

West Nile virus Infection Investigative Guidelines 2026

REPORT WITHIN 1 WORKING DAY

1. DISEASE REPORTING

1.1 Purpose of Reporting and Surveillance

1. To identify areas in which West Nile virus (WNV) is being transmitted.
2. To provide education to the general public on means of reducing mosquito habitats and the risk of mosquito bites.
3. To recommend preventive measures based on risk.

1.2 Laboratory and Clinician Reporting Requirements

Physicians and others providing health care must report confirmed or suspected cases to the Local Health Department (LHD) by telephone within 24 hours. Laboratories are asked to report within 1 working day, and to submit all samples for confirmation to the Oregon State Public Health Laboratory (OSPHL) to be forwarded to CDC.

1.3 Local Health Department Reporting and Follow-Up Responsibilities

1. Report all confirmed and presumptive cases to the Oregon Health Authority (OHA) by entry of data into Orpheus, within one working day of the initial physician or laboratory report.
2. Begin follow-up investigation within one working day. When possible, request medical records. Review and complete the relevant fields in Orpheus.
3. Facilitate transport of serum or CSF-positive samples to the Oregon State Public Health Laboratory (503-693-4100) for confirmatory purposes at CDC. Confirmatory testing is recommended for all commercial laboratory-positive results.
4. IgG positive and IgM negative tests are indicative of previous infection and these cases do not need to be investigated.

2. THE DISEASE AND ITS EPIDEMIOLOGY

2.1 Background

West Nile virus (WNV) 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. It was first identified in the U.S. in New York in 1999; the equine disease was first recognized in Egypt and France in the early 1960s.

2.2 Etiologic Agent

West Nile virus is a single-stranded RNA virus of the family Flaviviridae, genus Flavivirus. It is a member of the Japanese encephalitis virus serocomplex, which contains several medically important viruses associated with human encephalitis: Japanese encephalitis, St. Louis encephalitis, Murray Valley encephalitis, and Kunjin virus (an Australian subtype of West Nile virus). The close antigenic relationship of the flaviviruses, particularly those belonging to the Japanese encephalitis complex, accounts for the serologic cross-reactions observed in the diagnostic laboratory.

2.3 Description of Illness

Serosurveys indicate that about 80% of those infected with WNV are asymptomatic. Only 10–20% of individuals present with febrile illness of sudden onset, often accompanied by malaise, anorexia, nausea, vomiting, eye pain, headache, myalgia, rash, and lymphadenopathy; these symptoms generally last 3 to 6 days, and most cases are never diagnosed. Approximately half of hospitalized U.S. patients have had severe muscle weakness and encephalopathy. Approximately 10% of patients with encephalitis develop a poliomyelitis-like syndrome with flaccid paralysis. Neurologic presentations other than encephalitis or meningitis, which occur more rarely, include ataxia and extrapyramidal signs, cranial nerve abnormalities, myelitis, optic neuritis, polyradiculitis, and seizures. Myocarditis, pancreatitis, and fulminant hepatitis have been described in outbreaks occurring before 1990. Although recent outbreaks of West Nile virus seem to be associated with increased morbidity and mortality, severe neurologic disease remains uncommon. Two serosurveys conducted in New York City in 1999 and 2000 showed that approximately 1 in 150 infections resulted in meningitis or encephalitis, a result consistent with a 1996 Romanian serosurvey indicating that 1 in 140 to 320 infections led to these diseases.

2.4 Reservoirs

West Nile virus is maintained in an enzootic cycle involving culicine mosquitoes and different bird species; Corvids (crows, ravens, jays, magpies) seem to be most affected. After passing through three aquatic stages (egg, larva, pupa), adult mosquitoes begin to emerge in the spring in temperate regions. Viral amplification occurs in the bird–mosquito–bird cycle until early fall, when female mosquitoes bite infrequently. Many environmental factors affect this viral amplification cycle (for example, weather or climate, host and vector predators and parasites, and host immune status). When environmental conditions promote significant amplification, sufficient numbers of "bridge vector" mosquitoes—mosquitoes that bite both humans and birds—become infected in late summer and then pose an infection threat to humans. Year-round transmission is possible in more tropical climates. Through spring 2002, West Nile virus had been detected in 29 North American mosquito species; this number will undoubtedly increase as the virus spreads into new ecologic habitats. *Culex pipiens*, *Cx. quinquefasciatus*, and *Cx. tarsalis* mosquitoes live in the United States and some U.S. territories. In U.S. states, *Culex* mosquitoes can spread West Nile, St. Louis encephalitis, and eastern equine encephalitis viruses. *Culex* mosquitoes also spread Japanese encephalitis and Oropouche viruses, which do not spread regularly in U.S. states or territories. In Oregon, all the above-mentioned species are able and ready to bite!

