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SCIENTIFIC REPORT

Vector-borne diseases-knowledge maps

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The declarations of interest of all scientific experts active in EFSA's work are available at https://open.efsa.europa.eu/experts.

Abstract

This scientific report provides a structured overview of the main characteristics of 25 selected vector-borne diseases (VBDs) of potential relevance for the EU, including 12 diseases listed under the Animal Health Law (AHL) and 13 non-listed diseases. The objective is to compile and harmonise existing knowledge to support subsequent risk assessments, together with three complementary scientific reports on (1) vector competence and geographical distribution for the disease agents of arthropod species, (2) available surveillance and control measures for the VBDs and (3) potential pathways for introduction. Diseases were selected based on predefined eligibility criteria, including absence or uncertain status in at least half of EU Member States, the presence of competent vectors, susceptible domestic animal hosts and data availability. The report builds on EFSA's systematic literature review framework for Animal Disease Profiles, covering six knowledge areas: experimental infections studying infection dynamics and clinical manifestations in different hosts, pathogen survival and transmission modes, diagnostic test accuracy, vaccine efficacy, preventive and curative treatment efficacy and geographical distribution of the pathogens. Results are presented as standardised disease fact sheets, providing a harmonised and transparent evidence base to support EU-level risk assessments. While this report will be updated on an annual basis, continuously updated information, including interactive maps and full reference data sets, are available through EFSA's online Disease Profiles platform.

KEYWORDS

vector-borne diseases, diagnostic test performance, disease profiles, experimental infection studies, geographical distribution, risk assessment support, systematic literature review, vaccines and treatments

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SUMMARY

This scientific report addresses Term of Reference (ToR) 1.1 of the mandate by providing a structured overview of the main characteristics of 25 selected vector-borne diseases (VBDs) of potential relevance for the European Union (EU), including 12 diseases listed under the Animal Health Law (AHL) and 13 additional non-listed diseases.

Objective

The objective of this report is to compile and harmonise existing knowledge on the 25 diseases selected to provide a consistent evidence base for the subsequent risk assessments requested under ToRs 2, together with three additional scientific reports providing evidence on vector competence and geographical distribution, available surveillance and control measures and potential pathways for introduction.

The 25 VBDs were selected based on predefined eligibility criteria, including their current absence or uncertain status in at least half of the EU Member States, the presence of competent vectors, the involvement of susceptible domestic animal hosts and the availability of sufficient primary data. Diseases with clinical relevance in domestic animals were included. The report focuses on animal health, while acknowledging zoonotic aspects where relevant.

Methodology

The report builds on the systematic literature review (SLR) framework established for the EFSA Animal Disease Profiles. Six relevant knowledge areas were considered:

1. Experimental infections, investigating within-host infection dynamics and species-specific clinical manifestations.
2. Pathogen survival, including evidence on environmental persistence and transmission routes, where relevant.
3. Diagnostic tests performance, assessing the availability and performance characteristics of tests used for pathogen or antibody detection.
4. Vaccines, compiling evidence on the availability and effectiveness of vaccines in animal species.
5. Preventive and curative treatments, summarising evidence on pharmaceutical interventions used for prevention or treatment in animals.
6. Geographical distribution, documenting the occurrence of pathogens anywhere in the field worldwide.

For this mandate, the existing SLR databases were updated to include all selected diseases and literature published up to 31 December 2025. Evidence was extracted following standardised screening and data collection procedures, supported by semi-automated workflows. Where relevant, manual curation and expert judgement were applied to complement the SLR outputs, in particular for clinical signs, host range and vaccine availability.

Geographical distribution was compiled using data shared by the World Organisation for Animal Health (WOAH) for WOAH-listed diseases and SLR-derived evidence for non-listed diseases. This report provides a static synthesis of the evidence, while recognising the dynamic nature of VBD epidemiology.

Results

For each of the 25 selected VBDs, results are presented in short, standardised fact sheets. Each fact sheet includes the following sections: (1) disease overview and causative agent; (2) geographical distribution of the pathogen; (3) susceptible animal hosts; (4) transmission routes; (5) diagnostic tests; and (6) prevention and control measures.

These fact sheets summarise the key information relevant for EU level risk assessments and ensure consistency across diseases.

This report represents a snapshot of the current state of knowledge addressing ToR 1.1. In line with ToR 1.5, the information will be updated annually, with newly identified evidence highlighted in future updates.

More detailed and continuously updated information, including interactive maps and full reference data sets, is available through EFSA's online Disease Profiles platform. Readers are therefore encouraged to consult the online resources for the most up to date data beyond the publication cycle of this report.

1 | INTRODUCTION

1.1 | Background as provided by the requestor

In the last two decades, the EU has been significantly affected by various diseases of animals transmitted by arthropod vectors ('vector-borne diseases'), such as mosquitoes (e.g. West Nile fever), flies (e.g. lumpy skin disease), ticks (e.g. Crimean-Congo haemorrhagic fever) or *Culicoides* biting midges (e.g. bluetongue, epizootic haemorrhagic disease). The EU is also at risk of a wide range of serious vector-borne diseases such as Rift Valley fever or African horse sickness.

Recent data and epidemiological events show the increase of such vector-borne diseases (VBDs) either in the vicinity of the EU, in EU trading partners, or within the EU, concomitant with the progressive widening of the geographical extent of competent vectors such as biting midges and mosquitoes, some of them being able to transmit zoonotic pathogenic agents (e.g. *Aedes* and sandflies).

In April 2017, at the request of Directorate-General for Health and Food Safety (DG SANTE), EFSA published a scientific opinion on 36 VBDs, assessing their risk of introduction into the EU through movement of livestock or pets. This was considered a first screening, and it was already at that time recommended in the assessment that it should be updated.

In January 2020, also at the request of DG SANTE, and following reports of occurrence of the disease in North Africa, EFSA published a scientific opinion on epidemiological update and risk of introduction of Rift Valley fever (RVF) into Europe.

Since 2018, 12 VBDs have been listed under the Animal Health Law (AHL) and categorised by Commission Implementing Regulation (EU) 2018/1882[1] under various categories of listed diseases, depending on the level of intervention and the measures taken at EU level, and with reference to their vector species.

Those diseases largely differ one from another in terms of pathogenic agents, host species, vector species, as well as in terms of impact and zoonotic potential. However, it is relevant to consider them together as regards their specificity of being vector borne and what this entails in terms of risk assessment and risk management in view of the relative rapid evolution of the geographic distribution of vectors concerned.

It is relevant to ask support from EFSA and the relevant EU Reference Laboratories, to analyse the situation and get scientific advice assessing animal health risks linked with VBDs. The scientific advice should address in particular the likelihood of introduction of new VBDs in the EU and of spread of VBDs currently affecting the EU, the role of the climate evolution in this introduction or spread, and the potential evolution of the virulence or transmissibility of those VBDs. Considering the zoonotic nature of some of these VBDs, work in cooperation with the European Centre for Disease Prevention and Control (ECDC) appears relevant too.

This piece of scientific advice should explore and propose options to mitigate the risks of introduction and to address the suitable surveillance, prevention and control of VBDs in the EU, including vaccination.

1.2 | Terms of Reference as provided by the requestor

In the light of the above:

1 In accordance with Article 31 of Regulation (EC) No 178/2002, the Commission requests EFSA to provide scientific and technical assistance on the epidemiology of VBDs; the following aspects are of particular relevance for the scientific reports:

1.1 Provide a mapping/horizon scanning/compilation/description of the VBDs that are currently listed In the EU AHL (hereafter 'listed VBDs'), as well as other VBDs not listed but formerly assessed and deemed to have a potential impact and therefore deserving attention (hereafter 'non-listed VBDs'), including their geographic distribution in the EU, neighbouring regions or other regions presenting a particular risk due to epidemiological considerations;

1.2 Provide a mapping/horizon scanning/compilation/description in the EU and neighbouring countries of the currently known, as well as potential new, vectors competent for 'listed VBDs' and 'non-listed VBDs';

1.3 Provide a mapping/horizon scanning/compilation/description of the currently available surveillance, prevention and control measures for listed and non-listed VBDs in the EU; this includes the collection of data on the efficacy of these measures (e.g. vaccination efficacy, efficacy of biocidal treatments or repellents, animal treatments or insect nets or other husbandry practices);

1.4 Describe the potential pathways for listed and non-listed VBDs currently present in the EU to spread, and those not currently present in the EU to be introduced, including via intra EU movements or entry into the EU of animals, products of animal origin, plant material or means of transport, equipment, packaging materials, transport water and feed and fodder and other material, carrying viruses and/or vectors; and

1.5 Monitor the geographic spread and potential impact of listed and non-listed VBDs already circulating in the EU, considering among others their transmissibility (per se or linked to vector activity), virulence and zoonotic potential. The monitoring will include:

[1]Commission Implementing Regulation (EU) 2018/1882 on the application of certain disease prevention and control rules to categories of listed diseases and establishing a list of species and groups of species posing a considerable risk for the spread of those listed diseases (OJ L 308, 4.12.2018, p. 21).

1.5.1 Yearly update of the mapping requested in 1.1, 1.2 and 1.3;
1.5.2 Six-monthly newsletter with important highlights about possible changes in distribution, transmissibility, virulence, or zoonotic potential of listed and non-listed VBDs inside or outside the EU;
1.5.3 Contribution to monthly automated West Nile Fever monitoring reports in collaboration with ECDC.

2 In accordance with Article 29 of Regulation (EC) No 178/2002, the Commission requests EFSA to provide a scientific opinion on the risk posed by VBDs for the EU; the following aspects are of particular relevance for the scientific opinion:
2.1 Assess the probability of introduction (i.e. the probability of entry of the pathogen from extra or intra-EU origin, exposure and establishment) of listed and non-listed VBDs identified in 1.1, into previously free EU Member States, considering the relevant pathways identified in 1.4; describe possible options to prevent such introduction;
2.2 Assess the extent of spread of listed and non-listed VBDs in the previously free EU Member States, after local transmission has taken place, with a potential expected timespan for this spread;
2.3 Assess the impact of the introduction and potential further spread of listed and non-listed VBDs during 1 year after the introduction;
2.4 Critically assess the currently available risk mitigation measures for VBDs in the EU, in particular different biosecurity and surveillance systems, regionalisation and vaccination tools; and
2.5 Assess the need for the development of these and further measures within the EU, notably to enable safe intra-EU movements of animals from affected or non-affected areas.
Consider and describe the uncertainty related to any of the above.

1.3 | Interpretation of the Terms of Reference

This report addresses Term of Reference (TOR) 1.1 by providing a **structured overview of the main characteristics of vector-borne diseases (VBDs)** currently listed by Regulation (EU) 2016/429 (European Union, 2016) and Regulation (EU) 2020/687 (collectively referred to as the Animal Health Law, AHL) and not listed VBDs identified as having potential relevance due to their potential epidemiological impact ('non-listed VBDs'). Non-listed diseases were included if they met *all* the following conditions:

- The pathogen is **absent** or of **unknown status** in more than 50% of EU Member States.
- A **competent vector** is present in the EU.
- The pathogen has been **proven to infect** domestic animal species present in the EU.
- Clinical signs are present in animals *or*, if animals present no clinical signs, the disease can cause **disease in humans**.
- **Sufficient data are available**, i.e. primary data on pathogen distribution, pathogenesis in animals, epidemiology, and competent vectors.

The resulting 25 VBDs **that fulfilled the criteria provided above (12** listed **by Regulation (EU) 2016/429 and Regulation (EU) 2020/687; 13 not** listed) are **summarised** in Table 1. Information used to address these criteria was gathered through an initial scoping review of the scientific literature and complemented by expert judgement provided by the EFSA Working Group on Vector-Borne Diseases. The results were subsequently updated during report drafting based on findings from the systematic literature reviews conducted for TOR 1, including the classification of data availability (Table B1; Figure B1 in Appendix B). To address TOR 1.1, for each of the 25 VBDs, the main aspects of the **disease agent**, their main **hosts**, **transmission routes**, available **diagnosis tests** and **prevention and control options** are provided in dedicated sections in short fact sheets. These sections reflect the structure of the online EFSA's disease profiles, living documents which contain more detailed information on these aspects with interactive maps that are regularly updated. This report provides a static summary of the main characteristics of the VBDs as a basis for the annual update that is requested in TOR 1.5.1. In the updates, new evidence found since the previous reports will be highlighted and detailed information about the sources of the information and up to date maps will be provided on the online disease profiles. Further, a six-monthly newsletter with highlights on the 25 VBDs (TOR 1.5.2) as well as monthly monitoring reports on WNV (TOR 1.5.3) in collaboration with ECDC will be provided **as part of the monitoring activities requested in TOR 1.5**.

To address TOR 1.2, 1.3 and 1.4, three other dedicated scientific reports (SRs) have been prepared. These reports summarise the current knowledge on the:

- Competent vectors of the 25 VBDs (TOR 1.2, addressed by EFSA 2026a);
- Surveillance, prevention and control measures of the 25 VBDs (TOR 1.3, addressed by EFSA 2026b); and
- Risk pathways for their introduction into VBD-free countries in the EU (TOR 1.4, addressed by EFSA 2026c).

These three SRs, together with the present report describing the VBDs, serve as the evidence base ('dossier') for two scientific opinions (SOs) (Figure 1). In the first SO, the risk of introduction, spread and impact of the selected 25 VBDs will be assessed, thereby addressing TOR 2.1, 2.2 and 2.3 of the mandate. In addition, an **Expert Knowledge Elicitation** will be carried out to:

- Review and digest the compiled evidence;
- Critically assess the current risk mitigation strategies in the EU;
- Identify the most appropriate mitigation measures for the 25 selected VBDs under various epidemiological scenarios; and
- Evaluate the need for further development or adaptation of these and other mitigation measures, especially to support safe intra-EU movements of animals from affected or unaffected areas.

The **outcomes of the workshop** will be summarised and form the basis of the second Scientific Opinion that will address **TOR 2.4 and 2.5** of the mandate (Figure 1).

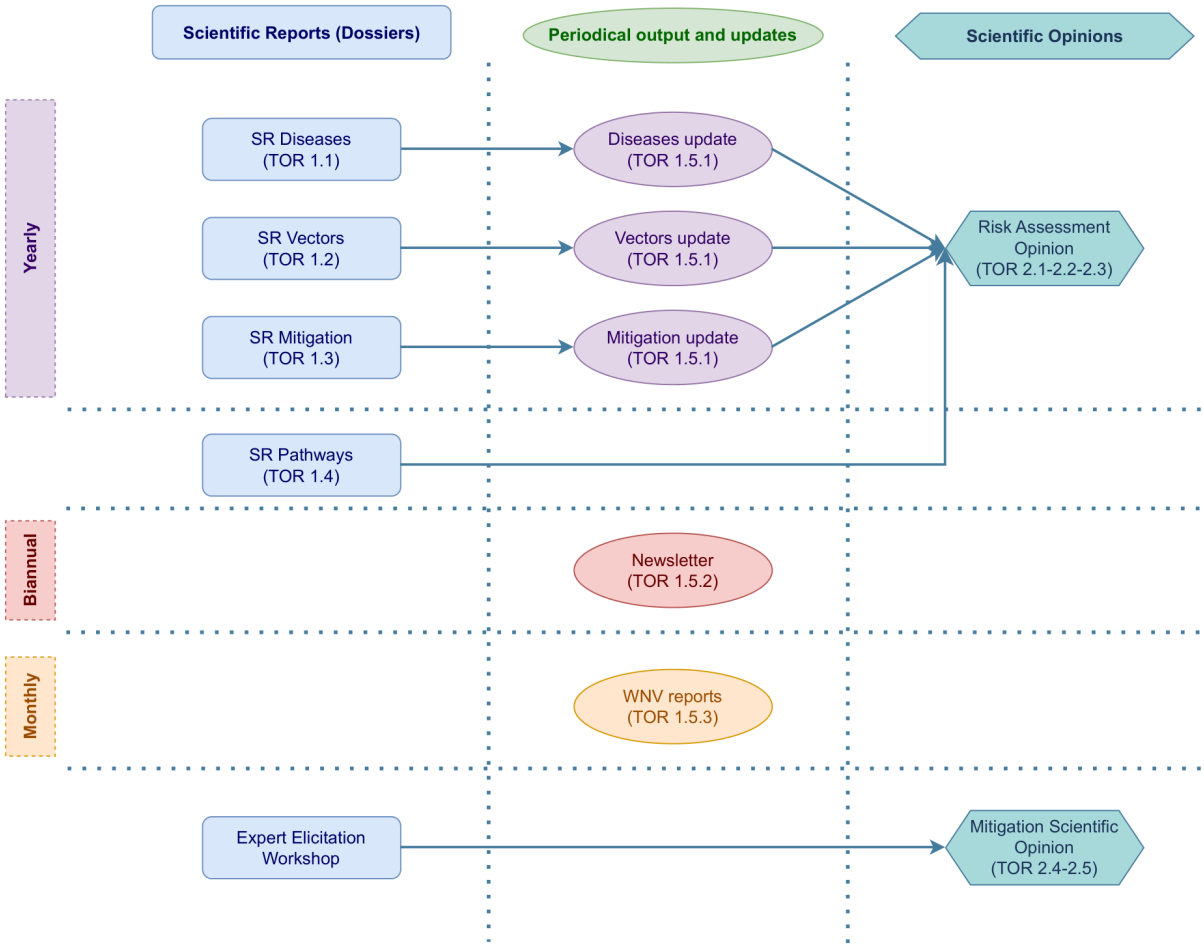


FIGURE 1 VBDs mandate workflow and outputs periodical update.

TABLE 1 Vector-borne diseases that met the eligibility criteria to be included in TOR 1.

Disease -agent	Listed disease in AHL*	Data availability**	Vector group	Pathogen present in EU	Vector(s) present in EU	Listed in WOAH	Zoonotic potential	Main amplifying hosts	Dead-end hosts	Clinical signs in animals
African horse sickness virus (AHSV)	A, D, E	++	Culicoides	✗	✓	YES		Equids		✓
Akabane virus (AKAV)		++	Culicoides	✗	✓	NO		Domestic ruminants		✓
Besnoitia besnoiti (*B. besnoiti*)		++	*Stomoxys*, Tabanidae***	✓	✓	NO		Cattle		✓
Bluetongue virus (BTV)	C, D, E	+++	Culicoides	✓	✓	YES		Ruminants, camelids	Dogs	✓
Borrelia burgdorferi s.l. (*B. burgdorferi*)		+++	Ixodidae	✓	✓	NO		Wide host range, rodents, birds	Dogs horses, humans	✓
Bovine ephemeral fever virus (BEFV)		++	Culicoides	✗	✓	NO		Cattle		✓
Cache Valley virus (CVV)		+	Culicidae	✗	✓	NO		Deer	Ruminants, horses, humans	✓

(Continues)

TABLE 1 (Continued)

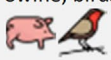
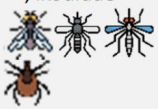
Coxiella burnetii (*C. burnetii*) 	E	+++	Ixodidae 	✓	✓	YES		Wide host range, ruminants 	Humans	✗
Crimean-Congo haemorrhagic fever virus (CCHFV) 		++	Ixodidae 	✓	✓	YES		Wide host range, ruminants 	Equids 	✗
Eastern equine encephalitis virus (EEEV) **** and Western equine encephalitis virus (WEEV)**** 	E	+	Culicidae 	✗	✓	YES		Birds 	Equids, humans 	✓
Epizootic haemorrhagic disease virus (EHDV) 	D, E	++	Culicoides 	✓	✓	YES		Cattle, cervids 		✓
Equine infectious anaemia virus (EIAV) 	D, E	++	Stomoxys, Tabanidae*** 	✓	✓	YES		Equids 		✓
Japanese encephalitis virus (JEV) 	E	++	Culicidae 	✗	✓	YES		Swine, birds 	Equids, humans 	✓
Leishmania infantum (*L. infantum*) 		+++	Phlebotominae 	✓	✓	YES		Canids, cats, lagomorphae , 	Equids 	✓

TABLE 1 (Continued)

Lumpy skin disease virus (LSDV)	A, D, E	++	*Stomoxys*, Tabanidae, Culicidae, Culicoides ***, Ixodidae 	✓	✓	YES		Cattle 		✓
Rift Valley fever virus (RVFV) 	A, D, E	+++	Culicidae 	✗	✓	YES		Ruminants, camelids 	Humans	✓
Schmallenberg virus  (SBV)		++	Culicoides 	✓	✓	NO		Ruminants 		✓
Shuni virus (SHUV) 		+	Culicoides Culicidae 	✗	✓	NO		Domestic ruminants, horses 	Humans	✓
St. Louis encephalitis virus (SLEV) 		+++	Culicidae 	✗	✓	NO		Birds 	Horses, humans 	✓
Tick-borne encephalitis virus (TBEV) 		++	Ixodidae 	✓	✓	NO		Wide host range, wild rodents 	Humans	✓

(Continues)

TABLE 1 (Continued)

Trypanosoma evansi (*T. evansi*)	D, E	++	Stomoxys***	✓	✓	YES	Unclear	Ruminants, camelids, equids	Humans, Pigs	✓
Trypanosoma vivax (T. vivax)		+++	Glossinidae, Tabanidae, *Stomoxys****	✗	✓	YES		Ruminants, camelids, equids		✓
Venezuelan equine encephalitis virus (VEEV)	D, E	+	Culicidae	✗	✓	YES		Horses, wild rodents	Humans	✓
Vesicular stomatitis virus (VSV)		+	Culicoides	✗	✓	NO		Cattle, equids, pigs	Humans	✓
West Nile virus (WNV)	E	+++	Culicidae	✓	✓	YES		Birds	Horses, humans	✓

Note: *Listed disease categories and definition as described in Regulation (EU) 2016/429; **Data availability based on the systematic literature reviews available on the Disease Profiles (https://animal-diseases.efsa.europa.eu/) and VectorNet for vector competence: +++well documented (> 100 publications), ++some papers available (51–100 publications), + only few papers available (0–50 publications); ***Mechanical transmission; ****EEEV and WEEV will be summarised together in this report, in line with the AHL and the WOAH terrestrial code. However, their risk will be assessed separately in the Scientific Opinion addressing TOR 2.1–2.3.

Table legend		
Pathogen type	Zoonotic potential	EU distribution
Virus Bacteria Parasite	Zoonotic Non-zoonotic	✓ Present in the EU ✗ Absent in the EU

2 | METHODOLOGY

A methodology for systematic literature review (SLR) has been previously consolidated, aiming at keeping the available knowledge regarding specific animal diseases up to date in seven specific areas of knowledge: experimental infections, pathogen survival, diagnostic tests performance, vaccines, preventive and curative treatments, vector treatments and geographical distribution. The protocol for this methodology documented by Dórea et al. (2022) can be applied systematically to update knowledge when needed. EFSA has kept dedicated databases storing data collected within those seven knowledge areas for 39 vector-borne diseases, updated regularly since 2015, and covering papers published since 1970.

For this specific mandate, the search strings were updated to add the four diseases not already covered (besnoitiosis, equine infectious anaemia, and infections by *Trypanosoma evansi* and *Trypanosoma vivax*), and all literature reviews were updated to include papers published up to 31 December 2025.

The regular SLR includes a title and abstract screening performed independently by two reviewers, a full-text screening performed by one reviewer (the list of eligibility criteria is described in Dórea et al., 2022) and a manual process of data collection. Semi-automated workflows set up using the statistical programming environment R ensure that all data fed into the literature review databased are used to update disease profiles available here: https://animal-diseases.efsa.europa.eu. Readers can find the full data sets in the references section of any of the profiles.

The disease profiles were used as the starting point to elaborate the fact sheets. In addition to the data collected through the literature reviews, evidence regarding the availability of vaccines for each disease was manually added, including vaccine types, commercial names and approval for the administration in the European Union and third countries. The list of disease hosts and common clinical signs was manually checked for completeness, as disease profiles are subjected to the eligibility criteria (e.g. review articles are not included in the SLRs).

Humans are not in scope for the SLR. Information regarding humans as host species was actively searched for this report, but data about morbidity, clinical signs and disease burden were not collected for humans.

All references of papers included in the SLRs, for each pathogen and thematic area, are included in Annex A. Throughout the document, information gathered through the SLRs, in particular when presenting results of compilation and analysis of large numbers of papers, will simply refer to Annex A for the bibliographical sources. Specific statements based on individual references will be cited directly in the document.

The extracted data of the SLR to which this report refers to are available at https://doi.org/10.5281/zenodo.19234573. All the regularly updated information is also available online in the individual disease profile for each VBD, which can be found at https://animal-diseases.efsa.europa.eu/

2.1 | Geographical distribution

2.1.1 | Diseases listed by World Organisation for Animal Health (WOAH)

The geographical distribution for diseases listed in the WOAH Terrestrial Animal Health Code (African horse sickness, bluetongue, Q-fever, Eastern equine encephalitis, Western equine encephalitis, epizootic haemorrhagic disease, equine infectious anaemia, Japanese encephalitis, lumpy skin disease, Rift Valley fever, *Trypanosoma evansi* infection (surra), Venezuelan equine encephalitis, West Nile fever, Crimean–Congo haemorrhagic fever, leishmaniasis (caused by Leishmania infantum) and *Trypanosoma vivax* infection) was compiled using information from that organization, shared publicly through the World Animal Health Information System (WAHIS, https://wahis.woah.org/). Countries are coloured according to the available information regarding their **stable disease situation**. This information is provided by countries through the WOAH monitoring system (semestral reports). The data displayed here reflect the information made available by countries to WOAH.

In the maps presented in this document, the semestral reports for the **last 5 years** were compiled. When different disease situations were reported within this period, the statuses are prioritised in the following order: *Present*, *Suspected* and *Absent*. That is, if in any of the semestral reports in the last 5 years, the country declared the disease status to be 'present', that country will be coloured as 'present' in this document, where only a snapshot of stable disease situations at the time of reporting were included. Countries are only coloured 'Absent' if the disease was never reported in the last 5 years. Where no information is available, this was made clear in the maps (disease absence is differentiated from absence of information regarding disease status).

In addition to the monitoring system, countries also provide immediate notifications through the WOAH early warning system. Data from these immediate reports are ***not*** displayed in this document. *For the newest, up to date information on geographical distribution, readers should always refer to the online version of these disease profiles (*https://animal-diseases.efsa.europa.eu/*)*.

2.1.2 | Diseases not listed by World Organisation for Animal Health (WOAH)

For those diseases where situation updates are not collected regularly by WOAH, this document shows the **geographical distribution of articles included in the SLR**. The SLR on geographical distribution for diseases not listed by WOAH aimed to provide a comprehensive picture of the geographical distribution. However, this is limited by the inclusion criteria used in the SLR, which excludes for instance secondary sources of information. Information not published in peer-reviewed

literature or not in English will not have been captured in the SLR. **The maps in this case reflect our knowledge of where the disease is documented**, but it is not possible to differentiate the absence of disease from the absence of information. Countries in which the studies available aimed specifically to prove disease absence (freedom from disease studies) are specifically highlighted in a different colour if any are available.

2.2 | Definitions and common characteristics

2.2.1 | Terminology

To ensure consistency across assessments, the following terminology describes key concepts, including host categories based on their role in the transmission cycle and pathogen detection. These definitions primarily refer to vector-borne transmission, while recognising that other routes (e.g. direct, vertical, venereal or oral) may occur.

2.2.1.1 | *Host types*

Any animal or human that can become infected with a pathogen following exposure through a competent vector or other route is considered a **susceptible host**. Detection of infection indicates susceptibility but does not necessarily imply a role in natural transmission. In this document, the susceptible hosts are grouped into three main types as shown in Figure 2:

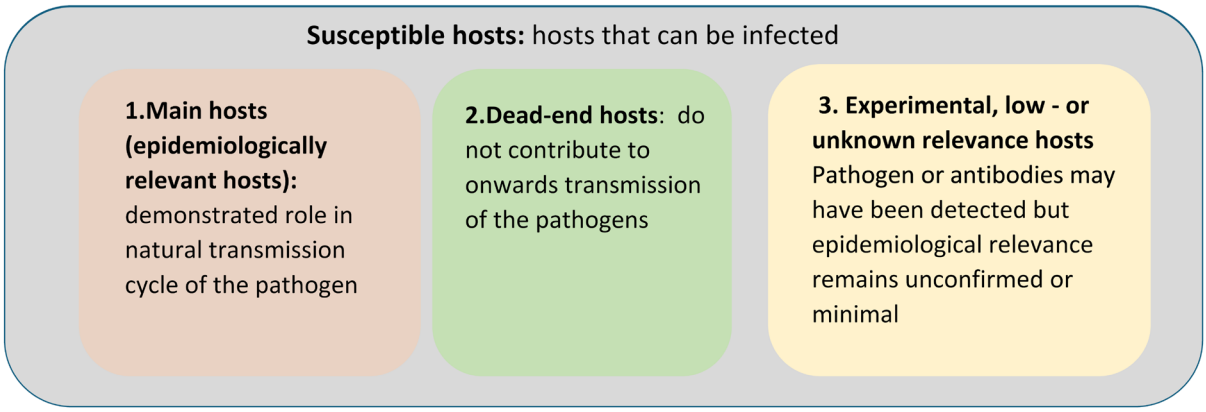


FIGURE 2 Summary of susceptible host categories based on their epidemiological role in VBD pathogen transmission.

1. **Main hosts (epidemiologically relevant hosts)**

Main hosts are species with a demonstrated role in the transmission cycle of the pathogen. These hosts typically develop pathogen titres sufficient to infect competent vectors and are regularly found infected under field conditions. Their contribution to pathogen maintenance, amplification or spread has been scientifically documented.

2. **Dead-end hosts**

Dead-end hosts are species that may become infected, but do not contribute to onward transmission of the pathogen. They generally fail to develop titres sufficient to infect vectors, or their infection does not result in epidemiologically meaningful pathogen dissemination. These hosts may experience clinical disease or seroconversion but do not sustain the transmission cycle.

3. **Experimental, low – or unknown relevance hosts**

Potential hosts are species that may be susceptible, but whose epidemiological relevance remains unconfirmed or minimal. This category includes:

- Species infected only under experimental conditions, without evidence of natural infection.
- Species occasionally infected in the field, but with:
  - Serological evidence only (e.g. antibodies without pathogen detection), or
  - Sporadic detections, with low pathogen titres insufficient to support vector infection or sustained transmission.

These hosts may be considered in hazard identification and surveillance but are not expected to play a significant role in the transmission dynamics of the disease.

2.3 | Common characteristics of vector-borne diseases

The diseases presented in the following assessment are transmitted by arthropod vectors and thus share several common characteristics which influence pathogen geographical distribution, occurrence and prevention.

2.3.1 | Geographical distribution

Disease transmission depends on the presence and activity of the vector species. For strictly vector-borne diseases, transmission cannot occur where the vector is absent.

2.3.2 | Seasonality

Transmission intensity varies seasonally, peaking when vector populations are most abundant. The seasonal pattern occurs because arthropod biology is highly sensitive to meteorological conditions, which affect their development, activity and abundance. In continental Europe, low winter temperatures can interrupt the disease transmission cycle because the vector species become inactive.

2.3.3 | Transmission

Vector-borne disease transmission also depends on vector species' host preference and the extrinsic incubation periods.

- Host preference refers to the vector's tendency to feed on a specific host species. This preference influences which host species are most likely exposed to infection and influences how pathogens circulate between animal and human populations. Depending on the susceptibility of the host species to infection, they may serve as epidemiologically relevant or dead-end hosts.
- The extrinsic incubation period is temperature-dependent and determines how long the pathogen needs to develop inside the vector before it can be transmitted. Higher temperatures shorten this period and allow the vector to become infectious more quickly, thus promoting disease transmission.

2.3.4 | Prevention and control

Information about specific vaccines and pharmaceutical treatments is detailed below for each specific pathogen. An overview of the prevention and control measures laid down by Animal Health Law (AHL) in the EU (Regulation (EU) 2016/429) and a summary of the effectiveness of the available risk mitigation measures for the 25 vector-borne diseases are provided in a dedicated Scientific Report (EFSA, 2026b).

3 | ASSESSMENT

All information compiled for each disease through the SLR processes are presented below. The data collected through the SLR are enhanced through manual curation to provide reading-friendly summaries of the knowledge available. As the literature reviews compiled a great number of references, the references are listed per disease and per literature review theme in Annex A.

When additional references were used, these are cited directly in the text. This can include, for example, review articles which are not eligible in the SLRs but can have been helpful to summarise known knowledge about the diseases.

3.1 | African horse sickness virus (AHSV)[2]

3.1.1 | Disease overview

African horse sickness virus (AHSV) causes African horse sickness, an infectious, non-contagious, *Culicoides*-borne viral disease affecting members of the Equidae family. The disease manifests severe respiratory and circulatory impairment through four main clinical forms: subclinical, subacute, peracute and acute (Mellor & Hamblin, 2004; Spickler, 2024a; WOAH, 2020a).

African horse sickness is a WOAH-notifiable disease, listed in the European AHL under categories A, D and E.

[2]Online version: https://animal-diseases.efsa.europa.eu/AHSV.

3.1.2 | Agent

AHSV is a non-enveloped, double-stranded RNA virus that belongs to the Orbivirus genus of the Reoviridae family. The virion is icosahedral, ~80 nm in diameter and has a genome of 10 linear segments encoding seven structural (VP1–VP7) and at least four non-structural proteins (NS1–NS4). VP2, the outer capsid protein, determines serotype specificity and induces neutralising antibodies (Howell, 1962; Mertens, 2005; Roy, 1996).

Nine serotypes (AHSV-1 to AHSV-9) have been identified, with partial antibodies cross-reactivity observed among certain pairs (e.g. 1 & 2, 3 & 7, 5 & 8, 6 & 9). No cross-reactions with other known orbiviruses have been reported (Howell, 1962; Mellor & Hamblin, 2004).

The virus shows strong stability under environmental conditions, remaining infective in refrigerated blood for extended periods and surviving heating at 55–75°C for 10 min. It is, however, inactivated by oxidising agents and prolonged heating (WOAH, 2020a).

3.1.3 | Geographical distribution

African horse sickness is endemic to sub-Saharan Africa. However, sporadic outbreaks have occurred in the Middle East, Mediterranean Europe and Asia.

According to the World Animal Health Information System (WAHIS) data (Figure 3), the agent was not reported in the EU in the last 5 years. Up to date maps based on WAHIS are available in the online version of the Disease Profile.[2]

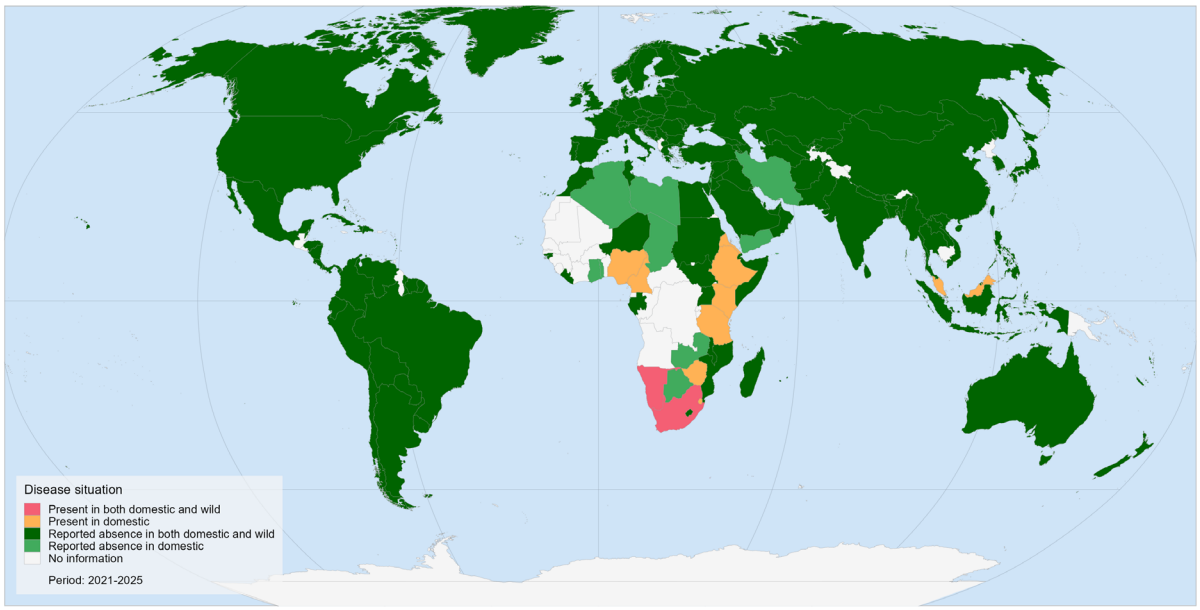


FIGURE 3 Geographical distribution of AHSV detected events (2021–2025), as reported to WOAH.

3.1.4 | Animal hosts

Susceptible hosts

Based on epidemiological knowledge of host–pathogen–vector interactions and outbreak reports, **the main hosts of AHSV are equids**. However, other susceptible species have been identified in the SLR, with the summary provided in Table 2.

TABLE 2 Susceptible host species of African horse sickness virus.

The SLR reported in the AHSV disease profile identified the following susceptible species (updated until 31/12/2025; for references, see Annex A)
FIELD

Epidemiological studies carried out in the field
• **Pathogen was detected in the following animal species:**
  • Canidae: *Canis lupus familiaris*
  • Equidae: *Equus caballus, Equus quagga*
• **Antibodies were detected in the following animal species:**
  • Canidae: *Canis lupus familiaris*
  • Equidae: *Equus caballus, Equus africanus asinus*
• **Outbreaks reported to WOAH included the following species:**
  • Equidae: *Equus quagga*

EXPERIMENTS
• **Experimental studies demonstrated infection in:**   • Equidae: *Equus caballus, Equus africanus asinus*

Clinical signs

African horse sickness occurs in four recognised clinical forms, with severity varying by equid species, immune status and viral strain.

- Pulmonary form (peracute): seen almost exclusively in horses; this form is the most severe. It is marked by high fever, acute respiratory distress and profuse frothy nasal discharge due to pulmonary oedema. Death occurs within 24–48 h, with case fatality close to 100%.
- Cardiac form (subacute): characterised by fever, swelling of the head, eyelids and supraorbital fossae, with variable dyspnoea. The course is longer than the pulmonary form, and case fatality is typically 50%–70%. This form occurs mainly in horses and occasionally in mules.
- Mixed form (acute): combines respiratory distress with oedema of the head and neck and is seen in horses and mules. Case fatality rates are high, averaging around 70%.
- Horse sickness fever (mild): clinical signs are limited to transient fever and mild malaise, followed by recovery. It predominantly affects donkeys and zebras but may also occur in partially immune horses.

Outcomes of a SLR of clinical signs in 24 study groups of equids are displayed in Figure 4. Predominantly, general, respiratory and cardiovascular clinical signs were reported.

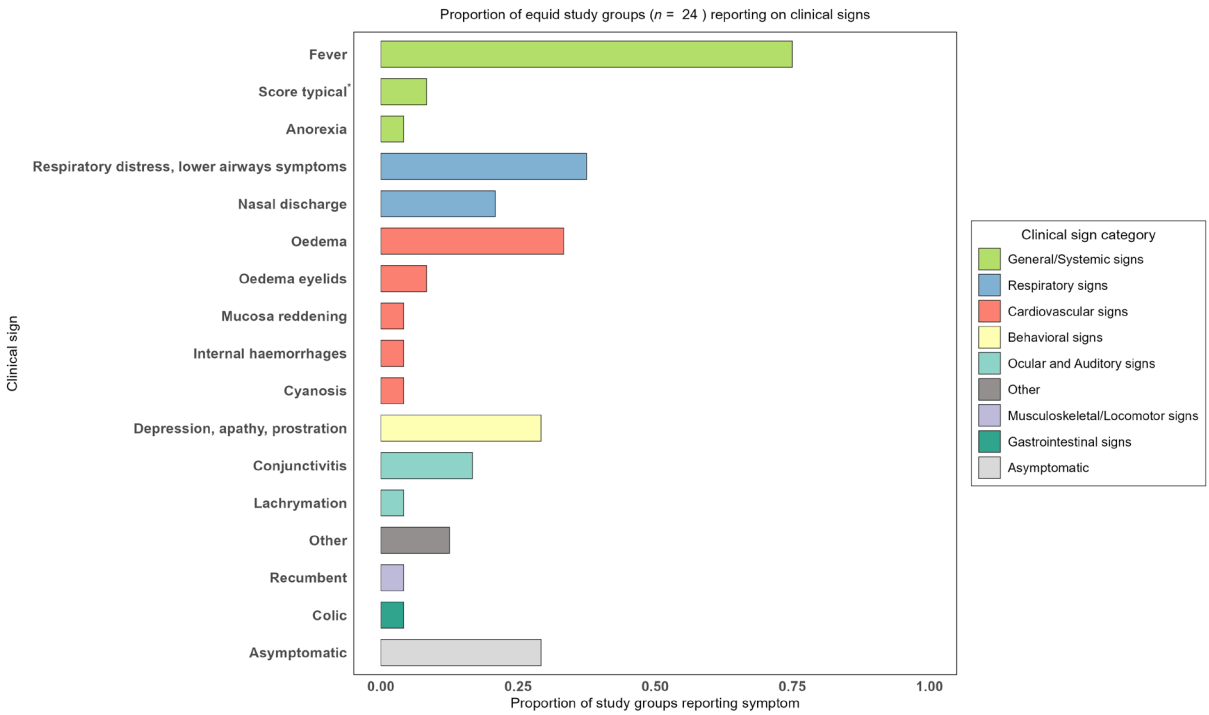


FIGURE 4 Clinical signs reported in the main hosts of AHSV. *Score typical: swelling of the head ('Dikkop'). Study group count per equid species: Horse $n = 23$; Donkey $n = 1$. The SLR was updated until 31 December 2025; for references, see Annex A.

Incubation period

The incubation period of AHS in equids is considered to range between 3 days and 2 weeks. The peracute pulmonary form tends to emerge more rapidly, with fever and respiratory signs appearing only a few days after infection. The cardiac form has a longer development, with oedema and circulatory signs often not evident until a week or more after exposure (Spickler, 2024a, 2024b, 2024c, 2024d; WOAH, 2020a).

In donkeys and zebras, infection is generally subclinical, and the incubation period is difficult to define, as animals may seroconvert without developing observable disease symptoms (Barnard, 1998).

Morbidity and case fatality

Horses are highly susceptible to AHSV, with case fatality ranging from 50% to 95%, depending on the clinical form. The pulmonary form is almost always fatal, while the cardiac and mixed forms are associated with case fatality rates of 50%–70%. In naïve horse populations, morbidity can be very high during outbreaks (up to 100% reported in naïve horses) (Mellor & Hamblin, 2004; Spickler, 2024a, 2024b, 2024c, 2024d; WOAH, 2020a).

Mules show intermediate susceptibility, with mortality around 50%. Donkeys are generally more resistant; mortality is usually 5%–10% in non-African breeds, while African donkeys often show only subclinical infections. Zebras rarely develop clinical disease (Barnard, 1998).

Other species, such as dogs, may die from peracute infection after consuming infected meat, but they are not relevant for transmission (Spickler, 2024a, 2024b, 2024c, 2024d).

Zoonotic potential

AHSV is not known to infect humans under natural conditions (WOAH, 2020a).

3.1.5 | Transmission

African horse sickness is primarily transmitted by biting midges of the genus *Culicoides* (Diptera; Ceratopogonidae). Evidence on the potential and likely competent *Culicoides* species and their geographic distribution can be found in a dedicated Scientific Report on vectors (EFSA, 2026a). The virus does not spread via direct contact between equids, although iatrogenic transmission through blood is possible.

3.1.6 | Diagnostic tests

Recommended tests (WOAH, 2025b) for agent detection are virus isolation and real-time PCR (RT-PCR).

For immune response detection, the recommended tests are ELISA (serogroup specific, based on VP7), complement fixation test (CFT) and virus neutralisation (VN).

Table 3 presents data on the sensitivity and specificity of diagnostic tests collected through SLR[3]; reported values correspond to the median sensitivity and specificity when multiple studies investigated the same test and are only included when explicitly stated in the publications.

TABLE 3 Median sensitivity and specificity of tests to detect AHSV/AHSV antibodies reported in literature included in the systematic literature review.

Target	Test	Species	Specificity	*N* animal groups	Sensitivity	*N* animal groups	References
Antigen	ELISA	Horse	100%	3	100%	3	Rubio et al. (1998)
Nucleic acid	Real-time RT-PCR	Horse	99.9%	1	97.8%	1	Guthrie et al. (2013)
Virus	Virus isolation	Horse	100.0%	1	72.1%	2	Guthrie et al. (2013); Laviada et al. (1992)
Antibody	Blocking ELISA	Horse	98.5%	2	99.2%	6	Durán-Ferrer et al. (2019)
Antibody	Competitive ELISA (C-ELISA)	Horse	100%	3	100%	4	Rubio et al. (1998); Hamblin et al. (1992); Kweon et al. (2003)
Antibody	ELISA	Horse	100%	3	100%	3	Rubio et al. (1998)
Antibody	Sandwich ELISA	Horse	100%	1	97.4%	1	El Hasnaoui et al. (1998)

[3]Eligibility criteria for the review can be found in the systematic literature review protocol (Dórea et al., 2022).

