

Epidemiology means how the parasite interacts with its host (the sheep or goat) and the environment.

Figure 2

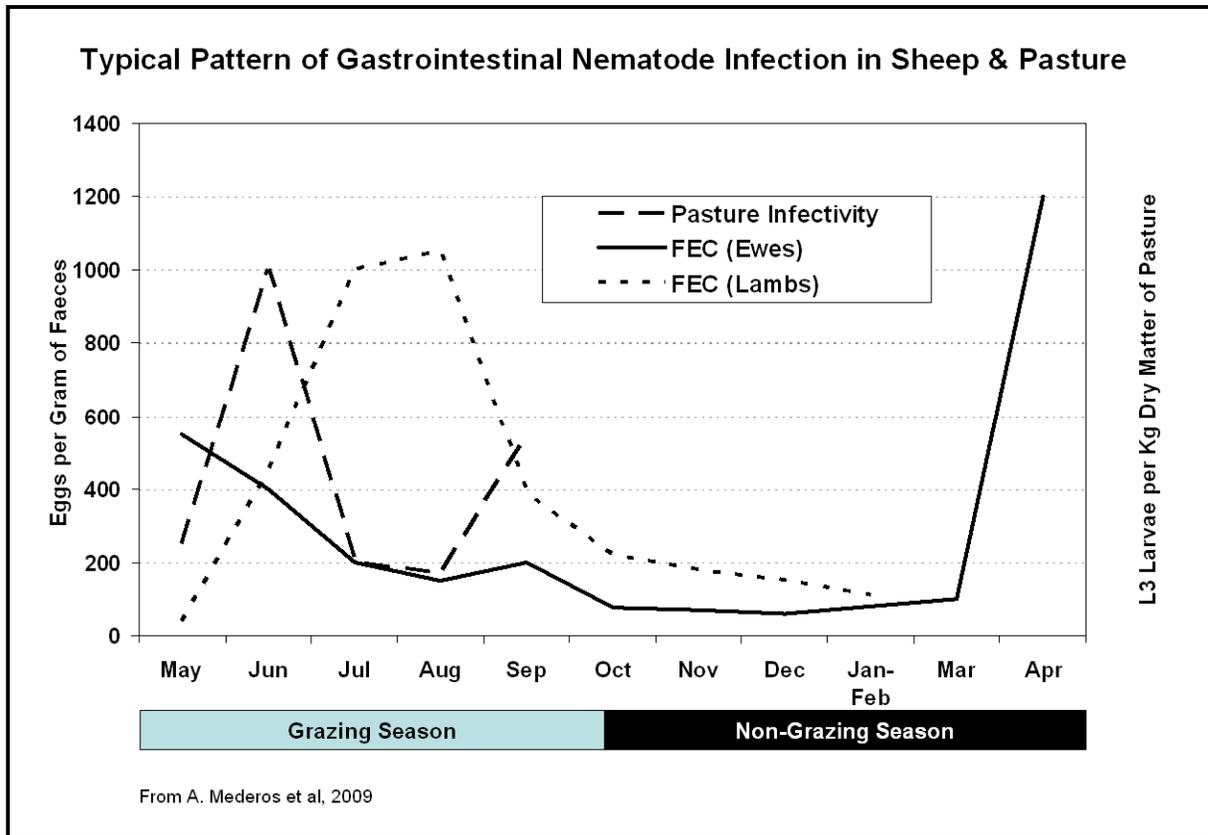


Figure 2 summarizes the typical level of gastrointestinal nematode parasite burden in lambs and adults, as well as the number of infective L3 on pasture, under conditions in central Canada. This graph is adapted from data obtained on farms in Ontario and Quebec in 2006 - 2008. Goats are similar. In the spring, adult sheep/goats serve to contaminate the pasture for lambs/kids, which then are the major source of pasture contamination. A hot dry summer will drop pasture contamination, while numbers will rise with the autumn rains.

NORMAL PATTERNS OF INFECTION IN ADULTS AND YOUNG-STOCK

YOUNG-STOCK IN THEIR FIRST GRAZING SEASON

These animals tend to have no natural immunity to gastrointestinal nematode (GIN) parasites. The L3 stage on pasture serves to infect naive lambs and kids. The level of L3 on pasture and the level of immunity in the young-stock will determine the level of disease seen. Over the grazing season, the loads in the lambs and kids 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 become hypobiotic or arrested. If lambs are not grazed, then they will not develop immunity until they are grazed as adults (e.g. yearlings).

ADULT SHEEP AND GOATS

Adult sheep tend to have a level of immunity to the GIN parasites if they have been previously grazed, but will still be infected and will contribute to pasture contamination. Adult goats develop immunity less well 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 “down-regulation” of immunity around the time of lambing or kidding (see below). This allows for increased egg production by adult female parasites, and thus serves to be one of the most important sources of pasture contamination to new born lambs and kids.

HYPOBIOSIS OR ARRESTED LARVAL DEVELOPMENT

After the L₃ larvae infect the host and moult to the L₄ stage, they may either develop into adults or stay at the L₄ 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 development of the free-living larval stages, e.g. autumn 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 L₃ larvae ingested in the fall - and sometimes late summer in the case of *Haemonchus* - arrest rather than develop to adults.

