

# Western Equine Encephalitis Fact Sheet

## 1. Disease overview

**Western equine encephalitis** is a **vector-borne viral** disease caused by **Western equine encephalitis virus** (WEEV), an alphavirus of the *Togaviridae* family. WEEV primarily circulates in an enzootic cycle involving wild birds and mosquitoes. Spillover infections can occur in equines and other mammals. In equines, WEEV can cause neurological disease of variable severity.

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

Western equine encephalitis is listed in the **European Animal Health Law** under category **E**.

## 2. Agent

WEEV 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. Its genome (~11.5 kb) encodes non-structural proteins involved in replication (nsP1–nsP4) and structural polyprotein that includes the capsid (C) and envelope glycoproteins E1 and E2.

WEEV is a natural recombinant of Eastern Equine Encephalitis virus (EEEV) and Sindbis virus. Several lineages have been identified, though little antigenic diversity is observed.

## 3. Geographical Distribution

Western equine encephalitis is historically endemic in western North America, particularly in regions of the United States and Canada. Sporadic equine outbreaks have also been documented in Argentina and Brazil.

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

WEEV is maintained in a **mosquito–bird–mosquito** cycle, particularly in wet areas. Passerine birds act as amplifying hosts, with high-titre viremia that infects mosquitoes. Equines are highly susceptible to WEEV but are considered dead-end hosts due to insufficient viremia for onward transmission.

Infections in other animals, including pigs, cattle, sheep and dogs, are typically asymptomatic. Poultry may develop viremia without clinical signs.

## 4.2. Clinical Signs

In **equines**, clinical presentation ranges from subclinical or mild disease to severe neurological symptoms. Signs include fever, anorexia, depression, ataxia, circling, head pressing, and incoordination. In severe cases, seizures, recumbency, and coma may occur.

In **birds**, clinical signs are rare. Chickens and turkeys may develop transient viremia following experimental infection, especially young birds.

Experimental WEEV infection in **rabbits** induces age- and route-dependent outcomes. Neonatal rabbits are susceptible to neurologic disease and mortality, especially following intracerebral inoculation, while adult rabbits typically remain asymptomatic. WEEV shows strong neurotropism, and infected rabbits mount an effective humoral immune response. Although not relevant in field transmission, rabbits serve as useful models for studying alphavirus neuropathogenesis and immunity.

### 4.2.1. Incubation Period

In equines, the incubation period for WEEV, defined as the interval between mosquito transmission and the onset of clinical signs, is typically **5–7 days**, but may vary depending on viral dose and host factors.

In experimentally infected poultry and pigs, viremia is usually detectable within 1–3 days, with no clinical disease observed.

### 4.2.2. Morbidity and mortality

In **equines**, morbidity during outbreaks varies, with clinical signs seen in approximately 10–40% of infected individuals. Neurological disease occurs in a subset of clinically affected horses. The mortality rate is approximately 20–50%, lower than for Eastern equine encephalitis virus but still significant.

In **poultry** and **pigs**, mortality is rare or absent under both field and experimental conditions.

### 4.2.3. Zoonotic Potential

Western equine encephalitis is a **zoonosis**, however animals do not transmit the virus directly to humans.

## 5. Transmission

WEEV is transmitted to vertebrate hosts through the bite of **mosquitoes**. The principal vector in North America is *Culex tarsalis*, which feeds on birds and mammals.

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

Birds develop sufficient viremia to infect mosquitoes and serve as reservoir hosts. Transmission occurs during mosquito-active months, typically spring through autumn. Equines and other mammals are incidental dead-end hosts and do not contribute to viral maintenance.

## 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.

Virus isolation is typically performed from central nervous system tissues in acutely affected or deceased equines.

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).

The CF test is frequently used for the demonstration of antibodies, although the antibodies detected by the CF test may not persist for as long as those detected by the HI or PRN tests. The latter is very specific and can be used to differentiate between Eastern, Western and Venezuelan virus infections.

## 7. Prevention and control

### 7.1. Vaccination

Inactivated vaccines against WEEV are available for equines. Annual vaccination is recommended in endemic regions, ideally prior to peak mosquito season.

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

### 7.2. Treatment

There is currently **no specific antiviral treatment** for WEEV infection. Supportive care in horses includes anti-inflammatory therapy, fluid support, and intensive nursing. Outcome is often poor once neurological signs are present.

Vector control (mosquito habitat reduction, repellents, insecticides) is critical for disease prevention. Reducing outdoor exposure during peak mosquito activity is recommended in endemic areas.