



ARBOVIRUSES

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Definition	<p>Arboviruses (arthropod-borne viruses) of significance to the equine population are:</p> <ul style="list-style-type: none"> • Eastern Equine Encephalitis (EEE) • Western Equine Encephalitis (WEE) • Venezuelan Equine Encephalitis (VEE) • West Nile Virus (WNV) <p>EEE, WEE, and VEE are alphaviruses in the family Togaviridae. West Nile virus is a flavivirus in the family Flaviviridae and is unrelated to the alphaviruses.</p>
Alphaviruses	<ul style="list-style-type: none"> • Eastern Equine Encephalitis (EEE) virus: In the United States, the highest number of cases occur in the Southeast states, but cases have occurred in all states east of the Mississippi River and as far west as Texas. Mosquito vectors for EEE include <i>Aedes</i> spp., <i>Coquillettidia perturbans</i>, and <i>Culiseta melanura</i>. • Western Equine Encephalitis (WEE) virus: Cases have principally occurred in the western US and very infrequently in parts of the Midwest. While large equine outbreaks have occurred historically, no equine cases have been reported in the last two decades. The most important mosquito vector of this disease is <i>Culex tarsalis</i>. • Venezuelan Equine Encephalitis (VEE) virus: geographic distribution is restricted predominantly to northern South America as well as Central America and southern Mexico. The epidemic subtypes of the virus IAB and IC emerge usually in Venezuela or Colombia. The disease last occurred in the US (Texas) in 1971. There is a continuing risk of the introduction of VEE. Several efficient vectors have been identified and include members of the genera, <i>Aedes</i>, <i>Anopheles</i> and <i>Culex</i> spp. <p>All alphaviruses (EEE, WEE, VEE) are reportable diseases; consult your State Animal Health Office when disease is suspected.</p>
Clinical Signs	<p>Highly variable; none pathognomonic</p> <ul style="list-style-type: none"> • Moderate to high fever 102.5–104.5°F (39.17–40.28°C) • Inappetence

- Diarrhea (VEE only)
- Dysphagia
- Head pressing
- Circling
- Blindness
- Dementia
- Seizures
- Rapid behavioral changes: somnolence, hyperexcitability, mania, self-mutilation
- Cranial neuropathy: nystagmus, facial nerve paralysis, and weakness of the tongue and pharynx
- Coma
- Death

Mortality:

- EEE: 75–95% (usually within 2–3 days of onset of signs)
- WEE: 20–40% (WEE affects horses less severely than EEE)
- VEE: 40–90%

Survivors: Alphavirus infection can result in long-term neurologic deficits. Horses can exhibit abnormal mentation and/or residual spinal cord abnormalities. Horses affected by EEE and VEE are most likely to exhibit these signs.

Incubation Period

- EEE: 3–7 days
- VEE: 2–4 days
- WEE: 2 days–3 weeks

Transmission

Indirectly, through bites from mosquitoes which become infected with virus following feeding on a viremic animal, most often an avian host. Multiple animal species (including snakes) may seroconvert with or without the appearance of clinical signs. However, only those that develop a viremia of sufficient magnitude and duration can be considered amplifying hosts.

- EEE/WEE: Birds and some snakes develop high-level viremia, leading to infection of feeding mosquitoes which then transmit the virus. Horses (and humans) become infected when high numbers of viremic birds and mosquitoes result in spillover to bridge vectors (mosquitoes that feed on birds as well as humans and horses). Infected horses do not develop viremia of significant magnitude for transmission and are therefore dead-end hosts. Humans can become exposed during necropsy procedures and handling of infected tissues. Appropriate precautions should be taken whenever handling suspect material.
- VEE: In contrast to EEE and WEE, horses infected with VEE develop viremia high enough to transmit to mosquitoes. Because they are

amplifying hosts, horses play an important role in the epidemiology of the disease in both horses and humans.

