

*Atlas of
Transboundary
Animal Diseases*

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African horse sickness

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ETIOLOGY

Classification of the causative agent

African horse sickness (AHS) is caused by a virus of the family *Reoviridae* of the genus *Orbivirus*. There are nine antigenically distinct serotypes of AHS virus (AHSV) identified by virus neutralization, but some cross-reaction has been observed between 1 and 2, 3 and 7, 5 and 8, and 6 and 9. No cross-reactions with other known orbiviruses have been observed.

Resistance to physical and chemical action

Temperature:

Relatively heat stable, especially in presence of protein. AHSV in citrated plasma still infective after heating at 55–75°C/131–167°F for 10 minutes. Minimal loss of titer when lyophilized or frozen at –70°C/–94°F with Parker Davis Medium. Infectivity is remarkably stable at 4°C/39°F, particularly in the presence of stabilizers such as serum and sodium oxalate, carbolic acid and glycerine: blood in OCG can remain infective >20 years. Can be stored >6 months at 4°C/39°F in saline with 10% serum. Fairly labile between –20°C/–4°F and –30°C/–22°F.

pH:

Survives pH 6.0–12.0. Readily inactivated below pH 6.0. Optimal pH is 7.0 to 8.5.

Chemicals/disinfectants:

Inactivated by formalin (0.1%) for 48 hours, β-propiolactone (0.4%), and binary ethyleneimine. Resistant to lipid solvents. Inactivated by acetic acid (2%), potassium peroxydisulfate/sodium chloride – Virkon® S (1%), and sodium hypochlorite (3%).

Survival:

Putrefaction does not destroy the virus: putrid blood may remain infective for >2 years, but virus is rapidly destroyed in meat by rigor mortis (lowering pH). Vaccine strains survive well in lyophilized state at 4°C/39°F.

EPIDEMIOLOGY

- Infectious disease is transmitted by *Culicoides* spp. that occurs regularly in most countries of sub-Saharan Africa
- At least two field vectors are involved: *Culicoides imicola* and *C. bolitinos*
- The disease has both a seasonal (late summer/autumn) and an epizootic cyclical incidence, with disease associated with drought followed by heavy rain
- Major epizootics in southern Africa are strongly linked with warm (El Niño) phase of the El Niño/Southern Oscillation (ENSO)
- Mortality rate in horses is 70%-95%, mules around 50%, and donkeys around 10%
- other than mild fever, infection in zebra and African donkeys is subclinical
- viremia may be extended in zebra (up to 40 days)

Hosts

- Usual hosts are equids: horses, mules, donkeys and zebra
- Reservoir host are believed to be zebras
- Antibody is found in camels, African elephants, and black and white rhinoceroses, but their role in epidemiology is unlikely to be significant
- Dogs have peracute fatal infection after eating infected horsemeat, but are not a preferred host by *Culicoides* spp. and unlikely to play a role in transmission

Transmission

- Not contagious by contact
- Usual mode of transmission is the biological vector *Culicoides* spp. *C. imicola* and *C. bolitinos* are known to transmit AHSV in the field; *C. imicola* appears to be the principal vector
- The North American species *C. variipennis* is an efficient vector in the laboratory

- Occasional mode of transmission: mosquitoes – *Culex*, *Anopheles* and *Aedes* spp.; ticks – *Hyalomma*, *Rhipicephalus*; and possibly biting flies – *Stomoxys* and *Tabanus*
- Moist mild conditions and warm temperatures favor the presence of insect vectors
- Wind has been implicated in dispersal of infected *Culicoides* in some epidemics
- Movement of *Culicoides* spp. over long distances (700 km over water, 150 km over land) via wind has been postulated

Sources of virus

- Viscera and blood of infected horses
- Semen, urine and nearly all secretions during viremia, but no studies have documented transmission
- Viremia usually lasts 4-8 days in horses but may extend up to 21 days; in zebras viremia may last up to 40 days
- Recovered animals do not remain carriers of the virus

Occurrence

AHS is endemic in the central tropical regions of Africa, from where it spreads regularly to Southern Africa and occasionally to Northern Africa. All serotypes of AHS occur in eastern and southern Africa. Only AHS serotype 9 and 4 have been found in West Africa from where they occasionally spread into countries surrounding the Mediterranean.

