

Venezuelan Equine Encephalitis Fact Sheet

1. Disease overview

Venezuelan equine encephalitis is a **vector-borne viral** disease caused by **Venezuelan equine encephalitis virus** (VEEV), an alphavirus of the *Togaviridae* family. The virus is maintained primarily in rodent–mosquito cycles; during outbreaks, equids can develop high viraemia and serve as amplifying hosts, while humans are incidental hosts who may also become viraemic but are not thought to drive transmission. In horses, disease ranges from fever and anorexia to neurological signs such as blindness, ataxia, convulsions, and death.

Venezuelan equine encephalitis is a **WOAH-notifiable disease**.

Venezuelan equine encephalitis is listed in the **European Animal Health Law** under categories **D**, **E**.

2. Agent

VEEV is an **enveloped, single-stranded, positive-sense RNA virus** within the genus *Alphavirus*, family *Togaviridae*. The virion is spherical (~70 nm), with an icosahedral capsid and lipid envelope carrying glycoproteins E1 and E2. The former is required for membrane fusion, while E2 mediates receptor binding and is the main target for neutralizing antibodies.

VEEV is classified into **six antigenic subtypes** (I–VI), with subtype I further divided into five **variants** (I-AB, I-C, I-D, I-E, I-F). Subtypes are further grouped into **enzootic strains** (subtype I variants I-D, I-E, I-F, and subtypes II–VI), which are maintained in rodent–mosquito cycles and typically cause mild or inapparent infection in equids, and **epizootic strains** (subtypes I-AB and I-C), which arise from enzootic progenitors and cause severe outbreaks in horses. Epizootic strains amplify in equids, while enzootic variants are generally non-pathogenic in equids but can cause human disease.

3. Geographical Distribution

Venezuelan equine encephalitis is historically endemic in parts of Central and northern South America, including Mexico, Colombia, Venezuela, and surrounding countries. Sporadic equine and human outbreaks have also been reported in Argentina and other regions of South America.

According to WAHIS data, the agent was not reported in the EU in the last 2 years.

For more detailed information and dynamic maps, visit the *Geographical Distribution* section of the online **disease profile** (accessible via the button in the top right corner).

4. Animal hosts

4.1. Primary animal species affected

Equids, particularly horses and donkeys, are the most clinically affected species and play a central role in **epizootic outbreaks**, as infected animals develop sufficient viremia to infect mosquitoes serving as **amplifying hosts**.

Other **domestic animals**, including cattle, swine, dogs, and poultry, may seroconvert without showing clinical signs, while rabbits, goats, and sheep can develop fatal disease.

Sylvatic rodents, particularly small wild species, act as **reservoir hosts** in **enzootic cycles**, maintaining the virus independently of equids. Various wildlife species – including bats, opossums, foxes, and wild birds – can also become infected in enzootic cycles.

4.2. Clinical Signs

In **equids**, the severity of the disease is strain-dependent; **enzootic strains** typically cause mild or subclinical infections, while **epizootic strains** are linked to severe neurological illness. Clinical presentations can be broadly categorized into four forms. The **subclinical** form, most often associated with enzootic strains, shows no obvious disease manifestation. A **moderate** presentation involves inappetence, fever, and depression, with viremia lasting 2 to 5 days, coinciding with the onset of fever and terminating with the production of neutralizing antibodies. The **severe-non-fatal** form is marked by persistent high fever, tachycardia, and a progression to more pronounced central nervous system signs such as paresis, muscle fasciculations, incoordination, and head-pressing, which can result in permanent neurological damage. Finally, a **fatal** presentation exhibits signs similar to severe disease but concludes in death, which can be sudden or occur hours after the onset of neurological signs.

Other **domestic animals**, such as cattle, swine, dogs, and poultry, are generally asymptomatic, although rabbits, goats, and sheep can develop fatal disease under experimental or epizootic conditions.

4.2.1. Incubation Period

In **equines**, the incubation period for VEEV, defined as the interval between mosquito transmission and the onset of clinical signs, typically ranges from 12 hours to 5 days. High fever appears within 12–24 hours and neurologic signs can manifest up to five days after infection and may persist for up to two weeks, with the duration and severity varying based on the specific virus strain and the equine species affected.

4.2.2. Morbidity and mortality

In **equines**, morbidity and mortality vary with the viral subtype. In epizootic outbreaks, morbidity can be very high (50–100%) in unvaccinated equine populations, with mortality reaching 50–70%. Enzootic strains generally cause much lower morbidity and mortality.

4.2.3. Zoonotic Potential

Venezuelan equine encephalitis is a **zoonosis**.

5. Transmission

VEEV is transmitted to vertebrate hosts through the bite of **mosquitoes**. In sylvatic and peri-urban environments, **enzootic strains** are maintained in **rodent–mosquito cycles** involving *Culex* mosquitoes (*Melanoconion* subgenus) and sylvatic rodents. **Epizootic strains** emerge through viral

mutations or recombination and are transmitted by a wider range of mosquito genera, including *Aedes*, *Deinocerites*, *Mansonia* and *Psorophora*, enabling explosive outbreaks. Equids infected with epizootic strains develop high-titre viremia sufficient to infect mosquitoes, which amplifies the virus and sustains equine epidemics.

→ For more information on vector distribution, visit the *Vector* section in the online disease profile.

6. Diagnostic tests

WOAH-recommended tests for the **detection of the agent**: Reverse transcription PCR (RT-PCR), virus isolation in cell culture or suckling mice and immunohistochemistry in brain tissue.

VEEV is often difficult to isolate from the brains of clinically affected equids. For successful virus isolation, blood samples should be obtained from febrile animals, particularly those in close proximity to confirmed encephalitic cases.

WOAH-recommended tests for the **detection of immune response**: IgM capture ELISA, indirect IgG ELISAs, Plaque reduction neutralisation (PRN), Haemagglutination inhibition (HI) and Complement fixation (CF).

Although enzootic VEEV subtypes and variants are non-pathogenic in equids, they induce antibody responses that may cross-react with epizootic VEEV strains in diagnostic assays. In addition, serological cross-reactions can occur between antibodies to Eastern and Western equine encephalitis viruses when using tests such as CF and HI.

7. Prevention and control

7.1. Vaccination

Both inactivated and live-attenuated vaccines are available for use in equids in endemic regions.

→ In the EU, there are no vaccines approved against VEEV.

7.2. Treatment

There is currently **no specific antiviral treatment** for VEEV infection. Supportive care in horses includes anti-inflammatory therapy, fluid support, and intensive nursing.

Vector control (mosquito habitat reduction, repellents, insecticides) and vaccination remain the most effective tools to prevent and control outbreaks. Reducing outdoor exposure during peak mosquito activity is also recommended in endemic areas.