Pennsylvania Game Commission Wildlife Disease Reference Library

Brain Worm

Other Names: Meningeal worm, *Parelaphostrongylus tenuis*

Cause

Parelaphostrongylus tenuis, commonly known as brain worm, is a nematode or roundworm parasite of white-tailed deer. The long, threadlike worms can be found in spaces and veins surrounding the brain. Adult male worms are greenish-yellow to brown in color and up to 6.2 cm (2.4 in) long and 0.2 mm (0.008 in) wide. Females are darker brown to red to black in color and up to 9 cm (3.5 in) long and 0.25 wide (0.01 in).

Significance

P. tenuis is common in white-tailed deer in eastern North America and it causes only occasional illness in this species. However, white-tailed deer can introduce the parasite to other susceptible species such as moose, elk, and caribou, in which it can cause deadly disease. The brain worm seems to play a significant role in preventing the establishment of mule, elk, and caribou populations in areas with high densities of white-tailed deer. *P. tenuis* may have contributed to the decline of moose in some areas of the United States and Canada, though this has not been proven. White-tailed deer can also introduce the parasite to livestock such as sheep, goats, alpacas, and llamas. Humans cannot become infected with this worm, and properly cooked meat of infected animals is safe to eat.

Photo courtesy of the Michigan Department of Natural Resources

Species Affected

White-tailed deer are the normal definitive host for this brain worm. *P. tenuis* can also infect moose, elk, caribou, mule deer, fallow deer,

The shaded area represents the known distribution of *P. tenuis* in North America. The dotted line is the western edge of white-tailed deer distribution. Image courtesy of Lankester 2001

bighorn sheep, pronghorns, domestic sheep, goats, llamas, camels, guinea pigs, and rarely, domestic cattle.

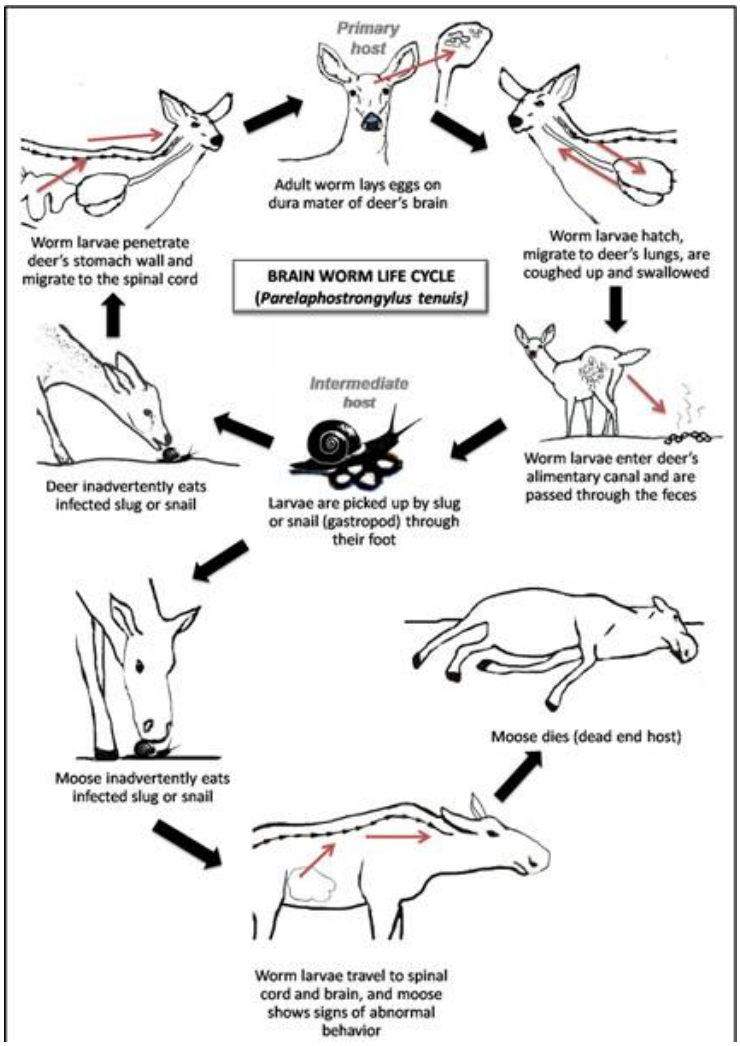
Distribution

P. tenuis is common in white-tailed deer populations throughout eastern North America. It is rare in the southeastern United States, and is not known to occur in western North America.

Transmission

Adult worms inhabit veins and venous sinuses on the surface of the brains of white-tailed deer. Females lay eggs that develop into first stage larvae (L1), which migrate through the bloodstream and become lodged in the small capillaries of the lungs. L1's move from the blood-filled capillaries into the air-space of the lungs. From the lungs, the larvae are coughed up and then swallowed. The L1's move through the digestive tract unchanged and are passed in the feces. The larvae must then penetrate or be eaten by a snail or slug, where they develop into second (L2) then third (L3) stage larvae. L3's infect new white-tailed deer hosts when the slug or snail is accidentally consumed along with vegetation. Once ingested, the L3's penetrate the stomach or intestinal wall and migrate through the body cavity to the spinal cord and to the brain. During this final migration to the brain, the larvae develop into adult worms.

Most white-tailed deer become infected during the first or second summer of life. Some deer are infected with only male or only female worms, so they do not produce larvae. Worms acquired by fawns likely stay with the deer for life, but deer probably do not acquire any more worms after the initial infection. While brain worms can infect many species, larvae are only produced and released in the feces of white-tailed deer and occasionally moose and elk.



Brain worm life cycle; courtesy of dec.ny.gov by Natalie Sacco

Clinical Signs

White-tailed deer typically do not show clinical signs of *P. tenuis* infection. When deer are infected with a large number of worms, temporary lameness, especially in fawns, circling, or other neurological signs may be observed.

When non-definitive hosts such as moose, and elk are infected they may exhibit signs of neurological disease including weakness, loss of coordination, head tilt, apparent blindness, circling, loss of fear, depression, weight loss, and finally paralysis and death.

Diagnosis

Definitive diagnosis can be reached by recovering and identifying adult *P. tenuis* worms or laboratory tests. Finding larvae in the feces is not sufficient for diagnosis because they are identical to muscle worm (*P. andersoni*) larvae.

Treatment

Anthelminthics (de-wormers) may help prevent larvae from developing into adult worms, but studies have not been conducted to prove their usefulness. Such treatment may be attempted for prevention in captive animals, but is probably not feasible for free-ranging wild populations.

Management/Prevention

This disease can be controlled by reducing white-tailed deer populations in regions where they share habitat with susceptible species. White-tailed deer should be prevented from interacting with susceptible captive animals, and snails and slugs should be kept out of enclosures. It is important that wildlife managers work to prevent the spread of *P. tenuis* into western North America by ensuring that infected animals are not introduced to unaffected areas.

Suggested Reading

Lankester, M. W. 2001. Extrapulmonary Lungworms of Cervids. Pages 228-278 *in* W. M. Samuel, M. J. Pybus, and A. A. Kocan, editors. Parasitic Diseases of Wild Mammals. Iowa State University Press, Ames, Iowa, USA.

Michigan Department of Natural Resources. Wildlife Disease. Brainworm. <http://www.michigan.gov/dnr/1,1607,7-153-10370_12150_12220-26502--,00.html>.