Handbook for the Control of Internal Parasites of Sheep



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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 in Canada. It is not meant to replace the unique relationship between the flock veterinarian and the producer and his / her flock, but only to augment the information available to them.

This handbook was written primarily for those working with sheep 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 in this country. We hope that you will find it of use in developing internal parasite control programs for sheep flocks.

IMPORTANT GASTROINTESTINAL NEMATODE PARASITES

This section contains a description of the most commonly found and important gastrointestinal nematode (GIN) parasites of sheep 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 cycle of the GIN is similar and described in Figure 1. In central Canada, the most common and the most pathogenic (disease causing) nematode parasites are *Teladorsagia circumcincta*, *Trichostrongylus* spp and *Haemonchus contortus*. *Nematodirus battus* which is also highly pathogenic has been reported 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.

Abomasum

Haemonchus contortus

Description: "Barber pole worm" "Blood 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. Eggs are typical of the Trichostrongyloidia Superfamily.



Courtesy J Avula

Epidemiology: This parasite also infects goats, llamas, deer and

occasionally cattle but in the latter, is likely a species-adapted strain. It over-winters only fairly on pasture in temperate climates as a 3^{rd} stage larvae (L₃). However it is a very prolific egg producer - each female worm can produce several thousand eggs every day - enabling marked pasture contamination by mid-July. Most pasture contamination is from infected lambs but may also be from the ewe during the periparturient egg rise associated with late pregnancy and lactation. The eggs can develop to the infective L₃ stage in as little as 5 days but 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 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. 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). These ewes can develop clinical disease from the emergence of these arrested L₄ larvae ("Type II haemonchosis") and also from the ingestion of L₃ on pasture.

- Clinical Disease: 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 (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 lambs will become profoundly anaemic. The bone marrow tries to compensate by increasing red blood cell production, but it is a losing battle. Sheep may drop dead on pasture with very severe infections (e.g. 30,000 worms). Sheep with lower levels of infection will be chronically anaemic, have low protein (hypoproteinemia) and "bottle jaw", have a poor appetite and exhibit weight loss. The conjunctival mucous membranes 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%). Haematocrits as low as 6% are not unusual. In Ontario, we have seen Type II haemonchosis. Ewes overwintering large numbers of arrested L₄ may develop severe disease in late April and early May - often before going to pasture - and even death may occur due to severe anaemia. Treatment in the fall with an anthelmintic effective against arrested larvae will prevent these losses.
- **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 very red from the response to the anaemia.

Teladorsagia circumcincta

- **Description:** "Brown stomach worm". Also known as "Ostertagia". Eggs are typical of the Trichostrongyloidia Superfamily.
- **Epidemiology:** This parasite also infects 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 Ostertagiasis"). 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 will develop in the spring and contribute significantly to pasture contamination with the periparturient rise in egg output.
- **Clinical Disease:** Infection is associated with intermittent diarrhea, weight loss or reduced gains, decreased appetite, and occasionally protein loss resulting in fluid accumulation under the jaw known as "bottle jaw". Plasma pepsinogen levels may be elevated due to abomasal damage and the pH of the abomasum is elevated (normal is 2.0 2.5) because of the 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 put to heavily contaminated pastures may experience severe disease due to the sudden massive infection; if sheep are put in the barn in the fall carrying massive numbers of arrested L_4 , Type II disease occurs in the spring when the L_4 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

- **Description:** "Stomach hairworm". The worms are < 0.5 cm in length and difficult to see with the naked eye. Eggs are typical of the Trichostrongyloidia Superfamily.
- **Epidemiology:** This parasite also infects goats, cattle and deer. Like *T. circumcincta*, disease is usually seen in the late summer or fall after a build-up of L_3 on pasture. The L_3 are well adapted to survive overwinter on pasture in the central Canadian climate. The arrested stage will develop in the spring and contribute significantly to pasture contamination with the peri-parturient spring rise in egg output.
- **Clinical Disease:** Diarrhea, 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 cause 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.

Small Intestine

Trichostrongylus colubriformis and T. vitrinus

- **Description:** Also known as the "Black Scour" or "Bankrupt" worm, they are small, light brown and hairlike and 0.5 to 0.75 cm in length. Eggs are typical of the Trichostrongyloidia Superfamily.
- **Epidemiology:** These parasites also infect goats and cattle. The parasite lives in the upper small intestine (duodenum and upper jejunum). The larvae burrow under the mucosa to develop and then burst out about 10 days after infection. This causes 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 and serve to infect the sheep in the spring grazing, if not already carrying an overwintered load of arrested L₃ parasites. While trichostrongyles can cause significant disease alone, the worst disease outbreaks are usually seen with concomitant infections with Teladorsagia.
- **Clinical Signs:** The parasite causes an enteritis (dark diarrhea) and hypoproteinemia (bottle jaw) with poor appetite and weight loss. Milder infections are associated with soft stools and poor growth rates.

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

Nematodirus battus, N. filicollis and N. spathiger



Courtesy J Avula

Description: "Thread-necked worm". They are slender worms approximately 1 to 1.25 cm in length. The anterior part of the worm is more slender than the posterior. The worms produce very large eggs in which the larvae develop to the L_3 stage. The eggs of *N. battus* are brown in colour whereas *N. filicollis* and *N. spathiger* are colourless, so can be differentiated under the microscope.

Epidemiology: These parasites also infect goats and occasionally calves. *N. battus*, which is more pathogenic, is not common in Canada. In contrast *N. filicollis* and *N. spathiger* are common but are only mildly pathogenic. Their life cycle is much different than other trichostrongylides.

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

- **Clinical Signs:** Mild infections of *N. filicollis* and *N. spathiger* may have no to mild signs of disease and most infections are seen with other gastrointestinal nematode parasites (GIN). *N. battus* will cause severe watery yellow-green diarrhea in lambs, 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 fecal egg counts may be of limited value in the face of clinical disease.
- **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 enteritis but marked villous atrophy of the intestinal lining.

The Typical Life Cycle of a Gastrointestinal Nematode Parasite

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.

Stages of the Life Cycle: Eggs passed in feces \rightarrow hatching $\rightarrow L_1$ larva (free-living) \rightarrow moults $\rightarrow L_2$ larva (free-living) \rightarrow moults but retains cuticle $\rightarrow L_3$ larva (does not feed and relies on stored nutrients for survival) \rightarrow pasture (free-living infective stage) \rightarrow moults inside animal $\rightarrow L_4$ larva (parasitic) \rightarrow L5 (immature adult, parasitic) \rightarrow adult (parasitic) \rightarrow lays eggs.

Pre-Patent Period: The period from ingestion of the L_3 stage to when eggs are detected in faeces.

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Figure 1.
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EPIDEMIOLOGY OF GASTROINTESTINAL NEMATODE PARASITES

Figure 2 summarizes the typical level of gastrointestinal nematode parasite burden in lambs and adults, as well as infectivity of L_3 on pasture, under conditions in central Canada. This graph is adapted from data obtained on farms in Ontario and Quebec.

Normal Patterns of Infection in Adults and Lambs

- **Lambs or Youngstock** in their first grazing season, tend to have no natural immunity to gastrointestinal nematode (GIN) parasites. The L_3 stage on pasture serve to infect naive lambs. The level of L_3 on pasture and the level of immunity in the lambs will determine the level of disease seen in the lambs. Over the grazing season, the loads in the lambs tend to increase and they become the major contributors to egg contamination on pasture. Towards the end of the grazing season, a proportion of the new GIN infection will not progress to adults, but will rather remain at the L_4 stage and become hypobiotic or arrested.
- **Adults** tend to have a level of immunity to the GIN parasites, but will still be infected and will contribute to pasture contamination. The phenomenon of periparturient egg rise has been well studied in ewes and is due to a relaxation of immunity around the time of lambing (see below). This allows for increased egg production by those parasites in the ewe, and thus serves to be the one of the most important sources of pasture contamination to newborn lambs.

Figure 2.



Hypobiosis or Arrested Larval Development

After the L_3 larvae infect the host and moult to the L_4 stage, they may either develop into adults or stay at the L_4 stage. At this stage, little disease is seen in the host and no eggs are passed. In this way, many immature parasites may collect in the host without clinical signs. The trigger for hypobiosis is thought to be unfavourable environmental conditions for egg hatching and larval development of the free-living stages, e.g. winter in temperate climates or dry periods. In Canada, arrested development is an important mechanism that allows for survival of *H. contortus*, as well as *T. circumcincta* and *Trichostrongylus axei*. It is believed that here most L_3 larvae ingested in the fall - and sometimes late summer in the case of Haemonchus - arrest rather than develop to adults.

Immunity of the Sheep and Parasite Burden

Acquired Immunity to Parasites

Lambs will develop immunity to parasites over-time. This period varies with the type of GIN but generally occurs over 4 to 6 months but varies between breeds and between animals. With immunity, the adult parasites are expelled but the sheep will continue to be infected with low numbers. However, without continued exposure to parasites, the sheep's immunity will wane and after 6 to 8 months, they can become susceptible again. Additionally, high challenges of GIN on pasture can overwhelm the sheep's immunity and cause disease. Immunity is also greatly affected by nutrition, particularly dietary protein as rumen non-degradable protein - also called "by-pass" protein. This is protein which is not digested by the rumen bacteria, but passes through the rumen and is digested in the abomasum and intestine. Examples of this type of protein is roasted soybeans and corn gluten. If the diet is supplemented with rumen by-pass protein, parasite resistance is improved. It is important to remember that adult goats do not develop immunity.

Periparturient Egg Rise (PPER)

Also called the "**Periparturient Relaxation of Immunity or PPRI**", this terms refers to the increase in eggs passed in the faeces of ewes around lambing time - which is traditionally in the spring months. It occurs because of a relaxation of immunity in the ewe associated with late gestation and lambing, allowing for maturation of arrested larvae, increased infection rate from ingestion of overwintered L_3 on pasture, and an increased rate of egg production from existing adult worms. The relaxation of immunity is thought to be because of nutritional stresses associated with late gestation. It typically extends from 2-4 weeks prior to lambing and extends up to 6-8 weeks after lambing. PPER's tend to be lower in single-bearing ewes compared to twin-bearing ewes, lower in mature ewes than first-time lambers, and lower when ewes are supplemented with by-pass protein sources. The level of PPER has not been studied in ewes which lamb in non-traditional times. We are currently conducting research into this issue by examining PPER on accelerated flocks. Our Canadian data suggests that PPER may be extended in dairy ewes. Perhaps this is due to a higher level of nutritional stress.

Genetic Resistance to Infection with GIN Parasites

Some sheep develop better immunity against parasites, and are more able to resist establishment of infection after developing this immunity. Within any population of sheep, there is variation in this ability, and a portion of that ability is genetic. Programs have been developed to identify those sheep carrying genes for resistance, either through ram selection (e.g. rams raised together and selecting those with lower faecal egg counts), or attempting to identify genetic markers in the DNA. It must be remembered that lambs with resistance genes, prior to developing immunity, are just as susceptible to GIN infection as lambs without resistance genes. The benefit comes later, after the lambs reach 4 to 6 months of age. As adults, these resistant sheep will shed fewer eggs, most importantly at the time of PPER, or if faced with an excessive burden on pasture. This reduction of contamination is where the benefit lies to genetic selection.

Resilience to Infection with Parasites

This is the sheep's ability to grow and thrive in the face of parasitic infection. These sheep are infected, and pass eggs which contaminate pasture. Within a population, there are individual sheep which exhibit resilience. But they may serve to contaminate the pasture for sheep that are not resilient nor immune. So for this reason, resistance is preferred to resilience.

