

Eastern, Western and Venezuelan Equine Encephalomyelitis

Sleeping Sickness

Eastern Equine Encephalomyelitis (EEE), Eastern Equine Encephalitis, Eastern Encephalitis

Western Equine Encephalomyelitis (WEE), Western Equine Encephalitis

Venezuelan Equine Encephalomyelitis (VEE), Peste Loca, Venezuelan Equine Encephalitis, Venezuelan Encephalitis, Venezuelan Equine Fever

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Etiology

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.

Eastern equine encephalomyelitis virus

Until recently, Eastern equine encephalomyelitis virus (EEEV) contained four genetic lineages. Lineage I was considered to be the North American variant of EEEV, while lineages II, III and IV were the South American variants. The latter three lineages have now become a new viral species, Madariaga virus. Unless otherwise specified, “EEEV” in this factsheet refers to all viruses formerly classified under this name, rather than lineage I viruses alone.

The North American EEEV seems to be more virulent than Madariaga virus in people, and under some conditions, it is also more pathogenic in experimentally infected nonhuman primates (e.g., marmosets), sparrows and rodents. Comparative studies in horses have not been published, but severe illness has been reported in this species in both North and South America.

Western equine encephalomyelitis viruses

The Western equine encephalomyelitis virus complex contains western equine encephalomyelitis virus (WEEV) and several closely related alphaviruses including Sindbis virus, Whataroa virus, Fort Morgan virus (and variants Stone Lakes virus and Buggy Creek virus), aura virus, and highlands J virus.. WEEV is the most important virus in this complex in the Western Hemisphere, although highlands J virus and Fort Morgan virus can affect some birds. Sindbis virus and Whataroa virus cause a febrile illness with polyarthritis in humans, but occur only in the Eastern Hemisphere, and are not discussed in this factsheet. Aura virus, found in South America, has not been linked to any illness in humans or animals.

Venezuelan equine encephalomyelitis viruses

The Venezuelan equine encephalomyelitis complex contains a number of viruses, which have been classified into 6 viral subtypes, I to VI, with subtype I further subdivided into five antigenic variants or serovars, AB to F. The currently recognized viral species in this complex are Venezuelan equine encephalomyelitis virus (VEEV), which contains variants AB, C, D and E in subtype I (i.e., variants I-AB, I-C, I-D and I-E), Mosso das Pedras virus (variant I-F), Everglades virus (subtype II), Mucambo virus (subtype III variants A, C and D), Tonate virus (subtype III variant B), Pixuna virus (subtype IV), Cabassou virus (subtype V) and Rio Negro virus (subtype VI). One isolate of Tonate virus, which was detected in the U.S. Rocky Mountains region in the 1970s, is also called Bijou Bridge virus. VEE complex viruses are sometimes referred to by their subtype and variant designation, rather than their species name.

VEE complex viruses are divided into epidemic (or epizootic) and enzootic (or endemic) groups, based on their epidemiological characteristics. All viruses except VEEV variants I-AB and I-C are considered to be enzootic. Enzootic VEE viruses occur in limited geographic areas, where they are maintained in cycles involving wild animals. They are not amplified in equids, and do not usually cause disease in these animals. In contrast, epidemic VEE viruses are detected only sporadically, are amplified in equids, and can cause extensive epidemics affecting both equids and

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humans. The origins of epidemic VEE viruses are uncertain, as I-AB and I-C viruses do not seem to be maintained in natural cycles between outbreaks. Some evidence suggests that they may arise when mutations in enzootic VEE viruses allow efficient amplification in horses, and die out once the epidemic ends.

One enzootic I-E virus strain, which has been detected in Mexico since the 1990s, differs from other enzootic viruses in that it affects horses. This virus has caused extensive outbreaks in Mexico, although it has not spread further. Like other enzootic VEE viruses, it is not thought to be amplified in equids.

Species Affected

Eastern equine encephalomyelitis

Passerine birds are thought to be the principal reservoir hosts for North American EEEV in natural cycles, but small mammals (e.g., rodents) might also amplify the virus. The primary reservoir hosts for Madariaga virus (South American EEEV) are still uncertain, but small mammals might play a more prominent role. Some experiments have suggested that reptiles (especially snakes) might help maintain EEEV over the winter. Domesticated mammals including equids are not important in virus amplification.

