Akabane virus infection Fact Sheet

1. Disease overview

Akabane virus (AKAV) causes Akabane virus infection, an arthropod-borne viral disease of domestic and wild ruminants, primarily cattle, sheep, and goats. The disease is characterized by reproductive losses resulting from transplacental infection, leading to abortions, stillbirths, and congenital malformations in newborns (Spickler, 2018; WOAH, 2025).

Akabane virus infection is not a WOAH-notifiable disease, and it is not listed in the European AHL.

2. Agent

AKAV is an enveloped, single-stranded, negative-sense RNA virus that belongs to the Orthobunyavirus genus of the Peribunyaviridae family. The virion is roughly spherical, 80–120 nm in diameter, and contains a tripartite genome composed of large (L), medium (M), and small (S) RNA segments. These segments encode the RNA-dependent RNA polymerase, glycoproteins Gn and Gc and, the nucleocapsid protein N and non-structural protein NSm and NSs. The glycoproteins Gn and Gc are responsible for host cell attachment and elicit neutralizing antibodies (Elliott, 1990; Elliott, 2014; Kobayashi et al., 2007). The virus is moderately stable under ambient conditions but is inactivated by lipid solvents, detergents, and standard disinfectants (CFSPH, 2015).

3. Geographical Distribution

AKAV is common in most tropical and subtropical areas between ~35°N and 35°S. It is endemic in much of Africa, Asia, the Middle East, and Oceania, including Japan and Australia (Kirkland et al, 2024).

Episodic incursions have occurred in southern Europe (Turkey), often associated with the movement of infected midges (Dağalp et al, 2021).

Akabane virus infection is not reportable to WOAH. Evidence from published studies describing natural infections with this agent, as well as field epidemiological studies, are collected in the EFSA's systematic literature review (updated until 31/12/2025) and summarized in Figure 1. For more detailed information, dynamic maps, and references visit the online disease profile (accessible via the button in the top right corner).

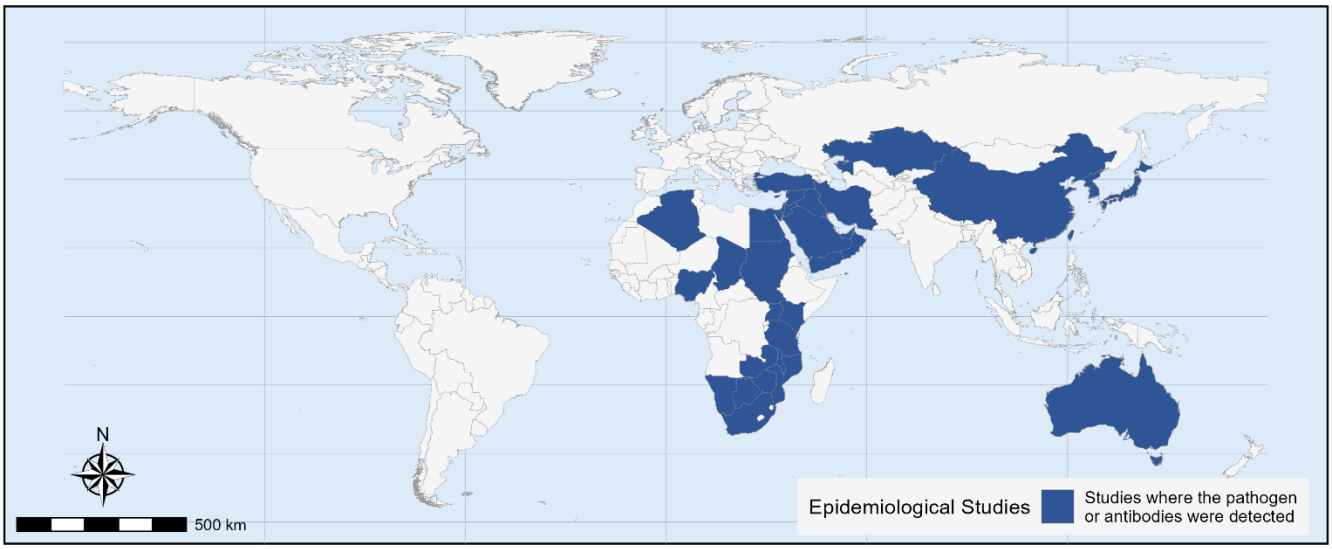


Figure 1. Geographical distribution of epidemiological studies addressing the occurrence of AKAV, as identified by the EFSA's systematic literature review (covering years 1970-2025).

