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Equine Encephalitides


Sleeping Sickness
Eastern Equine Encephalitis (EEE)
Western Equine Encephalitis (WEE)
Venezuelan Equine Encephalitis (VEE)

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Overview

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



www.fda.gov/oc/ohrt/equine.html

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In today’s presentation we will cover information regarding the agents that cause the equine encephalitides and their epidemiology. We will also talk about the history of these diseases, how they are transmitted, species that they affect, and clinical signs seen in humans and animals. Finally, we will address prevention and control measures, as well as actions to take if any of the equine encephalitides are suspected.

[Photo: Horses in field. Source: U.S. Department of Agriculture]

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Equine Encephalitides

- Eastern equine encephalitis (EEE)
- Western equine encephalitis (WEE)
- Venezuelan equine encephalitis (VEE)



www.fda.gov/oc/ohrt/equine.html

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Arboviruses are viruses that are transmitted by arthropods. Eastern, Western, and Venezuelan equine encephalomyelitis (EEE, WEE, VEE) are mosquito-borne, viral infections that can cause severe encephalitis in horses and humans. Some of these viruses also cause disease occasionally in other mammals and birds.

[Photo: Horses. Source: Brigitte Werner/Pixabay.com/public-domain]

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THE ORGANISM

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The Viruses

- EEE, WEE, and VEE viruses
 - Family *Togaviridae*
 - Genus *Alphavirus*
- Mosquito-borne
- Disease
 - Encephalitis in humans and horses
 - Other mammals and birds are occasionally affected



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Eastern, Western and Venezuelan equine encephalomyelitis result from infection by the respectively named viruses in the genus *Alphavirus* (family *Togaviridae*). In the human literature, the disease is usually called Eastern, Western or Venezuelan equine encephalitis rather than encephalomyelitis. These mosquito-borne viral infections cause severe encephalitis in horses and humans. Some of these viruses also cause disease occasionally in other mammals and birds.

[Photo: Electron micrograph of the Eastern equine encephalitis virus. Source: Dr. Fred Murphy and Sylvia Whitfield/CDC Public Health Image Library]

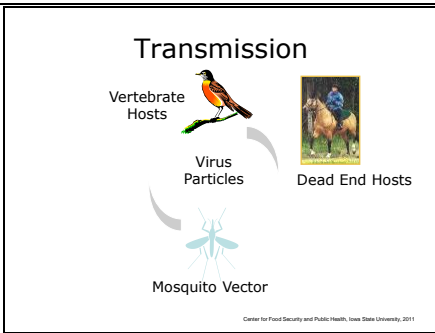
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TRANSMISSION

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Transmission for arboviruses begins when an infected female mosquito takes a bloodmeal from a reservoir vertebrate host, which in most cases is a bird. Viremia then sets in and is of a sufficient level and duration to affect other mosquitoes, thus propagating the cycle. The virus particles replicate in the salivary glands of the mosquito to be passed onto other vertebrate hosts, or dead end hosts, such as humans and horses, where overt disease occurs.

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Mosquito Life Cycle

- Four-stage life cycle
 - Egg, larva, pupa, adult
- *Aedes* species
 - Lay single eggs
 - Damp soil, later flooded
- *Culex* species
 - 100-300 eggs in raft
 - Lay eggs at night on water surface
- Survival requires wind protection
- Overwinter in egg stage

Center for Food Security and Public Health, Iowa State University, 2011

There are about 200 different species of mosquitoes in the United States, all of which live in specific habitats, exhibit unique behaviors, and bite different species of animals. Despite these differences, all mosquitoes share some common traits, such as a four-stage life cycle. The top image depicts the first stage of the life cycle of a mosquito, the egg. For *Aedes* species, an individual egg is laid one at a time on vegetation or damp soil that is later flooded by water. In the case of *Culex* species, an egg raft consisting of 100 to 300 eggs is laid at night on the water's surface (bottom image). It is essential for survival that the eggs are laid in an area that is sheltered from the wind. If a species overwinters, they do so in the egg stage. Eggs typically hatch to the larval stage within 48 hours.

[Photo: Mosquito life cycle. Source: Alameda County Mosquito Abatement at <http://www.mosquitoes.org/index.htm>]

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Mosquito Life Cycle

- Larvae live upside down in water; "wriggler"
 - Breathe via siphon tube
 - Molt 4 times
- Pupal stage is restful, non-feeding; "tumbler"
 - Breathe via "trumpets"
 - Splits to allow adult to emerge


Center for Food Security and Public Health, Iowa State University, 2011

Larvae ("wrigglers") come to the surface to breathe by utilizing a siphon tube while hanging upside down. They require large amounts of nutrients for maturation and feed on organic matter in the water. Over a 4 to 14 day period, depending on water temperature, they molt four times. The stages between molts are referred to as instars, and the larvae grow larger each stage, finally becoming a pupa after the 4th instar. The pupal stage can last 1 to 4 days, again dependent on water temperature, and resembles the butterfly in the cocoon stage, because this is where the mosquito develops into an adult. It is a very restful, non-feeding stage and the only movement is when it "tumbles" to protect itself. It utilizes two breathing tubes called "trumpets" and floats at the water's surface. During the summer, a *Culex* species pupa in the southern United States become an adult after two days in the pupal stage.

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Mosquito Life Cycle

- Newly emerged adult rests
- Female takes blood meal
 - Only females bite
 - Attractants for biting
 - Carbon dioxide, temperature, moisture, smell, color, movement
- Mating occurs a few days after flight
- Lifespan varies from 4 to 30 days



After the pupal skin splits, the newly emerged adult rests on the water surface long enough to dry off its wings in order to fly and harden its body parts. After a few days, the adult female begins to take blood meals and mate. Only female mosquitoes bite animals and humans and require a protein found in blood for egg production. Male mosquitoes are nectar feeders and do not bite humans. Carbon dioxide, temperature, moisture, smell, color, and movement are all attractants for biting mosquitoes, and humans usually are not their first choice. The entire lifespan varies with temperature and species and can range from four days to one month. [Photo: Mosquito. Source: CDC Public Health Image Library]

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Vectors of the Equine Encephalitides

Disease	Mosquito Vector
EEE	<i>Culiseta melanura</i> <i>Aedes</i> spp. <i>Culex (Cx.) nigrapalpus</i> <i>Coquilletidia</i> spp.
WEE	<i>Culex tarsalis</i> <i>Aedes melanimon</i> <i>Aedes dorsalis</i> <i>Aedes campestris</i>
VEE	<i>Culex (Melanoconion) spp.</i>

Center for Food Security and Public Health, Iowa State University, 2011

The mosquito vectors for EEE, WEE, and VEE are listed above.