Most infections in passerine birds appear to be asymptomatic, although a few species become ill after experimental inoculation. Clinical cases have been reported in some non-passerine birds including chukar partridges, pheasants, turkeys, ratites (emus, ostriches), psittacine birds, pigeons (*Columba livia*), house sparrows (*Passer domesticus*), egrets, glossy ibises (*Plegadis falcinellus*), whooping cranes (*Grus americana*) and African penguins (*Spheniscus demersus*). Among mammals, EEEV mainly causes disease in horses and other equids, but clinical cases have also been reported in sheep, cattle, dogs, South American camelids (llamas and alpacas), pigs, deer and a captive harbor seal (*Phoca vitulina*), as well as in some experimentally infected rodents and nonhuman primates. Some other species susceptible to infection, without reported disease to date, include goats, moose (*Alces alces*), certain rodents, bats, reptiles and amphibians.

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Passerine birds are the usual reservoir hosts for WEEV, but this virus may also cycle in blacktail jackrabbit (*Lepus californicus*) populations. Reptiles have been proposed as possible overwintering hosts. Domesticated mammals including equids are not important in virus amplification.

WEEV causes disease in equids and some species of birds such as emus, turkeys, pheasants and chukar partridges. Other species reported to be susceptible to infection (usually asymptomatic) include cattle; various small mammals including squirrels, other rodents and snowshoe hares (*Lepus americanus*); opossums; and snakes, tortoises and frogs.

Other WEE complex viruses

Highlands J virus mainly seems to infect wild birds. Although this virus is not known to be a significant cause of illness in mammals, it was isolated from the brain of at least one horse with encephalitis. It can also cause disease in experimentally infected young chickens and partridges, and turkeys of various ages.

Fort Morgan virus occurs in cliff swallows (*Petrochelidon pyrrhonota*) and house sparrows, and can affect house sparrow nestlings. It is not known to infect other species.

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Wild rodents are thought to be the usual reservoir hosts for enzootic VEE viruses, but birds may be involved in a few cycles (e.g., the Bayou Bridge variant of Tonate virus). Although rodent reservoir hosts in endemic areas seem to be unaffected, other wild and laboratory (or pet) rodents can become ill. Mice and hamsters are generally more susceptible than guinea pigs. Enzootic VEE viruses can also infect opossums (*Didelphis marsupialis*), bats and various other mammals including dogs. They are not known to cause any illness in equids, other domesticated livestock, dogs or cats, with the exception of one Mexican I-E variant, which is pathogenic for equids. Horses do not seem to be efficient amplifying hosts for any enzootic VEE viruses, including this variant.

Epidemic VEE viruses mainly affect equids and are also amplified in these animals. These viruses can infect wild and laboratory rodents, and cause severe disease in some species (including guinea pigs, mice and hamsters); however, there is no evidence that they are maintained in rodents or other animals between epidemics. Infections have also been reported in other mammals (e.g., pigs, cattle, goats, sheep, dogs, rabbits) and some birds, but most infections appear to be subclinical.

Zoonotic potential

Human illnesses have been reported after infection with EEEV, Madariaga virus, WEEV, epidemic VEE viruses and most enzootic VEE viruses. EEEV in North America is generally thought to be more virulent for humans than Madariaga virus in South America; however, both viruses can cause severe illness. Highlands J virus and Fort Morgan virus do not appear to affect people.

Humans infected with epidemic strains of VEEV can develop viremia sufficient to infect mosquitoes, but are not thought to be important in the epidemiology of this disease. People do not appear to transmit EEEV or WEEV to mosquitoes.

Geographic Distribution

All EEE and VEE complex viruses and most WEE complex viruses occur only in the Western Hemisphere.

WEEV has been isolated from western North America, including Canada, and as far south as Argentina. Highlands

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J virus circulates in the eastern U.S., while Fort Morgan virus (with its variants) is widespread in North America.

EEEV has been detected in eastern Canada, all U.S. states east of the Mississippi, and some additional states such as Arkansas, Minnesota, South Dakota and Texas. This virus is usually associated with swamps and marshes, and its distribution is not homogeneous: it is particularly common along the Gulf coast from Texas to Florida, along the Atlantic coast, and in some midwestern states around the Great Lakes. Madariaga virus occurs in parts of Central and South America, especially along the Gulf coast.

