

Diagnosis, Treatment, and Prevention of Common Small Ruminant Parasites

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Abstract

External parasites of small ruminants include lice, keds and mange mites. They cause pruritus, fleece damage, and in some instances blood loss anemia. The most important internal parasite of sheep and goats is *Haemonchus*, a blood sucking strongyle that can kill animals on contaminated pasture in a matter of weeks. Overuse of dewormers has caused the development of parasite resistance, and currently recommended programs emphasize selective treatment and eventual culling of animals that show severe clinical signs. Untreated animals are left in the herd to provide unselected parasites *in refugia*. Other strongyle species contribute to production loss by causing weight loss and diarrhea. Tapeworms are of minimal clinical importance but, regionally, liver flukes cause ill thrift or death. Fencing off ponds and stream is often more effective than deworming for fluke control. In regions where whitetail deer abound on pastures, the meningeal worm *Parelaphostrongylus tenuis* causes neurologic disease and sporadic losses of small ruminants and guard llamas.

Introduction

Every small ruminant herd health program must address parasite control. In addition to choosing effective drugs and appropriate dosage rates for the small ruminant species being treated, the veterinarian needs to emphasize nutritional programs that optimize immunity and barn and pasture management techniques that minimize exposure. These programs must be tailored for each geographic region and each individual herd within that region. Monitoring programs, especially fecal egg counts for strongyles, are critical to the success of the parasite control efforts.

External Parasites

External parasites of concern to small ruminants are lice, keds and mange mites. Pruritus is a hallmark of infestation with these parasites. Sucking lice (*Linognathus* spp) and biting lice (*Bovicola*, also called *Damalinia* spp) cause rubbing, scratching, chewing and

a ragged fleece. Keds (*Melophagus ovinus*), which are wingless flies and are often referred to as sheep ticks, cause pruritus and suck blood. Sucking lice and keds can cause significant anemia. Chorioptic mange and demodectic mange occur in both sheep and goats in the US. *Chorioptes* typically makes thick crusts on the pasterns and scrotum while *Demodex* mites distend individual hair follicles, making small nodules in the skin over the neck, shoulders and sides of goats. Sarcoptic mange is seen in goats, but not sheep in the US. *Sarcoptes* mites cause widespread lesions over the body and head. *Psoroptes cuniculi* ear mites are commonly found in the ears of goats. The mites cause goats to shake their heads and scratch at their ears, only rarely generalizing unto the skin of the body. Psoroptic mange of sheep (*Psoroptes ovis*) is a severe disease (sheep scab) that has been eradicated from the US.

Fleece partings of sheep usually reveal the presence of lice or keds. A flea comb will collect lice, nits and keds from the hair of goats, and the material can be placed in a zip-lock bag for examination under a dissecting microscope. *Demodex* are easily squeezed out of distended hair follicles and identified under the microscope by their cigar-shaped bodies. *Chorioptes* are large surface dwellers that can walk off the slide if a little insecticide is not mixed with a drop of oil to immobilize them. *Sarcoptes* are deep in the dermis and often missed even on deep skin scrapings. Identify mite species with a parasitology text. *Sarcoptes* have long unjointed pedicels, *Psoroptes* have long jointed pedicels, and *Chorioptes* have short pedicels. Certain mange infestations are reportable in some states and Canada.

Sucking lice, keds and burrowing mites can be treated with avermectins systemically. Goats require 150 to 200% of the cattle or sheep dose of ivermectin or moxidectin. A second treatment is usually required to kill parasites that hatch out later from eggs on or in the skin. Numerous parasiticides are available to apply topically by dipping, pouring, sprinkling, or dusting to treat lice and keds. Cattle pour-on products containing ivermectin or eprinomectin have been used off-label in small ruminants. Pyrethrin products such as permethrins are generally effective and labeled for small ruminants, but in Australia significant resistance of lice

