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## The West Nile Virus Epidemic in North America: 1999-2002

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The West Nile virus is a newly emerging viral disease in Canada. It was first diagnosed in North America in New York City in 1999. The West Nile virus (WNV) belongs to the family *Flaviviridae* and is an arbovirus transmitted between vertebrates by mosquitoes. Wild birds are considered the principal vertebrate host and mammals the dead-end host. The virus can infect a wide variety of mammals (dogs, camels, ruminants, rabbits, pigs, and bats), however, important clinical infections have been detected mainly in humans, horses, and birds. In horses and humans, infection can be subclinical or cause a wide range of signs. Diagnosis is mainly by IgM capture ELISA on serum collected close to the acute phase of the disease. There is no validated protocol for treatment; supportive therapy is usually given. Prevention is mainly by mosquito control and, in horses, by the use of vaccination. This issue of *Large Animal Veterinary Rounds* discusses the spread of the WNV epidemic and its effect on various animal species; particular emphasis is given to the diagnosis, treatment, and prevention of the disease in horses.

### An overview of WNV

WNV was first detected in North America in August of 1999. In the subsequent 3 years, it spread rapidly across the continent, killing many thousands of wild birds and causing serious disease in people and horses. In 2002 alone, WNV caused illness in at least 3,900 people in the United States and Canada, with over 200 fatalities. Over 14,700 American and Canadian horses became ill and many died, while as many as several hundred thousand wild birds, especially crows, were killed by WNV infection. Significant illness was reported in a wide range of other species, including 143 free-ranging North American bird, 8 wild, and 9 domestic mammalian species. By December 2002, the geographic range of the virus had increased from an initial small area around New York City in 1999 to include 44 states and 5 provinces. This WNV outbreak is a case study in what may well become regular events around the world. As human populations and human activities increase, there will be unintentional (or intentional) transportation of pathogens from one geographic area to another with subsequent inductions of epidemic disease.

### West Nile Virus

WNV is a virus of the Old World. Its name comes from the West Nile Province of Uganda, where the virus was first described in 1937.<sup>1-3</sup> WNV belongs to a closely-related group of viruses often referred to as the Japanese encephalitis serocomplex within the virus family *Flaviviridae*. The geographic range of WNV in the Old World is very wide: from southern Africa to north-central Europe and from the Atlantic coast to India and western China.

WNV is one of several viruses known as arthropod-borne viruses, or arboviruses, because all are transmitted among vertebrate hosts by arthropod vectors such as mosquitoes and ticks. Wild birds appear to be the principal vertebrate hosts for WNV, and the normal life history of the virus consists of cycles of transmission among birds and bird-feeding species of mosquito. Within its native range, WNV is not recognized as an important cause of illness or mortality among wild birds, but many species can be infected and carry enough virus in their blood to infect the mosquitoes that feed on them. In contrast, infected mammals generally do not carry much virus in their blood and thus do not act as effective sources of infection for mosquitoes. Thus, mammals are considered "dead-end" hosts for WNV, ie, hosts that may



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become infected and even diseased, but do not contribute to the maintenance of the virus in the environment or to the spread of the virus to other animals.

In temperate climates, viruses such as WNV typically follow a cycle of reproduction, amplification, and spread within bird populations from spring to late summer. Each spring, the virus may become active in an area either through arrival of infected migratory birds or emergence of dormant adult mosquitoes that were infected in the previous summer or fall. Through cycles of transmission between birds and bird-feeding mosquitoes, the number of infected wild birds and mosquitoes in the environment gradually builds to a maximum in late summer. At this same time, species of mosquito that feed on both mammals and birds also reach their maximum activity. Thus, the probability that mammals will become infected with WNV derived from the wild bird reservoir of virus is highest toward the end of summer and in early fall. The time of greatest risk of infection for people and horses begins in this mid-to late-summer period and ends following the reduction of mosquito activity in the fall.

