IMPORTANCE OF MOSQUITOES

Portions of this chapter were obtained from the University of Florida and the American Mosquito Control Association Public Health Pest Control website at http://vector.ifas.ufl.edu.

Introduction to Pests and Public Health: Arthropods are the most successful group of animals on Earth. They thrive in every habitat and in all regions of the world. A small number of species from this phylum have a great impact on humans, affecting us not only by damaging agriculture and horticultural crops but also through the diseases they can transmit to humans and our domestic animals. Insects can transmit disease (vectors), cause wounds, inject venom, or create nuisance, and have serious social and economic consequences.

Arthropods can be indirect (mechanical carriers) or direct (biological carriers) transmitters of disease. As indirect agents they serve as simple mechanical carriers of various bacteria and fungi which may cause disease. As direct or biological agents they serve as vectors for disease-causing agents that require the insect as part of the life cycle. In considering transmission of disease-causing organisms, it is important to understand the relationships among the **vector** (the disease-transmitting organism, i.e. an insect), the disease **pathogen** (for example, a virus) and the **host** (humans or animals). The pathogen may or may not undergo different life stages while in the vector. Pathogens that undergo changes in life stages within the vector before being transmitted to a host require the vector—without the vector, the disease life cycle would be broken and the pathogen would die. In either case, the vector is the means for the pathogen to pass from one host to another.

Mechanical transmission of disease. Mechanical transmission of disease pathogens occurs when the vector transports organisms, such as bacteria that cause dysentery, on their feet, body hairs, and other body surfaces to the host. There is no multiplication or development of the pathogen within the vector's body. The house fly, *Musca domestica*, is a mechanical (also known as passive) transmitter of bacillary dysentery. Mechanical transmission can be considered more or less accidental.

Biological transmission of disease. In this mode of transmission, an arthropod acquires the pathogen from one host. The pathogen then develops in the arthropod's body and is transmitted to another host. If the pathogen or **parasite** undergoes the sexual portion of its life cycle in a host, that host is the **primary** or **definitive host**, as in the mosquito that harbors malaria. For malaria, a human is the **intermediate host** in which the asexual stages of the parasite are found. Some diseases may require several hosts before completing the cycle of pathogen transmission. The hosts may be different species in different stages of the life cycle.

Obligatory vectors. Obligatory vectors of disease are those in which the pathogen develops from one stage to another. Malaria is an example of a disease that must be taken up by a vector (mosquito) in order for the pathogen (malaria) to develop from one stage to another. Without it the malaria parasite would die. Mosquitoes are often obligatory vectors of diseases.

Reservoir hosts. Reservoirs are defined as one or more host species that harbor a disease-causing pathogen over an extended period of time without showing symptoms of the disease. Disease transmission occurs when a vector feeds on a host that has the pathogen. After acquiring the pathogen, the vector then transmits it at a later date while feeding on another susceptible host. Birds are an example of reservoirs for mosquito-borne encephalitis that may affect humans and horses or other dead-end hosts that are not normally part of the transmission cycle. In many cases the disease is **endemic**, that is, it exists continually in reservoirs in a geographically defined area. Diseases that cycle in nature to non-reservoir hosts without involving man are called **zoonoses**. When a disease outbreak occurs in these non-human animals, it is called an **epizootic**.

Some vectors pass diseases to their offspring through eggs. This is known as **transovarial** transmission. When a female is infected with the disease organism, her eggs also become infected and the resulting larvae will carry the disease. The subsequent adult stage can then transmit the pathogen without needing to feed on an infected host. This occurs with several mosquito-borne viruses and with *Rickettsia rickettsii*, the bacterium causing Rocky Mountain spotted fever. In this instance the tick and the mosquito serve both as reservoirs and vectors.

Myiasis. Infestation of an animal, including man, by living maggots deposited on the skin or in a wound by flies is termed myiasis. Some insects, such as bot flies, breed solely in this manner. Screwworm infestations of livestock and occasionally humans are examples of this phenomenon. In the United States the primary screwworm fly has been eradicated, but the less economically important secondary screwworm fly still invades the flesh of wild animals such as deer.

Skin Irritation. Insects that cause skin irritations to humans include the puss, io moth, and saddleback caterpillars. These larvae have urticating hairs rather like those of stinging nettle plant. Cantharidin, present in the blood of blister beetles, can cause painful blistering when the beetle is crushed on the skin surface. These are not insects of direct public health concern, but are often topics of public enquiry.

Envenomization and Allergies. Many insects, some spiders, scorpions, and centipedes, have developed poisoning mechanisms to use in self-defense or in paralyzing their prey. Stings and bites may be intensely painful and irritating to humans but seldom cause death. Probably the most dangerous is the bite of the black widow spider, *Latrodectus mactans*. The brown recluse spider, *Loxosceles reclusa*, may inflict a serious bite, often resulting in so much dead tissue that skin grafting is needed. There have been deaths reported from both spiders. Stings of bees and wasps may be serious or even fatal to persons highly allergic to their venoms.

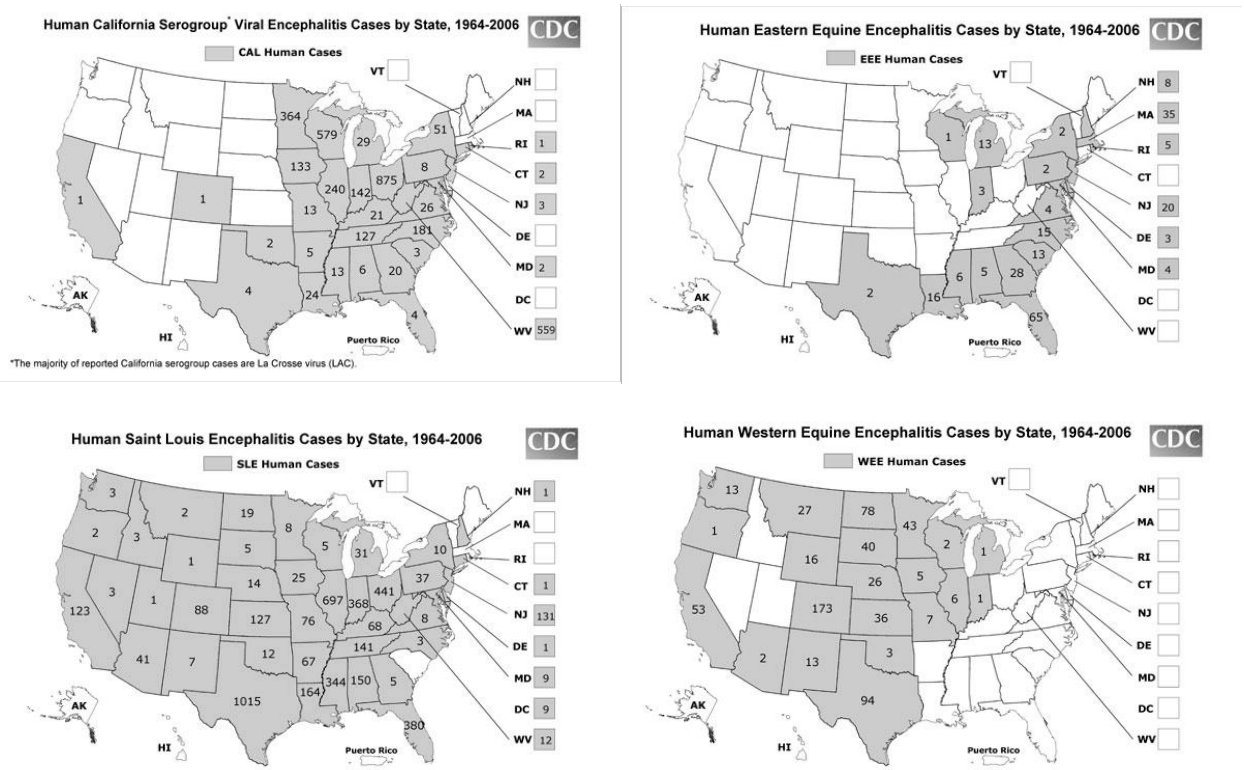
MOSQUITO-BORNE DISEASES

Aside from the irritation and annoyance that mosquitoes inflict on humans and livestock alike, the threat of disease pathogen transmission is always present. Mosquitoes are potent vectors of three types of organisms pathogenic to man and animals. These are (1) the plasmodia, causal organisms of the malarias, belonging to the Protozoa; (2) filarial worms of the genera *Wuchereria*, *Brugia* and *Dirofilaria,* the causal organisms of human lymphatic filariasis and dog heartworm; (3) viruses, especially the arboviruses causing such important diseases as yellow fever, dengue fever, and the encephalitides (Western Equine encephalitis, Eastern Equine encephalitis, St Louis

encephalitis, California serogroup viruses and West Nile virus). Of these diseases, West Nile virus represents the greatest perennial threat in the United States, whereas malaria and dengue are occasionally imported by travelers, and yellow fever is now quite rare due to the availability of a highly effective vaccine.