Normal Patterns of Infectivity on the Pasture

Refugia

This term is applied to the free-living stages of GIN on pasture, i.e. L_1 , L_2 and L_3 stages of larvae. Traditionally a higher proportion of the total parasite load on a sheep farm is on the pasture (80%) compared to the parasite load in the sheep (20%). This is important when understanding how anthelmintic resistance develops on a farm. We can deworm the sheep and only resistant worms are left in the animal, but the refugia is our source of susceptible parasites. Elimination or severe reduction of refugia may hasten the development of this resistance. However, a high level of refugia may be a primary factor in sheep developing clinical parasitism and perhaps even dying of its effects. It is important to learn how to ride the fine-line between too many parasites in refugia, and too few. Later in the handbook, when selective treatment is discussed, the term refugia may also be applied to include those sheep purposely not treated.

Effect of Environment on Survival of the Free-Living Stages

Temperature

The optimal temperature for hatching, larval development and L_3 survival varies by parasite. *Teladorsagia circumcinta* prefers 16° - 30° C; *Trichostrongylus colubriformis*, 22° - 33° C; and *Haemonchus contortus* requires the hottest temperatures at 25° to 37° C. But when temperatures range from 25° to 30° C, the development of all parasites is favoured. However, if the weather is hot, the L_3 may die more rapidly because their metabolic rate increases and they outlive their stored nutrients (L_3 cannot feed) before infecting a host. At temperatures < 10° C, larval development and moulting do not occur. At temperatures < 5° C, the metabolic rate of L_3 is very low - allowing prolonged survival, e.g. over-wintering on pasture.

Humidity

At faecal pellet or ground level the humidity should be > 80% to allow for development. Although L_1 and L_2 will not, the L_3 can survive dessication because of protection of the cuticle covering, even at freezing temperatures. Some species of L_3 (e.g. Teladorsagia but not Haemonchus) can enter a state of "anhydrobiosis" which allows them to survive severe cold and dessication, making them well-suited for Canadian conditions.

Assumptions Regarding Development and Survival of Free-Living Stages on Pasture

Survival Over-Winter on Pasture

When sheep are turned out to pasture in the spring that has been grazed the previous late summer - fall, it can be assumed that it is contaminated with L_3 that have survived over-winter. If pasture contamination was high the previous fall, then the level of L_3 from these species may be high in the spring as well. Teladorsagia and Trichostrongylus are particularly well suited to this survival. Snow cover throughout the winter enhances the survival rate. Several freeze-thaw cycles or prolonged, cold temperatures without snow cover may lower this survival rate. Haemonchus is generally assumed not to survive well at our latitudes, but there is early evidence that in Ontario, at least some L_3 of this parasite may survive to infect lambs



Courtesy - J Avula

the following season. Over-wintered L_3 of all GIN are considered to survive no longer than the end of June - but this depends on the temperature and humidity. A cool, wet spring may enhance survival, whereas a hot spring will shorten survival.

Survival on Pasture During the Grazing Season

The time from egg deposition to development to L_3 varies depending on temperature and humidity. Moderate temperatures and high humidity hasten development to L_3 . Cool temperatures will prolong



Courtesy J. Avula

Generations on Pasture

survival of L₃. Hot temperatures will shorten survival. The pasture itself will influence development and survival. Old pastures with a mat of dead grasses above the soil, will hold humidity longer as well as reduce temperature extremes, and therefore enhance development and survival. Heavy cropping (e.g. through pasture rotation) will reduce this mat and open the soil to sunlight and dessication - both limiting survival. Heavy morning dews or moisture that may be present after a rainfall, will allow migration of L₃ a few centimetres up the grass blades, enhancing infectivity of the pasture. Hot, sunny days will drive the L₃ down to the soil level, thus reducing infectivity.

One generation is the time from the eggs passed in the faeces, through hatching and development of the free-living larvae, infection of the sheep and then passage of eggs. Assuming that time from egg deposition to L_3 is 2 to 3 weeks and time from infection to egg production is 3 weeks, and the likelihood that optimal environmental temperatures are only present for 3 months at the most, there is limited time for build-up of L_3 on pasture during the first grazing season.

- **Teladorsagia and Trichostrongylus:** While these data are not known specifically for central Canada, it is unlikely that more than 2 generations of GIN occur under our conditions (from passing of eggs to infection and passing of eggs). Therefore it is likely that the most severe cases of parasitism on pasture are assisted by contamination from the previous grazing season and heavy stocking densities along with optimal summer conditions for L₃ development and survival.
- **Haemonchus**: While it is currently believed that overwintering on pasture under Canadian conditions is only moderately important at best, it survives overwinter very well in adult sheep in the hypobiotic state. Once the adult female worm matures in the spring, it is very prolific - capable of producing 10,000 eggs per day. Under warm, humid conditions L₃ will develop in as little as 7 days. This means that within one grazing season, the infectivity of the pasture may become very high and therefore risky to lambs by mid-July to early August, and occasionally to ewes.

DIAGNOSING GASTROINTESTINAL PARASITISM

Fecal Egg Counts (FEC)

Adult parasites will lay eggs, so FEC are a measure of the adult parasite population in the sheep. There is much animal-to-animal variation in FEC, so it is important to sample a random proportion of the group to get a clear picture of the parasite load in the animals.

Who

Sheep grazing pasture that are representative of the group. Do not sample sheep that have been held off feed, or that are off-feed due to illness. Ideally submit 10 samples representative of lambs and 10

samples representative of ewes. It is also important to sample lambs separately from adults, as counts will be very different - even on the same pasture.

Getting the Samples

The simplest way is to group the sheep into a corner of the pasture (with clean ground), hold them for 10 minutes or so and then release them. Pick up ten individual faecal samples (8 to 10 pellets each) that are fresh. Use a clean sandwich bag or disposable glove to collect, invert and tie off. Immediately place in a cooler with ice packs. Sheep can also be run into a chute and faecal samples collected from the rectum. Again, use a plastic bag or disposable glove. Apply a small amount of lubricant to the finger and gently tease out the faecal pellets. This latter method allows identification of the samples. Sheep for sampling should be randomly selected, e.g. not the first to be caught or through the chute. It is recommended to select sheep randomly so that the flock is properly represented.

Transportation of the Samples

It is very important that the samples be kept cool (< 5°C) but not frozen until they reach the laboratory. This is to prevent hatching which will lead to underestimation of the level of parasitism. Refrigerated samples should be analysed within 7 days of collection.

Analysis of the Samples

It is important that a trained person examine the samples to prevent confusion with air bubbles and pollen or other artifacts commonly found in faeces. It is also important that the samples be evaluated using a quantitative technique. The modified McMaster technique is one such method that will allow the number of eggs per gram (epg) to be reported. Qualitative counts (e.g. 1+, 2+, 3+) are not useful for differentiating between a moderate infection (e.g. 150 epg) or a severe infection (e.g. 1,000 epg) as both will be interpreted as 3+.

Pooled Versus Individual Samples

There is much animal-to-animal variation in egg output, with 30% of animals responsible for ~ 70% of the egg output. Pooling samples should be done at the laboratory to make sure that equal amounts of pellets are contributed by each sheep (minimum 4 grams of faeces each). Because of the animal-to-animal variation, it is important to select a minimum of 10 random animals to reduce the chance of not obtaining a representative sample from the group. Results from individual samples will allow the veterinarian to see the distribution of FEC within the group, but it is more expensive to run 10 individual samples rather than one pooled sample.

Significant Eggs Per Gram Levels

There is no recipe for determining a particular cut-point of FEC which indicates treatment is necessary. However, veterinarians will often use a threshold of 500 to 800 epg in an attempt to develop a parasite control program based on monitoring. The following cut-points have been used: < 250 epg - low; 250 to 800 epg - moderate; > 800 epg severe. However, there are several factors that need to be appreciated when deciding what cut-point to use:

Species of GIN

Haemonchus is a very prolific egg producer and is associated with rapid changes in the environment of pasture infectivity. If this species is predominant, the FEC can change very quickly, as can the level of disease in the lambs. Furthermore, even within the 3 week pre-patent period of the parasite, lambs can become very anaemic - before egg levels change significantly. Generally we do not know which type of parasite is contributing to the FEC. In summary, there is tremendous variation in the daily output of eggs depending on the parasite, and variation in the pathogenicity of different worm species. Larval culture and identification of GIN species can be done in specialized laboratories, although that service is not routinely provided in central Canadian diagnostic laboratories. A technique utilizing peanut agglutinin with a fluorescent dye is used in some laboratories to stain and identify the proportion of eggs which are of the haemonchus type.

Infection from Previous Season

Sheep that grazed the previous summer may have a significant hypobiotic load of L_4 *Teladorsagia* that are sitting in the abomasal walls waiting for more favourable climate conditions before developing to adults. In the spring, the massive re-emergence of these L_4 can be associated with significant disease - often called Type II "Ostertagiasis". The sheep have diarrhea and bottle jaw along with a negative FEC as the L_4 's have not yet reached the adult egg-producing stage. We have also seen a similar phenomenon with Haemonchus, when large #'s of arrested larvae emerge causing anaemia and sometimes death in ewes in the spring before going to pasture.

Grazing Heavily Infested Pastures

Naive sheep and lambs that graze very heavily infested pastures, may experience disease due to *Teladorsagia* and *Trichostrongylus* before the pre-patent period is complete. Like Type II disease, these animals will have watery diarrhea and bottle jaw with some deaths - along with a very low FEC.

Individual Variability in FEC

It has been shown that approximately 30% of lambs are responsible for 70% of the total egg production. This means that there is tremendous animal-to-animal variation in egg output - also called over-dispersion of the values. If means (averages) are used to determine how infected a group of sheep

are, there is a great risk of underestimating the level of infection. An example: 3 faecal samples have a count of 1,000 epg and 6 faecal samples have a value of 50 epg, this gives a mean value of 330 epg. In this example, if a cut-point of 500 epg is used, it might result in a decision not to treat when treatment should have been performed. Factors that should also be taken into account are the clinical condition of the sheep, as outlined below.

Clinical Changes in the Sheep

Diarrhea / Dag Scores

Faecal consistency (formed pellets, soft pellets, liquid diarrhea) may reflect parasite load, but some parasitic infections (e.g. acute haemonchosis) do not always exhibit diarrhea. Diet type also greatly influences faecal consistency- with lush grass causing diarrhea so



interpreting this must be done in the light of the type of pasture being grazed. Dag is defined as faecal contamination of the wool. Soft or diarrhetic stools will cling to the wool. A Dag score will give an approximation of faecal consistency or prevalence of diarrhea in the group of sheep. It should be noted that animals with diarrhea may actually have decreased FEC because the eggs are diluted so absence of eggs in an animal with diarrhea, does not mean that animal is not parasitized.

Poor Weight Gains / Weight Loss (Body Condition Score)

Gastrointestinal parasitism is associated with poor growth rates. The poor growth is primarily due to the decreased appetite from the parasite infection. Additional factors are the energy losses associated with the animal fighting the infection (i.e. immune response) and the losses of protein and blood that the parasites consume. Producers that weigh lambs on pasture, can track growth rates and, interpreting these data in the face

of nutritional constraints, can use this information (along with FEC) to determine if parasitism is clinically important. This may be one of the most sensitive indicators of significant levels of parasitism in individual sheep. A weigh scale set-up so that it is easy to run lambs through every few weeks during the highest risk periods, can allow for selective treatment of those lambs not gaining as expected. However, there are other causes of poor weight gains (e.g. poor pasture, coccidiosis, pneumonia) so that FEC should be done to confirm a parasitism problem.

