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Western Equine Encephalitis

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Overview

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

In today's presentation we will cover information regarding the agent that causes western equine encephalomyelitis and its epidemiology. We will also talk about the history of this diseases, how it is transmitted, species that it affects, and clinical signs seen in humans and animals. Finally, we will address prevention and control measures, as well as actions to take if western equine encephalomyelitis is suspected. [Photo: Horses in field. Source: U.S. Department of Agriculture]

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THE ORGANISM

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The Virus

- Western equine encephalomyelitis virus
  - Family Togaviridae
  - Genus *Alphavirus*
- Mosquito-borne
- Disease
  - Encephalitis in humans and horses

Western equine encephalomyelitis (WEE) results from infection by the respectively named virus in the genus *Alphavirus* (family Togaviridae). The Western equine encephalomyelitis virus (WEEV) is closely related to some other alphaviruses including Sindbis, Ft. Morgan Aura and Highlands J viruses; however, these viruses are considered to be distinct species. WEE is a mosquito-borne, viral infection that can cause severe encephalitis in horses and humans.

[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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HISTORY

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WEE History

- 1930
  - Isolated from horse brain
  - California; 50% case fatality rate
- 1933
  - *Aedes aegypti* experimentally infected with WEE
    - Virus transmitted to guinea pigs
    - Virus transmitted to horses (1936)
- 1938
  - Isolated from human brain

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 *Aedes aegypti* 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.

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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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EPIDEMIOLOGY

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WEE Epidemiology

- *Culex tarsalis*
  - Reaches high populations in 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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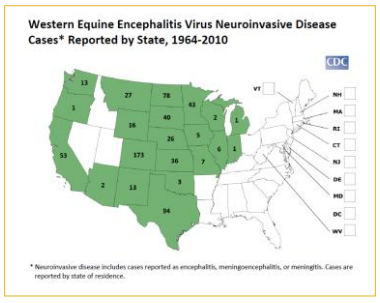

This map shows the distribution of human Western equine encephalitis virus neuroinvasive disease cases in the U.S. – reported by state between 1964-2010. For this period, WEEV neuroinvasive disease cases have been reported in Arizona (2), California (53), Colorado (173), Illinois (6), Indiana (1), Iowa (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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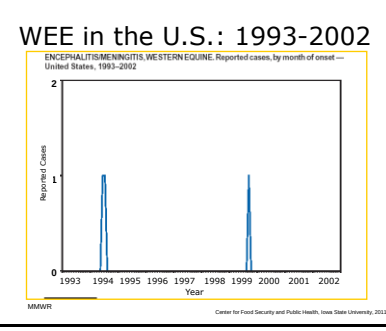

This graph depicts the number of reported WEE human cases in the United States from 1993-2002. During 1997, 35 strains of WEE virus were isolated from mosquitoes collected in Scotts Bluff County, Nebraska, but no human cases were reported. During the time period 1964-2002, an average of 18 human cases (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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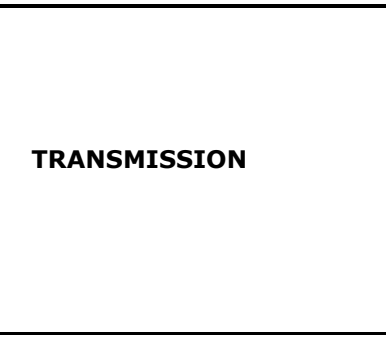

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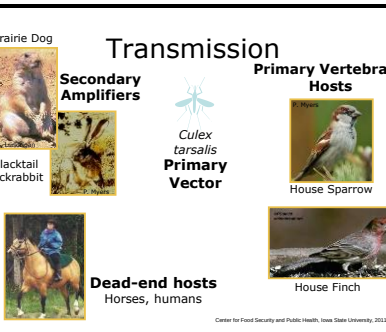

Transmission of WEE occurs primarily in areas west of the Mississippi River and involves a mosquito-vertebrate-mosquito cycle. *Culex tarsalis* is the primary vector for transmission to a variety of asymptomatic, primary amplifying hosts, namely the house sparrow (*Passer domesticus*) and the house finch (*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	*Culex tarsalis*	House sparrow, Red-winged blackbird, Magpie	Blacktail jackrabbit, Kangaroo rat
CA	*Culex tarsalis* *Aedes melanimon*	House sparrow House finch	Blacktail jackrabbit, Western gray squirrel
TX	*Culex tarsalis, Cx. quinquefasciatus* *Aedes vexans*	House sparrow	Blacktail jackrabbit, Prairie dog
NM	*Aedes dorsalis, Ae. campestris*		

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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DISEASE IN HUMANS

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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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DISEASE IN ANIMALS

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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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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: *Culeseta* mosquito. Source: Wikimedia Commons]

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Source Reduction

- Mosquito habitats
  - 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

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]

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Source Reduction Cont'd

- Drain or fill temporary pools with dirt
- Keep swimming pools treated and circulating
  - Avoid stagnant water
- Open marsh water management
  - Connect to deep water habitats and flood occasionally
  - Fish access

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]

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Surveillance

- Mosquito trapping and testing for viral presence
- Record keeping
  - Weather data, mosquito larval populations, adult flight patterns
- Sentinel chicken flocks
  - Blood test and ELISA to monitor seroconversion

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]

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Biological Control

- Predators, natural and introduced, to eat larvae and pupae
  - Mosquito fish
    - *Gambusia affinis, G. holbrooki*
    - *Fundulus* spp., *Rivulus* spp., killifish
- Other agents have been used but are not readily available
- Copepods

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

food. [Note: Copepods are tiny aquatic crustaceans (shrimp, crabs lobster, and relatives) that are widespread in both fresh and salt water habitats.]

[Photo: (Top) *Gambusia holbrooki* (Eastern mosquitofish). Source: Wikimedia Commons; (Bottom) Adult copepod. Source: University of Florida Extension at http://edis.ifas.ufl.edu/in490]

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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)
Bacillus thuringiensis israelensis (BTI)	Aquabac, Bactimos, LarvX, Teknar, Dunks (L)
Bacillus sphaericus	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 repellant. 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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Acknowledgments

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