The word "encephalitis" means an inflammation of the brain and possibly the spinal cord (encephalomyelitis). There are several possible causes for this inflammation, including mosquito-transmitted viruses, commonly referred to as arthropod-borne or arboviruses.

Distribution of Arboviral Encephalitis Cases in the US, 1964-2006 *



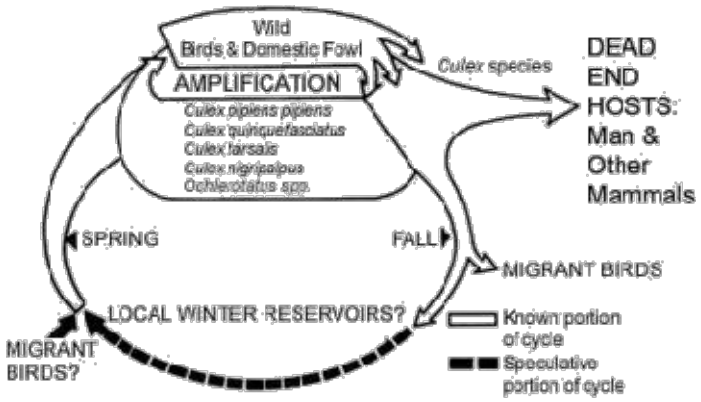
* West Nile Virus maps are in the WNV section.

St. Louis Encephalitis (SLE). This disease is caused by a flavivirus that has a natural transmission cycle involving several species of wild birds and a few species of *Culex* mosquitoes. The virus is briefly present in the blood of infected birds, and mosquitoes ingest the virus when taking a blood meal. After one to two weeks at summer temperatures, or longer at cooler temperatures, the virus multiplies and spreads to the mosquito salivary glands. It is then inoculated into the next host when the infected mosquito feeds.

Once infected, the mosquito can transmit the virus each time it takes, or attempts to take, a blood meal for the duration of its life. In the vertebrate host, usually a bird, the virus again reproduces and must reach a certain concentration in the blood to infect other susceptible mosquito species. In most cases, this level is reached less than a week after the infective bite occurs and lasts for only three to four days, after which the virus in the host blood is reduced below the level required

to infect the mosquito. To become infected and later transmit the virus, a mosquito must feed on the infected vertebrate during this three- to four-day period.

In some species of birds the virus level that develops is much higher than in other species. Those with higher levels are the most important sources of infection for mosquitoes. Also, some mosquito species are especially receptive and can become infected when feeding on blood with relatively low virus levels. These species are the most important in transmitting the virus to vertebrates. The SLE virus does not multiply in some refractory mosquito species and so is not transmitted even though the mosquito has fed on an infected bird. Also, the virus does not develop to high enough levels in the blood of some vertebrates to infect even the most susceptible mosquito species. These vertebrates are called "dead-end hosts" because the virus cannot be acquired from them by susceptible mosquitoes and transmitted to other vertebrates. Humans, non-human mammals, and some bird species are "dead-end hosts" for SLE. There is no person-to-person transmission. Vertebrates, birds, humans or other mammals infected with SLE virus do not always exhibit disease symptoms. Birds, even those that develop high levels of virus in their blood, may not exhibit any symptoms.



Life cycle of St. Louis encephalitis

The reaction of humans infected with SLE virus is quite variable and dependent on age, general health and other poorly understood characteristics. Infected humans may experience subclinical (inapparent) symptoms or the onset of fever, nausea, and vomiting with severe headaches within five to seven days, and severe central nervous symptoms that may produce temporary or permanent physical or mental disabilities, or even death. In general, the fatality rate is higher and the symptoms more severe in people over 50 years of age. In past epidemics, the fatality rates have ranged from four to 20 percent with most deaths occurring in the older age groups. Studies conducted in areas where epidemics have occurred indicate that for each person who developed symptoms of SLE virus infection (ranging from mild to severe), from 80 to 800 people had been infected with the virus but did not develop disease symptoms. The older age groups consistently exhibited the highest number and most severe symptoms of those infected during these epidemics.

In eastern and midwestern United States, epidemics of SLE have occurred in relatively densely populated urban and suburban areas in which suitable bird and mosquito species live in close association with humans. *Culex pipiens*, the northern house mosquito, is thought to be a primary

vector of SLE in areas where there are (1) many water-filled artificial containers that provide ideal breeding sites for mosquitoes, and (2) populations of birds, such as sparrows and starlings that live and nest in or near human residences. Water-holding structures commonly associated with humans (for example, storm sewer catch basins, cesspools, sewage lagoons, open septic tanks, polluted drainage ditches and water runoff impoundments, failed septic systems, drain fields and effluent from sewage disposal plants) also can produce huge numbers of these mosquitoes during the warm months of the year. But rural areas are also highly susceptible to transmission because pastures and other extensive temporary fresh-water breeding sites can produce large populations of non-domestic avian-feeding mosquitoes, e.g., *Culex nigripalpus*. In New Mexico and the western U.S., *Culex tarsalis* is considered the most important vector.

The probability of SLE transmission occurring is in direct proportion to the number of SLE virus infected birds and the abundance of *Culex* mosquitoes. Mosquito agencies monitor virus activity by sampling the blood of wild birds or, more commonly, sentinel birds caged in the natural habitat. SLE outbreaks may be more closely related to water accumulation patterns than to total rainfall (for example, drought conditions cause polluted streams to pool and produce *Culex* mosquitoes) and are most likely to occur from mid-summer to early fall, with case detection continuing until cold weather reduces mosquito abundance. SLE occurs throughout the United States; from 1964 through 2006 the number of reported cases annually has ranged from 2 to 1,967 (in 1975) for an average of 111 cases per year. Florida had an outbreak in 1990 resulting in 223 cases, and a localized outbreak occurred in northeastern Louisiana in 2001, resulting in 70 cases. In New Mexico, cases of SLE are sporadic rather than epidemic.

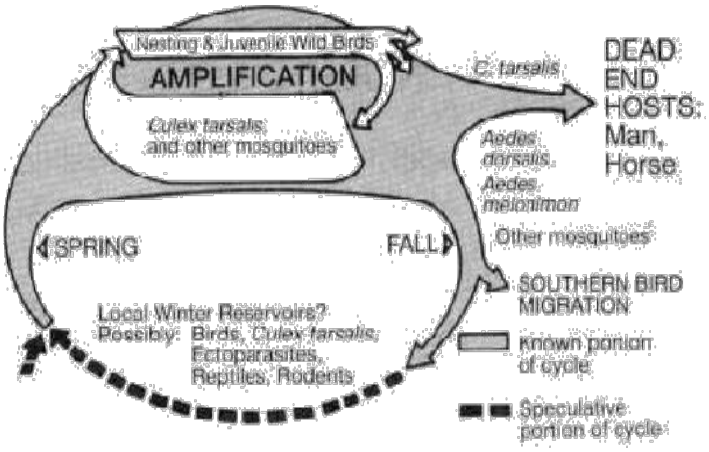
In temperate zones, *Cx. pipiens* females overwinter in locations that are protected from weather extremes such as outbuildings, attics, storm sewers and rodent burrows. All other stages are killed when exposed to freezing temperatures. After overwintering, these females lay eggs in rafts of 40 to 500 on the surface of organically polluted water. At summer temperatures, eggs hatch in one to two days and development to adult is completed in eight to 10 days, whereas in the cooler temperatures of spring and fall this development might require two or more weeks. Mosquito breeding occurs throughout the warm months of the year, and populations typically increase throughout the summer reaching their peak in late August. This species feeds primarily on birds, but there is some evidence that it may change its feeding habits from mid-August to September and feed on other animals, including humans. This, and favorable meteorological conditions, may explain in part why human outbreaks of SLE normally occur in late summer.