Enzootic VEE viruses have varying distributions in parts of Mexico, South and Central America. They are absent from Canada and most of the U.S.; however, Everglades virus (subtype II) occurs in Florida, and Tonate virus (variant III-B) was detected in Colorado and South Dakota in the 1970s. Enzootic VEEV I-E viruses pathogenic for equids have been detected only in Mexico; I-E viruses currently found in other parts of Latin America do not seem affect these animals.

Epidemics caused by epidemic VEE viruses (VEEV I-AB and I-C) tend to occur in northern South America, but also affect other parts of South and Central America. Some outbreaks have spread into North America.

Transmission

Eastern equine encephalomyelitis

In North America, EEEV is normally maintained in wild bird populations. *Culiseta melanura*, a mosquito that preferentially feeds on birds, is the most important vector in this sylvatic cycle. Other, mosquito species that feed on both birds and mammals (“bridge vectors”) may transmit EEEV to humans and domesticated mammals; however, recent evidence suggests that *C. melanura* may also play a direct, and perhaps significant, role. *Culex* spp. might be the main vectors for Madariaga virus (EEEV lineages II-IV) in South American sylvatic cycles. Other arthropods including chicken lice, chicken mites (Dermanyssidae) and assassin bugs can be infected with EEEV, and chicken mites can transmit the virus experimentally. How EEEV survives the winter in cold climates is still uncertain, but several mechanisms, including persistence in reptiles, prolonged persistence in birds, vertical transmission in mosquitoes, and periodic reintroduction by migrating birds have been suggested.

When birds are in close contact, EEEV can sometimes spread by methods not involving arthropods. This has been documented in captive game birds (e.g., pheasants), which can be infected by the oral route. The presence of large amounts of virus on the feathers of these birds suggests that transmission might occur by pecking, feather picking or preening. Cannibalism could also play a role. Emus can shed large amounts of virus in rectal and oral secretions, and in regurgitated material.

Horses, humans and other mammals are generally considered to be incidental (dead end) hosts for EEEV, but some horses develop a transient viremia sufficient to infect mosquitoes, and horse to horse transmission has been demonstrated by this route in the laboratory.

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WEEV is normally maintained in wild bird populations, and *Culex tarsalis* appears to be the most important vector for this virus in North America. WEEV can also be transmitted by other mosquitoes, especially some members of the genus *Aedes*. A sylvatic cycle between the mosquito *Aedes melanimon* and blacktail jackrabbits (*Lepus californicus*) has also been reported, probably after they become infected from the bird/mosquito cycle. Overwintering mechanisms for WEEV are uncertain, but similar mechanisms as for EEEV have been proposed.

Horses and humans infected with WEEV do not develop significant viremia, and are true dead-end hosts. This virus can cross the placenta in humans, and congenitally infected infants have been reported.

Other WEEV complex viruses

Highlands J virus is transmitted by *Culiseta melanura* mosquitoes, but the main vector for Fort Morgan virus is the cimicid swallow bug (*Oeciacus vicarius*), an ectoparasite of swallows.

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Enzootic VEE viruses are mainly thought to cycle between mosquitoes in the genus *Culex* and wild small mammals, especially rodents. In the North American Rocky Mountains, the cycle for Tonate virus (Bijou Bridge virus) was reported to involve birds and the swallow bug *Oeciacus vicarius*. Equids do not amplify enzootic VEE viruses.

Horses are the main amplifiers for epidemic VEE viruses. Other mammals do not seem to be epidemiologically significant in transmission, although sufficient viremia to infect mosquitoes has been reported in humans, and occasionally in other species (e.g., cattle, pigs, dogs). Many species of mosquitoes can transmit epidemic VEEV, and efficient vectors have been described in the genera *Aedes*, *Anopheles*, *Culex*, *Mansonia*, *Psorophora* and *Deinocerites*. Blackflies could be important mechanical vectors for epidemic strains during some outbreaks. Mites are also capable of transmitting these viruses mechanically. Ticks including *Amblyomma cajennense* and *Hyalomma truncatum* can be infected by both enzootic and epidemic VEEV strains, although their role in nature (if any) is unclear. Horses can shed epidemic VEEV in body fluids, and some authorities suggest that these viruses might be spread occasionally by direct contact or via aerosols. However, there are no reports of direct transmission between horses, or from horses to humans, in nature.

