



Extension FactSheet

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West Nile Virus Fact Sheet for Veterinarians

West Nile Virus (WNV) had never been seen in the Western Hemisphere prior to 1999. It has a geographic range greater than any other known arbovirus. It is found throughout Africa and north to central Europe and eastern Asia. The earliest epidemic of WN fever occurred in Israel in 1951 and 1952, and involved more than 500 hospitalized patients from several small outbreaks. The largest epidemic of WN fever on record occurred in South Africa during 1974. This epidemic involved an area of about 2,500 km² in the Karoo and southern Cape provinces and resulted in thousands of human infections. More recently, epidemics of WN fever have occurred in Romania (1996–97), Czechland (1997), Italy (1998), Russia (1999), and Israel (2000) so that it is considered a reemerging mosquito-borne disease. How WNV managed to be introduced into the United States is unknown, nor is it known how long it has been here. There is speculation that it was through the importation of birds, or possibly a mosquito transported to the United States by ship or airplane.

West Nile Virus is a member of the Japanese encephalitis virus complex of the genus *Flavivirus*, family *Flaviviridae*. This genus includes nine viruses distributed around the world. In the United States, the complex has two other representatives: Powassan and St. Louis encephalitis (SLE) viruses. Both cause encephalitis in humans. The epidemiol-

ogy of WNV is nearly identical to that of SLE virus. Both are principally carried by species of *Culex* mosquitoes and have birds as the reservoir. They differ in that WNV causes disease and mortality in humans, wildlife (birds, particularly crows and blue jays), and domestic animals, particularly horses. St. Louis encephalitis does not cause any remarkable disease in wildlife or mammals other than man.

Epidemiology

Early outbreaks of WNV in horses occurred in 1962 in France, and 1963 in Egypt. Much later, outbreaks occurred in 1996 in Morocco, 1998 in Italy, 2000 in France and Israel and in 1999 to 2005 in the United States. In France, they recorded a 10% morbidity with a 30% mortality rate. In Morocco, the case fatality rate was 44.7% (42/94) and in Italy it was 42% (6/14). In the United States in 1999, nine of 25 (36%) horses with clinical signs of the disease died or were euthanized. In 2000, there were 60 cases of WNV reported in horses from seven states and 23 horses either died or were euthanized (38%). During 2001, there were 738 confirmed horses that developed clinical signs of WNV infection in 19 states with ~25% fatalities. In 2002, there were >14,000 confirmed equine cases of WNV from 40 states; however, no mortality data is available. In 2003, there were 5,087 confirmed cases of WNV reported from

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40 states. In 2004, there were 1,341 confirmed cases in 36 states. In 2005, there were 1,143 confirmed cases of WNV in the United States, primarily in the western states. WNV is not on the list of reportable diseases; therefore, the number of reported is probably significantly lower than the actual number of infected horses.

West Nile Virus continues to be a serious threat to humans. The following table presents the number of human cases and fatalities in both the United States and Ohio.

Year	United States		Ohio	
	Cases	Deaths	Cases	Deaths
1999	62	7	0	0
2000	21	2	0	0
2001	66	9	0	0
2002	4,156	284	441	31
2003	9,862	264	108	8
2004	2,539	100	12	2
2005	3,000	119	61	2
2006	4,266	177	48	4
2007	3,623	124	23	3

Clinical Signs

In the Italian outbreak, all cases exhibited varying degrees of ataxia and weakness in the hind limbs. Asymmetric weakness was detected in the rear limbs of some horses. Some cases also had involvement of one or both forelimbs. In six cases, there was progression of clinical signs with ascending paresis leading to tetraplegia and recumbency within nine days. Depressed mental state and tremors were noted in a few cases; however, there were no behavioral or head posture abnormalities or cranial nerve involvement. In the U.S. cases, similar clinical signs were seen; however, more brainstem signs were present in some cases. For the horses that died or were euthanized, death usually occurred within five days.

Pathology

No gross pathologic lesions were detected. Histologically, all animals exhibited slight to moderate nonsuppurative encephalomyelitis, primarily in the spinal cord and lower brainstem affecting both grey and white matter. The most severe lesions were in the thoracic and lumbar spinal cord.

Differential Diagnosis

Rabies, botulism, EPM, CVM, EHV1, EDM and other encephalitis such as WEE, EEE and VEE, would all have

to be considered. Many of the cases have looked similar to commonly diagnosed neurologic diseases, particularly EPM. Considering the ascending paralysis, mentation changes and hyperesthesia in some cases, all horses that die or are euthanized should be sent for rabies diagnostics. Diagnostic rule-outs are necessary. See The Ohio State University web site given later in this fact sheet for aids in differential diagnosis.

Diagnosis

Due to the zoonotic potential of WNV, all horses that develop neurologic signs from June to October should be considered WNV suspects, particularly if the virus has been detected in Ohio close to the animal you are examining. **Whole blood, serum, and CSF (if collected) samples should be submitted to ODA/ADDL. Complete histories should be submitted with all samples (form available on the OSU web site).** If the animal is demonstrating rapidly progressive neurologic signs with recumbency, the animal should be submitted to the Ohio Department of Agriculture/Animal Disease Diagnostic Laboratory in Reynoldsburg, Ohio, for rabies and WNV testing. Other diagnostics include PCR of CNS tissues and immunohistochemistry. If you decide to perform a postmortem in the field, please refer to USDA guidelines. (See the OSU web site.)

Treatment

In the Italian outbreak, no treatments were effective. Similar findings occurred in the U.S. cases, as well. Regardless of the treatment, of the horses that survive at 6 month follow-up ~40% were still exhibiting neurologic deficits. Supportive care is the only therapeutic alternative.

Prevention

There are four vaccines available for prevention of WNV infection in horses. There is a killed vaccine that must be given in two doses initially, three to six weeks apart. Both doses should be completed at least four weeks prior to mosquito season. If the horse has already had the initial two doses, the first yearly booster should be given in early April. Horses that are stressed, such as show and race horses, should be given another booster in late July. Efficacy data from Ohio would suggest that <6% of horses that are properly vaccinated may die from WNV infection. The vaccine is considered very safe. There is also a new canarypox vectored vaccine that must be administered in two doses initially followed by annual boosters. Limited efficacy data is present at this time. In addition, there is a new DNA vaccine that has been released which has

demonstrated 100% efficacy in preventing clinical signs in foals. There is also a WNV chimera vaccine that was 95% efficacious in preventing clinical signs using a challenge model.

Reduce mosquito breeding sites; decrease exposure to adult mosquitoes; provide screened housing; use insect repellents; and reduce outdoor exposure. Local mosquito control authorities may be able to help in assessing the mosquito breeding risks associated with a specific property.

Contact Veterinarian

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What is the status of WNV in Ohio?

WNV has been confirmed in Ohio every year since 2001. Infected mosquitoes, birds, horses, and humans have been found in all Ohio counties. Therefore, the virus is present throughout the state. Contact your local health department in your area, or visit one of the web sites.

For the current status on WNV in Ohio and for more information, you can log on to the following web sites:

The Ohio State University:
<http://vet.osu.edu/1516.htm>

Ohio Department of Health:
<http://www.odh.ohio.gov/odhprograms/idc/zoodis/wnv/wnv1.aspx>

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