It is not known how SLE virus is maintained in nature during the winter months when vector mosquitoes do not actively feed and animal infections are not detected. There is some evidence that the virus can remain viable in overwintering *Cx. pipiens* adults and, presumably, be transmitted when they feed on susceptible vertebrates in the spring. Whether this is the primary overwintering mechanism is not yet known.

Western Equine Encephalitis (WEE). Western equine encephalitis is an alphavirus found mainly in the states west of the Mississippi River, and the upper Midwest states of Wisconsin, Illinois, Indiana and Michigan. Major, widespread outbreaks of WEE have occurred in the western parts of the United States and Canada. This disease occurs primarily in rural locations and has a natural transmission cycle that involves both mosquitoes and wild birds. *Culex tarsalis* and

Aedes melanimon are the most important vectors throughout the western United States and birds are the major host. East of the Mississippi River, *Cx. quinquefasciatus* is the suspected vector. A vaccine for horses is available, but no vaccine is available for humans.

Life Cycle of Western Equine Encephalitis



Western equine encephalitis can produce severe disease in both humans and horses, but both of these are "dead-end" hosts and are not involved in further transmission of the virus. Human WEE cases are usually first seen in June or July. Most WEE infections are asymptomatic or present as mild, nonspecific illness. Patients with clinically apparent illness usually have a sudden onset with fever, headache, nausea, vomiting, anorexia and malaise, followed by altered mental status, weakness and signs of meningeal irritation. Children, especially those under 1 year old, are affected more severely than adults and may be left with permanent sequelae, which is seen in 5 to 30% of young patients. The mortality rate is about 3%.

***Culex tarsalis*, the western encephalitis mosquito**

Infected wild birds infect the mosquitoes that feed on them, but the WEE virus is present in the infected bird's blood for only three to four days in the amounts needed to infect the feeding mosquito. Birds infected with WEE normally do not show any symptoms. Although several mosquito species are able to transmit this virus, *Cx. tarsalis* has been the major vector in past outbreaks of this disease. This species usually breeds in both permanent and temporary ground water with high organic content, such as sewage effluent. In many areas of the West, the species is most numerous where improper agriculture irrigation practices result in water accumulations. This mosquito feeds readily on both birds and mammals, so it serves as the major vector in the natural transmission cycle as well as transmitting WEE virus to humans and horses. As with SLE, it is not presently known how this virus is maintained in areas of the United States and Canada where adult mosquitoes are not active during the cold months of the year.

From 1964 through 2006, there were 640 reported cases of WEE in the U.S. The last widespread WEE activity in the West occurred in 1987. In that year New Mexico had one human case and 17 horse cases. Since 1987, there have only been five cases of WEE in the U.S.

Eastern Equine Encephalitis (EEE). Eastern equine encephalitis is an alphavirus maintained in nature by a bird-mosquito-bird cycle similar to SLE. It is distributed along the coastal states of the Gulf of Mexico and Atlantic Seaboard and occasionally occurs in the Midwest. Annual cases in the U.S. range from 0 to 15, for an average of 5 cases per year. Georgia, Louisiana, Massachusetts, and North Carolina reported cases of EEE in 2006.

Virus activity is confirmed by encephalitis monitoring programs conducted by mosquito control districts. These agencies periodically capture wild birds or maintain sentinel chicken flocks and collect blood samples for laboratory analysis to detect antibodies that were produced after they were infected by the EEE virus. High antibody levels in young birds indicate recent circulation of the virus, whereas antibodies in older birds might be the result of infection the previous year or earlier. Periodic outbreaks among non-vaccinated horses also provide evidence of local EEE viral activity. Equines are involved as "dead-end" hosts in the cycle along with man. Vaccines are available for horses and annual booster shots are recommended. There are no vaccines available for human use. EEE is more prevalent in horses and susceptible domesticated birds such as emus than in man and often produces fatal outcomes. Deaths due to EEE viral infections have been reported in red-winged blackbirds, house sparrows, whooping cranes and pheasants.

EEE can infect persons of any age, but young children and infants are particularly vulnerable to the disease. Seldom are more than 20 or 30 human cases reported nationally in any given year, although localized foci of human cases do occur. Mortality rates in human infections may exceed 50 percent. In fatal cases, death usually occurs within 48 to 72 hours after the onset of illness. Children and infants who survive are frequently afflicted with varying degrees of mental retardation and residual paralysis.

There are a number of mosquito species, especially *Culiseta melanura*, that are capable of maintaining and transmitting EEE to birds in nature. Since *Cs. melanura* is relatively uncommon and seldom bites man, other mosquitoes such as *Cs. inornata*, *Cx. quinquefasciatus* (southern house mosquito), *Aedes albopictus* (Asian tiger mosquito), *Ae. sollicitans* (tan salt marsh mosquito), *Ae. vexans*, *Ae. infirmatus*, *Ae. atlanticus*, and *Coquillettidia perturbans* are

important in transmission from birds to man and/or equines. Factors favoring infections in man are the buildup of virus in wild-bird populations and a high density of adult mosquito vectors.

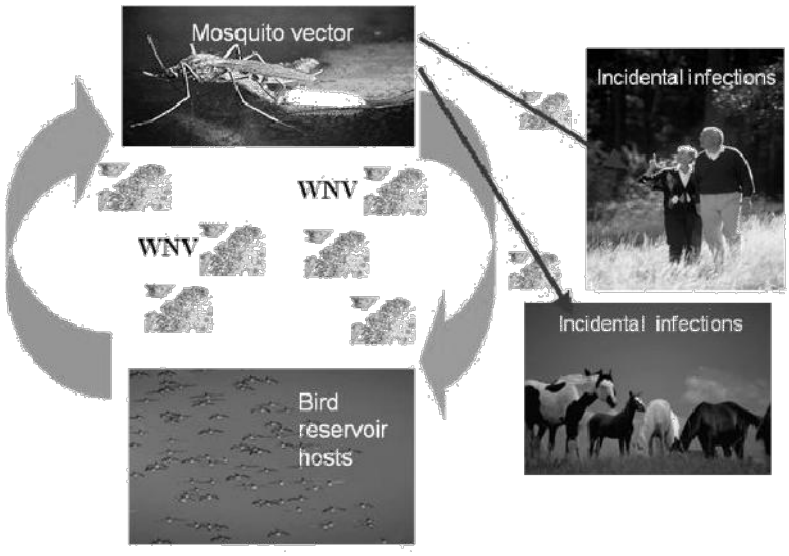
California (CE), LaCrosse (LAC), and Jamestown Canyon (JC) Encephalitis. These viruses of the California serogroup in the family Bunyaviridae are reported sporadically, averaging 80 cases per year in the US. Most cases are due to LaCrosse virus and occur primarily in Ohio, Indiana, Illinois, Minnesota, Wisconsin and West Virginia in children under 16 years of age. The disease is less severe than SLE and EEE, with a morality rate of less than 1%, but infected children often experience learning disabilities or personality changes. The natural cycles differ from SLE, EEE, and WEE in that natural hosts are small and medium-sized animals such as rabbits, hares, chipmunks and squirrels rather than birds. The vectors are mostly woodland *Aedes* mosquitoes, and most of the human cases have occurred in people who lived or worked in or near wooded areas. However, LAC virus seems also to be associated with container breeding mosquitoes, such as *Ae. triseriatus*, which readily maintain LAC virus in a transovarial transmission cycle. These viruses have been isolated from a number of species of woodland mosquitoes, particularly *Ae. triseriatus*, *Ae. canadensis*, *Ae. melanimon*, *Ae. trivittatus*, *Ae. stimulans* and *Ae. atlanticus*.

Venezuelan Equine Encephalitis (VEE). Like EEE and WEE viruses, Venezuelan equine encephalitis (VEE) is an alphavirus and causes encephalitis in horses and humans and is an important veterinary and public health problem in Central and South America. Occasionally, large regional epizootics and epidemics can occur resulting in thousands of equine and human infections. Epizootic strains of VEE virus can infect and be transmitted by a large number of mosquito species. The natural reservoir host for the epizootic strains is not known. A large epizootic that began in South America in 1969 reached Texas in 1971. It was estimated that over 200,000 horses died in that outbreak, which was controlled by a massive equine vaccination program using an experimental live attenuated VEE vaccine. There were several thousand human infections. A more recent VEE epidemic occurred in the fall of 1995 in Venezuela and Colombia with an estimated 90,000 human infections. Infection of man with VEE virus is less severe than with EEE and WEE viruses, and fatalities are rare. Adults usually develop only an influenza-like illness, and overt encephalitis is usually confined to children. Effective VEE virus vaccines are available for equines.

