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West Nile Virus in Canada with a Focus on the Prairie Provinces: 2002-2004

By Tasha Epp, DVM

West Nile virus (WNV) has spread across the North American continent, since its introduction in the summer of 1999. The virus first appeared in Canada in Ontario, Quebec, and Manitoba in 2001, Saskatchewan in 2002, and Alberta in 2003. At present, the virus is widespread in the southern portions of all the Prairie provinces, Ontario, Quebec, and Nova Scotia. WNV is an arthropod-borne virus, circulating primarily between birds and mosquitoes; however, it can affect mammals, including humans and horses as dead-end hosts. A complex interdependence between mosquitoes, climate, and bird species drives the amplification of the virus. Prevention is aimed at mosquito reduction and, in horses, improving immunity through vaccination. This issue of *Large Animal Veterinary Rounds* discusses the WNV epidemic in Canada with special reference to the disease in the Prairie Provinces.

Introduction

West Nile Virus (WNV) is one of several arboviruses or arthropod-borne viruses that are maintained in nature through transmission between susceptible vertebrate hosts (birds) by blood-feeding arthropods (mosquitoes).¹ In 1937, the virus was first identified in Africa and has since been implicated worldwide in human, horse, and bird epidemics of neurologic disease and death.²⁻³

In North America, wild birds of the corvid family are especially susceptible to the virus and their deaths have been used for tracking the geographic spread of the disease across the continent.⁴ The first reported cases of neurologic disease due to WNV in humans and birds occurred in early August of 1999 in the suburb of Queens, New York city, and in horses in late August on Long Island, New York.⁵⁻⁶ A narrative account of the emergence of the disease in New York can be found in the book *Secret Agents* by Madeline Drexler.⁵

Over the next 3 years, the spread of WNV was unexpectedly rapid across North America. In January/February of 2000, ribonucleic acid (RNA) of WNV was found in mosquitoes overwintering in storm and sewer systems of New York.⁷ By December 2002, 44 states and 5 provinces (Saskatchewan, Manitoba, Ontario, Quebec, and Nova Scotia) had reported cases of WNV in horses, humans, and birds.⁴ Persistence of the virus in temperate climates, the involvement of migratory birds, and the continued spread across the continent prompted speculation that the virus was well-established in North America.⁸

WNV transmission

In North America, WNV is maintained by a natural amplification cycle involving various species of birds and mosquitoes (primarily the genus *Culex*, but potentially other genera as well). Virus amplification is an increase in the quantity of virus within a host and/or an increase in the number of infected hosts. This process is most efficiently accomplished with specific species and climatic conditions as discussed in the following sections. There are differences for participation of mosquitoes



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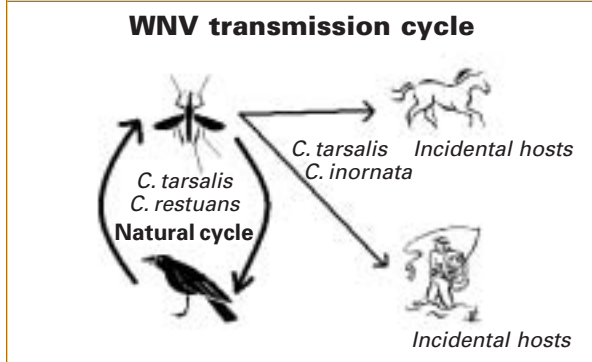
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Figure 1: Transmission cycle of West Nile virus. This includes the main mosquito species involved in the cycle on the Prairies. The natural cycle involves birds (multiple species, but largely corvids) and mosquitoes (Prairie species listed) with spill-over into the incidental hosts (humans and horses) during peak risk periods.



in the transmission cycle between Eastern and Western provinces/states (Figure 1).⁹