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SUMMARY OF EQUINE ENCEPHALITIDES

Distribution, Magnitude, and Outcomes

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Equine Encephalitides: Classification and Distribution

Disease	Family, Genus	Distribution
EEE	Togaviridae <i>Alphavirus</i>	Eastern U.S.
WEE	Togaviridae <i>Alphavirus</i>	Western U.S.
VEE	Togaviridae <i>Alphavirus</i>	Southern U.S.

This table lists the equine encephalitides along with their classification and distribution in the U.S.

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Human Risks and Outcomes

- Eastern equine encephalitis
 - Elderly most at risk
 - Case fatality rate: 33%
- Western equine encephalitis
 - Children <1 year most at risk
 - Case fatality rate: 3%
- Venezuelan equine encephalitis
 - Children most often affected
 - Fatalities are rare


EEE most often affects the elderly; the case fatality rate is 33%. As expected, EEE occurs mostly in the eastern regions of the U.S. WEE is reported in the western and central U.S. WEE causes severe disease in children under the age of one, and the mortality rate is about 3%. VEE cases are less severe than WEE and EEE and usually occur in children, but fatalities are rare.

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Animal Risks and Outcomes

- Case-fatality rate in horses
 - EEE ~ 90%
 - VEE ~ 50 to 90%
 - WEE ~ <30%
- Vaccine available in the U.S.



The case-fatality rate for horses varies depending on the specific encephalitic disease. EEE is the most fatal with an approximate 90% case-fatality. Next is VEE ranging from 50-90%, and lastly WEE causing death in <30% of the cases. Fortunately there is a trivalent, formalin-inactivated vaccine available for horses for WEE, EEE, VEE in the United States.

[Photo: Horses in field. Source: public-domain-image.com]

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EASTERN EQUINE ENCEPHALITIS

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EEE History

- 1831
 - Unknown encephalomyelitis virus affects horses in Massachusetts
- 1933
 - EEE first isolated from a horse
- 1937
 - EEE identified in ring-necked pheasants
- 1938
 - EEE first isolated from human brain


EEE was first isolated from a horse with encephalomyelitis in 1933, but it is thought that the disease dates back to 1831 to horses in Massachusetts. Birds are also susceptible to EEE as was discovered in ring-necked pheasants in 1937 in Connecticut. Since then the disease has been found to affect sparrows, pigeons, Peking ducks, Chukar partridges, emus, and ostriches, illustrating that species not indigenous to North America are susceptible. In 1938, EEE was first isolated from a human brain.

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EEE History

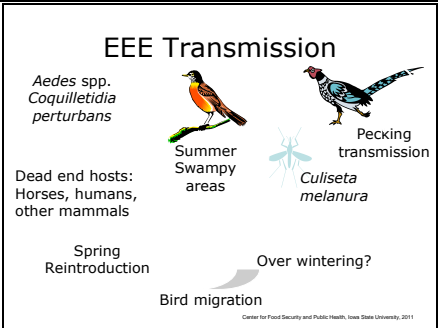
- 1942-1943
 - Michigan epidemic
- 1947
 - Southern Louisiana and Texas
 - 14,000 cases
 - 83% case fatality rate
- 1951
 - Isolated from *Culiseta melanura*



Most epidemics since then tend to occur along the eastern seaboard, from New Hampshire along the Atlantic Coast to the Gulf of Mexico states. However, Michigan had an epidemic in 1942 and 1943, demonstrating that the vector is not restricted to the east coast states. The largest known epidemic occurred in 1947 in southern Louisiana and Texas. Fourteen thousand horses and mules were affected, and nearly 12,000 died. This 83% case fatality rate reflects the typical disease in horses in North America. The disease was first thought to be transmitted by an *Aedes* species mosquito, but in 1951 EEE was isolated from *Culiseta melanura*. [Photo: Mare and foal. Source: Pixabay.com/public domain]

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Transmission of EEE occurs via a mosquito-vertebrate-mosquito cycle, with *Culiseta (Cs.) melanura* (an ornithophilic [“bird-loving”] mosquito feeding almost exclusively on songbirds) as the asymptomatic reservoir host. Birds are also able to spread the disease if they peck or eat diseased pen mates in captivity. *Cs. melanura* does not generally feed on mammals and requires secondary mosquitoes to transmit disease to humans and horses. *Cs. melanura* lives and breeds in freshwater and swamp areas during the summer, and feeds most actively 2 hours after sunset to sunrise. In late summer and early fall they can be found in drier uplands. The epidemic vector that spreads

disease to mammals and exotic birds varies for different regions of EEE prevalence, but *Coquilletidia (Cq.) perturbans* and several *Aedes* species are often involved. Disease most often occurs within 5 miles of the swampy areas where *Cs. melanura* and *Cq. perturbans* live and breed. *Cq. perturbans* is an opportunistic feeder that feeds on birds and mammals. Horses and humans are considered dead-end hosts of EEE virus because neither reaches a high enough level of viremia to infect mosquito vectors. Other mosquito species, such as *Aedes vexans* and *Culex nigripalpus*, can also transmit EEE virus. How EEE survives over winter is still unknown but *Cs. melanura* overwinter as larvae.

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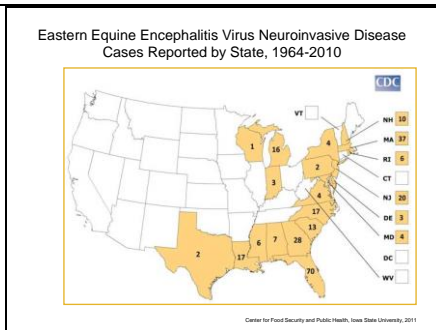
EEE Epidemiology

- 1964-2010
 - 270 cases total
 - Average 6 cases each year
 - Average 1 to 2 deaths each year
- Case-fatality rates
 - Human: 30 to 70%
 - Equine: 90%
- Equine cases usually appear first
 - Serve as sentinels for human disease

<http://www.cdc.gov/EasternEquineEncephalitis/tech/epi.html>

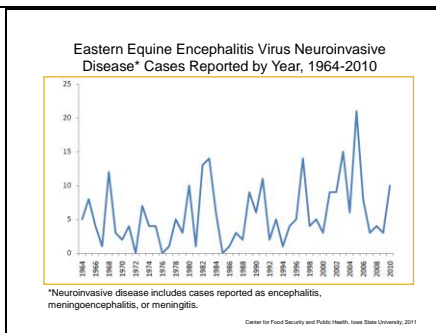
Since 1964, there have been a reported 270 cases of human EEE, averaging 6 cases per year, which is much smaller than the number of equine cases. The fatality rate is 30 to 70%, which is 1 to 2 human deaths annually, whereas horse mortality rates can be 90% or higher, with death occurring rapidly. EEE is a seasonal disease in most of North America, with outbreaks occurring in the late summer and early fall, reflecting the activity of the mosquito vector. Horses are usually the sentinel indicator of human disease.