Enzootic strains of VEE virus have a wide geographic distribution in the Americas. These viruses are maintained in cycles involving forest dwelling rodents and mosquito vectors, mainly *Culex* species. Occasional cases or small outbreaks of human disease are associated with these viruses; the most recent outbreaks were in Venezuela in 1992, Peru in 1994 and Mexico in 1995-96.

West Nile Virus. West Nile virus (WNV) is a flavivirus very similar to SLE. It was unknown in the Americas until 1999, when it appeared in the northeastern United States. It is unknown how the virus came to the North American continent. The most serious manifestation of West Nile virus infection is fatal encephalitis in humans and horses, as well as mortality in certain domestic and wild birds, particularly those in the Corvid (crow) family. Persons older than 50 years have the highest risk of severe disease. The virus, also found in parts of Eurasia and Africa, is found in birds and transmitted to humans primarily by *Culex* mosquitoes.

West Nile Virus Transmission Cycle



As in SLE, the majority (estimated at 80%) of people who are bitten by a mosquito with West Nile virus are asymptomatic. In those who do become ill, symptoms of the disease usually appear within 3 to 15 days after the bite of an infected mosquito. Most symptomatic infections manifest as West Nile fever, and symptoms include fever, headache and body aches, occasionally with skin rash and swollen lymph glands. In one California study, the median duration of symptoms of people with West Nile fever was one to two weeks, but the median time until patients reported they were "100 % back to normal" was 47 days. Some patients with West Nile fever report lingering symptoms such as headache, fatigue, and concentration problems.

About one percent of all people with WNV develop severe infection of the central nervous system. This is called West Nile neuroinvasive disease (WNND) and may result in encephalitis, meningitis, and/or acute flaccid paralysis. Symptoms of neuroinvasive disease include headache, high fever, neck stiffness, stupor, disorientation, coma, tremors, convulsions, muscle weakness, and paralysis. About 10% of WNND cases are fatal. Survivors of WNND sometimes experience long-lasting effects (up to a year or more) such as tremors, persistent headaches, fatigue, weakness, depression, slurred speech, and personality changes.

Although the majority of cases are acquired from the bite of an infected mosquito, transmission of West Nile virus has been proven to occur through organ transplants, blood transfusions, breastfeeding, and transplacentally. Two lab workers also acquired West Nile virus after receiving cuts on their hands while working with the virus. Testing by blood banks for viremic blood donors was initiated in 2003. In 2005 there were no transfusion-transmitted cases of WNV in the U.S., and in 2006 there were two. Organ donor testing was begun in 2005 to reduce the future risk of transplant transmission.

The maps on pages 11 and 12 show the introduction and spread of West Nile virus in the U.S. beginning in the fall of 1999 when 62 human cases with 7 deaths occurred in the New York City area following a widespread die-off of American crows in the region. Horse deaths were also

reported. In 2000, another 21 cases with two deaths were reported in New York, New Jersey and Connecticut. Increased surveillance along the entire eastern seaboard confirmed the spread of WNV to several states. Crows or other birds that had died of WNV were reported from as far north as New Hampshire and down to North Carolina. Research into possible mosquito vectors implicated not only *Culex* species; *Aedes* species were also possibly involved in the transmission of the virus.

During 2001 West Nile virus expanded its range southward by 750 miles to the tip of Florida and westward 800 miles to the western border of Arkansas. A total of 66 human cases with 9 deaths were reported from 10 states. Over 16,000 dead birds were tested, of which 44% were positive. Birds in the Corvid family made up 71% of the positive birds.

The table below shows the dramatic increase in annual cases of West Nile Virus beginning in 2002.

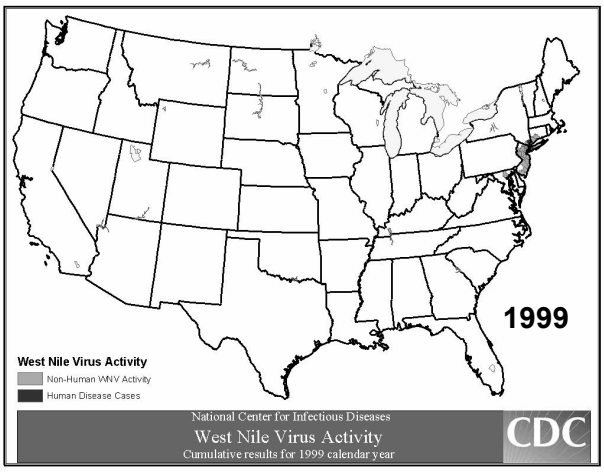
Reported West Nile Virus Disease Cases in Humans
United States, 1999 - 2007

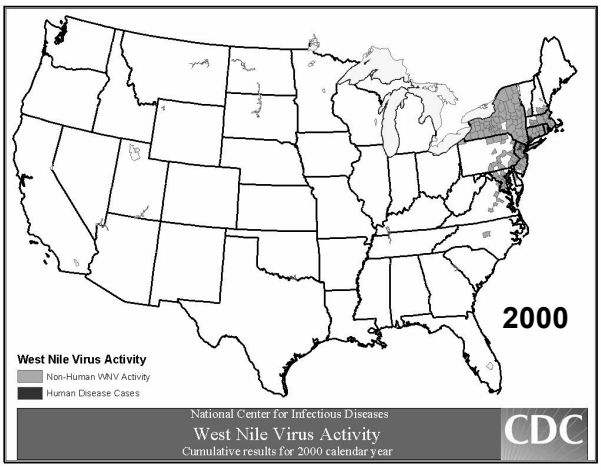
Year	Total	WNND	WNF/other	Deaths
1999-2001	149	142	7	18
2002	4,156	2,946	1,210	284
2003	9,862	2,866	6,996	264
2004	2,539	1,148	1,391	100
2005	2,949	1,288	1,661	116
2006	4,269	1,459	2,810	177
2007	3,576	1,187	2,389	115
Total	27,500	11,036	16,464	1

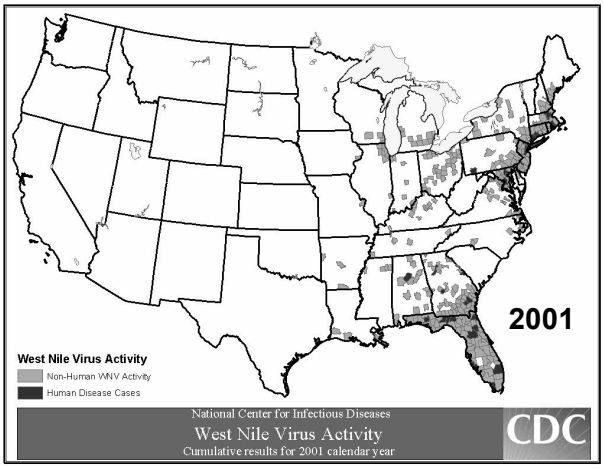
Over 14,000 horses were also diagnosed with West Nile virus in the U.S. in 2002. About 30% of the ill horses died or were euthanized. Symptoms in horses are: weakness, usually in the hindquarters; ataxia (muscle incoordination); paralysis; head tilt; circling; fever; behavioral changes; loss of appetite; and depression. An equine vaccine became fully licensed in February 2003. The vaccine should be given intramuscularly 3 to 6 weeks apart before the mosquito season, as it takes time for the antibodies to build up to a protective level. Yearly boosters are required.

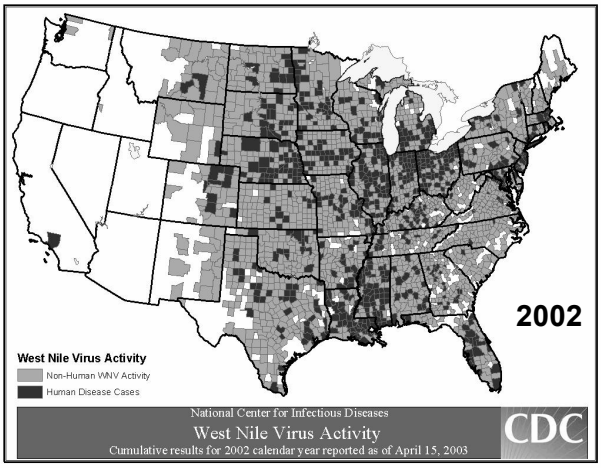
By the end of 2005, West Nile virus activity, whether in humans, horses, birds or mosquitoes, had been reported from all of the contiguous 48 states. Human cases were reported from 42 states with onset in every month of the year. The highest incidence of West Nile neuroinvasive disease was in the Western and Central states, and California reported the highest number of cases, 928.

