

The west nile virus essay



**ASSIGN
BUSTER**

A West Nile (WN) virus was first isolated in 1937 from the peripheral blood of a woman in the West Nile province of Uganda in Central Africa. Since then, WN viruses have been reported from North Africa (Egypt, Israel), East, Central, and South Africa, Asia (India, Pakistan), Borneo, Europe (Cyprus, France, Romania) and, most recently, the northeastern USA. Tests for antibody to WN suggest it has also been present in Thailand, the Philippines, Malaysia, Turkey, and Albania.

West Nile viruses are members of the virus family Flaviviridae and are closely related to Japanese encephalitis viruses from the Old World and St. Louis encephalitis (SLE) viruses from the New World. In addition, WN cross-reacts in a variety of serological tests, including the plaque reduction neutralization test, with Murray Valley encephalitis (MVE), Usutu, Kunjin, Kokobera, Stratford, and Alfuy viruses. It was this cross reactivity of the New York City (NYC) WN with SLE serologic reagents that initially confused this virus with SLE. Additional tests that used direct examination of the gene sequence of the NYC virus identified it as a WN-like virus, not SLE.

West Nile is represented by at least 2 distinct antigenic groups. One, the African-Middle Eastern group, contains WN isolates from the Congo, Egypt, Israel, Uganda, South Africa, Pakistan, France, and Eastern Europe. The second antigenic group contains WN isolates from India and South Africa.

The earliest reported epidemics caused by WN were in Israel. The first involved more than 500 clinical cases in 1950. Additional epidemics were reported from Israel in 1951, 1952, 1953, and 1957. The 1950 and 1957 epidemics were reported from a site 40 miles north of Tel Aviv, while the

1951 epidemic occurred at a site 15 miles southeast of Tel Aviv. In 1952, 1953, and 1954, cases were reported from both areas, indicting the ability of this virus to be extremely focal in its epidemic transmission patterns.

Epidemics of WN occurred in the Rhone delta region of France in 1962, 1963, and 1964. The largest epidemic of WN, involving thousands of clinical cases, was reported in South Africa in 1974. Epidemic activity was again reported in South Africa in 1983 and 1984. Human cases were reported in southeast Romania in 1996 and 1997. Most recently, WN virus(es) was apparently introduced into the northeastern region of the borough of Queens in NYC during the summer of 1999.

West Nile is more mobile than other closely related viruses like the SLE viruses in North America and MVE viruses in Australia. West Nile viruses have spread from Africa to Western Europe, the Middle East, Eastern Europe, and now North America. To date, close relatives of WN like SLE and MVE have remained confined to their home regions.

In humans, infection with WN can cause clinical or subclinical symptoms. Clinical symptoms vary from temporary fever to serious encephalitis. The disease can be severe in the elderly, but is usually mild in healthy adults and children. The incubation period for WN is 3-6 days. Onset of disease symptoms is usually sudden, beginning with a sustained, elevated fever. Clinical infection can include severe headache; a rash, usually on the trunk; and swollen lymph nodes. Symptoms of WN infection can also include eye, muscular and back pains, and gastrointestinal problems. In severe cases, there are often symptoms of encephalitis with eventual neurological

involvement and sometimes death. Humans experience a low-level viremia that lasts about 6 days. Mortality rates in humans range from 5 to 13%.

In domestic animals, clinical signs of WN infection have only been observed in horses, even though most horse infections are asymptomatic. However, in the WN epidemic in France during 1962-64, horses experienced 25% mortality. As with horses, cattle, sheep, and camels can also be infected with WN, but clinical symptoms and viremia capable of infecting arthropod vectors have not been reported from these hosts. Birds, however, do experience viremias capable of infecting arthropod vectors.

Mosquitoes and ticks serve as natural vectors of WN. Most virus isolates have been from mosquitoes, suggesting that they serve as primary vectors. *Culex univittatus* appears to be the major WN vector in Africa. *Culex pipiens* is a secondary vector in South Africa and may be the primary vector in Israel. Members of the *Cx. vishnui* complex are the primary vectors in India and Pakistan. West Nile infected ticks in the genera *Argas*, *Hyalomma*, and *Ornithodoros* have been collected in northern Africa and eastern Europe. The vector(s) responsible for the 1999 NYC outbreak have not yet been identified. The most likely candidates, however, are members of the *Cx. pipiens* species complex. Members of this complex have been implicated in West Nile outbreaks elsewhere in the world, and they are among the most common mosquitoes in NYC during the summer.