A few outbreaks have occurred outside Africa in the Near and Middle East (1959-1963), Spain (1966, 1987-1990), Portugal (1989), Saudi Arabia and Yemen (1997) and the Cape Verde Islands (1999). But recent northward expansion of the main African vector (Afro-Asiatic species *C. imicola*) and bluetongue virus into the Mediterranean Basin of Europe now threatens that region and beyond to AHS.

DIAGNOSIS

Incubation period is usually 7-14 days, but may be as short as 2 days. For the purposes of the OIE *Terrestrial Code*, the infective period for AHSV shall be 40 days for domestic horses.

Clinical diagnosis

- There are four principal manifestations of disease
- In the majority of cases, the subclinical cardiac form is suddenly followed by marked dyspnea and other signs typical of the pulmonary form
- A nervous form may occur, though it is rare
- Morbidity and mortality vary with the species of animal, previous immunity and the form of the disease
- Horses are particularly susceptible where mixed and pulmonary forms tend to predominate; mortality rate is usually 50% to 95%
- Mules: mortality is about 50%; European and Asian donkeys: mortality is 5%-10%; African donkeys and zebra: mortality is rare
- Animals that recover from AHS develop good immunity to the infecting serotype and partial immunity to other serotypes

Subclinical form (Horse sickness fever)

- Fever (40-40.5°C/104°F-105°F)
- Mild form; general malaise for 1-2 days
- Very rarely results in death

Subacute or cardiac form

- Fever (39-41°C/102-106°F)
- Swelling of the supraorbital fossa, eyelids, facial tissues, neck, thorax, brisket and shoulders
- Mortality usually 50% or higher; death usually within one week

Acute respiratory or pulmonary form

- Fever (40-41°C/104-106°F)
- Dyspnea, spasmodic coughing, dilated nostrils with frothy fluid oozing out
- Redness of conjunctivae
- Nearly always fatal; death from anoxia within one week

Mixed form (cardiac and pulmonary)

- Occurs frequently
- Pulmonary signs of a mild nature that do not progress, edematous swellings and effusions
- Mortality: about 70%-80% or greater

Lesions

Respiratory form

- interlobular edema of the lungs
- hydropericardium, pleural effusion
- edema of thoracic lymph nodes
- petechial hemorrhages in pericardium
- mucosa and serosa of small and large intestines may exhibit hyperemia and petechial hemorrhages

Cardiac form

- subcutaneous and intramuscular gelatinous edema
- epicardial and endocardial ecchymoses; myocarditis
- hemorrhagic gastritis

Differential diagnosis

- Anthrax
- Equine infectious anemia
- Equine viral arteritis
- Trypanosomiasis
- Equine encephalosis
- Piroplasmiasis
- Purpura hemorrhagica
- Hendra virus

Laboratory diagnosis

Samples

Virus isolation

- Unclotted whole blood collected in an appropriate anticoagulant at the early febrile stage and sent at 4°C/39°F to the laboratory
- Spleen, lung and lymph node samples collected from freshly dead animals are placed in appropriate transport media and sent at 4°C/39°F to the laboratory; do not freeze

Serology

- Preferably paired serum samples should be taken 21 days apart and kept frozen at -20°C/-4°F

Procedures

Virus isolation

- Cell cultures, such as baby hamster kidney-21 (BHK-21), monkey stable (MS) or African green monkey kidney (Vero)
- Intravenously in embryonated eggs
- Intracerebrally in newborn mice

Virus identification

- Enzyme-linked immunosorbent assay (ELISA) – rapid detection of AHSV antigen in spleen and supernatant from cell culture
- Virus neutralization (VN) – until recently the ‘gold standard’ for serotyping virus isolates, but takes 5 days
- RT-PCR assay for the specific detection of AHSV genome has been developed; this assay can be used to detect viral RNA in blood collected in EDTA, homogenized equid tissue, or mouse tissue and cell culture fluids
- Real-time PCR – detects all 9 serotypes

