INFO SERIES

September 2021

The Four Phases of Gastro-intestinal Worm Infection

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Another way to look at the life cycle of gastrointestinal nematode parasites is in four phases. Phase 1 is the Parasitic Phase which is the interaction between the animal and the worm. Phase 2 is the Contamination Phase which is the result of eggs that are passed in the feces during defecation. Phase 3 is the Free-Living Phase when larval stages develop and survive outside the animal. Phase 4 is the Infection Phase when available infective larvae are consumed during grazing. A number of factors affect what happens and influences control strategies during each of these phases.

PHASE 1: PARASITIC PHASE

During Phase 1, the worm has to develop and survive in the host. After ingestion, infective larvae lose their protective sheath and invade the mucosa (lining) of the abomasum, small intestine, or large intestine depending on which worm is involved. While in the mucosa, larvae develop to the next larval stage and then return to the surface of the gut mucosa where they become adult worms.

The animal's major defense mechanism against infection is the immune system. When infectious agents enter the body, the immune system reacts through a series of pathways that mobilize various components (antibodies, killer cells, etc.) that then attack and kill the invaders. These components act on the larval stages in the mucosa and the adults. How strong the immune response depends on several factors.

The immune system matures with age; therefore, young animals are relatively susceptible to infection and become more resistant with age. So, young animals usually harbor the heaviest infection levels and suffer the most severe consequences. Adult animals have developed stronger immunity and harbor fewer worms. One way infection level is measured is by quantifying the

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number of eggs being passed in the feces. Relatively high and low fecal egg counts are usually seen in young and adult animals, respectively. Young animals are more subject to clinical disease where signs of infection (diarrhea, rough hair coat, anemia, weight loss, bottle jaw, etc.) are seen. In older animals, infection usually becomes more subclinical where the only subtle sign may be weight loss. However, nutrition and/or stress can alter a host's immune competence. Under poor nutrition and stressful conditions, the immune system loses some effectiveness, cannot respond adequately, and, no matter what the age of the animal, the effects of infection can become worse (see Best Management Practices to Control Internal Parasites in Small Ruminants Fact Sheet: Nutritional effects on parasites, Steve Hart, October, 2018).

Of particular note in small ruminants is a phenomenon called the periparturient rise (PPR) in fecal egg output (see Best Management Practices to Control Internal Parasites in Small Ruminants Fact Sheet: The periparturient egg rise, Joan Burke, April 2020). This occurs at or around parturition and extends through most of the lactation period. Because parturition and lactation are stressful conditions, the dam's immune system is compromised. Furthermore, nutrients are partitioned preferentially to support mammary and fetal development and then lactation, which also decreases the animal's ability to generate an effective immune response to worm infection. Providing a high protein diet will help partitioning to support immune function.

Breeding and selecting for genetic resistance to infection is also possible, as within breed and between breed resistance has been demonstrated. The mechanism of resistance is predicated on improved immune response, thus fewer worms (see Best Management Practices to Control Internal Parasites in Small Ruminants Fact Sheet: Genetic selection, using cross breeding and estimated breeding values, Scott Bowdridge and Andrew Weaver, February 2019; On farm selection for resistance to parasites, Jim Morgan, January 2021).

The prepatent period (time between ingestion of infective larvae to egg laying adult females) of most worms is about 3 weeks, but this period can be extended (by a few months) for worms that have the capability to enter a period of delayed or arrested development called hypobiosis. This occurs during the season of the year when the environmental conditions are unfavorable for development and survival of the free-living larval stages. In warm climates, this happens either during summer or winter depending on the worm. In colder climates, all worms capable of hypobiosis will arrest in the winter.

PHASE 2: CONTAMINATION PHASE

The magnitude of pasture contamination during Phase 2 is affected by stocking rate (number of animals per grazing area), age of the animals, season of the year, and hypobiosis. The higher the stocking rate, the more feces are deposited on the grazing area and, thus, more eggs. The reverse is also true; lower stocking rates result in less feces and fewer eggs on pasture. More eggs are also passed from younger than older animals. Most worms have a definite seasonality, so during their "season," more eggs are produced and passed. Of particular note is the periparturient rise in fecal egg output. Thus, the existing female worms increase the number of eggs laid and deposited in the feces.

