

# West Nile Virus

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**West Nile Virus**  
West Nile Fever  
West Nile Disease  
Lordige  
West Nile Neuroinvasive Disease  
Near Eastern Equine Encephalitis

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**Overview**

- Organism
- History
- Epidemiology
- Transmission
- Disease in Humans
- Disease in Animals
- Prevention and Control

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In today's presentation we will cover information regarding the organism that causes West Nile fever and its epidemiology. We will also talk about the history of the disease, how it is transmitted, species that it affects (including humans, if applicable), and clinical and necropsy signs observed. Finally, we will address prevention and control measures for West Nile virus.


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**The Organism**

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**The Organism**

- Flaviviridae
  - *Flavivirus*
    - Single stranded RNA virus
- 2 genetic lineages
  - Lineage 1
    - 3 clades (1a, 1b, 1c)
- Infects humans, birds, mosquitoes, horses, and other mammals



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West Nile virus (WNV) is a single-stranded RNA virus of the family Flaviviridae, genus *Flavivirus*. WNV is a member of the Japanese encephalitis virus antigenic complex of arthropod-borne flaviviruses. West Nile virus has at least 2 genetic lineages. Lineage 1 is divided into 3 clades (1a, 1b, and 1c) and contains both virulent and attenuated viruses. Lineage 1a is responsible for many recent outbreaks. Clade 1b consists of Kunjin viruses, which are found in Australia, and clade 1c contains viruses found in India. Lineage 2 viruses, found mainly in Africa, often cause asymptomatic infections or mild disease. West Nile virus can infect humans, birds, mosquitoes, horses, and other mammals.

Image: Electron micrograph of West Nile virus isolated from brain tissue from a crow found in New York: CDC website.


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History

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**History**

- 1937: West Nile District, Uganda
  - First isolated
- 1950: Egypt
  - Ecology studied
- Additional outbreaks
  - 1951-54, 1957, Israel
  - 1962, 2000: France
  - 1973-74: South Africa
  - 1996: Romania, 1998: Italy



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WNV was first isolated from a feverish woman in the West Nile District of Uganda in 1937. Based on extensive studies done in Egypt in the 1950s, the ecology of the disease was found to vary greatly depending on the prevalence of the disease. At one epidemiological extreme are areas where WNV circulates in most years; uncomplicated WN fever is a mild and common childhood disease. Many people are immune in these areas, and WN fever epidemics and WN meningoencephalitis cases are rare. At the other extreme are the industrialized urban areas, where little or no previous WNV activity has occurred. When aging or immunologically naive populations encounter WNV for the first time, large numbers of neuroinvasive cases have been observed. There have been many West Nile outbreaks throughout the world. In 1957, nursing homes in Israel reported severe neurologic disease and death associated with West Nile fever. The outbreak in 1996 Romania seemed to spark the beginning of several outbreaks worldwide in large industrialized urban areas.


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Epidemiology

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**United States - 1999**

- New York City
  - 62 cases; 7 deaths
- Zoo birds, crows, horses also infected
- Caused by lineage 1a
  - NY99
- First appearance of WNV in the western hemisphere



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West Nile was first discovered in the United States in 1999. There were seven deaths and sixty-two cases (12% case fatality rate) in New York City and surrounding counties. Exotic zoo birds, crows, and horses were also found to be infected. This marked the first of appearance of WNV in the western hemisphere. The strain that entered the U.S., called NY99, appears to be related to a lineage 1a virus antigenically similar to a strain that circulated in Israel from 1997 to 2000.

Image: CDC website.

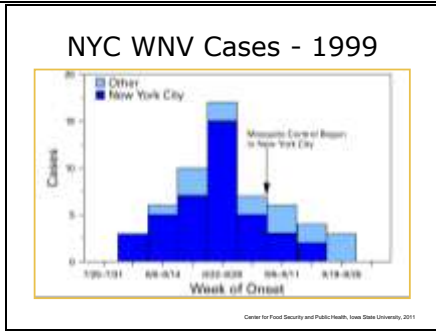
West Nile Virus

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This map shows the distribution of cases in New York City in 1999. The green dots indicate where WNV positive mosquitoes were found. The red dots indicate cases of WNV in humans, and the large circles show areas where WNV positive birds were found.