AHSV serotyping

- VN test has been the method of choice for serotyping as well as the ‘gold’ standard test for identifying AHSV’s isolated from the field using type specific antisera
- Recent development of a type-specific RT-PCR for identification and differentiation of the nine AHSV serotypes provides a method of serotyping isolates in tissue samples within a few hours. There was perfect agreement between the RT-PCR and the VN test
- Typing of nine AHS serotypes has also been performed with probes developed from a set of cloned full length VP2 genes

Serological diagnosis

Horses that survive natural infection develop antibodies against the infecting serotype within 8-12 days post-infection.

- Indirect ELISA (prescribed test in the *OIE Terrestrial Manual*)
- Complement fixation (prescribed test in the *OIE Terrestrial Manual*)
- Immunoblotting
- Virus neutralization: (alternative test in the *OIE Terrestrial Manual*) – used for serotyping
- Immunodiffusion
- Hemagglutination inhibition

PREVENTION AND CONTROL

No efficient treatment available

Sanitary prophylaxis

Free areas, regions and countries

- Identify the virus and serotype
- Establish strict quarantine zone and movement controls
- Consider euthanasia of infected and exposed equids
- Stable all equids in insect-proof housing, at a minimum from dusk to dawn when *Culicoides* are most active
- Establish vector control measures: destroy *Culicoides* breeding areas; use insect repellents, insecticides, and/or larvicides
- Monitor for fever at least twice daily: place pyrexial equids in insect-free stables or euthanize
- Consider vaccination
 - identify vaccinated animals
 - available vaccines are attenuated
 - produce viremia, and may theoretically reassort with the outbreak virus
 - may be teratogenic

Affected areas, regions and countries

- Annual vaccination
- Vector control

Medical prophylaxis

Vaccination of non-infected horses:

- Polyvalent live attenuated vaccine – commercially available in certain countries
- Monovalent live attenuated vaccine – after virus has been typed
- Monovalent inactivated vaccine – no longer commercially available
- Serotype specific subunit vaccine – currently in development



Photo 1. **African horse sickness:** Horse, head. Marked swelling of supraorbital fossa.
[Source: OVI/ARC]



Photo 2. **African horse sickness:** Horse, head. Non-pitting edema in the adipose tissue within the supraorbital fossa.
[Source: FV/UCM]



Photo 3. **African horse sickness:** Horse, conjunctiva. Severe edema.
[Source: OVI/ARC]

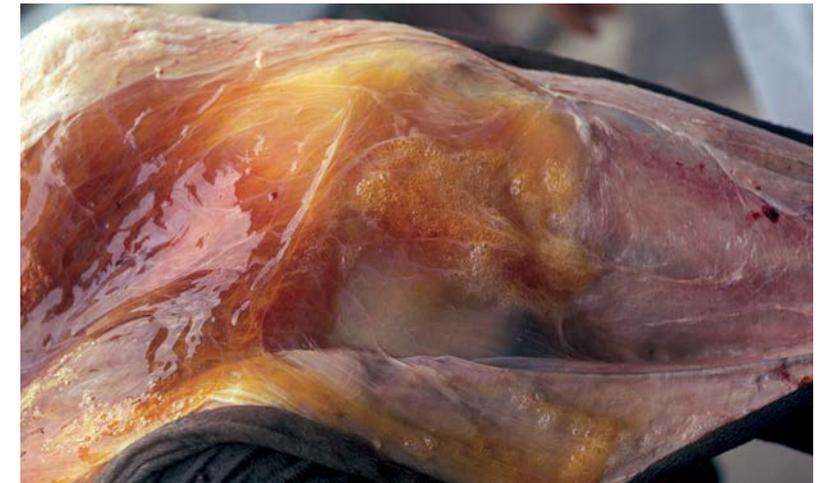


Photo 4. **African horse sickness:** Horse, shoulder. Severe subcutaneous edema.
[Source: OVI/ARC]



Photo 5. **African horse sickness:** Horse. Abundant white foamy nasal discharge.
[Source: PIADC]