to both organophosphates and pyrethrins has developed. Dairy animals should be treated with drugs that are approved for lactating cows. Pet animals have been treated with flea products labeled as safe for cats, although products approved for food animals would be preferable under the Animal Medicinal Drug Use Clarification Act. Treatment of sheep and fiber goats is most effective "off shears", as the chemicals will more easily reach the parasite and an elevation in skin temperature is detrimental to many of these parasites. Chorioptic mange can be particularly difficult to treat. Topical eprinomectin or lime sulfur solutions appear to be effective. Some goats are apparently allergic to *Chorioptes* mites, in that signs do not abate until corticosteroids are given. Secondary staphylococcal skin infections may require systemic antibiotics.

Coccidiosis

Coccidiosis is a major disease of young lambs and kids, especially those raised in confinement or grazing at high stocking rates. Numerous *Eimeria* species exist and pathogenicity is variable, but species differentiation requires close attention to microscopic measurements. The coccidia are host-specific. Pathogenic coccidia species that infect goats (*E. arloingi*, *E. ninakohlyakimovae*) do not infect sheep. Important sheep species include *E. crandallis* and *E. ovinoidalis* and do not infect goats. Rabbit and chicken coccidia do not infect small ruminants. The life cycle within the intestinal tract from ingestion to release of oocysts requires two to three weeks. Large numbers of pathogenic coccidia will seriously damage the intestinal mucosa, thereby limiting nutrient absorption and growth. The oocysts sporulate (and become infective) several days after being passed in the feces; moderate ambient temperatures and wet conditions are favorable to sporulation.

Youngsters over three weeks of age with diarrhea have coccidiosis until proven otherwise. Diarrheic feces adhered to the perineum and tail ("mucky butt") is a common presentation in lambs. Diarrhea may be accompanied by illthrift. Only rarely and with massive exposure is the diarrhea bloody. At necropsy there may be raised white foci in the wall of the intestine.

A quantitative fecal examination with identification of coccidial species present is helpful in determining if small ruminants have the disease coccidiosis or just coccidia in their feces. Fecal samples may be negative for oocysts early in the disease, while animals in balance with less pathogenic species may have 100,000 oocysts/gram. All adult small ruminants are expected to be shedding some coccidia.

Animals with clinical coccidiosis can be treated (off-label) with five days of oral sulfonamides (sulfa-

dimethoxine or sulfamethazine) at the cattle dosage or amprolium^a at 11.4 to 22.7 mg/lb (25 to 50 mg/kg) day. No treatments are available to kill the coccidia. Drugs just slow multiplication to allow the animal to develop immunity and decrease environmental contamination to protect others. A rebound of fecal oocyst counts occurs after treatment. It is typical to see higher counts 10 days after than before treatment. Once clinical signs appear, treatments have little effect on weight gain but do limit shedding. Treat all animals in the group, prophylactically.

Prevention programs for coccidiosis are two pronged: coccidiostats and hygiene. Routine medication of starter and creep rations is recommended for both goats and sheep. Two coccidiostats are labeled for goats. These are decoquinate^b at 0.23 mg/lb (0.5 mg/kg) body weight and monensin^c at 20 g/ton feed. Both have zero meat withdrawal. The coccidiostats labeled for sheep are lasalocid^d at 20-30 g/ton feed and decoquinate at 0.23 mg/lb (0.5 mg/kg) body weight. Distribution in loose salt has been effective for protecting lambs and kids by controlling shedding by the adults when minimal grain is fed. The safest drug for this purpose is decoquinate, which has been mixed at the rate of two pounds of 6% premix to 50 pounds of salt. Coccidiostats are not permitted in dairy animals or on organic farms.