### **One virus among many**

Although the arrival of WNV in North America has legitimately claimed considerable attention from public and veterinary health authorities as well as the media, it is not the unique or unprecedented pathogen that some media coverage may imply. Rather, it is an addition to a long list of arboviruses native to North America, several of which cause spectrums of infection and disease similar to WNV.<sup>6-8</sup> Some of these North American arboviruses live normally among wild birds and mosquitoes and can cause serious disease in people and horses. They include: eastern equine encephalitis (EEE), western equine encephalitis (WEE), St. Louis encephalitis, and Venezuelan equine encephalitis (VEE). Thus, North Americans have always been exposed to pathogens similar to WNV. This does not imply that WNV should be ignored. It is a new viral disease and its range and importance in North America, in the long term, remain unknown. WNV requires careful scientific attention so that its effects and associated risks can be properly defined and procedures can be designed to minimize its impact.

### **The North American WNV epidemic**

#### **1999**

WNV was first recognized in North America in August 1999. The first manifestation was unusual mortality in wild American crows around a large zoo in New York City, followed closely by mortality of birds in the zoo's own collection.<sup>4</sup> Soon after, New York City hospitals admitted several patients with symptoms of viral encephalitis.<sup>5</sup> The diagnosis of WNV infection was made first in the dead birds; human cases initially were attributed to the St. Louis encephalitis virus, a closely-related virus, but were reassessed once WNV had been identified in birds. In total, 62 people developed encephalitis due to WNV; 7 of these infections were fatal. At least 25 fatal cases of encephalitis in horses were recognized. Mortality of crows and other wild birds was enormous, but unquantified. Nearly all recognized infections in all species were confined to a radius of about 100 km from the centre of New York City, with a few

infected wild birds recognized further south, in southern New Jersey and Baltimore.

#### **2000**

Federal, provincial, and state public health authorities in eastern North America mounted intensive surveillance programs for WNV beginning in the late winter and spring of 2000. Testing for WNV in wild birds found dead proved to be the most effective way to detect virus activity in a geographic area. Although WNV is not known to cause significant mortality in wild birds in the Old World, many North American species, particularly members of the crow family (crows, ravens, magpies, jays) are highly susceptible and mortality rates after infection are very high. Surveillance in mosquitoes, sentinel chickens, horses, and humans was markedly less effective in detecting virus activity than surveillance in wild birds.

WNV expanded its range centrifugally in 2000, extending for a radius of about 400 km around the 1999 epicentre in New York City. A small number of infected birds were detected south of the contiguous affected area, in counties of central Virginia and North Carolina. In Canada, surveillance was instituted from Newfoundland to Saskatchewan, but no infected birds were detected. In the United States, 19 cases of encephalitis in people were recognized, 2 of which were fatal and all were close to the epicentre. At least 65 cases of encephalitis were recognized in horses spread across 7 states.

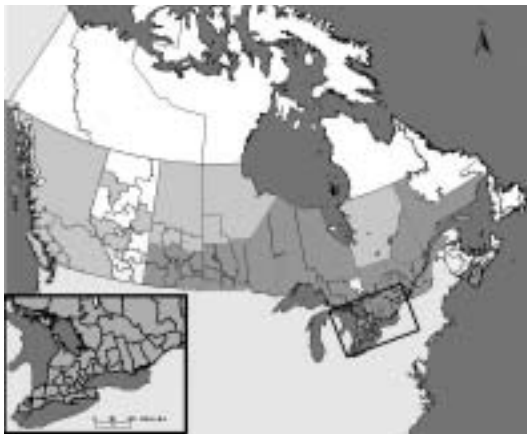
#### **2001**

The geographic range of WNV expanded greatly in 2001. The southern United States became a major area of activity, including nearly all of Florida and adjacent portions of Georgia and Alabama. The virus also spread north to southern Ontario, Michigan, Illinois, and Wisconsin, and west to just beyond the Mississippi River. The mechanism that extended the range of the virus is not known, but most likely it was a combination of the lateral movements of infected resident birds and mosquitoes, as well as the northward and westward movements of migratory birds that became infected in new areas of virus activity in the southern United States. Surveillance efforts in 2001 were larger than in 2000 due to a larger number of states participating and the ability of polymerase chain reaction (PCR) technology to test large numbers of specimens. Again, detection of virus in dead wild birds, particularly members of the crow family, proved to be the most effective means of virus surveillance. The geographic pattern of occurrence of the virus in wild birds was mirrored by the pattern of detection of infections in horses and humans some weeks or months later.