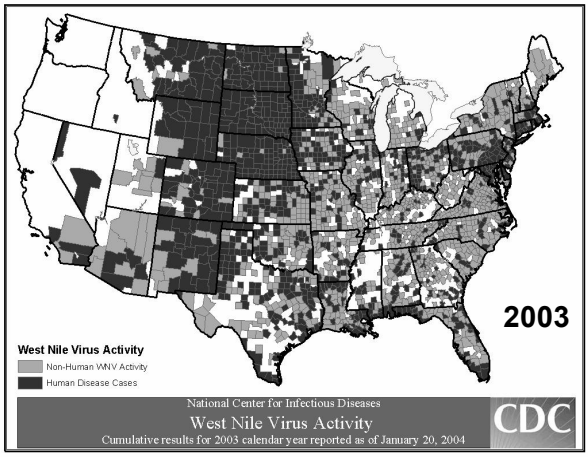
Progression of West Nile Virus in the U.S., 1999 - 2004

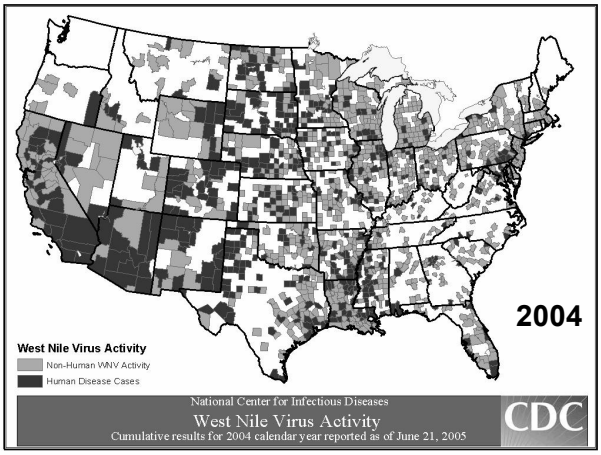




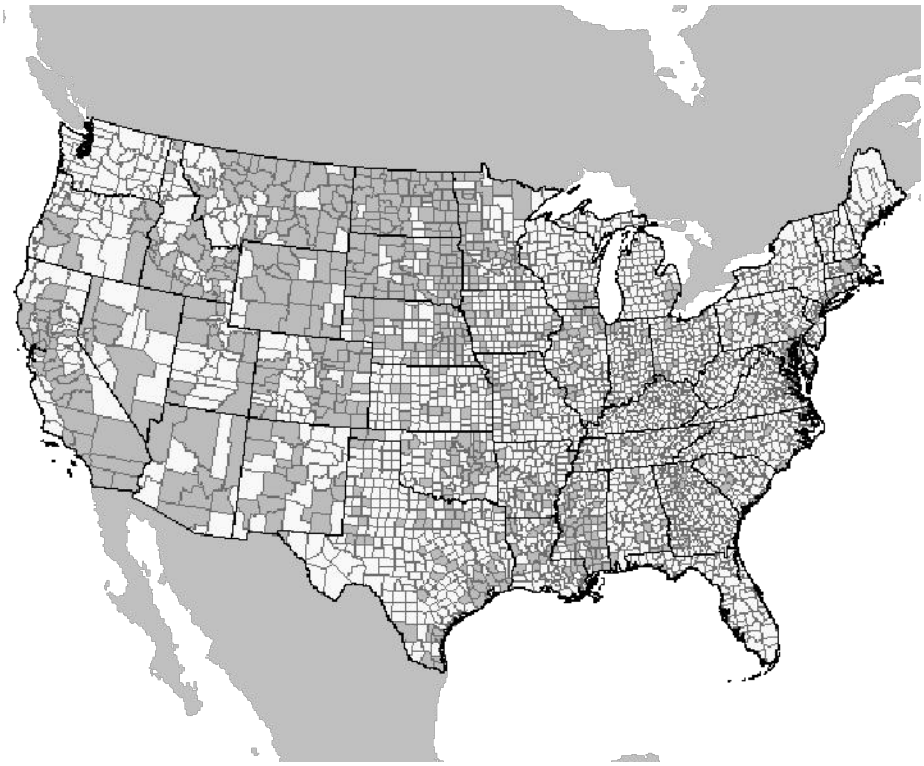








U.S. counties reporting West Nile Virus activity in 2007



West Nile Virus Surveillance in New Mexico, 2002 -- 2007. Surveillance methods include testing mosquito pools for the presence of the virus, and active surveillance for human and equine cases. Horse cases were the first indication of the presence of West Nile virus in the state in 2002, and 78 equine cases were reported from 14 counties, mostly east of the Rio Grande. While dead bird surveillance has proved useful in many areas of the country with high crow populations, it has not proven to be an effective surveillance tool in New Mexico. In 2002, only two birds tested positive for West Nile virus, a crow from Sandoval County and a blue jay from Chaves County.

Several mosquito control programs began collecting mosquitoes for arbovirus surveillance in 2002. Over 7000 mosquitoes were collected and sent to the state lab for testing for West Nile, Western Equine and St. Louis encephalitis viruses. Although none of the mosquitoes tested positive for West Nile virus, six pools were positive for WEE virus (from Curry, Roosevelt, Chaves, Sierra and Doña Ana counties).

New Mexico's first human case of West Nile virus had onset in July, 2003, and by the end of the year 209 human cases from 24 counties had been confirmed. Seventy-four cases were classified as neuroinvasive disease with either meningitis or encephalitis. There were four fatalities, ranging in age from 76 to 88 years. Over 400 horses were diagnosed with WNV in the state in 2003 from all but three counties, demonstrating that WNV was well established throughout the state.

2003: Seventy-nine mosquito pools from 14 counties were positive for WNV out of a total of 13,317 mosquitoes tested. The greatest number of positive pools was collected during the first

week of August. Twenty-two species of mosquitoes were collected and tested, but only three species, *Culex tarsalis*, *Culex quinquefasciatus*, and *Culex salinarius*, tested positive for WNV.

2004: The number of human cases in the state dropped to 88, of which 32 were neuroinvasive disease. Cases were reported from 16 counties; San Juan County had more than a third of reported cases (34). There were four fatalities, ranging in age from 68 to 82. The first case of the year had onset May 13, nearly two months earlier than the previous year. The first human cases preceded the first positive mosquito pool by five weeks.

During 2004, 128 mosquito pools from 11 counties were positive for WNV out of 71,663 mosquitoes tested. It was not until the second week of June that the first WNV positive mosquito pool was recovered; the greatest numbers of positive pools were reported during the last week of July. Twenty-seven species of mosquitoes were collected and tested, and ten species tested positive for WNV. Four species were significant in that they accounted for 89% of the mosquitoes tested and 87.5% of the positive mosquito pools. They were *Aedes vexans*, *Culex tarsalis*, *Culex quinquefasciatus*, and *Culex salinarius.*

2005: New Mexico reported 33 human cases of WNV from 12 counties. Twenty cases were classified as neuroinvasive disease and there were two fatalities. The median age of fever cases was 56 years, with a range of 22-66. The median age of neuroinvasive disease was 48.5 years, with a range of 21-78. This differs from previous years, when the median age of WNND was higher than that of fever cases. The two fatal cases were aged 50 and 78 years. As in previous years, the peak of human cases was mid-August. A total of 114,528 mosquitoes were tested from 15 counties. Of these, 75 mosquito pools from 8 counties tested positive for WNV.