Mosquitoes

Culex (C.) restuans and *C. tarsalis* are the primary mosquito species involved in the transmission cycle of WNV in Saskatchewan and likely the rest of the prairies as well.⁹ WNV has also been found in *Culiseta inornata*, but the role of *C. inornata* in transmission remains unknown. *C. restuans* feeds primarily on birds, but will occasionally have a mammalian blood meal. This mosquito is thought to be involved in the bird-to-bird amplification of the virus; it uses catch basins and water-filled tires for laying eggs. *C. tarsalis* prefers to bite birds, but readily switches to a mammalian blood source after the birds fledge (around early to mid July). This species prefers sedge meadow areas with shallow pools of stagnant water, such as hoof prints or ditches. It is known to bite several times, especially in hot weather; it is capable of producing multiple generations of mosquitoes in one season and is considered the main vector of virus amplification and transmission to mammals (including humans).

Southeastern Saskatchewan has always had higher numbers of *C. tarsalis*, probably due to a warmer climate and more wetlands.⁹ In 2003, however, *C. tarsalis* was found throughout the lower half of Saskatchewan as far north as Spiritwood and Meadow Lake. A massive mosquito trapping program found that the period of highest species abundance and documented presence of WNV in mosquitoes occurred between mid-July and mid-August. Curry provides a more complete story of the mosquitoes in Saskatchewan.⁹

In studies conducted in the northeastern United States, several *Culex* species have been implicated as the main vectors involved in the transmission cycle.¹⁰ *C. restuans* is thought to be important in bird-to-bird transmission in early summer with *C. pipiens* important in amplification of the virus later in the season. *C. melanura* appears to be

important in sylvatic (rural) transmission among birds, while *C. salinarius* is suspected in the transmission of WNV to horses and humans. In many urban areas of southeastern Canada (Ontario and Quebec) *C. pipiens* and *Aedes vexans* are thought to be the primary vectors for WNV transmission to humans.^{5,11}

Birds

In birds, WNV infection can range from viremia with no clinical illness to severe disease with variable periods of illness prior to death. Members of the corvid family (American crow, Raven, Magpie, Gray jay, and Blue Jay) are commonly reported as infected with WNV; they have high mortality rates that are used to track geographic viral spread. However, other species of birds are possibly involved in the transmission cycle. A species is considered a competent reservoir if it can develop a sufficient viral blood titer for a long enough time to allow transmission to mosquitoes.¹² Experimental research suggests that the House Sparrow and the Common Grackle are competent reservoirs of the virus and are able to transmit WNV to mosquitoes such as *C. tarsalis*. These species have sufficient virus to infect mosquitoes that feed on them, but may not show signs of illness or die from the infection. In addition, experimentally, birds have been shown to transmit the virus to cagemates through oral and cloacal fluids; it is speculated that this probably occurs in nature as well.

Climate

The virus amplification cycle in wild bird populations and the various mosquito species is controlled by environment. In the temperate climate of the northern United States and Canada, there is a specified window of risk for WNV infection. In Saskatchewan, the detection period of clinical cases for both humans and horses has been mid-July to mid-September, corresponding to the period of warm temperatures, less rainfall, and an active mosquito population. Changes in Saskatchewan climate patterns (eg, warmer winters, less rainfall, warmer minimum daily temperatures, longer frost-free periods, etc) are implicated in the expanded distribution of *C. tarsalis* from its 1970 limits north into the parkland or boreal transition ecoregions.⁹ Similar northern expansions have probably occurred in other provinces. An interdependence between climate and habitat suitable for mosquitoes, and bird species capable of amplifying the virus are the governing risk factors for both humans and horses.

Mammals

Mammals, such as horses and humans, are considered dead-end or incidental hosts for the virus because they do not produce enough virus to re-infect a mosquito and maintain the transmission cycle. However, they are capable of showing clinical symptoms.¹³ Horses can have two manifestations of infection with the virus: clinical signs with

neurological symptoms or inapparent infection. Several studies have suggested that the seroprevalence of infection (with or without symptoms) can be as high as 58% of those sampled.¹⁴ Of those infected, approximately 10% will show clinical symptoms that can be mild (eg, ataxia) or severe (eg, recumbency and paralysis). Of those showing symptoms, 25%-40% will die or be euthanized.