2.5 Sources and Routes of Transmission

The main route of human infection with West Nile virus is through the bite of an infected mosquito. Mosquitoes become infected when they feed on infected birds, which may circulate the virus in their blood for a few days. The virus eventually gets into the mosquito's salivary glands. During later part of the mosquito's blood meal, the virus may be injected into humans and animals, where it can multiply and possibly cause illness. In a very small number of cases, WNV also has spread through blood transfusions, organ transplants, breastfeeding and even during pregnancy from mother to baby.

There is no evidence that WNV virus can be transmitted to humans through consuming infected birds or animals. In keeping with overall public health practice, and due to the risk of known food-borne pathogens, people should always follow procedures for fully cooking meat from either birds or mammals. WNV is not spread through casual contact such as touching or kissing a person with the virus.

2.6 Incubation Period

The incubation period usually ranges between 3 to 14 days.

2.7 Period of Communicability

Affected persons develop a short-lived viremia (2-3 days) and are considered to be dead-end hosts, except in the rare situation where transmission occurs through blood transfusions, organ transplants, breastfeeding, and perinatal spread. Human-mosquito transmission does not occur due to the low concentration of viruses during the short-lived viremic stage.

2.8 Treatment

Treatment is supportive, often involving hospitalization, intravenous fluids, respiratory support, and prevention of secondary infections for patients with severe disease.

Ribavirin in high doses, interferon alpha-2b and IV immunoglobulin (IVIG) were found to have some activity against WNV *in vitro*, but no controlled studies have been completed on the use of these or other medications, including steroids, antiseizure drugs, or osmotic agents, in the management of WNV encephalitis.

3. CASE DEFINITIONS, DIAGNOSIS, AND LABORATORY SERVICES

3.1 Indication for testing

1. Febrile, aseptic meningitis with prior testing to rule out enteroviruses by PCR or culture (recommended)
2. Febrile encephalitis of unknown etiology
3. Flaccid paralysis, myelitis or neurological symptoms following a febrile illness
4. Onset of fever, rash, encephalitis or meningitis symptoms within two weeks of receiving blood products
5. Compatible illness and exposure history in a pregnant or breast-feeding woman
6. Clinically compatible illness during transmission season

3.2 Case Definition

Clinical criteria

A case that meets the above clinical criteria for neuroinvasive disease and one or more of the following laboratory criteria for a confirmed case:

Neuroinvasive disease

- Meningitis, encephalitis, acute flaccid paralysis, or other acute signs of central or peripheral neurologic dysfunction, as documented by a physician, **AND**
- Absence of a more likely clinical explanation. Other clinically compatible symptoms of arbovirus disease include headache, myalgia, rash, arthralgia, vertigo, vomiting, paresis and/ or nuchal rigidity

Non-neuroinvasive disease

- Fever (chills) as reported by the patient or a health-care provider, **AND**
- Absence of neuroinvasive disease, **AND**
- Absence of a more likely clinical explanation. Other clinically compatible symptoms of arbovirus disease include: headache, myalgia, rash, arthralgia, vertigo, vomiting, paresis and/ or nuchal rigidity

Confirmed

Neuroinvasive disease

A case that meets the above clinical criteria for neuroinvasive disease and one or more of the following laboratory criteria for a confirmed case:

- Isolation of virus from, or demonstration of specific viral antigen or nucleic acid in, tissue, blood, CSF, or other body fluid, **OR**
- Four-fold or greater change in virus-specific quantitative antibody titers in paired sera, **OR**
- Virus-specific IgM antibodies in serum with confirmatory virus-specific neutralizing antibodies in the same or a later specimen, **OR**
- Virus-specific IgM antibodies in CSF, with or without a reported pleocytosis, and a negative result for other IgM antibodies in CSF for arboviruses endemic to the region where exposure occurred.

Non-neuroinvasive disease

A case that meets the above clinical criteria for non-neuroinvasive disease and one or more of the following laboratory criteria for a confirmed case:

- Isolation of virus from, or demonstration of specific viral antigen or nucleic acid in, tissue, blood, or other body fluid, excluding CSF, **OR**

- Four-fold or greater change in virus-specific quantitative antibody titers in paired sera, **OR**
- Virus-specific IgM antibodies in serum with confirmatory virus-specific neutralizing antibodies in the same or a later specimen.

Presumptive

Neuroinvasive disease

A case that meets the above clinical criteria for neuroinvasive disease and the following laboratory criteria:

- Virus-specific IgM antibodies in CSF or serum but with no other testing.

Non-neuroinvasive disease

A case that meets the above clinical criteria for non-neuroinvasive disease and the laboratory criteria for a probable case:

- Virus-specific IgM antibodies in serum but with no other testing.