4. Animal hosts

4.1. Susceptible hosts

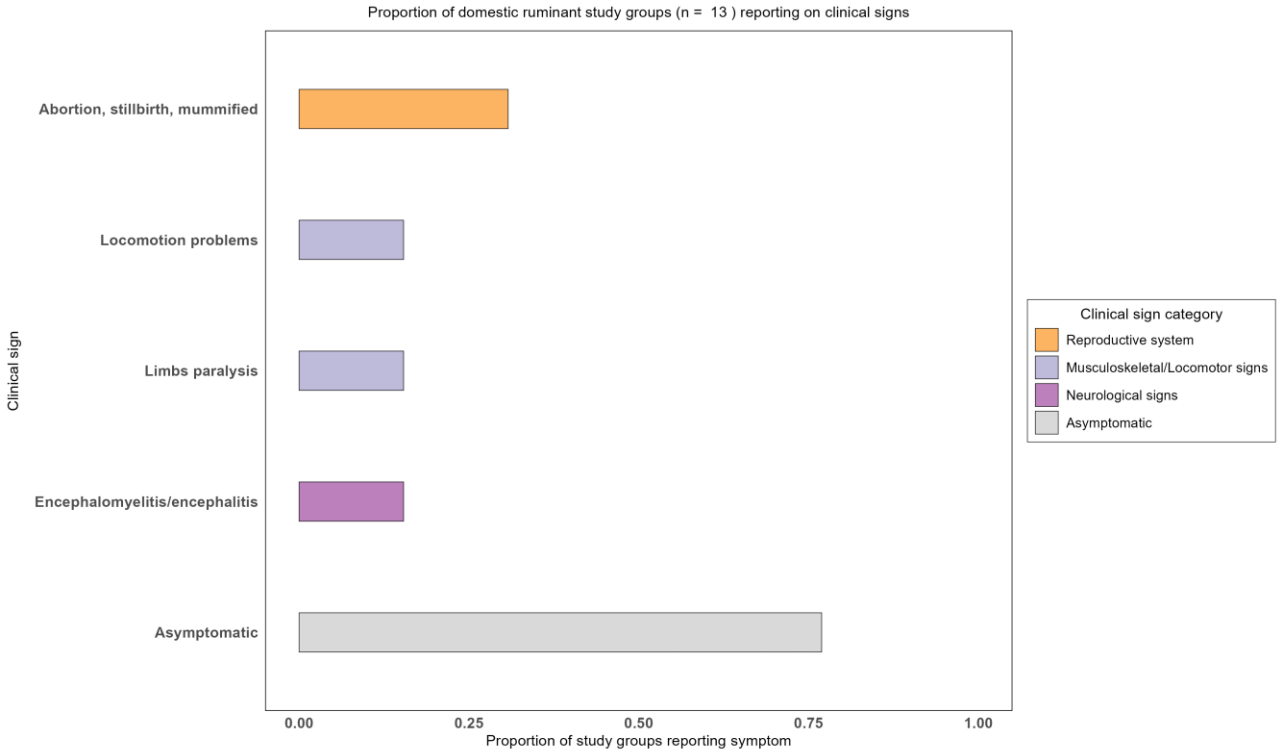
Based on epidemiological knowledge of host–pathogen–vector interactions and outbreak reports, the main hosts of AKAV are domestic ruminants. However, other susceptible species have been identified in the SLR. The SLR summary is given in Table 1.

Table 1. Susceptible host species of Akabane virus.

The systematic literature review reported in the AKAV disease profile, identified the following susceptible species (updated until 31/12/2025, for references see online disease profile)
FIELD
Epidemiological studies carried out in the field **Pathogen was detected in the following animal species:** • Bovidae: *Bos taurus, Capra hircus, Ovis aries* • Suidae: *Sus scrofa domesticus* **Antibodies were detected in the following animal species:** • Bovidae: *Bos taurus, Capra hircus, Ovis aries, Syncerus caffer, Tragelaphus scriptus, Taurotragus oryx, Aepyceros melampus, Kobus leche, Tragelaphus angasii, Hippotragus equinus, Hippotragus niger, Saiga tatarica, Damaliscus lunatus jimela, Damaliscus lunatus lunatus, Kobus ellipsiprymnus* • Camelidae: *Camelus dromedarius, Camelus bactrianus* • Elephantidae: No species specified • Equidae: *Equus caballus* • Suidae: *Sus scrofa domesticus, Potamochoerus larvatus, Phacochoerus africanus* **Outbreaks reported to WOAH included the following species:** • No species specified
EXPERIMENTS
Experimental studies demonstrated infection in: • Bovidae: *Bos taurus, Capra hircus, Ovis aries* • Phasianidae: *Gallus gallus domesticus*

4.2. Clinical Signs

Outcomes of a systematic literature review on clinical signs in 13 domestic ruminant study groups are displayed in Figure 2. Most study groups did not show any clinical signs or had reproductive clinical signs.



Study group count per domestic ruminant species: Cattle n = 6; Goat n = 4; Sheep n = 3. The SLR was updated until 31/12/2025, for references see the online disease profile.

Figure 2. Clinical signs reported in the main hosts of AKAV.

Clinical manifestations occur almost exclusively in offspring infected in utero. The outcome depends on the stage of gestation at which infection occurs.

Cattle: Infection of the dam during the 3rd to 6th month of gestation may result in abortions, stillbirths, mummification, or congenital malformations such as arthrogryposis, torticollis, scoliosis, hydranencephaly, and cerebellar hypoplasia. Infection during late gestation can lead to calves born alive but may show flaccid paralysis, inability to stand, or ataxia.

