CD Summary

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WEST NILE VIRUS — MOVING WESTER

SINCE ITS FIRST New-World sighting in New York in 1999, the West Nile virus (WNV) has spread south and west steadily and perhaps more rapidly than many public-health forecasters would have guessed. And although the epizootic area is yet 375 miles from our eastern frontier,* the betting in our office pool suggests that it will get here within a year. Even before then, Oregon travelers could be infected east of the Rockies and return home to be diagnosed, as has happened already to a Washington State resident. This issue of the *CD Summary* describes the clinical and epidemiologic features of WNV infection and outlines our plan for dealing with it when it arrives.

HISTORICAL BACKGROUND

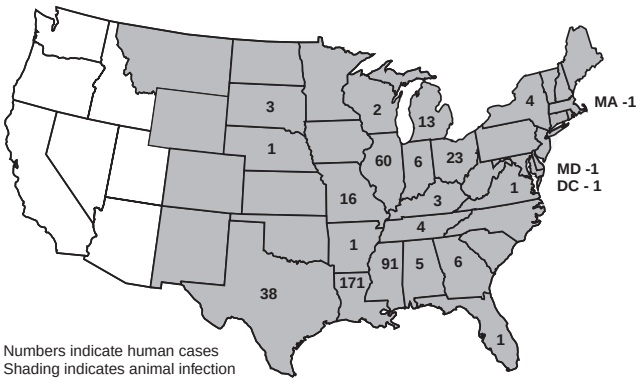
The virus in question was first isolated in 1937 from a febrile woman in the West Nile District of Uganda. WNV was recognized as a cause of severe human meningoencephalitis in elderly patients during an outbreak in Israel in 1957. Equine disease was first recognized in Egypt and France in the early 1960s. A flavivirus, West Nile virus is closely related to Japanese and St. Louis encephalitis viruses,[1] and persons infected by WNV may test positive for antibodies to St. Louis encephalitis virus. Until 1999, the West Nile and St. Louis viruses were considered Old World and New World variants of what was basically the same pathogen.

EPIDEMIOLOGY

Through December 2001, WNV had been detected in dead birds of at least 103 species, but members of the corvid family (crows, jays, and magpies) are thought to be the chief reservoir. Competent bird reservoirs will sustain an infectious viremia for 1 to 4 days after exposure, after which these hosts develop life-long immunity. Although infected birds can become ill or even die, most survive.

WNV is transmitted among birds and to mammals, including human beings, through the bites of infected mosquitoes. Mosquitoes become infected by feeding on viremic birds. Although several species of *Aedes* mosquitoes are susceptible to infection and appear able to transmit the virus, most virus isolations to date have been from *Culex* spp.,[2] which are prevalent throughout Oregon. There is no evidence that WNV can be passed through contact with infected birds; nor is there, to date, any evidence of person-to-person transmission. It is conceivable that dogs or cats could become infected by eating infected birds, but this is unproven.

West Nile virus infection
United States, 1 Jan — 27 August 2002



Infection has been found in many mammalian species, but they are not known to develop infectious-level viremias very often, and thus are probably "dead-end" or incidental hosts. Therefore, there is no reason to destroy an animal infected with West Nile virus. Of the non-human hosts, horses probably suffer the most: they develop ataxia, knuckling over, head tilt, muscle tremors, and sometimes inability to stand. Approximately 40% of cases in horses are fatal. A WNV vaccine is available for horses, but not for dogs, cats or human beings.

To date, 602 symptomatic human cases have been reported in the U.S. (453 of them during 2002). During 1999–2001, case counts tended to peak in late August and early September.[3]

CLINICAL FEATURES

Most people with WNV will have either no symptoms or only mild ones. A serosurvey conducted in 459 households near the epicenter of the 1999 outbreak in Queens, New York, found that 2.6% of residents had been infected, but only about 1 in 5 infected persons develop a febrile illness.[4] Symptoms begin 3-14 days after the bite of an infected mosquito and, along with fever, may include headache and myalgias, and occasionally skin rash and swollen lymph glands. About 1 in 150 infected persons develop meningoencephalitis, marked by headache, high fever, neck stiffness, stupor, disorientation, coma, tremors, and convulsions. In such cases, the cerebrospinal fluid almost always shows pleocytosis (leukocyte counts have ranged from 0-1,782 cells/mm^3), and elevated CSF protein (51-899 mg/dl).[3] Interestingly, muscle weakness and even paralysis can be prominent; these were found, respectively, in about 50% and 10% of hospitalized cases, and led in several cases to mistaken diagnoses of Guillain-Barré syndrome. Meningoencephalitis is more common in persons ⩾50 years of age. To date this year, 21 (4.6%) of the 453 reported cases have died.