Anaemia (Haemonchus)

A major clinical feature of haemonchosis is anaemia. In central Canada, we generally find that Teladorsagia and *Trichostrongylus* are the dominant parasites, but in late July to mid-August, in warm and wet summers - haemonchosis can be the most important type of parasitism on some farms. Lambs can be monitored during this period for evidence of anaemia. This can be done by taking a blood sample and measuring the proportion of red blood cells (packed cell volume or haematocrit). But it is more commonly done by assessing the colour of the conjunctival (around the eye) mucous membrane. The colour is normally pink but it can be pale pink to white in significant Haemonchus infections. This is known as the FAMACHA© system. It was developed in South Africa in regions where the primary type of GIN is Haemonchus, and is used successfully in the south-eastern USA where the epidemiology of parasites is similar. It allows the producer to monitor individual animals and to only treat



those that appear anaemic. Its drawback is that if other parasites are important, then it will fail to detect those infections. Other causes of anaemia may also confound the issue. Research performed to date in central Canada, strongly suggests that it is a poor indicator of parasite load. For this reason, the FAMACHA© system should only be used under the guidance of a veterinarian and only as an adjunct to FEC. There are other parasites that can causes anaemia: liver flukes being the most important - which



Body condition score

fortunately is not a problem yet in Ontario. If you believe that the FAMACHA © system would be useful on your property, contact your veterinarian for training on how best to use it.

Hypoproteinemia (Bottle Jaw)



Bottle Jaw

Almost all the GIN parasites feed on protein, also called albumin, that circulates in the blood and lymphatic system. In severe infections, the protein levels can drop very low and the fluid, which normally stays in the tissues, gathers under the skin and in the gastrointestinal lining. When fluid accumulates

under the jaw, this is termed "bottle jaw". E d e m a in the gastrointestinal lining causes poor absorption of nutrients and diarrhea. By

the time this is clinically apparent, parasitism is very advanced and the animal is in immediate danger of dying.

Courtesy J Avula

Listen to mon Carefully! Parasites

are <u>BAD</u>. They give You — Bottlejaw /

Necropsy and Worm Counts

If sheep are dying and internal parasites are suspected of being the cause, it is very important to confirm this diagnosis with a total (adult) worm count. Do not assume every dead lamb found at pasture is due to "worms", as de-worming when it is not required is not only an unnecessary expense, it may also contribute to anthelmintic resistance. A veterinarian can perform a field necropsy and attempt to identify abomasal and intestinal nematodes. *Haemonchus* are large and easy to see. *Teladorsagia* and *Trichostrongylus* are small and should be identified and counted in the laboratory using a microscope. The abomasal contents are removed and volume measured, and a sample of a known volume is removed and the worms counted. For example, if there are 10 worms counted in 1/100th of the volume, then the abomasum contained 1000 worms. A system recommended in the manual for the **Sustainable Control of Parasites in Sheep**¹ (SCOPS) from the UK is as follows:

2 points = Parasitism is likely affecting productivity 3 points = Parasitism is likely causing clinical signs and even death

| Teladorsagia sp: | 3000 worms | = 1 point |
|-----------------------|------------|-----------|
| Trichostrongylus spp: | 4000 worms | = 1 point |
| H. contortus: | 500 worms | = 1 point |
| Nematodirus spp: | 4000 worms | = 1 point |
| Immature worms: | 4000 worms | = 1 point |
| | | |

¹ Abbott KA, Taylor M, Stubbings LA. Sustainable Worm Control Strategies for Sheep, 2nd Edition. A Technical Manual for Veterinary Surgeons and Advisors.

ANTHELMINTIC DRUGS FOR SHEEP

Currently in Canada, there is only one anthelmintic drug licensed for use in sheep, specifically ivermectin. It is licensed both as a drench and an injection for sheep. However, veterinarians licensed by the province in which they practise, have the ability to prescribe anthelmintics that are licensed for use in other livestock species. But with this ability comes the responsibility for assuring safety (human and animal), efficacy and appropriate withdrawal times for meat and milk. Recently, the Small Ruminant Veterinarians of Ontario has sought advice from the Canadian Global Food Animal Residue Avoidance Database (CgFARAD) for appropriate withdrawals. SRVO will make those guidelines available to licensed veterinarians on request to help them in their recommendations. Anthelmintics are divided into broad spectrum - i.e. those able to kill a wide variety of parasites, and narrow spectrum - those only able to kill one or two types of parasites. Below is a listing of those anthelmintics that may be available for use in sheep in Canada, all of which are considered broad spectrum.

Benzimidazoles (BZ)

These are also known as "white" drenches. These chemicals are effective against all nematodes and adult tapeworms. The drug is deposited in the rumen and is slowly released into the gastrointestinal tract. They act on the intestinal cells of the nematode and the tegumental cells of the tapeworms, inhibiting uptake of glucose and causing starvation. They are also ovicidal - with activity against eggs being passed by the nematodes and tapeworms. Currently, fenbendazole is the chemical most commonly used from this group for sheep (Safeguard 10% suspension, Intervet Schering-Plough Animal Health) followed by albendazole (Valbazen, Pfizer Animal Health). Both of these products are available as drenches and are licensed for cattle. Albendazole also has activity against adult flukes, but should not be used during breeding or the first trimester of pregnancy because of toxicity to the fetus in early gestation. Generally, however, the BZ class of drugs are very safe with low levels of toxicity.

Imidazothiazoles (LV) and Tetrahydropyrimidines

This group contains levamisole, pyrantel and morantel. They are also known as "yellow" drenches. Levamisole is no longer manufactured in Canada (2005) and manufacture in the USA has been suspended since November 2008. It is also used as an immunomodulator in humans and for the treatment of specific types of cancer. It is not ovicidal and the difference between animal toxicity and efficacy is very narrow making overdose a potential issue. It works by paralysing the parasite so that it is removed rapidly from the gut. It works well against a broad range of adult worms but less so against the immature stages. However, it is particularly effective against lungworm. Signs of toxicity in animals include salivation, slow heart rate and muscle tremors with occasional death. Morantel can be used to treat GIN but is not effective against the immature forms. Pyrantel is rarely used in livestock but has a similar action as levamisole.

Macrocyclic Lactones (ML)

This group contains the avermectins (ivermectin, doromectin, eprinomectin) and the milbemycins (moxidectin). These compounds are derived from specific species of the Streptomyces genus and all act similarly. ML's have activity against most nematodes, but not tapeworms or flukes. They also has activity against some arthropod ectoparasites, specifically sucking lice and nose bots (*Oestrus ovis*), as well as keds

(*Melagophagus ovinus*) and mange (*Chorioptes, Sarcoptes* and *Psoroptes*). Because of this spectrum activity, drugs in this class are sometimes called endectocides. When administered, the drugs are stored in fat tissue and then slowly released into the body. These pharmacokinetic properties result in long meat and milk withdrawal times (Ivomec, Merial = 15 days meat withdrawal for sheep drench and 35 days for injectable). Moxidectin however, is the only drug in this class which is considered to have significant prolonged activity - approximately 35 days when administered as an injection, and 21 days when administered as a drench, against *Teladorsagia* and *Haemonchus*. The mode of activity is believed to be against neurotransmitter receptors specific to invertebrates. But it is considered quite safe for mammals.

Amino-Acetonitrile Derivatives (AAD)

The first product from this new class of drugs (monepantel), has been released recently (March 31, 2009) in New Zealand and the UK (Zolvix, Novartis Animal Health). This is the first new class of anthelmintics developed in 25 years and appears to have excellent activity against resistant strains of GIN as well as immature forms of nematodes, and in particular *Haemonchus*. The drug is also of low toxicity as it targets a unique, nematode-specific clad of acetylcholine receptor subunits.

Route of Administration

Drench versus injection

Drenches are deposited in the rumen so that proper absorption can occur. Injection of anthelmintics has been shown to result in a longer action - which may be favourable in some instances but may select for resistant nematodes because of prolonged sub-therapeutic drug levels. In general, drenches are preferred to injectable products.

Role of pour-on anthelmintics

There is evidence that pour-on products are not as well absorbed in sheep as in cattle. Because of the risk of sub-therapeutic dosing by this route, they are not recommended for use in sheep. Use of pour-on products as an oral medication is not recommended as the absorption, efficacy, duration of action and withdrawal times are not predictable and may increase the risk of anthelmintic resistance. Additionally injectable products have a different carrier that can affect its effectiveness.

Efficacy Against...

Hypobiotic Gastrointestinal Nematodes

Albendazole, fenbendazole, levamisole, doramectin, ivermectin and moxidectin all have some level of activity against the arrested L_4 stage of the abomasal nematodes. However the activity of the BZ and LV classes is considered is considered relatively poor compared to the ML class. Morantel has no activity against immature forms.

Persistent Activity

Only moxidectin has effective persistent activity against nematode parasites.

Tapeworms of Sheep

Albendazole and fenbendazole have excellent activity against the adult sheep tapeworm, *Moniezia*. These drugs are not effective against the cysticercus stage of *C. ovis* or *C. tenuicollis*.

External Parasites

The ML class is effective against sucking lice, mange, and nose bots but not effective against biting lice. While effective against keds, it is not effective against the pupae.

TREATMENT FAILURE

When a treatment fails to clear up a problem with internal parasites, many might be tempted to believe that this failure is due to anthelmintic resistance. This is only one reason for treatment failure (often called "drench failure") which can occur for several different reasons:

Use of the Wrong Anthelmintic

If an anthelmintic is used to treat a parasite for which it has no efficacy. A common example is the use of ivermectin to treat tapeworms.

Failure to Administer an Anthelmintic Properly

There are many reasons why an anthelmintic is administered in such a way that it will not work. It is the responsibility of the producer and veterinarian to make sure that the risk of this happening is minimized. The following is a list of how to prevent drench failure.

Weigh the Sheep

Using a calibrated livestock scale to prevent under-dosing (underestimating weight). If sheep are variable in weight, dose for the heaviest sheep in the group.

Select the Correct Dose

By reading the label for products approved for sheep. If not labelled for sheep, obtain the correct dose by veterinary prescription.



Courtesy J Avula

Use Drugs that Have a Canadian Drug Identification Number (DIN)

Drugs obtained through the internet (for example) may not contain what the label says it does as they may be manufactured in countries that do not have strict legislation on quality control.

Avoid Doubling the Dose

But if necessary because of anthelmintic resistance (see below), repeat the dose 12 hrs apart with BZ and ML drugs as this will lengthen the time in the GI tract when an effective dose is present.

Calibrate your Drench Gun Frequently

To assure delivering correct dose. Drench guns frequently do not actually deliver the amount listed on the syringe so measure and compare.

Drench Correctly

This is done by depositing the entire dose over the tongue, at the back of the throat. This will assure that the drench is swallowed into the rumen and is more slowly released. If administered in the front of the mouth, loss may occur by spitting or having the drug swallowed directly into the abomasum . Do not lift the head too much as that will prevent proper swallowing.



If Injection is Performed

Make sure that the automatic syringe is calibrated appropriately and that the entire dose is injected subcutaneously (not "intra-wool").

Don't Use by the Incorrect Route of Administration

Do not use a cattle pour-on product either as a pour-on or as a drench. Do not use an injectable product orally.

Hold Sheep Off Feed

Holding the sheep off-feed for 12 to 24 hrs before treatment, can increase the length of time that the concentration of the anthelmintic is effective. This is only effective if using BZ drugs, and should not be done if the ewes are in late gestation because of the risk of pregnancy toxaemia.