TABLE 3 (Continued)

Target	Test	Species	Specificity	*N* animal groups	Sensitivity	*N* animal groups	References
Antibody	Immunofluorescence Assay	Ass	83.3%	1	98.0%	1	El Hasnaoui et al. (1998)
Antibody	Immunofluorescence Assay	Horse	83.3%	1	98.0%	1	Laviada et al. (1992)

Note: The SLR was updated until 31 December 2025; for references, see Annex A.

3.1.7 | Prevention and control

Vaccination

Currently, there are no commercially available vaccines against AHS authorised in the EU, but in endemic regions, monovalent and polyvalent attenuated-live vaccines are available for horses, mules and donkeys (Guthrie et al., 2009; Spickler, 2024a, 2024b, 2024c, 2024d; WOAH, 2020a, 2025b).

Treatment

There is currently no specific antiviral treatment for AHSV infection, and management is primarily supportive on an individual basis (Spickler, 2024a, 2024b, 2024c, 2024d; WOAH, 2020a).

3.2 | Akabane virus (AKAV)[4]

3.2.1 | 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 characterised by reproductive losses resulting from transplacental infection, leading to abortions, stillbirths and congenital malformations in newborns (Spickler, 2018; WOAH, 2025b).

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

3.2.2 | 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 neutralising antibodies (Elliott, 1990, 2014; Kobayashi et al., 2007). The virus is moderately stable under ambient conditions but is inactivated by lipid solvents, detergents and standard disinfectants (Spickler, 2018).

3.2.3 | 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 & Lear, 2020).

Episodic incursions have occurred in southern Europe (Türkiye), 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 SLRs (updated until 31 December 2025) and summarised in Figure 5. All references can be found in Annex A and in the online version of the Disease Profile.[4]

[4]Online version: https://animal-diseases.efsa.europa.eu/AKAV.

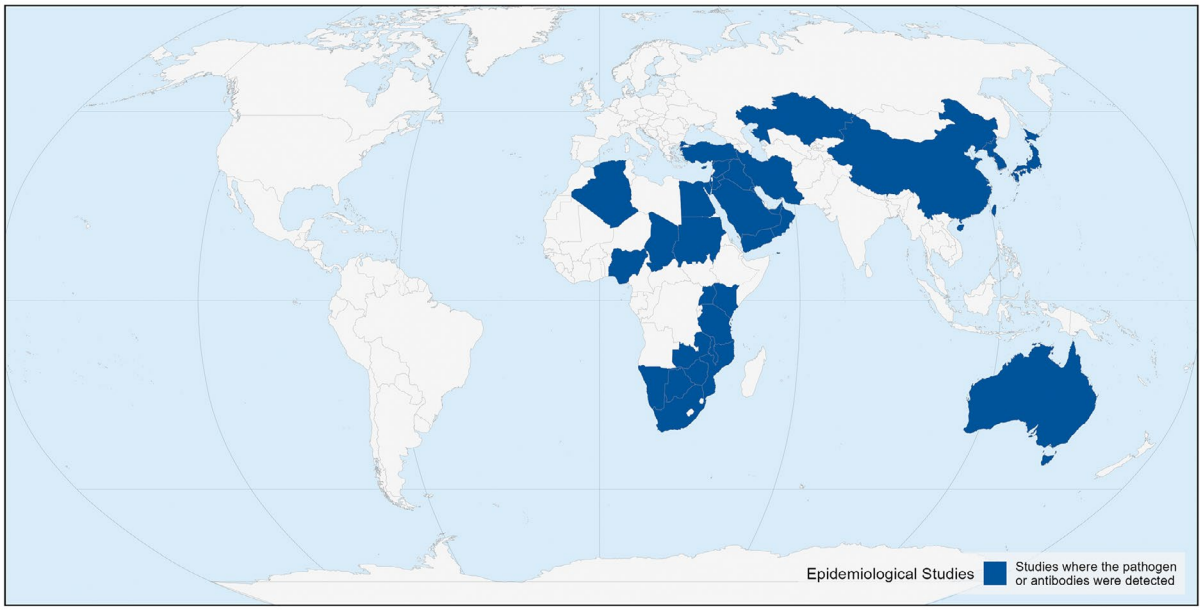


FIGURE 5 Geographical distribution of epidemiological studies addressing the occurrence of AKAV, as identified by the EFSA's systematic literature review (covering years 1970–2025).

3.2.4 | Animal hosts

Based on epidemiological knowledge of host–pathogen–vector interactions and outbreak reports, the main hosts of AKAV are domestic ruminants. However, other species have been identified in the SLR. The SLR summary is given in Table 4.

TABLE 4 Susceptible host species of Akabane virus.

The SLR reported in the AKAV disease profiles, identified the following susceptible species (updated until 31/12/2025; for references, see Annex A)
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*

Clinical signs

Outcomes of a SLR on clinical signs in 13 domestic ruminant study groups are displayed in Figure 6. Most study groups did not show any clinical signs or had reproductive clinical signs.

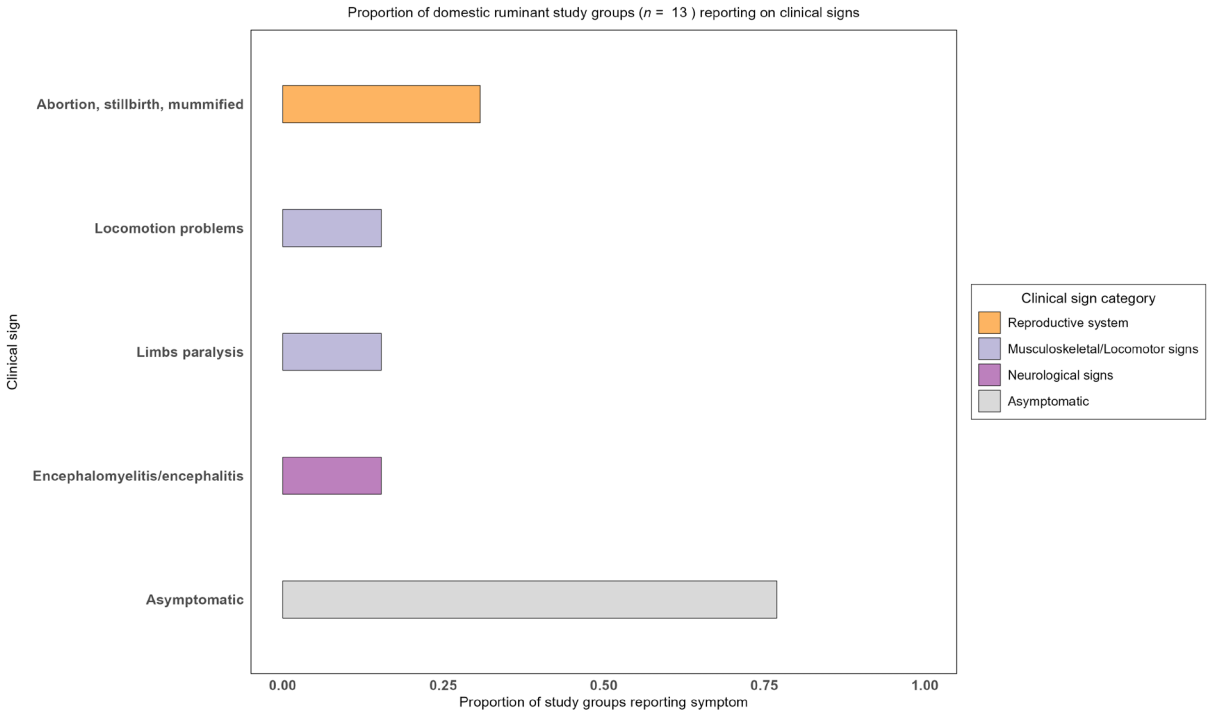


FIGURE 6 Clinical signs reported in the main hosts of AKAV. Study group count per domestic ruminant species: Cattle $n=6$; Goat $n=4$; Sheep $n=3$. The SLR was updated until 31 December 2025; for references, see Annex A.

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–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.

Incubation period

The incubation period in adult animals is not well defined due to the lack of clinical signs, but viraemia, which can lead to vertical transmission, usually occurs 1–6 days after infection (USDA, 2015; Annex A). Clinical disease in fetuses becomes apparent only at birth or abortion, weeks to months after maternal infection, depending on gestational timing (Kurogi et al., 1977; Spickler, 2018).

Morbidity and case fatality

In adult ruminants, most infections are asymptomatic, but clinical signs have been reported, as shown in Figure 6. 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; Annex A).

The primary impact is on the fetus, 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% and 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% (Spickler, 2018; USDA, 2015).

Zoonotic potential

AKAV is not known to infect humans under natural conditions.

3.2.5 | Transmission

AKAV is transmitted by biting midges of the genus *Culicoides* (Diptera; Ceratopogonidae). Evidence on the potential and likely competent *Culicoides* species and their geographic distribution can be found in a dedicated Scientific Report on vectors (EFSA, 2026a).

The virus is not spread by direct contact between animals or via fomite (USDA, 2015). 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).

3.2.6 | Diagnostic tests

WOAH-recommended tests (WOAH, 2025b) 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 fetal 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 fetal tissues may support diagnosis when molecular methods are unavailable, although sensitivity is lower than PCR-based assays (WOAH, 2025b).

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 fetal fluids from malformed or stillborn neonates before colostrum intake provides evidence of in utero infection (WOAH, 2025b).

To date, no diagnostic test evaluation studies meeting the eligibility criteria for inclusion have been identified through the SLR.

3.2.7 | Prevention and control

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 fetal infection and congenital malformations (Kirkland, 2015; Kurogi et al., 1978; WOAH, 2025b).

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

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).

3.3 | *Besnoitia besnoiti* (*B. besnoiti*)[5]

3.3.1 | Disease overview

Besnoitia besnoiti causes besnoitiosis, a parasitic disease which affects cattle. It is still unclear how besnoitiosis is transmitted under natural conditions. The most likely way of transmission within cattle herds is mechanical transmission by biting insects. An infection can progress asymptomatically, but can also lead to general illness, followed by a chronic disease, characterised by skin lesions and sand-like cysts in the mucous membranes and connective tissues (Lahondes et al., 2025).

Besnoitiosis is *not* a WOAH-notifiable disease, and it is not listed in the EU AHL.

[5]Online version: https://animal-diseases.efsa.europa.eu/Besnoitia.

3.3.2 | Agent

Besnoitia besnoiti is a cyst-forming, obligate intracellular protozoan belonging to the phylum Apicomplexa (family Sarcocystidae) (Coelho et al., 2023; Lahondes et al., 2025).

Besnoitia besnoiti parasites have different life stages. Tachyzoites are the proliferative stage and are present in the host species during acute disease. They are 6–7.5 × 2.5–3.9 μm and can reside within many different host cells. Bradyzoites are 6.0–7.5 × 1.9–2.3 μm and they persist in tissue cysts in chronically infected animals. Tissue cysts can contain hundreds of parasites (Cortes et al., 2014).

3.3.3 | Geographical distribution

Besnoitia besnoiti has been reported in Africa, South America and Asia and is emerging in Europe (Oryan et al., 2025). It has spread into Portugal, Spain, France, Greece and Ireland, and outbreaks have been reported in Germany, Italy, Hungary, Switzerland and Belgium (Basso et al., 2013; Delooz et al., 2021; EFSA, 2010; Oryan et al., 2025; Annex A).

Infection with *Besnoitia besnoiti* 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 SLRs (updated until 31 December 2025) and summarised in Figure 7. All references can be found in Annex A and in the online version of the Disease Profile[5].

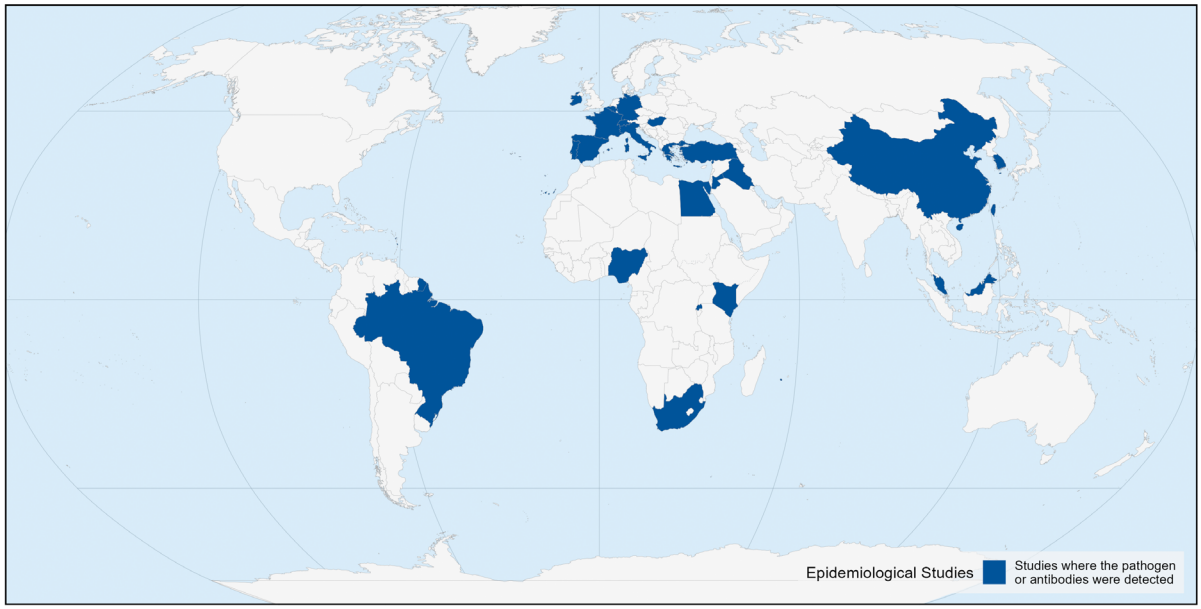


FIGURE 7 Geographical distribution of epidemiological studies addressing the occurrence of *Besnoitia besnoiti*, as identified by the EFSA's systematic literature review (covering years 1970–2025).

3.3.4 | Animal hosts

Based on epidemiological knowledge of host–pathogen–vector interactions and outbreak reports, the main hosts of *B. besnoiti* are cattle. However, other susceptible species have been identified in the SLR. The SLR summary is given in Table 5.

TABLE 5 Susceptible host species of *Besnoita besnoiti*.

The SLR reported in the *B. besnoiti* disease profiles, identified the following susceptible species (updated until 31/12/2025; for references, see Annex A)
FIELD
Epidemiological studies carried out in the field

• **Pathogen was detected in the following animal species:**
  • Bovidae: *Bos taurus*
• **Antibodies were detected in the following animal species:**
  • Bovidae: *Bos taurus, Ovis aries, Bos grunniens, Bubalus bubalis*
  • Cervidae: *Cervus elaphus, Capreolus capreolus*
• **Outbreaks reported to WOAH included the following species:**
  • No species specified

(Continues)

TABLE 5 (Continued)

The SLR reported in the *B. besnoiti* disease profiles, identified the following susceptible species (updated until 31/12/2025; for references, see Annex A)

EXPERIMENTS

- **Experimental studies demonstrated infection in:**
  - Bovidae: *Bos taurus*
  - Felidae: *Felis catus*
  - Leporidae: *Oryctolagus cuniculus*

Clinical signs

Outcomes of a SLR on clinical signs in 32 cattle groups are displayed in Figure 8. Most study groups showed general and cardiovascular clinical signs.

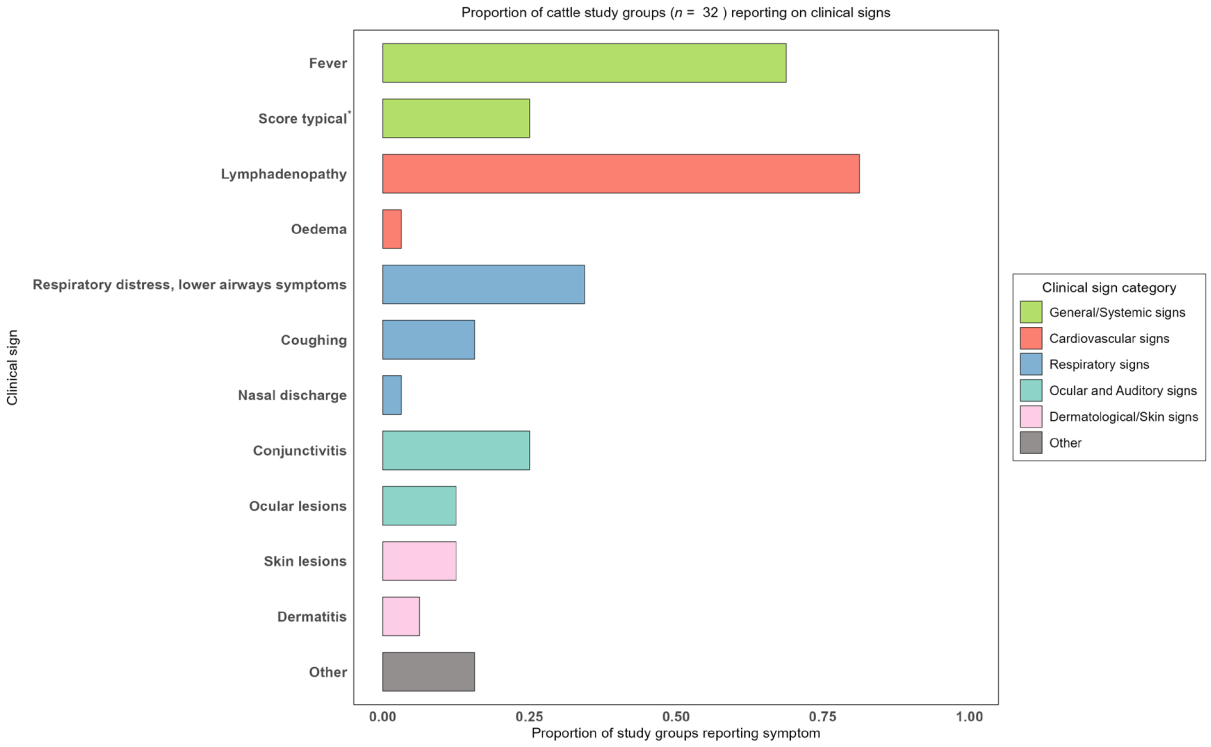


FIGURE 8 Clinical signs reported in the main hosts of *B. besnoiti*. *Score typical: visible scleral tissue cysts. The SLR was updated until 31 December 2025; for references, see Annex A.

Clinical signs in cattle develop in different phases. After infection, an acute phase is seen, which is caused by multiplication of tachyzoites. Affected animals have high fever (above 40°C) and show non-specific signs like depression, weight loss, photophobia, nasal and ocular discharge, lymph node swelling and decreased milk production. Oedema, with general swelling of the whole body, can be seen. Seven to 14 days after the start of the acute phase, a chronic phase develops, caused by cyst formation in the mucous membranes and connective tissues. These cysts can be seen by the naked eye in the conjunctiva, sclera, lips and vulval mucosa. Cyst forming can lead to hyperkeratosis, alopecia, folding of the skin and sometimes even shedding of the epidermis (elephantiasis/elephant skin). Male animals can become infertile because of necrotizing orchitis (Lahondes et al., 2025; Oryan et al., 2025; SLR).

Incubation period

The incubation period in cattle varies from 6 to 13 days (Delooz et al., 2021; Jacquiet et al., 2010; Oryan et al., 2025). In experimental infections in cattle, the incubation period varied between 1 and 40 days (Annex A).

Morbidity and case fatality

Most infected animals remain asymptomatic, with cysts in the mucous membranes as the only visible sign (EFSA, 2010; Lahondes et al., 2025; Oryan et al., 2025). A small proportion of infected animals develop clinical signs, varying from 1%–10% in endemic regions to 15%–20% in emerging regions (Lahondes et al., 2025). Severely affected animals might die. The case fatality rate during the chronic stage is around 10% (EFSA, 2010; Oryan et al., 2025).

Zoonotic potential

Besnoitia besnoiti is not known to infect humans under natural conditions.

3.3.5 | Transmission

It is still unclear how besnoitiosis is transmitted under natural conditions. It is assumed that it has an indirect life cycle, with a yet unidentified definitive host that is shedding oocysts.

The most likely way of transmission within cattle herds is mechanical transmission by biting flies. Furthermore, transmission via direct contact between animals with ruptured cysts on mucous membranes or skin lesions might be possible. Another option might be iatrogenic transmission when needles are reused. *B. besnoiti* has been found in bull's semen, so natural mating might also play a role in transmission (EFSA, 2010; Lahondes et al., 2025; Oryan et al., 2025).

Since most clinical signs are seen in summer, when cattle herds are out on the pasture and blood-sucking flies are most active and present, mechanical transmission by these insects seems to be a likely scenario.

Evidence on the potential and likely competent vector species and their geographic distribution can be found in a dedicated Scientific Report on vectors (EFSA, 2026a).

3.3.6 | Diagnostic tests

As the disease is not WOAH listed, there are no WOAH-recommended diagnostic tests.

In the acute phase of the infection, it can be difficult to diagnose the disease due to non-specific clinical signs. Characteristic clinical signs appear when tissue cysts start to develop in the chronic phase. Then, the disease can be diagnosed by histopathology of a skin biopsy. This method is considered the gold standard (EFSA, 2010; Lahondes et al., 2025; Oryan et al., 2025).

In acutely infected animals, conventional PCR or real-time PCR from skin samples is more sensitive than serological methods, because in this phase, no antibodies have been developed yet. However, in subclinically infected animals, with a low number of cysts, PCR might give a negative result (Oryan et al., 2025).

Indirect methods, detecting specific antibodies in the serum of infected animals, can be used for diagnosing clinical and subclinical infections. Immunofluorescence assays can be used for serological testing. They are more sensitive and specific than ELISA. However, ELISA tests are more appropriate when testing larger numbers of samples in epidemiological studies and diagnosing besnoitiosis in herds. It is recommended to confirm samples that test positive in ELISA with western blot because ELISA testing might result in false positives due to cross-reactions with other Apicomplexan parasites (Lahondes et al., 2025; Oryan et al., 2025).

Table 6 presents data on the sensitivity and specificity of diagnostic tests collected through SLR; reported values correspond to the median sensitivity and specificity when multiple studies investigated the same test and are only included when explicitly stated in the publications.

TABLE 6 Median sensitivity and specificity of tests to detect *B. besnoiti*/*B. besnoiti* antibodies reported in literature included in the systematic literature review.

Target	Test	Species	Sensitivity	*N* animal groups	Specificity	*N* animal groups	References
Antigen	ELISA	Cattle	91.7%	3	97.7%	3	García-Lunar et al. (2017)
Antigen	ELISA	Wild ruminants	95.7%	1	96.8%	1	
Antibody	ELISA	Cattle	88.3%	9	97.6%	9	Cortes et al. (2006); García-Lunar et al. (2013)
Antibody	Immunoblot	Cattle	90.0%	1	100%	1	Schares et al. (2010)
Antibody	I-ELISA	Cattle	100%	1	100%	1	Fernández-García et al. (2010)
Antibody	IFAT	Cattle	91.9%	5	95.4%	5	García-Lunar et al. (2013)
Antibody	Western blot	Cattle	90.2%	17	98.3%	17	Cortes et al. (2006); García-Lunar et al. (2013)

Note: The SLR was updated until 31 December 2025; for references, see Annex A.

3.3.7 | Prevention and control

Vaccination

There is no licensed vaccine available in Europe. Some countries (Israel, South Africa) have used live-attenuated vaccines. They protect animals from clinical disease but do not prevent the introduction of *B. besnoiti* into naïve herds. Furthermore, live-attenuated vaccines could lead to the introduction of the parasite into free herds, and vaccinated animals can become carriers of the parasite (Alvarez-García et al., 2013; Cortes et al., 2014; De Vos & Bock, 2000).

Treatment

There is currently no effective treatment for besnoitiosis in cattle (Lahondes et al., 2025; Oryan et al., 2025). It was found that oxytetracycline (OTC) prevented the development of orchitis in experimentally infected rabbits. OTC also seemed to have some therapeutic potential against the disease. OTC also prevented death in infected gerbils (Oryan et al., 2025; Shkap et al., 1985, 1987).

3.4 | Bluetongue virus (BTV)[6]

3.4.1 | Disease overview

Bluetongue virus (BTV) causes bluetongue (BT), an infectious *Culicoides*-borne viral disease that mainly affects ruminants and camelids. Infection with the virus results in a clinical picture that varies from very mild or subclinical clinical signs to a systemic haemorrhagic viral fever (WOAH, 2021a).

Bluetongue is a WOAH-notifiable disease, listed in the European AHL under categories C, D and E.

3.4.2 | Agent

Bluetongue virus is a non-enveloped, double-stranded RNA virus that belongs to the *Orbivirus* genus of the *Reoviridae* family. According to the World Organisation for Animal Health (WOAH), there are at least 27 officially recognised BTV serotypes, named BTV-1 to BTV-27 (WOAH, 2021a).

BTV has an icosahedral structure with three protein layers, composed of seven structural proteins (VP1-VP7), along with five non-structural proteins (NS1–NS5). The inner layer, composed of VP3 capsomers, encloses 10 dsRNA segments. The intermediate layer is composed of VP7, and the outer layer comprises two proteins: VP2 and VP5. VP7 determines serogroup specificity; it expresses antigens common to all BTV strains and serotypes included in the serogroup; and it is also used to detect anti-BTV antibodies in C-ELISAs. VP2 determines serotype specificity (WOAH, 2025b).

3.4.3 | Geographical distribution

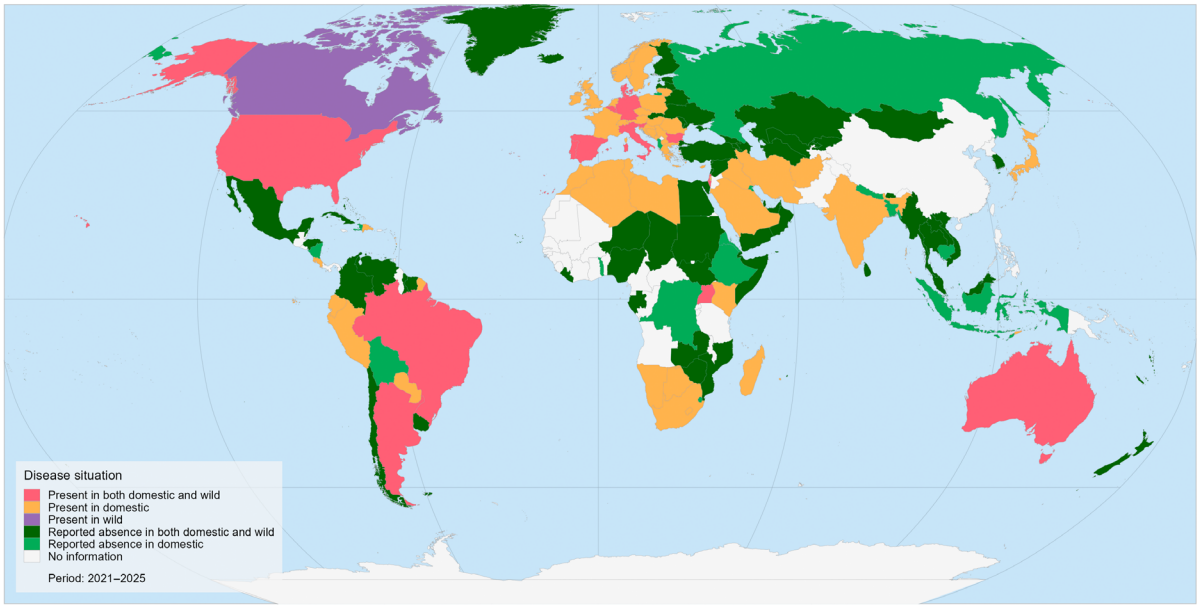


FIGURE 9 Geographical distribution of BTV-detected events (2021–2025), as reported to WOAH.

BTV has been detected across nearly all continents and is currently endemic in the EU. Figure 9 displays where the pathogen has been reported to WOAH in 2021–2025. The most reported serotypes in WAHIS in the past 2 years were BTV3, BTV8 and BTV4. Up-to-date maps based on WAHIS data are available in the online version of the Disease Profile.[6]

[6]Online version: https://animal-diseases.efsa.europa.eu/BTV.

3.4.4 | Animal hosts

Susceptible hosts

Based on epidemiological knowledge of vector ecology and outbreak reports, the main hosts of BTV are wild and domestic ruminants and camelids, whereas dogs are considered dead-end hosts. However, other susceptible species have been identified in the SLR. The SLR summary is given in Table 7.

TABLE 7 Susceptible host species of bluetongue virus.

The SLR reported in the BTV disease profile, identified the following susceptible species (updated until 31 December 2025; for references, see Annex A)

FIELD

Epidemiological studies carried out in the field

- **Pathogen was detected in the following animal species:**
  - Bovidae: *Bos taurus, Capra hircus, Ovis aries*
  - Camelidae: *Camelus dromedarius*
  - Canidae: *Canis lupus familiaris*
- **Antibodies were detected in the following animal species:**
  - Bovidae: *Bison bison, Bubalus bubalis, Bos taurus, Capra hircus, Ovis aries, Bos grunniens*
  - Camelidae: *Camelus dromedarius, Camelus bactrianus, Vicugna pacos, Lama glama*
  - Cervidae: *Odocoileus hemionus*
  - Canidae: *Canis lupus familiaris*
- **Outbreaks reported to WOAH included the following species:**
  - Bovidae: *Bison bonasus, Bos grunniens, Bos taurus, Capra hircus, Capra ibex, Ovis ammon, Ovis orientalis, Ovis aries, Rupicapra pyrenaica, Rupicapra rupicapra, Bubalus bubalis, Bubalus* spp.
  - Camelidae: *Vicugna pacos*
  - Cervidae: *Capreolus capreolus, Cervus elaphus, Dama dama, Mazama nana*
  - Suidae: No species specified
  - Hominidae: *Pan troglodytes*

EXPERIMENTS

- **Experimental studies demonstrated infection in:**
  - Bovidae: *Bos taurus, Capra hircus, Ovis aries, Bubalus* spp.
  - Camelidae: *Camelus bactrianus, Vicugna pacos, Lama glama*
  - Cervidae: *Odocoileus hemionus columbianus, Cervus elaphus, Cervus canadensis, Odocoileus virginianus, Muntiacus* spp.
  - Canidae: *Canis lupus familiaris*

Clinical signs

Both the severity and length of clinical manifestations vary depending on host species and breed, viral serotype or strain, husbandry conditions and the surrounding environment (Spickler, 2023a, WOAH, 2021a).

Sheep are considered the most clinically affected species – especially fine-wool breeds, such as Merino and crosses, and mutton breeds. BTV can cause widespread capillary damage, which leads to oedema, congestion, haemorrhage, inflammation, intravascular coagulation and necrosis.

- Respiratory signs: Nasal discharge (firstly serous, becoming mucopurulent within days, often forming crusts around the nostrils and muzzle), pulmonary oedema and second bacterial bronchopneumonia. Peracute cases can die after 7–9 days, due to severe respiratory signs that lead to asphyxiation.
- Skin: Hyperaemia observed in hair-free areas (groin, axillae and perineum). In chronic cases, there is wool breakage caused by an underlying dermatitis.
- Gastrointestinal tract: Diarrhoea, sometimes bloody.
- Gestation: Infection during early gestation often results in abortion or congenital malformations such as hydranencephaly ('dummy calf syndrome') or 'blue eyes' due to oedema in the eyes.
- Locomotion: Reluctance to move, due to inflammation of the coronary band. Chronic cases may display lameness and torticollis when there is skeletal muscle degeneration.
- Depression, recumbency and death are common in severe cases. Some sheep exhibit sudden death due to cardiac necrosis, even during apparent recovery.

In cattle, goats and African antelopes, infection is usually subclinical, acting as important amplifying reservoirs. However, when infected with a more virulent strain or in high doses, these species can manifest clinical signs similar to the ones of mild or severe disease in sheep.

Outcomes of a SLR of clinical signs in 229 study groups of ruminants and three study groups of camelids are displayed in Figure 10. Predominantly, general, respiratory and cardiovascular clinical signs were reported.

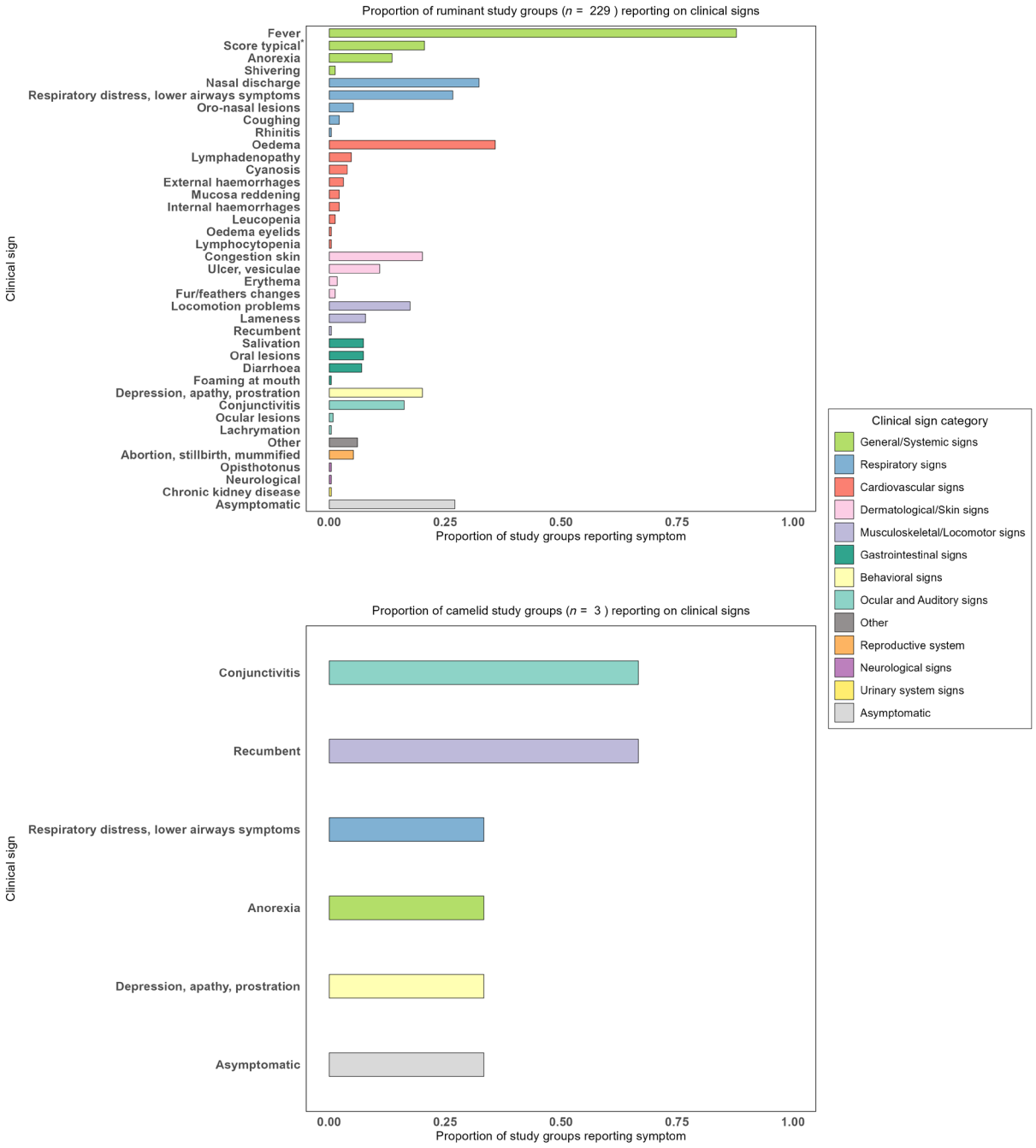


FIGURE 10 Clinical signs reported in the main hosts of BTV. *Score typical: blue tongue. Study group count per ruminant species: Sheep $n = 143$; Cattle $n = 44$, Goat $n = 19$; Muntjac deer $n = 6$; White-tailed deer $n = 5$; Black-tailed deer $n = 4$; Wapiti elk $n = 4$; Red deer $n = 3$; Buffalo $n = 1$. Camelid study groups: Alpaca $n = 1$; Camel $n = 1$; Llama $n = 1$. The SLR was updated until 31 December 2025; for references, see Annex A.

Incubation period

The incubation period of BTV can vary from 4 to 20 days, with the average duration lasting 4–7 days (Spickler, 2023a, 2023b; WOAH, 2021a). The SLR identified studies in alpaca, black-tailed deer, cattle, goat, llama, muntjac deer, sheep, wapiti elk and white-tailed deer, confirming this incubation range in all species (Annex A).

Morbidity and case fatality

Clinical morbidity varies a lot by host species, virus strain, breed and immunity. Exact rates are difficult to estimate because many studies report seroprevalences, and as the virus causes mainly a subclinical infection, the seroprevalence can be much higher than clinical morbidity (Spickler, 2023a, 2023b; USDA, 2016; WOAH, 2021a).

- In sheep, reported morbidity ranges from < 5% up to 50%–75% (or higher) in affected flocks. Mortality by virulent strains in naïve flocks can be very high (reports of up to ~70% or even higher). Young lambs are particularly vulnerable, often exhibiting more severe clinical signs and experiencing elevated case fatality rates of up to 30%.
- In cattle, morbidity is often low, as most infections are subclinical.

- In goats and camelids, morbidity is also usually lower than in sheep and clinical disease is often milder. Some species of wild ruminants can be highly susceptible.
- In wild deer and antelope species, case fatality rates can be as high as 90%.

Zoonotic potential

BTV is not known to infect humans under natural conditions.

3.4.5 | Transmission

BTV is primarily transmitted by biting midges of the genus *Culicoides* (Diptera; Ceratopogonidae). Wind can facilitate long-distance dispersal of infected vectors, and adult *Culicoides* can survive mild winters, contributing to virus persistence (Hendrickx et al., 2008; Wilson et al., 2008). Evidence on the potential and likely competent *Culicoides* species and their geographic distribution can be found in a dedicated Scientific Report on vectors (EFSA, 2026a).

Certain BTV serotypes appear to be transmitted to susceptible vertebrate hosts vertically, horizontally and through indirect contact (e.g. reused needles), without the interference of biological vectors (Bréard et al., 2018; Clarke et al., 2019). However, the impact these routes have on the epidemiology of this disease remains unclear (Spickler, 2023a, 2023b).

3.4.6 | Diagnostic tests

Recommended tests (WOAH, 2025b) for agent detection are the reverse-transcription polymerase chain reaction (RT-PCR) assay, the real-time RT-PCR and classical virus isolation. For immune response detection, the recommended tests are C-ELISA (serogroup specific), virus neutralisation (serogroup specific) and agar gel immunodiffusion (AGID).

Table 8 presents data on the sensitivity and specificity of diagnostic tests collected through SLR; reported values correspond to the median sensitivity and specificity when multiple studies investigated the same test and are only included when explicitly stated in the publications.

TABLE 8 Median sensitivity and specificity of tests to detect BTV/BTV antibodies reported in literature included in the systematic literature review.

Target	Test	Species	Sensitivity	*N* animal groups	Specificity	*N* animal groups	References
Nucleic acid	Real-time RT-PCR	Cattle	99.5%	1	98.5%	1	Vandenbussche et al. (2008)
Nucleic acid	Real-time RT-PCR	Sheep	99.6%	1	98.5%	1	
Antibody	AGID	Cattle	–	3	99.0%	3	Afshar et al. (1989)
Antibody	AGID	Goat	–	1	99.4%	1	
Antibody	AGID	Sheep	–	1	99.4%	1	
Antibody	C-ELISA	Bison	–	1	92.9%	1	Afshar et al. (1995)
Antibody	C-ELISA	Cattle	96.8%	15	99.3%	15	Niedbalski (2011); Niedbalski (2010); Vandenbussche et al. (2008); Cagienard et al. (2006); Afshar et al. (1992); Afshar et al. (1989)
Antibody	C-ELISA	Deer	–	1	99.8%	1	Afshar et al. (1995)
Antibody	C-ELISA	Goat	92.8%	3	96.0%	3	Bulut et al. (2006); Prabhakar et al. (2005); Afshar et al. (1989)
Antibody	C-ELISA	Llama	–	1	100%	1	Afshar et al. (1995)
Antibody	C-ELISA	Reindeer	–	1	99.6%	1	
Antibody	C-ELISA	Sheep	96.9%	14	99.4%	14	Vandenbussche et al. (2008); Bhanuprakash et al. (2007); Biteau-Coroller et al. (2006); Bulut et al. (2006); Shringi and Shringi (2005); Prabhakar et al. (2005); Lelli et al. (2003); Afshar et al. (1992); Afshar et al. (1989); Jyothi et al. (2022)
Antibody	I-ELISA	Sheep	89.7%	3	90.4%	3	Bhanuprakash et al. (2007); Jyothi et al. (2022); Biswas et al. (2025)
Antibody	I-ELISA	Goat	98.7%	1	69.9%	1	Biswas et al. (2025)
Antibody	SN test	Sheep	100%	1	100%	1	Lelli et al. (2003)

Note: The SLR was updated until 31/12/2025, for references see Annex A.

3.4.7 | Prevention and control

Vaccination

Both live-attenuated and inactivated vaccines against BTV are approved for use in certain countries. Vaccination is the most effective control strategy in endemic regions according to WOAH. However, immunisation is virus serotype-specific and does not confer cross-protection, complicating control efforts (WOAH, 2025b).

In the EU, inactivated vaccines are approved for serotypes 1, 3, 4 and 8 (EMA, 2025).

Treatment

There is currently no specific antiviral treatment for BTV infection. Management is primarily supportive and should be taken on early, by providing rest, soft and palatable food and clean water and implementing good husbandry practices. Secondary bacterial infections during the recovery period, such as Pasteurellosis, should be treated appropriately with antibiotic therapy.

Although treatment is symptomatic, vaccination, particularly of sheep in endemic areas, remains the most effective and practical control strategy against bluetongue disease. Evidence on the efficacy of pharmacological and insecticide treatment strategies can be found in a dedicated Scientific Report on mitigation measures (EFSA, 2026b).

3.5 | *Borrelia burgdorferi* s.l. (*B. burgdorferi*)[7]

3.5.1 | Disease overview

Borrelia burgdorferi s.l. causes an infectious, non-contagious, tick-borne bacterial disease known as Lyme disease. Animals can be a reservoir or have clinical manifestations of the disease (Spickler, 2020; WOAH, 2020b, 2025b).

Lyme disease is *not* a WOAH-notifiable disease, and it is not listed in the European AHL.

3.5.2 | Agent

Borrelia spp. are pathogenic bacterial spirochetes. Although they possess an outer membrane and are generally classified as Gram-negative, they are poorly visualised by standard Gram staining due to atypical cell envelope architecture. Their diderm structure comprises inner and outer membranes, but unlike typical Gram-negative bacteria, the outer membrane lacks lipopolysaccharide (LPS) and is instead enriched with diverse outer surface lipoproteins (Osps) that mediate host–pathogen interactions. Motility is driven by periplasmic flagella located between the membranes, enabling a characteristic corkscrew-like motion. A peptidoglycan layer, providing structural support, is situated in the periplasm adjacent to the inner membrane.

The classification of *Borrelia* species associated with Lyme borreliosis is broadly categorised into the following (Skar et al., 2024; Tatum & Pearson-Shaver, 2023):

- *B. burgdorferi* sensu lato: This complex encompasses a genetically diverse group of closely related *Borrelia* species, often referred to as genospecies, that are responsible for Lyme borreliosis globally. Key members of this complex include, but are not limited to, *B. burgdorferi* sensu stricto, *B. afzelii, B. garinii, B. bavariensis* and *B. spielmanii*. These distinct genospecies exhibit varying geographical distributions and clinical manifestations of disease across North America, Europe and Asia.
- *B. burgdorferi* sensu stricto: It is the predominant causative agent of Lyme borreliosis in North America.

3.5.3 | Geographical distribution

B. burgdorferi infection (Lyme disease) 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 SLR (updated until 31/12/2025) and summarised in Figure 11. All references can be found in Annex A and in the online version of the Disease Profile.[7]

[7]Online version: https://animal-diseases.efsa.europa.eu/Bburgdorferi.

