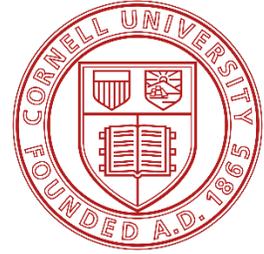


# Deer Worm Factsheet

## Prepared for sheep and goat producers



Courtesy of Cornell Sheep & Goat Program  
and Cornell Ambulatory Veterinary Services

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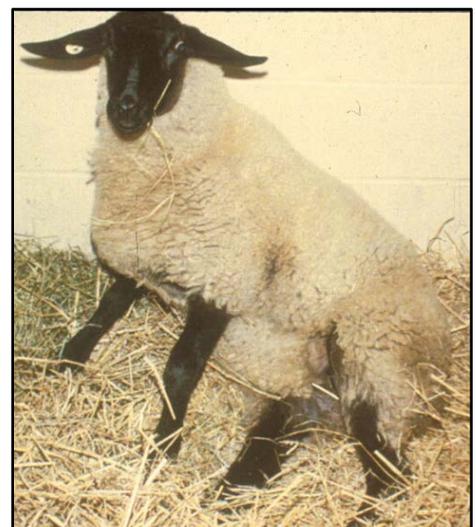
**What is deer worm?** *Parelaphostrongylus tenuis* (abbreviated *P. tenuis*), also called deer worm or meningeal worm, is a parasitic worm of ruminants. It is very common in white-tailed deer in the northeastern United States (up to 90% of deer harvested during hunting season have been shown to be infected), but does not cause disease in this species. The worm has an indirect life cycle, requiring land snails or slugs as intermediate hosts before being able to infect a ruminant.

**What animals are affected by the deer worm?** Although meningeal worm is best suited to white-tailed deer, many other animals that share grazing land with the deer, including sheep and goats, can be infected. In all species, except the white-tailed deer, infection with meningeal worm can cause serious neurologic disease.

**What is the life cycle of *P. tenuis*/How do sheep and goats become infected?** Immature deer worm larvae are shed in the feces of white-tailed deer and are either ingested by or burrow into a passing intermediate host (land snail or slug); over 2 to 3 months, they develop into a more mature, infective larval form. They will either remain in the snail or slug or can be left behind in its mucus trail. Sheep and goats that eat forage contaminated with the snail/slug or its mucus trail can become infected.

Once ingested by the animal, the mature larvae migrate out of the true stomach into the abdomen and follow nerves in the body wall until they reach the spinal cord. In the definitive host (white-tailed deer), the larvae then follow an orderly pattern of maturation in the grey matter of the spinal cord before migrating to the head where they live, reproduce, and lay eggs in the tissues surrounding the brain. However, in small ruminants (because they are not the definitive host) the larvae are unable to 'orient' themselves and thus follow an unpredictable and erratic course around the spinal cord and nearby nerves. This, in conjunction with the body's inflammatory response, causes damage to the central nervous system and leads to the external signs of disease discussed below.

**What are the signs of infection with deer worm?** There are two common signs that an infected sheep or goat may show. Many affected animals will have trouble moving around; early signs of deer worm infection include mild stumbling, knuckling and/or dragging the toes of one or both hind limbs, and general weakness of the hind limbs. In more advanced cases, the sheep/goat may suffer total paralysis of the hind limbs, leaving it in a dog-sitting posture or down and unable



*Figure 1. In more advanced cases, the animal may experience paralysis of the hind limbs*

to get up. The front limbs are less commonly affected, but can have the same problems as the hind limbs. Fortunately, even in a case of total limb paralysis, some animals still recover fully given proper treatment and/or time.

Another common sign of deer worm infection is excessive itching and rubbing of one area on the side of the body. Migrating larvae can irritate an individual nerve where it merges with the spinal cord, making the animal rub and/or bite incessantly at the area of skin to which the nerve runs. This leads to hair loss and occasionally a wound in the skin. Less commonly, infected animals will show signs of brain disease such as a head tilt, walking in circles, rapid eye flickering, and difficulty chewing. Appetite and body temperature typically remain normal in animals affected with the deer worm, which is not often the case with other common diseases of the brain in sheep and goats. It is important to note that not all affected animals will show all these signs. There can be a wide range of manifestations of deer worm infection, so it is important to be vigilant in watching for any problems with your animals.