Serological testing for WNV

There are many tests for WNV in birds, animals, and humans. The tests confirm WNV infection by identifying the virus directly or indirectly through finding antibodies to the virus. Tests can be performed on specimens including postmortem tissues, cerebrospinal fluid, whole blood, or serum. Since special biosecurity precautions are needed when testing for the virus itself, most WNV testing measures antibodies to the virus.

Multiple tests are available for horses depending on the type of sample, time-frame, and cost. The choice of a test is largely dependent on the local lab and the purpose of the test, whether surveillance or diagnosis of clinical disease. The polymerase chain reaction (PCR) is used for post-mortem tissues, primarily brain. The plaque reduction virus-neutralization test (PRNT) measures virus-neutralizing antibodies in both humans and horses, but is not widely available because it uses live WNV. The immunoglobulin (Ig) M-capture enzyme-linked immunosorbent assay (ELISA) is used to detect the IgM antibody in many mammals. There are also reagents available to perform an ELISA to detect IgG antibodies. The ELISA tests are widely used because they are cost effective, can be performed on serum, and have a quick turnaround time.

IgM antibody develops 8-10 days postinfection and persists for <2-3 months (average 1 month).⁶ This antibody is suitable for identifying recently infected horses that are “symptomatic” clinical cases of WNV, ie, displaying neurologic symptoms such as ataxia, recumbency, paresis or paralysis of limb(s), tremors, and/or seizures. It is less useful for surveillance because the antibody has a short duration in the horse and can be “missed” with sporadic or 1-time sampling. IgG antibody develops later and can be readily detected by 1 to 2 months postinfection. Using PRNT, antibodies have been documented to persist for >2 years, which suggests a long-lasting immunity to natural infection.¹⁵ IgG antibody is a good tool for surveillance of the historical extent of WNV spread, but it does not provide sufficient information to differentiate clinical WNV disease from existing titers due to previous exposure to the virus. The interpretation of IgG antibody is complicated by both the persistence of the antibody following natural infection and vaccination-associated antibody. Since this test is not distributed as a single standardized commercial kit, there can be inconsistencies in the reported antibody concentrations as a result of variation between batches of reagents and between different laboratories.

Figure 2: Geographic distribution of West Nile virus activity in horses (clinical cases) in Canada, 2002.



■ Areas with confirmed clinical cases.

Source: Compiled through various reporting agencies, eg, Ontario Ministry of Health and Long-Term Care, Manitoba Agriculture, SAFRR, and Alberta Agriculture, Food, and Rural Development (AAFRD).

Distribution of WNV

2002

Ontario reported over 100 cases of WNV in horses in 2002. The first reported case in a horse occurred in the Windsor-Essex region where the first case of WNV in a bird was reported in 2001.¹⁶ Quebec reported 3 horses in the Monteregie region and all 3 recovered.¹⁷ By the end of the 2002 season, Manitoba and Saskatchewan had reported their first cases of WNV. Dead bird surveillance and passive surveillance of clinical symptoms in horses showed a widespread distribution in these 2 provinces.

Manitoba recorded 270 positive cases of WNV in horses located in the southern third of the province and as far west as the Saskatchewan border (personal communication, Sheilagh Copeland, MB Ag). Of these, 65 were confirmed dead (24% fatality rate). Saskatchewan had approximately 29 cases of WNV in horses. The outcome for each case was not reported, but 6 of the positive cases were diagnosed using brain tissue samples (minimum 21% fatality rate). The cases were located primarily in the southeastern corner of the southern third of the province (personal communication, Leanne Forsythe, Saskatchewan Agriculture, Food, and Rural Revitalization [SAFRR]). Alberta had no reported cases of WNV in horses or birds (despite surveillance efforts directed at birds) in 2002 (Figure 2).

