Eastern Equine Encephalitis Fact Sheet

1. Disease overview

Eastern equine encephalitis is a **vector-borne viral** disease caused by **Eastern equine encephalitis virus** (EEEV), a highly pathogenic alphavirus of the *Togaviridae* family. EEEV primarily circulates in an enzootic cycle involving wild birds and mosquitoes. Spillover infections can occur in equines and other mammals. Equine infection is associated with severe, often fatal, neurological disease.

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

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

2. Agent

EEEV 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 (~11.7 kb) encodes non-structural proteins involved in replication (nsP1–nsP4) and structural proteins including capsid (C), envelope glycoproteins E1 and E2, and axillary proteins.

EEEV comprises **multiple genetic lineages** within the **EEEV complex**, with distinct geographic and pathogenic profiles. The **North American lineage** (Group I) is the most virulent, associated with severe neurologic disease and high mortality in equines and humans. In contrast, **South and Central American lineages** (Groups II–IV) are genetically distinct, enzootic in bird–mosquito cycles, and not known to cause clinical disease in mammals despite widespread circulation. The heightened virulence of Group I strains is linked to greater neuroinvasiveness and replication efficiency.

3. Geographical Distribution

Eastern equine encephalitis occurs predominantly in eastern North America, parts of the Caribbean, and sporadically in Central and 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

EEEV 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 EEEV but are considered dead-end hosts due to insufficient viremia for onward transmission.
Infection has also been reported in chickens, pigs, and deer, although clinical disease is rare outside of equines and select avian species.

4.2. Clinical Signs

In **equines**, EEEV infection ranges from nonspecific signs (fever, lethargy, anorexia) to severe neurological manifestations including ataxia, circling, head pressing, blindness, seizures, and recumbency. The disease has a rapid progression, and affected animals often deteriorate within 48–72 hours.

In **birds**, clinical signs are rare, but some species (e.g., pheasants, emus) may develop hemorrhagic or neurologic disease. In experimentally infected chickens, particularly young or immunologically naïve birds, the virus can cause detectable viremia, but clinical signs are rare.

Experimental infections with **pigs** have shown that they can develop low-level, short-lived viremia but typically do not show clinical signs.

4.2.1. Incubation Period

In equines, the incubation period for EEEV, defined as the interval between mosquito transmission and the onset of clinical signs, ranges from **3 to 10 days**.

The incubation period in experimentally infected pigs and poultry ranges from 1 to 5 days, depending on age, viral dose, and route of inoculation.

4.2.2. Morbidity and mortality

In **equines**, morbidity can be high during outbreaks, especially in unvaccinated populations. Mortality rates exceed 75–90%, making EEE one of the most lethal equine arboviral encephalitides. Survivors often exhibit long-term neurological deficits.

In **birds**, susceptibility varies by species. Some infected species may experience high mortality (up to 100%), while most remain asymptomatic.

4.2.3. Zoonotic Potential

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

5. Transmission

EEEV is transmitted to vertebrate hosts through the bite of **mosquitoes**. The primary enzootic vector is *Culiseta melanura*, which feeds mainly on birds. Bridge vectors, such as *Aedes*, *Coquillettidia*, and *Culex* spp., are responsible for transmitting the virus to equines and other mammals. Equines are incidental hosts and do not contribute to viral maintenance.

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

Transmission occurs seasonally, typically from late spring through early autumn, corresponding with mosquito activity.

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 EEEV are available for equines. Annual vaccination is recommended in endemic regions, ideally prior to peak mosquito season. Primary vaccination followed by timely boosters is essential to maintain protection.

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

7.2. Treatment

There is currently **no specific antiviral treatment** for EEEV 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.