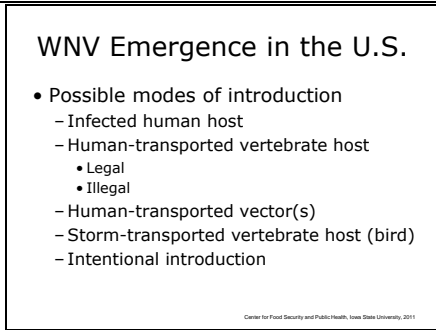
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This chart depicts the cases of infection by week occurring in New York City in 1999. Most of the cases occurred during the month of August. Mosquito control began the first week of September, and while cases were decreasing in number by that time, the control measures were likely to have helped stop transmission.

Source: MMWR October 22, 1999;48(41):944-946, 955.

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There are many speculations concerning the introduction of WNV into the United States. It is not known where the U.S. virus originated, but it is most closely related genetically to strains found in the Middle East. Possible modes of introduction include: infected human hosts, human-transported vertebrate hosts (both legal and illegal), human-transported vectors, storm-transported vertebrate hosts (such as birds), and intentional introduction.

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By 2003, WNV had spread across the U.S. to California. This map shows the number of cases of human disease in 2003. The states in red are those with human WNV cases; states in yellow (Maine) indicate avian, animal or mosquito infections.

Source: <http://www.cdc.gov/ncidod/dvbid/westnile/Mapsactivity/surv&control03Maps.htm>

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As of 2010, WNV continues to cause disease across the U.S. This map shows WNV activity reported to ArboNET, by state, in 2010.

Source: CDC.

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**Human WNV Activity**

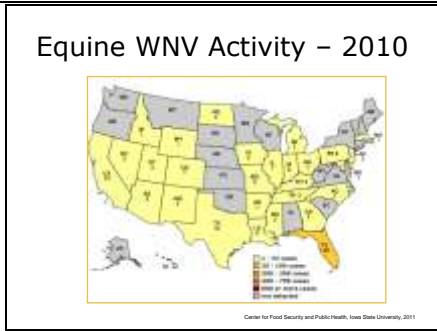
Year	Cases	Deaths
1999	62	7
2000	21	2
2001	66	9
2002	4156	284
2003	9862	264
2004	2539	100
2005	3000	119
2006	4269	177
2007	3630	124
2008	1356	44
2009	720	32
2010	1021	57

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Since the first detected case in 1999, the number of cases and deaths in humans has increased dramatically almost every year until the peak in 2003. This table shows the number of human WNV cases and deaths from 1999 to 2010.

Source: CDC.

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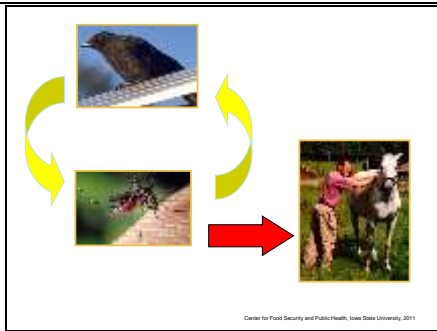
The spread of WNV in humans paralleled the spread of WNV in horses. WNV can now be found in horses throughout the U.S. This map shows the distribution of equine WNV cases in 2010.

Source: USDA APHIS.

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Transmission

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Birds (especially Corvids [ravens, jays, and crows]) are the primary vertebrate reservoir hosts for WNV. WNV is mainly transmitted by mosquitoes. In North America alone, there is evidence of infection in at least 59 species (including *Culex* spp., *Aedes* spp., and *Ochlerotatus* spp.). Some female mosquitoes feed only on birds (ornithophilic). However, some feed on birds and other species, transmitting WNV to human, horses, and other animals. The viremia that results in human, horses, and other animals not sufficient for WNV transmission, making them incidental hosts. This schematic gives a general representation of the transmission of West Nile Virus.

Image: Mosquito.


[http://commons.wikimedia.org/wiki/File:Aedes\\_albopictus\\_on\\_human\\_skin.jpg](http://commons.wikimedia.org/wiki/File:Aedes_albopictus_on_human_skin.jpg)

# West Nile Virus

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### Transmission

- Primary mosquito vector  
– *Culex* spp.
- Tick vectors  
– Asia, Russia  
– Role in transmission not clear




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*Culex* species are the most important vectors in maintaining the West Nile virus cycle. Many different types of mosquitoes throughout the world have also been found to transmit WNV. WNV has been isolated from ticks in Asia and Russia, but their role in virus transmission is still unclear at this time.

