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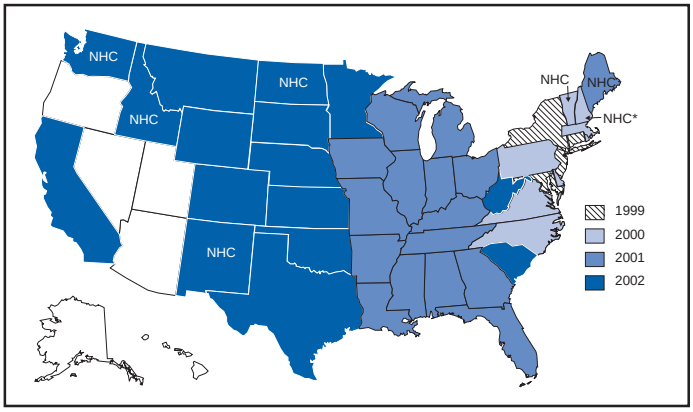
Provisional Surveillance Summary of the West Nile Virus Epidemic — United States, January–November 2002

This report presents provisional summary surveillance data about West Nile virus (WNV) activity that were reported to CDC during January 1–November 30, 2002, through the ArboNET surveillance network. In 2002, the reported numbers of human and animal infections increased, and the geographic range of WNV activity expanded substantially. These data underscore the need for intensive surveillance to detect and quantify WNV activity in areas where humans are at risk, public education to teach persons how to prevent mosquito bites, and sustained and integrated mosquito-control activities.

ArboNET is a web-based surveillance data network comprising 54 state and local public health departments and CDC. Specimens from ill humans and animals, dead birds, captive sentinel animals (mostly chickens), wild-caught birds, and mosquitoes were collected by state and local public health departments and other cooperating state and federal agencies and tested for WNV or WNV-specific antibody. Test results, the county and date of specimen collection or illness onset, and other data were entered into state and local public health department databases. Animal data were forwarded regularly to ArboNET through a secure data network; human cases were reported to CDC by telephone or facsimile only.

In 2002, WNV activity was reported from 2,289 counties in 44 states and the District of Columbia (DC) (Figure 1) compared with 359 counties in 27 states and DC in 2001 (*1*), and WNV virus was detected for the first time in 1,929 U.S. counties and 16 states. In 2002, a total of 3,389 human cases* of WNV disease were reported, compared with 149 during 1999–2001, and large numbers of WNV-infected birds, equines, and mosquitoes also were reported.

FIGURE 1. West Nile virus activity, by state — United States, 1999–2002



* No human cases.

Human Surveillance

In 2002, of the 3,389 reported cases of human WNV-associated illness, 2,354 (69%) persons had West Nile meningoencephalitis (WNME), 704 (21%) had West Nile fever (WNF), and 331 (10%) had an unspecified illness. Human cases were reported from 619 counties in 37 states and DC; five states (Illinois [774 cases], Michigan [475], Ohio [409], Louisiana [319], and Indiana [202]) accounted for 2,179 (64%) cases. Four of these five states (Illinois [492], Michigan [437], Ohio [277], and Louisiana [202]) together with Texas [164] accounted for 1,572 (67%) reported WNME cases. Illness onset dates ranged from June 10 to November 4

* Because surveillance data reported on state health department web sites but not yet reported to ArboNET are not included in this summary report, the number of human cases reported from 2002 in this summary might differ from those reported in preceding 2002 *MMWR* weekly updates.

reduction and personal protective measures to reduce mosquito exposure; 2) development of long-term, community-level, integrated mosquito surveillance and control programs (*10*); and 3) high-priority emphasis on the control of *Culex* mosquitoes, especially in urban and suburban areas.

Acknowledgments

This report is based on data prepared by ArboNET surveillance coordinators in local and state health departments and ArboNET technical staff, Div of Vector-Borne Infectious Diseases, National Center for Infectious Diseases, CDC.

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Laboratory-Acquired West Nile Virus Infections — United States, 2002

West Nile virus (WNV), a mosquito-borne flavivirus introduced recently to North America, is a human, equine, and avian neuropathogen (*1*). The majority of human infections with WNV are mosquito-borne; however, laboratory-acquired infections with WNV and other arboviruses also occur (*2–4*). This report summarizes two recent cases of WNV infection in laboratory workers without other known risk factors who acquired infection through percutaneous inoculation. Laboratory workers handling fluids or tissues known or suspected to be WNV-infected should minimize their risk for exposure and should report injuries and illnesses of suspected occupational origin to their supervisor.