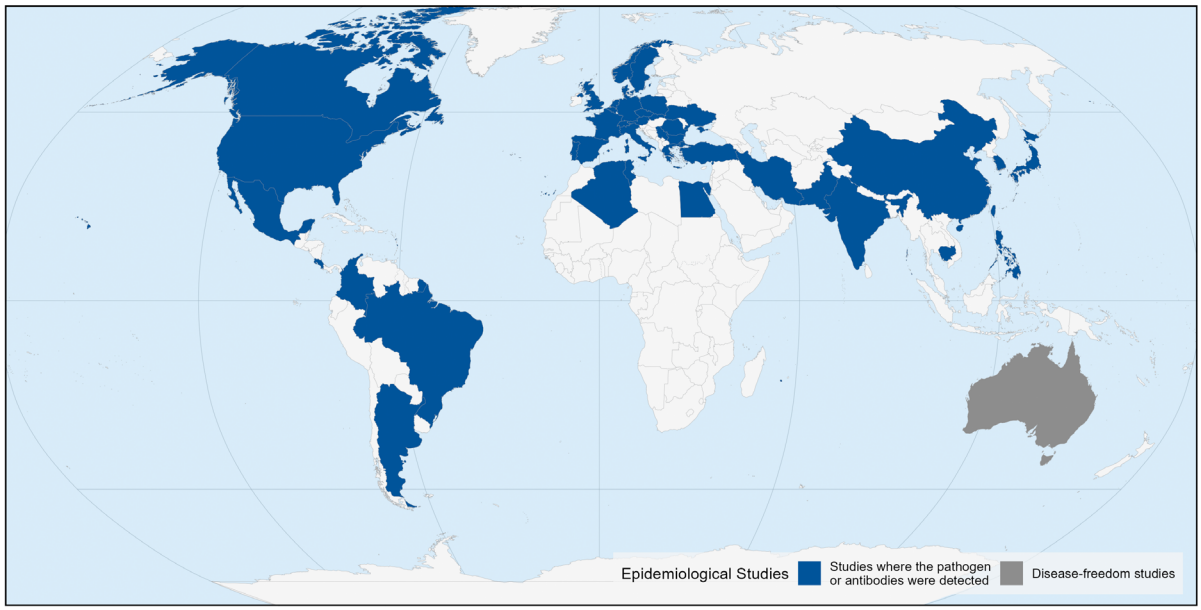


FIGURE 11 Geographical distribution of epidemiological studies addressing the occurrence of *B. burgdorferi*, as identified by EFSA's SLR (covering years 1970–2025).

3.5.4 | Animal hosts

Susceptible hosts

Based on epidemiological knowledge of host–pathogen–vector interactions and outbreak reports, *B. burgdorferi* has a wide range of vertebrate hosts with the most important being rodents and birds, whereas dogs, horses and humans are considered as dead-end hosts. The SLR on susceptible species summary is given in Table 9.

TABLE 9 Susceptible host species of *Borrelia burgdorferi* s.l.

The SLR reported in the *B. burgdorferi* disease profiles, identified the following susceptible species (updated until 31 December 2025; for references, see Annex A)
FIELD
Epidemiological studies carried out in the field

• **Pathogen was detected in the following animal species:**
  • Bovidae: *Bos taurus, Bison bison, Bison bonasus, Ovis aries, Capra hircus*
  • Camelidae: *Camelus bactrianus*
  • Canidae: *Canis lupus familiaris, Vulpes* spp.
  • Cervidae: *Odocoileus hemionus columbianus*
  • Chiroptera (Order): No families and species specified
  • Columbidae: No species specified
  • Cricetidae: *Myodes glareolus, Microtus agrestis, Microtus subterraneus*
  • Equidae: *Equus caballus*
  • Felidae: *Felis catus*
  • Fringillidae: *Fringilla coelebs, Coccothraustes coccothraustes*
  • Hominidae: *Homo sapiens*
  • Motacillidae: *Anthus trivialis*
  • Muridae: *Apodemus sylvaticus*
  • Muscicapidae: *Erithacus rubecula*
  • Mustelidae: *Meles meles, Mustela lutreola, Mustela putorius, Martes martes, Martes foina*
  • Prunellidae: *Prunella modularis*
  • Rodentia (Order): No families and species specified
  • Sciuridae: *Sciurus* spp.
  • Sittidae: *Sitta europaea*
  • Soricidae: *Sorex coronatus*
  • Troglodytidae: *Troglodytes troglodytes*
  • Turdidae: *Turdus merula, Turdus philomelos, Erithacus rubecula, Troglodytes troglodytes*

(Continues)

TABLE 9 (Continued)

The SLR reported in the *B. burgdorferi* disease profiles, identified the following susceptible species (updated until 31 December 2025; for references, see Annex A)

- **Antibodies were detected in the following animal species:**
  - Accipitridae: No species specified
  - Bovidae: *Bos taurus, Bubalus* spp., *Capricornis crispus, Ovis aries*
  - Camelidae: *Camelus bactrianus*
  - Canidae: *Canis lupus familiaris*
  - Cervidae: *Odocoileus virginianus*
  - Didelphidae: No species specified
  - Equidae: *Equus caballus*
  - Falconidae: No species specified
  - Felidae: *Felis catus*
  - Hominidae: *Homo sapiens*
  - Leporidae: No species specified
  - Muridae: *Mus musculus*
  - Mustelidae: No species specified
  - Phasianidae: No species specified
  - Sciuridae: No species specified
  - Strigidae: No species specified
  - Tapiridae: *Tapirus terrestris*
- **Outbreaks reported to WOAH included the following species:**
  - No species specified

EXPERIMENTS

- **Experimental studies demonstrated infection in:**
  - Canidae: *Canis lupus familiaris*

Clinical signs

Clinical disease is mainly important for humans. However, there are reports of Lyme disease manifestation in non-human primates, wildlife animals, dogs and cats.

Reports of clinical disease are infrequent, including synovitis, myositis, myocarditis in dusky-footed woodrats (*Neotoma fuscipes*); also, pneumonitis, encephalitis, perivascular lymphoplasmacytic infiltrates of kidneys and liver in white-footed mice (*Peromyscus leucopus*). Clinical signs of Borreliosis in domestic animals are often subtle and non-specific, with many infected animals remaining asymptomatic.

In dogs, common signs include shifting-leg lameness due to polyarthritis, fever, lethargy and enlarged lymph nodes. Severe but rare complications like Lyme nephritis can occur.

Horses may show lameness, stiffness and joint effusion. Less common but reported are neuroborreliosis (ataxia, behavioural changes), uveitis and skin nodules.

Cattle can exhibit fever, anorexia, decreased milk production and chronic weight loss. Stiffness and swollen joints have also been noted.

Cats rarely show clinical signs; when they do, they are typically mild, such as lameness, fever or lethargy.

Incubation period

In animals, the incubation period is not well defined, but clinical signs may appear several months after the infection.

Morbidity and case fatality

Although it is not trivial to accurately calculate a case fatality rate due to the long incubation period and lack of specific clinical signs, Lyme disease is rarely fatal (Spickler, 2020).

Zoonotic potential

Lyme disease is a zoonotic disease (CDC, 2024c).

3.5.5 | Transmission

Borreliosis is transmitted to vertebrate hosts through the bite of ticks. Evidence on the potential and likely competent tick species and their geographic distribution can be found in a dedicated Scientific Report on vectors (EFSA, 2026a). Ticks attach to an animal host reservoir, typically birds or other small mammals such as mice and voles, and feed for 3 to

7 days. Infected nymphs and adult ticks spread the bacteria each time they subsequently feed. The spread regularly increases during warmer and humid weather, typically in spring (Radolf et al., 2012; Rizzoli et al., 2011; Stanek et al., 2012).

3.5.6 | Diagnostic tests

There are no WOAH-recommended tests for this disease.

Table 10 presents data on the sensitivity and specificity of diagnostic tests collected through SLR; reported values correspond to the median sensitivity and specificity when multiple studies investigated the same test and are only included when explicitly stated in the publications.

TABLE 10 Median sensitivity and specificity of tests to detect *B. burgdorferi*/*B. burgdorferi* antibodies reported in literature included in the systematic literature review.

Target	Test	Species	Sensitivity	*N* animal groups	Specificity	*N* animal groups	References
Antibody	ELISA	Dog	96.7%	5	95.7%	5	Goldstein et al. (2014); Gerber et al. (2009); Barth et al. (2014); Chandrashekar et al. (2010); Sheets et al. (2000)
Antibody	Immuno-fluorescence Assay	Dog	51.5%	2	84.1%	2	Barth et al. (2014)
Antibody	Western blot	Dog	100%	1	100%	1	Sheets et al. (2000)

3.5.7 | Prevention and control

Vaccination

Two vaccines are used in dogs to prevent Lyme disease: whole cell lysate and recombinant outer surface protein A (OspA). Commercial versions are licensed in the EU.

In wildlife, experimental vaccination trials against *B. burgdorferi* have been conducted; the majority of individuals produced antibodies against the bacterium (Spickler, 2020; WOAH, 2020b).

Treatment

Pharmaceutical treatments applied to dogs for which the efficacy was tested include doxycycline, ceftriaxone and azithromycin. Additionally, insecticides applied to hosts (dogs) with tested efficacy include Imidacloprid + Flumethrin (Spickler, 2020; WOAH, 2020b; Annex A).

3.6 | Bovine ephemeral fever virus (BEFV)[8]

3.6.1 | Disease overview

Bovine ephemeral fever virus (BEFV) causes bovine ephemeral fever (BEF) or '3-day sickness', an acute, arthropod-borne viral disease of cattle and water buffalo. The disease is characterised by sudden onset of fever, lameness, stiffness and marked reduction in milk production, with high morbidity and generally low mortality (Spickler, 2016; Walker & Klement, 2015).

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

3.6.2 | Agent

BEFV is an enveloped, single-stranded, negative-sense RNA virus classified within the genus *Ephemerovirus*, family Rhabdoviridae. The virion is bullet-shaped, approximately 180 × 70–80 nm in size. The genome consists of a non-segmented RNA molecule encoding five structural proteins: the nucleoprotein (N), phosphoprotein (P), matrix protein (M), glycoprotein (G) and the RNA-dependent RNA polymerase (L). In addition, BEFV contains several accessory genes (α1, α2, β and γ) located between the G and L genes, which are characteristic of ephemeroviruses. The G glycoprotein is responsible for virus attachment and membrane fusion and induces neutralising antibodies, playing a central role in protective immunity (Kuzmin et al., 2009; Walker & Klement, 2015).

[8]Online version: https://animal-diseases.efsa.europa.eu/BEFV.

3.6.3 | Geographical distribution

BEFV is broadly distributed across Africa, the Middle East, Asia and Australia (Walker & Klement, 2015). Infection with BEFV 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 SLR (updated until 31 December 2025) and summarised in Figure 12. All references can be found in Annex A and in the online version of the Disease Profile.[8]

FIGURE 12 Geographical distribution of epidemiological studies addressing the occurrence of BEFV, as identified by the EFSA's SLR (covering years 1970–2025).

3.6.4 | Animal hosts

Based on epidemiological knowledge of host–pathogen–vector interactions and outbreak reports, the main hosts of BEFV are cattle. However, other species have been identified in the SLR. The SLR on susceptible species summary is given in Table 11.

TABLE 11 Susceptible hosts of bovine ephemeral fever virus.

The SLR reported in the BEFV disease profile, identified the following susceptible species (updated until 31 December 2025; for references, see Annex A)
FIELD
Epidemiological studies carried out in the field
• **Pathogen was detected in the following animal species:** • Bovidae: *Bos taurus*, *Bubalus* sp.
• **Antibodies were detected in the following animal species:** • Bovidae: *Bos taurus*, *Bison bison*, *Bos grunniens*, *Gazella gazella* • Cervidae: *Dama dama*, *Hydropotes inermis argyropus*, *Cervus elaphus*, *Capreolus pygargus*, *Cervus nippon*
• **Outbreaks reported to WOAH included the following species:** • No species specified
EXPERIMENTS
• **Experimental studies demonstrated infection in:** • Bovidae: *Bos taurus*

Clinical signs

Outcomes of a SLR of clinical signs in seven study groups of cattle are displayed in Figure 13. Predominantly, general and respiratory clinical signs were reported.

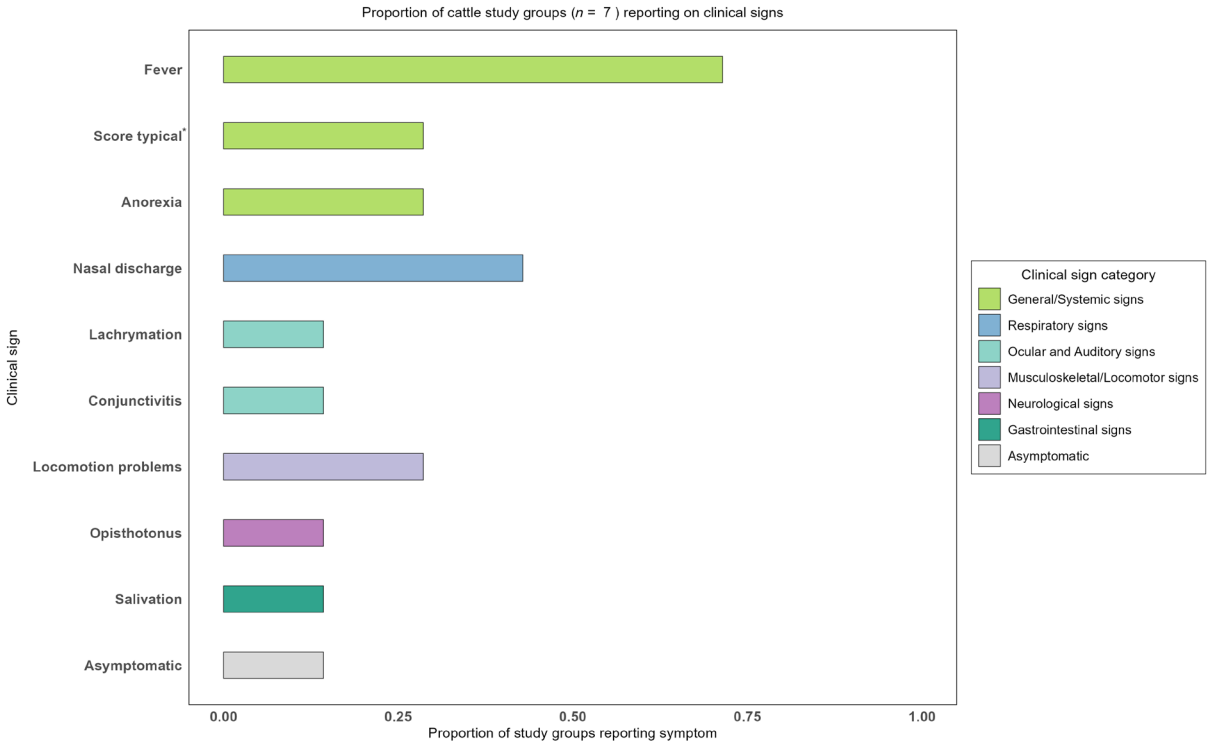


FIGURE 13 Clinical signs reported in the main hosts of BEFV. *Score typical: '3-day sickness' characterised by high fever, shivering, lameness, drop in productivity. The SLR was updated until 31 December 2025; for references, see Annex A.

The disease in cattle is characterised by sudden onset of fever, nasal discharge, locomotion problems, lameness, stiffness, recumbency and a reduction in milk production (Annex A; Aziz-Boaron et al., 2014; Walker & Klement, 2015).

Incubation period

Based on experimental studies, an incubation period ranging from 3 to 5 days in cattle has been observed (Annex A).

Morbidity and case fatality

During outbreaks of BEFV high prevalence and morbidities ranging from 20% to 80% have been reported with generally low case fatalities (Grobler et al., 2025; Walker & Klement, 2015). No fatalities due to infection were observed in experimentally infected cattle (Annex A).

Zoonotic potential

BEFV is not known to infect humans under natural conditions.

3.6.5 | Transmission

BEFV is transmitted primarily by biting midges of the genus *Culicoides* (Diptera; Ceratopogonidae). Evidence on the potential and likely competent *Culicoides* species and their geographic distribution can be found in a dedicated Scientific Report on vectors (EFSA, 2026a).

The disease occurs seasonally (mostly from later spring to autumn), often associated with increased vector activity and climatic conditions favourable to vector abundance. Experimental evidence indicates the absence of direct contact transmission with infected animals or fomites and the absence of transmission by mechanical arthropod vectors (reviewed by Walker & Klement, 2015).

3.6.6 | Diagnostic tests

Diagnosis of BEF relies mostly on the detection of the virus (virus isolation), virus RNA or host antibody responses. Real-time RT-PCR assays are used for the specific detection of BEFV RNA in blood or tissues, offering high analytical sensitivity and specificity in clinical and field samples and appear to be more sensitive than the conventional RT-PCR (Golender et al., 2025; Solanki et al., 2025). An evaluation of different diagnostic methods for detection of BEFV in naturally infected cattle found

that conventional RT-PCRs (targeting the L or G-genes) had moderate sensitivity (≤ 64%) and high specificity (> 89%); virus isolation has high specificity (99%) but poor sensitivity (30%) (Grobler et al., 2025).

For serological diagnosis, virus neutralisation tests (VNT) and various ELISA formats are used to detect specific antibodies against BEFV, including competitive ELISAs based on inactivated virus or recombinant nucleoprotein antigens. These ELISA tests showed, under laboratory conditions, a high sensitivity (97%–99%) and specificity (100%), compared with VNT (Benevenia et al., 2024).

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

3.6.7 | Prevention and control

Vaccination

There is no commercial vaccine authorised in Europe.

Outside Europe, there are three types of vaccines being used in the field: live-attenuated vaccines, inactivated vaccines and sub-unit vaccines (Walker & Klement, 2015). Live-attenuated vaccines have been shown to provide moderate to high protection (vaccine effectiveness) against clinical signs, with VE reported ranging from 60% to 90% (Gleser et al., 2023; Vanselow et al., 1995). Inactivated vaccines are safer but require multiple doses, with VE estimates ranging from 40% to 50% (Aziz-Boaron et al., 2014).

Treatment

There is currently no specific antiviral treatment for BEFV infection. Flunixin meglumine and phenylbutazone have been studied and used for BEF, but only as supportive, symptomatic treatments in cattle (Uren et al., 1989).

3.7 | Cache Valley virus (CVV)[9]

3.7.1 | Disease overview

Cache Valley virus (CVV) causes CVV infection, a mosquito-borne viral disease of domestic and wild ungulates, primarily sheep, goats, cattle and white-tailed deer. The disease is characterised by reproductive losses resulting from transplacental infection during early gestation, leading to abortions, stillbirths and congenital malformations in newborns, while adult animals typically remain asymptomatic (Boston University, 2025; Hughes et al., 2023; Waddell et al., 2019; WOAH, 2025b).

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

3.7.2 | Agent

CVV 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 neutralising antibodies (Hughes et al., 2023).

The virus is moderately stable under ambient conditions but is inactivated by lipid solvents, detergents and standard disinfectants (Boston University, 2025).

3.7.3 | Geographical distribution

Since its first isolation in 1956 in Cache Valley, Utah (USA), CVV has been detected throughout much of the United States and Canada, and in parts of Mexico, the Caribbean, South America, Africa and Australia.

CVV 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 SLRs (updated until 31 December 2025) and summarised in Figure 14. All references can be found in Annex A and in the online version.[9]

[9]Online version: https://animal-diseases.efsa.europa.eu/CVV.

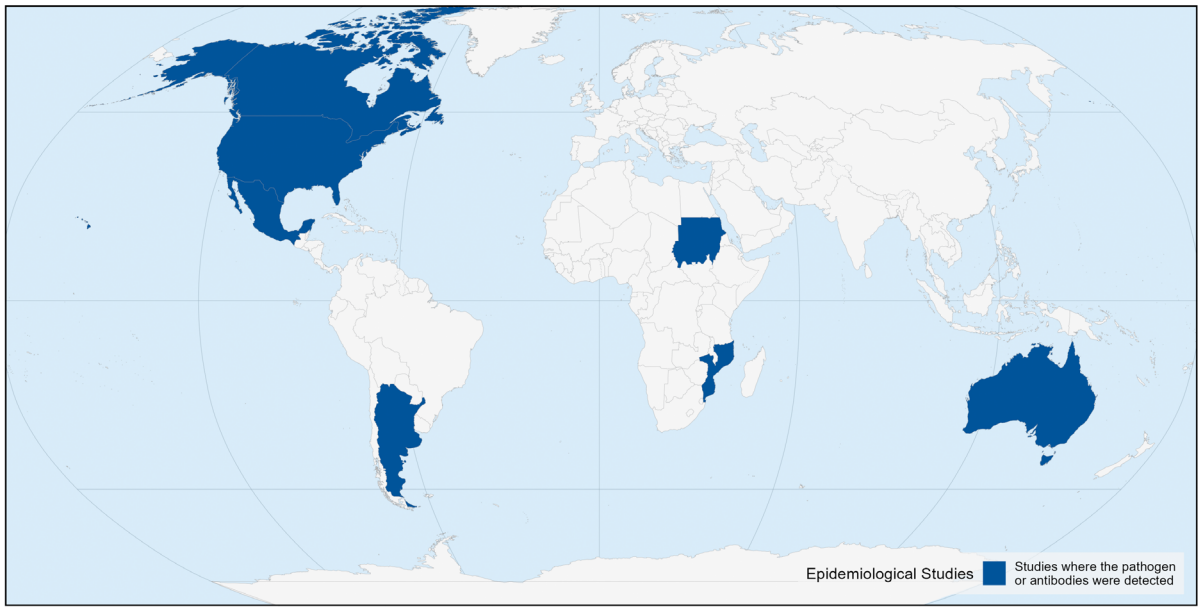


FIGURE 14 Geographical distribution of epidemiological studies addressing the occurrence of CVV, as identified by EFSA's systematic literature review (covering years 1970–2025).

3.7.4 | Animal hosts

Based on epidemiological knowledge of host–pathogen–vector interactions and outbreak reports, the main hosts of CVV are deer, whereas domestic ruminants, horses and humans are considered as dead-end hosts. However, other susceptible species have been identified in the SLR. The SLR summary is given in Table 12.

TABLE 12 Susceptible host species of Cache Valley virus.

The SLR reported in the CVV disease profile, identified the following susceptible species (updated until 31 December 2025, for references, see Annex A)
FIELD
Epidemiological studies carried out in the field

• **Pathogen was detected in the following animal species:**
  • Hominidae: *Homo sapiens*
• **Antibodies were detected in the following animal species:**
  • Bovidae: *Ovis aries*
  • Cervidae: *Odocoileus virginianus*
  • Hominidae: *Homo sapiens*
  • Leporidae: *Sylvilagus floridanus*
• **Outbreaks reported to WOAH included the following species:**
  • No species specified

EXPERIMENTS

• **Experimental studies demonstrated infection in:**
  • Bovidae: *Bos taurus, Capra hircus*
  • Cervidae: *Odocoileus virginianus*
  • Leporidae: *Oryctolagus cuniculus*
  • Suidae: *Sus scrofa domesticus*

Clinical signs

Clinical disease associated with CVV occurs almost exclusively in ruminant fetuses infected *in utero*, as adult animals typically remain asymptomatic. The severity and nature of fetal outcomes depend strongly on the stage of gestation at the time of maternal infection.

In sheep, infection of the ewe between approximately days 28 and 48 of gestation may result in abortions, stillbirths, mummified fetuses or a spectrum of congenital malformations. Predominant lesions include arthrogryposis and hydranencephaly, along with other central nervous system and musculoskeletal abnormalities such as hydrocephalus, micromyelia, limb deformities and severe muscle loss. Affected lambs may be born weak, unable to stand or die shortly after birth.

Although infection can occur in other ruminants, including cattle and goats, clinical disease linked to CVV has been documented primarily in sheep (Waddell et al., 2019).

Incubation period

Since adult animals infected with CVV rarely show overt clinical signs, the incubation period in adults is not clearly defined. However, after exposure, vertebrate hosts (especially ungulates) may develop transient viraemia sufficient to infect feeding mosquitoes. The exact timing of viraemia onset and duration remains poorly characterised, owing to limited longitudinal data in naturally infected animals (Hughes et al., 2023).

Morbidity and case fatality

In adult ungulates and other vertebrates, morbidity is generally negligible; most seropositive animals are asymptomatic. Mild or transient illness in adults appears to be uncommon and is not well documented (Hughes et al., 2023). Fatal outcomes in adults are rare or undocumented under natural conditions (Waddell et al., 2019).

In contrast, the primary impact of CVV is on the fetus in infected pregnant animals. In small ruminants, especially sheep, infection during the susceptible gestational window has been repeatedly associated with high rates of foetal loss, stillbirths or offspring born with severe congenital malformations. Because many malformed neonates are stillborn, die shortly after birth or are non-viable, the case fatality rate among these affected offspring can be very high (Waddell et al., 2019).

Zoonotic potential

Cache Valley virus infection is a zoonotic disease (WOAH, 2025a).

3.7.5 | Transmission

CVV infection is primarily transmitted by mosquitoes (Diptera; Culicidae). Evidence on the potential and likely competent vector species and their geographic distribution can be found in a dedicated Scientific Report on vectors (EFSA, 2026a).

Seasonal patterns of CVV transmission and seroprevalence appear to correspond to periods of higher mosquito activity, though the data do not yet define a strictly regular 'season' for outbreaks (Hughes et al., 2023).

For clinical disease to occur in offspring, transplacental transmission is key: infection of a pregnant dam results in virus crossing to the fetus producing the typical congenital malformations, stillbirths or fetal death associated with CVV. There is no published evidence that non-pregnant animals pass the virus venereally, via vertical transmission other than transplacental infection, or by other non-vector routes under natural conditions (Hughes et al., 2023; Waddell et al., 2019).

3.7.6 | Diagnostic tests

WOAH-recommended tests (WOAH, 2025b) for agent detection include virus isolation, immunofluorescence assay (IFA), immunohistochemistry (IHC), virus neutralisation (VNT) test and RT-PCR.

CVV can be isolated from the blood of febrile or viraemic adult animals, although the window for successful detection is short. Isolation from newborn fetuses is usually unsuccessful, because viral clearance typically occurs before birth as the fetal immune response develops. Detection of viral antigen or infectious virus can be achieved using IFA, IHC or VNT tests. In addition, both group-specific and virus-specific RT-PCR assays are available and provide sensitive tools for identifying *Orthobunyaviruses*, including CVV (WOAH, 2025b).

For immune response detection, the recommended tests are ELISA and VNT. The latter remains the reference method for confirming exposure and measuring functional neutralising antibodies. ELISA formats are also used for large-scale screening and surveillance. Because adult infections are usually subclinical, serology is the primary tool for identifying past infection at the herd level. Detection of antibodies in pre-colostrum fetal fluids or tissues from malformed or stillborn neonates provides strong evidence of utero infection (WOAH, 2025b).

To date, the SLR has not found studies evaluating diagnostic tests meeting the eligibility criteria for inclusion.

3.7.7 | Prevention and control

Vaccination

Experimental live-attenuated and inactivated vaccines against CVV have been developed, but none is yet commercially licensed for use in livestock (Hughes et al., 2023). A recent study compared a candidate live-attenuated CVV vaccine, which elicited a stronger and more durable neutralising response than the inactivated vaccine (Ayers et al., 2023).

Treatment

There is currently no specific antiviral treatment for CVV infection. As adult ruminants typically develop subclinical or mild transient infections, clinical management is rarely required. In cases involving congenital infection, affected neonates often present with severe neurological or musculoskeletal malformations that are incompatible with life. Supportive care offers no meaningful benefit in these cases, and euthanasia is generally recommended on welfare grounds.

3.8 | *Coxiella burnetii* (*C. burnetii*)[10]

3.8.1 | Disease overview

The bacterium *Coxiella burnetii* can infect mammals, birds, reptiles and arthropods. It causes Q-fever, an acute zoonotic febrile infectious disease that mainly affects ruminants and can cause abortions and stillbirths in cattle, sheep and goats. The bacterium is primarily transmitted through the inhalation of aerosols containing contaminated material, but in a lesser extent, the pathogen may be transmitted by ticks (Spickler, 2007).

Q-fever is a WOAH-notifiable disease, listed in the European AHL under category E.

3.8.2 | Agent

Coxiella burnetii (Coxiellaceae) is a Gram-negative, pleomorphic, obligate intracellular coccobacillus, phylogenetically related to *Legionella* spp. A key feature of its cell envelope is its lipopolysaccharide (LPS), which undergoes phase variation (Maurin & Raoult, 1999; Seshadri et al., 2003). The virulent Phase I form expresses a complete, smooth LPS typical of isolates obtained directly from infected hosts (Heinzen et al., 1999). In contrast, repeated passages in cell culture or embryonated eggs induce Phase II, characterised by a truncated, rough LPS structure and markedly reduced virulence (Heinzen et al., 1999). This LPS variation is central to the bacterium's antigenic properties, immune evasion and pathogenicity (van Schaik et al., 2013).

The pathogen *C. burnetii* undergoes a dimorphic developmental cycle, alternating between two morphological forms: the small cell variant (SCV) and the large cell variant (LCV) (Coleman et al., 2004; Omsland et al., 2009). The SCV is a metabolically quiescent, environmentally resistant form, exhibiting spore-like properties, including high resistance to desiccation, heat and chemical stress, and represents the infectious form of the bacterium (Coleman et al., 2004; Omsland et al., 2009). Upon entry into a host cell, the SCV differentiates into the LCV, which is larger, metabolically active and capable of replication (Omsland et al., 2009). This ability to persist as a highly resistant SCV contributes significantly to the organism's exceptional environmental stability and extremely low infectious dose (Maurin & Raoult, 1999; van Schaik et al., 2013).

3.8.3 | Geographical distribution

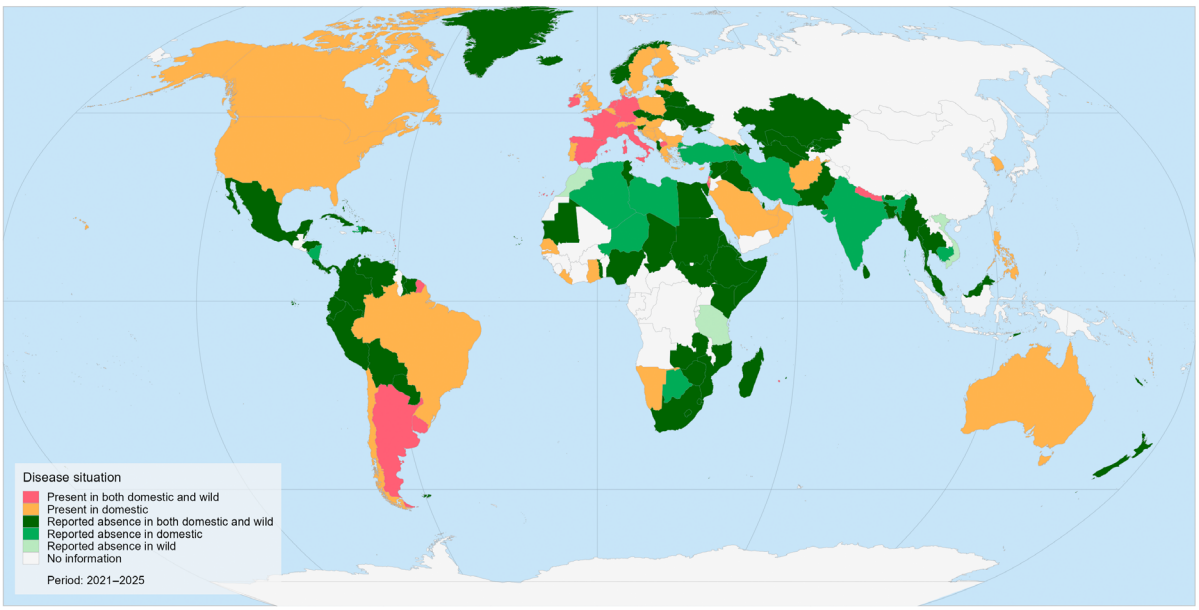


FIGURE 15 Geographical distribution of *Coxiella burnetii* detected events (2021–2025), as reported to WOAH.

[10]Online version: https://animal-diseases.efsa.europa.eu/Cburnetii.

C. burnetii has been detected across nearly all continents and is currently endemic in Europe. Figure 15 shows those countries where the pathogen has been reported in the last 5 years to WAHIS. Up to date maps based on WAHIS data are available in the online version.

3.8.4 | Animal hosts

Susceptible hosts

Based on epidemiological knowledge of host–pathogen–vector interactions and outbreak reports, *C. burnetii* has a wide range of vertebrate hosts, with the most important being domestic ruminants, whereas humans are considered as dead-end hosts. The summary of the SLR on susceptible hosts is given in Table 13.

TABLE 13 Susceptible host species of *Coxiella burnetii*.

The SLR reported in the Q-fever disease profile, identified the following susceptible species (updated until 31 December 2025; for references, see Annex A)

FIELD

Epidemiological studies carried out in the field

- **Pathogen was detected in the following animal species:**
- Bovidae: *Bos taurus, Capra hircus, Ovis aries, Bubalus* spp., *Gazella dorcas, Nanger dama, Gazella cuvieri*
- Cervidae: *Cervus elaphus*
- Equidae: *Equus caballus*
- Camelidae: *Camelus bactrianus, Camelus dromedarius*
- Canidae: *Canis lupus familiaris*
- Felidae: *Felis catus*
- **Antibodies were detected in the following animal species:**
- Bovidae: *Bos taurus*, *Bos indicus*, *Bos grunniens*, *Capra hircus*, *Ovis aries*, *Ovis orientalis, Saiga tatarica, Gazella Dorcas, Nanger dama, Gazella cuvieri*
- Suidae: *Sus scrofa domesticus, Sus scrofa*
- Equidae: *Equus caballus*
- Hominidae: *Homo sapiens*
- Cervidae: *Cervus elaphus, Dama dama, Capreolus capreolus*
- Camelidae: *Camelus bactrianus, Camelus dromedarius*
- Canidae: *Canis lupus familiaris*
- Muridae: *Mus musculus, Rattus rattus*
- Mustelidae: *Enhydra lutris*
- Otariidae: *Eumetopias jubatus, Callorhinus ursinus*
- Phasianidae: no species specified
- **Outbreaks reported to WOAH included the following species:**
- Bovidae: *Bos Taurus, Capra hircus, Ovis aries*

EXPERIMENTS

- **Experimental studies demonstrated infection in:**
- Bovidae: *Capra hircus, Ovis aries*

Clinical signs

Q-fever is typically a mild or subclinical disease in animals, primarily affecting cattle, sheep and goats (Maurin & Raoult, 1999; Spickler, 2007; WOAH, 2022). Infections are most often asymptomatic, but when clinical signs occur, they are predominantly associated with reproductive dysfunction in ruminants, especially late-term abortions (Agerholm, 2013; Spickler, 2007; WOAH, 2022). The most serious consequences include reproductive failure, notably late-term abortion, stillbirths, fetal mummification and fever (Maurin & Raoult, 1999; van den Brom & Vellema, 2009).

Outcomes of a SLR on clinical signs in 12 study groups of sheep and goats (no studies on cattle had passed the eligibility criteria) are displayed in Figure 16. Predominantly, clinical signs affecting the reproductive system were reported.

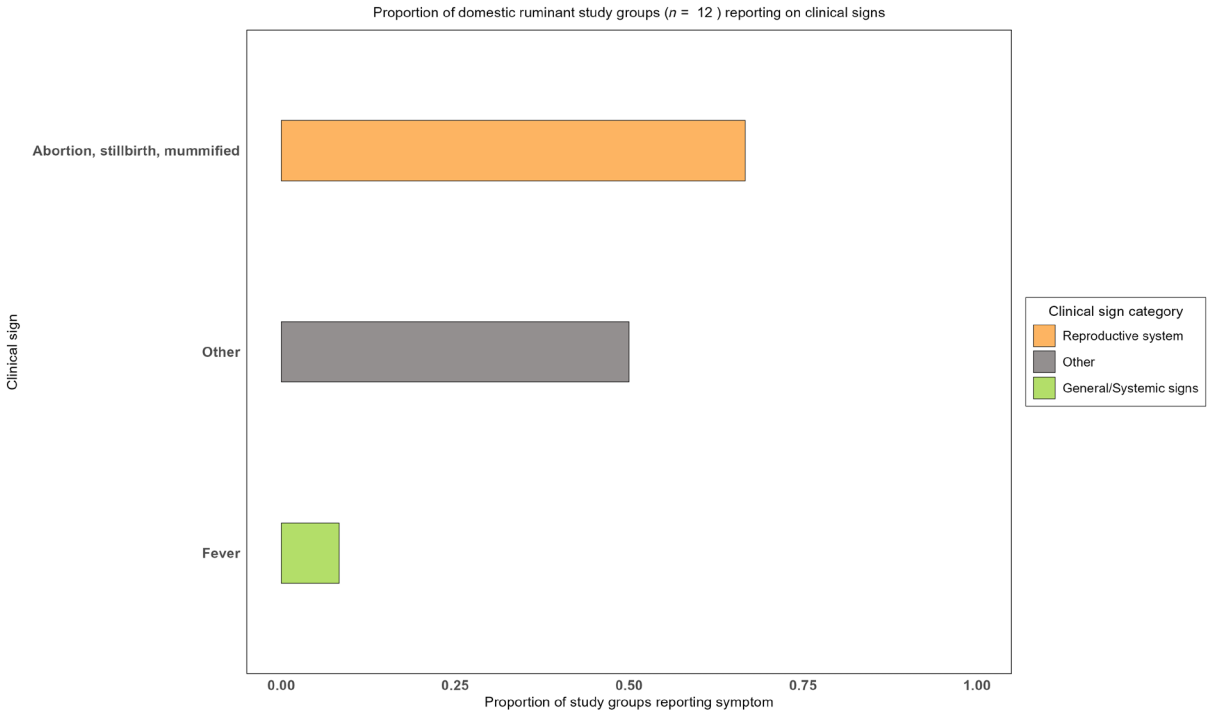


FIGURE 16 Clinical signs reported in the main hosts of *C. burnetii*. Study group count per domestic ruminant species: Goat $n = 9$; Sheep $n = 3$. The SLR was updated until 31 December 2025; for references, see Annex A.

In **sheep and goats**, the most significant clinical manifestation of *C. burnetii* infection is late-term abortion, often accompanied by the delivery of stillborn or weak offspring (Agerholm, 2013; Spickler, 2007; WOAH, 2022). While abortions may occur sporadically, *C. burnetii* is capable of causing sudden abortion outbreaks ('abortion storms') in previously unexposed flocks or herds, leading to substantial economic losses (van den Brom & Vellema, 2009). Severe necrotising placentitis is a frequent pathological finding in aborting animals (Agerholm, 2013). Non-specific clinical signs, such as transient malaise, anorexia or mild fever, may precede abortion but are generally mild and short-lived (Maurin & Raoult, 1999; Spickler, 2007; WOAH, 2022).

In cattle, C. *burnetii* infection is most commonly asymptomatic (Guatteo et al., 2011; WOAH, 2022). When clinical disease is observed, it primarily involves late-term abortion, stillbirths, weak calves, retained placenta, metritis and reduced fertility (Agerholm, 2013; Guatteo et al., 2011). Subclinical mastitis and bacterial shedding in milk have also been associated with *C. burnetii* infection in dairy cows, representing an important route for environmental contamination and zoonotic exposure (Guatteo et al., 2011; van den Brom & Vellema, 2009).

C. burnetii infections occur in many additional domestic and wild animal species, including companion animals, wildlife, birds and arthropods, most of which do not show clinical signs but may contribute to environmental maintenance and transmission of the bacterium (Maurin & Raoult, 1999; Spickler, 2007; WOAH, 2022).

Incubation period

The incubation period of Q-fever is generally variable and often difficult to determine due to the predominantly subclinical nature of the infection. However, in ruminants that develop clinical signs, particularly reproductive issues like abortion, signs typically manifest weeks to months after exposure (Spickler, 2007).

In experimental infections in goats, the incubation period has been shown to be as short as 3 days (Bouvery et al., 2003).

Morbidity and case fatality

Direct case fatality in animals due to *C. burnetii* is low. Animals, even those experiencing abortions, generally recover without severe systemic illness or fatal outcomes. Results obtained from the SLR resulted in one publication on experimental infection with *C. burnetii* meeting the inclusion criteria, which reported a fatality rate of 14% in goats (Arricau-Bouvery et al., 2005).

Zoonotic potential

Q-fever is a zoonotic disease (ECDC, 2025).

3.8.5 | Transmission

In animals, *C. burnetii* transmission is primarily through the inhalation of aerosols containing contaminated material (Maurin & Raoult, 1999; WOAH, 2022). High concentrations of the bacterium, up to a billion per cubic centimetre, are shed, particularly during parturition, in birth products (placenta, amniotic fluid), vaginal discharges, faeces and milk from infected ruminants (van Schaik et al., 2013).

The bacterium's small cell variant (SCV) exhibits spore-like resistance to heat, desiccation and environmental stress, allowing *C. burnetii* to persist for long periods in dust, bedding, manure and pasture environments, and to be dispersed over considerable distances by wind. This exceptional environmental persistence contributes significantly to the high infectivity and efficient airborne spread of Q-fever (Maurin & Raoult, 1999; Spickler, 2007; van Schaik et al., 2013).

Ticks may be involved in the mechanical transmission *of C. burnetii*, by passing the bacteria from an infected to a susceptible animal, and whose faeces contain the bacteria, thus also contaminating the environment (Celina & Cerný, 2022; Körner et al., 2021). Evidence on the potential and likely competent tick species and their geographic distribution can be found in a dedicated Scientific Report on vectors (EFSA, 2026a). While ticks may serve as vectors and play a role in maintaining *C. burnetii* in wildlife populations, their epidemiological importance in transmission within domestic livestock is generally considered secondary to aerosol transmission, especially during periods of birthing. The ingestion of contaminated materials, such as unpasteurized milk, can also lead to infection.

3.8.6 | Diagnostic tests

Recommended tests (WOAH, 2025b) for agent detection are PCR, culture and genotyping.

For immune response detection, the recommended tests are ELISA, indirect immunofluorescence assay and complement fixation test (CFT).

Table 14 presents data on the sensitivity and specificity of diagnostic tests collected through SLR; reported values correspond to the median sensitivity and specificity when multiple studies investigated the same test and are only included when explicitly stated in the publications.

TABLE 14 Median sensitivity and specificity of tests detecting *C. burnetii* or *C. burnetii* antibodies in studies included in the systematic literature review.

Target	Test	Species	Sensitivity	*N* animal groups	Specificity	*N* animal groups	References
Antibody	Complement fixation test	Goat	25.5%	3	99.7%	3	Muleme et al. (2016); Natale et al. (2012)
Antibody	ELISA	Cattle	82.0%	10	98.5%	10	Saegerman et al. (2022); Paul et al. (2013)
Antibody	ELISA	Goat	70.9%	2	88.5%	2	Muleme et al. (2016)
Antibody	I-ELISA	Cattle	97.0%	1	95.0%	1	Meletis et al. (2022)
Antibody	I-ELISA	Goat	100.0%	1	100.0%	1	Natale et al. (2012)
Antibody	Immunofluorescence Assay	Goat	88.8%	3	92.5%	3	Muleme et al. (2016)

Note: The SLR was updated until 31 December 2025; for references, see Annex A.

3.8.7 | Prevention and control

Vaccination

The EU has one inactivated vaccine available for commercial use in cattle, sheep and goats (Coxevac) (EMA, 2025).

Treatment

The treatment of infected animals is controversial. Some veterinarians advocate for the use of oxytetracyclines (Spickler, 2007). Others claim that antimicrobial therapy has little efficacy. There were no papers evaluating treatment efficacy that met the inclusion criteria[3] for the SLR.

3.9 | Crimean-Congo haemorrhagic fever virus (CCHFV)[11]

3.9.1 | Disease overview

Crimean-Congo haemorrhagic fever virus (CCHFV) causes Crimean-Congo haemorrhagic fever, a tick-borne viral disease. The virus is maintained in an enzootic cycle involving ticks and various wild and domestic animals. In animals, infection is generally subclinical, although seroconversion is frequent in areas where the virus circulates. Humans are the only species known to develop clinical disease (Ergönül, 2006; Spickler, 2019a; Whitehouse, 2004; WOAH, 2025).

Crimean-Congo haemorrhagic fever is a WOAH-notifiable disease, but it is not listed in the European AHL.

3.9.2 | Agent

CCHFV is a single-stranded, negative-sense RNA virus of the genus *Orthonairovirus*. The virus has a tripartite genome, comprising small (S), medium (M) and large (L) RNA segments. These segments encode essential structural and non-structural proteins including the nucleoprotein (NP), glycoproteins (Gn and Gc) and an RNA-dependent RNA polymerase (RdRp). The virus is enveloped and pleomorphic, typically ranging from 80 to 120 nm in diameter. The virus exhibits significant genetic variability, primarily driven by frequent segment reassortment and the error-prone nature of its RdRp during replication. Phylogenetic analyses have identified multiple distinct genotypes, with varying geographic distributions (Bente et al., 2013; Whitehouse, 2004).