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From 1964 through 2010, EEEV neuroinvasive disease cases were reported in Alabama (7), Delaware (3), Florida (70), Georgia (28), Indiana (3), Louisiana (17), Maryland (4), Massachusetts (37), Michigan (16), Mississippi (6), New Hampshire (10), New Jersey (20), New York (4), North Carolina (17), Pennsylvania (2), Rhode Island (6), South Carolina (13), Texas (2), Virginia (4), and Wisconsin (1). [Photo: Map reflecting the number of human cases of Eastern equine encephalitis virus neuroinvasive disease in the United States – reported by state, 1964-2010. Source: Centers for Disease Control and Prevention at <http://www.cdc.gov/EasternEquineEncephalitis/tech/epi.html>]

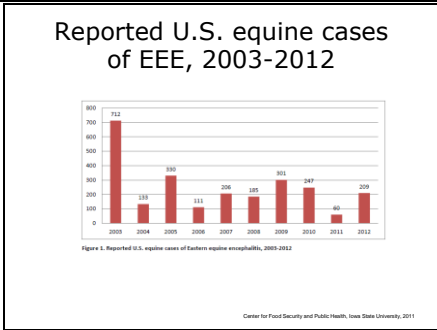
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Data Table: In the United States, the annual number of reported Eastern equine encephalitis virus neuroinvasive disease cases reported varies. From 1964 through 2010, an average of 6 cases were reported annually (range 0-21). This graph demonstrates how the number of cases can vary markedly from year to year. Note the cyclic, seasonal nature of the reported cases related to the summertime activity of the vector.

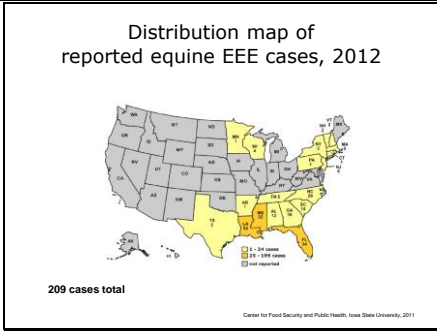
[Photo: Human Cases of Eastern Equine Encephalitis virus neuroinvasive disease in the United States – reported by year, 1964-2010. Source: Centers for Disease Control and Prevention at <http://www.cdc.gov/EasternEquineEncephalitis/tech/epi.html>]

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This graph shows the reported U.S. equine cases of Eastern equine encephalitis from 2003-2012. Source: U.S. Department of Agriculture, 2012 Summary of Eastern Equine Encephalitis Cases in the United States at <http://www.aphis.usda.gov/vs/nahss/equine/ee/>

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This map shows the distributio of the reported equine Eastern equine encephalitis cases reported in 2012 in the U.S. (209 cases total). Source: U.S. Department of Agriculture, 2012 Summary of Eastern Equine Encephalitis Cases in the United States at <http://www.aphis.usda.gov/vs/nahss/equine/ee/>

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- ### EEE in Humans
- Incubation period: 4 to 10 days
 - Mild disease uncommon
 - Fever, myalgia, headache, nausea, vomiting, abdominal pain, and photophobia
 - Seizure and coma in severe cases
 - Longer fever and flu-like symptoms before CNS signs results in a better outcome

The incubation period of EEE in humans is anywhere from 4 to 10 days following the bite from an infected mosquito. Milder disease is uncommon with EEE, and the time of onset of signs often indicates severity. Generally symptoms begin with a sudden fever, myalgia, headache, nausea, vomiting, abdominal pain, and photophobia. Severely affected individuals progress to seizure and coma. A long onset of fever and flu-like symptoms without CNS signs generally indicates a better prognosis.

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- ### EEE in Humans
- Survival rates associated with age
 - Highest in young adults: 70%
 - Lower in children: 60%
 - Lowest in elderly: 30%
 - Recovery can result in permanent brain damage
 - Diagnosis by serology
 - Treatment is supportive care

Outcome and quality of life following survival are also age-related, with survival rates being 70% in young adults, 60% in children, and lowest in the elderly at 30%. Those who recover may suffer permanent brain damage and require permanent institutional care. Diagnosis is often based on clinical signs, but is definitively made serologically with IgM capture ELISA. Seroprevalence at any titer, along with signs of a CNS infection is considered diagnostic because antibody levels in endemic areas are naturally low. Treatment is generally supportive and includes ventilation, minimizing cerebral edema, and maintaining electrolyte balance. There is no commercially available vaccine for humans.

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- ### EEE in Horses
- Incubation period: 5 to 14 days
 - Clinical signs in horses
 - Fever, anorexia, depression
 - CNS signs
 - Hypersensitivity, aimless wandering, head pressing, circling, ataxia, paresis, paralysis
 - Death may occur within days
 - Asymptomatic or mild infections also occur
 - Equine vaccine available

The incubation period for EEE is five to 14 days. The initial clinical signs include fever, anorexia and depression. In severe cases, this prodromal stage is followed by encephalitis; altered mentation, hypersensitivity to stimuli, involuntary muscle movements, impaired vision, aimless wandering, head pressing, circling, an inability to swallow, ataxia, paresis, paralysis and convulsions may be seen. Periods of excitement or intense pruritus can also occur. Laterally recumbent animals sometimes have a characteristic “paddling” motion. In addition, some animals may develop diarrhea or constipation, or have significant weight loss. Some affected horses die, particularly

when infected with EEE, within a few days. Asymptomatic infections or mild disease without neurologic signs may also occur. Equine vaccines are available for EEE.

