African Horse Sickness

Definition	African Horse Sickness (AHS) is a non-contagious, insect-borne infectious disease of equids. Although equids are the primary hosts, natural disease has been reported in other species. The disease is caused by African horse sickness virus that belongs to the family *Reoviridae*, genus *Orbivirus*. Nine antigenically distinct serotypes of the virus are known to exist, and while some are cross-protective (e.g. serotypes 6 and 9) others are not. Serotype 9 has been responsible for most outbreaks outside Africa. The disease is endemic in tropical equatorial regions of sub-Saharan Africa from which it spreads regularly to southern Africa and occasionally, to northern Africa. Outbreaks of AHS have also occurred outside of the African continent: in the Near and Middle East (1959-1963); Spain (1966, 1987-1990); Portugal (1989); Morocco (1989-1991); Saudi Arabia (1989); Saudi Arabia and Yemen (1997); the Cape Verde Islands (1999) and Thailand (2020). Although not endemic in South Africa, AHS reappears every year in the northeastern part of the country (NE Transvaal), from where it spreads southwards and not infrequently, westwards. African horse sickness is a highly important OIE listed equine disease and a transboundary disease in the USA. As such, any suspicion of AHS is immediately reportable to the United States Department of Agriculture (USDA) and State Animal Health Officials in all 50 states and territories.
Clinical Signs/Disease Manifestations	Four clinical manifestations of AHS have been described. Expression and severity of disease varies among members of the family Equidae. Horses and mules are most susceptible, experiencing severe disease and very high case-fatality rates. European and Asian donkeys are less susceptible, with mortality rates of 5-10%. African donkeys and zebras are least susceptible, rarely experiencing significant disease or mortality. **Pulmonary Form ("Dunkop")** • Acute to peracute form of the disease • Foals are particularly susceptible as protective maternal antibodies wane after 3 months of age

- Fever up to 41° C (106°F), accompanied by depression, profuse sweating, injection of the conjunctivae, severe pulmonary edema, dyspnea, paroxysmal coughing, copious frothy nasal discharge
- Onset of dyspnea is very sudden
- Disease progression may last hours to several days after onset of clinical signs
- Case-fatality rate is up to 95%

Cardiac Form ("Dikkop")

- Subacute form of the disease
- Fever up to 41° C (106°F), depression, supraorbital non-pitting edema, swelling, petechiation and eversion of the conjunctivae
- Edema of the head (eyelids, lips, cheeks, and tongue)
- Edema extending down the neck towards the chest (including neck, thorax, pectorals, and shoulders)
- Dysfunction of the upper airways and esophagus especially in cases with severe edema of the head
- Periodic recumbency and colic
- Disease progression lasts 4-8 days after onset of clinical signs
- Case-fatality rate is 50% or greater

Mixed Form

- Combination of pulmonary and cardiac forms of the disease
- Most frequently encountered form of the disease
- Initial evidence of pulmonary involvement followed by edematous swellings and effusions
- Acute paroxysms of coughing, copious, frothy nasal discharge, collapse
- Death from cardiac failure occurs 3-6 days after onset of clinical signs
- Case-fatality rate of 70% or greater

African Horse Sickness Fever

- Mildest (subclinical) form of disease, seldom diagnosed clinically
- Typically seen in partially immune horses and in donkeys and zebras
- Moderate malaise, fever of 40-40.5° C (104-105°F) lasting one to several days, anorexia, depression, occasionally mild conjunctivitis, dyspnea, and edema of the supraorbital fossa
- Rapid recovery of affected animals, death very rare.

Differential Diagnosis	Acute heart failure, acute pleuritis or pneumonia, equine viral arteritis, equine infectious anemia, purpura hemorrhagica, anthrax, plant toxicosis,

	chemical poisoning, heat stress, piroplasmosis, equine encephalosis, Getah virus infection, Hendra virus infection, and trypanosomiasis.
Incubation Period	The incubation period is usually 7-14 days although it can be as short as two days or even less with the peracute form of the disease. Experimental infections suggest that the incubation period can potentially be as long as 21 days.
Risk Factors	**Potential Routes of Virus Entry into a Country:** • Importation of infected equids from countries in which AHS is endemic or occurs regularly. Greatest risk is associated with zebras or donkeys, or horses with partial immunity. • Importation of infective animal products such as equine serum or semen from AHS affected countries. • Introduction of infected vectors, specifically *Culicoides* spp. via airplanes, ships. • Windborne carriage of infected vectors. **Factors Critical to Restricting Spread of AHS once Introduced into a Country:** • Delineation of area of infection. • Enforcement of movement controls of equids within, into, and out of the infected area. • Housing of all equids from dusk to dawn. • Implementation of insect control measures. • Daily monitoring of all equids for signs of the disease; any positive cases euthanized. • Vaccination of all susceptible animals with an appropriate monovalent or polyvalent AHS vaccine, as determined by USDA APHIS.
Transmission	Since AHS is a non-contagious infectious disease, it is not transmitted by direct contact between horses. It is an insect-transmitted disease, with midges (*Culicoides* spp.) serving as the primary vector. Climate and other factors influence the breeding of *Culicoides* spp.; thus, AHS is seasonal in occurrence with outbreaks frequently associated with a period of drought followed by heavy rains. The extrinsic incubation period of AHS virus in midges is eight days. There is no evidence of transovarial transmission or over-wintering of AHS virus in *Culicoides* larvae. Although mosquitoes, various biting flies and certain tick species may possibly play a role in the mechanical transmission of the virus, this has not been demonstrated under natural circumstances. Experimentally, AHS virus can be transmitted