3.9.3 | Geographical distribution

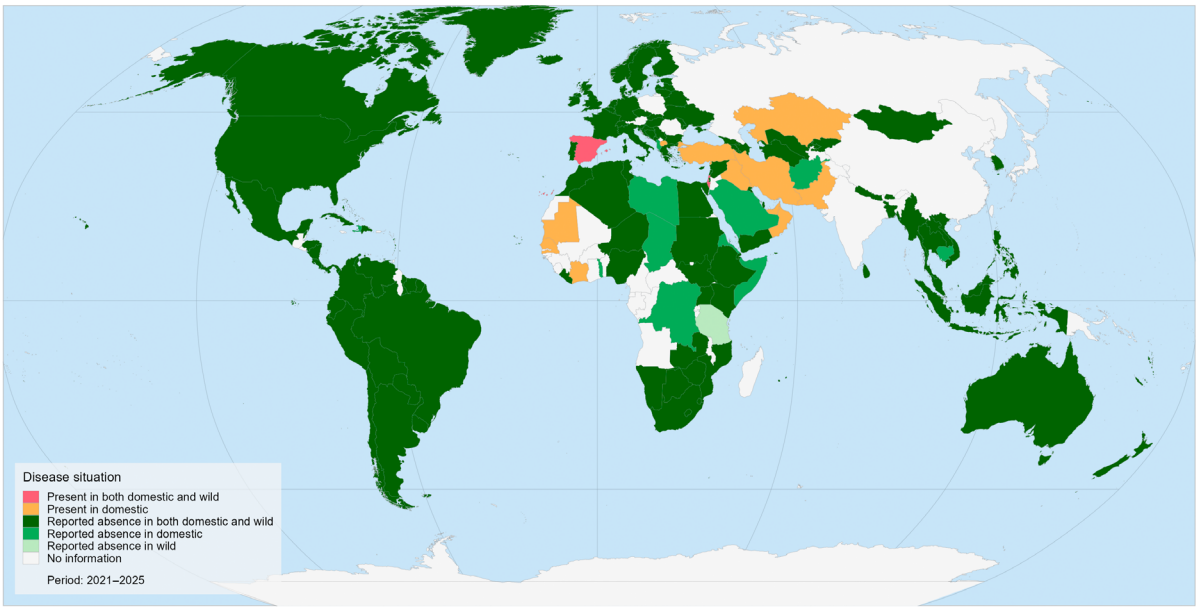


FIGURE 17 Geographical distribution of CCFHV detected events (2021–2025), as reported to WOAH.

Crimean-Congo haemorrhagic fever is endemic in Asia, Africa, the Middle East, the Balkans and has recently emerged in Spain. The disease mainly occurs in countries south of the 50th parallel north, which marks the ecological limit of its main tick vector. CCHFV has been reported in the EU over the past 5 years (Figure 17). Up-to-date maps based on WAHIS are available in the online version[11].

3.9.4 | Animal hosts

Susceptible hosts

Based on epidemiological knowledge of host–pathogen–vector interactions and outbreak reports, CCHFV has a wide range of vertebrate hosts with the most important being small wild terrestrial mammals and domestic ruminants, whereas equids are considered as dead-end hosts. However, other susceptible species have been identified in the SLR. The SLR summary is given in Table 15.

[11]Online version: https://animal-diseases.efsa.europa.eu/CCHFV.

TABLE 15 Susceptible hosts of Crimean-Congo haemorrhagic fever virus.

The SLR reported in the CCHFV disease profile, identified the following susceptible species (updated until 31 December 2025, references see Annex A)
FIELD
Epidemiological studies carried out in the field
• **Pathogen was detected in the following animal species:** • Bovidae: *Bos taurus, Capra hircus, Ovis aries* • Equidae: No species specified • Hominidae: *Homo sapiens*
• **Antibodies were detected in the following animal species:** • Bovidae: *Bos taurus, Capra hircus, Ovis aries, Syncerus caffer* • Camelidae: *Camelus dromedarius* • Canidae: *Canis lupus familiaris* • Equidae: *Equus caballus* • Hominidae: *Homo sapiens* • Suidae: *Sus scrofa*
• **Outbreaks reported to WOAH included the following species:** • Bovidae: *Bos taurus, Capra hircus, Ovis aries*
EXPERIMENTS
• **Experimental studies demonstrated infection in:** • Bovidae: *Ovis aries*

Clinical signs

No consistent clinical disease is observed in infected animals. Experimental infections in livestock have demonstrated short-term viraemia without observable signs of illness. Therefore, diagnosis relies on serological or molecular detection rather than clinical suspicion.

Incubation period

The incubation period in animals is not well defined due to the lack of clinical signs. The SLR identified three experimental studies in sheep, with the reported incubation varying from 3 to 8 days (Gonzalez et al., 1998; Li et al., 2024; Wilson et al., 1991).

Morbidity and case fatality

In livestock and wildlife, morbidity and case fatality are negligible, as infections are generally asymptomatic. However, the presence of high tick infestation rates and animal movement can influence the dynamics of virus circulation (Spickler, 2019a, 2019b).

Zoonotic potential

CCHFV infection is a zoonotic disease (WHO, 2025).

3.9.5 | Transmission

CCHFV is transmitted through the bite of infected ticks. Evidence on the potential and likely competent tick species and their geographic distribution can be found in a dedicated Scientific Report on vectors (EFSA, 2026a).

CCHFV circulates in a tick–vertebrate–tick enzootic transmission cycle. Transovarial and transstadial transmission in ticks allows for sustained circulation across life stages.

Animals become infected through tick bites, and the virus can be acquired by ticks during blood meals. While animals develop a transient viraemia (7–15 days), they do not transmit the virus directly to other animals or humans. Animal movement and seasonal tick activity contribute to virus spread in endemic areas. Transmission to humans occurs mainly through tick bites or contact with blood or tissues of infected animals (Bente et al., 2013; Ergönül, 2006; Spickler, 2019a, 2019b; Whitehouse, 2004).

3.9.6 | Diagnostic tests

Recommended tests (WOAH, 2025b) for agent detection: RT-PCR, Real-time RT-PCR and virus isolation in cell culture. Virus detection is typically limited to a short viraemic phase. Virus isolation requires BSL-4 containment and is rarely performed outside specialised laboratories.

For immune response detection, the recommended tests are IgM-capture ELISA, IgG-sandwich ELISA and competitive ELISA. The benefit of competitive ELISA is the capacity to investigate different animal species because they are host species independent. Virus neutralisation assays, generally considered to be highly specific, are rarely used for CCHFV diagnosis as members of the *Orthonairovirus* genus generally induce a weaker neutralising antibody response than members of other genera in the family Nairoviridae. Another drawback is the necessity to perform this assay in high biosafety containment because it uses live virus.

Serological tests are commonly used for surveillance in vertebrates. IgG ELISAs are the most widely used and can detect previous exposure. Cross-reactivity with other nairoviruses is possible and should be considered in the interpretation of results.

Table 16 presents data on the sensitivity and specificity of diagnostic tests collected through SLR; reported values correspond to the median sensitivity and specificity when multiple studies investigated the same test and are only included when explicitly stated in the publications.

TABLE 16 Median sensitivity and specificity of tests to detect CCHFV/CCHFV antibodies reported in literature included in the systematic literature review.

Target	Test	Species	Sensitivity	*N* animal groups	Specificity	*N* animal groups	References
Antibody	ELISA	Human	71.5%	4	93.2%	4	Cosgun et al. (2023)
Antibody	ELISA	Sheep	–	–	94.5%	2	Belij-Rammerstorfer et al. (2022)
Antibody	Indirect Elisa (I-ELISA)	Human	95%	2	99.1%	2	Shrivastava et al. (2022)
Antibody	Immunofluorescence Assay	Human	100%	1	100%	1	Cosgun et al. (2023)
Antibody	Lateral Flow Test	Human	39.7%	1	92.9%	1	Baniasadi et al. (2019)

Note: The SLR was updated until 31 December 2025; for references, see Annex A.

3.9.7 | Prevention and control

Vaccination

There are currently no licensed vaccines for CCHFV in animals.

Treatment

There is currently no specific treatment available for CCHFV infection in animals. Given the asymptomatic nature of infection, no therapeutic interventions are typically required.

3.10 | Eastern equine encephalitis virus (EEEV)[12]

3.10.1 | Disease overview

Eastern equine encephalitis virus (EEEV) is a highly pathogenic virus that causes Eastern equine encephalitis (EEE), a mosquito-borne viral disease which primarily circulates in an enzootic cycle involving birds and mosquitoes. Spillover infections can occur in equines and other mammals. Equine infection is associated with severe, often fatal, neurological disease (MacLachlan & Dubovi, 2017; Spickler, 2024a, 2024b, 2024c, 2024d; WOAH, 2025b).

Eastern equine encephalitis is a WOAH-notifiable disease, listed in the European AHL under category E.

3.10.2 | Agent

EEEV is an enveloped, single-stranded, positive-sense RNA virus that belongs to the *Alphavirus* genus of the Togaviridae family. 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 (Arrigo et al., 2010; Hasan et al., 2021).

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 case fatality 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 neuro-invasiveness and replication efficiency (Aguilar et al., 2008; Arrigo et al., 2010; CDC, 2024d; Hasan et al., 2021).

[12]Online version: https://animal-diseases.efsa.europa.eu/EEEV.

3.10.3 | Geographical distribution

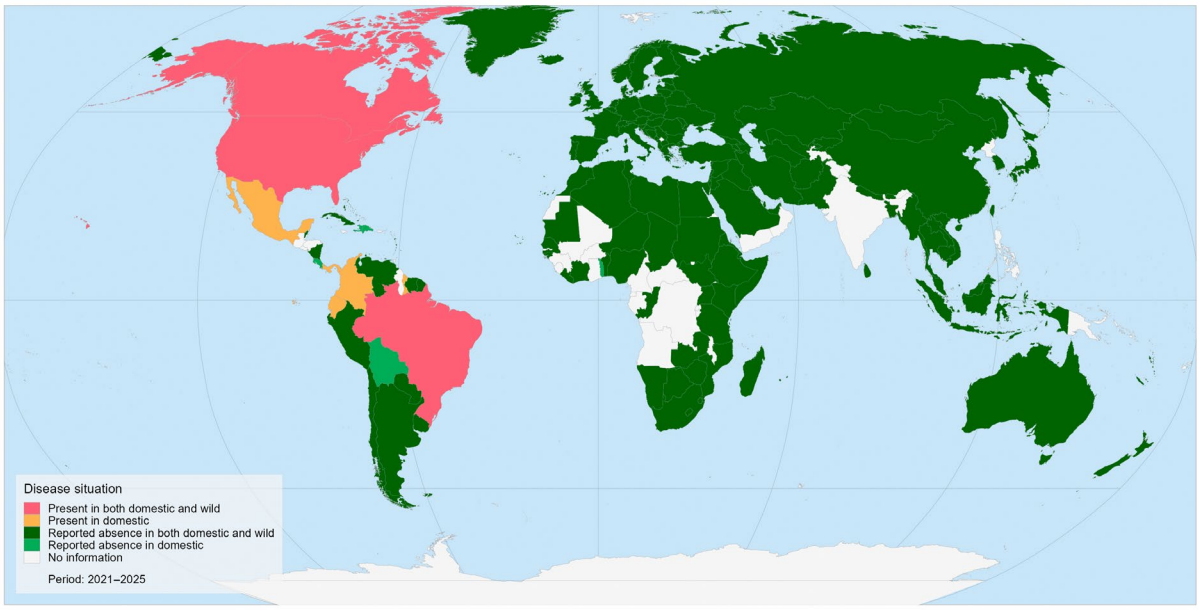


FIGURE 18 Geographical distribution of EEEV detected events (2021–2025), as reported to WOAH.

Eastern equine encephalitis occurs predominantly in eastern North America, parts of the Caribbean, and sporadically in Central and South America (Figure 18). According to WAHIS data, the agent was not reported in the EU in the last 5 years. Up-to-date maps based on WAHIS are available online.[12]

3.10.4 | Animal hosts

Susceptible hosts

Based on epidemiological knowledge of host–pathogen–vector interactions and outbreak reports, the **main hosts of EEEV are passerine birds** with humans and horses considered as dead-end hosts. However, other susceptible species have been identified in the SLR. The SLR summary is given in Table 17.

TABLE 17 Susceptible host species of Eastern equine encephalitis virus.

The SLR reported in the EEEV disease profile, identified the following susceptible species (updated until 31 December 2025; for references, see Annex A)
FIELD
Epidemiological studies carried out in the field
• **Pathogen was detected in the following animal species:** • Cervidae: *Odocoileus virginianus* • Equidae: *Equus caballus* • Phasianidae: *Gallus gallus domesticus*
• **Antibodies were detected in the following animal species:** • Cervidae: *Odocoileus virginianus, Alces alces* • Equidae: *Equus caballus* • Hominidae: *Homo sapiens*
• Outbreaks reported to WOAH included the following species: • Equidae: No species specified
EXPERIMENTS
• **Experimental studies demonstrated infection in:** • Phasianidae: *Gallus gallus domesticus, Meleagris gallopavo* • Anatidae: No species specified • Equidae: *Equus caballus*, • Bovidae: *Ovis aries, Bos taurus* • Suidae: *Sus scrofa domesticus*

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 h.

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

Experimental infections with pigs have shown that they can develop low-level, short-lived viraemia but typically do not show clinical signs (Elvinger et al., 1996). Due to a small number of study groups in the SLR investigating clinical signs, no graph was created.

Incubation period

In equines, the incubation period for EEEV 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.

Morbidity and case fatality

In equines, morbidity can be high during outbreaks, especially in unvaccinated populations. Case fatality rates exceed 75%–90%, making EEE one of the most lethal equine arboviral encephalitis. Survivors often exhibit long-term neurological deficits (MacLachlan & Dubovi, 2017; Spickler, 2024a, 2024b, 2024c, 2024d).

In birds, susceptibility varies by species. Some infected species may experience a high case fatality rate (up to 100%), while most remain asymptomatic (MacLachlan & Dubovi, 2017).

Zoonotic potential

Eastern equine encephalitis is a zoonotic disease (CDC, 2024a).

3.10.5 | Transmission

EEEV is transmitted to vertebrate hosts through the bite of mosquitoes (Diptera; Culicidae). Evidence on the potential and likely competent mosquito species and their geographic distribution can be found in a dedicated Scientific Report on vectors (EFSA, 2026a). Equines are dead end hosts and do not contribute to viral maintenance (Armstrong & Andreadis, 2013; CDC, 2024f; Scott & Weaver, 1989).

3.10.6 | Diagnostic tests

Recommended tests (WOAH, 2025b) for the detection of the agent: 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 from acutely affected or deceased equines.

Recommended tests (WOAH, 2025b) 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 detection 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.

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

3.10.7 | Prevention and control

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 (AAEP, 2024; Spickler, 2024a, 2024b, 2024c, 2024d).

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

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 (Deresiewicz et al., 1997; Spickler, 2024a, 2024b, 2024c, 2024d).

3.11 | Western equine encephalitis virus (WEEV)[13]

3.11.1 | Disease overview

Western equine encephalitis virus (WEEV) causes a mosquito-borne viral disease known as Western equine encephalitis. 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 (MacLachlan & Dubovi, 2017; Spickler, 2024a, 2024b, 2024c, 2024d; WOAH, 2025b).

Western equine encephalitis is a WOAH-notifiable disease and listed in the EU AHL under category E.

3.11.2 | 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 a structural polyprotein that includes the capsid (C) and envelope glycoproteins E1 and E2 (Hahn et al., 1988; Strauss & Strauss, 1994).

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 (Powers et al., 2001).

3.11.3 | Geographical distribution

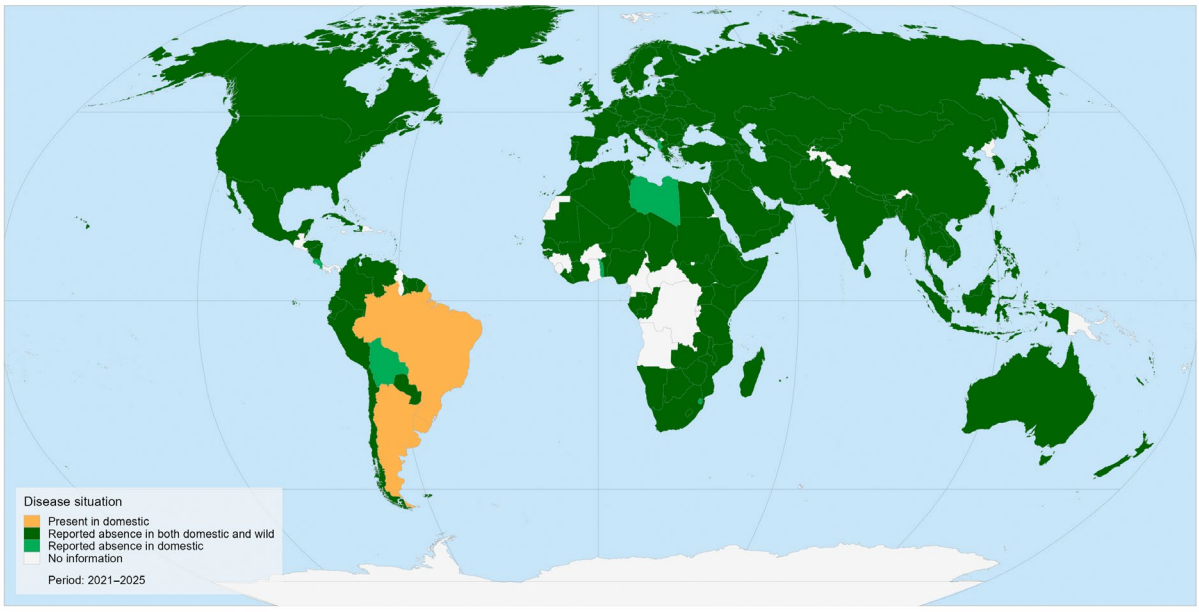


FIGURE 19 Geographical distribution of WEEV detected events (2021–2025), as reported to WOAH.

Western equine encephalitis is historically endemic in western North America, particularly in regions of the United States and Canada, although the disease does not seem to have been reported to WAHIS in the last 5 years (Figure 19). Sporadic equine outbreaks have been documented in Argentina and Brazil.

[13]Online version: https://animal-diseases.efsa.europa.eu/WEEV.

3.11.4 | Animal hosts

Susceptible hosts

Based on epidemiological knowledge of host–pathogen–vector interactions and outbreak reports, the main hosts of WEEV are passerine birds with humans and horses considered as dead-end hosts. However, other species have been identified in the SLR. The summary of the SLR on susceptible hosts is given in Table 18.

TABLE 18 Susceptible host species of Western equine encephalitis virus.

The SLR reported in the WEEV disease profiles, identified the following susceptible species (updated until 31 December 2025; for references, see Annex A)
FIELD
Epidemiological studies carried out in the field
• **Pathogen was detected in the following animal species:** • Equidae: *Equus caballus* • **Antibodies were detected in the following animal species:** • Canidae: *Canis lupus familiaris* • Equidae: *Equus caballus* • Hominidae: *Homo sapiens* • Rodentia (Order): No specific families nor species specified • **Outbreaks reported to WOAH included the following species:** • Bovidae: *Ovis aries* • Equidae: No species specified
EXPERIMENTS
• **Experimental studies demonstrated infection in:** • Anatidae: No species specified • Columbidae: No species specified • Leporidae: No species specified • Phasianidae: *Gallus gallus domesticus*

WEEV is maintained in a mosquito–bird–mosquito cycle, particularly in wet areas. Passerine birds act as amplifying hosts, with high-titre viraemia that infects mosquitoes. Equines are the most clinically affected by WEEV but are considered dead-end hosts due to insufficient viraemia for onward transmission.

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

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 viraemia following experimental infection, especially young birds.

Experimental WEEV infection in rabbits (Gresikova & Zavada, 1966) 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.

Incubation period

In equines, the incubation period for WEEV is typically 5–7 days, but may vary depending on viral dose and host factors. In experimentally infected poultry and pigs, viraemia is usually detectable within 1–3 days, with no clinical disease observed (Calisher, 1994; Spickler, 2024a, 2024b, 2024c, 2024d).

Morbidity and case fatality

In **equines**, morbidity during WEEV outbreaks is variable, with clinical disease reported in approximately 10%–40% of infected horses, depending on outbreak conditions, viral strain and host immunity (Calisher, 1994; Spickler, 2024a, 2024b, 2024c, 2024d). Among clinically affected horses, neurologic disease develops in only a subset, with some animals exhibiting

mild febrile illness while others progress to encephalomyelitis characterised by ataxia, paresis and altered mentation (Spickler, 2024a, 2024b, 2024c, 2024d).

The case fatality rate for equine WEEV infection is generally estimated at 20%–50%, which is substantially lower than that reported for EEEV but remains clinically and economically significant during outbreaks. Survivors typically recover without the severe long-term neurologic sequelae commonly observed following EEEV infection (Calisher, 1994; Spickler, 2024a, 2024b, 2024c, 2024d).

In **poultry** and **pigs**, WEEV infection is characterised by low pathogenicity, with mortality reported as rare or absent under both field conditions and experimental infection studies. Although transient viraemia can be detected following experimental inoculation, these species generally remain clinically normal, supporting their classification as incidental or dead-end hosts (Calisher, 1994).

Zoonotic potential

Western equine encephalitis is a zoonotic disease (CDC, 2024b).

3.11.5 | Transmission

WEEV is transmitted to vertebrate hosts through the bite of mosquitoes (Diptera; Culicidae). Evidence on the potential and likely competent mosquito species and their geographic distribution can be found in a dedicated Scientific Report on vectors (EFSA, 2026a).

In WEEV transmission cycles, **birds** develop sufficient viraemia to infect mosquitoes and serve as reservoir (amplifying) hosts, enabling sustained enzootic transmission. Virus circulation occurs primarily during mosquito-active months, typically spring through autumn in temperate regions of North America, reflecting the seasonal abundance and activity of mosquito vectors. Equines and other mammals, including humans, are considered dead-end hosts, as they develop insufficient or short-lived viraemia to infect mosquitoes and therefore do not contribute to viral maintenance or amplification in nature (Calisher, 1994; Weaver & Reisen, 2010).

3.11.6 | Diagnostic tests

Recommended tests (WOAH, 2025b) for the detection of the agent: 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.

Recommended tests (WOAH, 2025b) 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.

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

3.11.7 | Prevention and control

Vaccination

Inactivated vaccines against WEEV are available for equines, most commonly formulated as multivalent vaccines in combination with EEEV, Venezuelan equine encephalitis virus (VEEV) and/or West Nile virus (WNV). Annual vaccination is recommended in endemic regions, with vaccination ideally administered prior to the onset of peak mosquito activity to ensure protective immunity during periods of highest transmission risk (AAEP, 2024; Spickler, 2024a, 2024b, 2024c, 2024d).

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

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 (AAEP, 2024; Spickler, 2024a, 2024b, 2024c, 2024d).

3.12 | Epizootic haemorrhagic disease virus (EHDV)[14]

3.12.1 | Disease overview

Epizootic haemorrhagic disease virus (EHDV) causes epizootic haemorrhagic disease, a *Culicoides*-borne viral infection primarily affecting deer and cattle, causing high mortality in deer but often remaining subclinical in cattle. When clinical disease does occur in cattle, it typically involves fever, oedema, haemorrhages and erosions in the upper gastrointestinal tract and dysphagia (Spickler, 2019a, 2019b; WOAH, 2019a).

Epizootic haemorrhagic disease infection is a WOAH-notifiable disease, listed in the EU AHL under categories D and E.

3.12.2 | Agent

EHDV is a non-enveloped, double-stranded RNA virus that belongs to the *Orbivirus* genus of the Reoviridae family. The virion has three protein layers. The outer capsid contains two proteins, VP2 and VP5, with VP2 serving as the main determinant of serotype specificity. The core particle is a bilayered icosahedral structure composed of VP3 and VP7, where the latter is the serogroup-specific, immunodominant protein. Inside the core is the transcriptase complex, which includes VP1, VP4, VP6 and the segmented RNA genome. EHDV is closely related to bluetongue virus. Seven EHDV serotypes are currently recognised, with additional serotypes proposed. Ibaraki disease is caused by the Ibaraki strain of EHDV serogroup 2. The virus remains stable in blood and tissues at 20 and 4°C. It can be inactivated by heat treatment at 50°C for 3 h, 60°C for 15 min or 121°C for 15 min. EHDV is resistant to lipid solvents but is susceptible to several disinfectants, including β-propiolactone, 2% (w/v) glutaraldehyde, acids, alkalis (e.g. 2% sodium hydroxide), 2–3% (w/v) sodium hypochlorite, iodophores and phenolic compounds (Spickler, 2019a, 2019b; WOAH, 2025b).

3.12.3 | Geographical distribution

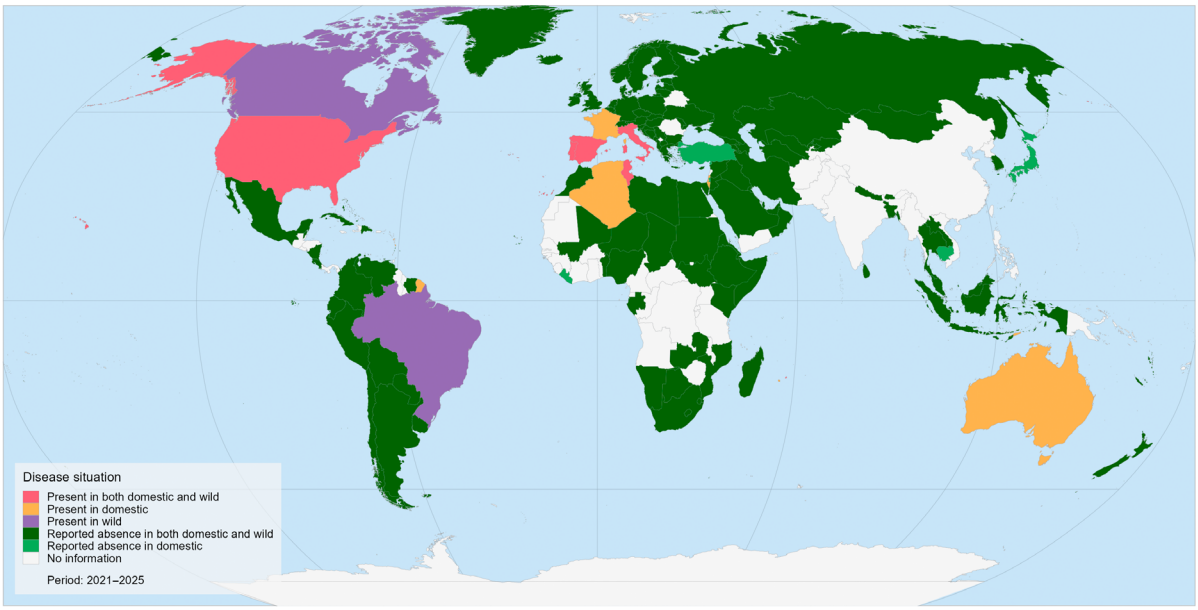


FIGURE 20 Geographical distribution of EHDV detected events (2021–2025), as reported to WOAH.

Cases of EHDV infection have been reported during 2021–2025 in domestic animals in North and South America, southwest Europe, northern Africa, Israel and Australia. Cases in wild animals have been reported from the same areas except for Australia (Figure 20). Up-to-date maps based on WAHIS are available in the online version.[14]

[14]Online version: https://animal-diseases.efsa.europa.eu/EHDV.

3.12.4 | Animal hosts

Susceptible species

Based on epidemiological knowledge of host–pathogen–vector interactions and outbreak reports, the main hosts of EHDV are cattle and cervids. However, other susceptible species have been identified in the SLR. The SLR summary is given in Table 19.

TABLE 19 Susceptible host species of epizootic haemorrhagic disease virus.

The SLR reported in the EHDV disease profile, identified the following susceptible species (updated until 31 December 2025, for references, see Annex A)
FIELD
Epidemiological studies carried out in the field
• **Pathogen was detected in the following animal species:** • Bovidae: *Bos taurus*, *Ovis aries* • Cervidae: *Odocoileus hemionus*, *Cervus elaphus*, *Odocoileus virginianus*
• **Antibodies were detected in the following animal species:** • Bovidae: *Bos taurus* • Cervidae: *Odocoileus hemionus*, *Cervus elaphus*
• **Outbreaks reported to WOAH included the following species:** • Bovidae: *Bos taurus*, *Capra hircus*, *Ovis aries* • Cervidae: *Cervus elaphus*, *Dama dama*, *Mazama gouazoubira*
EXPERIMENTS
• **Experimental studies demonstrated infection in:** • Bovidae: *Bos taurus*, *Capra hircus*, *Ovis aries* • Cervidae: *Odocoileus virginianus*, *Cervus elaphus*, *Dama dama*, *Capreolus capreolus*, *Cervus canadensis* • Suidae: *Sus scrofa domesticus*

Clinical signs

Outcomes of a SLR on clinical signs in 48 study groups of cattle and cervids are displayed in Figure 21. Predominantly general signs, no clinical signs or cardiovascular signs were reported.

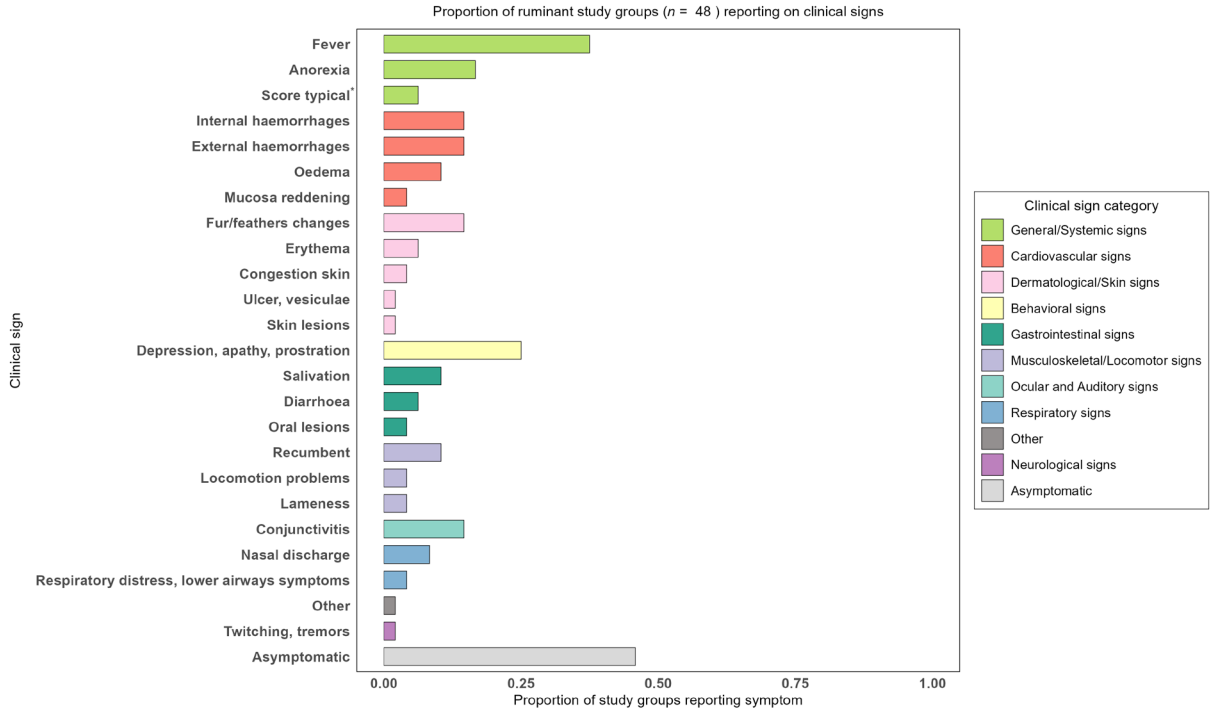


FIGURE 21 Clinical signs reported in the main hosts of EHDV. *Score typical: erosions in mouth, rumen and omasum. Study group count per species: Cattle $n = 24$; White-tailed deer $n = 19$; Wapiti elk $n = 2$; Fallow deer $n = 1$; Red deer $n = 1$; Roe deer $n = 1$. The SLR was updated until 31 December 2025; for references, see Annex A.

Deer: Clinical manifestations range from peracute to chronic disease. In the peracute form death often occurs within 36 h, sometimes without previous clinical signs (Spickler, 2019a, 2019b). Clinical signs are oedema of the head, neck, tongue, conjunctiva and lungs. In the acute form, clinical signs include fever, anorexia, diarrhoea, lethargy, lameness, haemorrhages and oedema in mucous membranes, skin and viscera. Erosions can be found in the mouth, rumen and omasum which can lead to excessive, sometimes blood-tinged, salivation. Death is common in the acute form. Those that survive the infection often have erosions, ulcers and scars in the rumen and omasum resulting in emaciation. In the chronic form, apart from damages in the rumen and omasum, growth rings or sloughing of the hoof wall may be seen. White-tailed deer is the most affected species, but similar clinical signs including death have been reported in some other cervids (Spickler, 2019a, 2019b).

Cattle: Most infections in cattle are subclinical (Spickler, 2019a, 2019b; WOAH, 2019a). Clinical signs in cattle include fever, anorexia, lameness, oedema, haemorrhages and ulcers and erosions in the mouth, lips, abomasum and coronets, eye and nasal discharge and salivation. Damaged striated muscles in the pharynx, larynx, oesophagus and tongue can lead to difficulty swallowing. This may lead to dehydration, emaciation and aspiration pneumonia. Abortions, stillbirths and foetal deformation have also been reported (Spickler, 2019a, 2019b).

Yak: Lameness, oral and nasal discharge have been reported in yaks infected with EHDV (Spickler, 2019a, 2019b).

Sheep: Reported clinical signs include fever, oedema of the head, lesions and hyperaemia in mouth and nose, lethargy, anorexia, lameness, abdominal distension and death. Clinical signs in experimentally infected sheep have been asymptomatic or showed mild clinical signs such as a rise in body temperature, buccal hyperaemia or ulceration of buccal mucous membranes (Spickler, 2019a, 2019b).

Incubation period

The incubation period in deer is reported to be 5–10 days in deer and 1–6 days in experimentally infected cattle (Annex A).

Morbidity and case fatality

In captive white-tailed deer, morbidity and case fatality might be up to 90% (Spickler, 2019a, 2019b; WOAH, 2019a). In wild white-tailed deer, mortality was estimated to be between 6% and 20% (Spickler, 2019a, 2019b). While the case fatality in experimentally infected white-tailed deer ranged between 25% and 60% (Quist et al., 1997).

In cattle, mortality rates of around 2%–10% have been reported (Spickler, 2019a, 2019b). In experimentally infected cattle, the fatality rates ranged between 25% and 100% (Spedicato et al., 2024).

Zoonotic potential

EHDV is not known to infect humans under natural conditions.

3.12.5 | Transmission

EHDV is transmitted by biting midges of the genus *Culicoides* (Diptera; Ceratopogonidae). Evidence on the potential and likely competent *Culicoides* species and their geographic distribution can be found in a dedicated Scientific Report on vectors (EFSA, 2026a).

The extrinsic incubation period is 10–14 days. The virus is not spread by direct contact between animals. In temperate climates, infection is most common during peak vector population, i.e. late summer and autumn. In tropical regions, outbreaks occur year-round. The midges can fly short distances but can be transported longer distances with the wind (Spickler, 2019a, 2019b; WOAH, 2019a).

Vertical transmission of the Ibaraki virus strain has been found in aborted fetuses in cattle. There is also one reported case of vertical transmission in cattle in the US (Spickler, 2019a, 2019b).

3.12.6 | Diagnostic tests

WOAH-recommended tests for agent detection are virus isolation in cell culture, RT-PCR and real-time RT-PCR. For agent detection, the recommended samples are whole blood, spleen, lungs, lymph nodes or liver (WOAH, 2025b).

Real-time RT-PCR is the preferred method for EHDV detection due to its high sensitivity and specificity, and no amplification is observed with related serotypes of bluetongue virus. There are commercial real-time RT-PCR kits that detect genome segment 9. There are also serotype-specific real-time RT-PCR kits available to distinguish the serotypes (WOAH, 2025b).

Real-time RT-PCR is a sensitive and specific method that can distinguish EHDV from bluetongue virus. Due to its ability to detect small amounts of virus, the results should be interpreted with caution since it does not necessarily indicate the presence of infectious virus and is sensitive to contamination. According to the SLR, the sensitivity and specificity of real-time RT-PCR in cattle are 100% and 95.5%, respectively (Schroeder et al., 2013).

For immune response detection, the recommended tests are competitive ELISA, virus neutralisation test (VNT), agar gel immunodiffusion (AGID) and complement fixation test (CFT).

Competitive ELISA detects serogroup-specific antibodies without cross-reactivity with other orbiviruses and is currently the preferred method. VNT detects and quantifies serotype-specific antibodies and is the reference method for this purpose, but it is time-consuming and labour-intensive and requires inclusion of all serotypes. AGID is a simple method for antibody detection but cannot distinguish between EHDV and BTV. The complement fixation test is serogroup-specific and can detect antibodies for 4–12 months after infection. (WOAH, 2025b).

3.12.7 | Prevention and control

Vaccination

In Japan, live-attenuated and inactivated vaccines have been developed and used to control the Ibaraki virus strain. In USA, autogenous inactivated vaccines have been developed for captive deer (WOAH, 2019a).

In the EU, an inactivated recombinant protein vaccine (serotype 8, VP2 protein) has been approved (EMA, 2026).

Treatment

There is currently no specific treatment for infection with EHDV (Spickler, 2019a, 2019b).

3.13 | Equine infectious anaemia virus (EIAV)[15]

3.13.1 | Disease overview

Equine infectious anaemia virus (EIAV) causes equine infectious anaemia, a persistent non-contagious viral disease that primarily affects members of the Equidae family. The disease is transmitted mechanically by biting insects and iatrogenically through blood-contaminated equipment. It presents with variable severity across three clinical forms (acute, chronic and inapparent). Infected equids frequently become lifelong viraemic carriers (Timoney, 2020; WOAH, 2025b).

Equine infectious anaemia is a WOAH-notifiable disease, listed in the European AHL under categories D and E.

3.13.2 | Agent

EIAV is an enveloped, single-stranded, positive-sense RNA virus in the *Lentivirus* genus of the Retroviridae family. The virion is pleomorphic, around 80–120 nm in diameter, with a diploid genome inside a conical core that encodes the structural proteins Gag, Pol and Env, as well as regulatory proteins Tat, Rev and S2. The surface glycoprotein gp90 determines viral antigenicity and is the main target of neutralising antibodies. EIAV does not have clearly defined serotypes analogous to those of orbiviruses; rather, variation occurs through genetic drift in the encoded envelope proteins, which influences virulence, antigenicity and immune evasion (Cook et al., 2013; WOAH, 2025b).

EIAV persists in infected equids as a lifelong viraemia and is highly stable in refrigerated blood, although it is inactivated by heat and common disinfectants (Cook et al., 2013; WOAH, 2025b).

[15]Online version: https://animal-diseases.efsa.europa.eu/EIAV.

3.13.3 | Geographical distribution

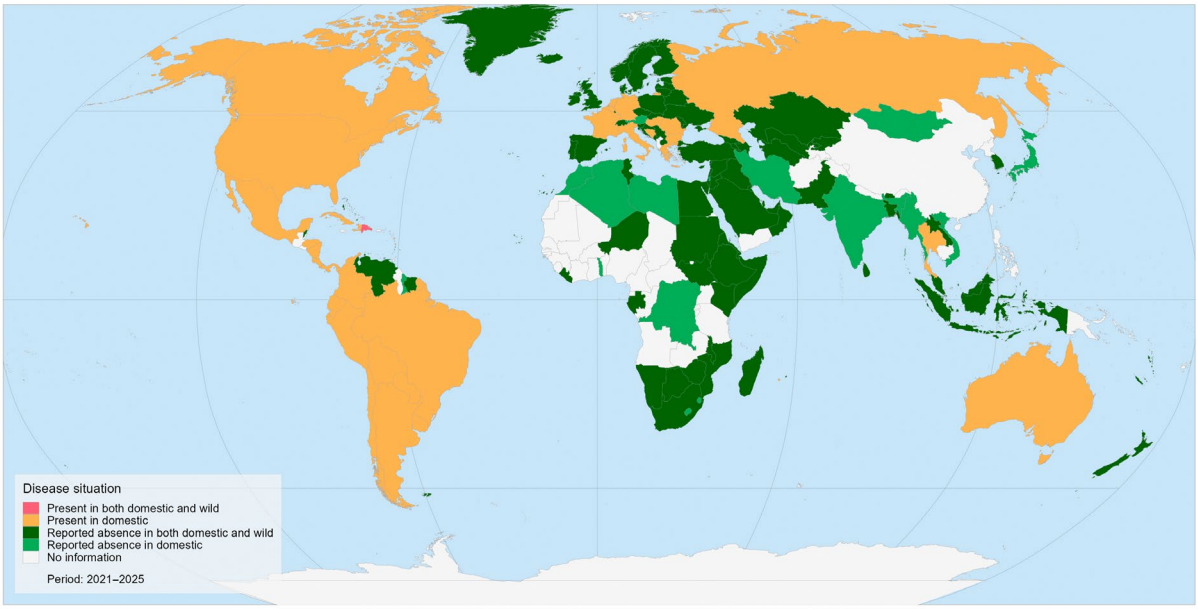


FIGURE 22 Geographical distribution of EIAV detected events (2021–2025), as reported to WOAH.

Equine infectious anaemia has a worldwide geographical distribution, but its prevalence varies significantly by region. The highest prevalence rates for EIAV are generally found in Central and South America. According to WAHIS, the disease has been reported in Europe in the last 5 years (Figure 22). Up-to-date maps based on WAHIS are available in the online version.[15]

3.13.4 | Animal hosts

Susceptible species

Based on epidemiological knowledge of host–pathogen–vector interactions and outbreak reports, the main hosts of EIAV are equids. Equids were the only susceptible hosts identified in the SLR (Table 20).

TABLE 20 Susceptible host species of Equine infectious anaemia virus.

Within the studies reviewed by the literature review in the EIAV disease profile, the following hosts were identified up to 31 December 2025. For references, see section Annex A.
FIELD
Epidemiological studies carried out in the field
• **Pathogen was detected in the following animal species:** • No species specified
• **Antibodies were detected in the following animal species:** • Equidae: *Equus caballus*
• **Outbreaks reported to WOAH included the following species:** • Equidae: No species specified
EXPERIMENTS
• **Experimental studies demonstrated infection in:** • Equidae: *Equus caballus*

Clinical signs

All equids are susceptible to EIAV and can become lifelong carriers. Clinical disease is most frequently recognised in horses, while donkeys and mules often show milder or subclinical infection. Indeed, donkeys may remain largely asymptomatic despite infection, and mules may also carry the virus without overt signs (Spickler, 2022a).

Outcomes of a SLR on clinical signs in 35 study groups of equids are displayed in Figure 23. Predominantly, general clinical signs were reported.

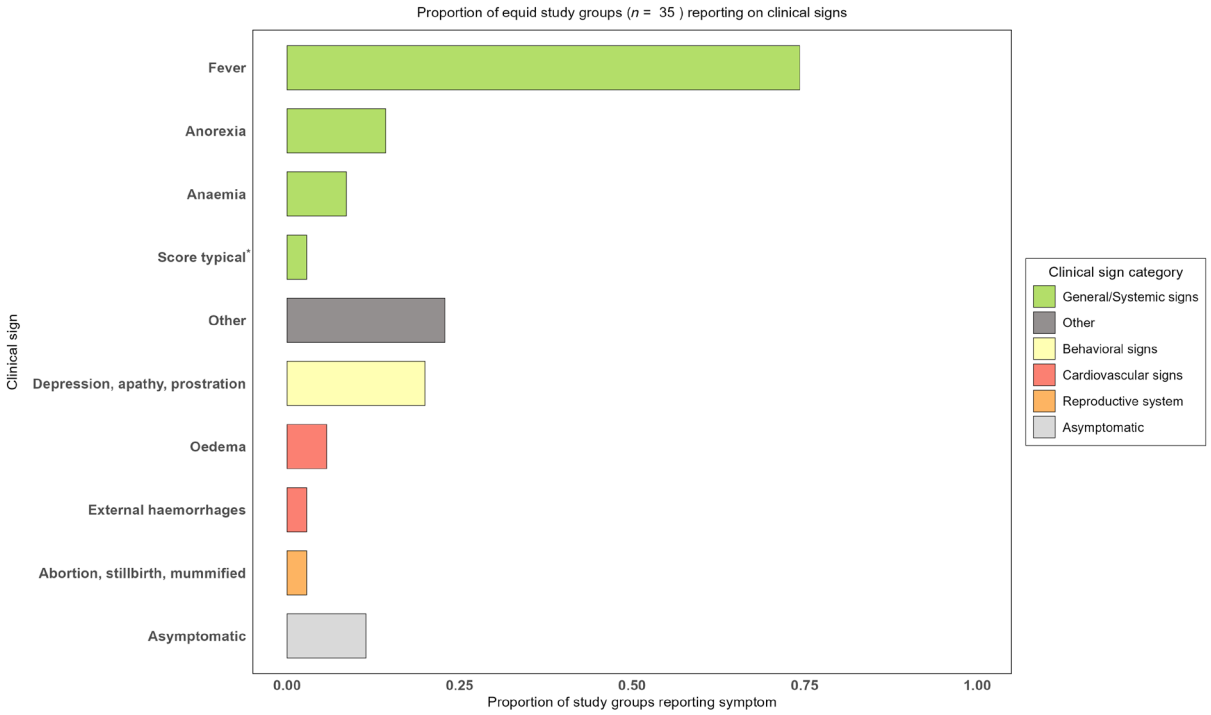


FIGURE 23 Clinical signs reported in the main hosts of EIAV. *Score typical: recurrent fever. Study group count per equid species: Horse $n = 33$; Ass $n = 2$. The SLR was updated until 31 December 2025; for references, see Annex A.

