

Culex mosquito laying eggs.

Photo: Centers for Disease Control

West Nile Virus continues to gain ground in United States

Horses, wild birds serve as biggest hosts for virus transmission from mosquitoes

By Michael Brashier, DVM, MS, Dipl. ACVIM
CONTRIBUTING AUTHOR

While new to us in the United States, West Nile Virus (WNV) was originally isolated in the 1930s in the West Nile district of Uganda making it one of the first arthropod spread viruses described. Historically this virus has caused occasional illness in humans and horses in Africa, the Middle East, and Asia with sporadic outbreaks in Europe. In endemic areas of Africa, up to 70 percent of the human population are seropositive with relatively few having shown clin-

ical signs, suggesting that the majority of infections are mild or subclinical. Veterinary surveillance in these areas is often lacking, so similar data for horses is unavailable, but evidence suggests that many infections in horses remain subclinical. However, WNV should still be considered an important pathogen in the horse.

The exact method of introduction into the United States is unknown. Speculation ranges from accidental transportation of mosquitoes or their larvae on ships, importation of infected birds, infected migratory birds blown off course by a storm, movement of an infected person, and even the importation or accidental transportation of infected frogs. However it arrived, suitable

vectors and hosts were available for the virus to propagate and spread. Various wild birds serve as the amplifying host and mosquitoes serve as the principal vector similar to some of the other arboviruses that are endemic to this area such as Eastern Equine Encephalitis virus.

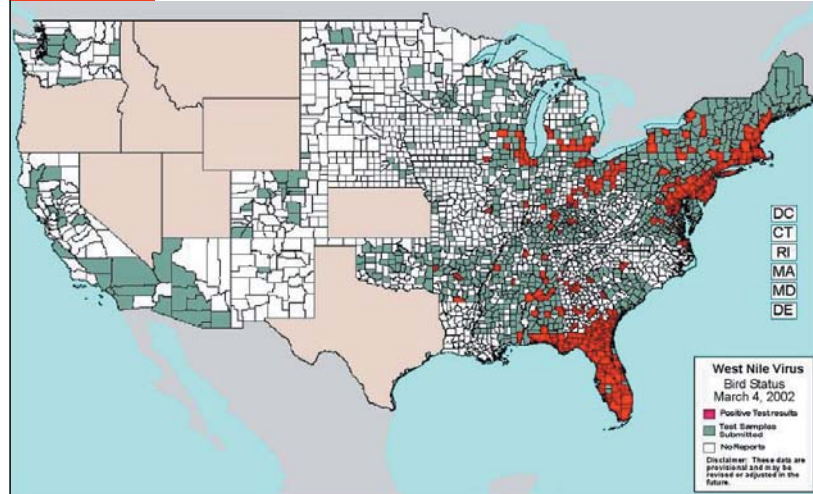
■ WNV hits the U.S.

Crows and other birds carrying antibodies to WNV are common in endemic areas, yet epizootics of clinical disease in wild, free ranging birds are apparently undocumented, suggesting that asymptomatic or mild infection is the norm in birds of those regions. However, the first evidence of WNV in the United States occurred in August of 1999 with crows dying by the hundreds in Long Island and the immediately bordering areas of New York, New Jersey and Connecticut. Clinical signs in these birds included ataxia, abnormal head position, circling and convulsions. Necropsy of these birds revealed meningoencephalitis, hemorrhage within the brain, myocarditis, splenomegaly and involvement of other organs. It may be that a mutation occurred allowing WNV to become more pathogenic for birds, or simply that it was able to cause more obvious disease because of the availability of a great number of naive hosts. In addition to the wild birds affected, during September 1999, 24 birds housed at the Bronx Zoo and the Queens Wildlife Center contracted the disease and died.

These included five flamingos, several ducks, two raptors, and three Asian pheasants. During the late summer and fall of 1999, there were 62 reported human cases with seven fatalities. There were also 25 ill horses with nine fatalities on Long Island during the same time frame. Thirty-eight equine cohorts from the same premises seroconverted without developing clinical disease.