Sheep and goats: Congenital defects are similar, with arthrogryposis and hydranencephaly as predominant lesions. Lambs may also present with domed skulls, mandibular deformities, or neurologic deficits.

In rare cases, adult cattle and sheep may exhibit transient fever, lethargy, or reduced milk yield, but overt systemic illness is uncommon.

4.2.1. Incubation Period

The incubation period in adult animals is not well-defined due to the lack of clinical signs, but viremia, which can lead to vertical transmission, usually occurs 1 to 6 days after infection (USDA, 2015; references in the online disease profile). Clinical disease in foetuses becomes apparent only at birth

or abortion, weeks to months after maternal infection, depending on gestational timing (Kurogi et al., 1977; Spickler, 2018).

4.2.2. Morbidity and case fatality

In adult ruminants most infections are asymptomatic, but clinical signs have been reported, as shown in Figure 3.4. Mild, transient fevers may occur but often go unnoticed. Fatality in adults is extremely rare; case fatality in experimentally infected cattle has been reported to be around 3% (Spickler, 2018).

The primary impact is on the foetus, where high rates of congenital abnormalities lead to significant mortality in newborns or require culling of severely affected animals. Reports from outbreaks indicate a high percentage of affected offspring among those born to non-immune dams infected during the susceptible period, with some reports citing morbidity rates in affected herds between 5 – 50% in cattle and 15 – 80% in sheep (USDA, 2015).

The case fatality in affected newborns is high. Most calves or lambs born with severe malformations are stillborn, die shortly after birth, or are humanely euthanized due to their inability to stand, nurse, or their poor quality of life. In such cases, the case fatality rate for affected neonates can be considered close to 100% (USDA, 2015; Spickler, 2018).

4.2.3. Zoonotic Potential

AKAV is not known to infect humans under natural conditions.

5. Transmission

AKAV is transmitted by biting midges of the genus Culicoides (Diptera; Ceratopogonidae). For more information on vector distribution, visit the Vector section in the online disease profile.

The virus is not spread by direct contact between animals or via fomite (USDA, 2105). Seasonal patterns of disease correspond closely to vector activity, with outbreaks typically occurring during or following warm, wet seasons. Windborne dispersal of infected Culicoides may facilitate long-distance spread (Spickler, 2018).

Transplacental transmission is the key mechanism leading to clinical disease in offspring. There is no evidence for venereal or vertical transmission in non-pregnant animals (Charles, 1994; Spickler, 2018).

6. Diagnostic tests

WOAH-recommended tests (WOAH,2025) for agent detection are virus isolation, fluorescent antibody test (FAT), immunohistochemistry (IHC), virus neutralisation (VNT) test and real-time RT-PCR.

Real-time RT-PCR is the preferred method for AKAV detection due to its high sensitivity and specificity, particularly useful given the short duration of viraemia in adult ruminants. Viral RNA can be detected in whole blood during acute infection or in foetal tissues such as brain, spinal cord, and placenta in cases of congenital malformations. Virus isolation remains a confirmatory technique but is less sensitive and more laborious, often successful only in samples collected early after infection or from vector pools. Immunohistochemistry on foetal tissues may support diagnosis when molecular methods are unavailable, although sensitivity is lower than PCR-based assays (WOAH, 2025).

For immune response detection, the recommended tests are ELISA and VNT.

ELISA assays, including commercial competitive ELISAs specific for AKAV, are suitable for large-scale screening and surveillance, providing a rapid means to assess herd exposure. The VNT remains the reference method for confirming serological results and determining functional neutralising antibody titres. Serological testing is particularly useful because infection in adult ruminants is often subclinical; therefore, antibody detection serves as the primary tool for confirming exposure and for monitoring vaccination or population immunity. Detection of antibodies in foetal fluids from malformed or stillborn neonates before colostrum intake provides evidence of in utero infection (WOAH, 2025).

To date, the systematic literature review has not found diagnostic tests evaluation studies meeting the eligibility criteria for inclusion.

7. Prevention and control

7.1. Vaccination

Live attenuated and inactivated vaccines against AKAV are available and widely used in endemic regions, particularly in East Asia and Australia, to prevent reproductive losses in cattle, sheep, and goats. Vaccination of breeding females prior to conception is recommended to ensure immunity during gestation, thereby preventing foetal infection and congenital malformations (Kirkland, 2015; Kurogi, 1978; WOAH, 2025).

Currently, there are no commercially available vaccines authorised in the EU.

7.2. Treatment

There is currently no specific antiviral treatment for Akabane virus infection. The infection in adult animals is usually asymptomatic or very mild, and they recover on their own. The severe impact of the virus is on the developing foetus, and once congenital defects have occurred, they are irreversible. The most effective strategy is to prevent susceptible pregnant animals from becoming infected during the critical stages of gestation (Spickler, 2018).

8. References

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