Images: (Upper) Mosquito larvae in water. (Lower): Female *Culex* mosquito laying eggs on water in an egg raft formation. CDC website.

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### *Culex pipiens*



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This map shows the distribution of *Culex pipiens* across the United States. Habitats determined by satellite data are shown in red. Mosquito distribution maps determined by means other than satellite surveillance are outlined in yellow.

Source: NASA. <http://www.gsfc.nasa.gov/topstory/20020828phap.html>

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### *Culex restuans*



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This map shows the distribution of *Culex restuans* across the United States.

Source: NASA. <http://www.gsfc.nasa.gov/topstory/20020828phap.html>

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### *Culex salinarius*



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This map shows the distribution of *Culex salinarius* across the United States.

Source: NASA. <http://www.gsfc.nasa.gov/topstory/20020828phap.html>

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### *Aedes vexans*



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This map shows the distribution of *Aedes vexans* across the United States.

Source: NASA. <http://www.gsfc.nasa.gov/topstory/20020828phap.html>

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**Transmission**

- Overwintering of mosquitoes
- Transovarial transmission
- Birds
  - Contact
  - Migratory transport



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Several additional factors may contribute to the maintenance of WNV in the U.S. Dormant mosquitoes that survive the winter may harbor WNV. Transovarial transmission has been demonstrated in some species of mosquitoes. Studies completed in birds indicate that contact transmission between birds may occur, and migratory birds may have a large role in transporting West Nile and its vectors to unaffected regions.

Image: *Culex quinquefasciatus*, shown blood-feeding on a human finger, has also been proven to be a vector associated with transmission of the WNV. CDC.

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**Human Transmission**

- Direct contact
  - Infected birds, tissues
- Laboratory acquired
- Blood transfusions
  - Screening implemented in 2003
- Organ transplants
- Transplacental transmission
- Breast feeding

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Humans usually become infected via mosquito bites, however, other modes of transmission exist. Some cases have occurred in people who handled infected birds or tissues from infected alligators. WNV has occurred in lab workers due to sharps injuries. WNV has also been found in the blood supply; however, screening was implemented in 2003. In 2002, an organ donor that had received WNV-infected blood spread the virus to four people receiving transplants. Rare cases of transplacental transmission and probable transmission in breast milk have also been reported.

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**Disease in Humans**

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**Disease in Humans**

- Incubation: 2 to 14 days
- Many WNV infections asymptomatic
- Two forms of disease
- **West Nile fever**
  - Most common form
  - Resembles influenza
  - Most infections resolve in 2 to 6 days
  - Persistent fatigue can occur

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The incubation period for WNV is approximately 2-14 days. Human illness has been classified into two forms: West Nile fever, which is relatively mild and flu-like, and West Nile neuroinvasive disease, which encompasses all cases with neurological signs. Many WNV infections are asymptomatic. West Nile fever is the most common form of the disease. This form resembles influenza, and is characterized by fever, malaise, weakness, headache and body aches. Anorexia, lymphadenopathy, nausea, diarrhea, vomiting, sore throat and conjunctivitis may also be seen. An erythematous, nonpruritic macular, papular or morbilliform skin rash occasionally develops on the neck, trunk, arms or legs. Most uncomplicated infections resolve in 2 to 6 days, but in some severe cases, persistent fatigue can last for a month or more.

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**Disease in Humans**

- **West Nile neuroinvasive disease**
  - Occurs rarely
    - Progression of West Nile fever
  - Can be severe and life-threatening
  - Three syndromes
    - Encephalitis
    - Meningitis
    - Acute flaccid paralysis
  - Persistent neurological dysfunction may occur


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A few patients with West Nile fever develop West Nile neuroinvasive disease. This form can be severe, and in some cases, it is life-threatening. Three syndromes – encephalitis, meningitis, and acute flaccid paralysis – are seen. Symptoms of more than one syndrome often occur in the same patient. West Nile meningitis is characterized by fever, headache, a stiff neck and photophobia. Patients with West Nile encephalitis have changes in consciousness, disorientation and/or focal neurological signs, which may include ataxia, incoordination, tremors, involuntary movements, and signs that resemble Parkinson’s disease (rigidity, postural instability and bradykinesia). Concurrent signs of meningitis are common, and seizures or coma may also occur. Some patients who recover have persistent neurological dysfunction.