Reinfection After Treatment

If the pasture that the sheep graze after treatment is infested with high levels of L_3 , then there can be apparent treatment failure. Most anthelmintics have no persistency, which means that very soon after treatment, the lambs are reinfected from the L_3 on pasture. If the challenge is high, then clinically they may appear as if they have not responded to the treatment. Depending on when the faecals are reexamined (e.g. 2 weeks later), the FEC may be very low indicating that the parasites within the lambs were killed - but that immature adults are numerous enough to cause disease. This is prevented by reducing the challenge after treatment through pasture management.

ANTHELMINTIC RESISTANCE (AR)

Around the world, AR is becoming very common - particularly in *Haemonchus* and *Teladorsagia*, and to all classes of anthelmintics. As a result, sheep rearing is being threatened in many countries and regions. By the time AR is clinically apparent (i.e. failure for treatment to improve the health of the animals being dewormed), it is well-advanced in the flock. Prevention of development of AR is critical for the survival of the sheep industry. The following will explain how AR develops and strategies to avoid its development.

Definition of AR

Resistance or AR is the heritable ability of the parasite to tolerate a normally effective dose of an anthelmintic. Usually a parasite is considered resistant if it survives a normal dose of a single anthelmintic. Parasites often survive if the treatment is administered incorrectly - this is not AR but treatment failure (see above). Because resistance is genetic, the parasite may be homozygous resistant, i.e. having two copies of the genes for resistance (RR), or heterozygous resistant, i.e. having only one copy of the gene for resistant to an anthelmintic than are heterozygous. Heterozygous resistant parasites are still susceptible to the correct dosing of an anthelmintic, but will survive if the animal is underdosed. However, a homozygous resistant parasite may not be affected at all although repeated dosing at a high rate, or dosing with two anthelmintics simultaneously, may be effective for



Courtesy J. Avula

awhile. The homozygous resistant parasite is rare in an unselected population of parasites. But once the selection has occurred, parasites do not lose their resistance.

How does AR Develop?

Pressure of Anthelmintic Use

Repeat dosing with an anthelmintic will often kill 95% or more of all GIN in an animal. But it is the surviving, genetically resistant population that will continue to lay eggs and contaminate the pasture. Repeat, frequent dosing, particularly if under-dosing occurs, will hasten the development of a resistant refugia. While sheep will clinically respond to a drench that is less than 95% effective, eventually the susceptible parasites are in the minority and the drench ceases to be clinically effective. This does not happen overnight, and may take years for a farm to get to this state.

Side Resistance

Resistance is generally shared by all drugs in an anthelmintic class. E.g. if the parasite is resistant to fenbendazole, then it is also resistant to albendazole. This is less true with the avermectins and moxidectin, where moxidectin may still be effective in the face of avermectin resistance, but often not for long.

Parasite Resilience

It appears that AR can develop more quickly if the population of parasites is already resistant to one or more classes of anthelmintics. It may be that those parasites have the ability to more quickly metabolize drugs than those that are 100% susceptible. This may play a role in the development of multiclass resistance on a given farm.

Having Low Refugia in the Face of an Aggressive Deworming Program

When we deworm the sheep, traditionally we are only treating a small proportion of the worms present on a farm, with those parasites "in refugia" not being subjected to deworming. This means that sheep have the opportunity to become infected again with susceptible parasites - thus lowering the risk of AR becoming a farm problem. However, common sense tells us that we want to reduce the challenge to sheep that comes from a heavily contaminated pasture. So it is a good idea to institute those practices that lower the level of parasites in refugia (see later in the handbook for those methods). However, if an aggressive deworming program is also instituted - the development of AR is accelerated. This is because sheep are dewormed and have less opportunity to become reinfected from refugia. A possible example of this is deworming ewes prior to lambing while in the barn, then turning them onto a clean pasture. The only parasite eggs dropped by the ewes will be resistant ones. See below for how this might work when practising pasture rotation.

Pasture Rotation

A common strategy is to "dose and move". This is to allow animals to not "carry" parasites into a clean pasture. But while this might appear to work for several years, this practice will hasten the development of AR. This is because if sheep are treated and returned to an infested pasture, they will become reinfected with a population of susceptible parasites that were already on the pasture. If they are treated and moved to a clean pasture, the only parasites to shed eggs into the new pasture are resistant strains and there is no mixing of populations when the animal is reinfected. It might take several grazing seasons to build-up a resistant population, but when it happens, severe losses may occur. See figures 3- a,b,c &d.

Introduction of Resistant Parasites

Purchase of sheep that contain large numbers of resistant parasites, may introduce AR to a sheep farm - which when combined with improper parasite control measures, will hasten the development of AR on a farm. Goats are a particular risk as AR tends to develop more quickly with this species. This is because many anthelmintics are metabolized more quickly in goats than sheep (increasing the risk of sub-therapeutic dosing) and that adult goats do not develop immunity as well as sheep do, so often carry more severe infections. Quarantine of new introductions and proper deworming of new introductions is an important strategy to prevent introduction of AR.

Improper Treatment

There are many ways to improperly administer a treatment (see above for prevention of treatment failure). By under dosing, heterozygous resistant parasites are more likely to survive, which will hasten the development of AR on a property.

The Canadian Situation

We are currently conducting a survey in Ontario to determine the prevalence of AR in parasitized sheep flocks. Our initial results are surprising and disappointing. It appears that many farms in Ontario may have multiple drug AR. These results will be published and are being communicated to veterinarians and producers.

Figure 3 a.

Anthelmintic Resistance & "Dose & Move" Strategy to "Safe" Pasture



TREAT

Treat with an anthelmintic and move to "safe" pasture, e.g. hay field, newly seeded pasture or not grazed by sheep in the previous season. Sheep will be almost "worm-free" - the only parasites left will be resistant, but loads are light so no clinical disease seen.

AFTER SEVERAL SEASONS

After several years of "dose and move", only resistant parasites will be left in the sheep to contaminate the pasture. Eventually, the refugia of resistant L3 on pasture will be sufficiently heavy so that sheep suffer from clinical gastrointestinal parasitism that is non-responsive to treatment.

 \bigcirc = susceptible parasites \diamondsuit = resistant parasites

Figure 3 b

Anthelmintic Resistance & "Dose & Move" Strategy to Contaminated Pasture



Figure 3 c



Figure 3 d



Detecting the Presence of AR in a Sheep Flock

If AR is suspected in a group of sheep, it is important to review the treatment protocols to make sure that the drug is being administered properly. To confirm AR on a property the following methods can be used:

Drench Response Test

This can be performed with only 1 faecal sample collection time but only suggests, rather than proves, that AR is present. The group is treated and faecal samples are collected from 10 randomly selected lambs after a period of time (7 days for LV, 10-14 days for BZ, 14-16 days for ML). Failure to achieve zero counts may indicate AR, or it may indicate treatment failure from other causes. Since this method is less labour intensive than the FECRT, it is often performed before carrying out the FECRT.

Faecal Egg Count Reduction Test (FECRT)

The FECRT is often used as the "gold standard" of determining if AR is present on a farm. It is scientifically sound when done correctly and will give an accurate picture of how effective anthelmintics are on a given farm. However, as you can see - the process is labour intensive - but gives the best information.

If you decide to perform a FECRT, you require a minimum of 30 lambs or young adults (first grazing season) with elevated faecal egg counts (individual minimum counts of 150 epg but 300 epg is preferred). Ten to 15 lambs are randomly assigned to control and treatment groups. It is necessary to use these many animals per group because of the normal variation in egg output between animals. If three anthelmintics are being evaluated (e.g. ivermectin representing the macrocyclic lactones (ML) and fenbendazole representing the benzimidazoles (BZ) and levamisole (LV)), then four groups (60 animals) are needed (e.g. control group (no treatment), ML group, BZ group and LV group). Individual faecal samples are obtained per rectum on day 0 (treatment day), lambs are weighed using a scale (to prevent underdosing) and treated appropriately by drenching. The controls are not treated but are sampled. All of the lambs are returned to the same pasture to graze. All the lambs are sampled again later (7 days for LV, 14 days for BZ and ML). The post-treatment faecal egg counts are compared to the control. Failure to reduce by 95% or greater compared to the control indicates resistance. Confidence intervals (CI) are also calculated and if the lower CI is < 90%, then AR is indicated as well.

Larval Development Assays (In Vitro Test)

In some countries, larval development assays are used to detect AR in the laboratory. They cannot be used to detect ML resistance, are expensive, and require a specialized laboratory to work properly. The assays are mostly used as a research tool in North America for prevalence surveys. Eggs are hatched and developed while exposed to an anthelmintic. The level of successful hatching, development or feeding is then measured. The positive aspects of the assays are that they require less labour on farm and don't require as many animals.

SUSTAINABLE INTEGRATED PARASITE MANAGEMENT

Goal

The goal of a parasite control program is to control the level of parasites on the farm to a level which has minimal production costs without allowing the development of anthelmintic resistance.

This is known as a sustainable Integrated Parasite Management or sIPM. Gastrointestinal parasites do not need be an issue on a sheep farm, if sound principles and an understanding of the epidemiology are used in developing a suitable control program. Use the following with your flock veterinarian to develop a program which is correct for your farm.

Maintain a Minimal Population of Susceptible GIN on Pasture

Refugia

As mentioned previously, on a sheep farm, there are two populations of parasites - the parasitic population which resides in the animal, and the free-living parasites which reside on pasture. Most of the time, the larger population is the free-living and is the population not exposed to the anthelmintic. This is called "refugia". The goal is to minimize the level of the free-living refugia so that lambs do not become parasitized, while maintaining the susceptibility of that population to anthelmintics.

In order to assure a safe refugia of susceptible parasites, use these suggestions to maintain a safe pasture.

Reduce the Level of Pasture Contamination

Rotate Pastures with Other Livestock Species

While cattle share some parasites with sheep, rotation with this species has been shown to lower pasture infectivity. Horses will also work. Co-grazing with cattle is less effective but may help.

Never Graze Sheep and Goats Together

Ideally, never graze goats on the same pastures as sheep either. Sheep and goats share the same parasites. Adult goats do not develop immunity to parasites and so will be a serious source of pasture contamination. Additionally, they metabolize anthelmintics more rapidly than sheep, require higher doses than sheep and because of that are at risk of developing AR more rapidly than sheep.

Rest Pastures That Are Heavily Contaminated

If a pasture was particularly heavily infected at the end of the previous grazing season, select it for plowing, reseeding, having and / or grazing with another species.

Do Not Spread Manure Onto Pasture

Although it is unlikely that infective larvae will survive in well-composted manure, poorly composted manure can be a source of parasites. It is possible that hay fields can be contaminated in the same way.

Use Low-risk Pastures for the Most Susceptible Animals

Graze weaned lambs on newly seeded pasture or hay fields. Annual pastures (e.g. turnips) that are plowed in at the end of the season will be beneficial.

"Dose and Move" versus "Dose and Stay"

Traditionally, the recommendation was to treat and then move the sheep to a new pasture. Research on risk factors for AR now shows that it is best to have the sheep remain on the pasture after treatment for up to 3-5 days to allow for a mild reinfection. This means that the parasite load in the animal is now a mix of susceptible parasites (from the reinfection) and a few resistant parasites (those left over from the deworming). This allows a susceptible refugia to be maintained. The different choices are presented in Figures 3 a, b, c & d.