Weaning, crowding, poor nutrition (including vitamin E/selenium deficiencies), wet environment, feet and feces in feeders all predispose to coccidiosis. Be sure that dietary energy, protein, copper, selenium and vitamin E are adequate to support immune function. Group youngsters to avoid more than two weeks spread in the age of the group. Do not house or graze younger lambs or kids where earlier born animals have heavily contaminated the environment with oocysts. Steam cleaning and drying will kill coccidial oocysts, but most disinfectants will not. Feeder designs that exclude lambs and kids, waterers that do not leak and lots of clean dry bedding are important control measures within the barn. For grazing animals, frequent moves to clean pasture (unused since the previous grazing season) will avoid the heavy exposures that lead to coccidiosis.

Haemonchus

The blood sucking abomasal strongyle *Haemonchus contortus* severely limits small ruminant production during warm, wet seasons. This parasite has been referred to as the barberpole worm; its gut full of red blood cells is wrapped around a uterus full of white eggs. A heavy infestation in a lamb may remove 20% of the circulating erythrocyte volume per day. The worm has a short generation time and is a heavy egg producer; 5,000-10,000 eggs are produced per worm per day. On heavily contaminated lush pasture, *Haemonchus* can infest and kill

its host in four weeks. Clinical signs include anemia, weakness, respiratory distress and lethargy. *Haemonchus* does not cause diarrhea, but other strongyle species that do may be present simultaneously. Their eggs as seen on fecal examination are indistinguishable.

A control program must be tailored to fit the climate, the pastures and the genetics of the herd. In the northeast, long frigid winters insure that the pastures are relatively worm free in the spring. If sheep and goats are dewormed before turned out to pasture, they will vacuum up the larvae that survived the winter. A repeat deworming three weeks later is analogous to emptying the vacuum cleaner bag. It is also imperative to deworm at parturition, as the phenomenon of periparturient rise greatly increases the shedding of eggs at this time. Other herd treatments will be dependent on weather and stocking rate and indicated by routine monitoring of fecal egg counts, as discussed below.

In the MidAtlantic states and the Southeast, many producers treat their herds monthly around the year. Resistance has developed to the anthelmintics used in these programs. No new drugs are on the horizon, and in some areas the parasites are now resistant to all three classes of anthelmintics: the benzimidazoles, the avermectins and the cholinergic agonists such as levamisole^e and morantel. Also very alarming is the frequent recommendation from owners and over-the-counter drug catalogs that moxidectin^f be used routinely in small ruminants. Parasitologists have considered this drug to be the reserve treatment when all others have failed—and with continued overuse resistance to moxidectin will become the norm; there will be no reserve drug. Organic herds are not permitted to use any anthelmintics, except for ivermectin, and that only when lives are in danger.

Dewormers should be rotated, probably on an annual basis. More frequent rotation merely speeds the development of resistance to multiple drugs. When treating a group, dose for the highest, not the average weight. Because of differences in drug metabolism, goats require 150 to 200% of the sheep drug dose per pound. Goats also have less inherent resistance than sheep, probably because they evolved as browsers, not grazers. The drugs should be given orally for fast kill of parasites and equally fast removal of the anthelmintic from the body. Avermectins should not be given by injection or as pour-ons, as the slow decline in drug concentration will select for establishment of resistant populations.

Delay the development of resistance by decreasing the frequency of exposure to dewormers. Because *Haemonchus* has a short generation time and is a heavy egg producer, animals can die of haemonchosis even with monthly deworming using effective drugs. Thus pasture management is critical to delay reinfestation. Tilling, harvesting hay, grazing with other species such as cattle

or horses, and allowing the pastures to go ungrazed for at least one year will all reduce pasture contamination. Rapid pasture rotation (every three days) followed by harvesting the regrowth for hay is ideal. The cleanest pastures should be reserved for young lambs and kids, which will have less immunity than adult animals. Moving the group to clean pasture after deworming will also delay reinfestation. Unfortunately, this selects for resistant worms. Parasitologists are currently recommending that some animals be left untreated so that they propagate strains of worms that are still susceptible to the available dewormers. Unselected worm populations are termed *refugia*.