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**Diagnosis in Humans**

- Serology
  - Serum or CSF
  - IgM capture ELISA
    - Cross reactions possible
  - Plaque neutralization test
- Detection of virus, antigen, or nucleic acids
  - RT-PCR
  - Immunohistochemistry



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In humans, West Nile virus infections are often diagnosed by serology. Diagnostic criteria include a rising titer or the presence of IgM in serum or cerebrospinal fluid (CSF). IgM in CSF indicates a recent infection; however, anti-WNV IgM can persist in the serum of some individuals for more than a year. For this reason, IgM in serum is suggestive but not definitive. Enzyme-linked immunosorbent assays (ELISAs) are the most commonly used serological tests. Cross-reactions can occur with closely related flaviviruses including yellow fever, Japanese encephalitis, St. Louis encephalitis or dengue viruses. For this reason, positive reactions in ELISAs or other tests may be confirmed with the PRN test. West Nile virus, viral antigens or nucleic acids can sometimes be detected in tissues, CSF, blood and other body fluids. WNV can usually be found in the blood of patients with West Nile fever, during the first few days after the onset of illness. Reverse-transcription polymerase chain reaction (RT-PCR) assays are often used to screen blood supplies for transfusion. However, viremia usually disappears before the onset of neurological signs, and viral RNA is generally absent from the serum of patients with neuroinvasive disease. CSF can be tested with RT-PCR, although this is rarely done in clinical practice. Immunohistochemistry to detect viral antigens is mainly used postmortem in cases of fatal neurological disease.

Image: Using a histochemical technique in processing this tissue specimen, this image reveals the presence of the West Nile virus. CDC Public Health Image Library.

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**Treatment in Humans**

- No specific therapy
- Supportive care
- Therapies in clinical trials
  - Interferon
  - Antisense nucleotides
  - IV immunoglobulin
  - Antiviral drugs



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No specific treatment, other than supportive care, is available. Intensive care and mechanical ventilation may be required in some cases. Various therapies including interferon, antisense nucleotides and intravenous immunoglobulin are being tested in clinical trials. Some antiviral drugs were promising in vitro, but most have been ineffective when tested in animal models or given to humans with severe disease. Screening for new drugs that may inhibit WNV is underway.

Image: <http://phil.cdc.gov/Phil/home.asp>

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**Disease in Animals**

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**Species Affected**

Horses*	Black Bear*	Bats*
Goats*	Wolf*	Llama*
Sheep*	Alpaca*	Cattle*
Dog*	Mountain Goat	Seal*
Rabbit	Alligator*	Cat*
Chipmunk	Gray Squirrels*	Deer*
Skunk*		
Crocodile*		



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A broad range of mammalian species are susceptible to natural or experimental infection with WNV. The role, if any, that mammals play in the WN virus transmission cycle is unknown. An asterisk indicates animal species that have been reported to show signs of WNV infection. Note this list is not exhaustive of all animal species that may be affected by WNV.

Image: Gray squirrel. <http://commons.wikimedia.org/wiki>.

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**Clinical Signs - Horses**

- Many asymptomatic
- Anorexia
- Ataxia
- Weakness
- Teeth grinding
- Convulsions
- Circling
- Tremors
- Difficulty swallowing
- Attitudinal changes
- Facial edema
- Colic
- Urinary dysfunction
- Complications
- Mortality
  - Many euthanized

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Most horses are infected asymptotically. In clinical cases, the illness is characterized by anorexia, depression and neurological signs, which may include ataxia, weakness or paralysis of one or more limbs, teeth grinding, aimless wandering, convulsions and/or circling. Tremors of the face and neck muscles are very common. Some animals have cranial nerve deficits, particularly weakness or paralysis of the face and tongue, which may lead to difficulty in swallowing. Attitudinal changes including somnolence, apprehension, hyperesthesia or periods of hyperexcitability are also common. Some horses with severe depression and facial paralysis may hang their heads; this can result in severe facial




edema. Coma, impaired vision and head pressing can be seen, but tend to be less common than in cases of encephalitis caused by alphaviruses. Colic and urinary dysfunction (from mild straining to stranguria) have also been reported. Fever is present in some but not all cases. Fatal hepatitis was seen in a donkey with neurological signs in France. Injuries, pulmonary infections acquired during prolonged recumbency, and other secondary effects can complicate the course of the disease. Some animals die spontaneously, but many severely affected animals are euthanized for humane reasons. Horses that recover usually begin to show improvement within seven days of the onset of clinical signs. Most but not all horses return to full function; approximately 10-20% may have residual defects such as weakness in one or more limbs, decreased exercise tolerance, muscle atrophy or behavioral changes.