Most people are infected by exposure to VEEV-infected arthropods, but cases have also been documented after laboratory accidents or exposure to aerosolized debris

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from the cages of infected laboratory rodents. Person-to-person transmission has never been reported, although VEEV has been detected in pharyngeal secretions and horizontal transmission is theoretically possible. VEEV can cross the placenta in pregnant women.

VEEV is reported to persist for a time in the environment, in dried blood and exudates. In a recent experiment, inactivation of 90% of an epizootic strain of VEEV on a glass surface took approximately 98 hours at room temperature (20-25°C) in the dark. Whether viruses in the environment would infect animals or humans this long is uncertain, as the researchers used various procedures such as sonication to recover as much bound virus from the glass as possible. The persistence of EEEV and WEEV in the environment is unknown, but EEEV has been isolated from feather quills for up to 6 days.

Infections in Animals

Incubation Period

The incubation period for WEE or EEE in horses is 5-14 days. The initial signs of VEE can occur 1-5 days after infection, although neurological signs usually appear around day 5.

Clinical Signs

Eastern and western equine encephalomyelitis complex viruses in equids

Eastern and western equine encephalomyelitis are very similar in horses, although the course of EEE may be shorter. Some animals may have asymptomatic infections or mild cases without neurological signs; however, in classic cases of encephalitis, an initial prodrome characterized by nonspecific signs (e.g., fever, anorexia and depression) is followed by neurological signs that may include altered mentation, hypersensitivity to stimuli, involuntary muscle movements, impaired vision, behavioral changes (e.g., aimless wandering, head pressing, circling), an inability to swallow, ataxia, paresis, paralysis and/or convulsions. Periods of excitement or intense pruritus have been reported, and 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 within a

few days, particularly when infected with EEEV. Horses that recover from encephalitis have a high incidence of residual deficits.

Other WEEV complex viruses in equids

Highlands J virus has been linked rarely with encephalitis in horses. Fort Morgan virus is not known to affect mammals.

Venezuelan equine encephalomyelitis in equids

Infections with epidemic VEE viruses may be asymptomatic, mild or resemble clinical EEE and WEE. In symptomatic horses, a febrile prodrome with depression, tachycardia, and inappetence is sometimes followed by neurological signs indicative of encephalitis. Some animals also have diarrhea and colic. Death can occur within hours after the onset of neurological signs; after a protracted illness accompanied by dehydration and extreme weight loss; or in animals without signs of encephalitis. Sudden death has also been reported. Animals that recover may have permanent neurological signs.

Enzootic VEE viruses usually infect equids subclinically or cause only mild, nonspecific clinical signs. However, an I-E strain found in Mexico can cause severe illness with encephalitis and high mortality.

Equine encephalomyelitis viruses in other mammals

Neurological signs caused by EEEV have been reported in various animals including llamas, alpacas, deer, sheep, cattle, dogs, pigs and a harbor seal. In one published report, all affected dogs were young (≤ 6 months of age), and the clinical signs included fever and diarrhea as well as signs of encephalitis. The clinical signs in these dogs progressed rapidly to recumbency, seizures and other CNS signs within 24-36 hours, and all affected dogs died or were euthanized. During outbreaks in pigs, the illness was most severe in nursing piglets, with reported signs including fever, lethargy, frank CNS signs and high mortality in some outbreaks. Emaciation, dyspnea and excessive salivation, as well as neurological signs, were documented in white-tailed deer (*Odocoileus virginianus*). A young sheep remained alert and maintained a good appetite until it was euthanized, despite fever and neurological involvement that progressed from front limb incoordination to forelimb and hindlimb paralysis with muscle fasciculation and paddling. Seizures were the main sign in a harbor seal, together with anorexia and lethargy; the latter signs may also have been related to molting.

