

AFRICAN HORSE SICKNESS

Aetiology Epidemiology Diagnosis Prevention and Control References

AETIOLOGY

Classification of the causative agent

Family Reoviridae, genus *Orbivirus*, 9 serotypes

Nine antigenically distinct serotypes of AHSV identified by virus neutralisation, 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 for 10 minutes. Minimal loss of titre when lyophilised or frozen at –70°C with Parker Davis Medium. Infectivity is remarkably stable at 4°C, particularly in the presence of stabilisers 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 in saline with 10% serum. Fairly labile between –20 and –30°C.
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 peroxymonosulfate/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 lyophilised state at 4°C.

EPIDEMIOLOGY

- Infectious disease is transmitted by *Culicoides* spp. that occur 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
 - Zebra may have extended viraemia (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 favour 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 viraemia, but no studies have documented transmission
- Viraemia usually lasts 4–8 days in horses but may extend up to 21 days; in zebras viraemia 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 (1959–63), Spain (1966, 1987–90), 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.

For more recent, detailed information on the occurrence of this disease worldwide, see the OIE World Animal Health Information Database (WAHID) interface [<http://www.oie.int/wahis/public.php?page=home>] or refer to the latest issues of the World Animal Health and the OIE *Bulletin*.

DIAGNOSIS

Incubation period is usually 7–14 days, but may be as short as 2 days

Clinical diagnosis

- Subclinical form: fever (40–40.5°C) and general malaise for 1–2 days
- Subacute or cardiac form: fever (39–41°C), swelling of the supraorbital fossa, eyelids, facial tissues, neck, thorax, brisket and shoulders. Death usually within 1 week
- Acute respiratory form: fever (40–41°C), dyspnoea, spasmodic coughing, dilated nostrils with frothy fluid oozing out, redness of conjunctivae, death from anoxia within 1 week
- A mixed form (cardiac and pulmonary) occurs frequently: pulmonary signs of a mild nature that do not progress, oedematous swellings and effusions, death from cardiac failure, usually within 1 week
- In the majority of cases, the subclinical cardiac form is suddenly followed by marked dyspnoea and other signs typical of the pulmonary form
- A nervous form may occur, though it is rare

Morbidity and mortality

- Morbidity and mortality vary with the species of animal, previous immunity and the form of the disease
- Pulmonary form: nearly always fatal; Cardiac form: usually 50% or higher; Mixed form: about 70–80% or greater. In contrast, horse sickness fever very rarely results in death

- Horses are particularly susceptible – 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 African horse sickness develop good immunity to the infecting serotype and partial immunity to other serotypes

Lesions

- Respiratory form: Interlobular oedema of the lungs, hydropericardium, pleural effusion, oedema of thoracic lymph nodes, petechial haemorrhages in pericardium
- Cardiac form: subcutaneous and intramuscular gelatinous oedema, epicardial and endocardial ecchymoses, myocarditis, haemorrhagic gastritis

Differential diagnosis

- Anthrax
- Equine infectious anaemia
- Equine viral arteritis
- Trypanosomosis
- Equine encephalosis
- Piroplasmosis
- Purpura haemorrhagica
- 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 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 to the laboratory; do not freeze

Serology

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

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

- ELISA – rapid detection of AHSV antigen in spleen and supernatant from cell culture
- Virus neutralisation (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, homogenised 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
- Complement fixation (prescribed tests in the OIE *Terrestrial Manual*)
- Immunoblotting
- Virus neutralisation: (alternative test in the OIE *Terrestrial Manual*) – used for serotyping
- Immunodiffusion
- Haemagglutination inhibition

For more detailed information regarding laboratory diagnostic methodologies, please refer to Chapter 2.5.1 African horse sickness in the latest edition of the OIE *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals* under the heading “Diagnostic Techniques”.

PREVENTION AND CONTROL

- No efficient treatment

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 pyrexical equids in insect-free stables or euthanise
- Consider vaccination
 - Identify vaccinated animals
 - Available vaccines are attenuated
 - Produce viraemia, 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

For more detailed information regarding vaccines, please refer to Chapter 2.5.1 African horse sickness in the latest edition of the *OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals* under the heading “Requirements for Vaccines and Diagnostic Biologicals”.

For more detailed information regarding safe international trade in terrestrial animals and their products, please refer to the latest edition of the *OIE Terrestrial Animal Health Code*.

REFERENCES AND OTHER INFORMATION

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- Mellor P.S. & Hamblin C. – African Horse Sickness: Review Article. *Vet. Res.* 35 (2004) 445–466

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The OIE will periodically update the OIE Technical Disease Cards. Please send relevant new references and proposed modifications to the OIE Scientific and Technical Department (scientific.dept@oie.int). Last updated October 2009.