#### **2002**

The range of WNV increased again in 2002. The virus was detected from Nova Scotia to Florida, westward across the continent continuously to Saskatchewan and New Mexico and, discontinuously, to northwestern Washington State and southern California (Figures 1 and 2). Again, massive wild bird mortality formed the basis of virus surveillance and the distribution of virus detected in birds was mirrored by cases in horses and people.

**Figure 1: Geographic distribution of West Nile virus activity in Canada during 2002.**



**LEGEND**

Birds submitted for diagnosis (by Health Region)

□	□	■
No	Yes	Positive

Source: Canadian Cooperative Wildlife Health Centre and Health Canada, <http://wildlife.usask.ca/english/frameWestNile.htm>

### Surveillance

Once WNV had been recognized in the New York City area, programs to rapidly detect virus activity were put in place in areas adjacent to the epicentre beginning in the fall of 1999. Because wild birds were the focus of surveillance, new partnerships were formed among public health, veterinary, and wildlife sectors to secure specimens, dissect out samples, and test tissues for virus. Laboratory capacity initially limited the scale of surveillance since only a few specialist centres had the materials, personnel, and procedures required to test for WNV. Immunohistochemistry (IHC) applied to fixed tissues was the first detection technology made widely available and it was used extensively in 2000. This is a slow and labour-intensive technique and only a portion of the dead birds collected could be tested by this means. PCR tests became available in 2000 and increasingly, during that year, they were applied in test centres. After 2000, PCR tests, verified selectively with virus isolation, became the basis for surveillance in wild birds and in other species. PCR has permitted testing of far more specimens than was possible with IHC. The sensitivity of surveillance was thereby improved considerably. For example, in Canada, only 185 birds of the 2288 collected in 2000 were actually tested for infection with the IHC technology available at the time; 3900 and 3600 birds were tested by PCR in 2001 and 2002, respectively.

In 2000, wild birds of many different species were collected for surveillance. However, it quickly became evident that, overwhelmingly, members of the crow family and particularly, the American crow and the blue jay, were especially susceptible to acute, lethal infection and their tissues contained large quantities of virus. Thus, crow family members, and in particular crows themselves, became the main focus of surveillance programs after 2000.

**Figure 2: Geographic distribution of West Nile virus activity in the United States during 2002.**



Source: Henry V. Huang, Environmental Risk Analysis Program, Centre for the Environment, Cornell University, <http://www.cfe.cornell.edu/erap/WNV/Maps/HH-USCan-2002Dec07.gif>

Mosquitoes also have been a focus of WNV surveillance.<sup>9</sup> Sound data regarding which species of mosquito are the main vectors for WNV are essential to understanding the ecology of the virus in North America and for devising approaches to its control. Many mosquito species have been found infected with WNV, including some dormant adults overwintering in protected sites in northern environments. This work is ongoing and must continue for several years before an ecological understanding of the virus will emerge.

### Disease and WNV

In mammals, including humans, it is clear that most infections with WNV result in no disease at all. Exposure is often widespread, but disease is rare. A small proportion of infected mammals do develop disease, however. This usually takes the form of inflammation of the central nervous system: encephalitis, meningitis, and/or myelitis. In humans, older people rather than younger people appear to be at greater risk of developing serious disease after infection, but the age range of affected people is very wide: from a few months to 99 years in the current epidemic. Bites from infected mosquitoes are the usual route for human infection. However, during the current epidemic, virus transmission also has occurred through blood transfusion, organ transplant, and breast milk from infected persons, events that have greatly amplified public concern about WNV.

In birds, infection with WNV may range from no disease at all to serious disease. Much remains to be learned about variations in susceptibility to disease from WNV among bird species and among individuals within a species. Members of the crow family suffer high rates of mortality. Often they die with virus present in a wide range of tissues, but no gross or microscopic lesions such as encephalitis. Other bird species (eg, hawks and owls) are also susceptible to fatal disease from WNV, but they usually die with inflammatory lesions in various tissues indicating a longer course between infection and death. Some bird species appear to tolerate infection without becoming diseased. It is very likely that these infected, but asymptomatic species will prove to be the most important species in the ecology of WNV, serving as major reservoirs of virus and sources of infection for mosquitoes. Much testing and experi-

**Figure 3:** Ataxic horse suffering from WNV infection; his front feet are stepping on each other while the hind legs are slightly base wide.



mental work needs to be done before it is known which North American bird species are the most ecologically important reservoir species.

