

Arthropod-borne Disease: West Nile Fever

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ABSTRACT

During the warm season months, nurse practitioners may see West Nile Virus (WNV) cases in their hospital or primary care work sites. Thorough understanding of WNV disease presentation and prevention is essential for diagnosis, treatment and patient education; especially since the neuroinvasive form can be severe. WNV is established in all 48 of the United States (US) contiguous states, as well as most countries worldwide. Extensive data from 1999 to present is available from the Centers for Disease Control (CDC).

Keywords: arthropod-borne infections, mosquito-borne infections, neuroinvasive disease, West Nile Fever, West Nile Virus

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INTRODUCTION

Since its appearance in New York City in 1999, West Nile virus (WNV) has become established in all 48 of the contiguous states of the United States, all Canadian provinces, as well as most countries worldwide.^{1,2} Alaska and Hawaii have not yet reported cases of WNV disease.¹ WNV infection can be severe. Since 1999 the Centers for Disease Control and Prevention (CDC) has collected data on a total of 39,557 cases, with 17,463 of total cases subclassified as the neuroinvasive form.² Currently, there is no human validated vaccine or treatment for WNV.¹ Public education focused on prevention is essential, as is early recognition of infection. Clinicians need to be aware of infection cycles in their locality; educate patients about protective and avoidance measures; and include this diagnosis in their differential of seasonal arthropod-borne infections.

The (reportable) disease associated with this virus is called West Nile fever (WNF). In the human host, the virus first multiplies in the skin zones, then has a visceral organ-infective stage, and finally passes to the central nervous system.³ Ironically, many individuals with WNF do not exhibit fever, and symptoms are mild or absent in about 70% to 80% of cases.^{4,5} Although most WNV infections resolve without further complications, individuals over 50 years of age and those who are immunocompromised have

greater morbidity and mortality.⁵ The neuroinvasive form of the disease has three principal forms, meningitis, encephalitis, and acute flaccid paralysis.⁶ These forms are more identifiable, usually include more comprehensive reporting, and are therefore considered the more reliable tracking statistic.¹

WNV was first identified in Uganda in 1937.¹ Disease outbreaks were infrequent and relatively mild until the 1990s when the frequency and severity escalated. Neuroinvasive presentations of WNF were noted as early as 1990 in Romania, and then Israel in 2000.⁷ Since its first appearance in the US in 1999, the CDC has tracked WNV and WNF in the US and across the globe, providing up-to-date information for health professionals, patients, and the community. A wealth of statistics, information, and links to additional resources are available on the CDC website.⁸ In 2000, an arboviral tracking system, ArboNET, was established by the CDC and state health departments to aid the documentation of this category of disease-producing viruses, including WNV, eastern equine encephalitis virus, Powassan virus, and St Louis encephalitis virus.¹ As WNV progressed in a westerly direction across the US, our information about the vectors, principal hosts, and pattern of incidence has been refined. Involvement of biologists, veterinarians, and national, state, and county public health personnel have all contributed to our improved information.

VIRUS CHARACTERISTICS AND TRANSMISSION

WNV is a single-strand RNA of the *Flaviviridae* family, genus *Flavivirus*.^{1,9} It is one of the arthropod-borne viruses (arboviruses) that can be transmitted through the bites of infected mosquitoes and ticks, primarily to mammals (eg, humans, horses, llamas, dogs, cats, or squirrels) and other hosts (eg, birds, chickens, or turkeys).¹⁰⁻¹² Although the virus has been detected in 65 different mosquito species in the US, the three principal mosquito vectors of WNV are *Culex pipiens* in the northern half of the country, *Cx quinquefasciatus* in the southern states, and *Cx tarsalis* in the western states; however, there is regional overlap in the distribution of these mosquitoes.^{1,13,14} WNV has complex host, host amplification, and transmission requirements. For example, when an appropriate bird host is infected by the bite of a mosquito carrying the virus, the virus will amplify in the bird, then an uninfected mosquito can then acquire the virus by biting the viremic bird; both infected mosquitoes can transmit the infection to humans or other hosts by its bite.^{11,13} The bird host is key to this cycle because of its ability to rapidly amplify the virus and usually survive the infection; the high levels of viremia in bird hosts are associated with higher rates of virus transmission.¹⁵ The American robin is known to be a significant amplifier host.¹⁵ Many adult bird species transfer maternal antibodies to their offspring, which can inhibit WNV amplification in young birds, and, although bird immunity is gradually developing, for unknown reasons large die-offs of specific flocks occur sporadically, especially crows and ravens.¹⁵

Direct bird and animal transmission of the virus to humans and other hosts usually does not occur but can occur if dead, infected birds are handled without precautions, or, with blood, tissue exposure from infected hosts—as in veterinary care.¹

Difficulty in predicting the seasonal timing and number of human cases is related to the complexity of host factors, namely bird migratory patterns; winter climatic factors (the virus must over-winter in a viable form); warm season weather patterns; and the number, type, and distribution of mosquitoes and birds in a region.^{1,15} Weather, especially temperature, has been shown to correlate with increased incidence of human disease, although prediction remains

exceedingly difficult.^{11,13,16} Further complicating the transmission cycle and case predictability is the female mosquito's ability to transmit the virus to its eggs, which then hatch already infected.^{13,14}

WNV infection in humans can occur through transfusion of blood and blood products; screening of the US blood supply began in 2003 after a post-transfusion case was identified.¹⁷ Although there have been a few cases of WNV transmission by donated organs and tissue, screening is not routinely done at this time.¹⁸

SURVEILLANCE

Most human cases of WNV in the US occur during July to September; with some incidence in April to June, but rarely in the winter months.² CDC year-to-year data clearly show fluctuations between years, with sporadic surges in human incidence (Table 1).¹⁹ Information about the yearly initiation of cases and the reasons for the variable case surges is a high priority and a variety of surveillance activities have been developed. ArboNET collects WNV data on human disease cases, viremic blood donors, animal cases, and virus presence in mosquitoes, mosquito larvae, dead birds, and sentinel chickens.¹ The US Geological Survey posts regularly updated information and maps from the ArboNET data.²⁰

Avian migrations are increasingly monitored year round by regions and countries to prepare for potential occurrence of WNV.¹ Other predictive surveillance activities use global tracking of human and nonhuman cases and global screening programs to identify seroconversions in nonhumans.^{1,16}

Limitations of the ArboNET data, especially regarding predictability, include: seroconversion in sentinel chickens, which typically occurs 3 to 4 weeks after transmission of the virus, can lag behind initial human cases; widespread use of equine WNV vaccines limits the data on equine WNV disease; and location and testing of live or dead wild birds is labor-intensive.^{1,11} Infection with WNV, even if asymptomatic, is believed to confer lifelong immunity and, therefore, positive antibodies in any host can be the result of current or resolved infection, further confusing predictability.^{11,13} In addition, surveillance data of nonhuman WNV may vary from year to year and not all states or regions collect similar data.¹

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