2003

Some notable changes occurred before the 2003 mosquito season. A WNV vaccine was licensed for use in Canadian horses. Nevertheless, WNV continued to move westward and most of the documented cases in Canada occurred in the Prairie provinces (Figures 3 and 4).

In Alberta, clinical cases of WNV were reported in humans, horses, and birds during the 2003 season. WNV in horses was declared a provincially reportable disease in

Figure 3: Geographic distribution of West Nile virus activity in horses (clinical cases) on the Canadian Prairies, 2003.



■ Areas with confirmed clinical cases, 2003.
Source: Compiled through various reporting agencies, eg, Manitoba Agriculture, SAFRR, and AAFRD.

Alberta to assist in tracking and studying the disease. Alberta reported 170 laboratory-confirmed horse cases; 59 died (35% fatality rate).¹⁸ Of the WNV positive horses, 11 were vaccinated; 4 of the affected and vaccinated horses died (36%). Cases were located over more than half of the province with most cases located in the central regions.¹⁹

Saskatchewan reported 133 laboratory-confirmed positive cases of WNV in horses. Follow-up information was obtained for 130 of these cases and 57 deaths were reported (44%). Nine of the cases were fully vaccinated and 4 of these died (44%). Cases were located in the southern half of the province with the majority of cases in the southwestern portion. Manitoba reported 47 cases of WNV in horses. Death loss was not reported. Cases were located over the southern half of the province.²⁰

Ontario reported 9 confirmed clinical WNV horse cases in 2003, while Quebec reported 3 confirmed clinical horse cases.^{17,21} Health Canada reported higher numbers for Ontario, Quebec, and the Prairie provinces; however, these data included all positive test results from laboratories whether the tests were IgG or IgM, and irrespective of whether the animal was demonstrating symptoms.

2004

With colder than usual temperatures during the spring and summer season, WNV activity was limited in 2004. Alberta reported 4 horse cases of WNV. Saskatchewan had no confirmed horse cases, but there were a few reports of horses with symptoms consistent with WNV (Saskatchewan Health, personal communication). Manitoba reported no clinical horse cases of WNV in 2004. Ontario reported 9 confirmed WNV cases in horses and Quebec reported 1 presumptive case in a horse.²² However, all provinces reported positive bird submissions.²³

Table 1: Percentage of total clinical horse cases documented in Canada by province for 2003. Almost 95% of cases in 2003 occurred in the three Prairie provinces.

2003 Surveillance	
Province	Case count
Nova Scotia	1 (0.3%)
Quebec	3 (0.8%)
Ontario	8 (2.2%)
Manitoba	47 (13%)
Saskatchewan	133 (36.7%)
Alberta	170 (47%)
Total positive cases	362

Sources: WCVN, AAFRD, Manitoba Health, Ontario Ministry of Agriculture and Food (OMAF), Quebec WNV Flash, and Health Canada.

Risk Factors for WNV

WNV has been intensively monitored since its introduction into North America. In Nebraska and Colorado, a study of horses clinically infected with WNV found that those infected early in the season (before mid-August) were almost twice as likely to die or be euthanized as horses developing symptoms after that date.²⁴ They also found that horses with severe symptoms (eg, an inability to stand) were 52 times more likely to die or be euthanized than horses with less severe symptoms (eg, ataxia). The researchers reported that previously vaccinated (either once or twice) horses with clinical signs, were more likely to survive than horses with clinical signs and no vaccination.

Researchers in Alberta reported that very few rural horse owners practiced any type of mosquito control on their premises.¹⁸ As well, almost all owners reported that clinically affected horses had been housed outside most of the time. Affected horses under the age of 15 had a better chance of recovery. No specific breed was more likely to be affected by the disease. In the Alberta surveillance program, there were too few clinically affected vaccinated horses to determine whether vaccination increased the probability of survival.