Equine infectious anaemia occurs in several clinical patterns, influenced by virus strain, equid species and immune status. In horses, there are three recognised clinical forms:

- Acute form: typically brief, lasting 1–3 days, and marked by sudden fever, depression and severe thrombocytopenia; some horses may develop jaundice, petechiae or epistaxis (Spickler, 2022a, 2022b). Although often mild and easily overlooked, rare peracute cases exhibit high fever, profound weakness and uncontrolled viral replication leading to circulatory collapse and death (Timoney, 2020).
- Chronic form (recurrent): characterised by intermittent febrile episodes accompanied by depression, thrombocytopenia, anaemia, jaundice, petechiae on mucous membranes, dependent oedema, increased heart and respiratory rates, muscle weakness and progressive weight loss (Spickler, 2022a, 2022b; Timoney, 2020). Episodes may be triggered by intercurrent disease, stress, heavy work or immunosuppression (Spickler, 2022a, 2022b), with intervals ranging from days to months and severity varying widely among individuals (Timoney, 2020).
- Subclinical form (inapparent carrier state): the most common long-term outcome, in which horses remain clinically normal or exhibit only subtle signs such as transient fever or mild inappetence. These animals serve as reservoirs of infection despite the absence of overt disease (Timoney, 2020).

Donkeys and mules are generally less severely affected, often showing minimal or no clinical abnormalities unless infected with highly adapted strains (Spickler, 2022a, 2022b).

Incubation period

The incubation period of equine infectious anaemia is variable, typically ranging from 1 week to 45 days, with many naturally infected horses developing signs between 15 and 45 days (Timoney, 2020). In some cases, the period may extend beyond this range, and clinical onset can be delayed until animals experience stress or concurrent illness that precipitates detectable disease (Spickler, 2022a, 2022b). Because early signs are often mild and transient, the true incubation period can be difficult to determine precisely.

Donkeys and mules, which frequently develop subclinical infections, may seroconvert without showing observable clinical signs, making their incubation period particularly challenging to define (Spickler, 2022a, 2022b).

Morbidity and case fatality

Horses are highly susceptible to EIAV. In endemic areas, herd-level seroprevalence has been reported to reach up to 70% on some farms (Spickler, 2022a, 2022b). Experimental infections demonstrate the impact of virulence and dose: highly virulent strains or high-dose inoculation have produced mortality rates approaching 80%, whereas low-virulence strains or low-dose exposure may result in predominantly inapparent infection (Cook et al., 2013). In natural settings, fatality is generally

low, with deaths occurring mainly in acute or peracute cases, while the majority of infected horses survive and transition into a lifelong inapparent carrier state (Cook et al., 2013; Timoney, 2020).

Mules and donkeys show lower morbidity and very low case fatality, with most infections remaining subclinical unless caused by highly adapted strains (Cook et al., 2013; Spickler, 2022a, 2022b; Annex A).

Zoonotic potential

EIAV is not known to infect humans under natural conditions.

3.13.5 | Transmission

EIAV is primarily transmitted via biting flies which mechanically transfer the virus from infected to susceptible equids (Cook et al., 2013; Timoney, 2020). Evidence on the potential and likely competent biting fly species and their geographic distribution can be found in a dedicated Scientific Report on vectors (EFSA, 2026a).

Direct transmission between horses through casual contact is considered rare, but iatrogenic transmission can occur through contaminated needles, surgical instruments or blood transfusions (CFSPH, 2022; Timoney, 2020). Movement of infected animals can facilitate long-distance spread of the virus. Seasonal and environmental factors influence vector abundance and activity, contributing to the episodic occurrence of outbreaks (Cook et al., 2013). Adult vectors can survive in favourable conditions, allowing EIAV to persist within populations even outside peak transmission seasons (Spickler, 2022a, 2022b).

3.13.6 | Diagnostic tests

WOAH-recommended test (WOAH, 2025b) for agent detection is virus isolation, although isolation from the blood of infected animals is only successful during the short periods of viraemia. Molecular assays such as RT-PCR can also detect viral RNA or proviral DNA and are considered complementary tools, particularly in animals with low or intermittent viraemia.

For immune response detection, the recommended tests are Agar Gel Immunodiffusion (AGID, Coggins test) and ELISA. The AGID test remains the reference method for confirming exposure and detecting antibodies, while ELISA is widely used for large-scale screening and surveillance. Because most infections in adult equids are subclinical, serology is the primary tool for identifying past infection at the herd level. Detection of antibodies in pre-colostral fetal fluids or tissues from stillborn or aborted fetuses provides strong evidence of in utero infection (WOAH, 2025b).

Table 21 presents data on the sensitivity and specificity of diagnostic tests collected through SLR; reported values correspond to the median sensitivity and specificity when multiple studies investigated the same test and are only included when explicitly stated in the publications.

TABLE 21 Median sensitivity and specificity of tests to detect EIAV/EIAV antibodies reported in literature included in the systematic literature review.

Target	Test	Species	Sensitivity	*N* animal groups	Specificity	*N* animal groups	References
Antibody	ELISA (blocking)	Horse	100%	1	97.0%	1	Hu et al. (2023)
Antibody	ELISA (competitive)	Horse	98%	5	100%	5	Espasandin et al. (2021); Nardini et al. (2017); Nardini et al. (2016)
Antibody	ELISA	Donkey	94.6%	2	96.1%	2	Villa-Mancera et al. (2024); Naves et al. (2019)
Antibody	ELISA	Horse	99.6%	13	97.6%	13	Ostuni et al. (2025); Odio et al. (2025); Villa-Mancera et al. (2024); Russi et al. (2023); Rodríguez-Domínguez et al. (2021); Naves et al. (2019); Fontes et al. (2018); Alvarez et al. (2015)
Antibody	ELISA	Mule	100%	1	91%	1	Naves et al. (2019)
Antibody	Fluorescence polarisation (FP)	Horse	93.7%	2	99%	2	Espasandin et al. (2021); Tencza et al. (2000)
Antibody	ELISA (indirect)	Equines	98%	2	99.9%	2	Ostuni et al. (2023); Scicluna et al. (2018)
Antibody	ELISA (indirect)	Horse	98%	3	100%	3	Nardini et al. (2017)

(Continues)

TABLE 21 (Continued)

Target	Test	Species	Sensitivity	*N* animal groups	Specificity	*N* animal groups	References
Antigen	AGIDT	Equines	97.5%	2	-	-	Oliveira et al. (2013)
Antigen	AGIDT	Horse	100%	1	100%	1	Piza et al. (2007)
Antigen	ELISA	Horse	96.1%	3	96.4%	3	Du et al. (2018); Reis et al. (2012); Piza et al. (2007)
Nucleic Acid	Real-time PCR	Horse	43.8%	1	99.1%	1	Cook et al. (2019)
Nucleic Acid	Reverse transcription-insulated isothermal PCR (RT-iiPCR)	Horse	80.8%	2	99.1%	1	Cook et al. (2019)

Note: The SLR was updated until 31 December 2025; for references, see Annex A.

3.13.7 | Prevention and control

Vaccination

No effective, globally accepted vaccines against EIAV are currently available. An attenuated live vaccine was developed and used in China from the 1970s through the 1980s, but this vaccine is no longer in use and has been discontinued worldwide (Thieulent et al., 2025; WOAH, 2025b).

In the European Union, as elsewhere, there are no licensed or authorised EIAV vaccines, and control strategies focus on serological surveillance (e.g. via the *Coggins/AGID test*) and biosecurity to prevent infection (WOAH, 2025b).

Treatment

There is currently no specific antiviral treatment or cure for equine infectious anaemia. Management of infected equids focuses on supportive care, including rest, stress reduction and treatment of secondary infections or complications such as anaemia or oedema (Cook et al., 2013; Spickler, 2022a, 2022b).

3.14 | Japanese encephalitis virus (JEV)[16]

3.14.1 | Disease overview

Japanese encephalitis virus (JEV) causes Japanese encephalitis, a mosquito-borne viral disease. JEV primarily affects pigs and birds. 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 (Spickler, 2023a, 2023b, 2019b; WOAH, 2025b).

Japanese encephalitis is a WOAH-notifiable disease, listed in the EU AHL under category E.

3.14.2 | Agent

JEV is an enveloped, single-stranded, positive-sense RNA virus that belongs to the *Orthoflavivirus* genus of the Flaviviridae family, which includes West Nile, Usutu and St. Louis encephalitis viruses. 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 (Schuh et al., 2014; Solomon et al., 2003; WOAH, 2019b, 2025b).

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 (Schuh et al., 2014; Solomon et al., 2003).

[16]Online version: https://animal-diseases.efsa.europa.eu/JEV.

3.14.3 | Geographical distribution

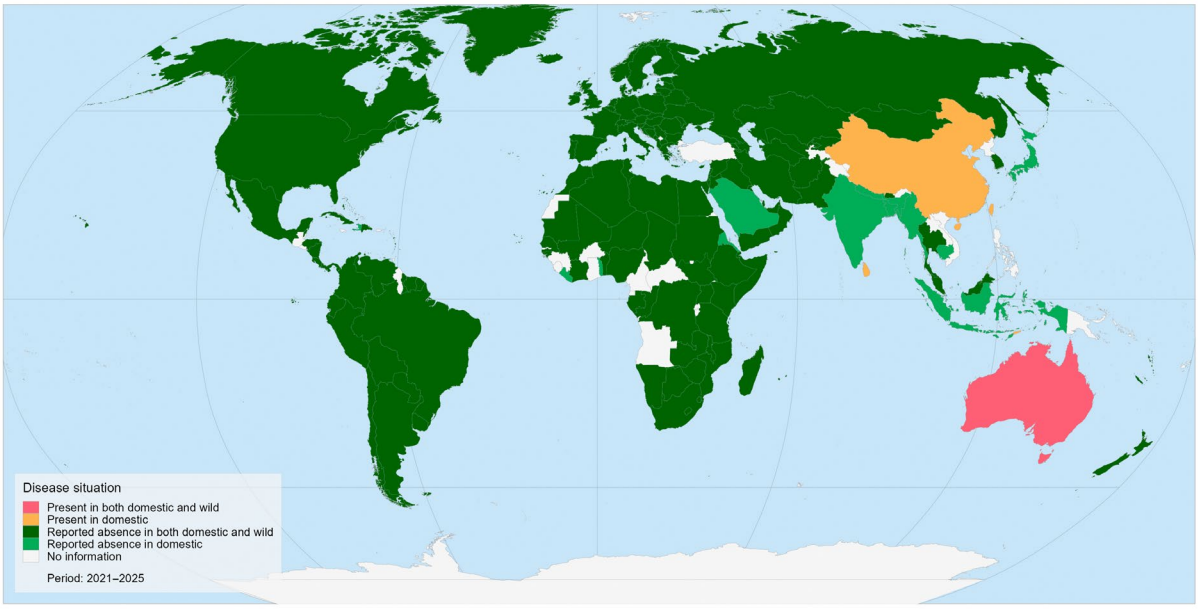


FIGURE 24 Geographical distribution of JEV detected events (2021–2025), as reported to WOAH.

Japanese encephalitis is endemic in some countries in Asia and Oceania. According to WAHIS, the disease has not been reported in Europe in the last 5 years (Figure 24). Up-to-date maps based on WAHIS are available in the online version.[16]

3.14.4 | Animal hosts

Susceptible hosts

Based on epidemiological knowledge of host–pathogen–vector interactions and outbreak reports, the main hosts of JEV are wild birds and Suidae, whereas humans and equids are considered as dead-end hosts. However, other susceptible species have been identified in the SLR (Table 22).

TABLE 22 Susceptible host species of Japanese encephalitis virus.

The SLR reported in the JEV disease profile, identified the following susceptible species (updated until 31 December 2025; for references, see Annex A)
FIELD
Epidemiological studies carried out in the field
• **Pathogen was detected in the following animal species:** • Bovidae: *Bos taurus, Capra hircus, Ovis aries, Bubalus* spp. • Canidae: *Canis lupus familiaris* • Equidae: *Equus caballus, Equus asinus* × *Equus caballus (Mule)* • Suidae: *Sus scrofa domesticus*
• **Antibodies were detected in the following animal species:** • Bovidae: *Bos taurus* • Canidae: *Canis lupus familiaris* • Equidae: *Equus caballus* • Hominidae: *Homo sapiens* • Phasianidae: *Gallus gallus domesticus* • Suidae: *Sus scrofa domesticus, Sus scrofa, Sus scrofa leucomystax*
• **Outbreaks reported to WOAH included the following species:** • Camelidae: *Vicugna pacos* • Suidae: No species specified
EXPERIMENTS
• **Experimental studies demonstrated infection in:** • Anatidae: No species specified • Columbidae: No species specified • Phasianidae: *Gallus gallus domesticus* • Suidae: *Sus scrofa domesticus, Sus scrofa*

Clinical signs

Outcomes of a SLR on clinical signs in 39 suid study groups and 22 bird study groups are displayed in Figure 25. In birds, most study groups did not report any clinical signs, while in swine most study groups reported general clinical signs or absence of clinical signs.

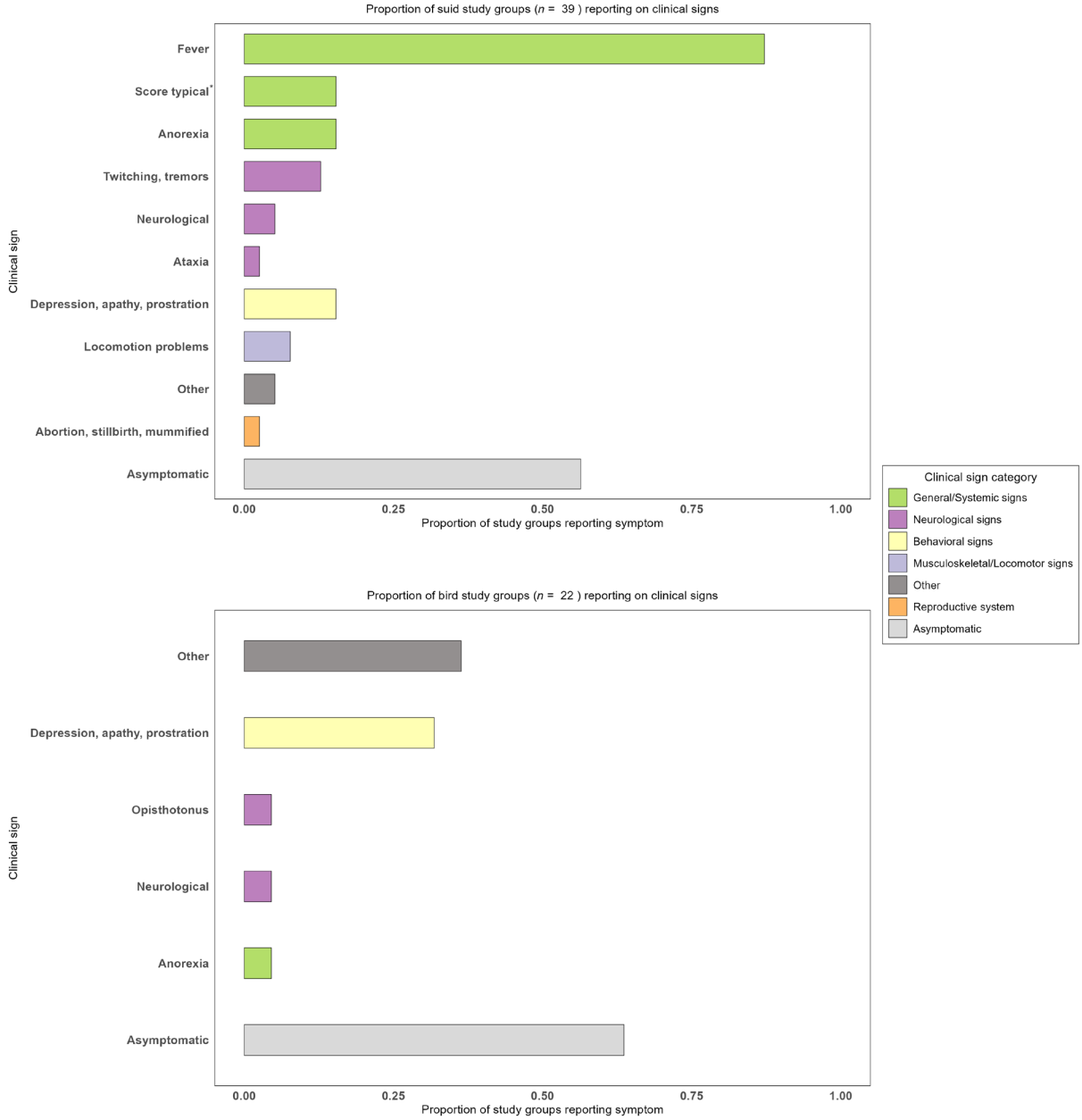


FIGURE 25 Clinical signs reported in the main hosts of JEV. *Score typical: stillbirths and mummified piglets; neurological signs in piglets. Study group count per suid species: Pig $n = 38$; Wild boar $n = 1$. Study group count per bird species: Duck $n = 18$; Chicken $n = 4$. The SLR was updated until 31 December 2025; for references, see Annex A.

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 fetuses usually at term. Boars may show reduced sperm count and motility. Live-born piglets may present 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 viraemia and occasional mild signs.

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

- 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.

Incubation period

In horses, the incubation period has been experimentally determined to be 8–10 days. For experimentally infected swine, signs of infection, fever and viraemia were observed 24 h post inoculation with other clinical manifestations apparent within 6 days post inoculation (Spickler, 2023a, 2023b; WOAH, 2019b).

Morbidity and case fatality

In pigs, morbidity is usually expressed as reproductive failure rather than clinical disease, and case fatality is negligible in adults. However, case fatality 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 (Spickler, 2023a, 2023b; WOAH, 2019b).

Zoonotic potential

Japanese encephalitis is a zoonotic disease (WHO, 2024a).

3.14.5 | Transmission

JEV is transmitted by mosquitoes (Diptera; Culicidae). Evidence on the potential and likely competent mosquito species and their geographic distribution can be found in a dedicated Scientific Report on vectors (EFSA, 2026a).

The virus is maintained in a bird–mosquito enzootic cycle. Several bird species (e.g. herons, egrets, sparrows, starlings, ducks and pigeons) act as natural hosts, while 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 viraemia to infect mosquitoes. Experimental studies have shown that young poultry can develop sufficient viraemia 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, vertical transmission or persistence in alternative hosts such as reptiles, amphibians or bats.

3.14.6 | Diagnostic tests

Recommended tests (WOAH, 2025b) for the detection of the agent: 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.

Recommended tests (WOAH, 2025b) 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.

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

3.14.7 | Prevention and control

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 are employed, primarily to protect breeding sows against reproductive losses (Spickler, 2023a, 2023b; WOAH, 2019b, 2025b).

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

Treatment

There is currently no specific antiviral treatment for JEV infection. Supportive care in horses includes anti-inflammatory therapy, fluid support and intensive nursing (Spickler, 2023a, 2023b; WOAH, 2019b).

When feasible, it is recommended to keep away swine from horses and humans to limit transmission and prevent outbreaks.

3.15 | *Leishmania infantum* (*L. infantum*)[17]

3.15.1 | Disease overview

Leishmania infantum causes Leishmaniosis, an infectious, non-contagious protozoan disease transmitted by sandflies (Diptera; Phlebotominae). Leishmaniosis affects several mammalian species, but dogs are considered the most relevant domestic reservoir (Spickler, 2022a, 2022b; WOAH, 2025a).

Leishmaniosis is a WOAH-notifiable disease, but it is not listed in the European AHL.

3.15.2 | Agent

L. infantum is an intracellular protozoan parasite belonging to the family Trypanosomatidae. It is observed in two different forms in nature. The promastigote form is found in the vector, is extracellular, flagellated, elongated, spindle-shaped and measures 15–30 µm in length and 5 µm in width. A long flagellum extends from the anterior end. The amastigote form is intracellular, found within macrophages of the vertebrate host. They are typically ovoid or spherical, measuring 1–5 µm in length and 1–2 µm in width. They have a large nucleus, a prominent kinetoplast and a short axoneme (Baneth et al., 2008; Kaye & Scott, 2011; Ready, 2014).

3.15.3 | Geographical distribution

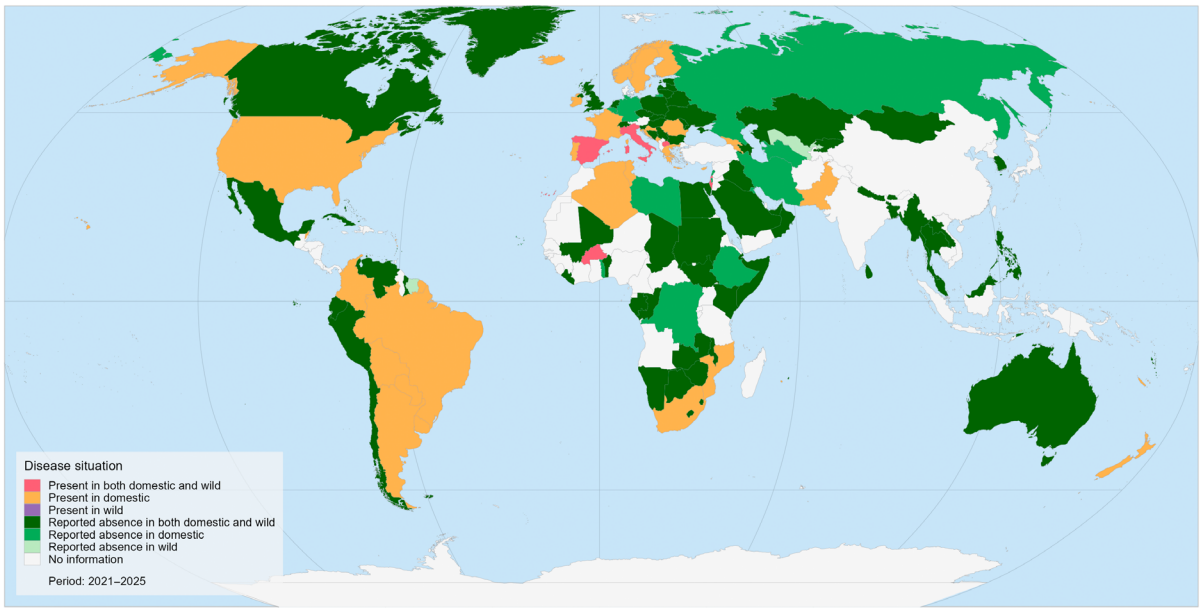


FIGURE 26 Geographical distribution of *L. infantum* detected events (2021–2025), as reported to WOAH.

L. infantum has been detected across almost all continents with reports in the EU in the 5 years (Figure 26). Up-to-date maps based on WAHIS data are available online.[17]

[17]Online version: https://animal-diseases.efsa.europa.eu/Linfantum.

3.15.4 | Animal hosts

Susceptible hosts

Based on epidemiological knowledge of host–pathogen–vector interactions and outbreak reports, the main hosts of *L. infantum* are lagomorphs, canids and cats, whereas equids are considered as dead-end hosts. However, other species have been identified in the SLR. The SLR summary is given in Table 23.

TABLE 23 Susceptible hosts of *Leishmania infantum*.

The SLR reported in the *L. infantum* disease profile, identified the following susceptible species (updated until 31 December 2025; for references, see Annex A)

FIELD

Epidemiological studies carried out in the field

- **Pathogen was detected in the following animal species:**
- Bovidae: *Capra hircus, Ovis aries*
- Canidae: *Canis lupus familiaris*
- Equidae: *Equus caballus*
- Felidae: *Felis catus, Lynx pardinus*
- Herpestidae: *Suricata suricatta*
- Hominidae: *Homo sapiens*
- Leporidae: *Lepus granatensis*
- Phasianidae: *Gallus gallus domesticus*
- Procyonidae: *Nasua nasua*
- **Antibodies were detected in the following animal species:**
- Bovidae: *Ovis aries, Bison bonasus*
- Canidae: *Canis lupus familiaris*
- Equidae: *Equus caballus*
- Felidae: *Felis catus, Lynx pardinus*
- Hominidae: *Homo sapiens*
- Leporidae: *Lepus granatensis, Lepus europaeus*
- Mustelidae: *Mustela lutreola*
- Procyonidae: *Nasua nasua*
- Suidae: *Sus scrofa domesticus*
- **Outbreaks reported to WOAH included the following species:**
- Canidae: *Canis lupus familiaris, Canis lupus*
- Felidae: *Felis catus*

EXPERIMENTS

- **Experimental studies demonstrated infection in:**
- Bovidae: *Ovis aries, Bos taurus*
- Canidae: *Canis lupus familiaris*
- Felidae: *Felis catus*

Clinical signs

Outcomes of a SLR of clinical signs in 74 dog study groups are displayed in Figure 27. Predominantly, dermatological and cardiovascular clinical signs were reported.

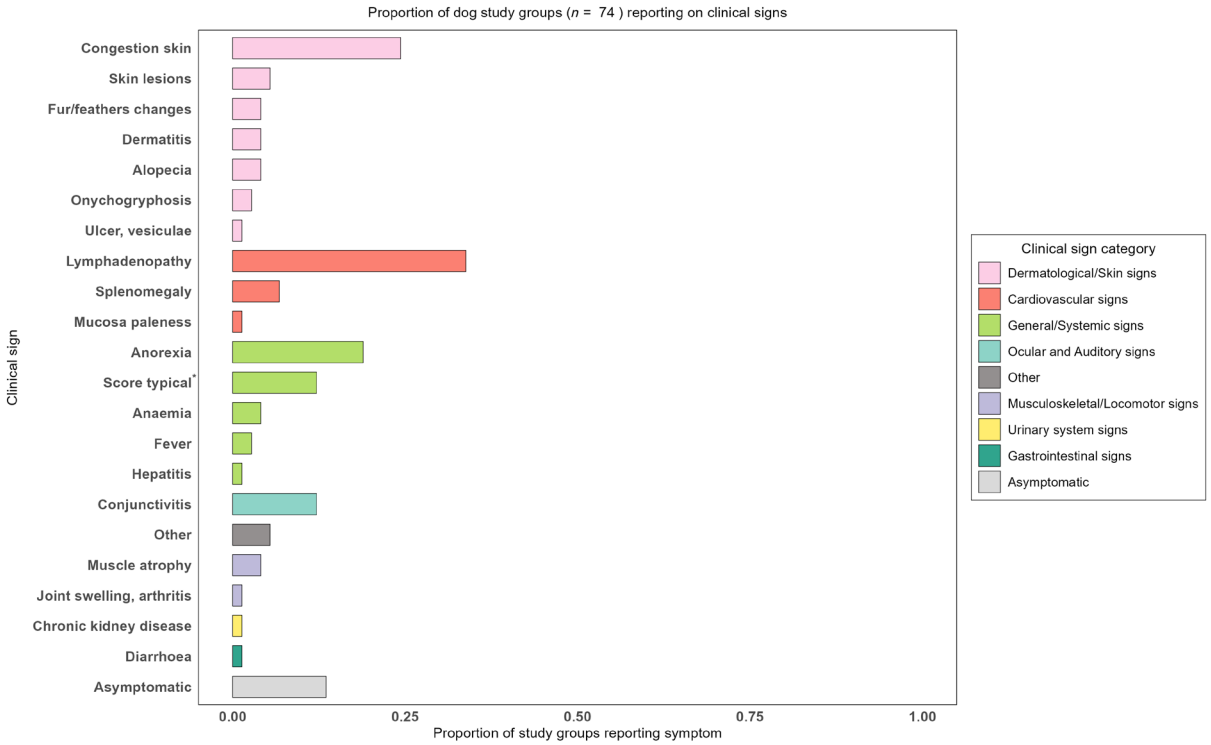


FIGURE 27 Clinical signs reported in the main hosts of *L. infantum*. *Score typical: weight loss, hair loss and skin lesions. Cat study groups not visualised due to small sample size ($n = 2$). The SLR was updated until 31 December 2025; for references, see Annex A.

Dogs are the most clinically affected species, which develop a chronic viscero-cutaneous disease known as canine leishmaniosis. The clinical outcome and progression of the disease result from complex interactions between the parasite and the host's immune system. The ability of host macrophages to effectively destroy the intracellular parasite is a critical determinant of the infection's course. Although the infection can be asymptomatic, both immune complex deposition and histiocytic reaction seem to be involved in a large range of symptoms:

- Skin: alopecia, dermatitis, skin lesions, mucosa paleness, coat changes, ulcer vesiculas and onychogryphosis.
- Kidneys: polydipsia, polyuria, kidney failure, vomiting (related to uraemia).
- Locomotion: joint swelling/arthritis, muscle atrophy.
- Digestive: diarrhoea, vomiting, blood in stool, weight loss, decreased appetite.
- Reproductive: in male, epididymitis and orchitis.
- Other: fever, conjunctivitis, ocular lesions, anaemia.

The median length of the clinical manifestation for dogs is 150 days, but results of experimental infections showed that it can last as long as 567 days.

Incubation period

The incubation period of leishmaniosis in dogs ranges from 28 to 540 days, with a median of approximately 3 months. The SLR identified experimental studies in cats, cattle, dogs and sheep (Annex A).

Morbidity and case fatality

Serological surveys included in the SLR reported percentage of seropositive animals of up to 30% in endemic areas (Annex A). The case fatality rate is around 10%, based on experimental infections (Annex A).

Zoonotic potential

Leishmaniosis is a zoonotic disease (WHO, 2023a).

3.15.5 | Transmission

Leishmaniosis is transmitted to vertebrate hosts through the bite of sandflies (Diptera; Phlebotominae). Evidence on the potential and likely competent vector species and their geographic distribution can be found in a dedicated Scientific Report on vectors (EFSA, 2026a).

Asymptomatic infection in dogs is widespread and contributes to maintaining the long-term presence of the parasite in endemic regions.

3.15.6 | Diagnostic tests

Recommended tests (WOAH, 2025b) for the detection of the agent are cytological examination, histological examination, classical isolation in culture and real-time PCR.

In animals exhibiting clinical signs and characteristic lesions, a definitive diagnosis is established by demonstrating parasites in stained smears of splenic, bone marrow or lymph node aspirates, skin scrapings or tissue biopsies. In low-grade infections, parasite detection requires in vitro isolation or PCR. Since morphological differences among *Leishmania* species are minimal, identification of isolates relies on biochemical and/or molecular methods. According to WOAH diagnostic manual, cytological examination shows high specificity, while sensitivity depends on the tissue examined: 93%–99% for spleen, 52%–85% for bone marrow and 52%–58% for lymph node aspirates. PCR sensitivity also varies with the sample, with bone marrow and lymph nodes being the most reliable, followed by skin, conjunctiva, buffy coat and peripheral blood.

Recommended tests (WOAH, 2025b) for the detection of immune response are immunofluorescence assay (IFA), ELISA, direct agglutination test and rapid immunochromatographic assay (RIA):

IFA is widely used because of its simplicity and genus-level specificity, although cross-reactions with *Trypanosoma cruzi* are common; in such cases, assays based on recombinant *Leishmania* antigens are preferable. Diagnostic performance reported in WOAH diagnostic manual includes a sensitivity of up to 96% and a specificity of 98%, though specificity decreases in areas endemic for Chagas disease. ELISA sensitivity varies with the antigen used (86%–99.5%), with specificity comparable to IFA (≈97%). Direct agglutination can reach 100% sensitivity and 98.9% specificity. In contrast, rapid immunochromatographic assays show medium to high specificity but low sensitivity (30%–70%). The rK39 dipstick achieves 97% sensitivity and 100% specificity in both symptomatic and asymptomatic dogs.

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

3.15.7 | Prevention and control

Vaccination

Two canine leishmaniasis vaccines (recombinant Q protein and bacterial DNA plasmid containing LACK gene) are commercially available in the EU (EMA, 2025).

Treatment

The SLR identified 21 pharmacological treatments. Meglumine antimonate and allopurinol were the most frequently reported therapeutic agents administered either as monotherapies, in combination with each other, or in association with additional compounds.

All references are available in the online version of the Disease Profiles and in Annex A.

3.16 | Lumpy skin disease virus (LSDV)[18]

3.16.1 | Disease overview

Lumpy skin disease virus (LSDV) causes lumpy skin disease (LSD), an arthropod-borne viral infection of domestic and wild bovines, primarily cattle and water buffaloes. The disease is characterised by fever, cutaneous nodules and lesions in the alimentary and respiratory tract, often accompanied by enlarged lymph nodes and oedema. Clinical outcomes range from subclinical infection to severe, sometimes fatal, systemic disease (Spickler, 2025a; WOAH, 2017a, 2025b).

Lumpy skin disease is a WOAH-notifiable disease, listed in the EU AHL under categories A, D and E.

3.16.2 | Agent

LSDV is a double-stranded DNA virus that belongs to the *Capripoxvirus* genus within the subfamily Chordopoxvirinae of the family Poxviridae. It is antigenically closely related to sheep and goatpox viruses. The virion is large, brick shaped and measures 293–299 nm in length and 262–273 nm in width. The central region of the genome contains open reading frames (ORFs) that encode proteins essential for virus replication and morphogenesis, while the more variable terminal regions show greater variability and encode proteins involved in virulence and host range. Phylogenetically, LSDV strains are broadly

[18]Online version: https://animal-diseases.efsa.europa.eu/LSDV.

divided into two main clusters. Cluster 1, comprising subclusters 1.1 and 1.2, includes field isolates from southern Africa, Kenya, the northern hemisphere, as well as vaccine strains. Cluster 2 contains recently identified recombinant viruses that contain genomic segments derived from both field and vaccine strains (Spickler, 2025a, 2025b; WOAH, 2017a, 2025b).

The virus can survive for a long time, up to months, in ambient temperatures protected from sunlight. In desiccated crusts, it can survive up to 35 days and in air-dried hides at least 18 days. LSDV is inactivated by sunlight, lipid solvents and several disinfectants (Spickler, 2025a, 2025b).

3.16.3 | Geographical distribution

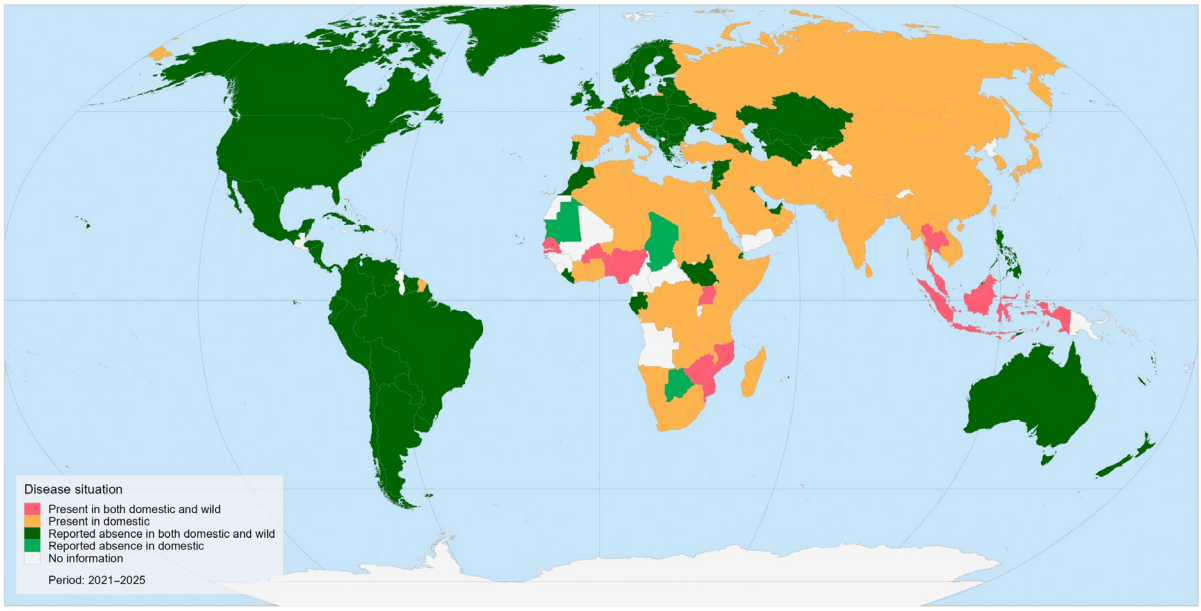


FIGURE 28 Geographical distribution of LSDV detected events (2021–2025), as reported to WOAH.

Lumpy skin disease is endemic in most African countries and Türkiye. According to WAHIS, outbreaks have also been reported from large parts of Asia and in France, Italy and Spain in the last 5 years (Figure 28). Up to date maps based on WAHIS are available in the online version of the Disease Profile.[18]

3.16.4 | Animal hosts

Susceptible species

Based on epidemiological knowledge of host–pathogen–vector interactions and outbreak reports, the main hosts of LSDV are cattle. However, other susceptible species have been identified in the SLR. The SLR summary is given in Table 24.

TABLE 24 Susceptible host species of lumpy skin disease virus.

The SLR reported in the LSDV disease profile, identified the following susceptible species (updated until 31 December 2025, for references, see Annex A)
FIELD
Epidemiological studies carried out in the field
• **Pathogen was detected in the following animal species:** • Bovidae: *Bos taurus, Capra hircus, Bubalus* spp.
• **Antibodies were detected in the following animal species:** • Bovidae: *Bos taurus, Syncerus caffer*
• **Outbreaks reported to WOAH included the following species:** • Bovidae: *Bos taurus, Bos frontalis, Bos gaurus, Bos javanicus, Capricornis sumatraensis, Bubalus* spp.
EXPERIMENTS
• **Experimental studies demonstrated infection in:** • Bovidae: *Bos taurus*

Clinical signs

Outcomes of a SLR on clinical signs in 23 cattle study groups are displayed in Figure 29. In most study groups, the clinical signs reported were predominantly generic and dermatological.

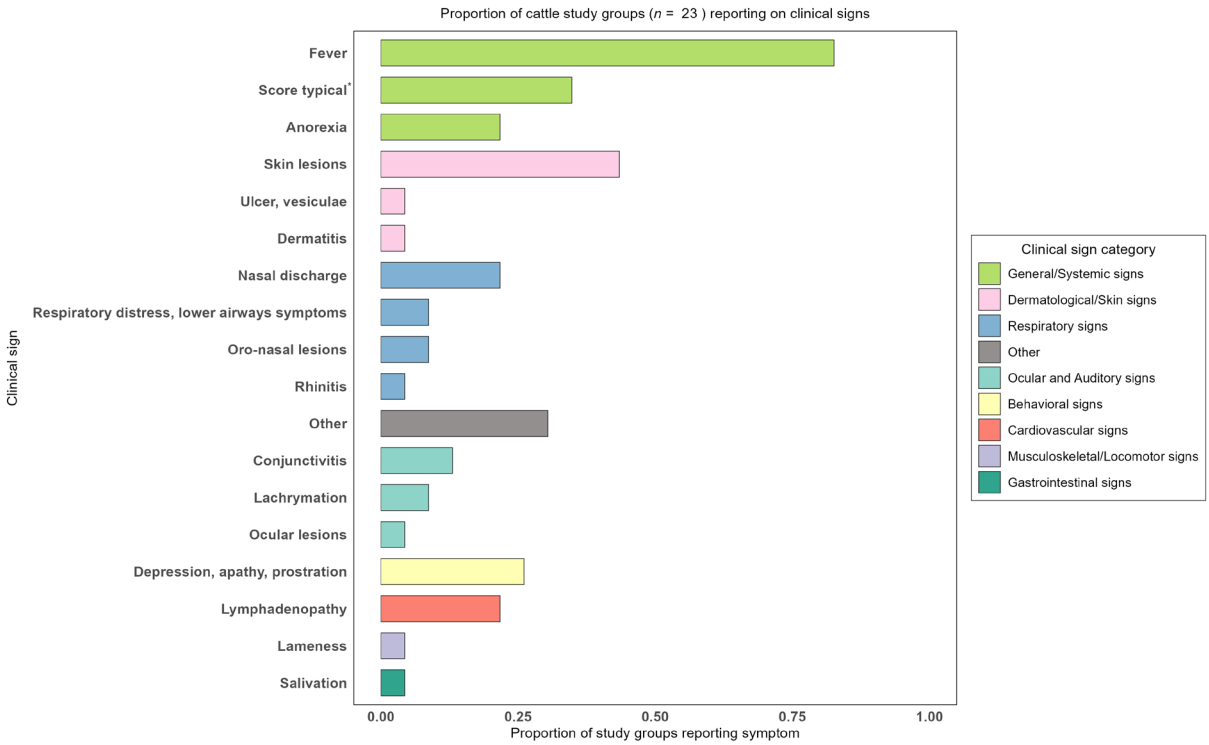


FIGURE 29 Clinical signs reported in the main hosts of LSDV. *Score typical: firm skin nodules. The SLR was updated until 31 December 2025; for references, see Annex A.

Clinical signs of lumpy skin disease in cattle range from subclinical to severe. Subclinical infections are difficult to detect, as affected animals may show only fever and enlargement of regional lymph nodes. Typical onset includes high fever (> 41°C), nasal discharge, conjunctivitis, reduced appetite, lethargy and decreased milk yield in lactating cows. Generalised lymphadenopathy is common. Cutaneous lesions usually appear within 48 h, most often on the head, neck, limbs, udder, genitalia and perineum. Nodules (0.5–8 cm) are initially firm, raised and well circumscribed, involving the epidermis, dermis, subcutis and sometimes underlying muscle. Early lesions may exude serum and show a grey-white cut surface. Many develop a necrotic, cone-shaped 'sit-fast'. Secondary bacterial infection is frequent, and healing may take months to years, often leaving scars. Mucosal lesions ulcerate quickly and may occur in the oral and nasal cavities, gastrointestinal tract, trachea and lungs, occasionally resulting in pneumonia. Pregnant cows may abort or deliver prematurely; bulls may experience temporary or permanent infertility. Ventral oedema (limbs, brisket, scrotum, vulva) may develop. Although most animals recover, convalescence can be prolonged due to emaciation and secondary infections.

Water buffalo seem to have less severe clinical signs than cattle. Similar signs of varying degree can be seen in various wild ungulates (Spickler, 2025a, 2025b).

Incubation period

The incubation period under field conditions is not well defined, but experimental infections in cattle indicate a range of 1–14 days, with a median of approximately 5 days. The incubation period in the field varies from 1 week up to 5 weeks. However, most sources state an incubation period of around 1 week (Spickler, 2025a, 2025b; WOAH, 2017a; Annex A).

Morbidity and case fatality

Morbidity rates in LSD outbreaks vary widely, ranging from 1%–2% to as high as 80%–90% in cattle (Spickler, 2025a, 2025b). The WOAH reports typical morbidity levels of 10%–20%. In experimentally infected cattle, case fatality rates average around 45% (range: 16%–50%) (Annex A).

Zoonotic potential

LSDV is not known to infect humans under natural conditions.

3.16.5 | Transmission

LSDV is primarily transmitted mechanically via biting midges of the genus *Culicoides* (Diptera; Ceratopogonidae), mosquitoes (Diptera; Culicidae) and biting flies (e.g. *Stomoxys calcitrans* and *Biomyia fasciata*), *Culicoides* midges, male ticks (*Riphicephalus appendiculatus* and *Amblyomma hebraeum*) and some non-biting flies. Evidence on the potential and likely competent vector species and their geographic distribution can be found in a dedicated Scientific Report on vectors (EFSA, 2026a).

The importance of different vectors probably varies in different areas because of their abundance and behaviour. LSDV in skin lesions is probably the main source of virus transmitted to arthropod vectors (Spickler, 2025a, 2025b; WOAH, 2017a, 2025b).

Direct contact between animals is considered to be a minor source of infection for cluster 1 LSDV. For cluster 2 LSDV; however, LSDV has infected cattle that shared an arthropod-free room without direct contact and no common feed and water troughs. Apart from skin lesions, LSDV can be shed from saliva, respiratory secretions, milk and semen. Cattle sharing water with cattle infected with cluster 1 have been infected with LSDV. Vertical transmission has been demonstrated experimentally through the transmission of infected semen during natural mating or artificial insemination (Akther et al., 2023).

Uterine transmission of LSDV is also possible (Spickler, 2025a, 2025b).

3.16.6 | Diagnostic tests

WOAH recommended tests (WOAH, 2025b) for agent detection are PCR, virus isolation (VI) and transmission electron microscopy (TEM).

VI is considered the reference standard test by WOAH. PCR is the preferred method for LSDV detection due to its rapidity and high sensitivity and can detect viral genome in skin lesions, saliva, nasal secretions, semen and blood. VI followed by PCR can confirm the presence of viable virus. TEM can identify poxviruses but cannot differentiate between poxvirus species.

For immune response detection, the WOAH recommended tests are virus neutralisation (VNT), western blot, immunofluorescence assay (specifically the indirect fluorescent antibody test, IFAT) and capripoxvirus ELISA.

Table 25 presents data on the sensitivity and specificity of diagnostic tests collected through SLR; reported values correspond to the median sensitivity and specificity when multiple studies investigated the same test and are only included when explicitly stated in the publications.

TABLE 25 Median sensitivity and specificity of tests to detect LSDV/LSDV antibodies reported in literature included in the systematic literature review.

Target	Test	Species	Sensitivity	*N* animal groups	Specificity	*N* animal groups	References
Antibody	C-ELISA	Cattle	–	–	99.2%	1	Milena et al. (2019)
Antibody	ELISA	Cattle	91%	1	87.0%	3	Milovanović et al. (2019)
Antibody	Immunofluorescence Assay	Cattle	88%	1	76%	1	
Antibody	VNT	Cattle	100%	1	100%	1	

Note: The SLR was updated until 31 December 2025; for references, see Annex A.