Photo 6. **African horse sickness:** Horse, lung. Severe pulmonary edema with hydrothorax; edema widening the interlobular septa and subpleural space.
[Source: PIADC]

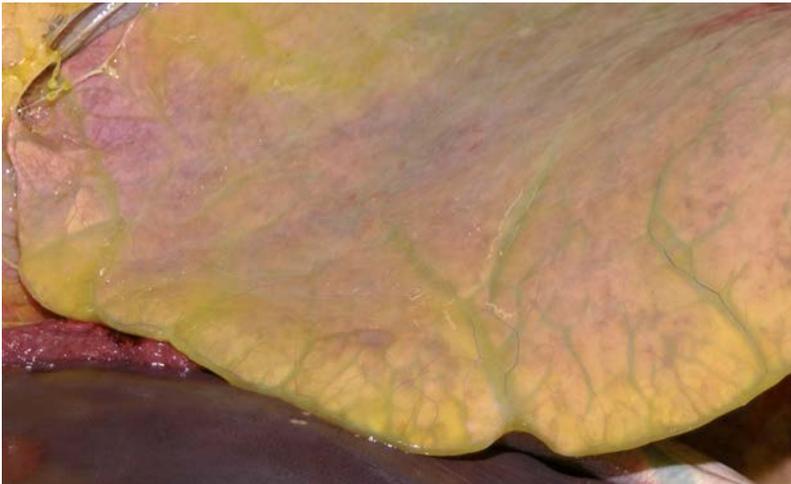


Photo 7. **African horse sickness:** Horse, lung. Marked pulmonary edema; edema expanding the interlobular septa and subpleural space.
[Source: PIADC]

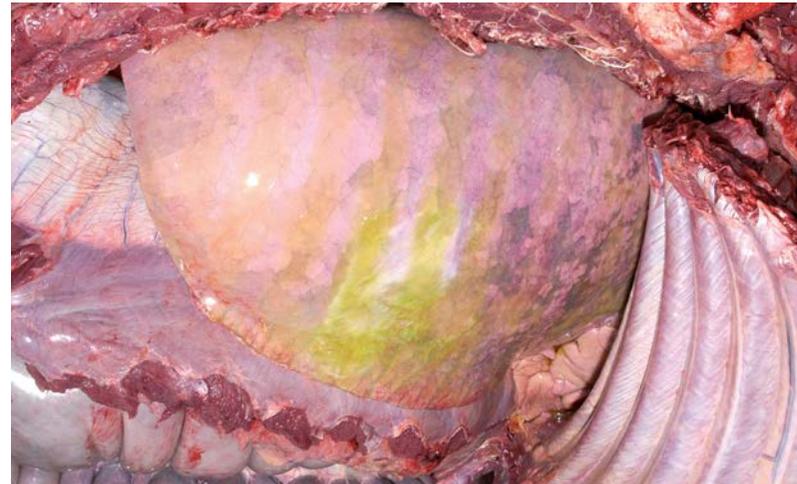


Photo 8. **African horse sickness:** Horse, lung. Severe pulmonary edema; rib impressions and yellow transudate on the pleura.
[Source: PIADC]

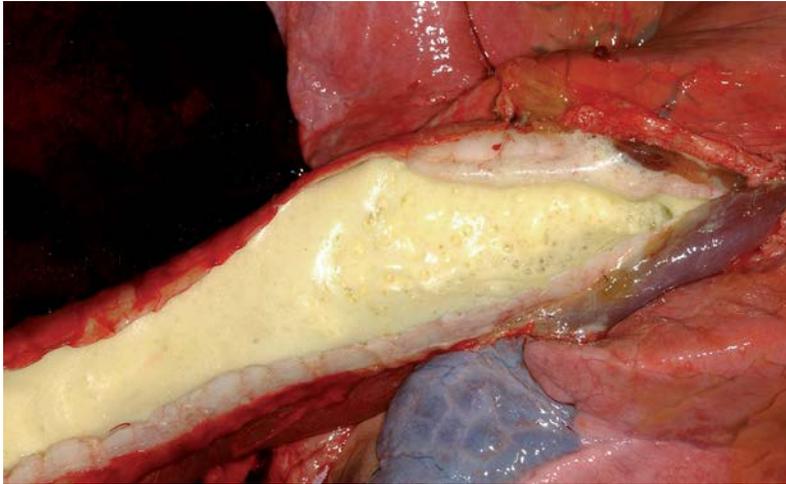


Photo 9. **African horse sickness:** Horse, trachea. Abundant white froth in trachea.
[Source: PIADC]