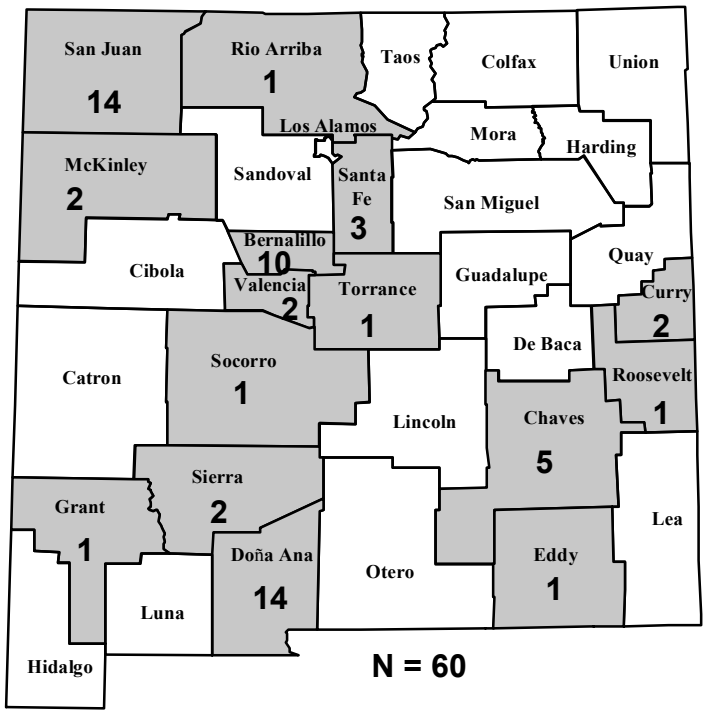
Thirty-eight species of mosquitoes were collected and tested, and nine species were positive for WNV. The positive species were: *Aedes vexans*, *Anopheles franciscanus*, *Culex erythrothorax*, *Cx. quinquefasciatus*, *Cx. salinarius*, *Cx. tarsalis*, *Ae. dorsalis*, *Ae. trivittatus*, and *Psorophora columbiae*. In addition to these, 1 pool of unidentified *Culex* species, and five pools of unidentified mosquitoes tested positive for WNV. The most significant species were *Culex tarsalis* and *Culex quinquefasciatus*; these two species comprised 39% of the total number of mosquitoes tested, and 73% of the positive pools. The earliest positive mosquito pool was collected five weeks before the first human case onset, at the end of April, and the last positive mosquito was collected in the third week of September.

2006: New Mexico reported 8 human cases of West Nile virus with a single fatality. These cases came from Roosevelt (1), Doña Ana (3), and San Juan (4) counties. Week of illness onset ranged from June 30 to October 8. Five of the cases were uncomplicated fever with a median age of 34; the median age of the three neuroinvasive cases was 49. Two horses tested positive for the virus, one in Santa Fe County and one in Otero County. In addition, one positive bird was reported from Colfax County and one from Chaves County. 25,367 pools of mosquitoes were tested from 14 different counties. Of these, 18 pools tested positive, with the first positive pool collected the second week of July. Mosquito populations peaked in early August, and West Nile rates were highest in late August. Eighteen species of mosquitoes were tested, but only *Culex tarsalis*, *Cx. quinquefasciatus*, and *Cx. salinarius* were positive for WNV.

Mosquitoes submitted to the state lab for arbovirus testing are also tested for Western Equine Encephalitis virus and St. Louis Encephalitis virus. Six pools of mosquitoes were positive for WEE: four pools of *Culex tarsalis*, and one each of *Culex salinarius* and *Aedes dorsalis*. These mosquitoes were collected from Bernalillo, Doña Ana, Sandoval and Sierra counties. There have been no more pools positive for SLE or WEE since 2005.

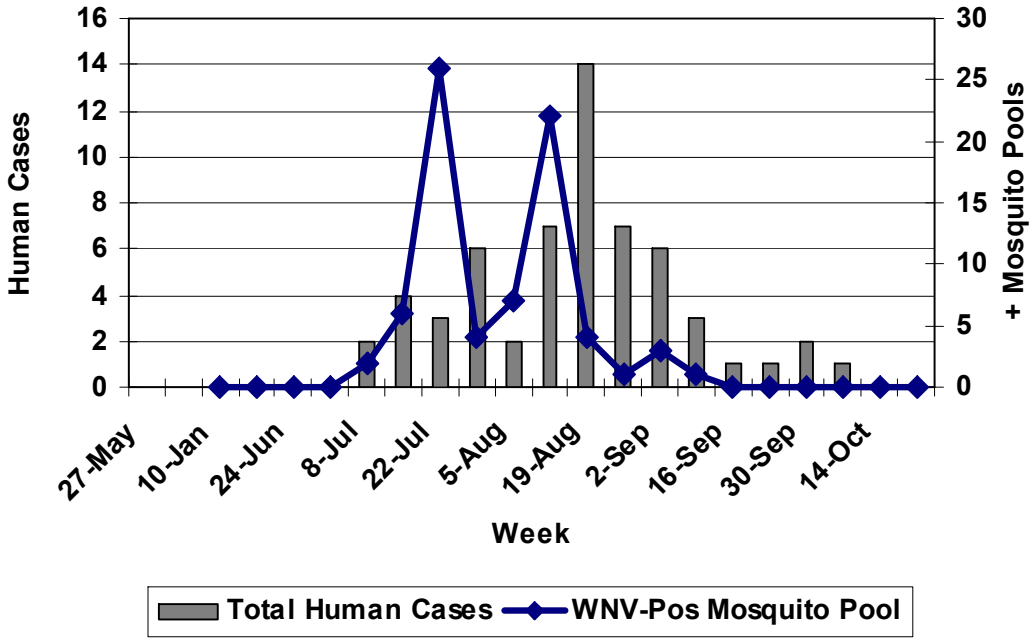
2007: This year showed an increase in rates of human West Nile illness for the first time since the introduction of the virus to New Mexico, with a total of 60 cases from 15 counties. Twenty-one of these cases were uncomplicated fever, with a median age of 46. The median age of the 39 neuroinvasive cases was 65. Three people died. In addition to the 60 people who became ill, three asymptomatic blood donors were detected. Seven of the nine counties where mosquitoes were collected and tested had West Nile virus positive mosquito pools, for a total of 76 positive pools out of 13,933 individual mosquitoes tested. Mosquito abundance and number of West Nile positive pools both peaked in mid-August. Nine mosquito species were tested; of these only *Aedes vexans*, *Culex quinquefasciatus*, and *Cx. tarsalis* pools were positive. Sixteen cases of West Nile infection in horses were reported from 12 counties.

Human WNV Cases by County, New Mexico, 2007

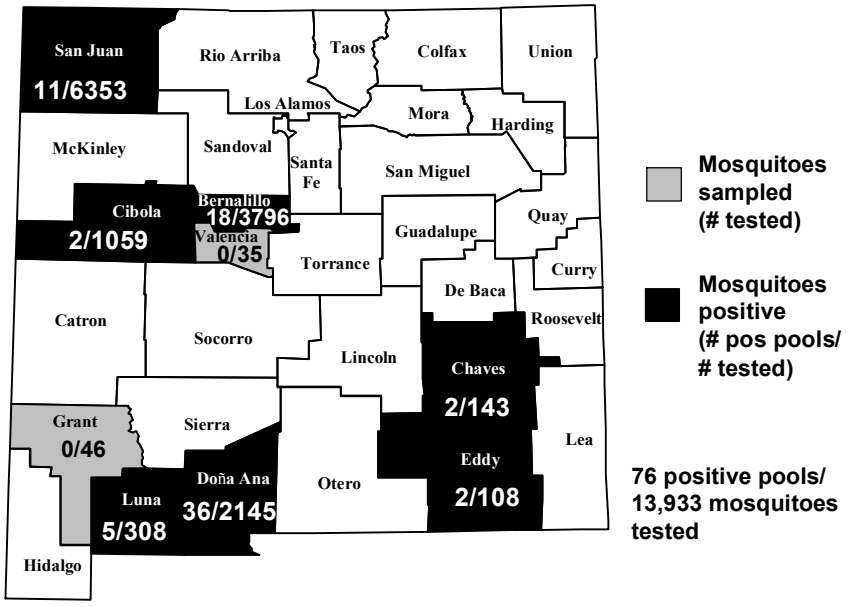


12/17/07; N = 60 cases

WNv Human Cases and Positive Mosquito Pools by Week, NM 2007



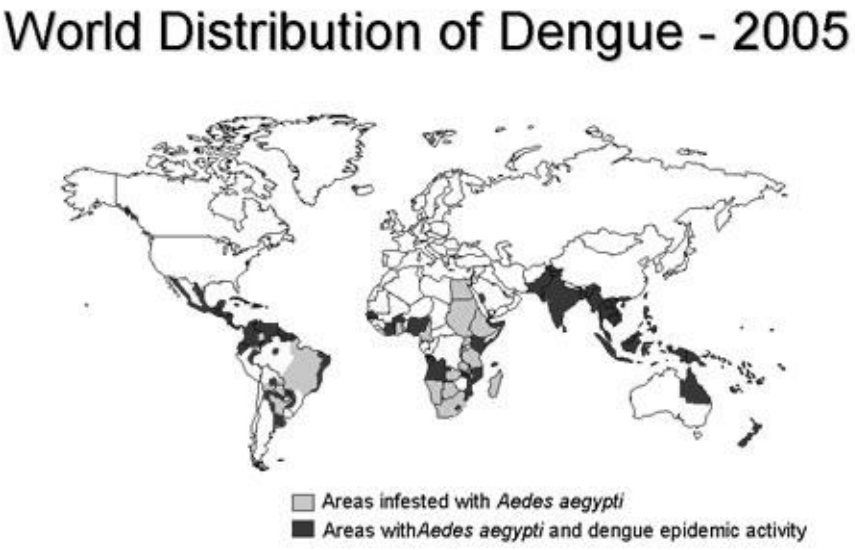
West Nile Virus Mosquito Surveillance, 2007