It is easy to import resistant parasites onto a farm with the purchase of carrier animals. All incoming small ruminants and camelids (such as guard llamas) should be dewormed before mixing with the group or being turned out on pasture. Ideally, a double dose of two different anthelmintics, from two different classes should be used. Levamisole is too toxic to permit doubling the dose. Fasting the animal overnight before giving a benzimidazole drug increases efficacy, as does treating three days in a row.

Monitor the parasite load and development of anthelmintic resistance with quantitative fecal examinations. A threshold for treatment should be 500 to 1000 eggs per gram. The lower threshold would be appropriate early in the grazing season, when it is important to limit pasture contamination. Do fecal egg counts on animals before deworming and repeat on the same animals 10 days later. If the counts do not decrease by at least 90% (some say 95%), resistance is present. Owners will see a clinical response to treatment of parasitized animals with as little as 30% efficacy, so do not trust “response to therapy” when judging efficacy.

In the future, owners should select for animals that are resistant to (or resilient to) *Haemonchus*. Studies have shown that 85% of the worms are in 15% of the sheep in a flock. After the strategic periparturient and pregrazing treatments have been carried out, packed cell volume should be monitored every few weeks by observing the color of the conjunctiva of the lower eyelid. Anemic animals are dewormed and marked while sheep or goats that remain healthy under the parasite pressure are left untreated (the FAMACHA system^g). An individual that requires several extra salvage treatments should be culled from the flock and the gene pool. Natural selection works the same way and has produced resistant sheep such as the Barbados blackbelly and the wild ruminants in large herds in Africa.

Other Intestinal Strongyles

Other species of importance to small ruminants include *Ostertagia*, *Trichostrongylus* and *Nematodirus*.

They lead to weight loss or poor production, diarrhea and hypoproteinemia which appears clinically as submandibular edema ("bottle jaw"). It is impossible to know which worm species are causing a clinical problem unless larval culture or postmortem examination of the gastrointestinal tract is performed. If an individual animal is heavily infested with parasites while the remainder of the flock appears to be healthy, test for paratuberculosis with the Johne's AGID and histology of the ileocecal junction. Clinical paratuberculosis is accompanied by marked interference with the host's immune response to parasites.

Tapeworms

Tapeworms (*Moniezia*) are commonly found in grazing small ruminants. Oribatid mites on the pasture serve as intermediate hosts. The segments may be noticed on the surface of fecal pellets, or clinging to the anus of pet animals. The tapeworms are most prevalent in young animals, yet do not appear to interfere with health and growth of lambs and kids. An exception has been proposed, that large masses of tapeworms may slow the passage of ingesta and allow the overgrowth of *Clostridium perfringens* in the intestinal lumen, thereby inducing enterotoxemia in unvaccinated animals. If owners are concerned about tapeworms, a benzimidazole such as fenbendazole or albendazole will treat the tapeworms while also addressing strongyles that may actually be causing illthrift.

Flukes

Fasciola hepatica is a common fluke in the Gulf Coast states and northwestern US. Parasitology texts claim that *Fasciola hepatica* does not occur in the Northeast, but the fluke has been seen on New York sheep farms. Aquatic snails are the intermediate host. Adults are less than 30 mm long. Eggs are large (130-150 μm x 63-90 μm) and do not float on routine fecal exam.

Acute fluke disease is caused by migration of massive numbers of immature *Fasciola* flukes through the peritoneal cavity and liver. Signs include abdominal pain, peritonitis and rapid death. **Chronic fluke disease** is caused by adults in the bile ducts. Typical signs are anemia, hypoproteinemia and weight loss. Liver enzymes may be elevated. Gastrointestinal strongyles and paratuberculosis are important differentials and the final diagnosis is often made at necropsy. **Black disease** (infectious necrotic hepatitis) occurs when migration of flukes activates *Clostridium novyi* spores in the liver, and an exotoxin causes coagulative necrosis. Sheep look sick for 10 to 60 minutes, and there is 100% mortality.