IMMUNITY AND PARASITE BURDEN

ACQUIRED IMMUNITY TO PARASITES

Lambs and less well kids 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 and by 4 to 6 months of age. However, this varies between species or breeds of animals and between animals in a flock. With immunity, the adult parasites are expelled but the animal will continue to be infected with low numbers. However, without continued exposure to parasites, the animal's immunity will wane and after 6 to 8 months, it can become susceptible again. Additionally, challenge with high numbers of GIN on pasture can overwhelm the animal'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 are fish meal, roasted soybeans and corn gluten. If the diet is supplemented with rumen by-pass protein, parasite resistance is improved. It is very important to remember that adult goats do not develop immunity as well as sheep.

PERIPARTURIENT EGG RISE (PPER)

Also called the “Periparturient Relaxation of Immunity” (PPRI), this term refers to the increase in eggs passed in the faeces 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. The down regulation of immunity is thought to occur because of nutritional stresses associated with late gestation. 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

We are currently conducting research into PPER in ewes lambing in the winter or fall compared to those lambing in the spring. The data suggest that ewes lambing in the winter also experience a PPER, while those lambing in the fall do not – this shows that season appears to play a role as well as nutrition in PPER. 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

Some sheep and goats develop better immunity against parasites, and are more able to resist establishment of infection after developing this immunity. 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. Within any population of sheep or goats, there is variation in this ability, and a portion of that ability is genetic. Programs have been developed to identify sheep carrying genes for resistance, either through ram selection (e.g. rams raised together and selecting those with lower faecal egg counts or higher levels of antibodies to parasites), or attempting to identify genetic markers in the DNA.

It must be remembered that lambs or kids with resistance genes, prior to developing immunity, are just as susceptible to GIN infection as those without resistance genes. The benefit comes later, after they have an opportunity to develop immunity – which may come more quickly in those animals with a better inherent ability to develop resistance. 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. This reduction of contamination is where the benefit lies to genetic selection.

RESILIENCE TO INFECTION WITH PARASITES

This is the animals' ability to grow and thrive in the face of parasitic infection. These sheep are infected, and pass eggs which contaminate pasture, but appear to be healthy. Within any population, there are individuals which exhibit resilience. But they serve to contaminate the pasture for animals that are not resilient or immune. So for this reason, resistance is preferred to resilience.

NORMAL PATTERNS OF INFECTIVITY ON THE PASTURE

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

Temperature

The optimal temperature for hatching, larval development and L₃ survival varies by parasite:

- *Teladorsagia circumcincta* 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 (e.g. > 28° C), the L₃ may die more rapidly because their metabolic rate increases and they outlive their stored nutrients (L₃ 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 L₃ is very low - allowing prolonged survival, e.g. over-wintering on pasture.

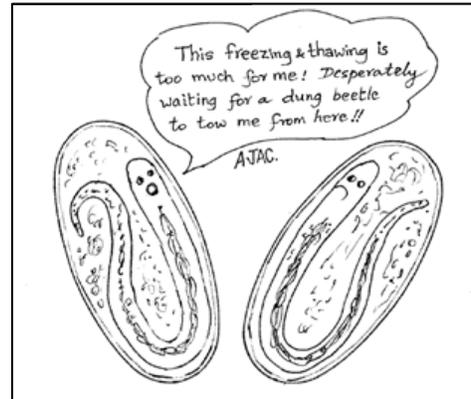
Humidity

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

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

Survival of L₃ Over-Winter on Pasture

When sheep or goats are turned out in the spring to a pasture that has been grazed the previous late summer - fall, it can be assumed that it is contaminated with L₃ that have survived over-winter. If pasture contamination was high the previous fall, then the level of L₃ 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. Several freeze-thaw cycles or prolonged, cold temperatures without snow cover may lower this survival rate. *Haemonchus* is generally assumed not to survive well in the central Canadian climate. 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 hot spring will shorten survival.



Development and Survival of L₃ on Pasture during the Grazing Season



As previously 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. Cool temperatures will prolong 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 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 the eggs passed in the faeces, through hatching and development of the free-living larvae, infection of the sheep or goat and then passage of eggs again in the faeces. Assuming that time from egg deposition to L₃ 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 L₃ on pasture during the first grazing season on a given pasture.

Teladorsagia and *Trichostrongylus*

While these data are not known specifically for central Canada, it is unlikely that more than 2 or at the most 3 generations of these parasites occur under our summer conditions (from passing of eggs to infection and passing of eggs). Therefore, it is likely that if severe parasitism from these parasites occurs on a farm, there was likely 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* does not appear to survive on pasture over the winter, it survives 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 5 to 7 days, allowing for multiple generations 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 by mid-July to early August, and to adult goats, and occasionally to adult sheep - if the loads are great enough.

SURVIVAL OF L₃ INDOORS

Very little is known about survival and transmission of GIN indoors. There is sufficient anecdotal evidence to suggest that transmission can occur but usually at very low levels. 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 to L₃, but again - lambs and kids would need to be fed on the ground to consume sufficient larvae to cause a significant infection. So, to lower the risk of GIN in this situation, it is important to minimize manure contamination of feed.