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### Diagnosis and Treatment - Horses

- **Diagnosis**
  - Live: serology
  - Dead: WNV detection at necropsy
    - Brain and spinal cord
    - RT-PCR, immunohistochemistry
- **Treatment**
  - No specific treatment, supportive care
  - Therapy is empiric



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In live horses, clinical cases are usually diagnosed by serology. A four-fold or greater increase in WNV-specific antibodies in serum, the detection of specific IgM in CSF, or the detection of specific IgM in serum confirmed by specific IgG in the same or a later sample are diagnostic. RT-PCR can be used to detect viral RNA in equine brain and spinal cord samples taken at necropsy. Immunohistochemistry is used as a postmortem test. It may detect WNV antigens in equine brain and spinal cord, and avian brain, heart, kidney, spleen, liver, intestine and lung. Because the CNS does not contain large quantities of virus, some infected horses are not detected by this test. No specific treatment is available, but animals may recover on their own if they are given supportive care. Supportive treatment has the goal of reducing inflammation in the CNS, preventing self-inflicted injuries and adverse effects from recumbency, and providing supportive nutrition and fluids. Therapy is empiric, and similar to the treatment of other causes of viral encephalomyelitis. Mild cases have sometimes recovered without treatment.

Image: [http://www.blm.gov/id/st/en/fo/jarbridge/wild\\_horses.html](http://www.blm.gov/id/st/en/fo/jarbridge/wild_horses.html).

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### Clinical Signs - Ruminants

- Frequently a single animal affected
- **Neurological signs**
  - Sheep, alpacas, reindeer, white-tailed deer
- Most affected animals die within 1 to 2 days
- Reproductive signs may be seen in sheep



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A few clinical cases have been reported in ruminants. Frequently, only a single animal has been affected on a farm. Occasionally, a few other animals became ill around the same time. Most sheep, alpacas, reindeer and white-tailed deer have had neurological signs that resembled the syndrome in horses. In many cases, these were the first signs observed in the animal. However, a prodromal syndrome of fever, anorexia and depression was reported in one alpaca; the fever disappeared by the time the neurological signs appeared. Sudden death without prior clinical signs was seen in a reindeer. Another reindeer had diarrhea for 1 to 2 weeks before the onset of neurological signs. Most affected animals have died, but one alpaca recovered from mild head tremors and ataxia. Death often occurs within 1 to 2 days, particularly in reindeer, but some animals have been ill for several days to a week. Experimentally infected sheep did not develop systemic signs, but some pregnant ewes aborted, had stillborn lambs, or gave birth to lambs that died soon after birth.

Image: <http://commons.wikimedia.org/wiki/>

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**Clinical Signs – Dogs, Cats**

- Dogs and cats
  - Often asymptomatic
  - Rarely
    - Fever, depression
    - Muscle weakness, spasms
    - Seizures, paralysis
    - Myocarditis
- Suspect WNV in animals exhibiting neurological and cardiac symptoms



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Small animals rarely exhibit clinical illness. Case reports of dogs and cats positive with WNV report fever, depression, muscle weakness, spasms, seizures and paralysis. Myocarditis can also be found. A serologic survey conducted in the initial epidemic area of New York showed a low infection rate of dogs and cats. WNV should be suspected in animals that exhibit neurological and cardiac symptoms.

Image: <http://commons.wikimedia.org/wiki/>

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**Clinical Signs – Dogs, Cats**

- Experimental infection
  - Mosquito bite: dogs
    - All dogs showed viremia, no clinical signs
  - Mosquito bite: cats
    - All cats showed viremia
    - All but one showed mild clinical signs
  - Infected prey: cats
    - All cats developed viremia
    - None showed clinical signs
- Conclusion
  - Readily infected, not amplifying hosts

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Dogs and cats have been experimentally infected with WNV. Four dogs and four cats were infected by mosquito bite: all four of the dogs developed viremia of low magnitude and short duration, and did not show clinical symptoms of disease; all four of the cats became viremic, and all but one showed mild, non-neurological signs of disease. During the period of viremia, WNV was not isolated from the saliva of either the infected dogs or cats. In addition, four different cats were exposed to West Nile Virus by consuming an infected mouse: viremia developed in these cats also, but none showed clinical signs of disease. The study showed that dogs and cats can readily be infected with WNV, and that prey animals may serve as an important source of infection. However, neither dogs nor cats would likely serve as amplifying hosts of the disease.

