Epidemiology of Gastrointestinal Parasites

We will examine how the parasite interacts with its host (the sheep or goat) and the environment and what determines how well the parasite survives and whether disease occurs in its hosts.

Figure 2 summarizes gastrointestinal nematode parasite burden in lambs and adults, as well as the number of infective L3 on pasture, under conditions in central Canada. This graph is adapted from data obtained on farms in Ontario and Quebec in 2006-2008. The abstract of this published work is included in Appendix 5. In the spring, adult sheep/goats serve to contaminate the pasture for youngstock, which then are the major source of pasture contamination through the grazing season. A hot dry summer will lower pasture contamination, while numbers will rise with the warm rains that often occur in late August - September.

Normal Patterns of Infection in Adults and Youngstock

Youngstock in Their First Grazing Season

Lambs and kids have no acquired immunity to gastrointestinal nematode (GIN) parasites, although some may have an innate (born with) immunity of varying levels. The L3 stage on pasture infects naive lambs and kids. The level of L3 on pasture, the level of immunity in the youngstock will determine the level of disease seen. Over the grazing season, the parasite load in the lambs and kids tend to increase and they quickly become the major contributors to egg contamination on pasture. Towards the end of the grazing season, a proportion of the ingested L3 will not progress to adults, but will rather become hypobiotic or arrested in development. If youngstock are not grazed, then they will not develop immunity until they are grazed as adults (e.g. yearlings). Immunity in sheep takes several months (up to 5 months) to develop. Goats do not develop immunity.

Adul Sheep and Goats

Adults, if they have been previously grazed, may carry infection over the winter as arrested L4, and will contribute to pasture contamination when turned out to graze in the spring from the emerging adult parasites. The level of egg shedding will depend on the parasite exposure and level of immunity they developed in the previous grazing season. Adult goats develop immunity less well than sheep and may have a more important role than adult sheep in contaminating pasture. The phenomenon of periparturient (around lambing / kidding) egg rise is due to a lowering of immunity (called “down-regulation”) around the time of lambing or kidding (see below). This allows for increased egg production by parasites, and thus serves to be one of the most important sources of pasture contamination in the spring to lambs and kids.

Hypobiosis or Arrested Larval Development

After the L3 larvae infect the host and moult to the L4 stage (for Trichostrongylus – L3) in the animal, they may either develop into adults or arrest as larvae. 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 disease. The trigger for hypobiosis is thought to be unfavourable environmental conditions for egg hatching and development of the free-living larval stages, e.g. the cooling
weather of autumn in temperate climates or the dry season in the tropics. In Canada, arrested development is an important mechanism that allows for survival of *H. contortus*, as well as *Teladorsagia* sp. and *Trichostrongylus* spp. It is believed that in Canada most L3 larvae ingested in the fall - and late summer in the case of *Haemonchus* - arrest rather than develop to adults.

**IMMUNITY AND PARASITE BURDEN**

**ACQUIRED IMMUNITY TO PARASITES**

Lambs and kids (though less well) will develop immunity to parasites over time. The actual length of time varies with the type of GIN but generally occurs over the first grazing season if long enough (4-6 months). Nursing lambs and kids tend to have no immunity but also have very little exposure to GIN parasites until they start to graze. Once they graze, immunity – at least in lambs – starts to develop. However, the time for immunity to develop in an individual varies between species or breeds of animals and between animals in a flock. There is a moderate heritability of this ability, which scientists are working to identify and exploit with specific genomics testing and breeding programs. When immunity develops, the adult parasites are expelled but the animal will continue to be infected with low numbers. This is called “self cure”.

Acquired immunity to parasites can be lowered by several factors. It is known that immunity to parasites is short-lived. Without continued exposure to parasites, the animal’s immunity will wane and after a few months, it can become susceptible again. We see this in Canada when sheep / goats in the spring will have poor immunity after being housed all winter. Additionally, challenge with high numbers of GIN on pasture can overwhelm the animal’s immune system and cause disease.

Immunity is also greatly affected by nutrition. Diets deficient in protein, particularly dietary protein as rumen non-degradable protein, also called “by-pass” protein, can lower the animal’s ability to mount an immune response to GIN parasites. This type of protein 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 are fishmeal, roasted soybeans and corn gluten. Supplementation with diets with higher protein is associated with lower egg counts in sheep; this is most easily seen at lambing and lactation. Some pasture plants containing condensed tannins (e.g. sanfoin and bird’s-foot trefoil) can also supply this type of protein. It is very important to remember that goats do not develop immunity as well as sheep.