*Figure 2. Another common sign is constant itching causing hair loss or even a wound often in a streak below the spinal column*

**How do we know that an animal is infected with deer worm?** To make the diagnosis of deer worm, we rely heavily upon the grazing history of the animal as well as the signs of disease it is showing. The fluid that surrounds the brain and spinal cord (cerebrospinal fluid) will also often have characteristic changes to it when viewed microscopically. An absolute diagnosis is made by examining the spinal cord of an affected animal after a necropsy has been performed.

**How do we treat?** There exist many approaches to treatment of *P. tenuis*, largely because no studies have previously been conducted to demonstrate what is most effective. Many have used ivermectin to treat migrating larvae, but this is thought to only be effective when the larvae are traveling in the stomach or abdominal cavity and not once they have reached the spinal cord. Ivermectin should not have the ability to cross the blood-brain barrier into the cerebrospinal fluid and thus may not have any effect on larvae already in the central nervous system (CNS). It may remove additional larvae that have not yet entered the spinal cord. Fenbendazole (Safe-guard<sup>®</sup>, Panacur<sup>®</sup>) is often used because it can penetrate into the CNS, killing larvae already in the spinal cord or brain. To help reduce inflammation and further damage to nervous tissue, steroids such as dexamethasone, or non-steroidal anti-inflammatory drugs such as flunixin meglumine (Banamine<sup>®</sup>), are also commonly utilized in treating infected animals. Some animals recover without any treatment, and not all treated animals inevitably recover. Withdrawal periods (the number of days after treatment that an animal's milk or meat cannot be used) vary widely for different drugs and need to be considered when deciding on a treatment.

**What is Cornell's involvement with deer worm research?** A four year study by the Cornell Sheep & Goat Program and Cornell Ambulatory Veterinary Services investigated the effectiveness of two different treatment protocols for naturally infected sheep and goats. Participating goat and sheep farmers knowledgeable about deer worm contacted the researchers when they observed a suspected case of deer worm infection; the veterinarians then determined if the animal qualified for the study. Enrolled animals were randomly assigned within herd to a treatment or control group with treatments alternating for succeeding animals from the same farm. **The treatment group received 5 days of oral fenbendazole (25 mg/kg), intramuscular dexamethasone (0.2 mg/kg for 3 days followed by 0.1 mg/kg for 2 days) and subcutaneous ivermectin (0.5 mg/kg); the control group received the same 5 day course of fenbendazole and dexamethasone with a similar volume of placebo**

**vehicle in place of ivermectin subcutaneously.** Farmers and study veterinarians were blinded to treatment. Neurologic examinations were conducted at time of enrollment and after the 5 days of treatment to assign animals a neurological score from 1 (unable to stand) to 5 (no detectable neurologic deficits or only an expert would notice) scale, with a score of 4 indicating likely to function in the herd for breeding though noticeably impaired.

Twenty goats and 18 sheep from 10 central NY farms were enrolled in the trial with 9 goats and 9 sheep in the control group and 11 goats and 9 sheep in the treatment group. After the 5-day treatment period, animals were classified as recovered if they required no further treatment to potentially remain in the breeding herd (score of 4). Six of 9 sheep treated with ivermectin recovered without further treatment but 3 had to be euthanized. Five of 9 sheep treated with the placebo recovered without further treatment; 2 required additional treatment, and 2 had to be euthanized. All 11 of the goats treated with the ivermectin recovered, while six of the nine treated with the placebo recovered without further treatment, and 3 required additional treatment.