Depending on location, some worm species undergo hypobiosis. This results in fewer adult worms during the hypobiosis period and fewer eggs deposited in feces. However, when hypobiotic larvae resume development (especially in the spring coinciding with parturition/ lactation) massive numbers become mature adults over a short period of time. The resultant egg production (contributing to the periparturient egg rise) and deposition in the feces can be very high which results in major pasture contamination. And, the increased worm burden can have severe adverse effects on the animal.

PHASE 3: FREE-LIVING PHASE

Development and survival of the free-living stages during Phase 3 depend on prevailing environmental (primarily temperature, moisture and available oxygen) and nutritional (available food supply for energy) conditions. All of these contribute to larval rate of development, stage survival and length of survival. Initially, the first stage larvae develop in the egg which then hatches. Development and survival to second-stage and finally third-stage (infective) larvae occur within the fecal mass. The first- and second-stage larvae are unprotected and need oxygen and energy (feeding on nutrients and microorganisms) to grow. The infective third-stage larva is enclosed in a protective sheath and does not feed.

Temperatures conducive for normal development and survival are between 65 to 85°F (18 to 30°C). Lower or higher temperatures reduce development and survival. Moisture is also crucial for development and survival. Because the initial development and survival occur within feces, moisture is usually adequate to complete development to the infective larvae. However, if the feces dry out quickly, due to high temperatures and/or physical disruption, the first- and second-stage larvae are susceptible to desiccation and will die. If feces remain intact, retain some moisture, and do not get too hot or too cold, infective larvae may remain alive for months. A moisture medium (rain or heavy dew) is necessary for infective larvae to migrate out of feces. They are relatively resistant to environmental conditions due to their protective sheath.

Temperature is usually the only factor that may adversely affect infective larvae. Generally, infective larvae can survive very low temperatures, but may die off during hard freezes. Sustained temperatures above 95°F (35°C) are usually lethal. The moisture conditions at ground level under forage cover usually are adequate for infective larvae to move around and survive. Since they don't feed, their length of survival depends on how fast they use up their energy reserves. So, the hotter it is, the faster they move and use up energy stores and survival is shorter. Eventually, infective larvae move up and down the forage when there is a moisture medium (i.e., advancing and receding dew). Rain also provides a moisture medium for larval movement on forage.

PHASE 4: INFECTION PHASE

Phase 4 is affected again by stocking rate in two ways. If the same animals are grazing as deposited the feces, the stocking rate determines how many eggs initially contaminated (Phase 2) the pasture and, consequently, how many infective larvae will be available for consumption. If the initial contaminating animals are removed and replaced by new animals, the new stocking rate will determine the level of exposure each animal has to infective larvae during grazing, i.e., the higher the stocking rate, the more chance of exposure and vice versa. It is well known that animals usually do not graze close to feces so exposure to larvae is reduced the further the distance between fecal deposits.

Eventually feces disintegrate, forage grows well with fecal fertilization, and animals will graze over the area where exposure can be high. For

the most part, infective larvae do not move much past 12 to 24 inches from feces or 2 to 3 inches up forage plants. So, the lower the animals graze forage and the closer grazed forage is to feces, consumption of infective larvae is increased and vice versa.

Natural sources of water, such as streams, ponds, or lakes provide moisture along the banks where forage can readily grow. When animals congregate to drink and consume the attractive forage, defecation in these areas usually leads to increased contamination and eventually more infective larvae. The same can be said for areas where supplements, especially hay, are fed on the ground if conditions are right for development and survival of the free-living stages. Similarly, trees provide an area for animal congregation and shade. Under all these situations, essentially a high stocking rate has been artificially created in a relatively small area where forage is kept closely grazed. One successful strategy to reduce reinfection rate is to strip-graze several small low infectivity pastures (sometimes at a high stocking rate) for short periods of time (a few days) and move to the next pasture, not returning to the same pasture for at least 45 or more days. This allows some die off of larvae before returning (see Best Management Practices to Control Internal Parasites in Small Ruminants Fact Sheet: Pasture management, Heather Glennon, December 2017).

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