As with other mosquito-borne viruses, many vertebrate species show evidence of natural infection with WN. Wild and domestic birds, however, show the most consistent evidence of infection. Hooded Crows and House

Sparrows in Egypt showed high antibody prevalence, and WN has been isolated from naturally infected Hooded Crows. West Nile was also isolated from horses showing signs of clinical encephalitis infection during the 1962-64 outbreak in France and from camels in Sudan. A WN virus was isolated from a tick collected on a camel in Central Kara-Kum in Eastern Europe. West Nile has been isolated from pigeons in Egypt, turtledoves in Turkey and wild bird species in Borneo, Cyprus, and Nigeria. Crows experimentally infected with WN showed high mortality. In nature, some crows must survive infection since antibody-positive individuals are often captured. A domestic pigeon infected with WN and showing clinical signs of illness was captured in Egypt. Domestic pigeons are abundant in urban settings and may prove to be particularly important in epidemics such as that observed in NYC during the summer of 1999.

WN viruses have been isolated from the peripheral blood of infected humans, especially during the first few days of clinical illness. Studies were done in Israel where consecutive daily blood samples were collected and analyzed for virus. West Nile virus isolation rates were 77% for the first day of infection, 27% for day 2, 18% for day 3, and 6% for day 4. Twenty-three strains of WN virus were isolated from febrile children in Egypt during the early 1950s.

The mechanism(s) by which WN was introduced into NYC is likely to be a topic of spirited debate. Several introduction mechanisms are possible. First, it is possible that WN has been maintained in low-level transmission cycles in the northeastern USA for many years and only became evident during the summer and early autumn of 1999. This surge of epidemic activity may have

been due to environmental conditions that pre-disposed the NYC area to the epidemic transmission of a flavivirus like WN. Urban epidemics of SLE in North America have typically occurred during extremely dry summers, conditions such as those that existed in NYC during the summer of 1999. Recent introduction of WN to the NYC area is also possible. A newly infected human may have traveled from Africa, Eastern Europe or some other location with a active WN transmission, arriving in NYC just as their viremia (the amount of virus in their peripheral blood) peaked. If an individual's viremia is high enough to infect vector mosquitoes (*Cx. pipiens* in NYC), some of the mosquitoes that feed on the infected blood will become infective. Newly infected mosquitoes require a temperature-dependent extrinsic incubation period (EIP), usually lasting at least 2 weeks, before they become infective. Virus replicates in mosquito tissue during the EIP, eventually infecting most of the internal organs including the salivary glands. Once the salivary glands are infected, the mosquito is usually able to transmit the virus to additional hosts during blood feeding. In NYC, these hosts may have included local wild birds: crows, pigeons, sparrows, gulls or as yet unidentified amplification hosts. This would thus establish an amplification and transmission cycle capable of moving virus out of the original transmission focus. The 1999 New York City focus was likely the Whitestone, Auburndale, and Flushing sections in the borough of Queens.

Another possibility is that a bird infected with WN virus may have been imported into the city either legally or illegally. Legally imported birds are quarantined for at least 30 days. This should ensure that infected birds do not come into contact with arthropod vectors. Illegally imported birds are not

quarantined and may have been a source of virus capable of infecting local mosquitoes. Finally, infected ticks or infected mosquitoes may have hitched a ride on an international air flight and exited at Kennedy International. Infected nymphs or adult ticks could also have hitched a ride while attached to a human traveler or a wild or domestic animal. These ticks might then have dropped off and later fed on a New York animal susceptible to WN virus, thus initiating a NYC transmission cycle. Finally, the virus may have been released accidentally from a legitimate scientific experiment or on purpose as an act of bio-terrorism. The accidental or purposeful release of any mosquito-borne pathogen would require timing that coincides with environmental conditions that favor virus amplification in vertebrate amplification hosts and transmission by suitable vectors, prior to any possible wide-spread transmission of the virus to humans. West Nile viruses possess distinct RNA profiles which can be characterized to discern different genetic forms. These profiles are sometimes quite specific and dependent on the original geographic location of the virus. Several recent studies have characterized the RNA profiles of WN viruses including several isolates from the NYC outbreak. The results show that all the NYC WN virus isolates were almost identical to one another. All the NYC WN isolates share very close affinity with a 1998 WN virus isolated from a goose in Israel. This finding that there is only a single genetic strain of WN in North America would certainly fit the possibility that the virus was recently introduced and it had not resided in North America long enough to diverge into additional strains. THE FUTURE OF WEST NILE VIRUS IN NORTH AMERICA. Regardless of its source, WN virus is likely here to stay in North America. If and when it spreads from the NYC metropolitan area remains to be seen. Of importance, has been the <https://assignbuster.com/the-west-nile-virus-essay/>

