Animals with lower levels of infection will be chronically anaemic, have low protein (hypoproteinemia) and “bottle jaw”, and have a poor appetite and weight loss. The conjunctival mucous membranes around the eye appear pale pink to white. The haematocrit (a measure of the proportion of blood that contains red blood cells) is often less than 12%, indicating severe anaemia (normal 25% to 35%).

In Ontario, **Type II Haemonchosis** often occurs in the spring, before the animals go to pasture. Ewes or does may develop severe disease in late April and early May, usually around lambing / kidding or when nursing. This is because of the large number of parasites that they picked up on pasture the previous grazing season and which overwintered in their abomasum, become active in the spring causing severe disease. These animals may not even have eggs in the faeces, because the parasites are not yet adults.

POST MORTEM

The carcass is very pale due to the anaemia. The abomasum contains numerous visible worms (2,000 to 20,000). The contents are dark brown from the seeping vessels and excreted digested blood. In severe, acute infections the abomasal mucosa may appear haemorrhagic. The marrow of the long bones is often very red from the response to the anaemia.

TELADORSAGIA CIRCUMCINCTA

WHAT DOES IT LOOK LIKE?

A small parasite, found in the abomasum, approximately 1 cm long, and barely visible to the naked eye. "**Brown stomach worm**". Also known as "Ostertagia". Eggs are typical of the Trichostrongyloidea Superfamily.

HOW DOES IT LIVE?

This parasite infects sheep and goats. Most severe infections occur in the late summer or fall but occasionally severe disease is associated with the re-emergence of the arrested L₄ larvae in the spring ("Type II Teladorsagiosis"). The latter is seen less commonly than with *Haemonchus*. The L₃ are well adapted to survive over winter on pasture in this climate and do so very successfully! The arrested stage carried in ewes and does will develop in the spring and contribute significantly to pasture contamination with the periparturient rise in egg output.

DISEASE IN THE SHEEP / GOAT

Disease is almost only seen in lambs and kids during the first season on pasture. Infection is associated with intermittent diarrhoea, weight loss or reduced gains, decreased appetite, and occasionally protein loss resulting in fluid accumulation under the jaw known as "bottle jaw". Plasma pepsinogen (a digestive enzyme in the abomasum) levels may be elevated due to abomasal damage, and the pH of the abomasum is elevated (normal is 2.0 - 2.5) because of damage to the glandular cells that secrete hydrochloric acid. This interferes with digestion and contributes to ill-thrift or weight loss. A load of 5,000 worms is considered to cause significant clinical disease. It is possible for severe disease to occur prior to eggs appearing in the faeces: lambs or kids put to heavily contaminated pastures may experience severe disease due to the sudden massive infection; if sheep or goats are put in the barn in the fall carrying massive numbers of arrested L₄, Type II disease may occur in the spring when the L₄ emerge.

POST MORTEM

The parasites invade the mucosa of the abomasum and cause swelling and redness of the abomasal folds. Scarring will occur with loss of gastric function - sometimes permanently in severe infections. The appearance of the lining of the abomasum is likened to Moroccan leather or a bird's eye pattern (swirls around a knot, which is the scar left by the worm).

TRICHOSTRONGYLUS AXEI

WHAT DOES IT LOOK LIKE?

"**Stomach hairworm**". An abomasal parasite. The worms are < 0.5 cm in length and very difficult to see with the naked eye. Eggs are typical of the Trichostrongyloidea Superfamily.

HOW DOES IT LIVE?

It is a parasite of sheep and goats but also infects cattle and deer. Like *T. circumcincta*, disease is usually seen in the late summer or fall after a build-up of L₃ on pasture. The L₃ are well adapted to survive over-winter on

pasture in the central Canadian climate. The arrested stage in sheep/goats will develop in the spring and contribute significantly to pasture contamination with the periparturient spring rise in egg output.

DISEASE IN THE SHEEP / GOAT

Diarrhoea, hypoproteinemia (bottle jaw), decreased appetite, weight loss are again all clinical features of severe infections with this parasite. Elevated plasma pepsinogen and abomasal pH are also important features. Again, 5,000 worms is considered to be associated with clinical disease.

POST MORTEM

The parasites invade the glandular mucosa, causing damage to the secretory cells. Plaques may be visible on the abomasal surface with chronic infections.

TRICHOSTRONGYLUS COLUBRIFORMIS AND T. VITRINUS

WHAT DOES IT LOOK LIKE?

Also known as the “**Black Scour**” or “**Bankrupt**” worms, they are small (0.5 to 0.75 cm in length), light brown and hair-like worms found in the small intestine (duodenum and upper jejunum). Eggs are typical of the Trichostrongyloidea Superfamily.