Deaths have been reported in various mammals including rabbits, goats, dogs and sheep during some VEE epidemics; however, laboratory experiments suggest that illnesses in most of these species are unusual. Fatal infections have been documented in experimentally infected rabbits; however, goats, sheep and dogs inoculated with epidemic VEE viruses had few or no clinical signs

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(although some dogs infected via mosquitoes developed leukopenia and lymphopenia in addition to fever). Susceptible rodents can develop nonspecific signs (e.g., lethargy, anorexia, weight loss) and/or neurological signs after inoculation, and nonspecific febrile illness has been reported in nonhuman primates.

Western and Eastern equine encephalomyelitis viruses in birds

WEEV and EEEV infections are asymptomatic in many birds; however, EEE outbreaks have been reported in several avian species, with syndromes ranging from neurological signs to hemorrhagic enteritis. Clinical signs reported in pheasants included fever, depression, weakness and profuse diarrhea, in addition to neurological signs such as incoordination, circling, tremors, and partial or complete paralysis of the legs. Chukar partridges infected with EEEV were dull and listless, typically found with ruffled feathers, sitting on their hocks with the beak on the ground, while lethargy, ataxia and paresis of the legs and neck were reported in whooping cranes. In a colony of African penguins, early signs of anorexia, mild lethargy and intermittent vomiting, were followed by persistent regurgitation, ataxia, seizures, and diarrhea that was mild in most birds but voluminous in a few. Most penguins recovered, but subtle, intermittent ataxia persisted in some birds. Hemorrhagic enteritis, with signs of depression, diarrhea (which may contain varying amounts of blood) and regurgitation, has been reported in ratites. The onset of disease is usually rapid in these birds, and the mortality rate high. EEEV can also cause depression, decreased egg production and death in turkeys. Although adult chickens are usually unaffected, experimentally infected, 2-week-old chickens developed severe depression, followed by abdominal distention and growth retardation. Some of these chickens died. In addition, EEEV has been isolated from psittacine birds with viral serositis.

WEE has been linked less often with disease in birds. WEEV-infected emus can be mildly to severely affected, with clinical signs that may include anorexia, lethargy, weight loss, watery diarrhea or hemorrhagic enteritis, neurological signs and sudden death. Turkeys can experience a drop in egg production and poor egg quality.

Highlands J virus has caused death in experimentally infected young chickens, turkeys and partridges, and nonspecific signs of illness and decreased egg production in adult turkeys. Fort Morgan virus can cause encephalitis and hepatitis in house sparrow nestlings, but is not known to affect other species.

Post Mortem Lesions

The gross lesions of equine encephalitis are usually nonspecific. Equids with VEE may have no lesions in the CNS or there may be extensive necrosis with hemorrhages. Necrotic foci are sometimes seen in the pancreas, liver and heart, but in general, the extracranial lesions are too

variable to be diagnostically useful. Congestion of the brain and meninges has been found in some cases of EEE and WEE, and antemortem trauma can result in ecchymotic hemorrhages with any of the encephalomyelitis viruses. Piglets experimentally infected with EEEV had multifocal necrosis and inflammation in the myocardium, in addition to encephalitis. Most birds affected by EEE or WEE have encephalitis, but hemorrhagic enteritis with multiple petechiae on the viscera has been reported in some species, including EEEV-infected emus.

Microscopic analysis of the brain tissue is often diagnostic. The typical lesion is severe inflammation of the gray matter; neuronal degeneration, infiltration by inflammatory cells, gliosis, perivascular cuffing and hemorrhages may be seen. WEE, EEE and VEE sometimes differ in the location and pattern of the lesions in the brain.

prevalence of infection was 64%, and 93% of clinically affected birds recovered with intensive supportive care. Symptomatic infections have been reported less often in WEEV-infected birds; however, the morbidity rate in eight flocks of WEEV-infected emus ranged from 15% to 50%, and approximately 9% of the birds died.

Venezuelan equine encephalomyelitis

Epidemic VEE viruses arise sporadically, but can cause epidemics that may last for several years. Up to 90% of susceptible equids may be infected, with morbidity rates ranging from 10-40% in some areas to 50-100% in others. Case fatality rates in horses are estimated to be 38-90%.

Most enzootic VEE viruses do not cause serious disease or deaths in horses, but some I-E strains in Mexico have caused limited outbreaks of encephalitis. During some of the initial outbreaks in the 1990s, the case fatality rates were 30-50%.