3.16.7 | Prevention and control

Vaccination

There are live-attenuated homologous and heterologous vaccines against LSDV. The homologous vaccines have been used successfully to control lumpy skin disease in the field and under experimental infection shown high levels of protection (WOAH, 2025b). The duration of immunity is at least 18 months. Side effects include a local reaction at the injection site, fever, reduced milk production and on rare occasions a 'Neethling' response. The heterologous live-attenuated vaccine is less effective in protecting cattle than the homologous (WOAH, 2025b).

Homologous inactivated vaccines against LSDV have been tested and developed. Although they are safe and tested, the duration of immunity is shorter. They require a booster vaccination after 1 month and then every 6 months (WOAH, 2025b).

Currently, there are no vaccines for LSDV authorised for regular veterinary use by the European Medical Agency, but the European Union keeps a vaccine bank for emergencies. During 2025, following the detection of outbreaks in Italy, France and Spain, OBP Neethling strain vaccines (Onderstepoort Biological Products) supplied from the EU LSD vaccine bank were used as part of the control strategy in all three countries (European Commission, 2025).

Treatment

In accordance with the EU AHL, susceptible species kept at in the affected holdings shall be culled to prevent further spread of the pathogen. Specific treatments for this disease are not compliant with the AHL.

3.17 | Rift Valley fever virus (RVFV)[19]

3.17.1 | Disease overview

Rift Valley fever virus (RVFV) causes Rift Valley fever (RVF), an infectious, non-contagious mosquito-borne viral disease. RVFV mainly affects domestic ruminants, particularly sheep, goats and cattle, and camelids. RVF causes significant economic losses due to abortion storms and high case fatality in young animals (Spickler, 2025a, 2025b; WOAH, 2019c, 2025b).

Rift Valley fever is a WOAH-notifiable disease, listed in the EU AHL under categories A, D and E.

3.17.2 | Agent

RVFV is an enveloped, single-stranded RNA virus that belongs to the *Phlebovirus* genus within the Phenuiviridae family. Its genome is segmented into three parts: the large (L), medium (M) and small (S) segments. The L segment encodes the RNA-dependent RNA polymerase, which is essential for viral replication. The M segment encodes two surface glycoproteins, Gn and Gc, which are involved in host cell attachment and membrane fusion. The S segment uses an ambisense coding strategy to produce both the nucleocapsid (N) protein and a non-structural protein (NSs). The N protein encapsidates the viral RNA, forming ribonucleoprotein complexes that are critical for genome stability and replication (Bouloy & Weber, 2010; Elliott, 1990; WOAH, 2019c, 2025b).

3.17.3 | Geographical distribution

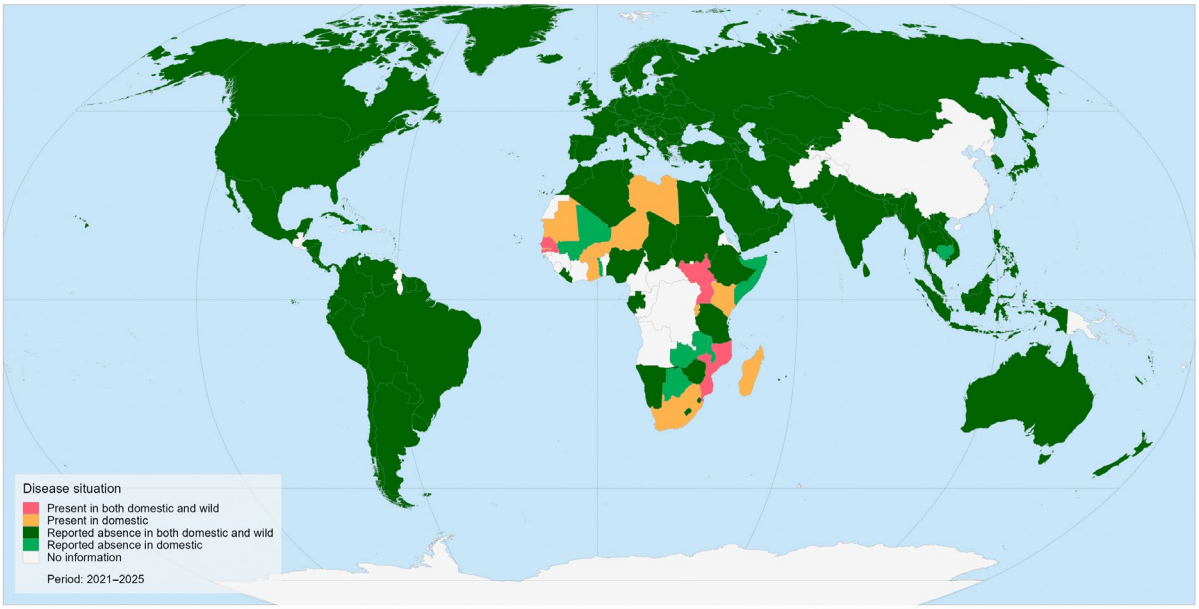


FIGURE 30 Geographical distribution of RVFV detected events (2021–2025), as reported to WOAH.

RVFV is historically endemic in sub-Saharan Africa, but outbreaks have occurred in the Middle East and the Indian Ocean Islands. The agent was not reported in the EU in the last 5 years (Figure 30). Up-to-date maps based on WAHIS are available in the online version of the Disease Profile.[19]

3.17.4 | Animal hosts

Susceptible species

Based on epidemiological knowledge of host–pathogen–vector interactions and outbreak reports, RVFV has a wide range of vertebrate hosts, with the most important being domestic ruminants and camelids, whereas humans are considered as dead-end hosts. The SLR on susceptible species summary is given in Table 26.

[19]Online version: https://animal-diseases.efsa.europa.eu/RVFV.

TABLE 26 Susceptible host species of Rift Valley Fever virus.

The SLR reported in the RVFV disease profile, identified the following susceptible species (updated until 31 December 2025; for references, see Annex A)
FIELD
Epidemiological studies carried out in the field
• **Pathogen was detected in the following animal species:** • Bovidae: *Bos taurus, Capra hircus, Ovis aries, Bos indicus, Syncerus caffer, Gazella dorcas* • Camelidae: *Camelus bactrianus, Camelus dromedarius* • Hominidae: *Homo sapiens* • Suidae: *Sus scrofa domesticus, Phacochoerus africanus*
• **Antibodies were detected in the following animal species:** • Bovidae: *Bos taurus, Capra hircus, Ovis aries, Bos indicus, Syncerus caffer, Bubalus* spp. • Camelidae: *Camelus bactrianus, Camelus dromedarius* • Equidae: *Equus caballus* • Hominidae: *Homo sapiens* • Phasianidae: *Gallus gallus domesticus* • Suidae: *Sus scrofa domesticus, Phacochoerus africanus*
• **Outbreaks reported to WOAH included the following species:** • Bovidae: *Bos taurus, Capra hircus, Ovis aries, Gazella dorcas, Bubalus* spp., *Hippotragus equinus, Oryx gazella* • Camelidae: No species specified • Leporidae: *Oryctolagus cuniculus*
EXPERIMENTS
• **Experimental studies demonstrated infection in:** • Bovidae: *Bos taurus, Capra hircus, Ovis aries, Bubalus* spp. • Camelidae: *Vicugna pacos* • Suidae: *Sus scrofa domesticus*

Clinical signs

Outcomes of a SLR on clinical signs in 41 ruminant study groups are displayed in Figure 31. Most study groups reported generic and reproductive clinical signs.

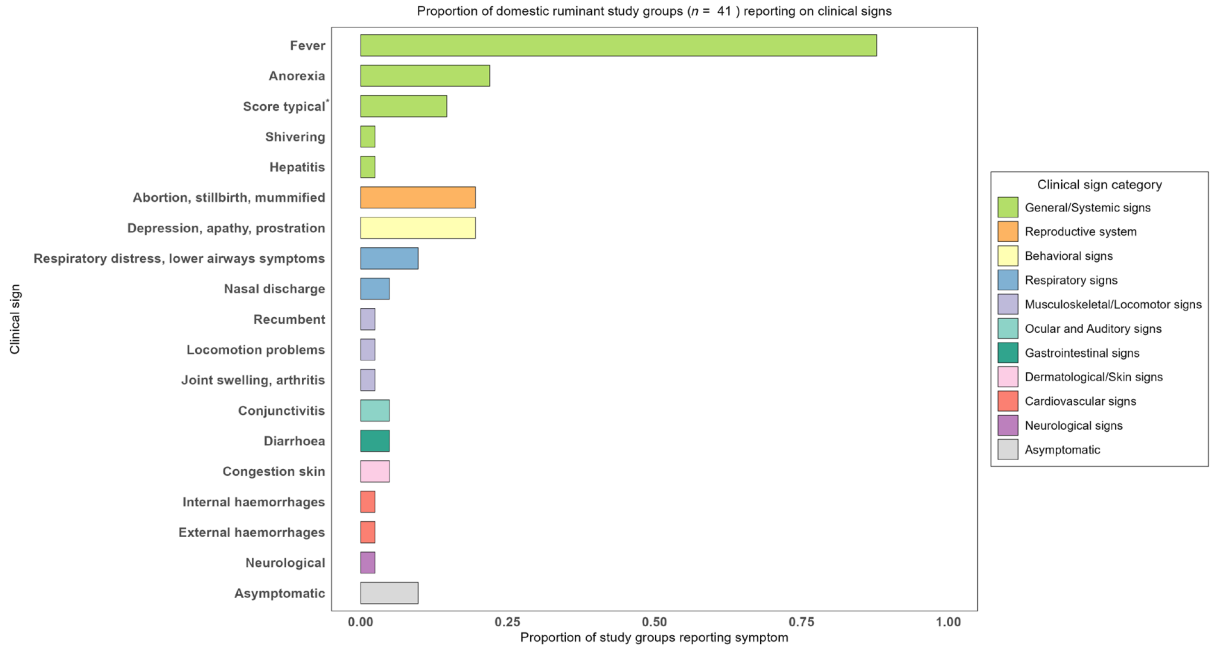


FIGURE 31 Clinical signs reported in the main hosts of RVFV. *Score typical: abortion storms. Study group count per domestic ruminant species: Sheep $n = 29$; Cattle $n = 7$; Goat $n = 4$; Buffalo $n = 1$. Camelid study groups not visualised due to small sample size (Alpaca $n = 1$). The SLR was updated until 31 December 2025; for references, see Annex A.

Clinical signals and severity can vary according to species, age and whether the animal is pregnant. Sheep are the most affected species with clinical signs duration of 4 days, followed by goats and cattle (3.5 and 1.5 days, respectively).

Young lambs and kids are the most susceptible individuals. Adult sheep and calves are highly susceptible as well. In general, RVFV has trophism for several cells in different organs, and the most observed lesion is multifocal, randomly distributed necrosis leading to a wide range of unspecific symptoms.

In naïve sheep and goats, RVFV can cause several non-specific clinical signs, including fever, lethargy, anorexia, nasal discharge and diarrhoea. Young animals are particularly vulnerable and may die suddenly. Pregnant animals often experience abortion, and newborns may be stillborn or die within a few days. In severe cases, hepatic necrosis can lead to jaundice and sudden death.

In cattle and camels, most RVFV-infected individuals show mild or subclinical signs. Fever, decreased milk production and anorexia may be observed. Pregnant animals may also abort, and neonatal mortality can occur. In rare cases, severe hepatic disease can develop, leading to jaundice, weakness and death.

In wild ruminants and other susceptible species, RVF is usually subclinical but can cause reproductive losses and occasional mortality during outbreaks involving highly virulent strains.

Other species can be infected but are resistant and do not show any clinical signs (Spickler, 2025a, 2025b; WOAH, 2019c; Annex A).

Incubation period

The incubation period of RVFV mostly varies according to the species. Experimental studies identified in the SLR showed a median incubation time of 2 days in sheep, goats and cattle, but up to 16 days in buffaloes. Clinical signs lasted up to 2 weeks (Annex A).

Morbidity and case fatality

Morbidity rates are generally high in susceptible livestock populations, often approaching 90–100%. Case fatality varies by species, age and breed, typically ranging from 3% to 20% in adult animals, but may reach as high as 100% in young lambs, kids or calves infected with highly virulent strains. Neonatal mortality is particularly severe, with young animals frequently succumbing within a few days of infection. Pregnant females are prone to abortion, with rates that can exceed 90% in naïve populations (Spickler, 2025a, 2025b; WOAH, 2019c; Annex A).

Zoonotic potential

Rift Valley fever is a zoonotic disease (WHO, 2024b).

3.17.5 | Transmission

RVFV is transmitted by mosquitoes (Diptera; Culicidae). Evidence on the potential and likely competent mosquito species and their geographic distribution can be found in a dedicated Scientific Report on vectors (EFSA, 2026a). Transmission can occur vertically in mosquitoes (transovarial transmission) or via iatrogenic routes through exposure to infected tissues or fluids, independent of the vector (Spickler, 2025a, 2025b; WOAH, 2019c).

3.17.6 | Diagnostic tests

Recommended tests (WOAH, 2025b) for the detection of the agent: Virus isolation, RT-PCR, ELISA for antigen detection and histopathology with immunohistochemistry.

Recommended tests (WOAH, 2025b) for the detection of immune response: ELISA, virus neutralisation test (VNT) and plaque reduction neutralisation test (PRNT).

Table 27 presents data on the sensitivity and specificity of diagnostic tests collected through SLR; reported values correspond to the median sensitivity and specificity when multiple studies investigated the same test and are only included when explicitly stated in the publications.

TABLE 27 Median sensitivity and specificity of tests to detect RVFV/RVFV antibodies reported in literature included in the systematic literature review.

Target	Test	Species	Specificity	*N* animal groups	Sensitivity	*N* animal groups	References
Antigen	B-ELISA	Cattle	99.0%	3	92.9%	3	Selvan et al. (2021)
Antigen	I-ELISA	Buffalo	98.3%	1	94.4%	1	Paweska, Smith, et al. (2003)
Antigen	I-ELISA	Cattle	98.1%	3	92.1%	3	Mohapatra et al. (2014); Pawęska et al. (2021)
Antigen	I-ELISA	Goat	99.2%	1	99.2%	1	Pawęska et al. (2021)
Antigen	I-ELISA	Sheep	99.2%	1	98.9%	1	

(Continues)

TABLE 27 (Continued)

Target	Test	Species	Specificity	*N* animal groups	Sensitivity	*N* animal groups	References
Nucleic acid	Real Time RT-PCR	Cattle	71.7%	1	97.4%	1	Odendaal et al. (2014)
Virus	Histopathology	Cattle	92.3%	1	94.6%	1	
Virus	Immuno-histochemistry	Cattle	99.4%	1	97.6%	1	
Virus	PRNT	Cattle	100.%	2	100.%	2	Upreti et al. (2018)
Virus	PRNT	Sheep	100.%	2	100.%	2	
Antibody	C-ELISA	Cattle	100%	14	95.1%	14	Upreti et al. (2018); Kortekaas et al. (2013); Paweska et al. (2005)
Antibody	C-ELISA	Goat	99.7%	1	99.6%	1	Paweska et al. (2005)
Antibody	C-ELISA	Sheep	95.6%	2	97.6%	2	Upreti et al. (2018); Paweska et al. (2005)
Antibody	Haemagglut-ination inhibition test (HIT)	Buffalo, Goat, Sheep, Cattle	100%	1	100%	1	Paweska, Smith, et al. (2003)
Antibody	I-ELISA	Cattle	96.6%	10	77.0%	10	Kortekaas et al. (2013); Fafetine et al. (2007); Paweska, Smith, et al. (2003)
Antibody	I-ELISA	Goat	98.7%	6	99.7%	6	Jäckel et al. (2013); Fafetine et al. (2012); Fafetine et al. (2007); Paweska, Smith, et al. (2003)
Antibody	I-ELISA	Sheep	98.8%	4	99.3%	4	Fafetine et al. (2012); Fafetine et al. (2007); Paweska, Smith, et al. (2003)
Antibody	Sandwich ELISA	Cattle	100%	3	100%	1	Pépin et al. (2010); Paweska, Burt, et al. (2003)
Antibody	Sandwich ELISA	Goat	99.8%	4	98.2%	2	Fafetine et al. (2012); Pépin et al. (2010); Paweska, Burt, et al. (2003)
Antibody	Sandwich ELISA	Sheep	80%	17	100%	15	Fafetine et al. (2012); Pépin et al. (2010); Paweska, Burt, et al. (2003)
Antibody	VNT	Buffalo	100%	1	100%	1	Paweska, Smith, et al. (2003)
Antibody	VNT	Cattle	100%	6	97.7%	6	Fafetine et al. (2007); Paweska et al. (2005); Paweska, Smith, et al. (2003); Selvan et al. (2021)
Antibody	VNT	Goat	100%	4	100%	4	Jäckel et al. (2013); Fafetine et al. (2007); Paweska et al. (2005); Paweska, Smith, et al. (2003)
Antibody	VNT	Sheep	100%	3	100%	3	Fafetine et al. (2007); Paweska et al. (2005); Paweska, Smith, et al. (2003)

Note: The SLR was updated until 31 December 2025; for references, see Annex A.

3.17.7 | Prevention and control

In accordance with the EU AHL, susceptible species kept at the affected holdings shall be culled to prevent further spread of the pathogen.

Vaccination

Commercial vaccines are available and used in Africa. Currently, there are no vaccines for RVFV authorised for veterinary use within the EU.

Treatment

There is currently no specific treatment for RVF infection (Spickler, 2025a, 2025b; WOAH, 2019c).

3.18 | Schmallenberg virus (SBV)[20]

3.18.1 | Disease overview

Schmallenberg virus (SBV) causes Schmallenberg disease, an infectious, non-contagious, *Culicoides*-borne viral disease. The main hosts are domestic and wild ruminants. Clinical presentation is generally mild, with reported signs including fever, anorexia and abortion (WOAH, 2017b, 2025b).

Schmallenberg disease is *not* a WOAH-notifiable disease, and it is not listed in the EU AHL.

3.18.2 | Agent

SBV is an enveloped, negative-sense, segmented, single-stranded RNA virus. It is classified within the Bunyaviridae family and the *Orthobunyavirus* genus. Members of the Bunyaviridae family are enveloped single-stranded negative-sense RNA viruses of 90- to 100-nm diameter with a genome segmented in three fragments. The latter are named according to their size, with 'L', 'M' and 'S' packages standing for large, medium and small segments, respectively (Garigliany et al., 2012). The Schmallenberg virus is a member of the Simbu serogroup viruses, which includes viruses such as Shamonda, Akabane and Aino. Investigations when Schmallenberg emerged showed that the M segment of the Sathuperi and Douglas orthobunyaviruses displayed higher identity with SBV, whereas the S and L segments were closer to Shamonda virus, suggesting that SBV was a reassortant virus between Sathuperi and Shamonda viruses (EFSA, 2014; Hoffmann et al., 2012; Wernike et al., 2014).

3.18.3 | Geographical distribution

SBV was first identified in Germany in 2011 and rapidly spread across much of Europe. Since then, circulation has been documented in multiple EU Member States and surrounding regions. Sporadic re-emergence continues, often linked to vector activity and climatic conditions. Since then, SBV has been reported in the Middle East, Africa and parts of Asia.

SBV infection is no longer reportable to WOAH. Evidence from published studies describing natural infections with this agent, as well as field epidemiological studies, is collected in the SLR (updated until 31/12/2025) and summarised in Figure 32. All references can be found in Annex A and in the online version of the Disease Profile.[20]

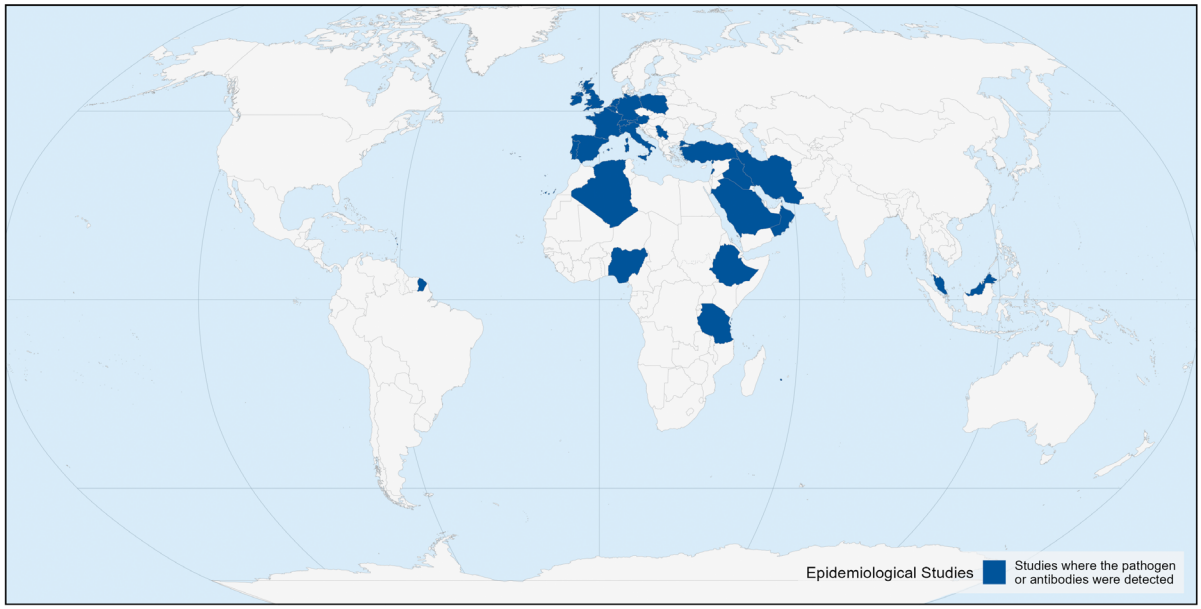


FIGURE 32 Geographical distribution of epidemiological studies addressing the occurrence of SBV, as identified by EFSA's SLR (covering years 1970–2025).

[20]Online version: https://animal-diseases.efsa.europa.eu/SBV.

3.18.4 | Animal hosts

Susceptible hosts

Based on epidemiological knowledge of host–pathogen–vector interactions and outbreak reports, the main hosts of SBV are ruminants. However, other susceptible species have been identified in the SLR. The SLR summary is given in Table 28.

TABLE 28 Susceptible hosts of Schmallenberg virus.

The SLR reported in the SBV disease profile, identified the following susceptible species (updated until 31 December 2025; for references, see Annex A)
FIELD
Epidemiological studies carried out in the field
• **Pathogen was detected in the following animal species:** • Bovidae: *Bos taurus, Capra hircus, Ovis aries* • **Antibodies were detected in the following animal species:** • Bovidae: *Bos taurus, Capra hircus, Ovis aries, Bison bison, Bison bonasus, Ovis orientalis, Rupicapra rupicapra* • Camelidae: *Vicugna pacos, Lama glama* • Cervidae: *Cervus elaphus, Dama dama, Rangifer tarandus, Capreolus capreolus, Cervus nippon* • Equidae: *Equus caballus* • **Outbreaks reported to WOAH included the following species:** • Bovidae: *Bos taurus, Capra hircus, Ovis aries*
EXPERIMENTS
• **Experimental studies demonstrated infection in:** • Bovidae: *Bos taurus, Capra hircus, Ovis aries* • Camelidae: *Vicugna pacos, Lama glama* • Phasianidae: *Gallus gallus domesticus*

Clinical signs

Outcomes of a SLR of clinical signs in 19 domestic ruminant study groups are displayed in Figure 33. Predominantly, the absence of clinical signs was reported in the study groups.

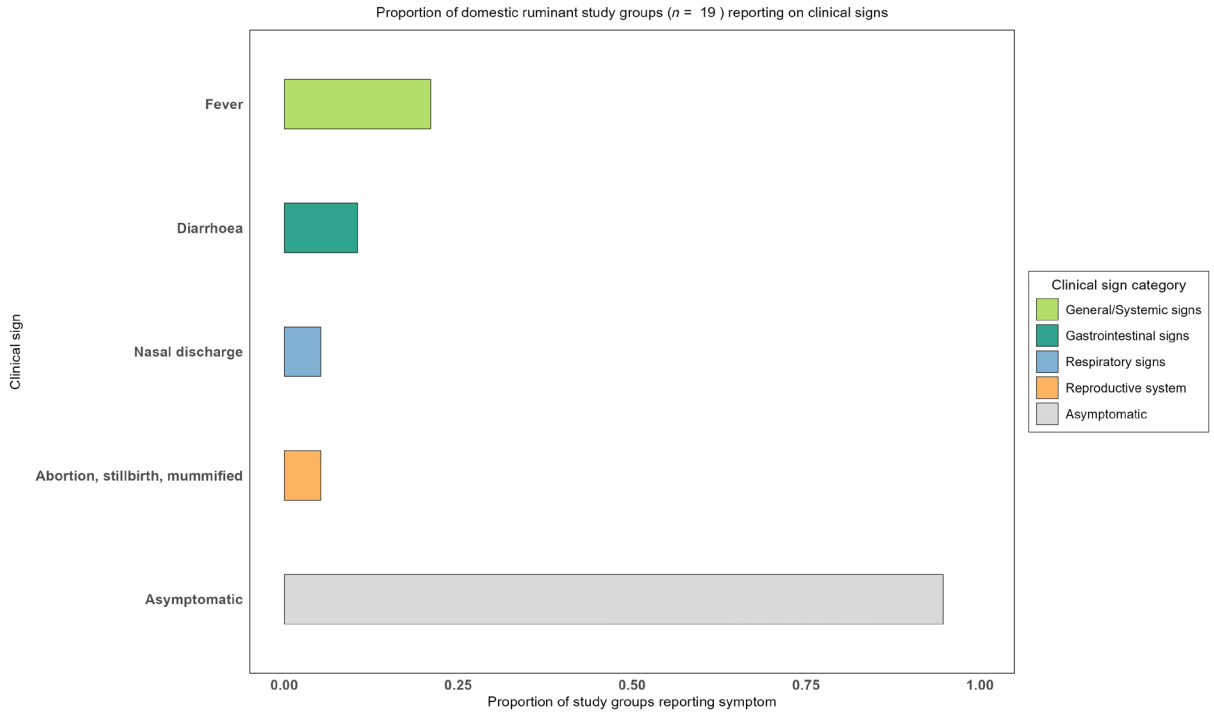


FIGURE 33 Clinical signs reported in the main hosts of SBV. Study group count per domestic ruminant species: Sheep $n = 11$; Cattle $n = 6$; Goat $n = 2$. The SLR was updated until 31 December 2025; for references, see Annex A.

Experimental infection in cattle and sheep generally resulted in no or only mild clinical signs. The presentation of clinical signs varies depending on species and age.

Adult ruminants typically exhibit a subclinical or mild acute disease, including fever, anorexia, diarrhoea, and decreased milk production. However, infection during pregnancy is associated with abortions, stillbirths and congenital malformations. Diarrhoea has also been documented in some instances within both cattle and sheep populations.

In neonates, congenital SBV infection can be associated with arthrogryposis, hydranencephaly, scoliosis, brachygnathia and other malformations. Affected offspring are often stillborn or die shortly after birth.

Incubation period

The incubation period of SBV mostly varies from 1 to 8 days post infection. Experimental studies included in the SLR report a median incubation time of 4–5 days in sheep and cattle. Experimental studies were also conducted in chicken, alpaca, goats and llama (Annex A).

Morbidity and case fatality

Morbidity in adult animals is generally low, with transient disease. Case fatality is primarily associated with affected neonates, particularly lambs, where losses can be high during outbreaks (WOAH, 2017b; Annex A).

Zoonotic potential

SBV is not known to infect humans under natural conditions.

3.18.5 | Transmission

SBV is transmitted to vertebrate hosts through the bite of certain species of *Culicoides* midges (Diptera; Ceratopogonidae). Vertical transmission in utero is the primary route leading to congenital disease. There is no evidence of direct horizontal transmission between animals (EFSA, 2014; Hoffmann et al., 2012; Wernike et al., 2014).

Evidence on the potential and likely competent *Culicoides* species and their geographic distribution can be found in a dedicated Scientific Report on vectors (EFSA, 2026a).

3.18.6 | Diagnostic tests

Recommended tests (WOAH, 2025b) for agent detection are virus isolation, immunofluorescence assay, immunohistochemistry (IHC), virus neutralisation test (VNT) and real-time RT-PCR.

Recommended tests (WOAH, 2025b) for the detection of immune response are ELISA and virus neutralisation test (VNT).

The infectious virus can be isolated using cell culture techniques. A variety of cell lines, including insect cells (KC and C6/36), hamster cells (BHK) and monkey kidney cells (Vero) have been successfully employed for this purpose.

Due to the non-specific nature of clinical signs, a differential diagnosis is necessary. For acute infections in adult animals, potential causes of high fever, diarrhoea and reduced milk production should be investigated. In cases of malformations of calves, lambs and kids, consideration should be given to other orthobunyaviruses, bluetongue virus, pestiviruses, genetic factors and exposure to toxic substances.

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

3.18.7 | Prevention and control

Vaccination

The EU has one inactivated vaccine available for commercial use in cattle and sheep.

Treatment

There is currently no specific treatment for Schmallenberg virus. Management focuses on supportive care for adults and preventing pregnancies during peak vector activity.

3.19 | Shuni virus (SHUV)[21]

3.19.1 | Disease overview

Shuni virus (SHUV) causes Shuni virus infection, an infectious, non-contagious insect-transmitted viral disease. The main hosts are domestic and wildlife ruminants and horses. Clinical signs include fever and neurological symptoms that can lead to death (McIntosh, 1980; Möhlmann et al., 2018; Steyn et al., 2020; van Eeden et al., 2012; WOAH, 2025b).

Shuni virus infection is *not* a WOAH-notifiable disease, and it is not listed in the EU AHL.

3.19.2 | Agent

SHUV is a negative-sense single-stranded RNA virus with a segmented genome. SHUV is a member of the Simbu serogroup, belonging to the genus *Orthobunyavirus* within the family Peribunyaviridae. SHUV is a spherical virus with a diameter of about 100 nm. The RNA genome encodes for four structural and two non-structural proteins. The small (S) genomic segment encodes for the nucleocapsid protein N and in an overlapping reading frame for the non-structural protein NSs. The medium (M) segment encodes for the glycoproteins Gn and Gc and the non-structural protein NSm. The large (L) segment encodes for the RNA-dependent RNA polymerase (McIntosh, 1980; Möhlmann et al., 2018; Steyn et al., 2020; van Eeden et al., 2012; WOAH, 2025b).

3.19.3 | Geographical distribution

SHUV virus infection is not reportable to WOAH. It has been reported from South Africa and Middle East countries. Evidence from published studies describing natural infections with this agent as well as field epidemiological studies are collected in the SLR and summarised in Figure 34. All references can be found in Annex A and in the online version of the Disease Profile.[21]

FIGURE 34 Geographical distribution of epidemiological studies addressing the occurrence of SHUV, as identified by EFSA's SLR (covering years 1970–2025).

3.19.4 | Animal hosts

Susceptible hosts

Based on epidemiological knowledge of host–pathogen–vector interactions and outbreak reports, the main hosts of SHUV are domestic ruminants and horses, whereas humans are considered as dead-end hosts. However, other susceptible species have been identified in the SLR. The SLR summary is given in Table 29.

[21]Online version: https://animal-diseases.efsa.europa.eu/SHUV.

TABLE 29 Susceptible host species of Shuni virus.

The SLR reported in the SHUV disease profile, identified the following susceptible species (updated until 31 December 2025; for references, see Annex A)
FIELD
Epidemiological studies carried out in the field
• **Pathogen was detected in the following animal species:** • Bovidae: *Bos taurus, Capra hircus, Ovis aries* • Equidae: *Equus caballus*
• **Antibodies were detected in the following animal species:** • No species specified
• **Outbreaks reported to WOAH included the following species:** • No species specified
EXPERIMENTS
• **Experimental studies demonstrated infection in:** • Bovidae: *Bos taurus, Ovis aries*

Clinical signs

In adult ruminants, infection with SHUV can be asymptomatic or may only cause mild clinical signs such as fever and diarrhoea. In horses and in calves, SHUV infection can cause fever and neurological signs, such as paralysis, ataxia and recumbency, and can lead to death.

In ruminants, abortion and malformation of fetuses have also been reported.

Incubation period

The incubation period in experimentally infected calves was 4 days (Sick et al., 2021; Annex A).

Morbidity and case fatality

No studies were found where the morbidity and case fatality of SHUV infections were assessed.

Zoonotic potential

Shuni virus infection is a zoonotic disease (Motlou & Venter, 2021).

3.19.5 | Transmission

SHUV is likely transmitted to vertebrate hosts through the bite of mosquitoes (Diptera; Culicicidae) and biting midges of the genus *Culicoides* (Diptera; Ceratopogonidae). Evidence on the potential and likely competent *Culicoides* species and their geographic distribution can be found in a dedicated Scientific Report on vectors (EFSA, 2026a).

Infected animals develop a viraemia, which lasts 3–4 days, and the virus can be acquired by vectors during blood meals. Infected vertebrates do not transmit the virus directly to humans (McIntosh, 1980; Möhlmann et al., 2018; Steyn et al., 2020; van Eeden et al., 2012; WOAH, 2025b).

3.19.6 | Diagnostic tests

There are no WOAH-recommended tests for SHUV specifically, but there are recommendations for the detection of Bunyaviruses (WOAH, 2025b):

Tests for detection of the agent are RT-PCR for orthobunyaviruses or Simbu serogroup viruses (this PCR needs to be followed by confirmation for SHUV by sequencing the PCR fragment), nested PCR with SHUV specific primers, virus isolation, infection of mice followed by virus isolation (and confirmation by PCR).

Viruses of the Simbu group, to which SHUV belongs, can be isolated very well from animals or vectors during viraemia, but isolation from pathological material is very difficult.

Tests for detection of immune response: Tests described for detection of antibodies are (indirect) immunofluorescence tests, complement fixation tests and (commercial) ELISA's. Most of these tests detect antibodies against the Simbu serogroup, but there is considerable cross-reactivity between the different viruses of the serogroup (Aino, Peaton, Schmallenberg, Shamonda and Tinaroo viruses), especially for tests that rely on the N protein. Serum neutralisation tests,

which detect neutralising antibodies directed against the glycoproteins, are more specific for a given virus species, but are labour-intensive. A more specific ELISA, based on the Gc-protein, was also developed.

The SLR has not found diagnostic tests evaluation studies meeting the eligibility criteria for inclusion.

3.19.7 | Prevention and control

Vaccination

There are currently no licensed vaccines for SHUV in animals.

Treatment

There is currently no specific antiviral treatment for SHUV infection management; it is primarily supportive on an individual basis.

3.20 | Saint Louis encephalitis virus (SLEV)[22]

3.20.1 | Disease overview

St. Louis encephalitis virus (SLEV) causes St. Louis encephalitis, a mosquito-borne viral disease maintained in an enzootic cycle between *Culex* mosquitoes and wild birds, which serve as amplifying hosts. In birds, infection is typically asymptomatic, though seroconversion is common in endemic areas. While humans are dead-end hosts and most infections are asymptomatic or mild, the virus can rarely cause severe, potentially fatal neuroinvasive disease, especially in older adults.

St. Louis encephalitis is *not* a WOAH-notifiable disease, and it is not listed in the EU AHL.

3.20.2 | Agent

SLEV is an enveloped, single-stranded, positive-sense RNA virus that belongs to the *Flavivirus* genus of the Flaviviridae family. The virion is spherical, 40–60 nm in diameter and carries a single RNA strand that is translated into one large polyprotein. This is then cut into the virus's structural components: capsid (C), premembrane/membrane (prM/M) and envelope (E). The E protein plays a key role in attaching to host cells and is the main target of neutralising antibodies.

The virus is moderately stable under ambient conditions but is readily inactivated by heat, lipid solvents, detergents and standard disinfectants (Fang, Brault and Reisen, 2009).

3.20.3 | Geographical distribution

SLEV is distributed widely across the Americas, from southern Canada to Argentina. SLEV 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 SLR and summarised in Figure 35. All references can be found in Annex A and in the online version of the Disease Profile.[22]

[22]Online version: https://animal-diseases.efsa.europa.eu/SLEV.

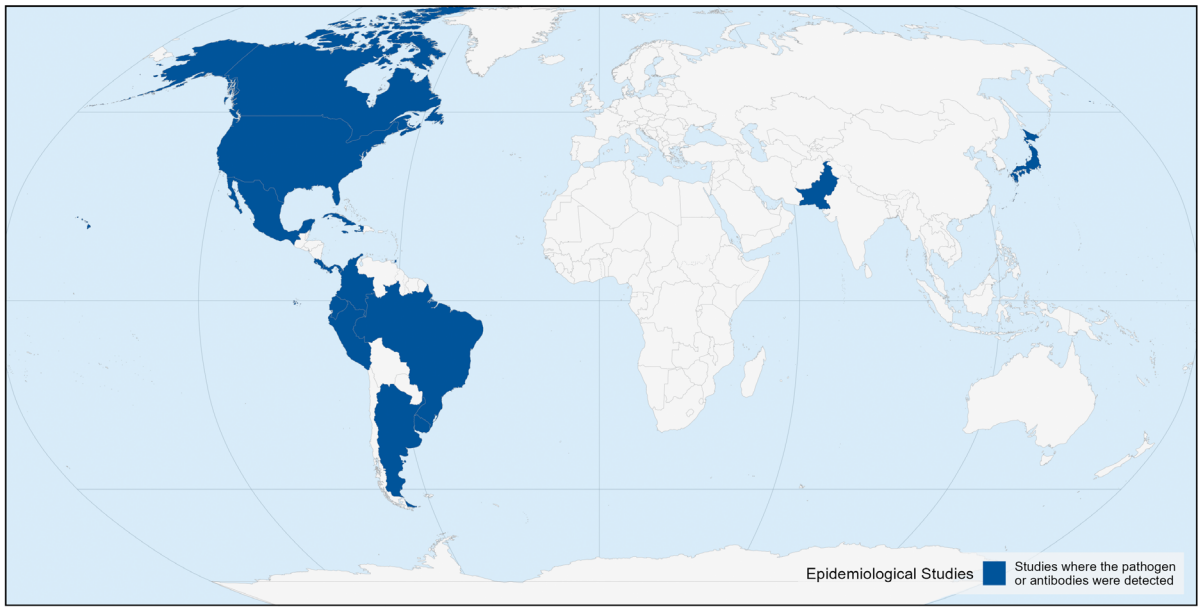


FIGURE 35 Geographical distribution of epidemiological studies addressing the occurrence of SLEV, as identified by EFSA's SLR (covering years 1970–2025).

3.20.4 | Animal hosts

Based on epidemiological knowledge of host–pathogen–vector interactions and outbreak reports, the main hosts of SLEV are wild birds, whereas humans and horses are considered as dead-end hosts. However, other susceptible species have been identified in the SLR (Table 30).

TABLE 30 Susceptible host species of Saint Louis encephalitis virus.

The SLR reported in the SLEV disease profile, identified the following susceptible species (updated until 31 December 2025; for references, see Annex A)

FIELD

Epidemiological studies carried out in the field

- **Pathogen was detected in the following animal species:**
  - Canidae: *Canis latrans, Vulpes vulpes*
  - Cervidae: *Odocoileus hemionus, Odocoileus virginianus*
  - Equidae: *Equus caballus*
  - Hominidae: *Homo sapiens*
  - Mephitidae: *Mephitis mephitis*
  - Phasianidae: *Gallus gallus domesticus*
  - Procyonidae: *Procyon lotor*
- **Antibodies were detected in the following animal species:**
  - Accipitridae: No species specified
  - Anatidae: No species specified
  - Bovidae: *Bos taurus, Capra hircus, Bubalus bubalis*
  - Canidae: *Canis lupus familiaris*
  - Cervidae: *Odocoileus virginianus*
  - Columbidae: *Columba livia domestica*
  - Equidae: *Equus caballus*
  - Falconidae: No species specified
  - Hominidae: *Homo sapiens*
  - Phasianidae: *Gallus gallus domesticus, Meleagris gallopavo*
  - Strigidae: No species specified
- **Outbreaks reported to WOAH included the following species:**
  - No species specified

EXPERIMENTS

- **Experimental studies demonstrated infection in:**
  - Phasianidae: *Gallus gallus domesticus*
  - Anatidae: No species specified
  - Columbidae: *Columba livia domestica*
  - Canidae: *Canis lupus familiaris*
  - Suidae: *Sus scrofa domesticus*

Clinical signs

SLEV primarily affects birds, which usually do not show clinical signs. Clinical illness in mammals is less common and asymptomatic. The only potential exception noted in research is the isolation of SLEV from a grey fox with encephalitis, suggesting that in rare cases, other mammals may develop clinical signs (Swanepoel & Burt, 2008).

Incubation period

The incubation period in vertebrate hosts is poorly known, as most infected animals remain subclinical. Serological surveys in horses frequently detect neutralising antibodies, yet molecular studies rarely identify active SLEV infection or virus in blood or tissue, consistent with low-level or transient viraemia and negligible outward signs of disease (Chalhoub et al., 2022; Silva & Romeiro, 2014).

In experimentally infected birds, viraemia typically develops within 1–4 days post-infection and lasts less than 5 days, providing a window for mosquito-mediated transmission (Monath et al., 1978; Reisen et al., 2003).

Morbidity and case fatality

Experimental infection studies in birds show that although many species become infected (i.e. seroconvert), clinical disease and mortality are rare or absent (Reisen et al., 2003).

Zoonotic potential

Saint Louis encephalitis is a zoonotic disease (CDC, 2024e).

3.20.5 | Transmission

SLEV is transmitted by mosquitoes (Diptera; Culicidae). Evidence on the potential and likely competent vector species and their geographic distribution can be found in a dedicated Scientific Report on vectors (EFSA, 2026a).

The virus is maintained in an enzootic cycle between mosquitoes and birds, which serve as the reservoir and amplifying hosts, while humans, horses and other mammals are considered dead-end hosts (Danforth et al., 2022). Transmission does not occur via direct contact between animals or through fomites. Seasonal patterns of human and animal infection generally correspond to peaks in mosquito activity, often during warm, wet months.

3.20.6 | Diagnostic tests

There are no WOAH-recommended tests specifically for SLEV, but a range of assays are used for the detection of flaviviruses in animals. Tests for detection of the agent include reverse transcription PCR (RT-PCR) targeting conserved regions of the SLEV genome, virus isolation in cell culture and inoculation of susceptible laboratory animals followed by confirmation of infection using PCR or immunohistochemistry (Howe et al., 1992). SLEV can be isolated most reliably from blood or tissues during the short period of viraemia in birds (Kramer et al., 2002); isolation from mammals is more difficult due to low-level and transient viraemia.

Tests for detection of the immune response include haemagglutination inhibition (HIA) assays, complement fixation (CF) tests and enzyme-linked immunosorbent assays (ELISAs). Most serological tests detect antibodies against flaviviruses broadly, and cross-reactivity is common, especially among closely related viruses such as West Nile virus (Chan et al., 2022). Serum neutralisation tests are more specific for SLEV albeit are labour-intensive.

The SLR has not found diagnostic tests evaluation studies meeting the eligibility criteria for inclusion.

3.20.7 | Prevention and control

Vaccination

There is no vaccine available for Saint Louis encephalitis. The infection in animals is usually asymptomatic or very mild, and they recover on their own.

Treatment

There is currently no specific antiviral treatment for St. Louis encephalitis.

3.21 | Tick-borne encephalitis virus (TBEV)[23]

3.21.1 | Disease overview

Tick-borne encephalitis virus (TBEV) causes tick-borne encephalitis, a zoonotic infectious, non-contagious, tick-borne viral disease. TBEV infects a wide range of hosts including rodents, wild and domestic ruminants, equines and dogs. While many domestic animals remain mostly asymptomatic, they play an important epidemiological role by serving as a potential source of human infection through close contact or consumption of raw milk (Bogovic & Strle, 2015; Lindquist & Vapalahti, 2008; Růžek et al., 2019; WOAH, 2020c).

Tick-borne encephalitis is *not* a WOAH-notifiable disease, and it is not listed in the EU AHL.

3.21.2 | Agent

TBEV is an enveloped, positive sense single-stranded RNA virus that belongs to the *Orthoflavivirus* genus of the Flaviviridae family. According to WOAH, there are five known virus subtypes: European (TBEV-Eu), Siberian (TBEV-Sib), Far Eastern (TBEV-Fe), Himalayan (TBEV-Him) and Baikalian (TBEV-Bkl). The genome of TBEV encodes three structural (capsid (C), membrane (M) and envelope (E) proteins) and seven non-structural (NS) proteins (NS1, NS2A, NS2B, NS3, NS4A, NS4B and NS5). The E protein is the main protein inducing a humoral response and is the main antigen used for serological testing (Bogovic & Strle, 2015; Lindquist & Vapalahti, 2008; Růžek et al., 2019; WOAH, 2020c).