Data from Saskatchewan are currently being analyzed, but are yielding similar results to both the Alberta and Colorado studies. Data were collected from both horses with and without clinical signs across the southern part of the province in 2003. The initial analysis suggested an association between vaccination use and a decreased risk of developing clinical signs.

Prevention strategies

WNV vaccination

In 2002, a killed WNV vaccine was approved for use in the USA and approved for Canada in 2003.

Information is accumulating that supports its use for the reduction of clinical illness and death from WNV infection. Reports from the USA have attributed the sharp drop in clinical horse cases – from over 15,000 cases (2002) to over 5,000 cases (2003) – to the extensive use of the vaccine.²⁵ However, immunity from natural infection is also likely to play a role in reducing incidence, but this has not yet been substantiated by any statistical study. There were anecdotal internet reports of abortions or foals born with defects after mares were vaccinated with the WNV vaccine. Statements by leading horse veterinarians from such institutions as the Universities of Wyoming, Kansas State, Colorado State and Ohio State do not support these allegations.²⁶ Equine owners are encouraged to report any concerns about potential vaccine involvement to the company or relevant government agencies. Vaccine efficacy was proven in accordance with standards set by the US Department of Agriculture (USDA) and the Canadian Food Inspection Agency (CFIA) that resulted in full licensure. This challenge study that involved 30 horses and resulted in 94% efficacy is on file at Wyeth Animal Health.

A new vaccine was introduced on the market in 2004. This vaccine uses recombinant vector vaccine technology. Genes for specific proteins from the WNV virus are isolated and inserted into the canarypox vector virus that is harmless to horses and other mammals. The horse immune system responds to the proteins, as it would to a WNV infection, and develops a protective immunity that is both cellular and humoral. Protection is said to be rapid (89% protection against viremia, 26 days after 1 vaccination), complete (100% protection against viremia, 14 days after 2 doses) and long lasting (90% protection against viremia after 1 year challenge). Again, these studies were done in accordance with standards set for licensure of vaccines and are on file with Merial.

Both vaccines have provided demonstrable protection against WNV by reducing viremia. To date, comparison of the 2 vaccines has not been done by an independent source; rather, comparisons have been published in informational sales pamphlets by both companies. The killed vaccine has now been packaged in combination with Western equine encephalomyelitis (WEE), Venezuelan equine encephalomyelitis (VEE), and tetanus vaccines, but is only distributed in 10-dose vials. The canarypox WNV vaccine comes in single-dose vials, but is not available in combination with any other horse vaccine.

Despite claims of 1 year protection, the American Association of Equine Practitioners (AAEP) recommends vaccination in high-risk areas every 4 to 6 months.²⁷ These areas correspond to regions where WNV poses a risk almost year round (eg, the southern US). In

Canada, clinical disease has only been reported between the months of July and October. As a result, vaccination should be timed to provide highest protection during the peak risk period (mid-July to mid-September). Considering the timing and length of the mosquito season on the Canadian Prairies, a vaccination given in June (booster or second of initial series) should provide protection for the high-risk period. If owners are concerned about protection in the early spring when mosquitoes emerge from hibernation and they vaccinate earlier than June, an additional booster shot should be given in July to enhance protection at the end of the high-risk period.

Conclusion

WNV is established in North America, although the risk of infection in humans and horses may change over time. Further research aimed at all components of viral multiplication can lead to better prevention strategies, for example, more effective mosquito control targeted at specific times, specific locations, and specific species. At present, vaccination remains the key to prevention of clinical disease in horses.

Acknowledgments: *This paper uses information from many government sources. Surveillance on a national scale can be difficult to obtain as multiple sources will have different numbers for the same season and time frame depending on the underlying source of information. Direct consultation with those involved in surveillance in each of the Prairie provinces and direct involvement in the Saskatchewan surveillance of horses aided the information gathering process.*

Tasha Epp, DVM, obtained her DVM from the University of Saskatchewan in 2000. She is presently working on completing her PhD in Veterinary Epidemiology through the WCVU.

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