Albendazole at 4.5 to 6.8 mg/lb (10 to 15 mg/kg) kills adult flukes, while extralabel clorsulon is effective against adults at 1.6 mg/lb (3.5 mg/kg) (sheep) or 3.2 mg/lb (7 mg/kg) (goats). Double this dose of clorsulon is used to kill eight week old flukes. As much damage can be done before the fluke matures enough to be susceptible to the drugs, it is very important to fence small ruminants out of swampy pastures to avoid exposure to the areas where the intermediate host snails live. The infective stage of the fluke is a long-lived metacercaria that attaches itself to grass.

Fascioloides magna is the large American deer fluke. Adults are 23 to 100 mm long. This fluke is commonly found in whitetail deer in the Adirondacks as well as in Canada, the Great Lakes region and elsewhere in the US. Aimless wandering of one or two flukes in a sheep's liver can be fatal. Infection is not patent in sheep. Albendazole is used at 6.8 mg/lb (15 mg/kg) to treat this fluke.

Dicrocoelium dendriticum has a fascinating life cycle that involves terrestrial snails and ants. It is a very small fluke (6 to 10 mm), and it causes bile duct hypertrophy, but is not severely pathogenic.

Parelaphostrongylus tenuis

The meningeal worm of the white-tail deer sheds larvae in the feces of infected deer. The larvae enter into snails and slugs where they develop into infective third stage larvae. If the mollusc is then consumed by a deer the larvae migrate from the abomasum across the abdominal cavity. They can reach the spinal cord in approximately 10 days. After further migration and development in the cord the parasite reaches the surface of the brain. Eggs pass from the venous sinuses to the lungs, and larvae are coughed up and swallowed to complete the cycle. The prepatent period is about three months.

In abnormal hosts such as the sheep and goat, the migrating larvae damage the spinal cord, often causing posterior weakness and ataxia. As they migrate forward, tetraparesis may occur, while some animals develop focal pruritic lesions which may indicate irritation to dorsal nerve roots. Occasionally the parasite arrives in the brain stem, causing clinical signs similar to those of listeriosis or caprine arthritis-encephalitis virus infection. In the Northeast, the majority of cases are seen in the fall, but *P. tenuis* has been diagnosed in all months of the year.

One popular treatment protocol involves five days of systemic ivermectin along with five days of fenbendazole at 11.4 to 13.6 mg/lb (25 to 30 mg/kg). An anti-inflammatory drug, such as dexamethasone (non-pregnant) or flunixin, is given at the same time to lessen adverse results of wounding or killing a parasite within

the central nervous system. Animals have recovered or died with this protocol - other small ruminants have recovered or died without any treatment. Prevention of cerebrospinal nematodiasis should be based on keeping deer out of the sheep and goat pastures or raising the domestic species in confinement, on hay. Many camelid owners treat monthly with ivermectin, but this practice must eventually lead to ivermectin resistance in abomasal and intestinal strongyles. Deer proof fences can be high or include a sloping outer fence to keep the deer from jumping into the pasture. Avoid grazing the edge of the woods or the water courses where deer drink. Instead, use deer infested fields for hay and graze closer to the buildings, perhaps with a guard dog.

Conclusions

External and internal parasites limit productivity of small ruminants around the world, but control meth-

ods must be tailored for the individual farm, its location and microenvironment, breeds and stocking rate. Environmental management and genetic selection are becoming increasingly more crucial in parasite control as drug resistance develops or producers opt for organic practices.

Footnotes

^a Corid®, Merial, Iselin, New Jersey

^b Deccox®, Alpharma, Fort Lee, New Jersey

^c Rumensin®, Elanco, Indianapolis, Indiana

^d Bovatec®, Alpharma, Fort Lee, New Jersey

^e Prohibit®, AgriLabs, St. Joseph, Missouri

^f Cydectin®, Fort Dodge Animal Health, Fort Dodge, Iowa

^g Discussed online at the Southern Consortium for Small Ruminant Parasite Control at <http://www.scsrpc.com/>