3.21.3 | Geographical distribution

TBEV is currently endemic in Europe. TBEV infection is not reportable to WOAH. Evidence from published studies describing natural infections with this agent, as well as field epidemiological studies, is collected in the SLR and summarised in Figure 36. All references can be found in Annex A and in the online version of the Disease Profile.[23]

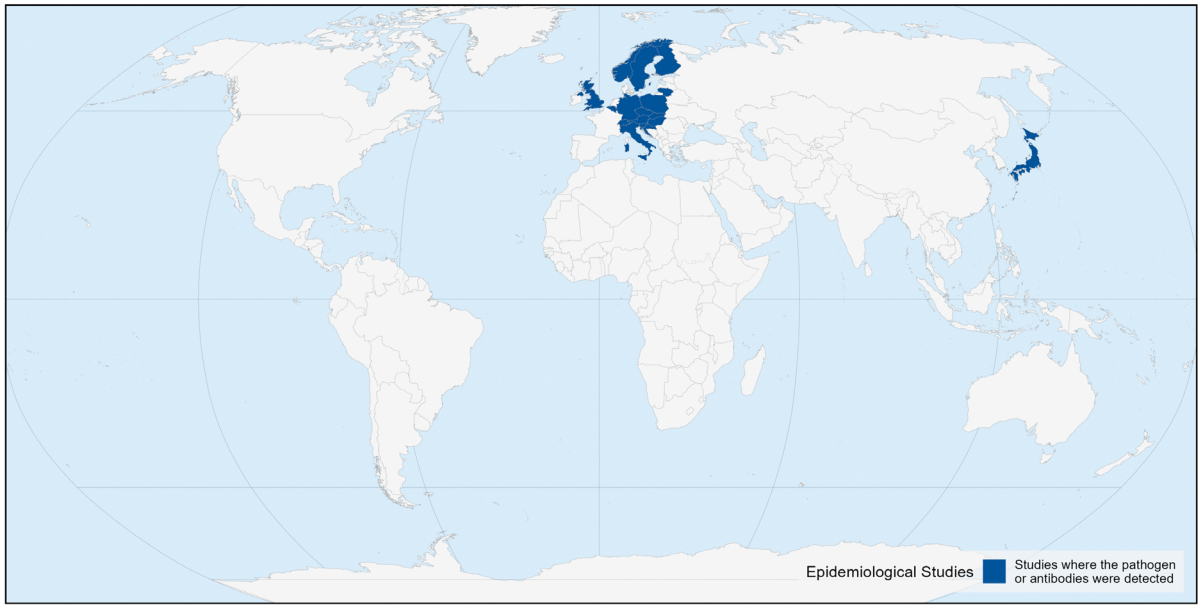


FIGURE 36 Geographical distribution of epidemiological studies addressing the occurrence of TBEV, as identified by EFSA's SLR (covering years 1970–2025).

3.21.4 | Animal hosts

Susceptible hosts

Based on epidemiological knowledge of host–pathogen–vector interactions and outbreak reports, TBEV has a wide range of vertebrate hosts with the most important being rodents, whereas humans are considered as dead-end hosts. However, other species have been identified in the SLR. The SLR summary is given in Table 31.

[23]Online version: https://animal-diseases.efsa.europa.eu/TBEV.

TABLE 31 Susceptible host species of tick-borne encephalitis virus.

The SLR reported in the TBEV disease profile identified the following susceptible species (updated until 31 December 2025; for references, see Annex A)
FIELD
Epidemiological studies carried out in the field
Pathogen was detected in the following animal species: • Bovidae: *Bos taurus, Capra hircus, Ovis aries* • Canidae: *Canis lupus familiaris* • Cervidae: *Capreolus capreolus* • Cricetidae: *Myodes glareolus, Microtus arvalis* • Muridae: *Apodemus agrarius, Apodemus flavicollis*
Antibodies were detected in the following animal species: • Bovidae: *Bos taurus, Capra hircus, Ovis aries* • Canidae: *Canis lupus familiaris* • Cervidae: *Capreolus capreolus, Cervus elaphus, Dama dama, Alces alces, Odocoileus virginianus, Cervus nippon* • Equidae: *Equus caballus* • Hominidae: *Homo sapiens* • Muscicapidae: *Luscinia megarhynchos* • Procyonidae: No species specified • Suidae: *Sus scrofa* • Sylviidae: *Sylvia atricapilla* • Turdidae: *Turdus merula*
Outbreaks reported to WOAH included the following species: • No species specified
EXPERIMENTS
Experimental studies demonstrated infection in: • Bovidae: *Ovis aries*

Clinical signs

Domestic animals infected with TBEV typically remain asymptomatic. Clinical signs, observed at a low frequency, have been reported predominantly in dogs and horses.

Described signs included hyperthermia, behavioural changes, apathy, ataxia/vestibular signs, cranial nerve deficits, cervical pain, paresis of one or more limbs, convulsion and seizures.

Livestock do not commonly show clinical disease but can develop robust, long-lasting and specific antibody response to infection. A rare event of disease in sheep has been reported, with an affected animal showing pyrexia, acute neurological signs, ataxia, torticollis, tremor, nystagmus, salivation and finally somnolence with inappetence and recumbency.

Clinical manifestation has been occasionally reported also in horses, including ataxia, tonic–clonic seizures, apathy and stupor, inappetence, mydriasis, convulsions of the legs, skittishness, bruxism and altered reactions to environmental stimuli.

An experimental study on bank voles (*Myodes glareolus*) reported distress and rapid weight loss as clinical signs in four out of 96 infected voles (Michelitsch et al., 2021).

Incubation period

TBEV infection in animals is usually asymptomatic and clinical cases are sporadic, so a precise incubation period is not well established in the peer-reviewed literature. One study has reported an incubation period of 5–9 days in dogs (Salat et al., 2021). In ruminants, appearance of the virus in milk has been demonstrated as early as day 2–3 post-infection, with excretion lasting up to 2 weeks (Hennechart-Collette et al., 2022).

Morbidity and case fatality

Seroprevalence studies indicate frequent infections in dogs, mostly asymptomatic and data to estimate morbidity rates are scarce. Reported case fatality rates in clinically affected dogs range from 16% to 50% (Annex A).

In experimentally infected voles, a morbidity rate of 4.17% (4/96) was observed (Hennechart-Collette et al., 2022). Similarly, one study experimentally infected lambs with TBEV and no clinical signs following infection were observed (Paulsen et al., 2019).

Zoonotic potential

Tick-borne encephalitis is a zoonotic disease (WHO, 2023b).

3.21.5 | Transmission

TBEV is transmitted to vertebrate hosts through the bite of ticks (Acari; Ixodidae). Evidence on the potential and likely competent tick species and their geographic distribution can be found in a dedicated Scientific Report on vectors (EFSA, 2026a).

In a natural cycle, small mammals like rodents and insectivores act as reservoir hosts.

Transmission to humans might occasionally occur through the consumption of raw milk from infected cattle, sheep or goat.

3.21.6 | Diagnostic tests

There are no WOAH-recommended tests for TBEV. There are diagnostic tests available for detection of either TBEV or specific antibodies against it.

For the detection of the agent, RT-PCR is used to identify viral RNA in various matrices, including blood, serum, organs and both bulk tank and individual milk samples. Virus isolation is also employed for the detection of the virus.

For immune response detection, the virus neutralisation test is available, along with commercial ELISAs and immunofluorescence assays. Serological tests have also been used to detect antibodies in milk samples.

Table 32 presents data on the sensitivity and specificity of diagnostic tests collected through SLR; reported values correspond to the median sensitivity and specificity when multiple studies investigated the same test and are only included when explicitly stated in the publications.

TABLE 32 Median sensitivity and specificity of tests to detect TBEV/TBEV antibodies reported in literature included in the systematic literature review.

Target	Test	Species	Sensitivity	*N* animal groups	Specificity	*N* animal groups	References
Antibody	Competitive ELISA	Wild boar	20%	1	84%	1	Trozzi et al. (2023)
Antibody	Indirect ELISA	Wild boar	23%	1	88%	1	

Note: The SLR was updated until 31 December 2025; for references, see Annex A.

3.21.7 | Prevention and control

Vaccination

There are currently no licensed vaccines for TBEV in animals.

Treatment

There is currently no specific antiviral treatment for TBEV infection, and management in dogs and horses relies on supportive care tailored to the severity of the clinical signs.

3.22 | *Trypanosoma evansi* (*T. evansi*)[24]

3.22.1 | Disease overview

Trypanosoma evansi is a flagellated protozoan parasite that causes the disease surra. It infects a wide range of wild and domestic animals. Among domestic species, the most affected vary by region but commonly include horses, camels, cattle and buffalo. Transmission is primarily mechanical by hematophagous flies. Iatrogenic transmission can occur, and carnivores may become infected by ingesting infected meat (Spickler, 2024a, 2024b, 2024c, 2024d; WOAH, 2013b).

Surra is a WOAH-notifiable disease, listed in the EU AHL under categories D and E.

3.22.2 | Agent

T. evansi is a unicellular flagellated protozoan parasite, belonging to the family Trypanosomatidae, genus *Trypanosoma*, section Salivaria. The parasite is morphologically characterised as a thin bloodstream trypomastigote with small terminal kinetoplast, but there are some akinetoplastic strains. Strains from different geographical areas and various host sources are morphologically indistinguishable. The trypanosomes measure 14–33 μm in length with a width of 1.5–2.2 μm. They possess a free flagellum and a small sub-terminal kinetoplast. *T. evansi* multiply in their mammalian hosts by longitudinal, binary fission (Brun et al., 1998; Desquesnes, Holzmuller, et al., 2013).

[24]Online version: https://animal-diseases.efsa.europa.eu/Tevansi.

3.22.3 | Geographical distribution

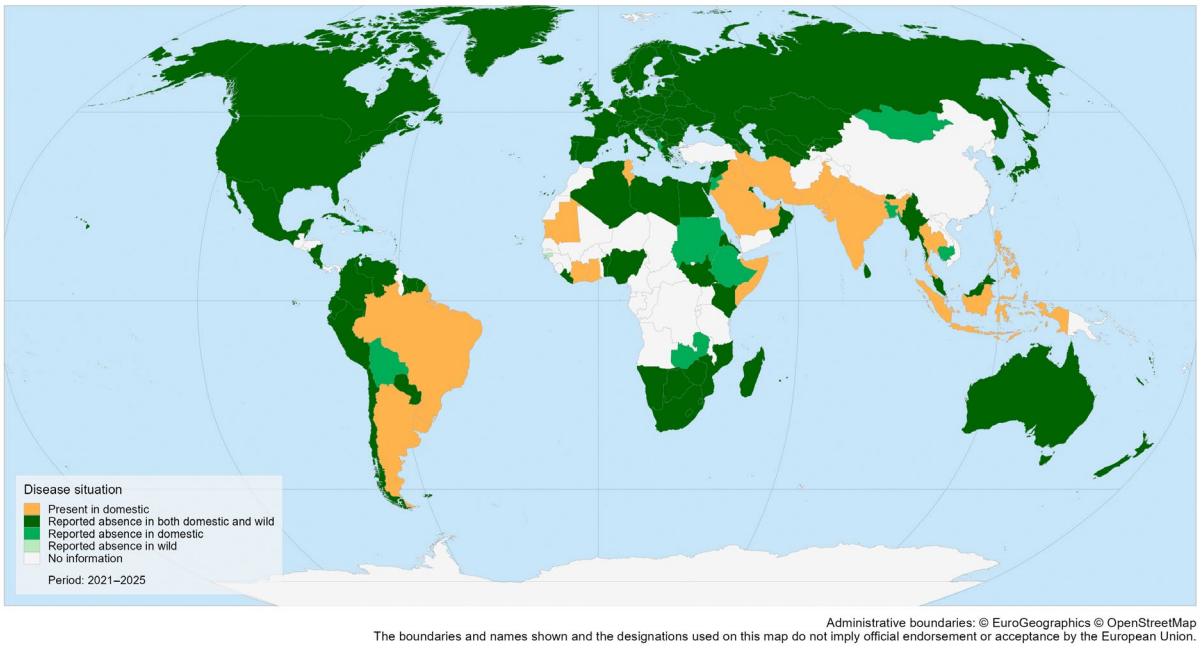


FIGURE 37 Geographical distribution of *Trypanosoma evansi* detected events (2021–2025), as reported to WOAH.

T. evansi has a wide geographical distribution and has been reported in Africa, Central and South America and Asia. Sporadic introduction into Europe, particularly in the Canary Islands and France, have also been reported in the past (Aregawi et al., 2019; Desquesnes, Holzmuller, et al., 2013). The distribution of reported occurrence in the last 5 years is shown in Figure 37. According to WAHIS data, the agent was not reported in the EU in the last 5 years. Up-to-date maps based on WAHIS are available in the online version of the Disease Profile.[24]

3.22.4 | Animal hosts

Susceptible species

Based on epidemiological knowledge of host–pathogen–vector interactions and outbreak reports, *T. evansi* has a wide range of vertebrate hosts, with the most important being ruminants, camelids and horses, whereas pigs and humans are considered dead-end hosts. The SLR summary is given in Table 33.

TABLE 33 Susceptible host species of *Trypanosoma evansi*.

The SLR reported in the *T. evansi* disease profile, identified the following susceptible species (updated until 31 December 2025; for references, see Annex A) identified the following susceptible species (updated until 31 December 2025; for references, see Annex A)

FIELD

Epidemiological studies carried out in the field

- **Pathogen was detected in the following animal species:**
  - Bovidae: *Bos taurus, Capra hircus, Ovis aries, Bubalus bubalis*
  - Camelidae: *Camelus bactrianus, Camelus dromedarius*
  - Canidae: *Canis lupus familiaris*
  - Equidae: *Equus caballus*
- **Antibodies were detected in the following animal species:**
  - Bovidae: *Bos taurus*
  - Camelidae: *Camelus dromedarius*
- **Outbreaks reported to WOAH included the following species:**
  - Bovidae: *Bos taurus*
  - Canidae: *Canis lupus familiaris*
  - Equidae: No species specified

EXPERIMENTS

- **Experimental studies demonstrated infection in:**
  - Bovidae: *Bos taurus, Capra hircus, Ovis aries, Bison bonasus, Bubalus bubalis*
  - Camelidae: *Camelus bactrianus, Camelus dromedarius*
  - Equidae: *Equus caballus, Equus caballus × Equus asinus (Mule)*
  - Suidae: *Sus scrofa domesticus*

Clinical signs

Outcomes of a SLR on clinical signs in 23 domestic ruminant and 11 equid study groups are displayed in Figure 38. Most study groups for both categories showed generic clinical signs.

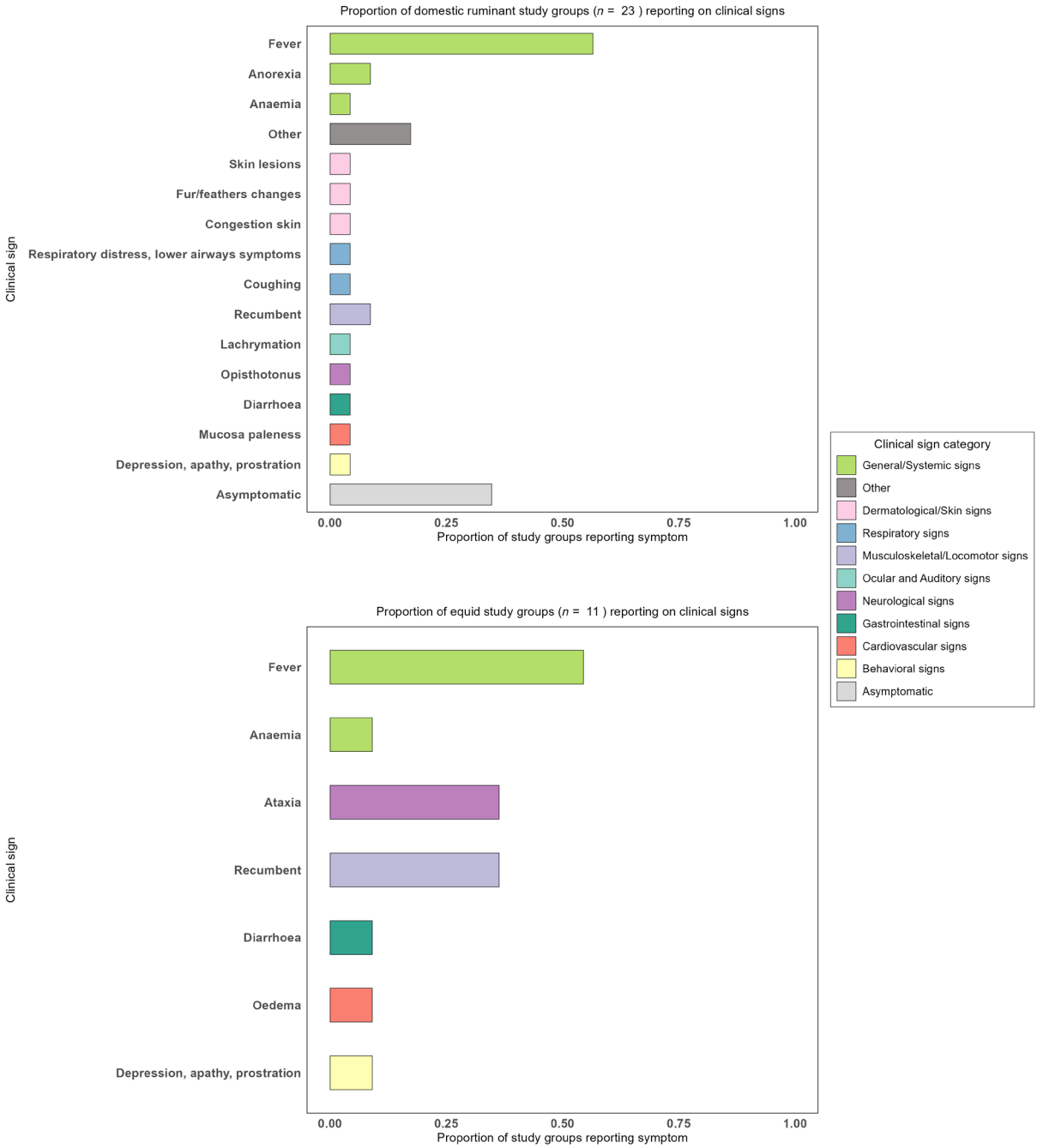


FIGURE 38 Clinical signs reported in the main hosts of *T. evansi*. Study group count per domestic ruminant species: Sheep $n = 10$; Cattle $n = 4$; Goat $n = 4$; Water buffalo $n = 4$; Buffalo $n = 1$. Study group count per equid species: Horse $n = 7$; Ass $n = 4$. Camelid study groups not visualised due to small sample size (Camel $n = 1$; Dromedary $n = 1$). The SLR was updated until 31 December 2025; for references, see Annex A.

T. evansi is mostly pathogenic for camels and horses and has high economic relevance for water buffalo in Asia. Frequent clinical signs include fever (can appear intermittently), anaemia, pale mucous membrane, loss of appetite and weight. Nervous signs, abortion, cachexia and death (mostly camels and horses) can be also observed. Overall, there is a large variety in the intensity of the expression of clinical signs depending on the host species and geographical location (Annex A; Desquesnes, Holzmuller, et al., 2013; Kim et al., 2024).

Incubation period

The SLR identified studies in buffaloes, camels, cattle, dromedary and sheep, all with a median incubation period of 1–2 days. In goats, the median incubation was also 2 days, but clinical signs could take up to 6 months to develop. Clinical signs lasted up to 2 months in sheep and cattle (Annex A).

Morbidity and case fatality

Overall, high morbidity (> 50%) and often high case fatality are expected in outbreaks affecting camels and horses, with fatalities close to 100% in acute untreated cases. Typically, chronic disease with lower case fatality is expected in cattle and buffalo, but production losses can still be substantial. Disease in other domestic species such as goats, sheep, pigs and dogs can range from subclinical to fatal (Aregawi et al., 2019; Desquesnes, Dargantes, et al., 2013; Desquesnes, Holzmuller, et al., 2013).

In experimental studies, median fatality rates as high as 100% for donkeys and camels have been reported. Median case fatality was also high for sheep and cattle (75%). In goats, the median fatality was 57%, but varied by animal group, and could reach 100%. Experimental studies in dromedary showed lower rates (median 10%), and no mortality was observed in horses (Annex A).

Zoonotic potential

T. evansi is a zoonotic disease, with human infection being rare and incidental (Desquesnes, Dargantes, et al., 2013; Joshi et al., 2005).

3.22.5 | Transmission

T. evansi is transmitted mechanically, primarily by biting flies. Evidence on the competent vector species and their geographic distribution can be found in a dedicated Scientific Report on vectors (EFSA, 2026a).

Following feeding from an infected host, the parasites survive for a few hours in the mouthparts of the vector; therefore, transmission takes place only when the vectors feed on new susceptible hosts within a short period of time (Desquesnes, Dargantes, et al., 2013). Iatrogenic transmission has also been associated with mechanical spread of *T. evansi (*Kim et al., 2024).

T. evansi can also locate extravascularly, which can lead to infection of carnivores by eating infected meat. Vampire bats (*Desmodus rotundus*) can both become infected with *T. evansi* but also act as vectors, able to transmit the parasite mechanically via their saliva (Desquesnes, Dargantes, et al., 2013; Ramírez et al., 2014).

Transplacental transmission of experimentally infected ewes was demonstrated. In addition, *T. evansi* DNA was detected in milk/colostrum from these ewes and used to orally infect mice, demonstrating potential transmission of *T. evansi* via this route (Campigotto et al., 2015).

3.22.6 | Diagnostic tests

WOAH recommended tests (WOAH, 2025b) for detection of the parasite are based on parasitological techniques involving direct microscopic examination of wet or dry-stained thick or thin blood films and the buffy-coat/microhaematocrit centrifugation technique (HCT). Detection of DNA by PCR or parasite-specific antigen using ELISA tests are also tests that confirm presence of the agent. Among all these tests, DNA detection by PCR has the highest sensitivity (78%–98%). The Se of the antigen ELISA test was around 79% (Annex A). Parasitological techniques have been shown moderate to low Se (39% to 71%) (Annex A; Holland et al., 2001; Monzón et al., 1990).

Serological methods such as ELISA, immunofluorescence assay (IFA) and the card agglutination test (CATT) are also recommended by WOAH. These tests have high Se (81%–95%) and specificity (> 95%) when applied in serum samples from camels or horses (Annex A; Camoin et al., 2019).

Table 34 presents data on the sensitivity and specificity of diagnostic tests collected through SLR; reported values correspond to the median sensitivity and specificity when multiple studies investigated the same test and are only included when explicitly stated in the publications.

TABLE 34 Median sensitivity and specificity of tests to detect *T. evansi*/*T. evansi* antibodies reported in literature included in the systematic literature review.

Target	Test	Species	Sensitivity	*N* animal groups	Specificity	*N* animal groups	References
Antigen	ELISA	Cattle and camel	79%	1	99%	1	Rudramurthy et al. (2018)
Antigen	ELISA	Dromedary	42.3%	2	100%	1	Sadek et al. (2021); Singh et al. (2004)
Antigen	ELISA	Water buffalo	80.0%	1	100%	1	Nguyen et al. (2014)
Antigen	Latex agglutination test	Camel	79.4%	2	99.5%	2	Hassan et al. (2005); Ngaira et al. (2003)
Nucleic Acid	Dry-LAMP	Dromedary	82.7%	1	–	–	Salim et al. (2018)
Nucleic Acid	Loop-mediated isothermal amplification (LAMP)	Pig	45.0%	1	100%	1	Thekisoe et al. (2005)
Nucleic Acid	PCR	Cattle	99.1%	2	90%	2	Freire et al. (2025); Maharana et al. (2019)
Nucleic Acid	PCR	Dog	100%	3	100%	3	Kaur et al. (2025); Azhahianambi et al. (2018)
Nucleic Acid	PCR	Dromedary	100%	4	100%	2	Sadek et al. (2021); Ali et al. (2011); Singh et al. (2004); Njiru et al. (2004)
Nucleic Acid	PCR	Equines	100%	1	94%	1	Devi et al. (2017)
Nucleic Acid	PCR	Pig	33%	1	100%	1	Thekisoe et al. (2005)
Nucleic Acid	PCR	Water buffalo	77.9%	2	100%	1	Parashar et al. (2015); Holland et al. (2001)
Parasite	Blood smear	Dromedary	16.7%	1	100%	1	Sadek et al. (2021)
Parasite	Thick blood smear	Camel	80%	1	100%	1	Hassan et al. (2005)
Parasite	Thin blood smear	Camel	80%	1	100%	1	Hassan et al. (2005)
Parasite	Thin blood smear	Cattle	73.7%	1	97.3%	1	Albohiri and Alsulami (2025)
Parasite	Thin blood smear	Dromedary	76%	3	100%	2	Selim et al. (2022); Ali et al. (2011); Singh et al. (2004)
Parasite	Thin blood smear	Pig	24%	1	100%	1	Thekisoe et al. (2005)
Parasite	Wet blood smear	Camel	80%	1	100%	1	Hassan et al. (2005)
Parasite	Wet blood smear	Dromedary	62.2%	2	100%	1	Ali et al. (2011); Singh et al. (2004)
Parasite	Wet blood smear	Water buffalo	25.1%	1	–	–	Holland et al. (2001)
Parasite	Buffy coat	Cattle	14.3%	1	99.7%	1	Kengradomkij et al. (2025)
Parasite	Buffy coat	Dromedary	100%	1	100%	1	Ali et al. (2011)
Parasite	Buffy coat	Water buffalo	38.6%	1	–	–	Holland et al. (2001)
Parasite	Direct microscopic examination	Cattle	34.6%	1	97.2%	1	Uzcanga et al. (2016)
Parasite	Microhaematocrit centrifugation technique (mHCT)	Cattle	37.2%	2	97.7%	2	Kengradomkij et al. (2025); Uzcanga et al. (2016)
Parasite	mHCT	Goat	92.7%	1	100%	1	Tejedor Junco et al. (2011)
Parasite	mHCT	Pig	38%	1	100%	1	Thekisoe et al. (2005)
Parasite	mHCT	Water buffalo	69.6%	1	–	–	Holland et al. (2001)
Parasite	Mini-anion-exchange centrifugation technique	Water buffalo	64.1%	1	–	–	Holland et al. (2001)
Parasite	Mouse inocolation test	Pig	65%	1	100%	1	Thekisoe et al. (2005)

(Continues)

TABLE 34 (Continued)

Target	Test	Species	Sensitivity	*N* animal groups	Specificity	*N* animal groups	References
Parasite	Mouse inocolation test	Water buffalo	74%	1	-	–	Holland et al. (2001)
Antibody	Card agglutination test (CATT)	Buffalo	77.4%	2	99.4%	2	Birhanu et al. (2015); Davison et al. (1999)
Antibody	CATT	Camel	78.8%	2	99.9%	2	Rogé et al. (2014); Ngaira et al. (2003)
Antibody	CATT	Cattle	77.5%	2	95.6%	4	Birhanu et al. (2015); Rogé et al. (2014); Reid and Copeman (2003)
Antibody	CATT	Dog	–	–	100%	2	Birhanu et al. (2015); Rogé et al. (2014)
Antibody	CATT	Dromedary	83%	5	99.6%	4	Selim et al. (2022); Boushaki et al. (2019); Birhanu et al. (2015); Ali et al. (2011); Njiru et al. (2004)
Antibody	CATT	Equihnes	100%	1	95.6%	2	Birhanu et al. (2015); Sumbria et al. (2014)
Antibody	CATT	Horse	–	–	100%	1	Rogé et al. (2014)
Antibody	CATT	Sheep	100%	2	96.3%	2	Birhanu et al. (2015); Rogé et al. (2014)
Antibody	CATT	Vicugna	–	–	100%	1	Birhanu et al. (2015)
Antibody	CATT	Water buffalo	73.8%	1	76.9%	1	Rogé et al. (2014)
Antibody	CATT	Zebu	30.7%	1	61.4%	1	Singla et al. (2015)
Antibody	ELISA, competitive	Equines	71.7%	1	99%	1	Mizushima et al. (2018)
Antibody	ELISA	Buffalo	78%	4	88.5%	4	Kumar et al. (2022); Davison et al. (1999)
Antibody	ELISA	Camel	98.9%	1	98.9%	1	Tran et al. (2009)
Antibody	ELISA	Cattle	95%	7	97.7%	7	Thi Nguyen et al. (2025); Kumar et al. (2022); Jaimes-Dueñez et al. (2019); Kundu et al. (2013); Reid and Copeman (2003)
Antibody	ELISA	Dromedary	100%	5	100%	5	Bossard and Desquesnes (2023); Kumar et al. (2022)
Antibody	ELISA	Equines	91.3%	7	61.5%	7	Kumar et al. (2022); Kumar et al. (2016); Reyna-Bello et al. (1998)
Antibody	ELISA	Pig	100%	2	97.5%	2	Kumar et al. (2022)
Antibody	IgG ELISA	Buffalo	89%	1	95%	1	Davison et al. (1999)
Antibody	ELISA, indirect	Cattle	76.2%	2	91%	2	Uzcanga et al. (2016)
Antibody	ELISA, indirect	Dromedary	91%	1	95%	1	Boushaki et al. (2019)
Antibody	ELISA, indirect	Equines	97.5%	1	100%	1	Camoin et al. (2019)
Antibody	LFIA	Buffalo	98.8%	1	94.4%	1	Birhanu et al. (2015)
Antibody	LFIA	Cattle	–	–	89%	1	Birhanu et al. (2015)
Antibody	LFIA	Dog	–	–	96%	1	Birhanu et al. (2015)
Antibody	LFIA	Dromedary	97.8%	1	95.8%	1	Birhanu et al. (2015)
Antibody	LFIA	Equines	–	–	100%	1	Birhanu et al. (2015)
Antibody	LFIA	Sheep	100%	1	96.3%	1	Birhanu et al. (2015)
Antibody	LFIA	Vicugna	–	–	83.8%	1	Birhanu et al. (2015)
Antibody	Latex agglutination test	Camel	92.2%	1	98%	1	Rogé et al. (2014)

TABLE 34 (Continued)

Target	Test	Species	Sensitivity	*N* animal groups	Specificity	*N* animal groups	References
Antibody	Latex agglutination test	Cattle	–	–	96.8%	1	Rogé et al. (2014)
Antibody	Latex agglutination test	Dog	–	–	100%	1	Rogé et al. (2014)
Antibody	Latex agglutination test	Horse	–	–	100%	1	Rogé et al. (2014)
Antibody	Latex agglutination test	Sheep	100%	1	100%	1	Rogé et al. (2014)
Antibody	Latex agglutination test	Water buffalo	68%	1	100%	1	Rogé et al. (2014)
Antibody	Microsphere-based immunoassay	Horse	97.9%	1	96%	1	Verney et al. (2022)
Antibody	Sandwich ELISA	Dromedary	92%	1	–	–	Pathak et al. (1993)

Note: The SLR was updated until 31/12/2025, references see Annex A.

3.22.7 | Prevention and control

Vaccination

There are no vaccines available against *T. evansi*.

Treatment

Trypanocidal drugs, such as diminazene diaceturate (DA), isometamidium chloride (IC), Cymelarsan and Quinapyramine sulfate, are used for treatment. Overall, the efficacy varies with host species, parasite strain/isolate, dose used and treatment regimen, with some resistance patterns (low efficacy), which may be associated with some specific isolates reflecting regional drug tolerance (Mekonnen et al., 2018; Annex A).

3.23 | *Trypanosoma vivax* (*T. vivax*)[25]

3.23.1 | Disease overview

Trypanosoma vivax is a protozoan parasite, part of the several species of the genus *Trypanosoma* which can cause trypanosomiasis. *T. vivax* affects multiple species of domestic and wild animals. The protozoan is transmitted cyclically by tsetse flies of the genus *Glossina* (restricted to sub-Sahara Africa and some regions of the Arabian Peninsula) or mechanically by biting flies, as well as through iatrogenic transmission (Fetene et al., 2021; WOAH, 2021b).

Trypanosomiasis caused by *T. vivax* is a WOAH-notifiable disease, but it is not listed in the European AHL.

3.23.2 | Agent

T. vivax is a unicellular flagellated protozoan parasite, belonging to the family Trypanosomatidae, genus *Trypanosoma*, section Salivaria. The parasite is morphologically characterised by a bloodstream trypomastigote stage with a terminal kinetoplast and well-developed undulating membrane (WOAH, 2021b). Trypomastigotes can multiply in the hosts and be mechanically transmitted by biting flies.

[25]Online version: https://animal-diseases.efsa.europa.eu/Tvivax.

3.23.3 | Geographical distribution

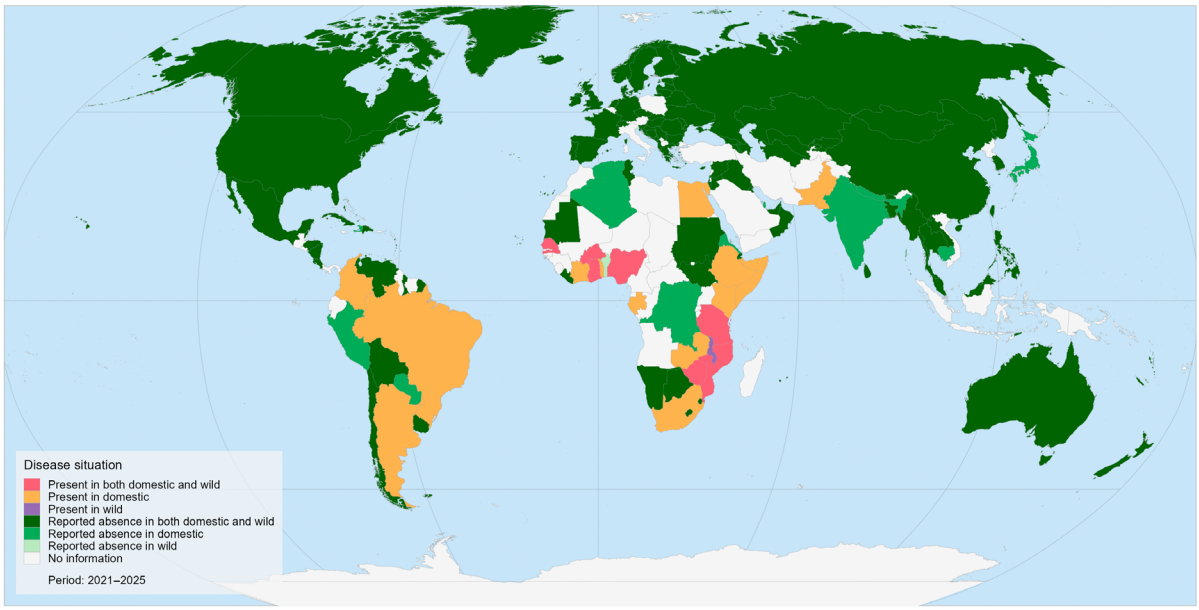


FIGURE 39 Geographical distribution of *Trypanosoma vivax* detected events (2021–2025), as reported to WOAH.

T. vivax has been reported in Africa, South America and Asia. This parasite has not been reported in Europe. Reported infections to WOAH for the period 2021–2025 are shown in Figure 39. In addition to those reports shown in this figure, epidemiological studies have reported the presence of *T. vivax* in Bolivia (Gonzales et al., 2007), Ecuador (Chávez-Larrea et al., 2024), Venezuela (Ramírez-Iglesias et al., 2017), Peru and Guyana (Fetene et al., 2021). All references and up-to-date maps based on reports to WOAH are available in the online version of the Disease Profile.[25]

3.23.4 | Animal hosts

Susceptible hosts

Based on epidemiological knowledge of host–pathogen–vector interactions and outbreak reports, the main hosts of *T. vivax* are domestic ruminants, equids and camelids. However, other susceptible species have been identified in the SLR. The SLR summary is given in Table 35.

TABLE 35 Susceptible host species of *Trypanosoma vivax* encephalitis virus.

The SLR reported in the *T. vivax* disease profiles, identified, or failed to identify the following susceptible species (updated until 31 December 2025, for references, see Annex A)
FIELD
Epidemiological studies carried out in the field

• **Pathogen was detected in the following animal species:**
  • Bovidae: *Bos taurus, Capra hircus, Ovis aries, Bubalus bubalis*
  • Camelidae: *Camelus bactrianus*
  • Equidae: *Equus caballus*
  • Suidae: *Sus scrofa domesticus*
• **Antibodies were detected in the following animal species:**
  • Bovidae: *Bos taurus*
  • Equidae: *Equus caballus*

• **Outbreaks reported to WOAH included the following species:**   • Bovidae: *Bos Taurus*
EXPERIMENTS

• **Experimental studies demonstrated infection in:**
  • Bovidae: *Bos taurus, Capra hircus, Ovis aries, Bubalus bubalis*
  • Equidae: *Equus caballus*

Clinical signs

Outcomes of a SLR of clinical signs in 36 study groups of domestic ruminants are displayed in Figure 40. Predominantly, general clinical signs were reported.

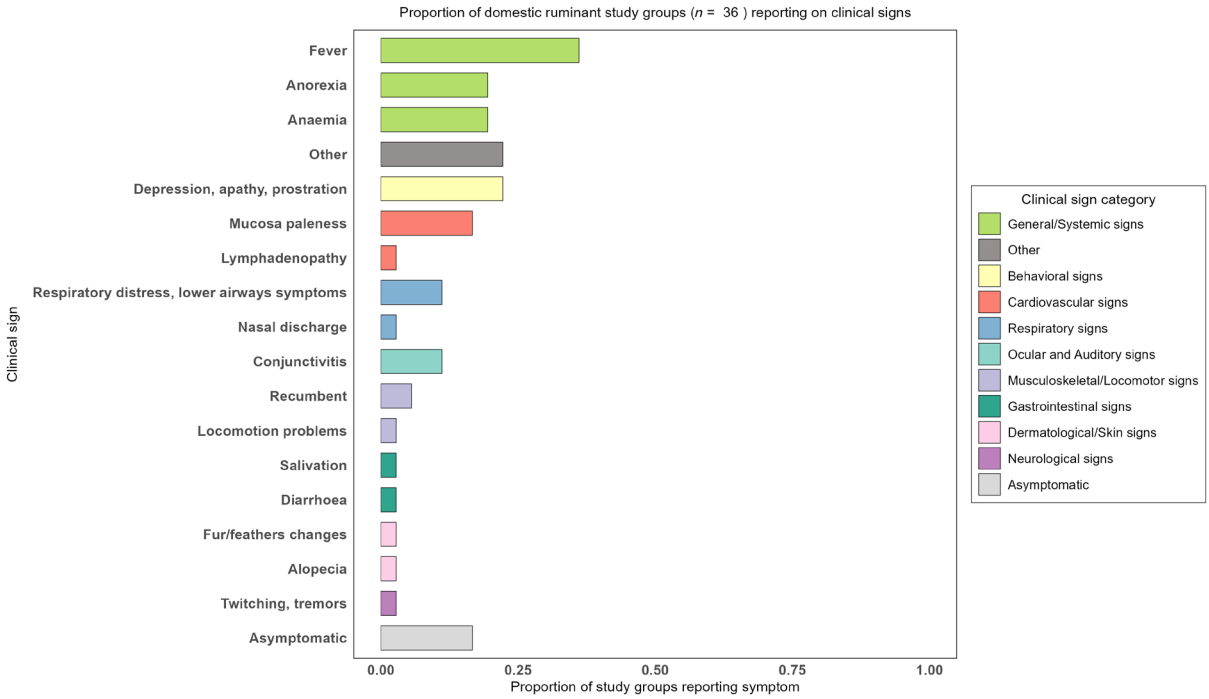


FIGURE 40 Clinical signs reported in the main hosts of *T. vivax*. Study group count per domestic ruminant species: Cattle $n = 16$; Goat $n = 9$; Sheep $n = 8$; Buffalo $n = 3$. Equid study groups not visualised due to small sample size (Horse $n = 2$). The SLR was updated until 31 December 2025; for references, see Annex A.

Following infection, animals, particularly cattle, goat and sheep, develop fever which is followed by anaemia, anorexia and prostration. Mucosal paleness, conjunctivitis and locomotion problems can also be observed. Decreased milk production and associations with abortions have also been reported for cattle, sheep and goat (Batista et al., 2009; Galiza et al., 2011; Riet-Correa et al., 2025). Diseased animals can show clinical signs for extended periods ranging from weeks to several months in these three ruminant species (Cadioli et al., 2012; Annex A).

Incubation period

The incubation period, experimentally measured in cattle, goat and sheep, can take from 2 to 7 days. The SLR identified experimental studies in buffalo, cattle, goat, horses and sheep (Annex A).

Morbidity and case fatality

Outbreak investigations have reported morbidities in cattle following mechanical transmission by hematophagous flies ranging from 3.6% to 80% with a median morbidity around 22.8%. Reported mortalities in these outbreaks (estimated as the observed mortality out of the total population) ranged from 2.1% to 10.6% (Riet-Correa et al., 2025).

In experimental studies, median fatality rates of 16.7% (interquartile ranges of 8.3–37.5) in cattle, 25% (7–100) in goats and 13.3% (10.0–16.7) in sheep have been reported (Annex A).

Zoonotic potential

T. vivax is not known to infect humans under natural conditions.

3.23.5 | Transmission

T. vivax is transmitted biologically or mechanically by hematophagous vectors of the order Diptera. In Sub-Sahara Africa and some regions of the Arabian Peninsula, *T. vivax* is mainly transmitted biologically by tsetse flies (*Glossinidae*). Other biting flies transmit *T. vivax* mechanically. Evidence on the potential and likely competent vector species and their geographic distribution can be found in a dedicated Scientific Report on vectors (EFSA, 2026a).

For the mechanical transmission, following feeding from an infected host, the parasites survive for a few hours in the mouthparts of these biting flies; therefore, transmission takes place only when the mechanical vectors feed on new susceptible hosts within a short period of time. Iatrogenic transmission has also been associated with mechanical spread of *T. vivax* (Riet-Correa et al., 2025).

3.23.6 | Diagnostic tests

WOAH recommended tests (WOAH, 2025b) for detection of the parasite are based on parasitological techniques involving direct microscopic examination of wet or dry-stained thick or thin blood films and the buffy-coat/microhaematocrit centrifugation technique (HCT). These tests have limited sensitivity around 52%, which depends on the time from sample collection to diagnostic testing, with the highest sensitivity reached when testing fresh blood samples. Specificity of these tests is in general high, but tests such as the HCT do not allow specific identification of the *Trypanosoma* species in areas where multiple species circulate (Masake et al., 1997).

Detection of DNA by PCR is the method with the highest specificity (close to 100%) and sensitivity (> 90%), particularly when using whole blood samples (Gonzales et al., 2006; Masake et al., 1997).

Serological methods such as ELISA and immunofluorescence assay (IFA) are also recommended by WOAH. These tests have overall moderate to high sensitivity (65%–89%) and specificity (> 80%).

Table 36 presents data on the sensitivity and specificity of diagnostic tests collected through SLR; reported values correspond to the median sensitivity and specificity when multiple studies investigated the same test and are only included when explicitly stated in the publications.

TABLE 36 Median sensitivity and specificity of tests to detect *T. vivax*/*T. vivax* antibodies reported in literature included in the systematic literature review.

Target	Test	Species	Sensitivity	*N* animal groups	Specificity	*N* animal groups	References
Antigen	ELISA	Cattle	28.55%	6	–	–	Eisler et al. (1998)
Antigen	Lateral flow immunochromatographic assay (LFIA)	Cattle	86%	1	32.5%	1	Richard Gashururu et al. (2021)
Nucleic Acid	PCR	Cattle	75.15%	2	100%	2	de Melo-Junior et al. (2024)
Nucleic Acid	RT-PCR	Cattle	93.42%	1	82.43%	1	Contreras-García et al. (2022)
Parasite	Blood smear	Cattle	10%	1	94%	1	Takeet et al. (2013)
Parasite	Thin blood smear	Cattle	28.9%	1	99.1%	1	Richard Gashururu et al. (2021)
Parasite	Wet blood smear	Cattle	38%	2	100%	2	de Melo-Junior et al. (2024)
Parasite	Buddy coat	Cattle	15.6%	3	–	–	Pillay et al. (2013)
Parasite	Microhaematocrit centrifugation technique (mHCT)	Cattle	40%	3	100%	3	Richard Gashururu et al. (2021); de Melo-Junior et al. (2024)
Antibody	ELISA	Cattle	82.35%	5	92.3%	5	Pinheiro et al. (2021); de Melo-Junior et al. (2024); Madruga et al. (2006)
Antibody	IgG ELISA	Cattle	85.3%	2	82.6%	2	Pinheiro et al. (2021)
Antibody	ELISA (indirect)	Cattle	62.1%	12	90.2%	4	Bontempi et al. (2024); Pillay et al. (2013); Magona et al. (2002)
Antibody	IFAT	Cattle	66.6%	2	90%	2	de Melo-Junior et al. (2024)
Antibody	LFIA	Cattle	69.6%	2	90%	2	de Melo-Junior et al. (2024)

Note: The SLR was updated until 31 December 2025; for references, see Annex A.

3.23.7 | Prevention and control

Vaccination

There are no vaccines available.

Treatment

Trypanocidal drugs such as diminazene diaceturate (DA), isometamidium chloride (IC), Imidocarb dipropionate (ID) and Ascofuranone (antibiotic) are used for treatment. High therapeutic efficacy (close to 100%) under experimental conditions has been reported for DA, IC and Ascofuranone, while lower efficacy (66.9%) was reported for ID (Bastos et al., 2020).