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Control

Measures to prevent mosquito bites, including the use of repellants and protective clothing (e.g., long pants and long-sleeved shirts) can reduce the risk of infection. Outdoor exposure should be limited at times when mosquitoes are active, especially during outbreaks. Mosquito abatement programs such as habitat modification (e.g. the removal of standing water sources around the home) and/or the application of larvicides or adulticides may reduce the risk of human infection. Improved irrigation management has decreased vector populations in California, where a primary mosquito vector for WEEV is associated with irrigation systems.

During VEE epidemics, controlling these viruses in horses can help prevent human infections. Even when equids are not important in the epidemiology of a disease (i.e., EEE or WEE), cases in horses can provide an early warning for human disease. Surveillance programs in birds (including sentinel chickens) are also helpful in predicting EEE outbreaks. .

Precautions should be taken to prevent exposure to body fluids when performing necropsies on horses. Containment level 3 is required for work with EEEV, WEEV or VEEV in the laboratory. Investigational VEEV and EEEV vaccines may be available for people at high risk of infection, but have limited availability, and are not without side effects.

Morbidity and Mortality

Eastern equine encephalomyelitis

In North America, the annual incidence of EEE varies from 0 to 36 cases, with an average of 5-10 cases per year in the U.S. since the 1960s. Approximately 4-5% of people who become infected with this virus are thought to develop EEE, but studies from the 1950s and 60s suggested that few people may be exposed. Clinical cases of encephalitis occur most often in people over 55 years of age and children younger than 15. Estimates of the case fatality rate vary from 30% to 75% (survival has improved in recent years), and permanent neurological deficits can occur in survivors. Only 10% of patients are estimated to recover fully, and many survivors with severe impairment die within a few years. Permanent neurological damage and death are particularly common in children.

Clinical cases caused by Madariaga virus are infrequently reported in Latin America. During a recent outbreak in Panama, there were no deaths among 13 confirmed clinical cases, although one person with suspected EEE died. Some affected people were, however, hospitalized with severe neurological signs, and sequelae were common in these cases. Antibodies to EEEV were detected in 3% of healthy people living nearby.

Western equine encephalomyelitis

WEE was relatively common in North America at one time. Between 1955 and 1984, an average of 34 confirmed cases were reported annually in the U.S., with a range of 0 to 172. Extensive epidemics were also seen at times, with more than 3000 cases in the U.S. and Canada in 1941, and 375 confirmed cases and nine deaths reported in California in 1952. However, clinical cases have rarely been reported in North America in recent decades. The reason for this decline is uncertain; however, it does not appear to be due to reduced virus virulence. Some studies suggest that seroprevalence in healthy people has also diminished (e.g., from 34% in 1960 to < 3% in the 1990s). WEE is uncommonly reported in Central and South America, but some cases might be attributed to other diseases common in tropical regions.

WEE is usually much milder than EEE in symptomatic cases; the overall case fatality rate is estimated to be 3-4%, although it was as high as 8-15% during a severe epidemic in 1941. Adults tend to be mildly affected or remain asymptomatic, but cases can be more severe in children and the elderly. Approximately 5-30% of young patients, and 56% of infants under a month of age, have permanent neurological damage. Except in infants (≤ 1 year), this damage mainly consists of persistent seizures.

Venezuelan equine encephalomyelitis

VEE can be widespread in human populations during epidemics, and more than 10% of the population in an area may be affected. During these outbreaks, cases usually begin weeks after the first illnesses are noted in horses. Serological studies suggest that enzootic VEE viruses might also cause significant numbers of clinical cases in Latin America; however, they may be misdiagnosed as other diseases such as dengue.

Most infections with epidemic or enzootic VEE viruses are mild or asymptomatic, with an overall case fatality rate estimated to be $\leq 1\%$ in healthy adults. Very young or elderly patients are more likely to develop severe disease. Mild to severe neurological signs may occur in 4-15% of symptomatic VEE cases, mainly in children. In these patients, estimates of the case fatality rate range from 10% to 35%, with the highest rates in children. The prognosis is considered to be excellent in patients who recover.

Internet Resources

Centers for Disease Control and Prevention (CDC)

Eastern Equine Encephalitis.

<http://www.cdc.gov/EasternEquineEncephalitis/>

CDC Diseases A to Z

<http://www.cdc.gov/az/a.html>