	between horses by injection of infected blood or organ suspensions, especially when administered by the intravenous route. While the virus is present in high concentration in blood and certain organs (e.g. lungs, spleen, lymph nodes), only trace amounts can be found in serum, tissue fluids and excretions. Viremia usually lasts 4-8 days in horses but may persist for up to 21 days. There is no evidence to indicate existence of the carrier state in recovered animals. Zebra, which are considered the natural reservoir of the virus in most regions in Africa, may remain viremic for up to 40 days.
Diagnostic Sampling, Testing and Handling	Diagnosis of AHS is virtually impossible during the febrile phase of the disease. A presumptive diagnosis is only feasible following development of the characteristic clinical signs. Federal and state animal health officials must be notified immediately where a case of AHS is suspected; their responsibility is to assist in the investigation and establish the appropriate diagnostic testing that needs to be undertaken. Samples from suspect cases of AHS should include unclotted whole blood, preferably collected during the febrile phase of the disease, and serum. Where possible, paired serum samples collected 2 weeks apart are recommended. Spleen, lung, and lymph node samples should be taken from freshly dead animals and placed in appropriate transport medium for PCR testing and/or virus isolation. Another set of tissue samples should be placed in 10% buffered formalin for histopathological examination. All samples for diagnostic investigation should be shipped overnight at 4° C to the USDA's National Veterinary Services Laboratories, Ames, Iowa and should be coordinated with state or federal animal health official. Current laboratory tests of choice for confirmation of diagnosis of AHS are the duplex real-time reverse transcription PCR assay for detection of the virus, and the VP-7 Blocking ELISA for determination of serum antibodies to the virus. Both tests have met the OIE requirements of validation for this disease. Demonstration of the typical macroscopic lesions of AHS on post-mortem examination are often sufficiently specific to support a diagnosis of the disease.
Recommended Action if AHS is Suspected	African Horse Sickness is an internationally reportable disease; contact your regulatory authority for reporting suspect cases. In the United States contact your State and/or federal animal health official. Federal Area Veterinarians in Charge https://www.aphis.usda.gov/animal_health/contacts/field-operations-districts.pdf

	State Veterinarians http://www.equinediseasecc.org/state-veterinary-offices
Specific Control Measures and Biosecurity Issues	A critical first step in the control of AHS is to expedite confirmation of the disease and identification of the virus serotype involved. On confirmation of a diagnosis and in conjunction with authorities, establish a strict quarantine zone and enforce movement controls within, into, and out of the infected area. In the likely event that it is decided to euthanize all infected and exposed equids, the carcasses must be disposed of as soon as possible while observing all appropriate biosecurity precautions in the process. Potentially contaminated areas must be disinfected, thoroughly cleaned and re-disinfected. African horse sickness virus is relatively heat stable and can remain viable at low temperatures for many years. It is resistant to putrefaction for an extended period. However, it is readily inactivated below pH 6.0 and by formalin and certain other chemicals. All equids must be stabled preferably in insect-proof housing from dusk to at least dawn. Insect control measures need to be implemented including destruction of *Culicoides* breeding sites, use of insect repellants and insecticides. The temperature of all equids should be taken twice a day. Any febrile equid(s) should be transferred to separate insect-proof accommodation, pending a decision on whether to euthanize the animal(s) or not. Decisions regarding potential implementation of a vaccination program and release of AHS cases and contact horses from quarantine will be at the direction of state and federal animal health officials.
Zoonotic Concerns	While humans are not natural hosts for AHS virus, under certain circumstances the virus can be transmissible to humans. Although recorded very infrequently, a neurotropic vaccine strain of the virus has caused encephalitis and retinitis in persons involved in the production of the commercial modified live vaccine against AHS.
Additional Resources	Spickler, AR, Roth, JA, Gaylon, J & Lofstedt, J. (2010) African Horse Sickness *In*: Emerging and Exotic Diseases of Animals 4th ED. Iowa State University, Ames, IA, 81-83 Fernandez, PJ & White, WR. (2010) African Horse Sickness *In:* Atlas of Transboundary Animal Diseases. OIE (World Organization of Animal Health) 99-106 2019 Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 9th ED. OIE (World Organization of Animal Health)

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