Reduce the Contamination of a Pasture by Using Pasture Rotation

However, understand that L_3 can survive for weeks or months if the environment is moist and temperate, and that L_3 can survive overwinter. Most pasture rotation systems require that sheep return repeatedly to the same pasture in a grazing season. Unless the frequency is < 2 weeks, eggs deposited when grazed previously will likely be hatched and the L_3 larvae will be waiting to infect. The following are some refinements on this management strategy:

Evasive Grazing

This technique requires knowledge regarding the speed of larval development given local conditions. For example, some work done in the Netherlands suggests that *H. contortus* becomes an issue after 3 weeks of grazing in the spring but after only 2 weeks of grazing in the summer. Models have been developed that take geography, weather and management practices into account to predict when pastures might becoming dangerous. These are commonly used in countries such as Australia, New Zealand and the UK. The model developed for the UK has been evaluated for its compatibility with Ontario data. The news is good - but adjustments will need to be



Courtesy J Avula

made for it to become practical. However, it should be remembered that during the summer pastures can remain very infective for up to 3 months making evasive grazing not practical as the only method for parasite control.

Strip Rotational Grazing

This is a form of evasive grazing and is relatively safe if the following hold true: 1) sheep are moved from the strip before eggs hatch and the larvae develop to L_3 stage (variable depending on weather, longer in cool weather but shorter in warm weather); AND 2) sheep do not return to the strip until the L_3 have died (variable depending on weather and moisture but may be up to several months if temperate and humid). On average the worst time period between grazing sessions is 3 weeks - the most likely time that the eggs have hatched and developed to L_3 . While short-term grazing will limit pasture contamination, returning to the strip several times in a season will result in the build-up of L_3 - just as if the pasture were set stocked (i.e. sheep put to the same pasture for the entire grazing season). While it may be prudent to strip graze in order to make optimal use of the pasture, monitor FEC closely.

Understand the Sources of Pasture Contamination

As the PPER in ewes is an important source of spring season pasture contamination, do not graze late gestation or early lactation ewes if possible. Lambs are the biggest source of pasture contamination by mid-summer. They should be the target of monitoring and possibly treating to reduce pasture contamination as well as disease. The following management practices can take advantage of understanding these sources.



Courtesy J Avula

Use Adult Sheep to "Clean" Pastures

Wean Lambs Early

If possible, do not graze lambs with ewes. Under some management situations, this is simple as lambs are often born well before the grazing season. If lambs are not grazed until after weaning (e.g. 50 to 60 days of age), then exposure to the PPER of ewes can be minimized.

Rotate Weaned Lambs Ahead of Ewes

After weaning, lambs should have "first access" to safe pastures. This way there is less risk from exposure to PPER contaminated pastures. Ewes are better able to tolerate heavily infested pastures.

If pastures are heavily contaminated and safe pastures are in short-supply, dry ewes not in late pregnancy can be grazed more safely than youngstock on these pastures and may help to lower the infectivity by grazing off L_3 . This should be done carefully and not without monitoring FEC.

Understand the Behaviour of the Free-Living Stages



Courtesy J Avula

Remember than L_1 and L_2 stages live within the faecal pellet and feed on the bacteria. If the pellet is exposed to moisture, then it breaks down and releases the L_3 to crawl onto the grass but exposes the $L_1 \& L_2$ to the environment where freezing temperatures or very hot, dry conditions can kill them more rapidly. L_3 dislikes dryness so will go down to the soil during hot days but migrate up when the dew is on the grass. But it is restricted on how high it can climb (usually not higher than 5 cm), so long grass is safer than short grass. This means that overgrazing pasture will increase the infection rate by forcing the sheep to graze close to the soil. Waiting until the dew is off the grass to graze short pastures is often not practical as a summer shower during the day could bring the L_3 back up the grass. Exposing the ground to sunlight may have some benefits as it

gives the L_3 less place to hide. This can be done by either de-thatching using a harrow when pastures get old to stir up the debris, or routinely planting new pastures and plowing in old pastures.

Use Anthelmintics Appropriately

To avoid treatment failure and development of AR, treat appropriately.

In summary:

- \star Weigh sheep. Dose for the heaviest sheep in the group.
- ★ Dose correctly by reading the label.
- ★ If not labelled for sheep, obtain the correct dose by veterinary prescription.
- ★ Use drugs with a Drug Identification Number (DIN) to be assured that the drug is effective
- ★ Goats are treated at 2X sheep dose (BZ and LV) or 1.5X sheep dose (ML)
- ★ Avoid doubling the dose but rather dose 12 hrs apart (BZ and ML only).
- ★ Calibrate your drench gun or automatic syringe frequently.
- ★ Drench correctly by depositing the entire dose over the tongue, at the back of the throat.
- ★ Oral drenches are preferred to injectable products.
- ★ Use the correct route of administration. Do not use a cattle pour-on product either as a pour-on or as a drench.
- ★ Holding the sheep off-feed for 12 to 24 hrs before treatment with a BZ, can increase the length of time that the concentration of the anthelmintic is effective.

Rotate Anthelmintic Classes Slowly

Consensus suggests not to rotate more frequently than annually. Rapid rotation will lead to multiple class AR.

Combining Anthelmintic Classes

In many parts of the world, there are commercial deworming products that contain more than one anthelmintic. These were developed for sheep farms with AR and will temporarily improve efficacy of those drugs. However, unless AR has been shown to be present on your farm, and unless prescribed by a veterinarian - do not combine anthelmintic classes.

Treat Animals Selectively

"Targeted Treatments"

This means to treat sheep that need it when they need it. This is done by monitoring FEC and clinical evidence of disease and then treating the group. Increasing the interval between anthelmintic treatments reduces the development of AR. Times to monitor (and possibly treat):

Ewes 1 Month Prior to Lambing

This is to eliminate or reduce the PPER which is considered one of the most important sources of pasture contamination for lambs. For spring lambing ewes - this is an obvious time. More research needs to be done for winter and fall lambing ewes to determine the level of PPER for out-of-season lambing and its significance to the ewes. Under some management conditions, e.g. if ewes lamb and nurse lambs indoors, it may not be necessary to deworm at this time. However, on some farms the ewes carry

significant loads and may suffer disease or decreased productivity if not dewormed. For these reasons, it is important to make the decision on whether or not to deworm with your flock veterinarian and to monitor faecal egg counts in those ewes nursing lambs.

Lambs at Mid-Summer

The exact week may vary depending on the warmth and humidity of the summer and how early summer arrives in our northern climate. Generally early to mid-July is the earliest that we routinely see clinical evidence of disease. Mostly it is slightly later - late July to August, which appears to be the highest risk period in our climate for haemonchosis. However, if the pasture is heavily contaminated with overwintered L₃ (e.g. *Teladorsagia*), then disease in the lambs may occur earlier. By checking FEC in lambs (and ewes if grazing together) in early to mid-July, and treating when high counts are found (or in the case of haemonchosis, evidence of anaemia can also be used), GI parasitism should not become clinically evident. If the FEC is negative, but lambs are showing severe clinical signs of parasitism, consult your flock veterinarian to determine if this is a Type II disease situation (see above) or if another disease is present (e.g. coccidiosis).

Repeat Monitoring

If when the lambs are monitored, the FEC is below the cut-point to treat, resample in mid-summer at least every 4 weeks and perhaps more frequently, particularly if *Haemonchus* has been a problem in the past.

Monitor after Treatment

If lambs are treated, depending on the drug used - monitoring should be done every 4 (BZ and LV) to 6 (ML) weeks. If the lambs appear parasitized after treatment, resample at 14 days to determine if treatment failure occurred (see above).

Monitor According to Farm History

By knowing the farm history, the time of monitoring can be adjusted. E.g. if the previous summer, lambs had elevated FEC in the first week of July, then monitoring should be done in mid-June.

Monitor in the Fall?

By October there is often no reason to use FEC to determine infection. Although the lambs may be parasitized, most of the development is now to the arrested L_4 stage which will not produce eggs. Performing FEC at this time will underestimate the infection.

"Targeted Selective Treatments"

This means treating only those individual sheep that need it and is based on the knowledge that in any given population of sheep, only a proportion actually require deworming. This is called "over-dispersion" as mentioned previously. The trick is to correctly identify which sheep require treatment. The producer has only a few options to be able to do this effectively and economically.

Using Fecal Egg Counts

Unless the flock size is very small, it is not economical to perform individual FEC on all animals in order to detect the "big shedders", i.e. those 30% of animals that shed 80% of the eggs. There is no method of determining parasite egg load in faeces other than using a laboratory based-test.

Using the FAMACHA © System

The FAMACHA © system can be used very effectively to select individual sheep for treatment of haemonchosis - but is not effective at detecting infection of other GIN species. It could be used on farms that know when *Haemonchus* becomes a problem (e.g. late July, early August) but should be combined with FEC. Sheep that score 3, 4 or 5 would be drenched and then everybody monitored every 2 to 3 weeks during the high risk period.

Using Evidence of Diarrhea

Dag scores indicating diarrhea may be helpful when the producer can eliminate other reasons for scouring (e.g. coccidosis or lush pasture) and may work best when combined with monitoring weight gains. However some research suggests that by the time the lambs have diarrhea, significant clinical disease is occurring - i.e. waiting until they have diarrhea is too late.

Using Weight Gains

Routine weighing of lambs (e.g. every 2 - 4 weeks) can identify those lambs that are not gaining as fast as their cohorts, one reason for which may be GIN parasitism. One method of using this information is to only deworm the lighter lambs and leave the heavier lambs untreated. More research is needed regarding the appropriate deviation of average daily gain (ADG), e.g. if the normal ADG is 0.35 kg/day, how much less is a "trigger" for treatment? A producer may get a feel for what growth should be expected from the lambs on a particular type and growth of pasture. That may be more useful than a scientific formula. Under the new RFID and automated weighing systems, it would be relatively easy for producers to select poor gaining lambs for treatment on a relatively frequent basis. The same type of system is used frequently for lambs in feedlots. Body condition score was not found to be helpful in a recent Canadian study.

Using Milk Production and Lambs Nursing

For dairy ewes, there has been some research into the effect deworming has on milk production but it is not conclusive as it is in dairy goats. However, there is evidence that ewes nursing multiple lambs shed more eggs than ewes nursing singles. This is likely due to differences in nutritional stresses between the two groups. Deworming only ewes with multiples - either before lambing based on pregnancy scanning, or after lambing based on number of lambs nursing - is one way to target those ewes that likely have the highest PPER.

Treatment of Breeding Animals at Housing in the Fall

This may reduce the arrested L_4 that overwinter and are thus available for a PPER the next spring in housed ewes. The recommendation of treatment pre-breeding should only be done if monitoring or poor condition suggests that the ewes are parasitized.

Quarantine and Treatment of New Introductions

Purchased sheep or goats may introduce parasites, and possibly AR. While performing a FEC may determine if infection is present, it may be more prudent to effectively treat the animal(s) while in isolation. This is done by dosing with an effective anthelmintic and keeping in quarantine for at least a few days. Once the FEC is negative, the animal is turned out to a contaminated pasture to pick up non-resistant GIN so any residual "imported" GIN are diluted. If the FEC is not negative, then treating with 2 anthelmintics simultaneously could be done under the supervision of the flock veterinarian.

Investigate Treatment Failure

Determine if due to AR, drench failure or another reason.

Re-establishing a Susceptible Parasite Refugia

If AR has been identified on a sheep property, is it possible to re-establish a susceptible refugia? The jury is still out on this one. Two different ways are suggested to accomplish this.