question of whether this virus will persist through the winter, either in a vertebrate reservoir, or perhaps in an overwintering mosquito. Current observations indicate that both likelihoods for persistence have indeed occurred. Three pools of *Culex* species collected during January and February 2000 at Fort Totten, New York City (northeastern Queens) yielded WN virus RNA using a very sensitive assay called reverse transcriptase polymerase chain reaction (RT-PCR). On subsequent analysis using a Vero cell culture, one of the three RT-PCR positive pools was found to contain live WN virus. This was then confirmed using a WN-specific monoclonal antibody staining of infected cells and by virus gene sequencing.

In mid-February 2000 WN virus was isolated from a Red-tailed Hawk found moribund near Bronxville, New York (north of NYC and east of Yonkers, NY). These data indicate that, as of mid-winter 2000, live WN virus was still extant in the NYC metropolitan area.

Factors that may facilitate dispersal of WN include the movement of infected humans, infected vectors (ticks and mosquitoes), and infected amplification hosts (domestic birds, wild resident birds, and wild migratory birds). It is not difficult to envision ways in which this virus may quickly move around the country. Many cities have large populations of vector mosquitoes capable of transmitting this virus. These include *Cx. pipiens* in the northern half of the country, its close relative *Cx. quinquefasciatus* in the south, *Cx. tarsalis* in the west, and *Cx. nigripalpus* in the deep south. These species of mosquitoes are certainly among the most likely North American candidates to transmit WN to birds and humans. There are likely many other mosquito and tick species that could play a role in keeping WN circulating within a region. If

WN is introduced into another region of the USA, it will, in time be observed in domestic avian populations. Nation-wide vigilance for WN is essential.

An important question concerning WN movement and transmission throughout North America is: What can be done to monitor the movement and introduction of WN in new cities and localities throughout North America? The answer is simple: surveillance, surveillance, surveillance, and more surveillance. Vector surveillance, amplification host surveillance, meteorological surveillance, and virus surveillance. Mosquito and vector control programs throughout the USA already have considerable experience monitoring WN's close relative, SLE (see the page on SLE). St. Louis encephalitis has been a continuing problem particularly in the upper mid west, Florida, Louisiana, Texas, and California where there have been many severe outbreaks. A comprehensive integrated arboviral surveillance program for SLE in Florida has been proposed and implemented (see Day and Lewis 1992). Programs similar to this should be considered for areas that are at risk for arboviral transmission, especially WN, SLE, and dengue viruses. The dengue viruses present another potential problem for humans, especially in the southern USA. It is only through vigilant surveillance that epidemics can be recognized before local, state, and federal health officials are blind-sided by the unexpected appearance of large numbers of infected humans in places like NYC. Accurate risk assessment of a vector borne epidemic will give all public health authorities time to institute control strategies and public awareness campaigns that will reduce the impact of an epidemic.

A second important question is: What can be done to minimize the impact of WN, or other vector borne pathogens, when they do become established in a region. It is essential that the most efficient and effective control or risk management strategies be applied. Authorities will make their decisions about what strategies to use based on scientific information about the pathogen and vectors involved, and local or regional environmental conditions. It is generally accepted that it would be enormously costly and very difficult to vaccinate large human populations to prevent a vector borne epidemic, even were vaccines for these viruses available. Vaccines are not currently available for the vast majority of arthropod-borne pathogens including WN and SLE.

Personal protection against biting arthropods, particularly when they are infected with dangerous pathogens, remains one of the most important ways to avoid disease. Avoid mosquitoes. Make sure screens are in good repair to prevent mosquitoes from entering houses. If you must enter areas where there is a threat of encountering infected mosquitoes, wear protective clothing. Finally, use a personal insect repellent that provides a reasonable Complete Protection Time (CPT). The CPT is the total time following repellent application that the treated individual will remain bite free. For example, under normal conditions the CPT for a 5% formulation of DEET (diethyl toluamide, presently the most effective insect repellent) is approximately 2 hours. The CPT for a 24% DEET formulation is more than 4 hours.