S I d e 2 8	<p>EEE in Birds</p> <ul style="list-style-type: none"> • Asymptomatic in most bird species • Clinical signs <ul style="list-style-type: none"> - Depression, tremors, leg paralysis, somnolence - Emus, ostriches <ul style="list-style-type: none"> • Hemorrhagic enteritis, emesis - Death 24 hours after onset • Vaccination <ul style="list-style-type: none"> - Some birds are vaccinated for EEE 	<p>EEE virus infections are asymptomatic in most species of birds, but serious or fatal infections can occur in some species. Birds infected with EEE exhibit depression, tremors, leg paralysis, and somnolence, resulting in death after 24 hours. Emus and ostriches may only present with hemorrhagic enteritis and emesis. In some areas, some bird species may be vaccinated for EEE.</p>
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S I d e 2 9	<p>Diagnosis</p> <ul style="list-style-type: none"> • Ante mortem: serology <ul style="list-style-type: none"> - Virus neutralization - Hemagglutination inhibition - ELISA - Complement fixation - Virus isolation • Post mortem <ul style="list-style-type: none"> - Virus identified in tissues (brain) - Immunohistochemistry, ELISA, RT-PCR 	<p>In horses, EEE can be diagnosed by serology. Commonly used tests include virus neutralization (the plaque reduction neutralization or PRN test), hemagglutination inhibition, ELISA and complement fixation. A definitive diagnosis usually requires a fourfold rise in titer in paired samples. The identification of specific IgM in the ELISA is also useful; a presumptive diagnosis may be obtained with a single sample, particularly when a combination of serologic tests is used. In horses, EEE may also be diagnosed by virus isolation. At necropsy, EEE virus may be found in tissues, particularly the brain, with immunohistochemistry, ELISA or RT-PCR.</p>
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S I d e 3 0	<p>WESTERN EQUINE ENCEPHALITIS</p>	
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S I d e 3 1	<p>WEE History</p> <ul style="list-style-type: none"> • 1930 <ul style="list-style-type: none"> - Isolated from horse brain - California; 50% case fatality rate • 1933 <ul style="list-style-type: none"> - <i>Aedes aegypti</i> experimentally infected with WEE <ul style="list-style-type: none"> • Virus transmitted to guinea pigs • Virus transmitted to horses (1936) • 1938 <ul style="list-style-type: none"> - Isolated from human brain 	<p>WEE was first isolated from a horse brain in 1930 when nearly 6,000 horses fell ill with a CNS disease in the San Joaquin Valley of California. The case-fatality rate was about 50% in that particular epidemic. In 1933, researchers were able to experimentally infect <i>Aedes aegypti</i> mosquitoes and transmit the virus to guinea pigs. The virus was experimentally transmitted to horses in 1936; however, it wasn't until 1938 that WEE was isolated from a human brain.</p>
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WEE History

- 1941
 - Natural infection found in mosquito *Culex tarsalis*
 - Epidemic in Canada and northern U.S.
- 1942
 - *Culex tarsalis* identified as the vector
- 1943
 - Confirmed as mosquito-borne disease
 - Birds identified as reservoir host

Culex tarsalis mosquitoes were found to be naturally infected with WEE in 1941 in the state of Washington. A major epidemic also occurred that year involving 2,792 cases in Manitoba and Saskatchewan, Canada and the north central United States. Case-fatality rates averaged 12.4 per 100,000. By 1942, evidence confirmed *Culex tarsalis* was an important vector of the virus. By 1943, WEE was thought to be mosquito-borne, utilizing birds as their reservoir host. Throughout the 1940's, many studies proved the distribution of WEE to include much of the western United States.

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WEE Transmission

The diagram illustrates the WEE transmission cycle. It shows Secondary Amplifiers (Prairie Dog, Blacktail Jackrabbit, Kangaroo rat) and Primary Vertebrate Hosts (House Sparrow, House Finch). The Primary Vector is *Culex tarsalis*. Dead-end hosts include Horses and humans. The diagram is credited to the Center for Food Security and Public Health, Iowa State University, 2011.

Transmission of WEE occurs primarily in areas west of the Mississippi River and involves a mosquito-vertebrate-mosquito cycle. *Culex tarsalis* is the primary vector for transmission to a variety of asymptomatic, primary amplifying hosts, namely the house sparrow (*Passer domesticus*) and the house finch (*Carpodacus mexicanus*). Other passerine birds, such as the red-winged blackbird and magpie, are also amplifier hosts for WEE. The blacktail jackrabbit, kangaroo rat, Western gray squirrel, and prairie dog are all mammals that serve as amplifiers for WEE in various parts of the United States. Humans and horses are dead-end hosts for WEE and do not contribute to virus amplification. *Culex tarsalis* breeds in agricultural areas, such as irrigation ditches and other aquatic areas rich with vegetation. WEE virus was isolated from field collected larvae of *Aedes dorsalis*, providing evidence that vertical transmission may play an important role in the maintenance cycle of an alphavirus. WEE virus has been isolated occasionally from some other mosquito species present in the area.

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WEE Transmission

State	Vector	Avian host	Mammalian Host
CO	<i>Culex tarsalis</i>	House sparrow, Red-winged blackbird, Magpie	Blacktail jackrabbit, Kangaroo rat
CA	<i>Culex tarsalis</i> <i>Aedes melanimon</i>	House sparrow House finch	Blacktail jackrabbit, Western gray squirrel
TX	<i>Culex tarsalis</i> , <i>Cx. quinquefasciatus</i> <i>Aedes vexans</i>	House sparrow	Blacktail jackrabbit, Prairie dog
NM	<i>Aedes dorsalis</i> , <i>Ae. campestris</i>		

© 2009, CDC

This table depicts the various vectors responsible for transmission of WEE and their avian and mammalian hosts for different states west of the Mississippi River. *Culex tarsalis* is the primary vector for transmission in Colorado, California, and Texas to a variety of asymptomatic, primary amplifying hosts, namely the house sparrow (*Passer domesticus*) and the house finch (*Carpodacus mexicanus*). Other important mosquito vector species include *Aedes melanimon* and *Culex stigmatosoma* in California; *Ae. dorsalis* in Utah and New Mexico; *Ae. campestris* in New Mexico; *Culex quinquefasciatus*, *Ae. vexans*, *Ae. nigromaculis*, and *Psorophora columbiae* in Texas.