Treatment is supportive. Interferon a-2b has been shown to inhibit the toxicity of WNV to Vero cells *in vitro*,[5] but trials in human beings have not been published.

* as the crow flies from Shepherd, Montana.

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SURVEILLANCE IN OREGON

For many years, Oregon's vector-control districts have quietly conducted surveillance for arboviruses. Mosquitoes are collected and tested (in pools of 20-50 of the same species) by polymerase chain reaction (PCR) at the Oregon State Public Health Laboratory (OSPHL) for St. Louis and Western Equine encephalitis viruses. Last year, testing for WNV was added, and >2000 pools were tested; neither WNV nor the other arborviruses were identified. In addition, flocks of sentinel chickens (10 birds per coop) have been maintained by various vector-control districts throughout the state. During 2000, one of the flocks seroconverted, becoming positive for antibody to the agent of Western Equine encephalitis, and to prevent further spread, stagnant water pools were drained and mosquito adulticides were employed. To increase surveillance in 2002, 8 more districts obtained sentinel chicken flocks in mosquito-rich areas. Of the >1000 blood samples tested in the last year and a half, none have seroconverted. Finally, to date in 2002, 10 birds of the corvid family have been submitted to Oregon State University's Veterinary Diagnostics Laboratory for PCR testing for WNV, and none had the infection.

Oregon law requires reporting of WNV and other arthropod-vector-borne infections to local public health officials within 1 working day,† and we are interested in receiving reports of *any* human encephalitis of unknown etiology. OSPHL can test serum and CSF for WNV, along with St. Louis and Western Equine encephalitis viruses; it can also send specimens to CDC to test for more exotic arboviruses.

WHAT DO WE DO WHEN IT GETS HERE?

Having observed the Patton-like dash of WNV across the country, we think it inevitable that WNV will arrive into God's country: so let's review our options. Although one might be tempted to douse the state with pesticides, a number of less costly and safer mosquito-control activities should be undertaken first. These include reducing the number of mosquito breeding sites, particularly in populated areas, by eliminating standing water; and applying larvicide to *Culex* larval habitats early. Vector-control officials have planned carefully to reduce breeding sites using larvicides already approved by the Oregon Departments of Agriculture and Fish & Wildlife.

CDC recommends the following to those concerned about WNV:

- Apply insect repellent sparingly to exposed skin. The more DEET‡ a repellent contains the longer time it can protect you from mosquito bites.
- Repellents may irritate the eyes and mouth, so avoid applying repellent to the hands of children.
- Whenever you use an insecticide or insect repellent, be sure to read and follow the manufacturer's Directions for Use, as printed on the product.
- Spray clothing with repellents containing permethrin or DEET since mosquitoes may bite through thin clothing. Do not apply repellents containing permethrin directly to exposed skin. If you spray your clothing, there is no need to spray repellent containing DEET on the skin under your clothing.
- When possible, wear long-sleeved shirts and long pants whenever you are outdoors.
- Place mosquito netting over infant carriers when you are outdoors with infants.
- Consider staying indoors at dawn, dusk, and in the early evening, which are peak mosquito biting times.
- Install or repair window and door screens so that mosquitoes cannot get indoors.
- To avoid helping mosquitoes breed in your environment, drain standing water. Routinely empty water from flower pots, pet bowls, clogged rain gutters, swimming pool covers, discarded tires, buckets, barrels, cans, and other items that collect water in which mosquitoes can lay eggs.

For more information, see *http://www.healthoregon.org/acd/wnile/home.htm* or *http://www.cdc.gov/ncidod/dvbid/westnile/index.htm*.

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†Oregon Administrative Rule 333-018-0015(5)(c)

‡ *N*,*N*-diethyl-*m*-toluamide