African Horse Sickness Fact Sheet

1. Disease overview

African Horse Sickness is an **infectious**, **non-contagious**, **vector-borne viral** disease affecting members of the *Equidae* family. It is caused by **African Horse Sickness virus** (AHSV), an orbivirus of the family *Reoviridae*. The disease manifests with severe respiratory and circulatory impairment and presents with varying severity across its four clinical forms.

African horse sickness is a **WOAH-notifiable disease**.

African horse sickness is listed in the **European Animal Health Law** under categories **A, D, E**.

2. Agent

AHSV is a **non-enveloped**, **double-stranded RNA virus** of the genus *Orbivirus* within the family *Reoviridae*. 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 neutralizing antibodies.

Nine serotypes (AHSV-1 to AHSV-9) have been identified, with partial 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.

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 minutes. It is however inactivated by oxidizing agents and prolonged heating.

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 WAHIS data, the agent was not reported in the EU in the last 2 years.

For more detailed information and dynamic maps, visit the *Geographical Distribution* section of the online **disease profile** (accessible via the button in the top right corner).

4. Animal hosts

4.1. Primary animal species affected

All equids are susceptible to AHSV. **Horses** are the most severely affected species, while **mules** show intermediate susceptibility. **Donkeys** and especially **zebras** often experience subclinical

infection; zebras are considered the principal reservoir hosts and play an important role in maintaining virus circulation.

Although very rare, **dogs** may develop peracute, often fatal disease after ingestion of infected horsemeat, but they are not preferred hosts for *Culicoides* spp. and are unlikely to contribute to transmission.

Serological evidence of exposure has been reported in a wide range of other species, including wild carnivores such as hyenas, jackals, African wild dogs, cheetahs and lions, likely through feeding on infected equids. Antibodies have also been detected in several herbivores, including dromedary camels, sheep, goats, African elephants, and rhinoceros.

4.2. Clinical Signs

African horse sickness occurs in **four recognized 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 edema. Death occurs within 24–48 hours, with mortality close to 100%.

Cardiac form (subacute): characterized by fever, swelling of the head, eyelids, and supraorbital fossae, with variable dyspnea. The course is longer than the pulmonary form, and mortality is typically 50–70%. This form occurs mainly in horses and occasionally in mules.

Mixed form (acute): combines respiratory distress with edema of the head and neck and is seen in horses and mules. Mortality rates are high, averaging around 70%.

Horse sickness fever (mild): Clinical signs are limited to transient fever and mild malaise, followed by recovery. Predominantly affects donkeys and zebras but may also occur in partially immune horses.

4.2.1. Incubation Period

The incubation period of African horse sickness, defined as the interval between infection and the onset of clinical signs, in equids is considered to range between **3 and 21 days** and depends on host species, immune status, and route of infection.

In **horses**, clinical signs usually appear within 3 to 7 days after infection, but in some cases the period may extend up to two 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.

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

4.2.2. Morbidity and mortality

Horses are highly susceptible to AHSV, with mortality ranging from **50–95%**, 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.

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.

Other species, such as **dogs**, may die from peracute infection after consuming infected meat, but they are not relevant for transmission.

4.2.3. Zoonotic Potential

AHSV is not known to infect humans under natural conditions.

5. Transmission

African horse sickness is primarily transmitted by biting midges of the genus *Culicoides*. The virus does not spread via direct contact between equids, though iatrogenic transmission through blood is possible. Seasonal and climatic factors influence vector abundance, driving the cyclic occurrence of outbreaks. Wind can facilitate long-distance dispersal of infected vectors, and adult *Culicoides* can survive mild winters, contributing to virus persistence.

→ *Culicoides* midges have been reported in the EU in the past 2 years.

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

6. Diagnostic tests

WOAH-recommended tests for **agent detection** are virus isolation and reverse transcription 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).

The following table presents data on the sensitivity and specificity of several diagnostic tests from studies deemed eligible according to EFSA's systematic literature review protocol; 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:

Target	Test	Species	Specificity	N studies	Sensitivity	N studies
Antigen	ELISA	Horse	100%	3	100%	3
Nucleic acid	RT-PCR	Horse	99.9%	1	97.8%	1
Virus	Virus isolation	Horse	100.0%	1	72.1%	2
Antibody	Blocking ELISA	Horse	98.5%	2	99.2%	6
Antibody	Competitive ELISA	Horse	100%	3	100%	4
Antibody	ELISA	Horse	100%	3	100%	3
Antibody	Sandwich ELISA	Horse	100%	1	97.4%	1
Antibody	IFAT	Ass	83.3%	1	98.0%	1
Antibody	IFAT	Horse	83.3%	1	98.0%	1

7. Prevention and control

7.1. Vaccination

Monovalent and polyvalent attenuated-live vaccines are available for horses, mules, and donkeys in endemic regions.

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

7.2. Treatment

There is currently **no specific antiviral treatment** for AHSV infection, and management is primarily **supportive** on an individual basis. In endemic areas, control relies on vector management, movement restrictions, quarantine, and outbreak management.

In accordance with the Animal Health Law (AHL) in the EU, susceptible species kept at in the affected holdings shall be culled to prevent further spread of the pathogen.