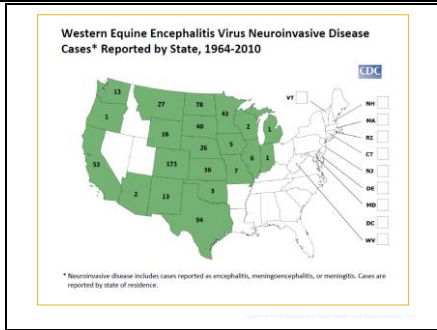
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WEE Epidemiology

- *Culex tarsalis*
 - High populations mid- to late-summer
 - Epidemics associated with cool, wet spring
 - Wind can carry mosquitoes 800 miles in less than 24 hours
- Cases appear in June-August
 - 639 cases since 1964
 - 1989-1997: No human deaths

Culex tarsalis mosquitoes generally reach their highest population density in mid- to late-summer. Human and horse cases of WEE soon follow. Epidemics are often associated with cool spring temperatures and increased precipitation for vector abundance. Wind trajectories have been followed and it is suggested that mosquitoes breed in the winter months near the Gulf of Mexico and then are carried to northern Texas and Oklahoma in the spring. In early summer, *Culex tarsalis* is carried north to Kansas, Nebraska, South Dakota, Minnesota, Wisconsin, and Manitoba, reflecting the pattern of outbreaks that occurred in 1981 and 1983. These mosquitoes can travel 780 to 840 miles (1250 to 1350 km) in less than 24 hours. Because of vector population, most cases are seen from June to August. There have been 639 cases of human WEE since 1964 in the United States but no deaths were reported from 1989 to 1997. [Photo: *Culex tarsalis* mosquito. Source: CDC Public Health Image Library]

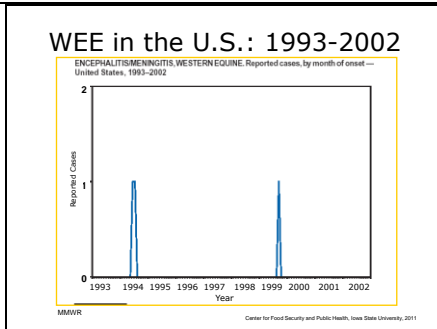
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This map shows the distribution of human Western equine encephalitis virus neuroinvasive disease cases in the U.S. – reported by state between 1964-2010. For this period, WEEV neuroinvasive disease cases have been reported in Arizona (2), California (53), Colorado (173), Illinois (6), Indiana (1), Iowa (5), Kansas (36), Michigan (1), Minnesota (43), Missouri (7), Montana (27), Nebraska (26), New Mexico (13), North Dakota (78), Oklahoma (3), Oregon (1), South Dakota (40), Texas (94), Washington (13), Wisconsin (2) and Wyoming (16).

[Image from Centers for Disease Control and Prevention at

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This graph depicts the number of reported WEE human cases in the United States from 1993-2002. During 1997, 35 strains of WEE virus were isolated from mosquitoes collected in Scotts Bluff County, Nebraska, but no human cases were reported. During the time period 1964-2002, an average of 18 human cases (range 0-172) were reported each year in the United States. (Data from the Summary of Notifiable Diseases 2002, CDC website.)

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WEE in Humans

- Incubation: 5 to 10 days
- Resembles EEE but usually asymptomatic or mild in adults
- Clinical signs
 - Sudden onset of fever, headache, nausea, vomiting, anorexia, malaise
 - CNS signs in children less than 1 year
 - Altered mental status, weakness, irritability, stupor, coma

The incubation period is 5 to 10 days for WEE. WEE resembles EEE but is usually asymptomatic or mild in adults, with nonspecific signs of illness and few deaths. Children under 1 year of age are affected more severely than adults, and the elderly and immunosuppressed are also more susceptible. Clinical symptoms often include a sudden onset of fever, headache, nausea, vomiting, anorexia, and malaise. Patients who progress to central nervous system signs have an altered mental status, weakness, vertigo, photophobia, and drift into a stupor or coma. Infants less than 2 months of age are irritable, convulse, and have tremors. As a patient ages, the signs occur less frequently; however 5 to 30% of young patients are often left with permanent neurological sequelae and require permanent institutionalization or home care.

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WEE in Humans

- Prognosis
 - Poor for young clinical patients
 - Case-fatality rate: 3 to 15%
 - Death within one week of clinical onset
- Diagnosis difficult from blood, CSF
 - Post mortem virus isolation from brain
- Treatment is supportive care
- Vaccine available for military personnel only

The mortality rate with WEE ranges from 3 to 15% depending on the source, and death will occur within the first week after onset of illness. Diagnosis from blood or CSF is difficult during the illness and often is confirmed by isolation from the brain following a post mortem exam. Acquiring acute and convalescent sera and monitoring for fourfold or greater increase in antibody titer is ideal, but is often not obtainable due to the clinical course of the disease. Treatment involves supportive care, and although there is a vaccine available, it is generally only administered to military personnel.

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WEE in Animals

- Asymptomatic
 - Blacktail jackrabbit, kangaroo rat, Western gray squirrel, prairie dog, birds
- Horses with clinical signs
 - Fever, depression, altered mentation, head pressing, ataxia, dysphagia
 - Progress to paralysis, convulsions, death
 - Mortality rate <30%

Vertebrate mammalian hosts, such as the blacktail jackrabbit, kangaroo rat, Western gray squirrel, and prairie dog, are generally asymptomatic and only serve as amplifiers of the disease. Birds are also uncommonly affected. Horses are dead-end hosts for WEE and may be asymptomatic. When present, clinical signs in equines initially include fever, depression, quiet demeanor progressing to altered mentation, head pressing, impaired vision, ataxia, and the inability to swallow. Paresis and paralysis generally precede convulsions, and death can occur within 2 to 3 days following the onset of clinical signs. Mortality is generally <30%, but those that develop neurological signs and recover still have a poor prognosis.

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WEE in Animals

- Diagnosis
 - Serology
 - Can differentiate EEE and WEE using the virus neutralization or ELISA tests
 - Post mortem
 - Immunohistochemistry, ELISA, RT-PCR
- Treatment is supportive care
- Vaccine available

As for EEE, serology is useful for diagnosing WEE in animals. Cross-reactions can occur between EEE and WEE antibodies in the complement fixation and hemagglutination inhibition tests; however, these viruses can be differentiated by virus neutralization or antigen-capture ELISA. At necropsy, WEE virus may be found in tissues, particularly the brain, with immunohistochemistry, ELISA or RT-PCR. There is no treatment besides supportive care for this disease. A vaccine is available.