From this initial introduction into the United States, WNV has spread rapidly. During 2000, WNV was isolated from birds across 12 states, several hundred mosquito pools, and was reported to have caused 21 human cases with two deaths and 60 equine cases with 23 deaths across seven states in the northeast. By late fall of 2001, WNV had been identified in 80 bird species and eight mammalian species from 27 states covering most of the eastern half of the United States and resulting in thousands of clinical cases (see Table 1). Wild birds of the order Passeriformes have accounted for the majority of clinical cases with corvids (jays and crows) accounting for the majority of cases in this order. During 2001, there were 738 reported equine cases with 651

dvm Table 1: West Nile Virus Bird Status—March 2002



Sources: USGS, CDC & CINDI

verified through diagnostic testing at the National Veterinary Services Laboratory (NVSL) of the Animal and Plant Health Inspection Service (APHIS) from 20 states (see Table 2, p. 47.) Of the 470 horses for which an outcome had been reported, 156 (33.2 percent) died or were euthanized. Florida had the greatest number of cases with nearly 500 cases and 82 deaths. It is unknown how many horses

may have seroconverted without demonstrating clinical illness. USDA-APHIS used a case definition of compatible clinical signs, as described in this article, and one of the following tests: viral isolation, a four-fold or greater increase in plaque reduction neutralization test (PRNT) from paired sera, or positive IgM and PRNT in a single serum sample.

Continued on next page ►

Update on Avian West Nile Virus

As the West Nile Virus continues to spread across the United States, veterinarians and researchers are finding it is affecting both wild and pet bird populations. In 2001, a total of 4,604 crows and 1497 other birds with WNV infection were reported from 27 states and the District of Columbia.

“We’re learning that West Nile Virus is here to stay,” says Dr. Branson Ritchie, professor of avian and exotic medicine at the University of Georgia College of Veterinary Medicine. “It has caused a number of casualties in bird populations but I think we’ll see it become more a seasonal problem. Bird owners are going to have to take precautionary measures to protect their birds.”

Dr. Susan Clubb, dipl. ABVP (Avian), a Florida practitioner is monitoring the progression of WNV in psittacine birds and whether vaccination is effective.

Very preliminary observations suggest that West Nile Virus is a health risk for psittacine birds but the morbid-

ity and mortality rates appear to be very low. Additionally, the case mortality rate appears to be low and older birds seem to be more severely affected, she says.

Establishing a definitive diagnosis for West Nile Disease has been challenging for veterinarians. Virus isolation has been unrewarding which might indicate transient infections occur in psittacines. Several cases exhibiting acute onset of clinical signs were seropositive indication possibly rapid seroconversion, she reports in the preliminary findings of her study. It also appears by the findings of this study that psittacines may in fact react similarly to chickens as described in a study released in 2000.

Clubb says initial impressions of vaccine efficacy are promising. No adverse reactions were observed upon vaccination. The majority of the birds vaccinated seroconverted. However, she is still analyzing the data and further analysis will be reported later this year.

■ Potential vectors?

Of the other mammalian species, including several species of rodents, ruminants, and dogs, that have been documented to contract WNV, almost all develop only a short-lived, low level viremia resulting in seroconversion without the development of clinical signs. They are not thought to be important in maintenance of the virus in the environment. Except for experimentally infected sheep, in which fever, abortion and occasional encephalitis have been reported, clinical disease appears to be limited to primates, horses and some species of birds.

■ Equine WNV

West Nile Virus appears to have a pronounced CNS tropism in the horse. The amount of viral antigen is scant and the lack of WNV in extraneural tissues in



Flaviviruses share a common size (40-60nm), symmetry (enveloped, icosahedral nucleocapsid), nucleic acid (positive-sense, single stranded RNA approximately 10,000-11,000 bases), and appearance in the electron microscope.

horses is distinctly different from the widespread production of antigen seen in infected birds. Because of this difference,

horses appear to be a dead-end host.