### WNV disease in horses

WNV disease in horses is of considerable public and veterinary concern. Although the disease has been reported in a variety of domestic species, horses are particularly susceptible. While there are many more subclinical than clinical cases, some horses can show severe clinical signs and the mortality rate in these affected horses is high.

### Clinical signs

The incubation period is typically 6–10 days, however, in some cases it may be as long as 20 days.<sup>10</sup> Horses infected with WNV can be unaffected. In a recent study, only 1 of 12 horses experimentally infected with WNV showed clinical signs.<sup>11</sup> Symptomatic horses show a wide range of signs, ranging from mild peripheral neurologic disease to encephalitis.<sup>12</sup> Anecdotal reports suggest signs are more common in older horses. Infected horses may have a mild fever, an acute onset of ataxia in all 4 limbs (Figure 3), hypermetria, head tremors and lip twitching (Figure 4), hypersensitivity to touch or sound, somnolence, weakness, recumbency, and seizures.<sup>13,14</sup> Anisocoria and slow pupillary light responses, anorexia, altered mentation, fine and coarse fasciculations over different parts of the body, drooping of the lower lip, stupor, blindness, complete flaccid paralysis of one or more limbs, muzzle edema, symmetrical or asymmetrical weakness of the tongue, head tilt, and dysphagia have also been reported.<sup>12,14–16</sup>

**Figure 4:** Lip twitch in a horse with neurological symptoms of WNV infection.



### Diagnosis

Antemortem diagnosis of WNV disease is based on a combination of a physical examination to detect neurologic signs and serologic evaluation.<sup>12</sup> Although the clinical signs are variable, WNV infection should be particularly suspected when signs of acute-onset weakness or ataxia, tremors, and mentation changes are observed.

A number of tests are available to confirm recent infection in suspect horses. In many circumstances, the IgM capture ELISA test is most appropriate. This test is performed on serum and it detects the early immunologic response to infection that develops days postexposure. It is usually positive during the clinical phase of WNV infection in horses. Based on limited information, vaccination does not appear to interfere with testing.<sup>15</sup> The plaque-reduction neutralization test (PRNT) can also be used to measure virus-neutralizing antibodies in the serum or the cerebrospinal fluid (CSF). This test measures the IgG response and 2 serum samples collected 10 to 14 days apart are required for diagnosis. Recent infection is indicated by seroconversion with a 4-fold or greater rise in titer.<sup>17</sup> Paired sera must be submitted because previous exposure or vaccination interferes with the test results.<sup>15</sup> Virus isolation is not routinely performed because of the short viremic stage and the presence of low levels of the virus.<sup>17</sup> In rare instances, it is possible to isolate the virus from the tissue of the horses dying of the disease before the IgM capture ELISA becomes positive. Postmortem diagnosis can be achieved using PCR detection of virus or, in some cases, virus isolation using multiple unfixed

sections of the cerebrum, cerebellum, brainstem, and representative samples of the spinal cord. The IgM capture ELISA on serum may be useful in detecting exposure in cases from which virus has cleared. During shipping, the samples should be kept chilled and care must be taken because of the zoonotic potential of WNV.<sup>17</sup>

Other laboratory tests are less specific, less sensitive, and less useful. CSF analysis may be xanthochromic,<sup>12</sup> and have increased mononuclear cells and total protein.<sup>15</sup> However, in some affected horses it is normal.<sup>12,18</sup> Complete blood cell count results are variable.<sup>15</sup> Serum biochemical analysis may reveal mild to moderate increases in blood urea nitrogen, creatinine, or muscle enzymes.<sup>15</sup>

### **Treatment**

There is no validated protocol for treating horses with WNV.<sup>12</sup> Basically, treatment is supportive and aims to reduce pain and inflammation, avoid injury, and minimize the deleterious consequences of recumbency.<sup>17</sup> Non-steroidal and steroidal anti-inflammatory medication, as well as, dimethyl sulfoxide (DMSO) have been used, but their effectiveness has not been established. Some advocate the use of corticosteroids, but these should probably be avoided since they are immunosuppressive. Fluid and nutrient support, as well as sling support can be beneficial in individual cases.<sup>17</sup>