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VENEZUELAN EQUINE ENCEPHALITIS

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Viral Strains

Subtype	Cycle	Pathogenic
I-A I-B I-C	Epizootic/ Epidemic	Highly virulent for equines
I-D II I-E III I-F IV V VI	Enzootic/ Endemic	Not for horses Limited cases in humans

Venezuelan equine encephalomyelitis (VEE) virus has a complex classification system due to its large number of subtypes. Subtype I includes six variants, three of which are epidemic variants. The distinction is important from an epidemiological standpoint as some subtypes cause severe disease and epidemics. Variants A,B, and C of subtype I (i.e., I-A, I-B, and I-C) are considered epizootic (or epidemic) strains and are highly virulent to equines. Subtype I-A originated in donkeys in Trinidad; I-B originated in humans in Honduras. [Note: These two subtypes are usually referred to as I-AB because of their almost identical fingerprints.] I-C originated in horses in Venezuela. The variants D, E, and F of subtype I (i.e., I-D, I-E, and I-F) and subtypes II, III, IV, V, VI are considered enzootic (or endemic) strains and are not pathogenic for equines. There have been infrequent limited outbreaks from these strains in humans. Enzootic (endemic) strains have a wide geographic distribution in the Americas, but the pathogenic form has not been seen in the United States since 1971. VEE viruses have been classified into six subtypes based on antigenic analysis: subtypes I-D, I-E, and I-F; subtype II (Everglades virus - which was the only subtype found in the United States); subtype III (Mucambo virus A,B,C); subtype IV (Pixuna virus); subtype V (Cabassou virus); and subtype VI (AGso-663 virus).

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VEE Viral Strains


<ul style="list-style-type: none"> • Epizootic/Epidemic <ul style="list-style-type: none"> - I-A, I-B, and I-C - Disease in humans and horses - Transmission by many mosquito species - Natural reservoir unknown - Horses and donkeys act as amplifiers 	<ul style="list-style-type: none"> • Enzootic/Endemic <ul style="list-style-type: none"> - Disease in humans - Transmission mainly by <i>Culex (Melanoconion)</i> species - Natural reservoir is rodents living in swamps and forests
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The epidemic strains of VEE (i.e., I-A, I-B, and I-C) cause disease in humans and horses, while the enzootic strains (i.e., I-D, I-E, I-F, II, III, IV, V, VI) only cause intermittent disease in humans. Enzootic strains have been isolated from mosquitoes, whereas epidemic strains have not been identified since 1973, questioning whether they are still present in nature. Epidemic strains utilize a large number of mosquito species as a means to spread disease; vertebrates, primarily horses and donkeys amplify the virus. The natural reservoir is unknown. Enzootic strains have a wide geographic distribution in the Americas and are maintained in wild animals, specifically rodents living in rain forests and swamps. They are transmitted by fewer mosquito vectors, mainly *Culex (Melanoconion)* species.

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VEE History

- 1938
 - Isolated from horse brain
- 1962-1964
 - Outbreak in Venezuela
 - 23,000 human cases
- 1967
 - Outbreak in Colombia
 - 220,000 human cases
 - Over 67,000 horse deaths



Outbreaks of VEE-type diseases have occurred in the Western Hemisphere since the 1920s, and the virus was finally isolated in 1938 from a horse brain. Annual outbreaks occurred throughout the 1960s in Colombia and Venezuela. A long standing epidemic from 1962 to 1964 in Venezuela caused more than 23,000 human cases of VEE with a case-fatality rate of 0.6%. In 1967, a major epidemic hit Colombia, causing 220,000 human cases and over 67,000 horse deaths. [Photo: Horses. Source: U.S. Department of Agriculture]

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VEE History


- 1969-1971
 - Largest recorded outbreak
 - Covered area from Costa Rica to Rio Grande Valley in Texas
 - Thousands of human encephalitis cases
 - Over 100,000 horses died
- 1995
 - Venezuela and Colombia
 - Over 90,000 human cases

The largest recorded outbreak of VEE began in 1969 in Guatemala and covered a geographic region from Costa Rica to the Rio Grande Valley of Texas. Human cases of encephalitis were in the thousands, and over 100,000 horses died as a result of VEE. Small, occasional outbreaks have occurred in Peru and Mexico, but in fall of 1995 there were over 90,000 human cases in Venezuela and Colombia.

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VEE Epizootic Transmission

Other species naturally infected but not amplifiers




Dead-end hosts
Humans

Primary Vector
multiple mosquito species



Vertebrate Host
Horses




Center for Food Security and Public Health, Iowa State University, 2011


Epizootic strains of VEE virus can be transmitted by a large number of mosquito species, such as *Mansonia titillans*, *M. indubitans*, *Psorophora confinnis*, *Ps. discolor*, *Deinocerites pseudus*, *Aedes thelcter*, *Ae. sollicitans*, *Ae. taeniarhynchus*, *Ae. scapularis*, and *Ae. aegypti*. Horses and donkeys are the principal amplifying hosts for VEE, while humans are dead-end hosts. There are other domestic species of animals that have been naturally infected with the virus, such as cattle, swine, dogs, and chickens, but there is not enough evidence to suggest they are silent amplifiers or reservoirs of disease. Wild species, such as cotton rats, opossums, gray foxes, bats, and wild birds, have been isolated as carriers of the virus in an outbreak, but again their role is unclear as amplifiers or maintainers of VEE.

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VEE Enzootic Transmission

Primary Vector
Culex
(*Melanoconion*)
species





Vertebrate Host
Rodents



Dead end hosts
Humans

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Enzootic transmission of VEE occurs between mosquitoes (primarily *Culex [Melanoconion]* species) and rodents who live in or near swamps and rain forests. Humans are dead-end hosts for VEE.

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VEE in Humans

- Incubation period: 1 to 6 days
- Usually acute, mild, systemic disease
- Clinical signs
 - Fever, chills, headache, myalgia
 - Coughing, vomiting, diarrhea
 - CNS signs
 - Encephalitis occurs in 4% of children
 - Less than 1% of symptomatic adults
- Death is rare

www.cdc.gov/nczod/dzdx/vee

In humans, VEE is usually an acute, often mild, systemic illness. The clinical signs may include fever, chills, generalized malaise, severe headache, photophobia and myalgia particularly in the legs and lumbosacral region. Coughing, sore throat, nausea, vomiting and diarrhea may also be seen. Approximately 4% of children develop mild to severe encephalitis; neurologic disease occurs in less than 1% of symptomatic adults. VEE usually resolves within two weeks, with acute symptoms subsiding after 4 to 6 days, and deaths are rare.

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VEE in Humans

- Pregnant women
 - Fetal encephalitis, placental damage, abortion/stillbirth, congenital disease
- Diagnosis
 - Paired sera with rising titer
 - ELISA IgG or IgM
- Treatment
 - Supportive care
- No vaccine available

www.cdc.gov/nczod/dzdx/vee

In pregnant women, this disease can affect the fetus; fetal encephalitis, placental damage, abortion/ stillbirth or severe congenital neurologic anomalies may be seen. Diagnosis is difficult but involves paired serum samples for rising antibody titer, or ELISA monitoring IgG or IgM. Treatment involves supportive care; there is no vaccine available.