Case Reports

Case 1. In August 2002, a microbiologist working in a U.S. laboratory was performing a necropsy on a blue jay submitted as part of a state's WNV surveillance program. The microbiologist worked in a Class II laminar flow biosafety cabinet under biosafety level 2 (BSL-2) conditions (*5*) and lacerated a thumb while using a scalpel to remove the bird's brain. The wound, a superficial cut over the dorsal surface of the interphalangeal joint, was cleansed and bandaged. Four days after injury, the microbiologist had acute symptoms of headache, myalgias, and malaise followed by chills, sweats, dysesthesias, recurring hot flashes, swelling of the post-auricular lymph nodes, and anorexia. Two days later, the microbiologist noted a maculopapular rash that began on the face; extended to the trunk, arms, and legs during the next 3 days; and then disappeared gradually. The microbiologist continued to work during illness and had intermittent chills, sweats, dysesthesias, and hot flashes for approximately 1 week before recovering fully. On the third day of illness (7 days post-injury), the microbiologist sought medical care from a physician and reported no history of recent mosquito bites, prolonged outdoor activities, or recent blood transfusion. On physical examination, the patient was afebrile with erythema on the cheeks, but the examination was otherwise normal. Serial serum samples taken from the patient and submitted to CDC for WNV serologic testing revealed evidence of an acute WNV infection. The initial specimen (collected 3 days after illness onset) was negative for WNV-specific IgM or neutralizing antibodies. Specimens collected 13 and 21 days after illness onset both were positive for WNV-specific IgM antibody; the latter specimen was positive for WNV-specific neutralizing antibody, with a titer of 160; the specimen collected 13 days after illness onset was not tested by neutralization. The brain of the blue jay tested positive at CDC for WNV RNA by real-time polymerase chain reaction (TaqMan®) using two primer/probe sets.

Case 2. In October 2002, a microbiologist working in a U.S. laboratory who was harvesting WNV-infected mouse brains in a Class II laminar flow biosafety cabinet under BSL-3 conditions (*5*) punctured a finger with a contaminated needle. The wound was cleansed and bandaged. The microbiologist's body temperature was measured several times each day, and 3 days after injury, the microbiologist had upper respiratory infection (URI) symptoms without fever or chills. The next day, URI symptoms continued with malaise, fatigue, chills, and a low-grade fever (100.9° F [38.3° C]). That evening, the patient took an over-the-counter cold medication. The next morning, the patient awoke without fever or chills but with continued URI symptoms and a

dry cough and hoarseness that persisted for >1 week, although the patient missed only 1 day of work. At no time did the patient notice a skin rash, an increase in the usual degree of joint pain, or a stiff neck. The patient reported no history of recent mosquito bites, prolonged outdoor activities, or recent blood transfusion. The patient had a history of exposure to multiple flaviviruses or flavivirus antigens (i.e., had had dengue fever and had received yellow fever and Japanese encephalitis vaccines). Serial serum samples taken and submitted to CDC for WNV serologic testing revealed evidence of an acute WNV infection. WNV-specific IgM antibody was absent from both the initial specimens (1 day after injury and 3 days before fever onset) and a specimen collected 2 days after fever onset. Anti-flaviviral IgG antibody was detected in both of these specimens by enzyme-linked immunosorbent assay (ELISA), but no change in the intensity of IgG activity was observed. A serum specimen collected 10 days after illness onset was positive for WNV-specific IgM antibody and showed a sharp increase in the intensity of anti-flaviviral IgG antibody by ELISA. Neutralizing antibody test results are pending.

Reported by: *G Campbell, MD, R Lanciotti, PhD, Div of Vector-Borne Infectious Diseases, National Center for Infectious Diseases; B Bernard, MD, H Lu, MD, Div of Surveillance, Hazard Evaluations and Field Studies, National Institute for Occupational Safety and Health, CDC.*

Editorial Note: This report documents two recent laboratory-acquired WNV infections in the United States. On the basis of the timing of the events described, WNV infection of the two microbiologists resulted from exposure through percutaneous inoculation in laboratories. Illnesses in both laboratory workers were mild and self-limited, which is typical of illnesses in WNV-infected persons (*1*). These cases confirm that laboratory workers are at risk for occupationally acquired WNV infection (*2–4*), including West Nile meningoencephalitis.

In the second case, although the presence of heterologous flavivirus antibodies did not prevent WNV infection, these heterologous antibodies might have provided some degree of cross-protection that moderated the clinical severity of the infection. Laboratory workers should not assume that immunity to other flaviviruses will protect them from WNV infection or its more severe clinical consequences (*6*).

During the 2002 WNV epidemic and epizootic in the United States (*7*), the number of laboratories and laboratory workers involved in arboviral diagnostic and reference activities has increased substantially. Therefore, the potential for laboratory-acquired WNV infections has increased. Laboratory-acquired arboviral infections are most likely underreported, and few recent data are available (*3,4*). In 2001, a suspected case of laboratory-acquired WNV infection was reported in New York (*8*). Laboratory workers involved in necropsies or other procedures involving materials potentially infected with WNV should use every precaution to minimize their risk for exposure to fluids or tissues during handling, including standard droplet and contact precautions; using and disposing of needles, scalpels, and other sharp instruments safely; and minimizing the generation of aerosols.