Source: Emerging Infectious Diseases, [www.cdc.gov/eid](http://www.cdc.gov/eid) Vol. 10, No.1, Jan 2004.

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**Clinical Signs - Wildlife**

- Birds
  - Commonly found dead (e.g., Corvids)
- Bats, chipmunks, skunks, and domestic rabbits
  - Majority do not develop clinical signs
- Gray Squirrels
  - Lethargy, paw biting, vocalization, ataxia, circling, encephalitis, myocarditis

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West Nile virus has been detected in nearly 300 species of North American birds since 1999. Although birds, particularly crows and blue jays, infected with WN virus can die or become ill, most infected birds do survive. Bats, chipmunks, skunks, and domestic rabbits usually do not develop clinical signs. Neurological signs have been reported in squirrels, such as lethargy, paw biting, vocalization, ataxia, and others.


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Prevention and Control

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**Vaccination**

- Several commercial vaccines available for horses
  - Consult label for instructions
  - Usually 2 doses, 3 to 6 weeks apart
  - Annual revaccination
- Vaccines sometimes used off-label to protect birds




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Several commercial vaccines are available for horses in the U.S. and other countries, and one vaccine has been licensed for geese in Israel. Consult the manufacturers label for instructions. Horses usually receive two doses, three to six weeks apart, and then an annual booster. Vaccines are sometimes used “off label” to protect sensitive birds or other species. For instance, in an effort to minimize the impact of West Nile virus on endangered California condors, captive condors have been vaccinated since 2003, and attempts have been made to vaccinate wild chicks in the nest.

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**Mosquito Management**

- Surveillance
- Source reduction
- Personal protection
- Biological control
- Larvicide
- Adulticide



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All of these methods play important roles in successful mosquito management. Surveillance, source reduction, and personal protection are techniques used by individuals in order to monitor mosquito populations, reduce their numbers, and limit human exposure. Biological control, larvicide, and adulticide control methods are techniques most commonly employed on a large, community-wide basis.

Image: [www.common.wikimedia.org](http://www.common.wikimedia.org)

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**Surveillance**

- Dead bird testing
- Sentinel chicken flocks
- Mosquito collection
  - Test for pathogens
  - Account for species
- Larval and adult mosquitoes
  - Map habitats
  - Record keeping



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States may test dead birds for WNV. In addition, sentinel caged chicken flocks may be periodically tested for WNV, as they are good indicators of increased virus activity. Mosquitoes can be tested for WNV and other pathogens, alerting us to emerging diseases. Mosquito species counting programs are also useful surveillance methods. When established mosquito larval and adult threshold populations are exceeded, control activities are initiated. Seasonal records are kept in addition to weather data in order to predict mosquito larval occurrence and adult flights.

Image: Working with a sentinel chicken flock in Iowa.

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**Source Reduction**

- Eliminating larval habitats
  - Tires, bird baths, containers, rain gutters, unused swimming pools



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Source reduction consists of elimination of larval habitats or rendering of such habitats unsuitable for larval development. Empty standing water in old tires, cemetery urns, buckets, plastic covers, toys, or any other container. The water that stands in old tire piles is the ideal site for mosquito breeding. No matter how a tire sits on the ground it is always capable of collecting rain water. Empty and change the water in bird baths, fountains, wading pools, rain barrels, and potted plant trays at least once a week. Drain or fill temporary pools with dirt. Keep swimming pools treated and circulating, and keep rain gutters unclogged.

Image: <http://commons.wikimedia.org/wiki/>

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**Source Reduction**

- Making habitats unsuitable for larval development
- Public education
- Marsh water management
  - Drain, fish access, gated




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Many mosquitoes that carry West Nile Virus need to lay their eggs in a raft on standing water. By emptying containers such as the one pictured, you are doing your part in making the habitat unsuitable for egg laying and thus larval development. Public education is an important component of source reduction. Other forms of source reduction include open marsh water management, in which mosquito-producing areas on the marsh are connected by shallow ditches to deep water habitats to allow drainage or fish access; and rotational impoundment management, in which the marsh is minimally flooded during summer but is flap-gated to reintegrate impoundments to the estuary for the rest of the year.

