

# Eastern (EEE), Western (WEE), and Venezuelan (VEE) Equine Encephalitides

**Category:** Equine Neurologic Viral Diseases

**Agents:** EEEV, WEEV, VEEV (Family:  
*Togaviridae*, Genus: *Alphavirus*)

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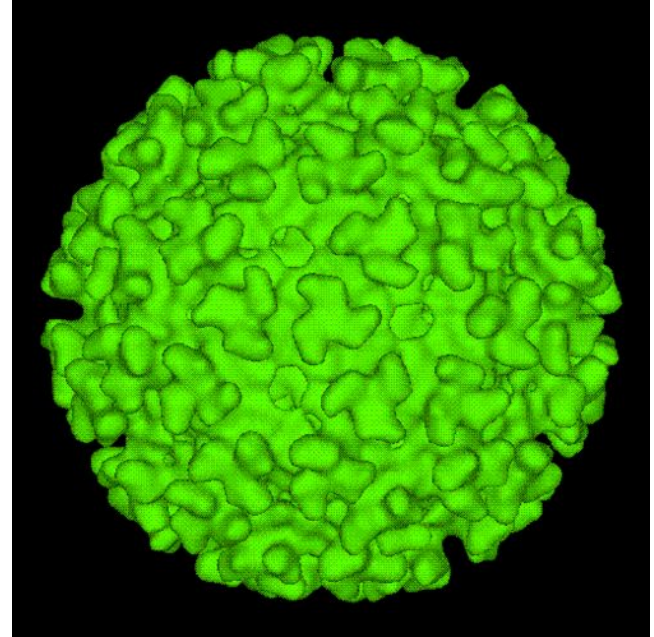


# I. Introduction

- Arboviral encephalomyelitis affecting horses and humans.
  - Occur in *North* and *South America*.
- Transmitted from **sylvatic reservoirs** to horses and humans via mosquitoes.
- South American EEEV variant now designated as **Maradiaga Virus (MDV)**.

## II. ETIOLOGY

- **EEEV, WEEV, VEEV:** Single-stranded, enveloped, positive-sense RNA viruses.
  - **Family:** *Togaviridae*
  - **Genus:** *Alphavirus*
- **Reservoirs:**
  - Birds → EEEV
  - Rodents → Endemic VEEV
- In epizootic VEEV, mutations occur causing equids to become reservoirs.



A computer-generated model of the surface of an *Alphavirus* derived by cryoelectron microscopy.

### III. Pathophysiology

- Causes **destructive encephalomyelitis** from **neuronal viral replication**.
- Leads to severe inflammation with polymorphonuclear infiltration.
  - **EEEV**: Most neurovirulent, causing rapid neuronal damage.
  - **WEEV**: Milder disease; Highlands J variant persists in Florida.
  - **VEEV**: Unique—**horses act as amplifying hosts** during outbreaks.

## IV. Epidemiology

- **Endemic to North & South America.**
  - Outbreaks fluctuate with weather and mosquito populations.
- Expanding northward (USA & Canada).
  - **EEEV:** Eastern USA.
  - **WEEV:** Western USA.
  - **VEEV:** Southern Texas, Central & South America.

## V. Signalment

- No breed or sex predisposition.
- **Young horses** more at risk for EEE.
- **Risk Factors:**
  - Poor vaccination coverage.
  - High mosquito density.
  - Undervaccinated horses entering endemic regions.



A foal (young horse) with its mare (adult female horse). **Young horses** are more susceptible to Eastern Equine Encephalomyelitis (EEE).

## VI. Clinical Signs (Early/Prodromal Stage)

- During the **early (prodromal) stage**, occurring 48–96 hours before neurologic signs, horses may show:
  - **Fever;**
  - **Depression;**
  - **and** **inappetence**
- Some may also exhibit *mild lameness* or *abdominal discomfort* prior to the onset of neurologic disease.

## VI. Clinical Signs (Neurologic Stage)

- Shows signs of **diffuse brain involvement**, such as:
  - **Somnolence** (*drowsiness*)
  - **Dementia** (*mental dullness*)
  - **Head pressing, ataxia** (*incoordination*), **blindness, circling, and seizures**
- May involve the **spinal cord or brainstem**, causing **weakness** or **paralysis**.
- **Severe cases** progress to **colic-like signs, reduced alertness, recumbency, and death**.
- Other possible signs: **abortion, mouth sores, lung bleeding, and nosebleed**.



## VII. Diagnosis

### Serology

- **IgM capture ELISA** titer  $\geq 400 \rightarrow$  **confirmatory**.
- **Fourfold rise** in titer between acute and convalescent samples  $\rightarrow$  **positive**.
- **EEEV:WEEV titer ratio**  $\geq 4$  = **suspicious**;  $\geq 8$  = **strongly indicative** of EEEV infection.
- EEE-affected horses rarely survive long enough for paired sampling.

### Cerebrospinal Fluid (CSF)

- **Neutrophilic pleocytosis** (*increased neutrophils*).
- **Elevated total protein** concentration.

## VIII. Differential Diagnosis

- **West Nile Virus (WNV) encephalomyelitis** — tends to cause milder cerebral and more spinal cord signs.
- **Rabies** — consider with acute neurologic signs and behavioral changes.
- **Leukoencephalomalacia** — due to moldy corn ingestion.
- **Hepatic encephalopathy** — associated with liver dysfunction.

## VIII. Pathologic Findings

- **Gross:**

- Congested meninges.
- Petechial hemorrhages of brain/spinal cord.
- Cerebral edema with possible herniation of brain tissue.

- **Histologic:**

- Meningoencephalomyelitis with neuronal degeneration and gliosis.
- Perivascular and neuroparenchymal infiltrates.
- Possible cardiac lesions.

## IX. Treatment

- **Supportive care only:** *No specific antiviral therapy is available.*
- **Fluid and metabolic support:** Maintain hydration and correct metabolic imbalances.
- **Prevent self-induced trauma:** Take precautions to avoid injuries caused by neurologic deficits.
- **Corticosteroids:** May provide benefit in mild cases (*anecdotal evidence*).

## X. Prognosis

- **EEEV:** 85–100% mortality.
- **WEEV:** 20–40% mortality.
- **VEEV:** 40–80% mortality.
  - Survivors may retain neurologic deficits (*ataxia, depression, behavioral changes*).
- Reinfection possible; *vaccination* recommended.

# XI. Prevention and Control

- **Vaccination:** Essential preventive measure.
- **Vector Control:** Reduce mosquito exposure.
- **Reporting:** Notify health authorities of cases.
- Use PPE during outbreaks, especially with VEEV.

# XI. Zoonotic Potential

- **EEEV & WEEV:** Horses are dead-end hosts; they do not transmit the virus to humans.
- **VEEV:** Horses can act as **active amplifiers**, posing a **risk of transmission to humans** during epizootics.

## Biosecurity Measures

- Avoid contact with blood, ocular, or nasal secretions from infected horses.
- Follow strict **rabies necropsy protocols** when handling carcasses.
- Use appropriate **personal protective equipment (PPE)** during treatment, handling, or necropsy.

## XII. Key Takeaways

- Equine arboviral infections primarily affect the **central nervous system** and have **high fatality rates**.
- EEEV is the most **virulent**, while VEEV poses a significant **zoonotic risk**.
- **Prevention** relies on **vaccination** and **mosquito/vector control**.
- **Early detection, prompt supportive care, and timely reporting** are critical to saving lives and controlling outbreaks.



# References

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