HOW DO THEY LIVE?

These parasites infect sheep, goats and cattle. The larvae burrow into the mucosa of the small intestine to develop and then burst out about 10 days after infection. This may cause severe damage to the intestinal wall with loss of blood and protein. Most disease occurs in the late summer and fall from the build-up of infestation on pasture. L3 can over-winter on pasture very well and serve to infect lambs and kids in the spring grazing. They can also overwinter in the animals as arrested L4 parasites. While trichostrongyles can cause significant disease alone, the worst disease outbreaks are usually seen with co-infections with Teladorsagia.

DISEASE IN THE SHEEP / GOAT

The parasite causes an enteritis (dark diarrhoea) and hypoproteinemia (bottle jaw) with poor appetite and weight loss. Milder infections are associated with soft stools and poor growth rates. Affected lambs and kids may have manure (dag) sticking to the back end and tail – evidence of diarrhoea.

POST MORTEM

The small intestine will have patches of erosion and loss of the normal villous lining.

WHAT DOES IT LOOK LIKE?

“Thread-necked worm”. They are slender worms approximately 1 to 1.25 cm in length and live in the upper part of the small intestine. The front part of the worm is more slender than the rest of the parasite. With a heavy infestation, they appear like a cotton-ball in the upper small intestine. The worms produce very large eggs in which the larvae develop to the L₃ stage. The eggs of *N. battus* are brown in colour whereas *N. filicollis* and *N. spathiger* are colourless, so they can be differentiated under the microscope. These parasites infect sheep, goats and occasionally calves.



HOW DO THEY LIVE?

N. battus, is the most disease-causing species of this genus, but is not common in Canada. In contrast, *N. filicollis* and *N. spathiger* are common but cause only mild or no disease. Their life cycle is very different from other trichostrongyloides.

N. battus: The eggs will only hatch after a prolonged period of cool weather followed by more mild weather in which the temperature stays above 10° C. Usually, eggs laid in the summer do not hatch until the following spring or possibly even for 2 years, so that the biggest risk period for infection and disease is the late spring (May and June). Lambs and kids on pasture in the spring are most at risk of disease.

N. filicollis: The eggs tend to accumulate on pasture and hatch in the late autumn.

N. spathiger: eggs hatch more quickly and several generations of infection can occur in one grazing season.

It is common, however that lambs and kids housed indoors or in dry lots can become infected indicating that the parasite can complete its life cycle without pasturing. Adult sheep and goats appear to have very good immunity and don't seem to play a role in infecting their offspring.

DISEASE IN SHEEP AND GOATS

N. battus will cause severe watery yellow-green diarrhoea in lambs and kids, often accompanied by dehydration and thirst - and in severe infections, death. Clinical signs may appear before eggs are produced (pre-patent period of 14 to 16 days), so faecal egg counts may be of limited value in the face of clinical disease. Mild infections of *N. filicollis* and *N. spathiger* may have no to mild signs of disease -most infections are seen with other gastrointestinal nematode parasites (GIN).

POST MORTEM

Large numbers of thread-like “cotton balls” of worms will be found in the small intestine. Some species tunnel into the mucosa. Severe infections are accompanied by signs of mild reddening of the intestine (enteritis) and marked villous atrophy of the intestinal lining.

SUMMARY OF THE STAGES OF THE LIFE CYCLE

The eggs of the parasite are passed in the faeces and the L1 develops within...

The eggs hatch within the faecal pellet to L1 larvae →

L1 larvae (free-living) feeds within the pellet and moult to L2 →

L2 larvae (free-living) feeds within the pellet and moult to L3 →

L3 larvae retains the L2 cuticle. This stage does not feed and relies on stored nutrients for survival. This stage migrates outside the faecal pellet on pasture (free-living infective stage) and is ingested by the sheep / goat when grazing.

Inside the gastrointestinal tract the L3 moults to L4 →

L4 larvae which is parasitic and feeds on the sheep / goat and moults to L5 →

L5, the immature adult stage which is parasitic, matures to an adult →

The adult stage which is parasitic, lays eggs which are passed in the faeces.

The cycle starts over again.

Figure 1 shows the typical life cycle of the gastrointestinal nematodes *Teladorsagia circumcincta*, *Haemonchus contortus* and *Trichostrongylus* spp. For these parasites, there is no intermediate host. The **Pre-Patent Period** is the period from ingestion of the L3 stage to when eggs are detected in faeces, usually 16 to 21 days for most GIN.