Diagnostic Sampling, Testing and Handling

IgM-capture ELISA: Single serum sample (red top tube) shipped refrigerated to laboratory by overnight courier. Results of $\geq 1:400$ are confirmatory in horses exhibiting clinical signs consistent with EEE/WEE. Detection of IgM in CSF (if available) is even more conclusive, but death may occur in EEE cases prior to an intrathecal antibody response.

Virus neutralizing antibody titers in paired serum samples in horses that survive: Acute and convalescent samples (red top tubes) collected 2–4 weeks apart and shipped refrigerated to laboratory by overnight courier. Four-fold increase in titers between samples is considered confirmatory in horses exhibiting clinical signs consistent with EEE or WEE and not having been recently vaccinated.

CSF analysis—fluid should be collected in EDTA tube and red top (RT) tube:

- Fluid analysis will be performed in CSF collected in EDTA tube
- CSF WBC count—moderate mononuclear pleocytosis ([CSF Sampling](#))
- CSF total protein—usually >70 mg/dl
- EEE—neutrophilic pleocytosis

PCR and viral isolation may be attempted on CSF of clinically affected horses from EDTA tube for EEE and WEE, but laboratory should be contacted prior to sample collection.

Note: CSF fluid requires prompt evaluation when cytology and fluid analysis is requested. When this is not feasible, direct smear slide mounts may be made, allowed to dry, and express shipped in slide mailers at room temperature to the laboratory.

Post-mortem

A rabies prevention protocol should be followed for ALL horses demonstrating signs of encephalitis that undergo a post-mortem examination. Most causes of viral encephalitis in the horse are also zoonotic for humans. [Rabies Testing Protocols](#)

Post-mortem sample collection requires appropriate precautions to avoid exposure. [Necropsy Procedures](#)

Histopathology: Fix at least one-half of the brain for histopathology. Fresh brain should be submitted for concomitant virus isolation, immunochemistry, and rabies testing. [Brain Removal Information](#)

Practitioners performing necropsies in the field are encouraged to contact a veterinary diagnostic laboratory to which they plan to submit samples for further testing such as histopathology and pathogen identification to be certain they collect the appropriate samples and handle the samples in a manner that will optimize making a definitive diagnosis. For some situations such as neurologic cases, submission of the entire carcass to the diagnostic laboratory



	for post-mortem examination is recommended due to the time and labor required to perform a complete exam and collection of samples from the equine CNS.
Shedding of Virus Following Resolution of Clinical Signs	<ul style="list-style-type: none"> • EEE and WEE infected horses do not develop significant viremia and therefore do not shed virus • VEE infected horses develop significant viremia between 2 and 5 days after infection. Horses can shed virus into the nasopharynx and in the milk (lactating mares)
Environmental Persistence	Alphaviruses are enveloped, but are susceptible to drying, ultraviolet light, and detergent.
Specific Control Measures	<p>Vaccination</p> <ul style="list-style-type: none"> • Infection is preventable with vaccination. Although WEE and VEE have not occurred recently in the US, vaccination should continue as risk of re-emergence exists • After an initial vaccination series (per manufacturer's instructions), administer an annual booster 2–4 month prior to mosquito season. In endemic areas, booster at 4-month intervals during mosquito season. Immunity is short lived (4–6 months), thus requiring frequent boosters • Pregnant mares should be vaccinated one month prior to foaling to increase colostrum protection (See AAEP Vaccination Guidelines) to the foal <p>Vector control</p>
Release of Animals from Isolation	No restrictions need to be placed on recovered animals.
Biosecurity Issues for Receiving Animals	None
Zoonotic Potential	Human risk of exposure is via bite of infected mosquitoes that acquired virus from birds (not horses; exception: VEE) or through handling CNS tissue and/or fluids of infected animal. Precautions are indicated when performing necropsy examinations of neurologic horses.
Resource Information	<p>Long MT, Gibbs EPJ. Equine alphaviruses. In Sellon D, Long M. Equine Infectious Diseases, 2nd ed. Philadelphia, PA: Saunders Elsevier 2014.</p> <p>Long MT. West Nile virus and equine encephalitis viruses: new perspectives. Vet Clin North Am Equine Pract. 2014. 30(3):523-42.</p>