Introduce Lambs Infected with Susceptible GIN

The refugia is reduce through either leaving the pasture fallow for a long period of time, grazing with another species such as cattle or horses (not goats), or plowing and reseeding. Lambs that have been purposely infected with susceptible GIN are then introduced to seed the pasture with susceptible refugia. Then the sheep population of that farm is grazed on these pastures and the new infection will dilute the level of resistant parasites carried by those sheep. Down sides to this are to invest in reducing the refugia and then locating lambs with heavy loads of susceptible GIN.

Infect Sheep with Susceptible GIN from Refugia

This method requires deworming the flock with AR parasites with multiple dewormers and multiple times to reduce the parasite load as much as possible. Then they are moved to a heavily contaminated pasture with susceptible refugia. The down side to this method is to locate such a pasture. It is also dubious that returning the sheep to the AR pastures will effectively dilute the refugia. The producer will need to be very careful to preserve any susceptible parasites if possible.

Other Methods of Control

To reduce the use of chemical anthelmintics, some of the following methods have been used. Regardless of what methods are employed, make sure they are science-based and can work on your farm.

Genetic Selection

The breeding of resistant or resilient sheep can be done by selecting a breed (e.g. some hair breeds such as Barbados Blackbelly, but not Katahdin) or selecting individuals within a breed - usually rams that have lower FEC or other measures. Gene marker tests have been marketed in some countries to help identify sheep that will have lower FEC. Remember that immunity is acquired and resistant sheep still need to be infected to develop this immunity. Heritability for this trait is moderate, so a producer could use FEC in ram lambs (comparison within a group) as a criteria for selecting a replacement ram, but genetic progress within a flock will be moderate to slow.

Another approach would be to cull adults that have high FEC and don't develop good immunity as they grow older. Lambs from these animals would be sent to market rather than retained as replacements. Resilient sheep will be infected and will contaminate a pasture with eggs, but will not have a significant production losses. Within a population, there will be resilient and susceptible sheep so that selection

must be done by using good records, measures of anaemia, FEC and growth monitoring in order to avoid losses and to select the correct animals.

Condensed Tannin Containing Pasture Plants

Grazing pastures seeded with plants that contain high levels of condensed tannins (CT), have variably shown to reduce shedding of eggs in the faeces. In North America, most research has been published on the legume *Sericae lespedeza* (SL), a warm climate plant. The mechanism may be 2-fold. While there may be a direct effect by CT on the ability of the adult parasite to produce eggs and for those eggs to develop to infective larvae in the faeces, at least some of the effect is from the elevated levels of by-pass protein available to the animal. Animals fed SL also have an improved immune response over animals on a control diet. Low levels of CT in the diet have been shown to increase reproductive performance and wool growth independent of parasite load. However, high levels in the diet decrease feed consumption and have a negative effect on performance.

Two temperate climate plants with some potential benefits are Bird's Foot trefoil (*Lotus corniculatus*) and Sulla (*Hedysarum coronarium*). At least experimentally, sainfoin (*Onobrychis coronarium*) has been reported as both beneficial and of no benefit. There are other CT plants and tree extracts (Quebracho extract for example) that are promising and may be a helpful adjunct to other control measures. More research is needed using species that grow well in Canada.

Nematophagus Fungi

A fungus *Duddingtonia flagrans*, grows in faeces - sending out hyphae that will trap and kill the freeliving forms of GIN in the faecal pellets. While these fungi occur naturally, in order to get them into the faeces in sufficient quantity to be effective, the spores must be fed to the sheep daily for a minimum of 60 days. The intent is to feed at turn-out for a period of time to disrupt the build-up of L_3 on pasture until the season is advanced enough that disease will not occur in that grazing season. Research has focussed on feeding ewes during the PPER - the major source of spring pasture contamination, which should then spare the lambs. This could be viewed as preferable to using anthelmintics at this time which will select for AR. At this point, daily dosing is not practical for grazing sheep and a bolus is being developed that will pay-out over a longer term.

Copper Oxide

There has been much published on the use of copper oxide wire particles to control GIN. In sheep, it appears only effective in reducing infections due to *Haemonchus contortus* and only temporarily. It does not appear to improve weight gains (over controls). It does elevate liver copper levels in sheep. Given the copper status of sheep in central Canada where copper toxicosis is already an issue, use of copper oxide wire particles is not advised.

Vaccination

A large international research effort is currently underway to develop a vaccine against *Haemonchus*. A particular antigen known as H-gal-GP appears to confer protective immunity against *H. contortus* when lambs are vaccinated. Issues to resolve include purifying and manufacturing sources of this antigen and performing field trials to determine what conditions the vaccine best performs.

Alternative Dewormers

There have been many "alternative" or "natural" deworming products recommended over the years. Some are toxic to the sheep as well as the parasites (e.g. nicotine). Some do not work in controlled, peer reviewed studies (garlic, papaya seeds). Diatomaceous earth has been used as an anthelmintic but there is no scientific evidence that it is efficacious. It may be useful for control of external parasites but more research needs to be done to show sufficient efficacy and safety. It is dangerous to humans to handle. There are other herbal plants that have been hypothesized to be effective parasiticides, (e.g. Neem oil) but at this time there is insufficient supportive scientific evidence for this claim, and safety for both animals and humans has not been demonstrated.

Parasite Control on Organic Sheep Farms

There is no evidence that sheep cannot be raised organically and not suffer from clinical parasitism, but producers must invest heavily into monitoring and principles of sIPC. A recent study performed on conventional and organic sheep farms in Canada found little difference between the level of parasites on these two types of farms, although there was tremendous variation in parasite loads between individual farms. Producers must remember though that reserving treatment only in the face of clinical disease is not appropriate for proper control of GIN parasitism and has welfare implications for the flock. Use of unproven, potentially toxic "alternative" methods is also not the goal of a well-managed organic flock.

The Organic Production Systems—General Principles and Management Standards (CAN/CGSB 32.310-2006 Amended October 2008) states that

6.7.4 Medical treatment for sick or injured livestock shall not be withheld to preserve their organic status. All appropriate medications shall be used to restore livestock to health when methods acceptable to organic production fail.

Section 6.7.9 states:

Organic livestock operations shall have a comprehensive plan to minimize parasite problems in livestock.

- a. The plan will include preventive measures such as pasture management and faecal monitoring, as well as emergency measures in the event of a parasite outbreak.
- b. By way of derogation, when preventive measure fail, the operator may use parasiticides not listed in CAN/CGSB-32.311, *Organic Production Systems Permitted Substances Lists*, provided that:
 - i faecal samples or animal examination indicate that an individual or group of animals are infected with parasites;
 - ii the operator has received written instructions from a veterinarian indicating the product to be used, the individual or group of animals to be treated, the dose and route of administration;
 - iii withdrawal times are twice the label requirement or 14 days, whichever is longer;
 - iv slaughter animals under one year of age can be treated only once and slaughter animals over a year of age can receive a maximum of two treatments. Slaughter animals that require further treatment will lose organic status;
 - v dairy animals needing more than two treatments per year (antibiotics and/or anthelmintics) will

lose their organic status and shall go through a 12-month transition. These dairy animals shall never be organic for slaughter purposes;

- vi under this derogation, a dam may be treated during gestation;
- vii a producer will create a written action plan (including timing), describing how they will adjust/change their parasite control plan to avoid similar emergencies.

CAN/CGSB-32.311, Organic Production Systems - Permitted Substances Lists indicates that parasiticides are allowed.

Producers should always check with their certification body for proper interpretation of the regulations prior to treating animals with products not listed in CAN/CGSB-32.311-2006 Amended October 2008 and December 2009.

OTHER IMPORTANT NEMATODE PARASITES

Below are descriptions of other important nematode 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 Trichostrongyloidia 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 become hypobiotic (arrested) in the late fall. Lambs 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 Trichostrongyloidia Superfamily.

Epidemiology: This parasite can also infect goats. At this time, we have not found evidence of this parasite so it is likely not common in central Canada. The infective L_3 can penetrate the skin where they migrate to the lungs and then migrate to the digestive tract. Ingested L_3 do not migrate to the lungs. The prepatent period is 4 to 8 weeks. This parasite prefers more tropical climates, and heavy burdens are

not common under Canadian climatic conditions.

- **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 usually not severe.
- **Clinical Signs**: In very high levels of infection, young lambs (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.

Large Intestine

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 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 L_3 can over-winter on pasture and the L_4 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 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 Trichostrongyloidia 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 L_3 penetrate the mucosa of the small or large intestine and form nodules where they develop to the L_4 stage. They may remain in the nodules for up to 1 year. When the L_4 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. Egg appearance is specific to the genus.
- **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 world wide, 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 L_1 larvae are passed in the faeces where they moult to L_3 . 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.

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 L_1 larvae are coughed up and swallowed, then passed in the faeces. They penetrate the foot of the mollusc, mature and reside there for 2 to 3 weeks until eaten by a sheep. The mollusc is digested releasing the L_3 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. Eggs are laid in the capillaries 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-tails, 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 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 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: cestodes, both adult tapeworms and larval (intermediate stage) tapeworms; protozoa, which includes coccidia (Eimeria) and Cryptosporidium; 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 Eimeria spp but it takes only a few days to complete the life cycle and produce oocysts. The oocysts are infective in fresh faeces, are very resistant to environmental factors, and build-up quickly in lambrearing areas.
- **Clinical Signs:** Diarrhea in lambs, usually greater than 1 week of age up to 3-4 weeks. Affected lambs can become very unthrifty because of villous atrophy, malabsorption, and maldigestion. They can become severely dehydrated, depressed and cachexic and in some outbreaks, can be a significant cause of lamb 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.

Eimeria crandallis and E. ovinoidalis (Coccidiosis)

- **Description:** "Coccidia". There are 11 different species of coccidia in sheep but not all are considered to be pathogenic. There are subtle differences in the morphology and size of their oocysts which help to distinguish pathogenic from non-pathogenic. Only the 2 most pathogenic species will be presented here. Coccidia are microscopic, protozoal parasites of the intestine.
- **Epidemiology:** These parasites are specific to sheep and are not shared with goats or other animals. The complicated life cycle, which involves asexual reproduction (schizogony or merogony) and sexual reproduction (gametogony), is presented in Figure 4. Sporulation of the oocysts, necessary for successful infection, takes a few days in ideal weather but several weeks if the weather is cool. The prepatent period for *E. crandallis* is 15 to 20 days, and for *E. ovinoidalis* is 12 to 15 days.

While the coccidia invade the cells of both the small and large intestine, most of the dangerous damage is to the large intestine because cellular turnover (healing) is slower than in the small intestine. Adult sheep develop immunity to disease but continue to shed low numbers of oocysts in the faeces. The oocysts are very resistant to dessication. Newborn lambs ingest the oocysts and within a few weeks produce large numbers of oocysts which contaminate the environment. Later born animals are exposed to a very high level of contamination and are most at risk of developing clinical disease. Other stresses (e.g. poor nutrition or milk supply) or diseases (e.g. pneumonia, contagious ecthyma) put the lamb more at risk of clinical coccidiosis.

In central Canada, the worst outbreaks of disease are usually seen in nursing lambs born to prolific ewes in indoor lambing situations. Diagnosis based on faecal egg counts should be interpreted carefully, as non-pathogenic species can boost counts very high. Evidence of disease along with elevated faecal oocyst counts is helpful, although severe disease can be present before the prepatent period is finished. Speciation of the oocysts will help to determine if the counts are significant if no disease is evident.