**PERIPARTURIENT EGG RISE (PPER)**

This phenomenon is due to the “periparturient relaxation of immunity” (PPRI), and refers to the increase in eggs passed in the feces of ewes and does from a few weeks before giving birth through the nursing period (around 8 weeks). This typically takes place in the spring months and occurs because of a down-regulation (lowering) of immunity in the late-pregnant female which allows for the following: arrested larvae to mature to egg-producing adults; ingestion of overwintered L3 on pasture to more likely result in infection; and an increased rate of egg production from existing adult worms. All of this results in a dramatic increase in pasture contamination in the spring at lambing / kidding and while nursing. Nutritional stresses in late pregnancy and lactation will increase the likelihood of PPER being high. PPER tends to be lower in single-bearing females compared to those with multiples, lower in mature females than first-timers, and lower when females are supplemented with by-pass protein sources.

**PPER in out-of-season lambing flocks**

Findings from research conducted in Ontario flocks on accelerated lambing programs found that ewes lambing in the winter also experience a PPER, while those lambing in the fall already had very high levels of infection and PPER was not apparent. The abstract describing this work is presented in Appendix 5. Thus PPER will occur regardless of season although it appears to be less of a feature in fall lambing ewes, perhaps because the parasites have not yet become hypobiotic. Canadian data also suggest 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

Immunity to parasite infections may be innate (i.e. the animal is born with the ability to manage parasite infections) or acquired after exposure to parasites. Both are important. Some sheep and goats develop better immunity against parasites, and are more able to resist establishment. This can be more pronounced in some breeds, e.g. some tropical hair breeds of sheep are known to develop immunity more quickly than some northern wool breeds. Also, within any population of sheep or goats, there is variation in this ability, and a portion of that ability is genetic. This trait is considered moderately heritable.

Programs have been developed to identify sheep that carry this genetic ability, 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. Both of these methods have drawbacks:

- There is tremendous variation in day-to-day faecal egg counts (FEC) and so to use FECs, a large number of animals are needed to detect significant differences in egg output that are due to genetics.
- Additionally, the animals need to be exposed to significant burdens of parasites to have high enough FEC to detect a difference between animals.
- Gene markers may detect animals of one breed with parasite resistance but in another breed, these markers don’t work.

Newer methods of measuring immunity instead (e.g. CarLA Saliva Test, AgResearch NZ) have shown promise. Fewer animals are needed to detect differences and lambs with superior performance can be detected as little as 3 months after going to pasture. Research is being conducted in Ontario to determine if this technology can be used here.

It must be remembered that lambs or kids with superior ability to develop acquired immunity, are susceptible to GIN infection until that immunity is developed and so need to be monitored for infection. The benefit comes later, after they have an opportunity to develop immunity. As adults, these resistant animals will shed fewer eggs, most importantly at the time of PPER, or if faced with an excessive burden on pasture. Their offspring are also more likely to be able to manage parasite challenges.

RESILIENCE TO INFECTION WITH PARASITES

This is the animals’ ability to thrive in the face of parasitic infection. These animals are infected, and pass eggs that contaminate pasture, but appear to be healthy. Within any population, there are individuals that exhibit resilience. But they serve to contaminate the pasture for animals that are not resilient or immune. It is also less heritable than the trait for superior immunity. This means that while some animals aren’t affected by increased pasture contamination, a larger part of the flock is affected and may suffer disease. So for this reason, resistance is preferred to resilience.

NORMAL PATTERNS OF INFECTIVITY ON PASTURE

EFFECT OF ENVIRONMENT ON DEVELOPMENT AND SURVIVAL OF THE FREE-LIVING STAGES

Temperature

At temperatures < 10°C, egg hatching, larval development and moulting do not occur. Parasite larvae develop at temperatures above this but prefer warmer temperatures for optimal development. The optimal temperature for hatching, larval development and L3 survival varies by parasite:

- *Teladorsagia circumcincta* prefers 16°C - 30°C;
- *Trichostrongylus colubriformis*, 22°C - 33°C; and
- *Haemonchus contortus* prefers the warmest temperatures at 25°C to 37°C.

Northern spring weather conditions may not be favourable to rapid egg hatching and larval development. But when temperatures range from 25°C to 30°C during the summer, the development of all parasite larvae is favoured. When summer weather is hot, while the other parasites usually take 3 weeks from egg to L3, *Haemonchus* may develop in as
little as 5 days. As our climate continues to warm, all GIN parasites – but in particular Haemonchus – will enjoy more favourable conditions for development.