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VEE in Horses

- Incubation period: 1 to 5 days
- Horses most susceptible
 - Fever, anorexia, depression, flaccid lips, droopy eyelids and ears, incoordination, and blindness
 - Death 5 to 14 days after clinical onset
- Case-fatality rate: 50 to 90%
- In utero transmission results in abortion, stillbirth

www.cdc.gov/nczod/dzdx/vee

Incubation period in animals is 1 to 5 days, and horses are the most susceptible to epidemic VEE viral infection. Clinical signs include fever, anorexia, depression, flaccid lips, droopy eyelids and ears, head pressing, circling, incoordination, and blindness. Death usually occurs 5 to 14 days after clinical onset, and case-fatality rate ranges from 50 to 90%. In utero transmission can occur, leading to dead or stillborn foals.

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VEE in Animals

- Most domestic animals do not show clinical signs or amplify the virus
- Experimentally
 - Infected rabbits and dogs die after inoculation
 - Laboratory animals susceptible
 - Act as sentinels
 - Guinea pigs, mice, hamsters
- Enzootic strains do not cause disease in animals

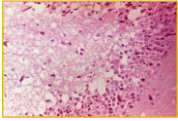
www.cdc.gov/nczod/dzdx/vee

Most other domestic animals, including swine, cattle, and chickens, have not shown clinical signs after natural infection, but rabbits and dogs that were experimentally infected died shortly after inoculation. Laboratory animals vary in their response to the disease, but many are susceptible and act as sentinels for disease prevalence. While guinea pigs experience fever and fatalities without central nervous system signs, mice will experience meningoencephalomyelitis and death. Enzootic strains only cause disease in humans.

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VEE in Animals

- Diagnosis
 - Virus isolation
 - Serology
 - Paired sera with rising titer
 - ELISA IgG or IgM
- Treatment
 - Supportive care
- Vaccine available for horses



VEE can be diagnosed by virus isolation or serology. VEE virus can often be recovered from the blood during the early, febrile stage of disease, but animals with neurologic signs are not usually viremic. Diagnosis is made via serial serum samples monitoring a rise in antibody titer, or by using ELISA for IgG or IgM. Because of the nature of the CNS signs, treatment involves supportive care. Prevention is a better option, and a vaccine is available in the U.S. for horses.

[Photo: A photomicrograph of mouse brain tissue after dying of Venezuelan encephalitis. It reveals neural necrosis and edema. Source: CDC Public Health Image Library.]

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VEE as a Biological Weapon

- Aerosolized VEE
- Human and equine disease occur simultaneously
- Flu-like symptoms in humans
- Possible neurological signs in horses
- Large number of cases in a given geographic area

If a bioterrorism attack were to occur using a viral encephalitis agent, experts feel the most likely agent for weaponization would be Venezuelan equine encephalomyelitis virus. The virus particles would be aerosolized and disseminated, with human disease as the primary event. Equines would also be susceptible, but disease would most likely occur simultaneously, without animals acting as sentinels. Disease symptoms in humans would resemble the flu and be hard to distinguish, so basis of an outbreak would likely be a large number of sick individuals and horses in a given geographic area. Horses may or may not exhibit neurological signs, depending on how the virus was weaponized.


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PREVENTION AND CONTROL






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Management of Mosquito-Borne Diseases

- Source reduction
- Surveillance
- Biological control
- Chemical control
 - Larvicide
 - Adulticide
- Educating the public
 - How to protect themselves



Prevention and control of mosquito-borne diseases involves source reduction, surveillance, biological control, chemical control (larvicides and adulticides), and educating the public on how to protect themselves. [Photo: *Culex* mosquito. Source: Wikimedia Commons]

<p>S I d e 5 7</p>	<p style="text-align: center;">Source Reduction</p> <ul style="list-style-type: none"> • Mosquito habitats <ul style="list-style-type: none"> - Make unavailable or unsuitable for egg laying and larval development • Minimize irrigation and lawn watering • Punch holes in old tires • Fill tree holes with cement • Clean bird baths, outside waterers, fountains 	<p>By trying to eliminate the source of mosquitoes, humans and animals can decrease their risk of exposure. Efforts should be concentrated on making habitats for egg laying and larval development unsuitable. Less irrigation should be utilized or ditches managed so that water does not sit undisturbed for more than 2 days. Other actions include punching holes in old tires to encourage drainage, filling tree holes with cement, and cleaning bird baths and outside animal waterers at least once a week. [Photo: Domestic mosquitoes are often found breeding in old discarded tires. Source: CDC Public Health Image Library]</p>
<p>S I d e 5 8</p>	<p style="text-align: center;">Source Reduction Cont'd</p> <ul style="list-style-type: none"> • Drain or fill temporary pools with dirt • Keep swimming pools treated and circulating <ul style="list-style-type: none"> - Avoid stagnant water • Open marsh water management <ul style="list-style-type: none"> - Connect to deep water habitats and flood occasionally - Fish access 	<p>Further source reductions include draining or filling temporary pools with dirt and keeping swimming pools treated and circulating to avoid stagnant water; eliminating puddles in gutters, around faucets, air conditioners, and septic tanks; and managing open marshes by connecting mosquito areas and shallow ditches to deep water habitats that allow drainage or fish access. [Photo: Domestic mosquitoes are seen here breeding in jars of rainwater. Source: CDC Public Health Image Library]</p>
<p>S I d e 5 9</p>	<p style="text-align: center;">Surveillance</p> <ul style="list-style-type: none"> • Mosquito trapping and testing for viral presence • Record keeping <ul style="list-style-type: none"> - Weather data, mosquito larval populations, adult flight patterns • Sentinel chicken flocks <ul style="list-style-type: none"> - Blood test and ELISA to monitor seroconversion 	<p>Many states and local governments utilize surveillance programs when there are established risk factors for human disease present. This may include mosquito trapping and testing for viral presence in a given area. When established mosquito larval and adult threshold populations are exceeded, control activities can be initiated. For example, heavy winter snow fall followed by heavy spring rains can lead to flooding and more standing water for mosquitoes to lay eggs upon. Seasonal weather patterns and historical records are kept to predict mosquito larval occurrence and adult flights. Instituting surveillance programs using sentinel chicken flocks and mosquito trapping and testing are ways to monitor disease prevalence in a given area. Blood testing birds, either wild or young, unexposed chickens, and monitoring viral seroconversion or antibody titer allows authorities time to alert the general public if there is concern. These are common practices for EEE. [Photo: Sentinel chicken flock. Source: Danelle Bickett-Weddle/CFSPH]</p>
<p>S I d e 6 0</p>	<p style="text-align: center;">Biological Control</p> <ul style="list-style-type: none"> • Predators, natural and introduced, to eat larvae and pupae <ul style="list-style-type: none"> - Mosquito fish <ul style="list-style-type: none"> • <i>Gambusia affinis</i>, <i>G. holbrooki</i> • <i>Fundulus</i> spp., <i>Rivulus</i> spp., killifish • Other agents have been used but are not readily available • Copepods  	<p>Biological control involves using different predators that eat mosquito larvae and pupae. The mosquito fish, <i>Gambusia affinis</i> and <i>G. holbrooki</i> are the most commonly used supplemental control because they are easily reared. They are indiscriminate feeders, though, and may eat other things, such as tadpoles, zooplankton, aquatic insects and other fish eggs. Some naturally occurring fish, such as <i>Fundulus</i> spp., <i>Rivulus</i> spp., and killifish, play an important role in controlling mosquitoes in open marsh water and rotational impoundment management. There are other agents, such as fungi, protozoa, and nematodes, that have been tried but are not readily available. A predacious copepod, <i>Mesocyclops longisetus</i>, preys on mosquito larvae and is a candidate for local rearing with <i>Paramecium</i> spp. for food. [Note: Copepods are tiny aquatic crustaceans (shrimp, crabs lobster, and relatives) that are widespread in both fresh and salt water habitats.] [Photo: (Top) <i>Gambusia holbrooki</i> (Eastern mosquitofish). Source: Wikimedia Commons; (Bottom) Adult copepod. Source: University of Florida Extension at http://edis.ifas.ufl.edu/in490]</p>

