



THE NORTHEAST WILDLIFE DISEASE COOPERATIVE

<http://sites.tufts.edu/nwdc>

Rabies

Cause

Rabies is caused by viruses from the *Lyssavirus* genus.

Significance

Rabies is a great public health concern because it can be transmitted to humans by the bite of infected animals and is nearly 100% fatal without post-exposure treatment. In the 1960s, wildlife became the primary source of rabies exposure for domestic animals and humans. Cases of human rabies in the United States have declined with the vaccination of domestic animals. With shrinking wildlife habitat and more opportunities to encounter wildlife, avoiding wild animals that are behaving abnormally is very important.

Species Affected

All mammals can potentially become infected with the rabies virus. In North America, the major wildlife reservoirs for this disease are raccoons, coyotes, skunks, bats, and foxes. Unvaccinated domestic or feral cats and dogs are also at risk of acquiring and transmitting rabies.

Distribution

Rabies is currently found on every continent except Antarctica and was likely introduced to North America from Europe around the 1700s. The United Kingdom and Japan have successfully eradicated rabies. Some other island nations are also free of the disease.

There are multiple strains of the virus associated with specific wildlife species. The raccoon strain is most

common in the eastern United States. Skunk rabies is more common in the central United States and California, while fox rabies is found in Texas, Arizona, and Alaska. Although raccoon rabies is very common in New England, this strain of the virus has only been reported in three cases of human infection. Most human cases in the United States and Canada are caused by bat rabies. Bats are a particular concern because their teeth are so small that people often do not realize that they have been bitten.

Transmission

Rabies is most commonly transmitted through the saliva via the bite of an infected animal. Rabies may also be transmitted by non-bite exposures, which include infected saliva entering an open wound, the eyes or the mouth. Rabies transmission has been reported in rare cases via inhalation of infected particles, and transplantation of infected tissue.



Distribution of major rabies strains in the United States and Puerto Rico in 2008 (Blanton et al. 2010)

The incubation period of rabies is usually 1-3 months, but this can vary greatly depending on the species and the location of the exposure site on the body. The rabies virus travels slowly through the nervous system to the brain. Once in the brain, the virus replicates and spreads to the salivary glands and other tissues. If the exposure site is farther from the brain (i.e. toward the legs or feet), it will take longer for the virus to travel to the brain, thus giving the host a longer incubation period.

Clinical Signs

Once the rabies virus has reached the brain, the infected host will begin to exhibit non-specific neurologic signs. Infected animals may exhibit abnormal behavior such as excitability, self-mutilation, apparent lack of fear, or aggression. Other clinical signs include loss of coordination, paralysis, difficulty breathing, excess salivation, and difficulty swallowing. Rabies will eventually lead to coma and death within 1 to 10 days of the onset of clinical signs. Sometimes infected animals will appear normal. Clinical signs can vary widely and resemble many other neurological conditions, so a diagnosis cannot be reached based on clinical signs alone. Generally speaking, abnormal behavior is the most common sign.

Diagnosis

The only way to confirm a rabies infection is to examine the brain of a suspected infected animal. Rabies suspects must be euthanized and submitted for laboratory testing. The brain must be intact and should be refrigerated (not frozen) before testing. The brain tissue is tested using a fluorescent antibody test (FAT), which detects antibodies against the rabies virus.

Treatment

Post-exposure treatment has a high success rate if administered quickly after exposure to the rabies virus and before the onset of clinical signs. The exposure site should be immediately cleaned with soap and water to reduce the amount of virus particles and post-exposure treatment should be administered as soon as possible.

If treatment is not administered prior to the appearance of clinical signs, there is no generally accepted further treatment for rabies. However, 1-3 individuals are thought to have survived in recent years with induced coma and supportive treatment.

Management/Prevention

There is a vaccine available for use in both people and animals. Prevention through vaccination has been very successful in domestic animals. People with greater risk of exposure such as veterinarians, wildlife biologists, wildlife rehabilitators, and animal handlers should receive pre-exposure vaccinations.

Oral Rabies Vaccine (ORV) programs have been implemented throughout the country to prevent further geographic spread of the virus.

This program aims to vaccinate wildlife against rabies by distributing oral rabies vaccines within fishmeal bait. ORV programs have successfully eliminated fox rabies in several countries in Europe

and in Texas. Since 1997, USDA Wildlife Services has been working with local, state, and federal parties in the eastern US to develop and monitor ORV programs. They established a vaccination zone from Maine to Alabama to prevent the westward and northern spread of raccoon rabies. Every year, millions of baited vaccines are distributed throughout the eastern United States.



Raccoon eating Oral Rabies Vaccine.
Photo courtesy of www.cdc.gov

Suggested Reading

Blanton, J. D., D. Palmer, and C. E. Rupprecht. 2010. Rabies surveillance in the United States during 2009. *Journal of the American Veterinary Medical Association* 237: 646-657.

Bureau of Epidemiology, Division of Infectious Disease Epidemiology. 2010. Pennsylvania annual animal rabies testing report 2010. Pennsylvania Department of Health, Harrisburg, Pennsylvania, USA.

Centers for Disease Control and Prevention (CDC). 2011. Rabies.

www.cdc.gov/rabies/index.html

Jackson, A. C. 2008. Rabies. *Neurologic Clinics* 26: 717-726.

Michigan Department of Natural Resources. Wildlife Disease. Rabies.

www.michigan.gov/dnr/0,1607,7-153-10370_12150_12220-27259--,00.html

Rupprecht, C. E., K. Stohr, and C. Meredith. 2001. Rabies. Pages 3-36 in E. S. Williams and I. K. Barker, editors. *Infectious diseases of wild mammals*. Iowa State University Press, Ames, Iowa, USA.

Slate, D., T. P. Algeo, K. M. Nelson, R. B. Chipman, D. Donovan, J. D. Blanton, M. Niezgod, C. E. Rupprecht. 2009. Oral rabies vaccination in North America: opportunities, complexities, and challenges. *PLoS Neglected Tropical Diseases* 3: 1-9.

Wildlife Services. 2007. Cooperative Rabies Management Program National Report 2007. United States Department of Agriculture Animal and Plant Health Inspection Service.

Williams-Whitmer, L. M., and M. C. Brittingham. 1996. Rabies. *Wildlife Damage Control* 1. The Pennsylvania State University, University Park, Pennsylvania, USA.