Statistical analysis indicated that higher pretreatment neurological scores improved outcome ( $P = 0.002$ ). The effect of ivermectin was almost significant ( $P = 0.073$ ). However, all 11 goats treated with ivermectin were categorized as recovered. Therefore, differences between species on the effect of ivermectin could not be tested with the binary logistic regression used for analysis. The better outcomes for goats are probably explained by closer observation of goats (coincidentally all goats were returned to barns at night while all sheep were left in pastures 24/7) which resulted in higher pre-treatment neurological scores. **Given the importance of pretreatment score on the odds of recovery, close observation of animals at high risk for *P. tenuis* infestation is warranted to ensure early treatment.** Studies with larger numbers of animals are needed to definitively state whether including ivermectin in the treatment protocol improves outcome. Inclusion of ivermectin for 5 days increases the drug withdrawal period for the standard protocol to 96 days or more according to FARAD, the Food Animal Residue Avoidance Databank. If there is no concern about the withdrawal period, we cannot rule out the possibility that ivermectin may be beneficial in the treatment of highly valuable animals.

The Cornell Sheep & Goat Program and Dr. Judith Appleton's lab at the Baker Institute for Animal Health also cooperated on a study to determine if sheep can become resistant to *P. tenuis* when established infections in early life are followed by later exposures. In October 2013, 12 ewe lambs in the Cornell sheep flock were each orally dosed with 20 L3 (stage 3 larvae) of *P. tenuis* to induce immunity (Infected). Twelve cohorts of the treated ewes were orally given the suspension medium only (Control). Due to a shortage of L3 to challenge the Control and Infected ewes in October 2014, only 4 of 12 Control ewes and 5 of 12 Infected ewes were challenged (given 100 L3 orally); the unchallenged ewes were given the suspension medium. All trial ewes were kept in a barn for the entire 2-year experiment to prevent natural exposure to *P. tenuis*. Results (Figure 3.) support the potential effectiveness of vaccines developed from killed *P. tenuis* L3. Alternatively, recombinantly-produced proteins found on the surface of *P. tenuis* L3 might be used to make effective vaccines.

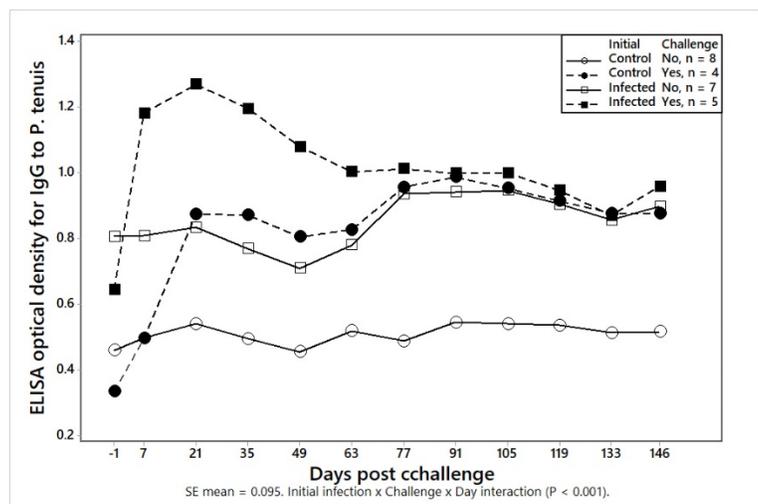


Figure 3. Effect of days post challenge on serum antibody concentrations to *P. tenuis* in ewes initially in a control group or group dosed with 20 L3 at 7 months of age. Roughly half of each group was challenged with 100 L3 at 19 months of age (Day 0).

**Can infection be prevented?** Regular, frequent deworming to prophylactically treat for deer worm is costly and time-demanding; perhaps more importantly, it inadvertently selects for stomach and intestinal worms resistant to the drug, rendering that dewormer ineffective at controlling these damaging gastrointestinal parasites. There is no selection for resistant deer worm larvae because they never complete their life cycle in sheep and goats.

The most effective way of preventing infection from deer worm is to reduce exposure of sheep and goats to infected snails and slugs and to limit deer access to grazing pastures. Pastures bordering woodlands are more likely to be frequented by deer and low, moist areas provide a more conducive environment for deer worm larval development. Fields the first year after forest clearing are especially high risk. Avoiding these areas, especially in wet seasons or after the leaves have fallen, will help reduce exposure and may decrease the chances of deer worm infection. Daily patrolling by guardian dogs during the winter and grazing season and other deterrents such as human activity and/or deer-proof fencing to discourage deer from bedding down in pastures will also reduce the incidence of disease.

***Thank you to the Beakman Fund for Research in Meat Goat Health for funding to prepare this factsheet.***