Western equine encephalomyelitis (WEE) virus was initially isolated from sick horses in 1930 and from a fatal human case in 1938. This virus causes an acute febrile illness in equines and humans, characterized in its most severe form by signs and symptoms of inflammation and injury of the meninges, brain and spinal cord. Large outbreaks occurred in the north central United States in 1941 and in the Central Valley of California in 1952. Both sporadic cases and small epidemics continue to occur throughout the western states.

While the exact number of equine cases is generally not known, it can be conservatively estimated that for every reported human case there are several hundred horse cases. The occurrence of human disease is always associated with equine encephalitis in the same area, and an equine epidemic often precedes the appearance of human cases. Where most horses have been vaccinated against WEE, this early warning of viral transmission may be less obvious.

WEE occurs in mid to late summer in the Coachella Valley, with the case incidence being higher in the rural population than in the urban residents. About one-third of the reported cases are in children under 5 years of age. Infants under 1 year of age are most susceptible to developing severe encephalitis. Among the reported adult cases, the attack rates are higher in males than in females. Infection is often abortive or undifferentiated, and there is a fairly low case-fatality rate of 3 to 4 percent.

The incubation period of WEE is usually 5 to 10 days. The onset of the illness can be sudden (especially in adults) or characterized by a 2 to 4 day period of lethargy, fever, and headache (especially in children). Recovery is generally complete with rare instances of permanent neurological symptoms.

Western equine encephalomyelitis virus is maintained in a primary enzootic (animal to animal) transmission cycle involving wild birds and mosquitoes. In the western United States, the temporal and spatial distribution of WEE is restricted to the distribution and abundance of its known vector, the mosquito *Culex tarsalis*. While *Culex tarsalis* can be found breeding in a wide variety of standing water, habitats range from unattended swimming pools, irrigated date and citrus orchards to salt marshes along the Salton Sea.

Fairly substantial population levels of *Culex tarsalis* can be found during the summer, and active-breeding populations can be found throughout the winter in Southern California.

Wild birds serve as the basic viral reservoir hosts during the epidemic season. Both nestling and adult birds of many species serve as effective viremic hosts. Some species, such as the house finch and house sparrow, appear to play especially important roles. Domestic fowl develop viremias sufficient to infect *Cx. tarsalis* but probably contribute relatively little to viral amplification.

The over-wintering mechanisms of WEE are not completely understood, although several theories have been investigated. Among those over-wintering mechanisms suggested are viral reservoir in local bird species, viral reservoir in hibernating arthropod vectors and vertebrates, and viral reintroduction by migrating birds. Horses and humans are “dead end” hosts for WEE virus; viremia levels are insufficient to serve as a source for viral infection.

The mosquito and vector control districts located in known WEE endemic areas utilize a variety of quantitative and qualitative surveillance methods to determine the seasonal and temporal distribution of the virus, vector, and bird populations. Among the numerous employed methods of surveillance are: mosquito population assessments using CO2-baited traps and standard New Jersey Light Traps, mosquito larval surveillance, blood sampling from sentinel chickens, and mosquito sampling for virus detection.