The Subcommittee on Arbovirus Laboratory Safety of the American Committee on Arthropod-Borne Viruses recommends four biosafety levels for laboratories that handle arboviruses, comprising combinations of laboratory practices and techniques, safety equipment, and laboratory facilities (*2*). Laboratory investigations that involve handling of live WNV should be conducted under BSL-3 containment (*2,9*). However, because of concerns that strict BSL-3 containment for handling human or animal specimens in the clinical diagnostic setting would severely limit the number of laboratories capable of detecting WNV infections in a timely manner, BSL-2 facilities can, with modest modification of their procedures, achieve an acceptable level of safety for the conduct of certain routine diagnostic procedures involving live WNV, including bird necropsies (*9,10*).

Participating laboratory employees should receive training that reinforces awareness of potential occupational hazards and risks and that stresses the importance of timely reporting of all injuries and illnesses of suspected occupational origin. After unintentional laboratory incidents of potential exposure to WNV-infected materials, an exposed person should cleanse any wound or exposed skin immediately and thoroughly, receive first aid, and then report the incident to a supervisor, as was done in the two cases described in this report. No antivirals or other drugs are known to be effective in the prevention or treatment of WNV infection. A baseline serum specimen should be obtained and stored. If the worker has an illness within the 2 weeks after the exposure, prompt medical evaluation, consultation with public health authorities, and collection of additional serum samples for virologic and serologic analysis are recommended.

CDC encourages the reporting of all laboratory-acquired arboviral infections to local, state, and federal public health authorities, regardless of clinical manifestations. Additional information and consultation about WNV are available from CDC's Division of Vector-Borne Infectious Diseases, telephone 970-221-6400 or 970-266-3592 or at http://www.cdc.gov/ncidod/dvbid/westnile.

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Intrauterine West Nile Virus Infection — New York, 2002

West Nile virus (WNV), a mosquito-borne flavivirus and human neuropathogen, is epidemic in the United States (*1*). In 2002, newly recognized mechanisms of person-to-person WNV transmission were described, including possible transmission from mother to infant through breast milk (*2,3*). WNV has not been previously associated with intrauterine infection or adverse birth outcomes. This report describes a case of transplacental WNV transmission. Pregnant women should take precautions to reduce their risk for WNV or other arboviral infection and should undergo diagnostic testing when clinically appropriate.

On August 29, 2002, a previously healthy woman aged 20 years in the estimated 27th week of pregnancy was admitted to a New York hospital with a 2-day history of fever, severe headache, blurred vision, abdominal and back pain, and vomiting. On examination, she had a fever of 102.7° F (39.3° C); the fetal heart rate was elevated. A computerized tomographic scan of the patient's head, a fetal sonogram, and routine analyses of blood and amniocentesis fluid were normal. A urine culture grew *Proteus mirabilis* and *Escherichia coli.* Intravenous antibiotics were administered.

Four days after admission, the fever had resolved, and the patient had pain and weakness of the legs. Neurologic examination indicated symmetric weakness of the legs and hyporeflexia of the legs and arms. No cranial nerve abnormalities were noted. Electromyelography (EMG) was not completed. On September 14, despite persistent lower extremity paresis, she left the hospital against medical advice.

On September 16, the patient was readmitted following a fall. She was afebrile, but physical examination revealed weakness in both legs. Fetal monitoring results were normal. Serum was positive for IgG antibodies to rubella virus and herpes simplex virus (HSV), and laboratory tests showed no evidence of syphilis or infection with human immunodeficiency virus (HIV). Serum also was positive for flavivirus IgM and IgG by immunofluorescence assay. Additional serum and CSF specimens were obtained during the week ending October 12. Serum was positive for WNV-specific IgM antibodies. CSF analysis indicated lymphocytic pleocytosis (11 white blood cells/mm^3, 87% lymphocytes, 8% monocytes, and 5% neutrophils), elevated protein (63 mg/dL), and the presence of WNV-specific IgM antibodies. Polymerase chain reaction (PCR) tests of CSF for WNV, enterovirus (EV), and HSV were negative. EMG studies indicated widespread involvement of the lower motor neurons or their proximal axons, with the legs affected more severely than the arms. A diagnosis of meningoencephalitis was made.

Approximately 5 weeks later, the patient delivered a live infant (estimated gestational age: 38 weeks). Serum obtained from the mother at the time of birth was positive for WNV-specific IgM and neutralizing antibodies. The infant's birth weight and general clinical examination were normal. An ophthalmologic examination revealed bilateral chorioretinitis, and MRI of the brain indicated severe cerebral abnormalities, including severe bilateral white-matter loss in the temporal and occipital lobes and cystic change in one temporal lobe consistent with focal cerebral destruction. Cord blood and infant heel-stick blood samples were positive for WNV-specific IgM and neutralizing antibodies. CSF was WNV-specific IgM antibody–positive but was contaminated with red blood cells. The presence of WNV-specific IgM antibody in the infant's serum and CSF confirmed intrauterine infection with WNV. Serum was cytomegalovirus (CMV) IgM antibody–negative but IgG-positive, and serologic tests were negative for lymphocytic choriomeningitis virus infection and toxoplasmosis. PCR tests of CSF for WNV, EV, and HSV were negative. Urine CMV culture was negative. Gross and histopathologic examinations of the placenta, umbilical cord,