3.24 | Venezuelan equine encephalitis virus (VEEV)[26]

3.24.1 | Disease overview

Venezuelan equine encephalitis virus (VEEV) causes Venezuelan equine encephalitis, a mosquito-borne viral disease. 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 (Spickler, 2024a, 2024b, 2024c, 2024d; WOAH, 2020d).

Venezuelan equine encephalitis is a WOAH-notifiable disease, listed in the EU AHL under categories D and E.

3.24.2 | Agent

VEEV is an enveloped, single-stranded, positive-sense RNA virus belonging to the *Alphavirus* genus within the Togaviridae family. 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 neutralising 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 (Strauss & Strauss, 1994; Weaver et al., 1992, 2004; Young & Johnson, 1969).

3.24.3 | Geographical distribution

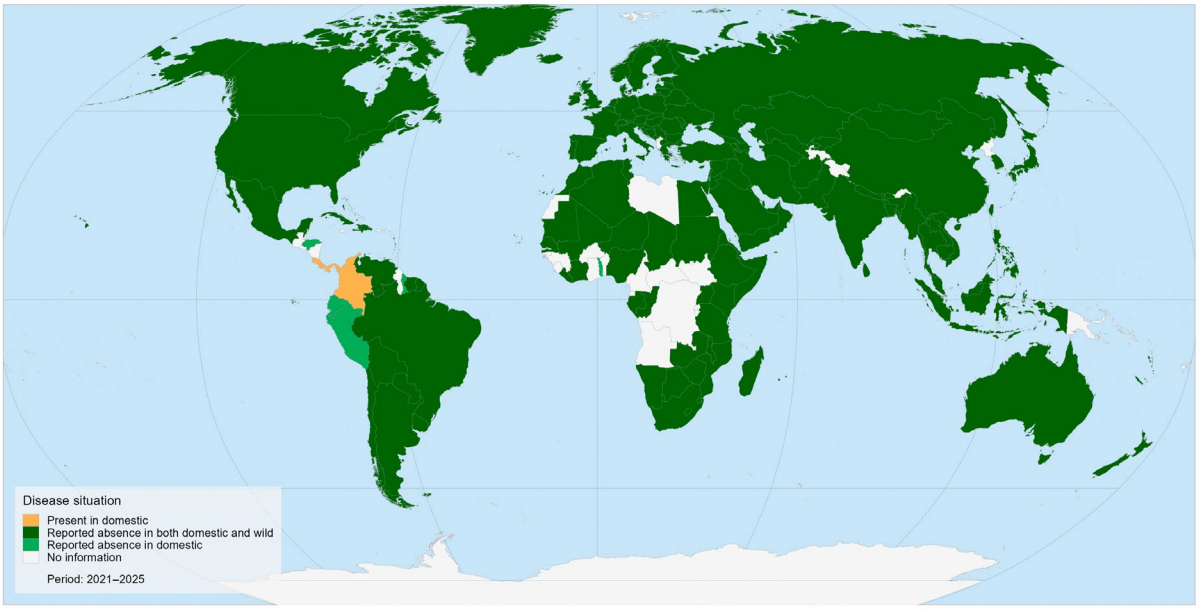


FIGURE 41 Geographical distribution of VEEV detected events (2021–2025), as reported to WOAH.

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 5 years (Figure 41). Up-to-date maps based on WAHIS are available in the online version of the Disease Profile.[26]

[26]Online version: https://animal-diseases.efsa.europa.eu/VEEV.

3.24.4 | Animal hosts

Susceptible hosts

Based on epidemiological knowledge of host–pathogen–vector interactions and outbreak reports, the main hosts of VEEV are rodents and horses, whereas humans are considered dead-end hosts. However, other susceptible species have been identified in the SLR (Table 37).

TABLE 37 Susceptible host species of Venezuelan equine encephalitis virus.

The SLR reported in the VEEV disease profile, identified the following susceptible species (updated until 31 December 2025; for references, see Annex A)
FIELD
Epidemiological studies carried out in the field

• **Pathogen was detected in the following animal species:**
  • Suidae: *Sus scrofa domesticus*
• **Antibodies were detected in the following animal species:**
  • Equidae: *Equus caballus*
  • Rodentia (Order): No families specified
• **Outbreaks reported to WOAH included the following species:**
  • Equidae: No species specified

EXPERIMENTS

• **Experimental studies demonstrated infection in:**
  • Bovidae: *Bos taurus*
  • Equidae: *Equus caballus*
  • Leporidae: *Oryctolagus cuniculus, Lepus europaeus*
  • Suidae: *Sus scrofa domesticus*

Clinical signs

Outcomes of a SLR on clinical signs in 27 equid study groups are displayed in Figure 42. Most study groups in both categories showed generic and neurological clinical signs.

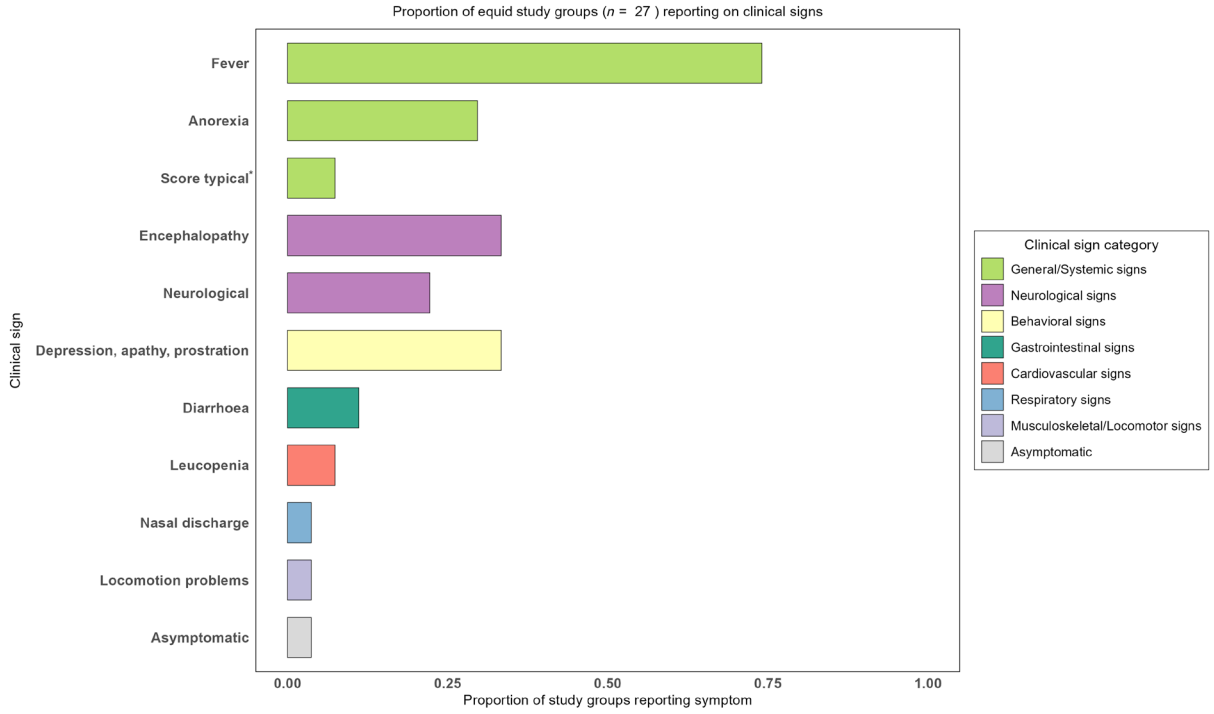


FIGURE 42 Clinical signs reported in the main hosts of VEEV. *Score typical: fever, neurological signs. Study group count per equid species: Horse $n = 24$; Ass $n = 3$. The SLR was updated until 31 December 2025; for references, see Annex A.

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 categorised 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 viraemia lasting 2–5 days, coinciding with the onset of fever and terminating with the production of neutralising 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.
- 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.

Incubation period

In equines, the incubation period for VEEV typically ranges from 12 h to 5 days. High fever appears within 12–24 h and neurologic signs can manifest up to 5 days after infection. Signs may persist for up to 2 weeks, with the duration and severity varying based on the specific virus strain and the equine species affected (Spickler, 2024a, 2024b, 2024c, 2024d; WOAH, 2020d; Annex A).

Morbidity and case fatality

In equines, morbidity and case fatality vary with the viral subtype. In epizootic outbreaks, morbidity can be very high (50%–100%) in unvaccinated equine populations, with case fatality rate reaching 50%–70%. Enzootic strains generally cause much lower morbidity and case fatality (Spickler, 2024a, 2024b, 2024c, 2024d; WOAH, 2020d; Annex A).

Zoonotic potential

Venezuelan equine encephalitis is a zoonotic disease (WHO, 1971).

3.24.5 | Transmission

VEEV is transmitted to vertebrate hosts through the bite of mosquitoes (Diptera; Culicidae). Evidence on the potential and likely competent mosquito species and their geographic distribution can be found in a dedicated Scientific Report on vectors (EFSA, 2026a). In sylvatic and peri-urban environments, enzootic strains are maintained in rodent–mosquito cycles. Epizootic strains emerge through viral mutations or recombination and are transmitted by a wider range of mosquito genera enabling explosive outbreaks. Equids infected with epizootic strains develop high-titre viraemia sufficient to infect mosquitoes, which amplifies the virus and sustains equine epidemics (Spickler, 2024a, 2024b, 2024c, 2024d; WOAH, 2020d).

3.24.6 | Diagnostic tests

Recommended tests (WOAH, 2025b) for the detection of the agent: 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.

Recommended tests (WOAH, 2025b) 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.

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

3.24.7 | Prevention and control

Vaccination

Vaccination is the most effective tool to prevent and control outbreaks. Both inactivated and live-attenuated vaccines are available for use in equids in endemic regions (Spickler, 2024a, 2024b, 2024c, 2024d; WOAH, 2020d).

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

Treatment

There is currently no specific antiviral treatment for VEEV infection. Supportive care in horses includes anti-inflammatory therapy, fluid support and intensive nursing (Spickler, 2024a, 2024b, 2024c, 2024d; WOAH, 2020d).

3.25 | Vesicular stomatitis virus (VSV)[27]

3.25.1 | Disease overview

Vesicular stomatitis virus (VSV) causes vesicular stomatitis, a vesicular disease affecting primarily horses, cattle and pigs. In livestock, clinical signs are indistinguishable from foot-and-mouth disease. VSV is transmitted by direct contact (including fomites) but may also be transmitted by *Culicoides* biting midges (Diptera; Ceratopogonidae) (WOAH, 2013a, 2025b).

VSV infection is a WOAH-notifiable disease, but it is not listed in the EU AHL.

3.25.2 | Agent

VSV is an enveloped, non-segmented, single stranded, negative-sense RNA virus that belongs to the family Rhabdoviridae, genus *Vesiculovirus*. The virion is bullet-shaped, approximately 70 nm in diameter and 170–200 nm in length. The genome of the virus encodes five structural proteins the nucleoprotein (N), phosphoprotein (P), the matrix protein (M), the glycoprotein (G) and the RNA-dependent RNA polymerase (L). The G glycoprotein mediates virus attachment and membrane fusion and is the principal antigen inducing virus-neutralising antibodies. (Kuzmin et al., 2009; Liu et al., 2021).

There are two distinct serogroups of VSV recognised: New Jersey (NJ) and Indiana (IND). There are four different serological complexes (subtypes) within the IND serogroup: IND-1 (classical IND), IND-2 (cocal virus), IND-3 (alagoas virus) and IND-4 (Morreton virus) (Liu et al., 2021).

3.25.3 | Geographical distribution

The WAHIS does not collect and report information on VSV since 2014. The virus is present endemically in the Americas, with studies reporting its presence in the USA and South America. Evidence from published studies describing natural infections with this agent as well as field epidemiological studies are collected in the SLR and summarised in Figure 43. All references can be found in Annex A and in the online version of the Disease Profile.[27]

FIGURE 43 Geographical distribution of epidemiological studies addressing the occurrence of VSV, as identified by the EFSA's SLR (covering years 1970–2025).

[27]Online version: https://animal-diseases.efsa.europa.eu/VSV.

3.25.4 | Animal hosts

Susceptible hosts

Based on epidemiological knowledge of host–pathogen–vector interactions and outbreak reports, the main hosts of VSV are cattle, equids and pigs, whereas humans are considered as dead-end hosts. However, other susceptible species have been identified in the SLR. The SLR summary is given in Table 38.

TABLE 38 Susceptible host species of vesicular stomatitis virus.

The SLR reported in the VSV disease profile, identified, or failed to identify the following susceptible species (updated until 31 December 2025; for references, see Annex A)
FIELD

Epidemiological studies carried out in the field
• **Pathogen was detected in the following animal species:**
  • No species specified
• **Antibodies were detected in the following animal species:**
  • Cervidae: No species specified
  • Equidae: *Equus caballus*
• **Outbreaks reported to WOAH included the following species:**
  • Bovidae: *Bos taurus, Ovis aries, Capra hircus*
  • Equidae: *Equus caballus*
  • Suidae: No species specified

EXPERIMENTS

• **Experimental studies demonstrated infection in:**
  • Bovidae: *Bos taurus*
  • Equidae: *Equus caballus*
  • Suidae: *Sus scrofa domesticus*

Clinical signs

Outcomes of a SLR of clinical signs in 48 pig, 15 cattle and 8 horse study groups are displayed in Figure 44. Predominantly, dermatological clinical signs were reported in all the study groups.

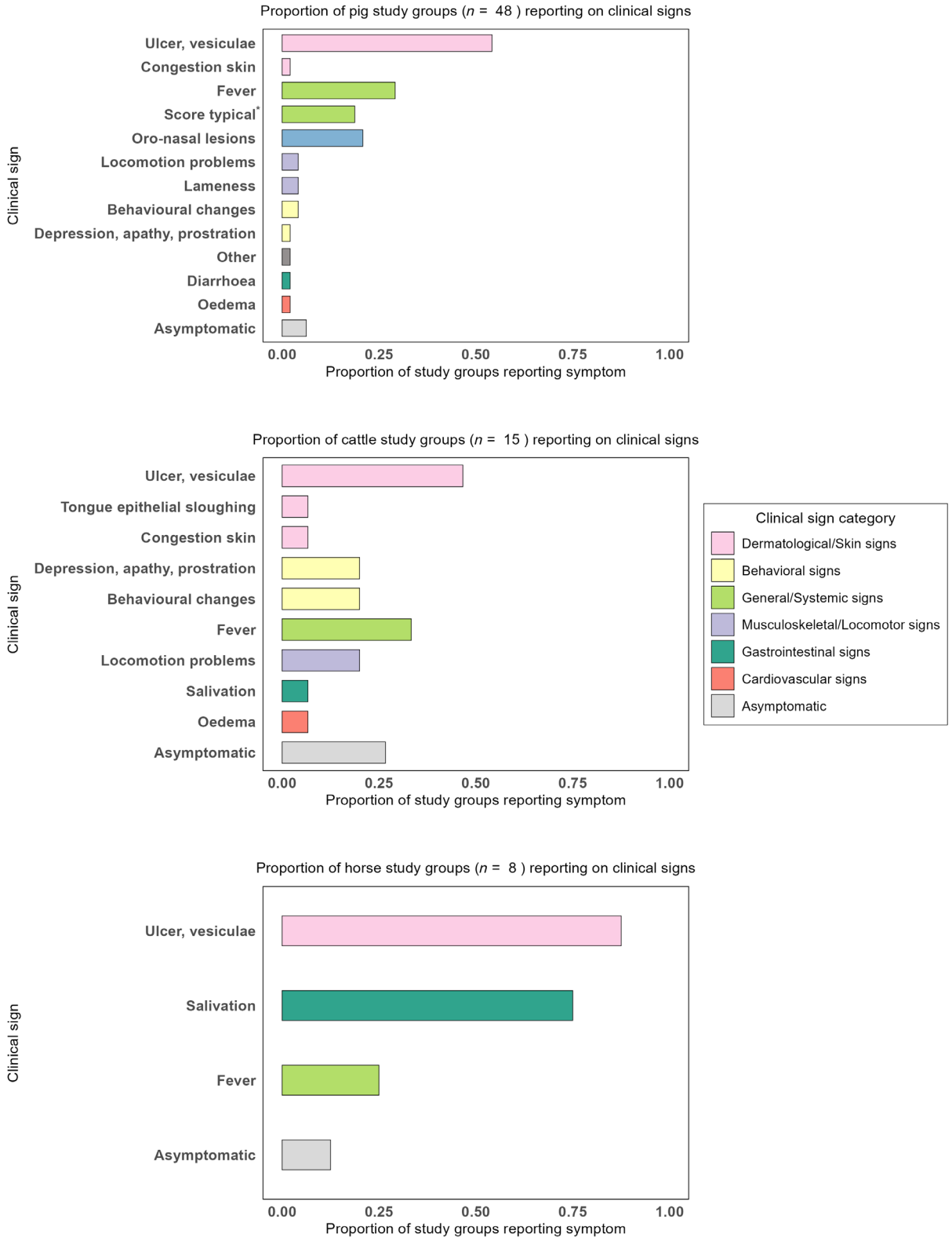


FIGURE 44 Clinical signs reported in the main hosts of VSV. *Score typical: skin lesions. The SLR was updated until 31 December 2025; for references, see Annex A.

Clinical signs associated with VSV resemble those of foot-and-mouth disease with the exception that VSV also affects horses. Clinical manifestation in horses, cattle and pigs is characterised by fever, vesicular formation that progresses to erosions and ulcerations in the oral cavity, lips, snout (pigs) and distal limbs (coronary band). As a result, excessive salivation, lip smacking, lameness and prostration can be observed (Annex A). Clinical signs can be observed for a median period of 9–10 days (Annex A).

Incubation period

The median incubation period observed in experimental studies is around 2 (1–3) days for horses and cattle. In pigs, the median incubation period is also 2 days, but incubation as long as 7 days has been reported (Annex A).

Morbidity and case fatality

Serological studies in affected premises have reported a high fraction of seropositive cattle or horses not showing clinical signs (Mumford et al., 1998). Similar morbidities are expected for pigs. Fatality in horses and cattle is reported to be rare (Reis Jr et al., 2009). In experimentally infected pigs, a fatality of 8.3% was observed (Annex A).

Zoonotic potential

VSV infection is a zoonotic disease (WOAH, 2013a).

3.25.5 | Transmission

The mechanisms of VSV transmission are not fully understood; however, both direct contact and insect-mediated transmission have been reported. Direct contact transmission appears to occur through contact with vesicular fluid, saliva or contaminated fomites with the virus entering a susceptible host through abrasions of the skin or mucous membranes. Biting midges of the genus *Culicoides* (Diptera; Ceratopogonidae) are potential biological vectors of VSV. Mechanical transmission by vectors is also possible. Evidence on the potential and likely competent vector species and their geographic distribution can be found in a dedicated Scientific Report on vectors (EFSA, 2026a).

Humans may become infected through direct contact with infected animals or tissues, but human-to-human transmission has not been demonstrated (Reis et al., 2009).

3.25.6 | Diagnostic tests

WOAH-recommended tests (WOAH, 2025b) for agent detection include virus isolation, ELISA for antigen detection, complement fixation test (CFT) and RT-PCR. Sensitivities (Se) and specificities (Sp) higher than 90% have been reported for the RT-PCR and the antigen ELISA test. Lower Se relative to the ELISA test has been reported for CFT (SLR; Alonso et al., 1991; Ferris et al., 2012).

WOAH-recommended tests for the detection of immune response: Liquid-phase blocking ELISA, competitive ELISA, virus neutralisation test (VNT) and complement fixation test (CFT). The sensitivity and specificity for VNT and ELISA were well above 90% (SLR; Workman et al., 1986; WOAH, 2025b).

Table 39 presents data on the sensitivity and specificity of diagnostic tests collected through SLR; reported values correspond to the median sensitivity and specificity when multiple studies investigated the same test and are only included when explicitly stated in the publications.

TABLE 39 Median sensitivity and specificity of tests to detect VSV/VSV antibodies reported in literature included in the systematic literature review.

Target	Test	Species	Sensitivity	*N* animal groups	Specificity	*N* animal groups	References
Antigen	C-ELISA	Cattle	94.1%	2	98.75%	2	Allende et al. (1992)
Antibody	VNT	Horse	98.4%	1	97.4%	1	Allende and Germano (1993)

Note: The SLR was updated until 31 December 2025; for references, see Annex A.

3.25.7 | Prevention and control

Vaccination

There are no commercial vaccines available for VSV.

Treatment

There is currently no specific antiviral treatment for VSV infection.

3.26 | West Nile fever virus (WNV)[28]

3.26.1 | Disease overview

West Nile virus (WNV) causes West Nile fever (WNF), a mosquito-borne viral disease. Birds are the main hosts for WNV with regular spillover events affecting humans and horses. WNF includes a variety of clinical signs, from asymptomatic to flu-like, encephalitis and death (Spickler, 2023a, 2023b; WOAH, 2025b).

West Nile fever is a WOAH-notifiable disease and is listed in the EU AHL under category E.

3.26.2 | Agent

WNV is an enveloped, single-stranded, positive-sense RNA virus belonging to the *Orthoflavivirus* genus within the Flaviviridae family. The virion has a spherical shape, with surface proteins arranged in an icosahedral-like symmetry. Its genome, approximately 10–11 kb in length, encodes 11 proteins: four structural proteins (C, Pr, M and E) and seven non-structural proteins (NS1, NS2A, NS2B, NS3, NS4A, NS4B and NS5). Phylogenetic analyses have identified up to nine distinct lineages of WNV based on genetic differences in the envelope and non-structural protein genes, with lineage 1 and lineage 2 being most prevalent and associated with severe human disease. In Europe, lineage 2 has been the predominant strain (Lu et al., 2024; May et al., 2011; Mukhopadhyay et al., 2005; Petersen et al., 2013).

3.26.3 | Geographical distribution

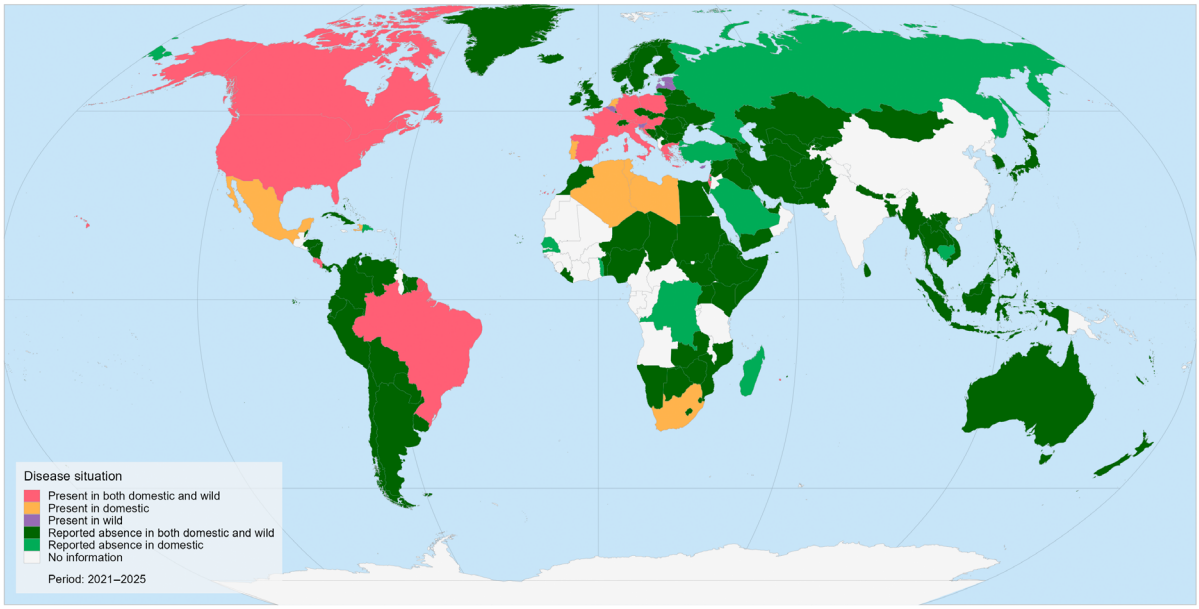


FIGURE 45 Geographical distribution of WNV detected events (2021–2025), as reported to WOAH.

WNV has been reported in North and South America, Africa, the Middle East, South-East Asia and Europe. In the EU, WNF cases have been detected over the past 5 years (Figure 45). Up-to-date maps based on WAHIS are available in the online version of the Disease Profile.[28]

3.26.4 | Animal hosts

Susceptible hosts

Based on epidemiological knowledge of host–pathogen–vector interactions and outbreak reports, the main hosts of WNV are passerine birds, whereas humans and horses are considered dead-end hosts. However, other susceptible species have been identified in the SLR. The SLR summary is given in Table 40.

[28]Online version: https://animal-diseases.efsa.europa.eu/WNV.

TABLE 40 Susceptible host species of West Nile virus.

The SLR reported in the WNV disease profile, identified or failed to identify the following susceptible species (updated until 31 December 2025; for references, see Annex A)

FIELD

- Epidemiological studies carried out in the field

Pathogen was detected in the following animal species:

- Accipitridae: No species specified
- Columbidae: No species specified
- Equidae: *Equus caballus*
- Falconidae: No species specified
- Hominidae: *Homo sapiens*
- Phasianidae: *Gallus gallus domesticus*
- Strigidae: No species specified
- Suidae: *Sus scrofa domesticus*

Antibodies were detected in the following animal species:

- Accipitridae: No species specified
- Bovidae: *Bos taurus, Capra hircus, Ovis aries*
- Camelidae: *Camelus dromedarius, Camelus bactrianus*
- Canidae: *Canis lupus familiaris*
- Columbidae: No species specified
- Equidae: *Equus caballus, Equus africanus asinus*
- Falconidae: No species specified
- Felidae: *Felis catus*
- Hominidae: *Homo sapiens*
- Phasianidae: *Gallus gallus domesticus*
- Strigidae: No species specified

Outbreaks reported to WOAH included the following species:

- Accipitridae: *Aquila heliaca, Accipiter gentilis, Accipiter nisus, Buteo buteo, Gyps fulvus*
- Anatidae: *Aythya nyroca, Chenonetta jubata*
- Bombycillidae: No species specified
- Cacatuidae: *Cacatua galerita*
- Camelidae: No species specified
- Canidae: *Canis lupus familiaris*
- Charadriidae: No species specified
- Columbidae: *Streptopelia decaocto, Columba livia, Columba palumbus*
- Corvidae: *Corvus corax, Corvus cornix, Corvus corone, Corvus frugilegus, Coloeus monedula, Pica pica, Garrulus glandarius*
- Equidae: No species specified
- Falconidae: *Falco cherrug, Falco rusticolus, Falco sparverius, Falco tinnunculus*
- Fringillidae: *Serinus canaria*
- Hirundinidae: *Delichon urbicum*
- Laridae: *Larus michahellis, Ichthyaetus melanocephalus*
- Paridae: *Cyanistes caeruleus, Parus major*
- Passeridae: *Passer domesticus*
- Pelecanidae: *Pelecanus rufescens*
- Phalacrocoracidae: *Phalacrocorax carbo*
- Phasianidae: *Gallus gallus domesticus*
- Phoenicopteridae: *Phoenicopterus chilensis, Phoenicopterus roseus, Phoenicopterus ruber*
- Psittacidae: *Psittacula krameri, Pionus maximiliani*
- Psittaculidae: *Glossopsitta pusilla*
- Scolopacidae: *Numenius arquata*
- Spheniscidae: *Spheniscus demersus, Spheniscus humboldti*
- Strigidae: *Otus scops, Strix nebulosa, Asio otus, Bubo bubo, Bubo scandiacus*
- Sturnidae: *Acridotheres tristis*
- Turdidae: *Turdus merula*

EXPERIMENTS

- **Experimental studies demonstrated infection in:**
  - Anatidae: No species specified
  - Bovidae: *Ovis aries*
  - Canidae: *Canis lupus familiaris*
  - Columbidae: No species specified
  - Corvidae: No species specified
  - Equidae: *Equus caballus*
  - Felidae: *Felis catus*
  - Fringillidae: *Serinus canaria*
  - Leporidae: No species specified
  - Phasianidae: *Gallus gallus domesticus, Meleagris gallopavo*
  - Strigidae: No species specified
  - Suidae: *Sus scrofa domesticus*

Clinical signs

Outcomes of a SLR on clinical signs in 21 bird study groups are displayed in Figure 46. Most study groups showed behavioural and neurological clinical signs.

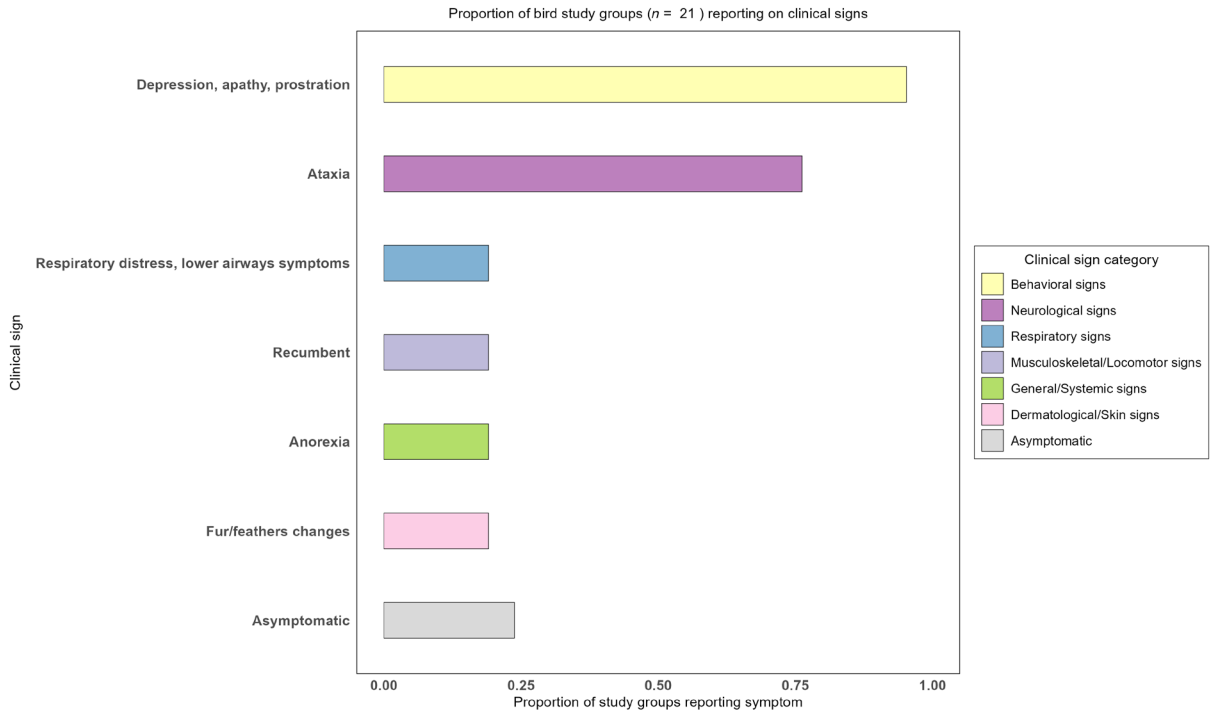


FIGURE 46 Clinical signs reported in the main hosts of WNV. Study group count per bird species: Goose $n = 15$; Turkey $n = 2$; Canary $n = 1$; Chicken $n = 1$; Duck $n = 1$; Pigeon $n = 1$. The SLR was updated until 31 December 2025; for references, see Annex A.

The clinical manifestation of WNF ranges from subclinical disease to influenza-like symptoms, encephalomyelitis/encephalitis and death.

In birds, WNV infection in susceptible species can cause several non-specific clinical signs, including weakness, lethargy, dehydration, emaciation, feather loss or fluffed feathers, recumbency, drooped head and anorexia. Neurological signs may also develop, such as head tilt, nystagmus, ataxia, tremors, paresis and seizures. In severe cases, WNV infection can be fatal.

In horses, most WNV-infected individuals show no clinical signs or only mild influenza-like illness with fever. In some cases, the infection may also lead to neurological complications. Neurological signs occur in approximately 10% of horses displaying influenza-like signs and may include loss of coordination, stumbling, falling, generalised weakness and muscle twitching. Affected horses may experience seizures, drooping lips, repetitive lip smacking and a lowered head position. Teeth grinding and heightened sensitivity to touch or sound can also be observed. In severe cases, the horse may become recumbent and unable to rise.

Incubation period

The incubation period for WNV is usually 2–6 days but can reach up to 21 days (Spickler, 2023a, 2023b; Annex A).

Morbidity and case fatality

In **birds**, morbidity and case fatality rates vary significantly between species. Some species appear resistant, while others suffer fatal neurological disease. In highly susceptible species, such as corvids, morbidity and case fatality rates may approach 100% during an outbreak in a local population. However, some bird species, including waterfowl and chickens, rarely show clinical signs of disease. In **horses**, the morbidity rate of WNF in a naïve population varies between studies and viral lineage. In Europe, approximately 10%–20% of infected horses display clinical signs of WNF, such as fever and decreased general demeanour. Among horses exhibiting clinical signs, around 1%–10% develop West Nile encephalitis. The case fatality rate due to neurological WNF is approximately 30% (Spickler, 2023a, 2023b; Annex A).

Zoonotic potential

West Nile fever is a zoonotic disease (WHO, 2024c).

3.26.5 | Transmission

WNV is transmitted to vertebrate hosts through the bite of mosquitoes (Diptera; Culicidae). Evidence on the potential and likely competent vector species and their geographic distribution can be found in a dedicated Scientific Report on vectors (EFSA, 2026a).

WNV is sustained through an enzootic transmission cycle between mosquitoes and birds. Most WNF cases occur during the seasons more favourable for mosquito activity, typically from spring to autumn. The main introduction route into new areas is through migratory birds. Horses and humans are considered dead-end hosts for WNV because they develop low viraemia, meaning the virus does not reach levels sufficient for further transmission (Hayes et al., 2005; Kramer et al., 2008; Petersen et al., 2013).

3.26.6 | Diagnostic tests

Recommended tests (WOAH, 2025b) for the detection of the agent: Nested RT-PCR, real-time RT-PCR, immunohistochemstry and isolation in tissue culture.

In general, virus isolates are more easily obtained from avian specimens and mosquitoes, with horses and humans being lesser sources. Various bird tissues, including the brain, heart and liver, can be successfully used for virus isolation. However, detecting the virus in live, clinically ill horses is challenging due to fleeting viremia. For deceased encephalitic horses, brain tissue (especially the hindbrain and medulla) and the spinal cord are preferred specimens for virus isolation.

Recommended tests (WOAH, 2025b) for the detection of immune response: IgM capture ELISA, IgG indirect and competitive ELISAs, plaque-reduction neutralisation (PRN) and virus neutralisation test (VNT).

The IgM capture ELISA is effective for detecting recent natural exposure to WNV in equines, as WNV-specific IgM antibodies typically appear 7–10 days post-infection and persist for 1–2 months. Most horses with West Nile encephalitis test positive by the time clinical signs emerge. Neutralising antibodies are detectable by 2 weeks post-infection and may persist for over a year.

In birds, serological tests include ELISA, haemagglutination inhibition, virus neutralisation and plaque reduction neutralisation (PRN) assays. Some assays may cross-react with related flaviviruses, such as Usutu virus, St. Louis encephalitis virus or Japanese encephalitis virus. Among these, PRN is the most specific for WNV. When cross-reactivity is a concern, comparative titres against related flaviviruses should be assessed.

Interpretation of serological results must account for vaccination history, especially when using PRN, VNT or IgG ELISA. The IgM capture ELISA can also be applied to birds and other species if appropriate species-specific capture antibodies (e.g. anti-chicken IgM) are available. PRN is suitable for use across species, including avian hosts.

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

3.26.7 | Prevention and control

Vaccination

Prophylactic immunisation with vaccines is the most effective control strategy against WNF in endemic areas (Spickler, 2023a, 2023b; WOAH, 2025b). Currently, vaccines against WNF are available only for horses, with two inactive and one live vaccine approved in the EU (EMA, 2025).

Treatment

There is currently no specific antiviral treatment for WNV infection. Management is primarily supportive and should be initiated early, focusing on rest and good husbandry practices. Horses with neurological signs often require specialised hospital care (Petersen et al., 2013; Spickler, 2023a, 2023b; WOAH, 2025b).

GLOSSARY AND ABBREVIATIONS

Disease glossary

Causative Agent	Abbreviation	Disease	Abbreviation
African horse sickness virus	AHSV	African horse sickness	AHS
Akabane virus	AKAV	Akabane	AKA
Besnoitia besnoiti	*B. besnoiti*	Besnoitiosis	Besno
Bluetongue virus	BTV	Bluetongue	BT
Borrelia burgdorferi s.l.	*B. burgdorferi* s.l.	Lyme Disease	Lyme

Causative Agent	Abbreviation	Disease	Abbreviation
Bovine ephemeral fever virus	BEFV	Bovine ephemeral fever	BEF
Cache Valley virus	CVV	Cache valley/Bunyamwera disease	Cache
Coxiella burnetii	*C. burnetii*	Q-fever	Q-fever
Crimean Congo haemorrhagic fever virus	CCHFV	Crimean Congo haemorrhagic fever	CCHF
Eastern equine encephalitis virus	EEEV	Eastern equine encephalitis	EEE
Epizootic haemorrhagic disease virus	EHD V	Epizootic haemorrhagic disease	EHD
Equine infectious anaemia virus	EIA	Equine infectious anaemia	EIA
Japanese encephalitis virus	JEV	Japanese encephalitis	JEV
Leishmania infantum	*L. infantum*	Leishmaniosis	*Leishmania*
Lumpy skin disease virus	LSDV	Lumpy skin disease	LSD
Rift Valley fever virus	RVFV	Rift Valley fever	RVF
Schmallenberg virus	SBV	Schmallenberg	SB
Shuni virus	SHUV	Shuni	Shuni
St Louis encephalitis virus	SLE	St Louis encephalitis	SLE
Tick borne encephalitis virus	TBEV	Tick borne encephalitis	TBE
Trypanosoma vivax	*T. vivax*	Trypanosomiasis	Tryp
Trypanosoma evansi	*T. evansi*	Surra	Surra
Venezualan equine encephalitis virus	VEEV	Venezualan equine encephalitis	VEE
Vesicular stomatitis virus	VSV	Vesicular stomatitis	VS
West Nile virus	WNV	West Nile fever	WNF
Western equine encephalitis virus	WEEV	Western equine encephalitis	WEE

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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APPENDIX A

Protocol: Translation of Terms of Reference (TORs) into subquestions, evidence needs and reporting methods (Table A1)

TABLE A1 Protocol for the assessment of TOR 1.1.

ToR1.1.	Question	Evidence needs	Data collection	Reporting methods
Disease overview	What type of pathogen causes the disease? Is it listed under the EU AHL?	Legal status (AHL/WOAH), short description and historical context of vector-borne disease agent	Information extracted from DP "Disease overview" section	Narrative summary
Agent	What is the taxonomy (family, genus, species/serogroup/serotype)?	Taxonomy, subtype classification, antigenic/phylogenetic markers as reported in DP	Information extracted from DP "Agent" section	Narrative summary
Geographic distribution	Is the pathogen present in the EU? What is known about global distribution?	EU presence/absence, global distribution, trends over time	Information extracted from DP "Geographic distribution" section	Narrative summary of evidence provided from **SLR geographic distribution** in DP. Refer to DP for SLR review protocol and up-to-date distribution maps
Animal hosts	Which animals are primary hosts and reservoir hosts? Clinical signs? Incubation period? Morbidity/case fatality? Zoonotic status?	Host lists, reported cases, studies with confirmed infection, clinical descriptions, incubation period	Extracted data from DP sections "Animal hosts" and "Zoonotic aspects"	Narrative summary of quantitative and/or qualitative evidence collected through **SLR on experimental infections** in DP. Expert judgement to complete gaps
Transmission	Through which transmission routes can the VBD agent be transmitted?	Transmission routes, vector group	Extract data from EFSA profile "Transmission" and "Vectors" section	Narrative summary of quantitative and/or qualitative evidence collected through **SLR on experimental infections** and **SLR on vector competence** in DP
Diagnostic tests	Which diagnostic tests are available for pathogen and immune response detection in hosts? What is their performance?	Diagnostic methods accuracy data (Se/Sp)	Extract data from DP "Diagnostic tests" section, including evidence tables	Narrative summary of quantitative and/or qualitative evidence collected through **SLR on diagnostic tests**
Vaccination	Which vaccines are used worldwide? Effectiveness/safety? Which are approved in the EU?	Vaccine types, effectiveness and safety evidence, EU authorisations	Extract data from DP "Vaccination" section, noting products authorised in EU	Narrative summary of quantitative and/or qualitative evidence collected through **SLR on vaccinations**
Treatments	Specific medicinal treatments for affected animals?	Effectiveness of available therapeutic options	Extract data from DP "Treatment" section (if populated)	Narrative summary of quantitative and/or qualitative evidence collected through **SLR on treatments**

Note: EFSA Disease Profile version: https://animal-diseases.efsa.europa.eu/ – dd/mm/yyyy.

Abbreviations: AHL, animal health law; DP, disease profile; SLR, systematic literature; WOAH, World Organization of Animal Health.

APPENDIX B

Classification of data availability for selected vector-borne diseases

TABLE B1 Number of publications identified through systematic literature reviews for select pathogens.

VBD	Publication count	Data availability
AHSV	66	++
AKAV	77	++
B. besnoiti	82	++
B. burgdorferi s.l.	486	+++
BEFV	67	++
BTV	380	+++
C. burnetii	217	+++
CCHFV	73	++
CVV	24	+
EEEV	31	+
EHDV	72	++
EIAV	55	++
JEV	79	++
L. infantum	560	+++
LSDV	87	++
RVFV	317	+++
SBV	84	++
SHUV	16	+
SLEV	212	+++
T. evansi	72	++
T. vivax	121	+++
TBEV	98	++
VEEV	31	+
VSV	33	+
WEEV	18	+
WNV	191	+++

Note: +, 0–50; ++, 51–100; +++, > 100.

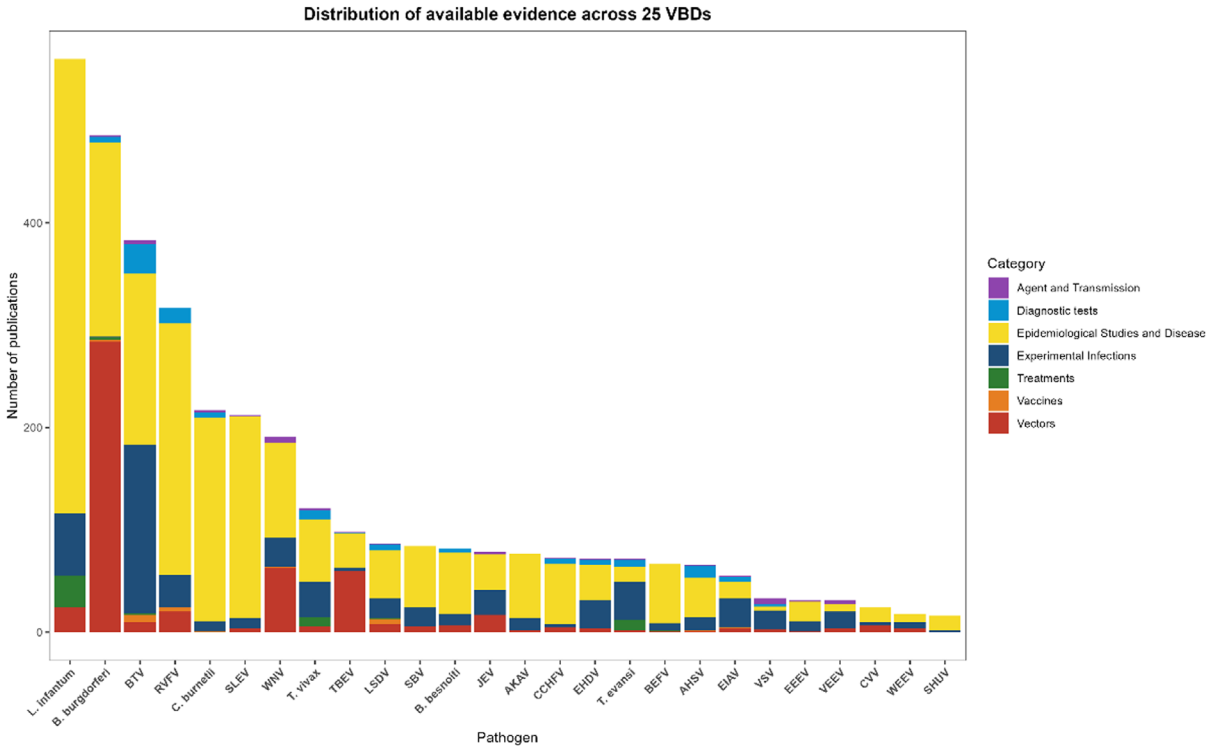

FIGURE B1 Available evidence by category for the 25 vector-borne diseases (WEEV and EEEV are considered together). *Y*-axis refers to the publications that were considered eligible in the SLRs.

ANNEX A

References of Systematic literature reviews supporting the vector-borne diseases knowledge maps

Annex A is available under the Supporting Information section on the online version of the Scientific Report.