CD Summary

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WHY DIDN'T NOAH SWAT THOSE TWO MOSQUITOES?

Since West Nile virus (WNV) was first introduced into the United States in 1999, it has spread relentlessly across the continent, with animal or human cases now identified in every state. WNV gives every indication that it will become an endemic disease in the US, continuing to sicken wildlife, domestic animals and humans every summer.[1] While Oregon has only had a few human and animal cases to date, we expect that this year may be a different story.

HISTORY

West Nile virus, a mosquito-borne flavivirus, was first isolated from a patient in the West Nile region of Uganda in 1937. For the next 60 years, it remained a little-understood cause of febrile illness and sporadic encephalitis in parts of Africa, Europe, and Asia. It first appeared in the Western Hemisphere in 1999, when patients with West Nile encephalitis were diagnosed in New York City. Since that time, nearly 6,700 human cases of illness and 620 deaths due to WNV have been reported in the US. Given that the reported cases are only the tip of the iceberg, it is estimated that 940,000 persons have been infected with WNV in the US, of whom 190,000 have become ill. In addition, WNV infection has been identified in 58 mosquito, and 284 bird species in the US.

THE DISEASE

Clinically, WNV infection ranges from asymptomatic infection, to fever and, at its most serious, encephalitis and death. Most persons (perhaps 80%) who become infected with WNV develop no clinical illness or symptoms. Of the remaining 20% who do develop symptoms, most develop what has been termed West Nile fever, characterized (not surprisingly) by fever; headache; fatigue; rash on the trunk; and swollen lymph glands. The incubation period for WNV infection ranges from about 2 to 14 days, although longer incubation periods have been documented in immunosuppressed persons.

In its most severe form West Nile virus causes neuroinvasive disease, which occurs in 1 of 140 cases. About 25% to 35% of these patients develop meningitis alone, and 60% to 75% encephalitis or meningoencephalitis. Symptoms of West Nile meningitis include fever, headache, and stiff neck. West Nile encephalitis is characterized by altered mental status or focal neurologic findings, including limb paralysis and cranial nerve palsies. West Nile poliomyelitis, a flaccid paralysis syndrome associated with WNV infection, is less common than meningitis or encephalitis. This syndrome is generally characterized by the acute onset of asymmetric limb weakness or paralysis without sensory loss. Many survivors of neuroinvasive disease have long-term disabilities. The risk of neuroinvasive disease increases with age and appears to be substantially higher among organ-transplant recipients than in the general population.

The prevalence of immunity to WNV varies by location and population (age and sex). At one extreme are some endemic areas of Africa with WNV antibody prevalence of roughly 50% in children and 90% in adults. In contrast, the low seroprevalence in Queens, New York (3% in 1999) and on Staten Island and Suffolk County, New York and Fairfield County, Connecticut (0–1% in 2000), suggests significant background immunity did not result from the recent epidemics in those areas.[3]

TRANSMISSION

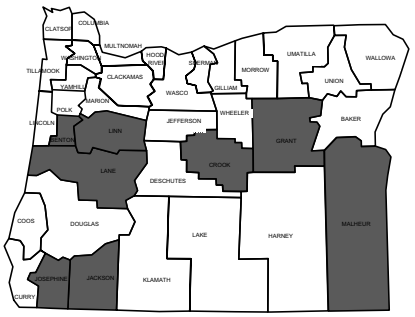
Nearly all WNV infections result from mosquito bites; however, transmission via transplanted organs and transfused blood, transplacental transmission, and occupational transmission by means of percutaneous exposure have occurred. Screening of the US blood supply was initiated in 2003. To date, over 1,000 viremic donors (6.3% of all reported cases) have been identified (818 in 2003 and 188 in 2004).

WNV is maintained in nature in a mosquito-bird-mosquito transmission cycle, primarily involving *Culex* sp. A recent study suggests that two species, *Culex pipiens* (second most common mosquito species collected in Oregon in 2004) and *Culex restuans* Theobald (*Diptera: Cilicidae*), not previously considered important in transmitting WNV to humans, may be responsible for up to 80% of human WNV infections in this region.[4] Birds are the natural reservoir (amplifying) host for WNV; horses and humans are dead-end hosts.