Equine surveillance for West Nile virus has become much less useful than it was when the virus first invaded New Mexico. Rates of illness in horses have dropped from hundreds (in 2003) to dozens (2004 to present). We speculate that the vastly reduced number of reported cases may be due to 1) increased use of the WNV equine vaccine; 2) veterinarians not submitting blood samples once they recognize signs of WNV in a horse; and 3) increasing natural immunity in the equine population.

Arbovirus surveillance in humans, horses, and mosquitoes is ongoing. When looking at states that have had WNV for several years, some have shown a large decrease in human cases after having a large number of cases the previous year, while others have had even larger numbers of cases the second or third year after the virus was first detected in their state. Our public health message continues to encourage prevention of West Nile virus by use of effective insect repellents, avoidance of outdoor activities if possible when mosquitoes are active, elimination of standing water around the home, and encouraging integrated mosquito management by community mosquito control programs.

Dengue. Dengue is a viral disease, commonly called "breakbone fever." It is often characterized by severe headache, pain behind the eyes, high fever, backache, pain in the joints, and a severe rash, with convalescence that may require several weeks. These symptoms generally occur five to six days after an infected mosquito has bitten a susceptible person. In uncomplicated cases, death rarely occurs. However, four strains of dengue virus are recognized, each of which produces life-long immunity against the infecting virus in humans after infection. Exposure to infection by a second strain of dengue virus in an already immune individual may result in a more severe form of dengue known as dengue hemorrhagic fever (DHF) with accompanying dengue shock syndrome (DSS). Increased dengue hemorrhagic fever has been experienced in the Western Hemisphere in the last 20 years, with outbreaks occurring in the Caribbean region.



Dengue epidemics were common in the 1800's and early 1900's in the United States. Before 1980 the last major epidemic of dengue in the continental U.S. occurred in Louisiana in 1945 with 62 confirmed cases, but authorities estimated that there were probably several hundred that were unapparent and/or unreported cases. However, in 1980, Texas recorded 23 locally acquired cases of dengue and new cases have occurred sporadically until 1999 when 18 cases were reported. Although not endemic now in most of the United States, potential dengue vectors are present in the southern United States and the virus is commonly imported in persons entering the country from endemic areas of the tropics. Conditions for dengue transmission are present, but it is unlikely that this would occur at the levels observed at the turn of the last century, when hundreds or thousands of cases are estimated to have occurred in some major cities. Presently, there is no vaccine available to prevent dengue infections.

Dengue is transmitted in the U.S. by *Ae. aegypti* in a mosquito-man-mosquito cycle. (Other potential vectors are *Ae. albopictus* and *Oc. japonicus*). This species is found in close association with humans, breeding in natural and artificial water-holding containers around dwellings. After biting an infected person, the female mosquito requires eight to ten days for viral development before it is capable of transmitting the virus to man. Once infected, the mosquito remains infective for the remainder of her life.

In 2001, Ted Brown collected two adult *Aedes aegypti* from Doña Ana County, confirming the presence of this mosquito in New Mexico. Since then, New Mexico State University has collected *Ae. aegypti* during mosquito surveys in Doña Ana County.

***Aedes aegypti*, the vector of both dengue and yellow fever.**

Yellow Fever (YF). Yellow fever is caused by a virus closely related to the dengue virus. In fact, YF infections produce dengue-like symptoms in humans; however, the effects of YF are normally much more severe. The fatality rate may reach 50% or more in epidemics. The virus is presently found in Africa and South America. It is absent from the continental U.S., where the last epidemic of YF occurred in New Orleans in 1905.

As with dengue, the YF virus is transmitted in urban areas by *Ae. aegypti*. Unlike dengue, however, the probability of YF virus reaching the U.S. is low. This may be due in part to the fact that YF, along with cholera, smallpox, and plague, are quarantinable diseases. This means that the Centers for Disease Control and Prevention in Atlanta, Georgia, are continually monitoring outbreaks of YF in the Western hemisphere. Yellow fever vaccines are used to prevent disease in humans going to YF endemic areas. It is also required of people moving from YF endemic areas to YF-free areas where *Ae. aegypti* is present.

Malaria. On a worldwide basis, malaria remains the most important human disease transmitted by mosquitoes. It is estimated that there are 400 million human cases of malaria in the world (mostly in Asia and Africa), with over two million human deaths annually. Most of the deaths are children under 10 years of age. In Africa, more than one in every 20 children dies from malaria. It is believed that malaria was introduced into the North American continent during colonial days. Tens of thousands of cases occurred in the U.S. before the 1930s, but there are no reliable statistics available for the period. In the 1930s, approximately 100,000 cases were reported annually; in the early 1940s, the number of cases was reduced dramatically due to the work of public health agencies using DDT during and after World War II and to the Tennessee Valley Authority (TVA) source reduction program.

Since the late 1950s a few hundred to a few thousand new cases have been recorded annually, almost entirely attributable to travelers and military returnees from malarious areas. These cases were contracted outside the U.S., but symptoms appeared following re-entry into the country.

Until recently, secondary infection contracted in the U.S. has been rare. However, there are periodically documented cases of human malaria being transmitted in the U.S. from infected hosts to uninfected hosts by indigenous mosquitoes (in California, Florida, New Jersey, New York and Texas). Anti-malarial prophylaxis is effective and most non-resistant forms of malaria are responsive to treatment. However, resistant strains of malaria are widespread and difficult to treat.

Human malaria is caused by any of four species of *Plasmodium*, a protozoan parasite that causes fever, chills, sweating, and headache. Anemia results from destruction of red blood cells by malaria parasites in their various stages. If not treated, it may cause shock, renal failure, acute encephalitis, coma, and death. The four human malaria parasites are:

- *Plasmodium falciparum*; most severe form; circumtropical; resistant strains exist
- *P. vivax*; tropical and temperate zones
- *P. malariae*; Africa and SE Asia; also in chimpanzees
- *P. ovale*; mildest, rarest form; West African coast

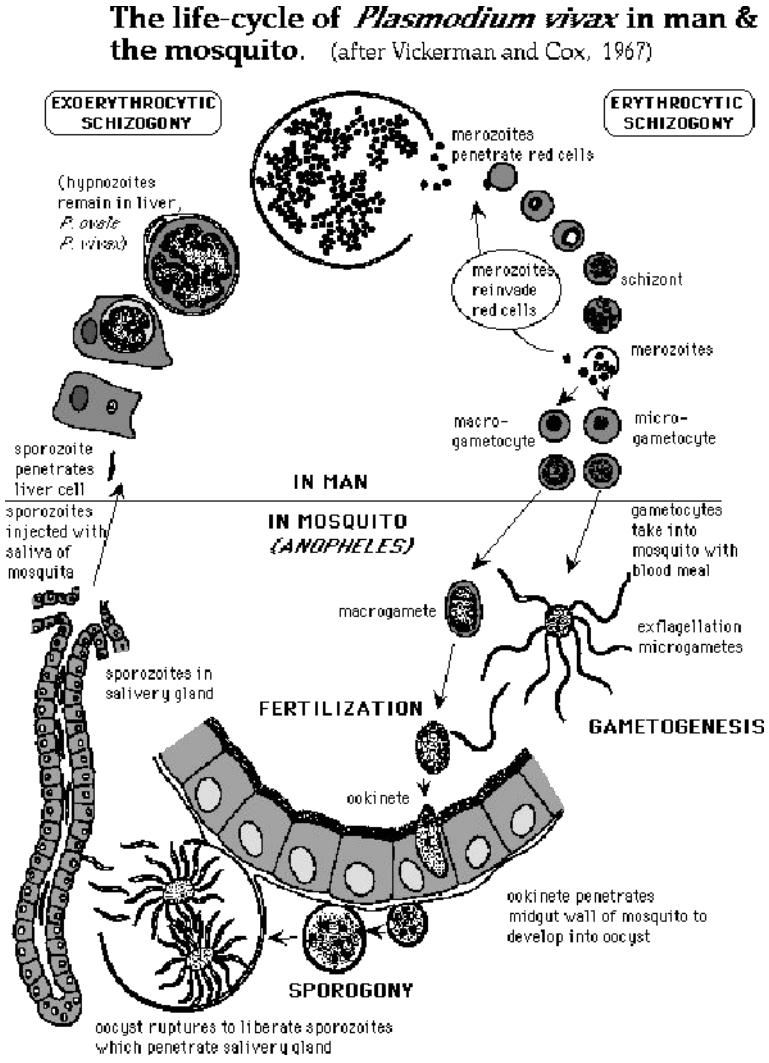
The disease is transmitted by several species of *Anopheles* mosquitoes. In the eastern U.S., some members of the *An. quadrimaculatus* species complex are important vectors. Another species, *An. crucians*, is also a vector but probably to a lesser degree. In the western U.S., the major vectors are *An. hermsi* and *An. freeborni*. These species are widespread and are most abundant from April through September. Worldwide, approximately 85 – 90 of the 370 *Anopheles* species are vectors of malaria.

