AFRICAN HORSE SICKNESS

Aetiology Epidemiology Diagnosis Prevention and Control References

AETIOLOGY

Classification of the causative agent

African horse sickness (AHS) is caused by a virus of the family *Reoviridae* of the genus *Orbivirus.* There are 9 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 titre 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 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/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
  - viraemia 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 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, 4 and 2 have been found in North and 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), 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. 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 dyspnoea 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 1 week

Acute respiratory or pulmonary form

- Fever (40–41°C/104–106°F)
- Dyspnoea, spasmodic coughing, dilated nostrils with frothy fluid oozing out
- Redness of conjunctivae
- Nearly always fatal; death from anoxia within 1 week

Mixed form (cardiac and pulmonary)

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

Lesions

- Respiratory form:
  - interlobular oedema of the lungs
  - hydropericardium, pleural effusion
  - oedema of thoracic lymph nodes
  - petechial haemorrhages in pericardium
  - mucosa and serosa of small and large intestines may exhibit hyperaemia and petechial haemorrhages

- Cardiac form:
  - subcutaneous and intramuscular gelatinous oedema
  - epicardial and endocardial ecchymoses; myocarditis
  - hemorrhagic 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/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) or insect cells (KC)
- Intravenously in embryonated eggs
- Intracerebrally in newborn mice

Virus identification

- Enzyme-linked immunosorbent assay (ELISA) – rapid detection of AHSV antigen in blood, spleen and supernatant from cell culture
- Virus neutralization (VN) – until recently the 'gold standard' for typing as well as identifying virus isolates, but takes 5 days
- RT-PCR is a highly sensitive technique that allows the detection of a very low number of copies of RNA molecules
- Real-time PCR – detects all 9 serotypes

AHSV typing

- VN test has been the method of choice for typing as well as the 'gold' standard test for identifying AHSV's isolated from the field using type specific antisera
- Development of a type-specific gel-based RT-PCR and real-time RT-PCR using hybridisation probes for identification and differentiation AHSV genotypes provides a rapid typing method for AHSV in tissue samples and blood. There is a good correlation between the results obtained with the type-specific RT-PCR and the VN test, however, the sensitivity of these assays is lower than that obtained with the diagnostic group-specific real-time RT-PCR Typing of nine AHSV types 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.

- Blocking ELISA (prescribed test in the OIE *Terrestrial Manual*)
- Indirect ELISA (prescribed test in the OIE *Terrestrial Manual*)
- Complement fixation (prescribed test in the OIE *Terrestrial Manual*)

Virus neutralization **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 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 pyrexic equids in insect-free stables or euthanize
- 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

- At present only the live attenuated AHS vaccines (polyvalent or monovalent) are commercially available
- 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 Animal*s under the heading "Requirements for Vaccines".

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*.

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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 April 2013.