- **Clinical Signs:** Disease can be acute and severe, particularly in nursing lambs exposed to high levels of oocysts. Dysentery with severe diarrhea, dehydration and depression are seen with a significant case fatality rate. Disease may be more chronic with soft faeces that often clings to the wool of lambs (daggy). Growth is retarded due to malabsorption of nutrients. Lambs are slow growing and unthrifty.
- **Post Mortem:** The large colon and caecum are reddened, with watery and sometimes bloody content. The small intestine may have villous atrophy. Sometimes the giant schizonts or meronts can be seen grossly.

Coccidiosis - Its Treatment and Control

Control of this parasite has focussed on 2 issues: 1) prevent build-up of the oocysts (eggs) in the environment through environmental hygiene and reduction of shedding by adults and 2) control the infection in lambs until immunity is strong enough.

Environmental Hygiene

The oocyst has the ability to survive in the environment for several months and perhaps longer. It will become infective in 1-3 days so the opportunity for build-up in the environment is massive. Between groups of lambs, the pens should be cleaned out, all organic debris removed and the walls, feeders and floors washed down. Steam cleaning is preferred if possible. While it is very difficult to kill the oocysts, this will greatly reduce the level of contamination. Feeder and waterer design are critical in preventing faecal soiling of food and water.

Shedding from Immune Animals

Adult ewes will intermittently shed oocysts in the faeces their whole lives but numbers increase greatly prior to lambing due to the PPER. In operations in which stocking densities are high indoors, feeding a coccidiostat prior to lambing may reduce this PPER and thus reduce environmental contamination. There may be some benefit to continuing this treatment until the lambs are weaned.





Other Risk Factors

Lambs that are ill for another reason (e.g. pneumonia, contagious ecthyma) or are stressed (high stocking densities, temperature fluctuations, high humidity, poor nutrition), are more susceptible to developing disease and severe infections.

Prophylactic / Metaphylactic Treatment

Lambs can be infected from early in life, but don't often exhibit severe disease until 5 to 8 weeks of age, unless the environment is very contaminated in which case they may become clinically ill as early as 3 weeks of age. Medicating with a coccidiostat or coccidiocidal drug is frequently used to control infection in young lambs until they have achieved immunity, which generally happens by 4 months of age. The principles are simple: begin medication prior to the animal becoming heavily infected; and make sure that the animal receives a minimal therapeutic dose on a daily basis until it develops immunity. The only product currently licensed for sheep in Canada is lasalocid (Bovatec, Alpharma Animal Health Canada). The product is to be incorporated into a total lamb ration (lamb creep) and delivered at 36 mg lasalocid / kg feed (36 parts per million or ppm) and offered free choice. The therapeutic dose is 1 mg lasalocid / kg bw daily so that it is critical that the feed be offered and consumed to allow for this.

Treatment Failure

There may be several reasons for this: lambs are not consuming sufficient medication; or the medication is being overwhelmed with environmental challenge. Rarely there may be resistance, but the first 2 reasons should be investigated first.

An example of the importance of intakes: A 10 kg lamb (22 lb) needs to be medicated with 10 mg lasalocid daily in the feed. This means that if the feed contains 36 mg/kg, the lamb must consume (10/36)

= 0.28 kg of feed daily. If the lambs are eating less than this, it may be because the ewes are milking heavily and / or the lambs are ill (e.g. pneumonia, diarrhea, contagious ecthyma) and thus not consuming sufficient feed. High stocking densities in indoor confinement operations can also lead to a challenge which may overwhelm the medication. The worst outbreaks tend to be indoor with nursing lambs born to prolific ewes.

Products Not Licensed for Sheep in Canada

There are many other products for treating and controlling coccidiosis in lambs but all should be used by veterinary prescription only.

Monensin (Rumensin, Elanco) is often incorporated into complete rations at 11 ppm or in limit fed rations at 22 ppm. Again the therapeutic dose is 1 mg/kg bw daily, but the drug is more toxic than lasalocid and intakes need to be carefully controlled.

Decoquinate (Deccox, Alpharma Animal Health Canada) is very safe and effective but it is critical that intakes reach at least 1 mg/kg bw daily or the drug is not effective. Note, this is twice the dose approved for cattle. Decoquinate is not considered a growth enhancer whereas monensin and lasalocid improve feed efficiency as well as control coccidiosis. This may be a consideration for producers growing lamb for markets that forbid the use of growth enhancer drugs.

Totrazuril (Baycox, Bayer Animal Health) is an oral medication approved for use in swine. The drug kills both the free-living and intracellular stages. It is most effective when used as a drench about 2-3 weeks prior to when the lambs would break with coccidiosis, e.g. 4-5 weeks of age. However totrazuril is very persistent and in other countries where approved for sheep, has a very long meat withdrawal (42 days in the UK). Only use under the direction of a veterinarian.

Amprolium is often used as a treatment (Amprol, 9.6% solution, Huvepharma) at a dose of 10 mg/kg bw (each ml contains 96 mg of drug) daily for 5 days. Higher doses may be scripted by the flock veterinarian to reduce the number of treatment days. It can be also incorporated in the feed or water for control. Prolonged treatment, however is associated with toxicoses, in particular disease which mimics polioencephalomalacia.

Many oral sulphonamide products are useful for the treatment of coccidiosis as well. However, long-term use can be associated with kidney damage. Again, none of these products should be used without a veterinarian's prescription to prevent inappropriate treatments and drug residues.

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 the forage mite ingests them. The eggs then hatch and the larvae migrate to the body cavity of the mite where they develop into cysticercoids (a tapeworm head in a fluid-filled cyst). 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 cysticeroids can overwinter in the mites.

- **Clinical Signs:** This tapeworm is generally believed not to cause significant disease in sheep. 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 typical of a fluke infection.
- **Epidemiology:** The definitive hosts are sheep, goats, cattle, horses, deer and humans. 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 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 sheep. 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 sheep have 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 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 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 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 Great Lakes region and eastern Manitoba. 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.
- **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 and they do not enter the bile ducts.

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 climb to the top of blades of grass, where they are more likely to be eating by grazing ruminants. The flukes are very long-lived and infections can be cumulative. The eggs are distinctive 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 but heavy infections may cause ill thrift. One case has been reported of mortality from 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.

IMPORTANT EXTERNAL PARASITES OF SHEEP

External parasites in sheep can be an annoyance and irritation and can be responsible for animal discomfort and some economic losses. However, the most dangerous external parasites (psoroptic mange and some blow flies) are not a problem in this country at this time. Below covers the most common and most important of the external parasites of sheep that occur in Canada.

Lice

Bovicola ovis

- **Description:** "chewing lice". Also known as *Damalinia ovis*. The lice are reddish brown with a large head. They are small - 3 mm in length. From egg through 2 nymph moults to egg laying adult, takes 2 to 3 weeks. The lice are found through the wool as small yellowish specks. The nits (eggs) are adhered to the wool.
- **Epidemiology:** The host is sheep only. The adults only live 1 month but lay 1 to 3 eggs per day. They can reproduce both sexually and asexually and so populations can explode quickly up to ½ million under severe conditions. They prefer hanging onto wool in the head and neck region but can move quickly and are found over the whole body.
- **Clinical Signs:** The lice chew and bite the wool, scales and scabs and produce pruritus (itching) of the sheep. The sheep will scratch and lose wool. Sometimes sheep can be seen with wool in their teeth caused by chewing and biting to relief the pruritis.
- **Treatment:** Treatment must always be done twice usually 14 days apart to allow for the nits to hatch. The following are approved for the treatment of chewing lice in sheep: 0.6% rotenone powder; 5% carbaryl powder; malathion liquid (500 gm in 100 L of water). Macrocyclic lactones (e.g. ivermectin) are not effective against chewing lice.

Linognathus ovillus

- **Description:** "long-nosed louse" "sheep face louse". This is a sucking louse and is found mainly on the face. It is long, narrow, black and about 2.5 mm in length. The eggs are also black but are hard to see.
- **Epidemiology:** The only host is the sheep. The adult lays one egg per day and the life cycle takes about 20 to 40 days to complete. They are not as common as chewing lice but severe infestations can occur. They can transmit a bacteria called *Eperythrozoon ovis*, which infects red blood cells and causes anaemia and fever.
- **Clinical Signs:** The parasite is found mainly on the face, but can be present anywhere in the body. They cause pruritus and rubbing leading to dermatitis. Because they suck blood, they can cause anaemia if present in large numbers.
- **Treatment:** As with chewing lice. Macrocyclic lactones are not labelled for sucking lice but there is some efficacy against this parasite. Contact your vet about whether it is appropriate to use it.

Mange

Chorioptes bovis

- **Description:** "leg and scrotal mange". Also known as *C. ovis, C. caprae, C. cuniculi* and *C. equi* which reflects the other hosts it will infect (goats, cattle, horses and rabbits). The mites do not burrow into the skin but do cause irritation.
- **Epidemiology:** The mites are most often found on the pasterns and dew claws but do the most damage when present on the scrotum. The disease is most commonly seen in the winter months and signs decline in the spring.
- **Clinical Signs:** The skin becomes thick, scabby and itchy. The scrotum has a leathery appearance. The inflammation will heat up the testicles thereby causing testicular hypoplasia and sub or infertility.
- **Treatment:** Ivermectin has some effect but some cases do persist despite repeated treatment. Moxidectin, not licensed for sheep in Canada, is more effective - likely because of the persistency of the drug. Contact your veterinarian to discuss this further.

Psoroptes ovis

Description: "sheep scab". These are burrowing mites that occur over the entire body.

- **Epidemiology:** This parasite has not been diagnosed in North America in several decades and is believed to be exotic to this continent. It is a highly contagious disease in the UK, where the infection is common. It is only controlled by treating the entire group and preventing reintroduction through biosecurity (new introductions, fence-line contact or community pastures, fresh wool products). Endectocides with persistence (e.g. moxidectin) are effective. If insecticides are to be used, the entire sheep must be plunged dipped to get the entire body and head. Repeated treatments may be necessary.
- **Clinical Signs:** Sheep are very pruritic over their entire bodies, with great sores and wool loss although milder disease occurs in endemic situations. Untreated sheep are a welfare concern.

Sarcoptes scabiei

- **Description:** "scabies". This is a burrowing mite and prefers the non-wooled areas of the body, e.g. face, ears, groin and axilla. It is not common in Canada.
- **Clinical Signs:** The parasite causes intense itching to the point where the sheep would rather scratch than eat making this disease a welfare issue.

Treatment: Contact your veterinarian if you suspect this mite. Macrocyclic lactones have some effect.

Parasitic Flies

Melophagus ovinus

- **Description:** "sheep ked". This is a wingless fly, 0.5 to 0.8 cm in length. It has a tick-like appearance but is not related to ticks. It prefers living on the neck, shoulders and belly.
- **Epidemiology**: Keds can also infect goats but not commonly. The fly cannot live off the sheep very long and transmission is by direct contact. The keds will remain close the body of the sheep except in warm weather (> 21 C°) when they may be found at the top of the wool fibres, so transmission is more likely in the summer. The eggs hatch inside the female and develop through the 3 larval stages inside the fly. Once "born" the larvae pupate and can be seen in the wool as reddish-brown 3 to 4 mm long pupae. This form is very resistant to insecticides. Adult keds emerge in 3 weeks although this may be longer in the winter months. However, ked populations only build up slowly as a female only gives birth to 20 larvae in her lifetime. The ked feeds on blood through biting the skin, and heavy infestations will cause anaemia.
- **Clinical Signs:** The most obvious sign is pruritus and tearing at the wool as with chewing lice. The wool is discoloured a pinkish-brown by the excrement that contains digested blood. The wounds caused by the bites can be secondarily infected.
- **Post Mortem:** Other than anaemia and pruritus, the biggest economic issue is the destruction of the hide by a condition called "cockle" which is a response to the irritation caused by the biting of the ked. Cockle is a discolouration of the hide with raised nodules (scars) and affected hides are discarded. While this is a loss to the hide industry, it is less important to the producer. But the pruritus is worrisome to the sheep and so should be appropriately managed.
- **Treatment:** Shearing will remove the drug resistant pupae. Topical insecticides (pyrethroid (Ectiban 25 Fly Killer, Engage Animal Health) + see chewing lice) and avermectins (not licensed for this parasite) will greatly reduce the population if treated at the same time as shearing. Otherwise, repeated treatments with topical insecticides is required to control the infestation.