After an incubation period of one to two weeks, clinical disease in the horse presents as ataxia (85 percent) characterized by a base-wide stance, hypermetria, stumbling, circling and leaning against a supporting structure; depression or apprehension (50 percent); hindlimb weakness or paresis in nearly 50 percent with progression to tetraparesis and/or re-

cumbency in a significant proportion of these; and muscle fasciculations (40 percent) involving the face, trunk or limbs. Some cases will progress to convulsions and paddling. The febrile episode is frequently missed and has been documented as little as 25 percent of the time. Case mortality rate seems to be between 25 and 40 percent. Recovery may be as short as two to three weeks, but continued improvement has been documented over several months in more severely affected animals. Major differential rule-outs include: Equine Herpes Virus 1 induced myeloencephalitis, Eastern or Western Equine Encephalitis virus, Equine Protozoal Myeloencephalitis, and otitis media/interna.

Lacking a specific anti-viral pharmacotherapeutic agent, treatment is supportive. Non-steroidal anti-inflammatory drugs are indicated. In severe cases, a dose of a short-acting steroid such as prednisolone sodium succinate may be warranted in initial therapy to help reduce inflammation and restore function to neurons that have been disabled due to inflammation. Prolonged use should be avoided to minimize the impact on lymphocyte function. Intravenous dimethyl sulfoxide may prove useful also in decreasing CNS inflammation and edema. The horse should be deeply bedded and in as safe an area as possible (padded stall would be ideal) to prevent injury should it stumble and fall when trying to ambulate or rise. Slings, fluid and nutrient support may be beneficial in severe cases.

Frequently no gross lesions are detected in affected horses. Gross lesions that have been reported are CNS hemorrhage and meningeal congestion. Histopathologic lesions are non-specific and include perivascular cuffing, multifocal gliosis, and hemorrhage. The location of the most severe histologic lesions has varied in different reports. In an outbreak in Italy where the predominant presenting complaint was spinal ataxia with infrequent encephalitic signs, the most severely affected region of the CNS was the ventral horns of the spinal cord with relatively fewer and milder lesions

noted in the brain. In an early report from the North American outbreak, encephalitic signs appeared to be more common than that in the Italian outbreak and histologic lesions were reported primarily in the brain.

Even though the horse is environmentally considered a dead-end host, due to the zoonotic potential of WNV, veterinarians should exercise caution when sampling CNS tissue for necropsy. USDA-APHIS recommends the following:

1. Keep the use of needles and sharp instruments to a minimum.

2. Do NOT use mechanical saws to obtain spinal cord samples.

3. Procedures that create an aerosol should be done in a way to minimize the dispersal of the aerosol particles.

4. Wear Tyvek® disposable coveralls or, at a minimum, a solid-front, water-resistant, long-sleeve gown.

5. Wear three pairs of gloves. The innermost pair should be latex or other disposable gloves. Substantial waterproof gloves (e.g., Playtex® kitchen gloves) should be worn over the innermost pair. The gloves should be long enough for the gown sleeves to be tucked inside the gloves; duct tape may be useful for keeping sleeves inside gloves. The outermost pair of gloves should be metal or Kevlar®, e.g., a Whizard® Hand Guard (steel/Kevlar®) glove from Koch® (800-456-5624) or a locally purchased filleting glove. **THIS OUTER PAIR OF GLOVES MUST BE WORN** throughout the necropsy procedure.

6. Wear a face shield or goggles to protect mucous membranes, and wear a disposable "half mask" HEPA respirator (3M® 8293) to avoid aerosol infection.

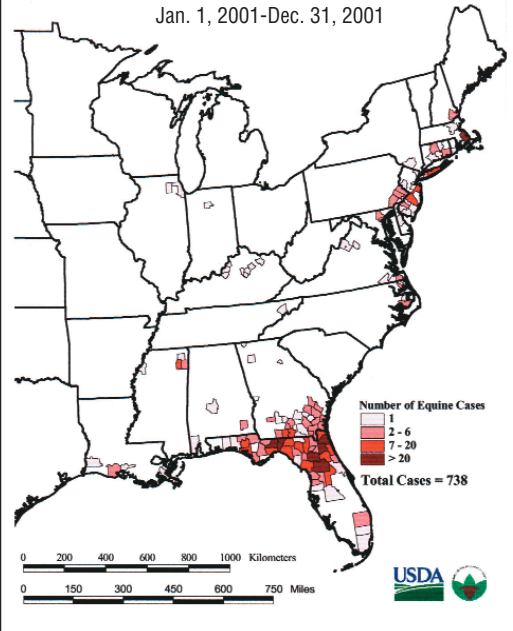
■ Ounce of prevention

A conditionally licensed vaccine for the prevention of WNV is available. A conditionally licensed vaccine has to have met standards for safety and purity, and have a reasonable expectation for efficacy with

