Alphaviruses that are related to VEE include the viruses that cause Eastern Equine Encephalitis (EEE) and Western Equine Encephalitis (WEE). EEE is a very serious, but quite rare arbovirosis in the east of the USA, but also occurs sporadically in Central and South America. An incubation period of 7-10 days, fever, meningism, severe encephalitis and a mortality rate that can be as high as 50% characterise the disease. The epidemic potential became evident in 1938. After a severe storm in Boston, Massachusetts there was a major outbreak with a high mortality rate. What was the connection between the storm and the disease? Birds form the reservoir. The virus is transmitted between birds by mosquitoes such as Culiseta melanura. This mosquito lays its eggs in dark underground hollows in an acid soil, such as root hollows in marsh cypresses or red maple trees. It is an unusual habitat for oviposition (egg-laying). The larvae are not in open water and are not easy to find. Such places easily become water-logged after heavy rainfall. In this way huge numbers of mosquitoes can appear simultaneously. Transmission between birds then increases. More than 75 different types of bird can be infected. When mosquitoes that bite both birds and man are infected (such as Aedes vexans, Coquilletidia perturbans), the infection can be transmitted to man. Culex tarsalis is also important in transmission. Horses and donkeys can be infected. In these animals the course of the infection is often dramatic and death among horses can precede an epidemic. Surveillance is carried out with sentinel birds. If there is a threat of an epidemic, insecticides are sprayed, e.g. by ULV (ultra low volume spraying).

In the west of the USA WEE occurs sporadically in man and animals. In other regions of the USA and South America WEE also occurs, but until now apparently only in animals. It is not known whether this is to do with the different antigenic types in North and South America. Most of the infections in adults are pauci- or asymptomatic. After an incubation period of 5-10 days there is a gradual onset of fever, malaise, headache, neck stiffness and dizziness. In serious cases this develops into stupor, coma, flaccid and spastic paralysis. There is pleocytosis in the cerebrospinal fluid as well as an increase in the protein content. Children often have permanent neurological sequelae. The mortality rate among symptomatic patients is about 10%.