3.3 Laboratory Confirmation

The diagnostic method is the detection of IgM antibody to WNV in serum collected within 4–14 days of illness onset or cerebral spinal fluid (CSF) collected within 8 days of illness onset using the IgM antibody capture enzyme-linked immunosorbent assay.

Since IgM antibody does not cross the blood-brain barrier, the presence of IgM antibody in CSF strongly suggests central nervous system infection.

IgG positive and IgM negative tests are indicative of previous infection and these cases do not need to be investigated.

WNV belongs to the Flavivirus genus, which also includes dengue, yellow fever, Japanese encephalitis, Zika, and others. These viruses are antigenically related — their envelope proteins share structural similarities. If a patient was recently infected with or vaccinated against another flavivirus such as Japanese encephalitis, their immune system may produce antibodies that cross-react with WNV antigens.

3.4 Services Available at the Oregon State Public Health Laboratories

OSPHL will send samples to CDC to perform confirmatory PRNT testing.

4. ROUTINE CASE INVESTIGATION

4.1 Case Interview

Interview the case (or parents) and others who may be able to provide pertinent information.

1. Identify Source of Infection

Obtain a travel history and about mosquito exposures during the likely exposure period to assist vector control. It is also important to ask about receiving blood products or about organ or tissue transplants.

2. Identify Potentially Exposed Persons

Determine if patient donated blood or organs, breastfed, or gave birth during the communicable period. If patient donated blood or organs, inform the blood or tissue bank of the potential exposure. In cases of potential mother-to-infant transmission, monitor the infant for compatible signs and symptoms for 14 days after last possible exposure.

4.2 Environmental Evaluation

Generally none, although in outbreak settings where a vector (mosquito control) district is found, an investigation may be warranted to identify factors favoring transmission.

5. CONTROLLING FURTHER SPREAD

5.1 Education

Potentially exposed persons should be instructed to watch for fever, rash, lethargy, headache, loss of appetite, or vomiting. Should signs or symptoms develop within the next two weeks, they should seek medical care as needed and advise the physician of the potential for exposure. Persons exposed should be advised not to donate blood. Pregnant women should discuss breast-feeding with their medical care provider.

5.2 Isolation

No isolation needed.

5.3 Case Follow-up

No case follow-up needed.

5.4 Protection of Contacts

None.

5.5 Precautions

Precautions against the disease are based on protection against mosquito bites.

Reduce the number of mosquitoes in breeding sites outdoors by draining sources of standing water.

- At least once or twice a week, empty water from flowerpots, pet food and water dishes, birdbaths, swimming pool covers, buckets, barrels, and cans.
- Check for clogged rain gutters and clean them out.
- Remove discarded tires, and other items that could collect water.
- Be sure to check for containers or trash in places that may be hard to see, such as under bushes or under the home.
- Place mosquito netting over infant carriers when outdoors.
- 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

Reduce exposure to mosquitos

Repellants are an important tool to assist people in protecting themselves from mosquito-borne diseases. CDC recommends the use of products containing active ingredients which have been registered by the U.S. Environmental Protection Agency (EPA) for use as repellents applied to skin and clothing. EPA registration of repellent active ingredients indicates the materials have been reviewed and approved for efficacy and human safety when applied according to the instructions on the label.

Repellents for use on skin and clothing

CDC evaluation of information contained in peer-reviewed scientific literature and data available from the EPA has identified several EPA registered products that provide repellent activity sufficient to help people avoid the bites of disease carrying mosquitoes. Products containing these active ingredients typically provide reasonably long-lasting protection:

- Install or repair window and door screens so that mosquitoes cannot get indoors
- DEET (Chemical Name: N,N-diethyl-m-toluamide or N,N-diethyl-3-methyl-benzamide)

- Picaridin (KBR 3023, Chemical Name: 2-(2-hydroxyethyl)-1-piperidinecarboxylic acid 1-methylpropyl ester)
- Oil of Lemon Eucalyptus* or PMD (Chemical Name: para-Menthane-3,8-diol) the synthesized version of oil of lemon eucalyptus
- IR3535 (Chemical Name: 3-[N-Butyl-N-acetyl]-aminopropionic acid, ethyl ester)

EPA characterizes the active ingredients DEET and Picaridin as "conventional repellents" and Oil of Lemon Eucalyptus, PMD, and IR3535 as "biopesticide repellents", which are derived from natural materials. For more information on repellent active ingredients see (<https://www.epa.gov/insect-repellents/skin-applied-repellent-ingredients>).

UPDATE LOG

September 2025 – Guidelines and template updated (Emilio DeBess).

March 2009 – Changes included expansion of repellent information under 5.5 Precautions (Emilio DeBess).