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Chemical Control

- Essential when:
 - Source reduction not effective
 - Surveillance shows increased population of virus-carrying mosquitoes
- Requires properly trained personnel
- Larvicides, adulticides
- Toxic to many birds, fish, wildlife, aquatic invertebrates, honeybees
- Human exposure is uncommon

Chemical control is often warranted when source reduction is not enough and surveillance shows an increased population of virus-carrying mosquitoes. All insecticide use requires proper training by the personnel applying it, and can be targeted at the immature (larvicides) or adult (adulticides) mosquitoes. While it is limited, there is a risk of toxic effects on nontarget organisms, such as birds, fish, wildlife, aquatic vertebrates, and honeybees, so low levels of pesticide and proper training of applicators are used. Humans are often concerned with the use of chemicals, but low application rates, ultra low volume (ULV) methods, spraying at night while people are indoors, and notifying the public prior to application all decrease exposure risks.

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Chemical Control

- Federal Food Drug and Cosmetic Act limits the quantity of adulticide used
 - Due to wind drift onto agricultural crops
- Method used varies
 - Type of target mosquito
 - Type of targeted habitat
 - Aerial spraying covers wide area
- Funding provided by state or local government
 - Rarely federal

To further prevent human exposure, the Federal Food Drug and Cosmetic Act (FFDCA) limits the quantity of poisonous or deleterious substances added to food, specifically adulticides carried by wind drift over agricultural crops. The method selected depends on the type of mosquitoes that need to be controlled and the targeted habitat. Aerial spraying can cover a wide geographic area to control nuisance mosquitoes in emergency situations. Costs for such application are often covered by state or local emergency funds, and rarely by federal funds unless a natural disaster has occurred.

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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

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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Larvicides

Name	Product (Larvae, Pupae, Adult)
Temephos	Abate (L)
Methoprene	Altosid (L)
Oils	BVA, Golden Bear (L, P)
Monomolecular film	Agnique (L, P)
<i>Bacillus thuringiensis israelensis</i> (BTI)	Aquabac, Bactimos, LarvX, Teknar, Dunks (L)
<i>Bacillus sphaericus</i>	VectoLex (L)
Pyrethrins	Pyrenone, Pyronyl (A, L)

This chart depicts the various types of larvicides used in the United States, with their chemical or biological name, as well as the commercial product name. There may be others on the market that this chart does not cover.

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Adulticides

- Necessary when other control measures unsuccessful
- Least efficient
- Proper type and time of application helps efficacy
 - Ultra low volume (ULV) foggers
 - 1 ounce per acre
 - Small droplets contact and kill adults



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 that settle on the ground or vegetation 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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Adulticides	
Chemical Name	Product
Malathion	Fyfanon, Atrapa, Prentox
Naled	Dibrom, Trumpet
Fenthion	Batex
Permethrin	Permanone, AquaResilin, Biomist, Mosquito Beater
Resmethrin	Scourge
Sumithrin	Anvil

This chart displays the various types of chemicals used as adulticides, namely the organophosphates, malathion, and naled. Natural pyrethrins, fenthion, and synthetic pyrethroids, such as permethrin, resmethrin, and sumithrin, and their product names are also listed.

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Personal Protection

- Stay inside during the evening when mosquitoes are most active
- Wear long pants and sleeves
- Use mosquito repellent when necessary
 - Follow label directions
 - DEET
 - Do not use on pets



Humans can protect themselves in two ways: reduce contact with mosquitoes and reduce the population of infected mosquitoes in the environment. Personal protection involves reducing time outdoors in the early evening hours when mosquitoes are most active, wearing long pants and long sleeved shirts, and applying mosquito repellent containing DEET to exposed skin areas. DEET can be sprayed on clothing, but this is unnecessary because the underlying skin is protected from insect bites by the clothing. **DEET should not be used on pets.** [Photo: Applying mosquito repellent. Source: Radford Davis/CFSPH]

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Personal Protection

- Make sure window and door screens are "bug tight"
- Replace your outdoor lights with yellow "bug" lights
 - Bug zappers are not very effective
- ULV foggers for backyard use
- Keep vegetation and standing water in check around the dwelling

It is important to protect yourself by making sure mosquitoes cannot enter your home. Check window screens for holes and make sure they are bug tight so as not to allow entry. Replacing your outdoor lights with yellow bulbs decreases the attractiveness of many bugs to entry ways. Bug zappers are not specific to mosquitoes and are not much help with control. Ultra low volume foggers can be purchased for backyard use to decrease the mosquito population in the event that people will be outdoors during mosquito feeding hours. Keep vegetation and standing water in check around the dwelling to avoid larval habitats.

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Internet Resources

- CDC Division of Vector Borne Infectious Diseases-Arboviral Encephalitides
 - <http://www.cdc.gov/ncidod/dvbid/arbor/>

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