OREGON DATA

In 2004, five Oregonians tested positive for WNV infection: three persons were symptomatic with West Nile fever, and two had asymptomatic infections. One was identified through the American Red Cross as a blood donor. Four of

WEST NILE VIRUS, OREGON 2004



County	Human	Birds	Horses
Benton	0	1	0
Crook	0	1	0
Grant	0	0	1
Jackson	1	11	4
Josephine	0	5	4
Lane	0	2	0
Linn	0	0	1
Malheur	4	3	22
Total	**5**	**23**	**32**

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the five lived in Malheur County and one in Jackson County; four were female. All patients recovered fully. In addition, 32 horses and 23 birds tested positive for WNV—most of them found in Malheur and Jackson Counties.

Funding from CDC has enhanced Oregon's surveillance for WNV. Animal sample collection (birds, mosquitoes and sentinel chickens) is underway. Vector control districts and county environmental health agencies are assisting the public with dead bird collection. Bird collection samples should be fresh (dead within 24 hours) and kept refrigerated until testing is done. Vector control districts specialize in female mosquito collection, identification and testing.

Horses presenting with clinical symptoms of West Nile virus can be tested at no cost to the veterinarian. Refrigerated serum samples with clinical information and location of the animal to be tested must be submitted to process the sample. Bird carcasses and horse serum samples will be tested at the School of Veterinary Medicine, Diagnostics Laboratory in Corvallis, Oregon (OSUVDL, see box for web site).

DIAGNOSIS AND TREATMENT OF HUMAN CASES

The most efficient diagnostic method in humans is detection of IgM antibody to WNV in serum collected within 8 to 14 days of illness onset or CSF collected within 8 days of illness onset using the IgM antibody-capture, enzyme-linked immunosorbent assay (MAC-ELISA). Specimens from human patients with suspected WNV neuroinvasive disease will be given priority for testing at the Oregon State Public Health Laboratory (OSPHL). West Nile Fever cases and other patients with mild symptoms can be privately tested and if positive confirmed at OSPHL. For testing at OSPHL, a form (see box below) must accompany the sample describing the patient's clinical symptoms

To date, therapy for WNV infection has been mostly supportive, since no currently available antiviral or other drug has proven efficacy. Experimental treatments under consideration for human therapy include ribavirin, interferon 2 alpha, anti-WNV immunoglobulin and anti-sense therapy (see box below for information on clinical trials).

PREVENTION

Avoiding mosquito bites is by far the most effective way of preventing WNV infection. Mosquito bites can be avoided by the use of repellants with either DEET,* Picaridin or oil of lemon eucalyptus. Other options include wearing protective clothing (long sleeves, socks, and long pants) when outdoors. In addition, many of the mosquito species that spread WNV bite primarily from dusk to dawn. It is advisable to either stay indoors during these hours or to use protective clothing and repellent.

The term "Integrated Pest Management" (IPM) best represents how to keep humans removed from the biting mosquito population. IPM includes removing and preventing unnecessary standing water and repairing window and door screens. Using chemical pesticides is another significant aspect of IPM, and includes larviciding and adulticiding.

* (DEET);*N*,*N*-diethyl-*m*-toluamide, now called *N*,*N*-diethyl-3-methylbenzamide

No human vaccine for WNV is yet available, but several laboratories are currently conducting vaccine research. Both inactivated and DNA-based vaccines have been developed for use in horses. Based on the attack rate and case fatality among the equine population, vaccine therapy is strongly recommended.

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1. Glaser A. West Nile virus and North America: an unfolding story. Rev Sci Tech 2004;23:557–68.
2. Petersen LR, Hayes EB. Westward ho?—The spread of West Nile virus. N Engl J Med 2004;351:2257–9.
3. Campbell GL, Marfin AA, Lanciotti RS, Gubler DJ. West Nile virus. Lancet Infect Dis 2002;2:519–29.
4. Kilpatrick AM, Kramer LD, Campbell SR, Alleyne EO, et al. West Nile virus risk assessment and the bridge vector paradigm. Emerg Infect Dis 2005;11:425–9.

RESOURCES

For more information about West Nile Virus:

- Contact us at 503/731-4024 or call our toll-free information line: 1-866/703-4636.
- Visit: *http://www.oregon.gov/DHS/ph/acd/diseases/wnile/wnile.shtml* or *http://www.cdc.gov/ncidod/dvbid/westnile/index.htm*
- For specimen collection information and the form: *http://www.oregon.gov/DHS/ph/acd/diseases/wnile/survey.shtml*
- OSPHL lab forms: *http://www.oregon.gov/DHS/ph/phl/forms.shtml*
- OSUVDL: *http://www.vet.orst.edu/*
- For information about clinical trials visit: *http://www.cdc.gov/ncidod/dvbid/westnile/clinicalTrials.htm*