

Arboviruses

Definition

Arboviruses (arthropod borne viruses) of significance to the equine population are:

- Eastern Equine Encephalitis (EEE)**
- Western Equine Encephalitis (WEE)**
- Venezuelan Equine Encephalitis (VEE)**
- [West Nile Virus \(WNV\)](#)

EEE, WEE, and VEE are alphaviruses. West Nile virus is an arbovirus but is unrelated to alphaviruses.

Alphaviruses

Eastern Equine Encephalitis virus (EEE): The focus of this disease is usually the East coast with cases occurring as far west as Ohio. The mosquito vector for **EEE** includes members of the *Aedes* sp. and *Coquillettidia perturbans*.

Western Equine Encephalitis virus, as the name suggests, principally occurs in the Midwest and Western United States. While large equine outbreaks have occurred, no equine cases were reported from 1999-2006. The most important vector of this disease is *Culex tarsalis*.

Venezuelan Equine Encephalitis virus has a geographical distribution restricted predominantly to Central and South America, although U.S. incursions have occurred, and the risk of introduction persists. Several efficient vectors have been identified and these include common genera, *Aedes*, *Anopheles* and *Culex* spp. **VEE is a reportable disease; consult State Veterinarian when disease is suspected.**

Clinical Signs—highly variable; none pathognomonic

Moderate to high fever 102.5-104.5°F (39.17-40.28°C)

Depression/somnolence

Inappetance

Diarrhea (VEE only)

Dysphagia

Head pressing

Circling

Blindness

Dementia

Seizures

Rapid personality change: 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 abnormalities in horses that survive. 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

EEE: 3 – 7 days

VEE: 2 – 4 days

Transmission

Indirect, bites from mosquitoes having become infected with virus while feeding on viremic animal or avian host. Multiple animal species may seroconvert or develop clinical signs, however only those that develop significant viremia are reservoir hosts.

EEE/WEE/WNV: Only birds appear to develop a significant level of virus in their blood and can thus transmit the respective disease. An infected horse is a dead-end host; viral exposure can occur through necropsy procedures and handling of tissues. Appropriate precautions should be taken.

VEE: Horses do develop a high enough viral titer to transmit to mosquitoes, therefore, horses have a reservoir role in VEE infections. During epizootics, horses are important amplifiers of the virus.

Diagnostic Testing

MAC- ELISA test—single serum sample (red top tube)

Results of >1:400 are confirmatory in horse(s) exhibiting clinical signs consistent with EEE/WEE.

Paired serum neutralizing antibody titers: 2 samples (red top tube) collected 2-4 weeks apart

Four-fold increase in titers between samples is considered confirmatory in horse(s) exhibiting clinical signs consistent with EEE and not having been recently vaccinated.

CSF analysis:

CSF WBC count—usually > 7 cells/ul

CSF total protein—usually >70 mg/dl

PCR may be attempted on CSF of clinically affected horses. Test sensitivity is limited.

Note: CSF fluid requires prompt evaluation. When this is not feasible, direct smear slide mounts may be made and express shipped on cold packs to the laboratory.

Post-mortem:

A rabies protocol should be followed for ALL horses demonstrating encephalitis that undergo a post-mortem. Most causes of viral encephalitis in the horse are also zoonotic for humans.

Post-mortem sample collection requires appropriate precautions to avoid exposure.

[Click here for 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. [Click here for brain removal document.](#)

Shedding Time of Organism Past Resolution of Clinical Signs

VEE horses develop significant viremia between 2 and 5 days after infection. Horses do not shed this virus, but mosquitoes can obtain virus by ingesting a blood meal from these horses.

Environmental Persistence

This enveloped virus is susceptible to drying, ultraviolet light, and detergent.

Specific Control and Treatment Measures

Vaccination

After initial vaccination series (per manufacturer's instructions), administer annual booster prior to mosquito season. In endemic areas, booster at 4 month intervals during mosquito season.

Vector control

Release of Animals from Isolation

No restrictions need be placed on recovered animals.

Biosecurity Issues for Receiving Animals

None.

Zoonotic Potential

Human risk of exposure via bite of infected mosquito or through handling CNS tissue and/or fluids of infected animal. Precautions are indicated when performing necropsy examinations of neurologic horses.

Link to *A Review of Equine Zoonotic Diseases: Risks in Veterinary Medicine (J.S. Weese)*:
<http://www.aaep.org/proceedings/02proceedings/910102000362.pdf>