Although warm weather hastens parasite development to L3, it may conversely lower the length of survival of those larvae. When the weather is hot (e.g. > 28°C), the L3 may die more rapidly because their metabolic rate increases and they outlive their stored nutrients (remember L3 cannot feed).

Conversely cool weather, which slows larval development, will also significantly increase survival of L3 from weeks to months because of the lowered metabolic rate. At temperatures < 5°C, the metabolic rate of L3 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. L3, but not L1 or L2 can survive desiccation (drying) because of protection of the cuticle covering, even at freezing temperatures.

### ASSUMPTIONS REGARDING DEVELOPMENT AND SURVIVAL OF FREE-LIVING STAGES ON PASTURE

**Survival of L3 over-winter on pasture**

Some species of L3 (e.g. Teladorsagia and Trichostrongylus but not Haemonchus) can enter a state of “anhydrobiosis” that allows them to survive severe cold and desiccation (drying), making them well suited for surviving the freeze-thaw cycles of our Canadian winters.

When sheep or goats are turned out in the spring to a pasture that has been grazed the previous late summer or autumn, it can be assumed that it is contaminated with L3 that have survived over-winter. If pasture contamination was high the previous autumn, then the level of L3 from Teladorsagia and Trichostrongylus will be high in the spring as well. Snow cover throughout the winter enhances survival. Several freeze-thaw cycles or prolonged, cold temperatures without snow cover may lower this survival rate. Haemonchus does not survive effectively when temperatures dip below 0°C. Over-wintered L3 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 warm / dry spring will shorten survival.

### Development and survival of L3 on pasture during the grazing season

As mentioned, the time from egg deposition to development to L3 varies depending on temperature and humidity. Moderate temperatures and high humidity hasten development to L3. Moderate temperatures will prolong survival of L3. It is likely that a proportion of L3 present in June and July, are still alive in September. 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 grazing (e.g. through pasture rotation) will reduce this mat and open the soil to sunlight and desiccation - 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.
Generations of GIN on pasture

One generation is the time from when the eggs are passed in the feces, through hatching and development of the free-living larvae, infection of the sheep or goat and then passage of eggs again in the feces. Assuming that time from egg deposition to L3 is 2 to 3 weeks (up to 6 weeks if cool) and time from infection to egg production is 3 weeks – so a total of 5 to 6 weeks on average. It is likely that optimal environmental temperatures are only present for 4 months in most of Canada (mid-May, June, July, August, mid-September – 16 weeks), so there is limited time for build-up of L3 on pasture during the first grazing season on a given pasture. This may change as our climate warms.

Teladorsagia and Trichostrongylus

In central Canada, it is unlikely that more than 3 generations of these parasites occur under our summer conditions. Therefore, it is likely that if severe parasitism from these parasites occurs on a farm, there was heavy contamination from the previous grazing season as well as high stocking densities in the current grazing season - along with optimal summer conditions for L3 development and survival, i.e. warm and moist conditions.

Haemonchus

Although Haemonchus doesn’t survive on Canadian pasture over the winter, it survives very well in adult sheep in the hypobiotic (arrested) state. Once the adult female worm matures in the spring, it is capable of producing 5,000 to 10,000 eggs per day. Under warm, humid conditions L3 will develop in as little as 4-5 days, allowing for multiple generations per grazing season (one generation could be as short as 3 ½ weeks) and therefore massive pasture contamination. This means that within one grazing season, the infectivity of the pasture may become very high and therefore risky to lambs and kids – and even adults - by mid-July to early August.

Survival of L3 indoors

Very little is known about survival and transmission of GIN indoors. There is sufficient anecdotal evidence to suggest that infection can occur in the barn. Certainly lambs and kids housed in drylot situations (i.e. where they have access to a yard without pasture), particularly if fed on the ground – may pick up GIN infection. Inside a barn, in the summer it may be possible for eggs to hatch and develop to L3, but again – lambs and kids would need to be fed on the ground to consume sufficient larvae to cause a significant infection. Water bowls and troughs contaminated with feces are another source of infection. Very rarely are parasite burdens acquired indoors of clinical significance. To lower the risk of GIN in this situation, it is important to minimize manure contamination of feed and water.

Next section is “Diagnosing Gastrointestinal Parasitism”