Photo: Centers for Disease Control

Table 2: Summary of Equine Cases of clinical West Nile Virus Infection

Jan. 1, 2001-Dec. 31, 2001



not the WNV vaccine will need to be given more frequently than once a year. Other preventive measures involve controlling exposure to the mosquito vector. Limit turnout during the dawn and dusk periods when mosquitoes are most active. The use of fans in stalls can discourage mosquito activity as they are not as strong a flier as stable flies. The use of screened enclosures and the use of insect repellants can also limit mosquito-feeding activity. Elimination of mosquito larval habitats will reduce mosquito numbers. Even small pools of stagnant water such as found in discarded tires provide an adequate habitat.

For larger pools and ponds, the addition of larval predatory fish will help reduce mosquito numbers.

After a review of the literature, it is still unclear at what risk pet birds may be at for contracting and developing a clinical case of WNV. It is apparent that members of the order Passeriformes are able to become infected, but except for corvids, few seem to develop disease. There are occasional reports of disease in raptors, but they are few. This author was unable to lo-

cate literature sources documenting susceptibility or lack thereof for members of the psittacine family. It would seem prudent though to generally limit outdoor exposure. Domestic fowl, turkeys and chickens, while susceptible to infection and seroconversion appear to rarely, if ever, develop clinical disease. Other pets such as cats and dogs are also reported to be clinically unaffected.

Since WNV has proven vectors and hosts available in the United States and has now survived several winters, it appears that it is here to stay. As the population of naïve hosts dwindles, the incidence of the disease in both horses and birds will hopefully diminish as well. Still, it should be considered in the differential diagnosis list for horses developing neurologic signs, and appropriate measures to protect horses from the disease should be instituted. **DVM**

ongoing efforts to verify efficacy. Label recommendations state that two injections should be administered intramuscularly three to six weeks apart initially. Experience with other vaccines against equine viral encephalitic infections indicates that annual boosters may not be adequate. This is especially true of EEE vaccines for horses residing along the Gulf Coast. Only testing and experience will dictate whether or



Dr. Brashier is an associate professor and chief of equine medicine and surgical service at Mississippi State University College of Veterinary Medicine. He received his veterinary degree from the Louisiana State University College of Veterinary Medicine in 1986. Dr. Brashier then completed a residency program in large animal internal medicine and a master's program in 1993 at the University of Minnesota College of Veterinary Medicine. He is a diplomate of the American College of Veterinary Internal Medicine. He joined the faculty at Mississippi State in 1994.

Suggested reading

- Cantile C, Di Guardo G, Eleni C, et al. Clinical and neuropathological features of West Nile Virus equine encephalomyelitis in Italy. *Equine Veterinary Journal* 2000;32:31-35.
- Cantile C, Del Piero F, Di Guardo G, et al. Pathologic and immunohistochemical findings in naturally occurring West Nile Virus infection in horses. *Veterinary Pathology* 2001;38:414-421.
- Ritchie BW. West Nile Virus—a recent immigrant to the United States. *The Compendium of Continuing Education for the Practicing Veterinarian* 2000;22:576-586.
- Senne DA, Pedersen JC, Jutto DL, et al. Pathogenicity of West Nile Virus in Chickens. *Avian Diseases* 2000;44:642-649.
- Snook CS, Hyman SS, Piero FD, et al. West Nile Virus encephalomyelitis in eight horses. *Journal of the American Veterinary Medical Association* 2001;218:1576-1579.
- Steele KE, Linn MJ, Schoepp RJ, et al. Pathology of fatal West Nile Virus infections in native and exotic birds during the 1999 outbreak in New York City, New York. *Veterinary Pathology* 2000;37:208-224.
- Swayne DE, Beck JR, Zaki S. Pathogenicity of West Nile Virus for Turkeys. *Avian Diseases* 2000;44:932-937.
- USDA-APHIS maintains a WNV web page. This may be accessed at www.aphis.usda.gov and clicking on the West Nile Virus link under the Hot Issues topic list.