In malaria, the mosquito is an obligatory vector, in which the parasite must complete part of its life cycle in the mosquito and part in the human host. An infected female *Anopheles* mosquito injects the asexual form of the parasite (sporozoites) into a person's blood stream as she feeds. The sporozoites travel through the blood stream to the liver, entering the liver cells. After 6 to 25 days, merozoites are formed and leave the liver to enter the bloodstream and attack the red blood cells. This developmental stage is called the trophozoite. As the trophozoite grows it destroys its red blood cell to release merozoites that invade more red blood cells. This produces the fevers and chills associated with malaria as these parasites and their toxins are released into the bloodstream periodically. Male and female gametocytes of the parasite are also released from the red blood cells and are then ingested by another female *Anopheles* mosquito when she bites a victim.

In the mosquito, the gametocytes (the sexual forms of the malaria parasite) unite to form a zygote which then travels to the wall of the mosquito's gut, penetrates the lining cells, and comes to rest on the outer wall of the gut. The resulting oocyst begins producing sporozoites. The sporozoites invade the salivary glands of the mosquito where they are injected into the next host for the cycle to repeat.

Eradication of malaria in most areas is made more difficult by vectors becoming resistant to pesticides, resistance to antimalarial drugs in some areas, lack of skilled vector control workers, and lack of drugs and resources. The spread of malaria parasites is facilitated by the ease of worldwide travel these days. Other factors influencing the spread of malaria include vector capability and longevity, anthropophilic tendencies (does the mosquito species present bite

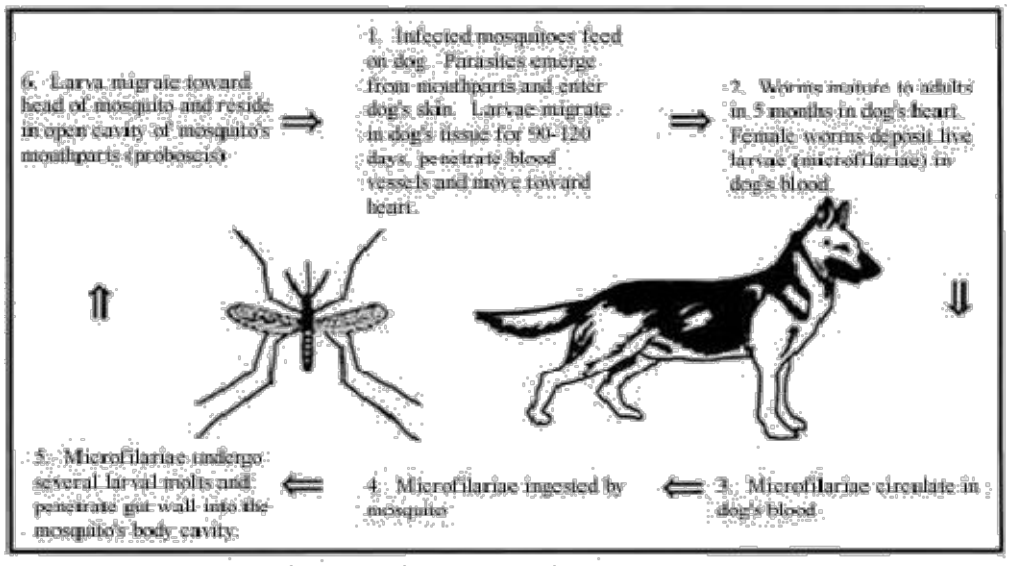
humans?), population density, education of the public, use of antimalarial chemoprophylaxis, chemical, physical, and cultural (i.e., screens on windows) vector control methods, and access to medical care.



Dog heartworm. Dog heartworm (*Dirofilaria immitis*, a filarial worm) is a serious disease for all breeds of dogs in temperate and tropical climates. Infection rates in some states have been reported to be as high as 80% in dogs over two-and-a-half years old, and almost 100% in dogs over five years old. Humans can also become infected, but the worms are unable to develop or mature because humans are not their natural hosts. Several genera of mosquitoes can transmit the parasite to dogs (e.g., *Aedes*, *Anopheles*, *Culex*, and *Psorophora*). However, in nature, *Cx. quinquefasciatus* is considered the most important vector. Other incriminated vectors of dog heartworms include *Ae. albopictus*, *Ae. sierrensis*, *Ae. taeniorhynchus*, *Ae. vexans* and *Cx. salinarius*. A study in Bernalillo County showed that *Aedes vexans* had the greatest potential for heartworm transmission, compared to *Culex quinquefasciatus* and *Cx. tarsalis*.

The life cycle of the dog heartworm involves two factors: mosquitoes and dogs. Mosquitoes ingest the immature worms called microfilariae while taking blood from dogs. These immature worms develop through three larval stages in the mosquito in nine to 14 days. After they have developed into infective or third stage larvae, the microfilariae are transmitted via the mosquito's mouthparts to a dog when the mosquito feeds again. The larvae grow and migrate through the host's subcutaneous tissues and large blood vessels and eventually enter the right ventricle of the heart. In the heart, the larvae grow into adult worms measuring about 11 inches for the females, and 6 inches for the male. The adult female lays no eggs but produces microfilariae that circulate in the blood, completing the cycle.

In areas where dog heartworm infection is prevalent, dog owners should provide mosquito-proof sleeping quarters for their animals. Additionally, the owners should seek the assistance of a veterinarian to administer a program of chemical prophylaxis. Highly effective preventive medication is readily available and easily administered.



Simplified life cycle of dog heartworm.

Filariasis. Although not currently present in the United States, filariasis is endemic in some islands of the Caribbean and is undoubtedly imported occasionally from endemic areas in the Western Hemisphere, Africa and Asia. Transmitted from person to person by *Mansonia*, *Culex*, *Anopheles,* and *Aedes* species, the filarial nematode parasites (*Wuchereria bancrofti* and *Brugia malayi*) live in the human lymphatic system. They cause extreme enlargement of soft tissues, called elephantiasis. Mosquitoes pick up the microfilariae by feeding on infective humans. After developing through the larval stages, the infective (third stage) larvae leave the mosquito and enter the human host as the female mosquito penetrates the skin in search of blood. Filariasis represents a minimal threat to subtropical areas in the United States.

Acquired Immune Deficiency Syndrome (AIDS). When the AIDS virus was initially discovered in humans, one of the first questions asked of public health officials was, "Is the virus that causes AIDS transmitted by bloodsucking insects?" There have been exhaustive laboratory tests conducted by the Centers for Disease Control and Prevention and other similar agencies around the world using a wide range of blood feeding insects and arthropods in attempting to answer this question. Currently, there is no scientifically-based, credible evidence to suggest or imply that mosquitoes or other biting arthropods can transmit the virus to humans. The epidemiological evidence also strongly argues against vector-borne transmission of AIDS: in Africa, where both AIDS and malaria are prevalent, AIDS is confined to sexually active adults and IV drug users, not appearing in children nor at random in local populations, as would be expected of true vector/mosquito-borne diseases.