Fortunately, the USA has some of the best mosquito and arthropod control programs in the world. Vector control and personal protection against vectors and the diseases they carry are the best way to avoid infection with

vector-borne pathogens. For example, strategies that might be effective against WN in NYC include: the source reduction of mosquito breeding sites; focal applications of insecticides directed against adult and immature mosquitoes; public service announcements to educated residents about the vector, the disease, and disease avoidance; tips to help prevent home-invasion by infected vectors; and information about the most effective means of personal protection. Recent SLE epidemics and outbreaks in Florida have proved that one of the most effective means of reducing human infection is to widely disseminate accurate information through the media in an effort to educate the public. An individual's first line of defense during a vector-borne disease emergency is knowledge and personal protection. The U. S. centers for Disease Control and Prevention (CDC) has initiated the National WN Virus Surveillance System for 2000 as a result of the WN virus introduction to North America. The objectives of this surveillance system are: To monitor the potential geographic and temporal spread of WN virus over the eastern and southern U. S.? To further develop national public health strategies for WN virus surveillance.? To develop a more complete regional picture of the geographic distribution and incidence of the other clinically important arboviruses in the eastern and southern U. S.? To provide national and regional information to public health officials as well as the public.? To evaluate the use of cooperative agreement funds and the need for additional resources. ANDERSON, J. F., et al. 1999. Isolation of West Nile Virus from mosquitoes, crows, and a Cooper's Hawk in Connecticut. *Science* 286: 2331-2333. BIGLER, W. J., E. LASSING, E. BUFF, et al. 1975. Arbovirus surveillance in Florida: Wild vertebrate studies, 1965-1974. *Journal of Wildlife Diseases* 11: 348-356. BIGLER, W. J., E. LASSING, E. BUFF et al. 1976.

Endemic eastern equine encephalomyelitis in Florida: A twenty-year analysis, 1955-1974. *American Journal of Tropical Medicine and Hygiene* 25: 884-890.

CHAMBERLAIN, R. W. 1980. History of St. Louis encephalitis, In *St. Louis encephalitis*, T. P. Monath (ed.). American Public Health Association, Washington D. C., pp. 3-61.

CDC UPDATE. 2000. Surveillance for West Nile virus in overwintering mosquitoes, New York, 2000. *MMWR* 49: 178-179.

DAY, J. F. AND G. A. CURTIS. 1993. Annual emergence patterns of *Culex nigripalpus* females before, during and after a widespread St. Louis encephalitis epidemic in south Florida. *Journal of the American Mosquito Control Association*. 9: 249-255.

DAY, J. F. AND G. A. CURTIS. 1994. When it rains they soar-and that makes *Culex nigripalpus* a dangerous mosquito. *American Entomologist* 40: 162-167.

DAY, J. F. AND G. A. CURTIS. 1999. Blood feeding and oviposition by *Culex nigripalpus* (Diptera: Culicidae) before, during, and after a widespread St. Louis encephalitis virus epidemic in Florida. *Journal of Medical Entomology* 36: 176-181.

DAY, J. F. AND L. M. STARK. 1999. Avian serology in a St. Louis encephalitis epicenter before, during, and after a widespread epidemic in South Florida, USA. *Journal of Medical Entomology* 36: 614-624.

DAY, J. F. AND A. L. LEWIS. 1992. An integrated approach to St. Louis encephalitis surveillance in Indian River County, Florida. *Florida Journal of Public Health* 4: 12-16.

HUBALEK, Z. AND J. HALOUZAK. 1999. West Nile Fever-a reemerging mosquito-borne viral disease in Europe. *Emerging Infectious Diseases* 5: 643-650.

KRAUSE, R. M. (ed). 1998. *Emerging Infections*. Academic Press, New York, New York.

LANCIOTTI, R. S., et al. 1999. Origin of the West Nile Virus responsible for an outbreak of encephalitis in the Northeastern United States. *Science* 286: 2333-2337.

TABACHNICK, W. J. 1998. *Arthropod-borne pathogens: Issues for*

<https://assignbuster.com/the-west-nile-virus-essay/>

understanding emerging infectious diseases. In R. M. Krause (ed.), *Emerging Infections*. Academic Press, New York, New York, pp. 411-430. Bibliography: The 1999 Introduction of the West Nile Virus to North America Year: 1999.

Authors: Jonathan F. Day and Walter J. Tabachnick Unit: Florida Medical Entomology Laboratory.

Citation: Florida Medical Entomology On-line Publications PH1004.

University of Florida, IFAS.

<http://www.ifas.ufl.edu/veroweb/online/WNile/WNilemain3.htm>.