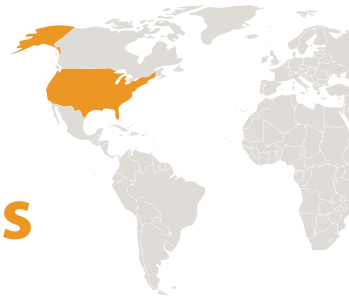


West Nile Virus in the United States

Coincidence or fate?

Merve Ulubas

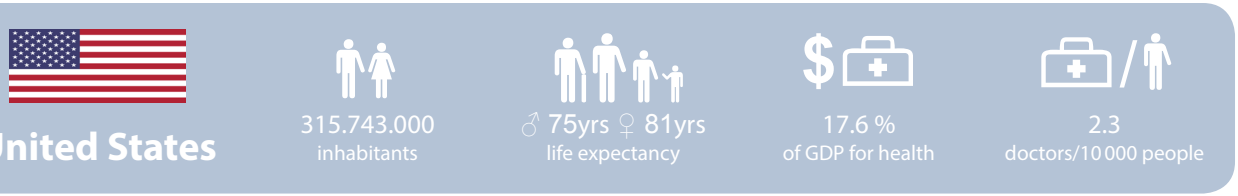
In January this year, health officials reported 1118 cases of West Nile Disease and 41 deaths across 38 states in the United States(US), almost half of which occurred in Texas. The Centers for Disease Control and Prevention (CDC) speak of the largest outbreak in the US in years (1). Every year, several states are invaded by symptomatic presentations of disease caused by this virus. How did the virus get to the US in the first place? And more importantly, can it be prevented?

West Nile Virus

The West Nile Virus (WNV) is a RNA arbovirus, part of the Japanese encephalitis virus group. The virus resides in African, Asian and European regions. Generally, viral initiation in humans leads to silent infection. The primary vector of WNV in the US is the *Culex pipiens L.* mosquito, of which the females' blood feed on birds and humans. Other vectors are *Aedes* and *Anopheles* mosquitoes. Once infection in an individual mosquito has been established, transmission occurs vertically. The cycle includes mosquito-bird-mosquito transmission, although birds as natural hosts are usually not symptomatic. Transmission to humans occurs through mosquito bites, and the peak period of virus transmission to humans in the US is between (mid)summer and autumn. During the winter months, the virus is assumed to survive through hibernation of mosquitoes. To date, the exact survival mechanism has not been resolved.

Presentation of disease

During infection, WNV is symptomatic in approximately 20 to 40 percent of people. The incubation period, the period between infection and manifestation of symptoms, ranges from 2 to 14 days. It usually presents as West Nile fever and encephalitis, or as meningitis. The degree of symptoms range from an uncomplicated fever to fatal neuro-invasive disease. This may lead to symptoms such as lowered consciousness and lethargy. Furthermore, acute flaccid paralysis and movement disorders may occur. The definitive diagnosis is made through the detection of IgM in serum or cerebrospinal fluid, which can be found during the first 10 to 28 days of infection, as well as polymerase chain reaction (PCR) tests.



United States

Interestingly, WNV causes the most prevalent neuro-invasive disease in humans in the northern states of the US. But how did the virus come to the US and why did it progress in the following years? Up until now, the mode of introduction in the US is speculative. To gain a better understanding, knowledge of WNV's history is essential. WNV was isolated for the first time in 1937 in a patient in Uganda near the West Nile, hence the name. Migration of infected birds may have led to cases of WNV in Asia and Europe in the 1950s and 1960s. WNV was introduced in the US in 1999, when an epidemic of meningo-encephalitis was reported in New York City (NYC), leading to 59 hospitalized cases and 7 deaths. From there on, the virus extended to Ontario, Canada and several other eastern states in the US. The spreading trend developed during the following years, when 142 cases of the neuro-invasive WNV disease, including 18 deaths, occurred in the US. In 2011, out of 107 registered patients in New York state, 60 of them had neuro-invasive disease, 7 of which proved fatal. In addition- an estimated 785 patients with neuro-invasive disease were diagnosed with WNV in Texas that same year.

NYC outbreak: source of introduction to USA?

It turned out that the two first cases described in the outbreak of 1999 in NYC lived within a radius of 3.2 kilometres of each other and in the proximity of two major international airports in NYC. It is believed that transmission may have been caused by the importation of infected vectors, such as birds. Interestingly, during this epidemic, an increased number of deaths among birds were reported in the same region

Prevention

No vaccine against the West Nile presentations has been developed to date, although governmental and medical agencies are working on it. Currently, the emphasis of policy lies on prevention. Any form of disease by WNV can be prevented through public education about avoidance of mosquito bites. For example, this is achieved by advocating long sleeved clothing, gazed screens on doors and windows and insect repellents. Another advice is to avoid breeding spots for mosquitoes, such as stagnant water. Furthermore, organised centre vector mosquito control plays a vital role in prevention. This can be implemented by exploring mosquito breeding sites and controlling their larvae spread.

Conclusion

Although the West Nile Virus usually resides in Asian, African and European countries, the spread of the virus to parts of the US is thought to be caused by introduction of mosquito and bird vectors. Various outbreaks have occurred in different cities and states across the US, of which NYC was the first in the late 1990s. An important task at hand is public education about prevention of WNV and multi-centred vector control, since vaccination is not possible yet.

Further reading

Campbell GL et al. West Nile Virus. Lancet Infect Dis 2002;2:519-29.

About the author

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