

EFSA - Disease profiles

Japanese Encephalitis Fact Sheet

1. Disease overview

Japanese encephalitis is a **vector-borne viral disease** caused by **Japanese encephalitis virus** (JEV), a flavivirus related to West Nile, Usutu, and St. Louis encephalitis viruses. The disease is transmitted by mosquitoes and primarily affects pigs and birds, which serve as amplifying and reservoir hosts, respectively. Occasionally, JEV can cause neurological disease in equids and humans, which are however considered dead-end hosts. Infection in animals is often subclinical, although reproductive losses in pigs are an important outcome.

Japanese encephalitis is a **WOAH-notifiable disease**.

Japanese encephalitis is listed in the European Animal Health Law under category E.

2. Agent

JEV is an **enveloped**, **single-stranded**, **positive-sense RNA** virus belonging to the genus *Orthoflavivirus*, family *Flaviviridae*. The virion is spherical (~50 nm), with a lipid envelope containing glycoproteins that mediate cell entry. The genome is ~11 kb in length and encodes a single polyprotein cleaved into three structural proteins (C, prM/M, and E) and seven non-structural proteins (NS1, NS2A, NS2B, NS3, NS4A, NS4B and NS5). **NS1** elicits strong antibody responses and is used in diagnostic assays.

JEV is classified into **five genotypes (GI–GV)** based on sequence variation in the E gene, with GI and GIII currently predominant in Asia. Despite genetic diversity, all genotypes belong to a **single serotype**, and immunity induced by one provides cross-protection against others.

3. Geographical Distribution

Japanese encephalitis is endemic throughout south and southeastern Asia, parts of the western Pacific and Australia.

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

Pigs are the most important **amplifying hosts**, developing high-titre viremia sufficient to infect mosquitoes. **Birds**, particularly ardeid wading birds, also serve as major **reservoirs**, sustaining long-

term viral circulation in natural habitats. **Equids** are susceptible to infection and may develop neurological disease but are considered incidental dead-end hosts. Other **domestic animals**, such as cattle, sheep, goats, chicken, ducks, dogs and cats, may seroconvert but usually do not develop clinical signs or contribute to transmission.

4.2. Clinical Signs

In **pigs**, Japanese encephalitis is often subclinical in young animals but can be a significant reproductive disease in adults. In sows, losses can be substantial, with abortions, stillbirths, or mummified foetuses, usually at term. Boars may show reduced sperm count and motility. Live-born piglets may present with neurological signs such as tremors and convulsions and often die shortly after birth. In non-pregnant females, infection typically results in mild febrile illness or subclinical infection. Natural infection in swine induces long-lasting immunity.

In **birds**, particularly ardeid species, infection is typically asymptomatic, though experimental infections have demonstrated viremia and occasional mild signs.

In **horses**, infection is usually subclinical, but when disease occurs, it often presents in localized clusters. Three syndromic forms are recognized:

- **Transitory type**: moderate fever lasting 2–4 days, accompanied by inappetence, impaired locomotion, and congested or jaundiced mucosa, with rapid recovery within a few days.
- **Lethargic type**: febrile episodes with marked stupor, teeth grinding and chewing motions, difficulty swallowing, petechiated mucosa, incoordination, neck rigidity, impaired vision, paresis, and paralysis. Recovery generally occurs within about a week.
- **Hyperexcitable type**: high fever with profuse sweating, muscle tremors, aimless wandering, behavioural changes such as aggression, and signs of blindness. Neurological sequelae may follow in recovering animals.

4.2.1. Incubation Period

In **horses** the incubation period, defined as the interval between mosquito transmission and the onset of clinical signs, has been experimentally determined to be 8–10 days. For experimentally infected **swine**, signs of infection, fever, and viremia were observed 24 hours post inoculation with other clinical manifestations apparent within 6 days post inoculation.

4.2.2. Morbidity and mortality

In **pigs**, morbidity is usually expressed as reproductive failure rather than clinical disease, and mortality is negligible in adults. However, mortality may reach nearly 100% in non-immune infected piglets. In **equids**, morbidity rates reported from field cases vary from less than 1% to 1.4%. Case fatality rate is typically around 5-15%, but it can reach 30-40% during outbreaks, with survivors occasionally showing residual neurologic deficits. In **birds** and **other domestic animals**, infections are usually subclinical.

4.2.3. Zoonotic Potential

Japanese encephalitis is an important **zoonosis** and a leading cause of viral encephalitis in humans in endemic regions. However, animals do not directly transmit the virus to humans.

5. Transmission

JEV is transmitted by **mosquitoes**, typically of the *Culex* genus. *Culex tritaeniorhynchus* is known as an important vector as it breeds in rice paddies and wetlands and feeds on a wide range of hosts, including birds, pigs, and equids. Several additional species of *Culex*, *Aedes* and *Mansonia* mosquitoes

are competent vectors under experimental conditions, and vertical transmission in mosquitoes has been documented.

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

The virus is maintained in a **bird-mosquito enzootic cycle**, with **ardeid birds** (herons and egrets) acting as natural reservoirs and **pigs** serving as important amplifying hosts that bridge transmission to equids and, occasionally, to humans. **Horses** and humans are dead-end hosts because they do not develop sufficient viremia to infect mosquitoes. Experimental studies have shown that young poultry can develop sufficient viremia to infect mosquitoes, but their role in natural transmission is uncertain. Although experimental evidence suggests that pigs may shed virus or transmit it via semen, these routes have not been confirmed in the field.

The mechanisms of **overwintering** remain unclear but may involve survival of infected mosquitoes, transovarial transmission, or persistence in alternative hosts such as reptiles, amphibians, or bats.

6. Diagnostic tests

WOAH-recommended tests for the **detection of the agent**: Reverse transcription PCR (RT-PCR), Real-time RT-PCR, virus isolation in cell culture or suckling mice and immunohistochemistry in brain tissue. In horses, sampling focused on brain regions such as the corpus striatum, cortex, and thalamus, and sometimes blood or spinal cord, is advised due to typically low isolation rates.

WOAH-recommended tests for the **detection of immune response**: IgM capture ELISA, indirect IgG ELISA, Plaque reduction neutralisation (PRN) and Virus neutralisation (VNT). IgM ELISA is particularly valuable for identifying recent infections, as antibodies are detectable within days after onset. Indirect IgG ELISA is useful for screening populations, though it may cross-react with other flaviviruses. PRNT remains the most specific assay, especially for confirming cases in areas where multiple flaviviruses co-circulate, while VNT offers a reliable alternative in well-equipped laboratories.

7. Prevention and control

7.1. Vaccination

Vaccines are available for both horses and swine in endemic countries. In **horses**, inactivated vaccines are used to prevent encephalitis and its neurological consequences. In **pigs**, both inactivated and live-attenuated vaccines employed, primarily to protect breeding sows against reproductive losses.

 \rightarrow In the EU, there are no vaccines approved against JEV.

7.2. Treatment

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

Vector control (mosquito habitat reduction, repellents, insecticides), is recommended to prevent outbreaks, and when feasible, swine should be kept away from horses and humans to limit transmission.