Oestrus ovis

- **Description:** "nose bots". The parasitic stage of this fly is the larvae which inhabit the nasal passages of the sheep, causing irritation. The flies the free-living stage- are greyish brown slightly more than 1 cm in length with a coat of short, brown hairs and small black spots on the abdomen. The larval stage will grow up to 3 cm in length, are whitish with black, horizontal bands, and the head has black hooks near the mouth.
- **Epidemiology:** Goats can also be a host. The female fly squirts a jet of liquid containing larvae at the external nares of the sheep as they fly by. This jet can contain up to 25 larvae and are very tiny (1 mm) at this time. The larvae start to migrate up the nose through the sinuses causing mucous production through irritation. They attach to the lining of the passages using the oral hooks, which further irritates the sheep. The L₁ moult to L₂ in the nasal passages and the L₂ moult to L₃ in the frontal sinuses. Once the L₃ are fully grown, they are sneezed out where they pupate on the ground and then emerge as flies. Time from infection to dropping out is variable with a shorter time in the summer (2)

weeks) for up to 9 months in the winter. The fly only survives 2 weeks in the summer but can lay up to 500 larvae. During the summer, 2 to 3 generations can occur so that the sheep has continual infections over the summer months. Over-wintering occurs in the sheep, where the L_1 and L_2 remain quiet until the weather becomes warmer in the spring, when they will mature to L_3 to complete the cycle again.

- **Clinical Signs:** The signs associated with larval development and migration are sneezing, mucous production and rubbing their nose in irritation. Sometimes severe infections cause nose bleeds. Occasionally the larvae will die in the sinus cavities and secondary bacterial infection may penetrate to the brain. Heavy infections may cause ataxia from the irritation. However, the fly also causes the animal distress. As the fly approaches, the sheep will bunch, stamp their feed and bury their heads towards the ground to avoid the flies laying the larvae. They are very irritated and will not graze while a fly is present. Sometimes this behaviour is called "gadding" and can be associated with weight loss if bot flies are plentiful.
- **Treatment:** Ivermectin is approved for use to treat nose bots and will kill all larval stages. It has been recommended that if nose bots are a particular problem, a treatment mid-summer and one in the mid-winter to kill the over-wintering larvae, will effectively control this parasite. Systemic organophosphates are also effective but must be used only with the advice of a veterinarian as they can be toxic both to the animal and the person handling them.

Fly Strike

- **Clinical Signs:** Fly strike, or myiasis, is caused by a number of species of flies that lay eggs on wounds, wet skin or in dead tissue. The larvae (maggots) may consume live tissue or the debris (blood, pus, necrotic tissue) of the wound. A low infestation of maggots cause no signs in the sheep beyond odour and the presence of the maggots, but severe infestations cause depression, fever and anorexia because of the tissue damage and absorbed toxins. The toxaemia can cause liver and kidney failure depending on the severity. The affected sheep stand apart from the flock and may have a dark wet appearance to the fleece where they are "struck".
- **Treatment:** Affected animals should be identified and have the wool clipped from the affected area. The maggots need to be killed or they will pupate to flies if just removed from the wound. Many topical insecticides will work but none is approved for this purpose in sheep.
- **Control:** To prevent future infestations is more problematic because there must be persistency of the product in the wool. Synthetic pyrethroids and organophosphates have been used for this purpose and are applied by hand spraying, plunge dipping, or jetting. Applications of these products can be done 2 X in the summer (e.g. May and August) to give protection throughout the high risk period. However, nothing is approved for this purpose in sheep. Other measures such as proper wound treatment, shearing before pasturing, tail docking and preventing diarrhea by controlling internal parasites will all help prevent fly strike.

Lucilia sericata

Description: "greenbottle" blowfly. The flies are ~ 1 cm in length with a greenish to bronze sheen. They are attracted to the odour of decomposing flesh and will lay eggs on dead animals or live animals with

wounds. Female flies must have a protein meal before they lay eggs. L. cuprina - important in Australia and New Zealand - is thought only to be a problem in warm climates.

Epidemiology: The eggs are hatched on the wool and the larvae crawl down to the wound. They secrete enzymes which break down tissue and liquify it so it can be eaten. The odour caused by this infestation will attract more blowflies, so left untreated - a minor infection can become a severe one which will overwhelm the animal. While wounds play an important role (e.g. tail-docking), so will wet, soiled fleece from diarrhea, birthing fluids or heavy rainfall conditions. Foot rot or foot scald can also attract blowfly strike.

Calliphora spp, Phormia regina, Protophormia terranovae

Description: "Bluebottle fly", "blackbottle fly". Most of the Calliphora flies occur only in Australasia, but a few occur in Canada. They are usually secondary invaders to a fly strike.

CONTROL OF THE INTERMEDIATE STAGE OF DOG TAPEWORMS

There are many types of dog tapeworms and some of these have an intermediate stage which can infect sheep. The damage they do to the sheep is usually limited to the internal organs and / or carcass and the effect is usually economic. However these economic losses can be devastating with regards to losses associated with carcass condemnation. One of the types is also dangerously zoonotic. All can be controlled using similar measures.

Important Dog Tapeworms - Intermediate Stage

Echinococcus granulosus

- Description: "Hydatid disease". This tapeworm of dogs and wild canids (coyotes and wolves) is highly zoonotic. The tapeworm in canids is short (~½ cm in length) making it difficult to see in the intestine. The intermediate stage is a hydatid cyst rather than cysticercus. These cysts form in the liver or lungs of the sheep (or human) and can grow very large up to 20 cm. Inside these cysts are tens to hundreds of tapeworm larvae (hydatid sand), each one capable of growing to a tapeworm. If the cyst forms in the abdomen, it may grow very large, containing several litres of fluid.
- **Epidemiology:** Many ruminants can be the intermediate host (along with humans) but sheep have been historically implicated in maintaining dog infections. The dog can by infected with thousands of adult tapeworms without signs and infected sheep rarely show signs. But humans with cysts develop signs of respiratory disease (lung) or liver disease. If one ruptures, the person may die of anaphylaxis. Children that play with infected dogs are particularly at risk. Dogs become infected because they are allowed to scavenge infected sheep. Evidence of cysticercuses in sheep indicates that the management of sheep and dogs is conducive to Echinococcus infection. At present this parasite occurs in northern Quebec and Ontario, but not in the southern parts of these provinces.

Cysticercus tenuicollis

Description: This is the intermediate stage of the dog tapeworm *Taenia hydatigena*.

- **Epidemiology:** Dogs, wild canids (wolves, fox, coyotes), weasels and stoats are the final host (i.e. adult tapeworm occurs in these species). Sheep, goats, deer and cattle are the intermediate hosts. The dogs consume the infected intermediate host (e.g. sheep offal) and become infected. The adult tapeworm sheds segments which contain thousands of eggs, in the faeces. The eggs contaminate pasture or feed which the sheep eats. If the sheep eats an entire segment, then death may occur from severe liver damage. The eggs hatch and the larvae migrate for about 4 weeks, eventually to the liver and abdominal cavity where each larva forms a cysticercus. This is a bladder-like structure that contains one embryonic tapeworm or protoscolex (head only). These cystic structures are fairly large (1 to 3 cm) but do not harm the sheep. Eventually the cysticercus will die and scar if not consumed.
- **Clinical Signs:** There are no clinical signs in the sheep (with the exception of a massive infection which may cause liver failure) or the dog.
- **Post Mortem:** Larval tracts, bladder-like cysts and scars can be seen in the liver, causing condemnation of that organ. While not economically devastating, the presence of this infection indicates a farm-level problem with management of dead-stock (scavenging) or offal from slaughtered animals. This sets up the possibility of infection with *Cysticercus ovis* (see below) which can be economically devastating.

Cysticercus ovis

Description: "Sheep Measles". This is the intermediate stage of the dog tapeworm *Taenia ovis*.

Epidemiology: The dog and wild canids (wolves, coyotes, foxes) are the final host. Sheep and goats are the intermediate hosts (not deer). This is an emerging disease in Canada. The adult tapeworm is long and sheds segments that each contain over 70,000 eggs. The segments are found in the faeces but also on the coats of dogs. They have been know to travel up to 80 metres across pasture. The eggs contaminate pasture and feed. Sheep consume the eggs which hatch in the digestive tract. The larvae migrate to the muscles (skeletal, heart, diaphragm, masseter muscles) where they form small cysts ~ 1 cm in size. After 2-3 months, the cysts are infective to dogs. This life cycle is portrayed in Figure 5. The cysts may die but some can remain viable in the sheep for over 1 year. While sheep eventually develop resistance to the tapeworms, a new crop of naive lambs continues the cycle - as long as dogs and wild canids are allowed to scavenge or eat sheep. If a dog is allowed to eat an infective cyst, the prepatent period is 6 to 9 weeks.

Clinical Signs: There are no clinical signs in the sheep or the dog.

Post Mortem: The cysts are apparent at slaughter and depending on the number and distribution, cause the carcass to be condemned. The disease is not zoonotic, but affects meat quality so an outbreak - causing condemnation of a large percentage of carcasses - can be economically devastating to the industry.

Figure 5.



Control and Prevention of Cysticercus ovis

Control of the infection in sheep is done by controlling the infection in your farm dogs and preventing infection of coyotes, wolves and foxes. Once a sheep is exposed to the tapeworm eggs, there is no available method of preventing development of the cysts – either through medication or vaccination.

Control is done by:

- proper deadstock management to prevent scavaging of carcasses by all canids including guard dogs, neighbours dogs and coyotes
- reduce predator losses through a variety of means.
- routine deworming of all farm-dogs with medications effective against tapeworms (see below)
- assuring that farm dogs are only fed safe forms of dog food (see below)

Routine Deworming of the Dog

Talk to your vet about what option is best for your operation, generally all dogs with access to sheep or goats should be de-wormed monthly

- Droncit injectable; (Praziquantel)
- Droncit tablets; (Praziquantel)
- Lopatol tablets; (Nitroscanate)

- Drontal Plus tablets; (Praziquantel + Pyrantel pamoate + Febantel)
- Cestex tablets; (Esiprantel)

If Sheep Carcasses are to be Fed to Dogs

- Freezing: freeze carcass to -10 degrees Celsius for 7 days OR
- Cooking: cook meat to an internal temperature of 72 degrees Celsius

GLOSSARY OF ABBREVIATIONS

| AAD | Amino-Acetonitrile Derivatives | FECRT | Faecal egg count reduction test |
|------------|-----------------------------------|-------|---------------------------------|
| ADG | Average daily gain | LV | Levamisole |
| AR | Anthelmintic resistance | GIN | Gastointestinal nematodes |
| BZ | Bendizimadoles | ML | Macrocyclic lactones |
| СТ | Condensed tannins | PPER | Periparturient egg rise |
| epg FEC | eggs per gram Faecal egg count | SL | Sericae lespedizia |
| | | | |

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