Photo 10. **African horse sickness:** Horse, lung. Severe pulmonary edema.
[Source: PIADC]

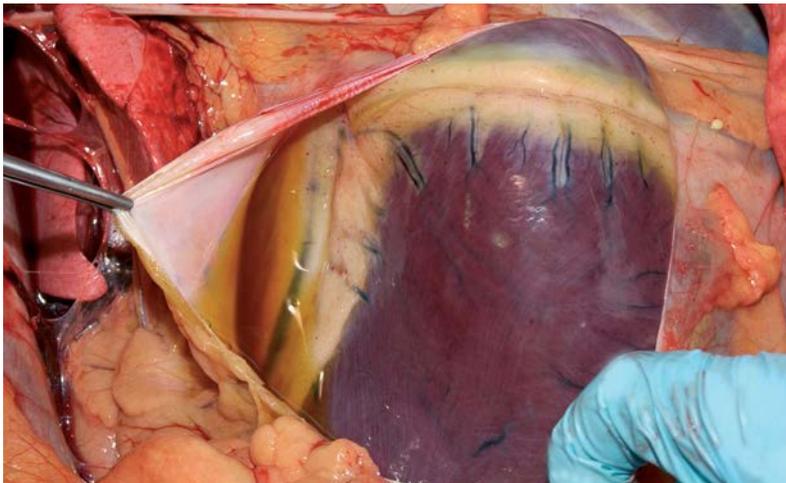


Photo 11. **African horse sickness:** Horse, heart. Moderate hydropericardium.
[Source: PIADC]



Photo 12. **African horse sickness:** Horse, heart, left ventricle. Severe endocardial hemorrhages.
[Source: PIADC]

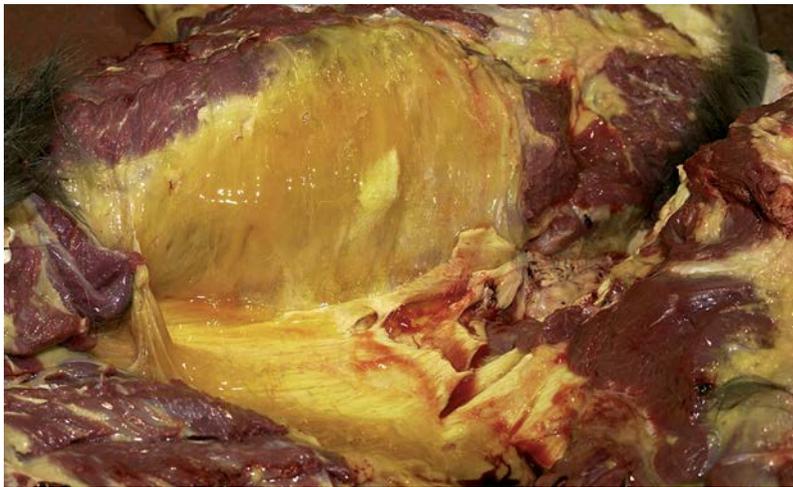


Photo 13. **African horse sickness:** Horse, cervical muscles and ligamentum nuchae. Severe intermuscular edema. [Source: PIADC]



Photo 14. **African horse sickness:** Horse, cervical muscles. Intermuscular edema. [Source: PIADC]



Photo 15. **African horse sickness:** Horse, cervical muscles. Subcutaneous and intermuscular edema. [Source: PIADC]



Photo 16. **African horse sickness:** Horse, large colon. Serosal petechiae and congestion. [Source: PIADC]