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**Personal Protection**

- Reduce time outdoors
  - Especially evening hours
- Long pants and sleeves
- Use mosquito repellent
  - 35% DEET
  - Do not use DEET on animals
- Keep window screens intact
- Use yellow "bug" light bulbs in outdoor light fixtures



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West Nile Virus infection can be prevented by reducing contact with mosquitoes. Personal protection measures include reducing time outdoors, particularly in early evening hours, wearing long pants and long sleeved shirts, and applying mosquito repellent containing DEET to exposed skin areas. Do not use DEET products on your pets. The concentration of DEET in mosquito products is too high to be safe for cats and dogs. Exposed pets may develop severe neurological problems. Use dog- and cat-approved mosquito repellent products on pets, but note that not all products labeled for use on dogs can be used on cats (American Veterinary Medical Association website). Make sure window and door screens are intact (bug tight). Replace your outdoor lights with yellow (bug) lights.

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**Biological Control**

- Utilizes predators, both natural and introduced, to eat larvae and pupae
  - Mosquito fish
    - *Gambusia affinis*, *G. holbrooki* most common
    - *Fundulus* spp., *Rivulus* spp., killifish
- Other agents have been used but are not readily available
  - Fungus, protozoa, nematodes
  - Copepods

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Biological control involves using different predators that eat mosquito larvae and pupae. The mosquito fish, *Gambusia affinis* and *G. holbrooki* are the most commonly used supplemental control because they are easily reared. They are indiscriminate feeders and may eat other things, such as tadpoles, zooplankton, aquatic insects, and other fish eggs. Some naturally occurring fish such as *Fundulus* spp., *Rivulus* spp., and killifish play an important role in controlling mosquitoes in open marsh water and rotational impoundment management. There are other agents such as fungus, protozoa, and nematodes that have been tried but are not readily available. A predacious copepod, *Mesocyclops longisetus*, preys on mosquito larvae and is a candidate for local rearing

with *Paramecium* spp. for food.

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**Larvicides**

- Use when source reduction and biological control not feasible
- More effective and target-specific
- Less controversial than adulticides
- Applied to smaller geographic areas
  - Larvae concentrate in specific locations


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Larvicides are used when immature mosquito populations become larger than source reduction can manage or biological control can handle. They are often more effective and target-specific than adulticides, making them less controversial. They can be applied to smaller geographic areas than adulticides because larvae are often concentrated in specific locations, such as standing water.

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**Adulticides**

- When other control measures unsuccessful
- Least efficient
- Proper type and time of application helps efficacy
  - Ultra Low Volume foggers
    - 1 ounce per acre
  - Small droplets contact and kill adults



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Despite the efforts listed in previous slides, there are times when the environment prevails or humans are unable to prevent large swarms of mosquitoes. Adulticide use then becomes necessary. It is often the least efficient control program but ultra low volume spray either on the ground or aerially can reduce the population when the proper type and time of application is followed. Effective adult mosquito control with adulticides requires small droplets that drift through mosquito areas and come in contact with adults to kill them. Large droplets settle on the ground or vegetation and do not contact mosquitoes and may cause undesirable effects on nontargeted organisms. Insecticides are applied in a concentrated form at very low volumes such as 1 oz (29.6 mL) per acre. Excessive wind and updrafts reduce control, but light wind is necessary for drifting spray droplets.

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**Biosafety**

- Mosquito avoidance precautions
  - Bug spray, long sleeves, etc.
- Wear gloves or double plastic bags to collect dead birds
- Wash hands after handling
- Manipulate carcasses in biosafety cabinet when possible for necropsy

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This is a list of recommendations from Office of Laboratory Security, Canada Health Department for field investigations and handling dead animals and their tissues that may be infected with WNV.

Website: [www.hc-sc.gc.ca/pphb-dgspsp/ols-bsl/wnvbio\\_e](http://www.hc-sc.gc.ca/pphb-dgspsp/ols-bsl/wnvbio_e)

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### Additional Resources

- U.S. Department of Agriculture (USDA)  
– [www.aphis.usda.gov](http://www.aphis.usda.gov)
- Centers for Disease Control and Prevention (CDC)  
– <http://www.cdc.gov/ncidod/dvbid/westnile/index.htm>
- Center for Food Security and Public Health  
– [www.cfsph.iastate.edu](http://www.cfsph.iastate.edu)

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