### **Prognosis**

It was once estimated that roughly 10% of affected horses show severe clinical signs and for those that do, the prognosis is poor.<sup>10</sup> However, the clinical outcome of infected horses ranges from complete recovery to intractable encephalitis and death.<sup>17</sup> The mortality rate appears to be 25%–40%.<sup>14,15</sup> Favourable criteria include mild clinical signs, rapid improvement, and recovery from recumbency within 2–4 days.<sup>15</sup> Unfavourable criteria include severe clinical signs and recumbency, especially for more than 3–5 days.<sup>15</sup>

### **Prevention**

#### **Vector abatement**

Efforts should be made to reduce the population of mosquito vectors and to minimize the exposure of susceptible horses to mosquitoes.<sup>17</sup> Mosquito population reduction can be achieved by using larvicidal or adulticidal chemical treatments and by removing mosquito-breeding sites. Physical and chemical barriers can be placed between horses and mosquitoes in order to decrease the exposure during the epizootic.<sup>17</sup> Summer-weight blankets and mosquito repellents can be helpful.<sup>17</sup> Horses should be stabled at night and during other periods of great mosquito activity, and the barns rendered as vector-free as possible.<sup>16,17</sup>

#### **Movement restrictions**

Horses are not likely to serve as a source of the virus for mosquitoes.<sup>17</sup> However, there are still concerns about

the potential threat posed by close proximity to infected animals.<sup>17</sup> Only restriction of movement of sick horses is justified since there is no evidence that WNV produces a long-term viremia in horses.<sup>17</sup> In a recent experiment with one species of mosquito, mosquitoes did not become infected after feeding on experimentally infected horses in the viremic stages of the disease.<sup>11</sup> Horses that are outdoors at night are at higher risk of infection.<sup>16</sup> The European Union has placed import restrictions on United States horses originating from specific states.<sup>17</sup>

### **Vaccination**

The available EEE, WEE, and VEE vaccines are not cross protective for WNV infection.<sup>17</sup> Currently, there is only one conditionally licensed vaccine. It is a killed, whole cell, adjuvanted vaccine. Two unpublished studies have been released. In a safety study using 649 horses and ponies, 1.2% of the vaccinated horses showed local or systemic reactions within 2 weeks after the second vaccination. Therefore, the vaccine is considered safe. The second study was an immunogenicity study. A plaque reduction test showed a significant increase in the neutralizing antibodies 14 days following the second vaccination.

The vaccine is available in 10-dose tanks and a booster dose is required 3–6 weeks after the initial dose. Once the tank is opened, it should be used as soon as possible. It has been recommended that the initial and booster be given at least 4 weeks before the start of the mosquito season.<sup>15</sup> However, the duration of immunity appears to be short and the period when clinical signs are seen is later than the start of the mosquito season. Therefore, it might be more beneficial to time the second vaccination for 2 weeks before the period when first reports of equine cases are likely. The optimal frequency of subsequent vaccine boosters is unknown at this time.<sup>15</sup> In Florida, a small number of fully vaccinated horses developed the disease.<sup>15</sup> Also, many horses received only one injection during the outbreak and a significant number of them developed the disease.<sup>15</sup> Horses that have clinical signs do not benefit from vaccination and vaccination may confound testing.<sup>15</sup> Vaccination of recovered horses is probably not necessary in the year following infection because they are likely to have an immunity against WNV.<sup>15</sup> However, the duration of this protection is unknown at the present time.

The Canadian Food Inspection Agency states that vaccinated horses may not be eligible for export to countries that require negative serological test for WNV and that veterinarians must maintain all records pertaining to the use of the vaccine for 6 years.

### **Editor's Note**

In an oral report at this year's American Association of Equine Practitioners (AAEP) meeting, data were presented suggesting that horses only receiving 1 dose of vaccine had very little protection. Following 2 vaccinations, protection appeared to be relatively short-lived and only in the second month following vaccination were no cases of

clinical WNV infection reported. Protection appeared to wane rapidly in the fifth and sixth months postvaccination.

The optimal timing and frequency of WNV vaccination is not yet established. Based on the AAEP data, a first dose in April or May with the second vaccination at the end of June, might be a good combination. Protection should be at its peak in August and be relatively good in September, months when there are usually a large number of cases. If finances are not a limiting concern, horses vaccinated before the start of mosquito season could receive a third vaccination in July to provide additional protection in August and September.

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