

**AFRICAN HORSE SICKNESS
STANDARD OPERATING PROCEDURES:
1. OVERVIEW OF ETIOLOGY AND ECOLOGY**

FAD PReP

**Foreign Animal Disease
Preparedness & Response Plan**



**United States
Department of
Agriculture**

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The Foreign Animal Disease Preparedness and Response Plan (FAD PReP) Standard Operating Procedures (SOPs) provide operational guidance for responding to an animal health emergency in the United States.

These draft SOPs are under ongoing review. This document was last updated in **August 2013**. Please send questions or comments to:

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FAD PreP
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African Horse Sickness (AHS)

Etiology & Ecology Quick Summary

Disease

African horse sickness, perdesiekte, pestis equorum, peste equine, peste equina Africana.

Mortality & Morbidity

Very high mortality in horses, depending on the clinical form of disease. Mild or subclinical disease in zebra and African donkeys. High mortality in other equids.

Susceptible Species

Members of the equidae family: horses, mules, donkeys, and zebras. Dogs die peracutely after eating infected horse meat but do not play a role in the epidemiology of AHS.

Zoonotic Potential?

Not a threat to public health.

Reservoir Hosts

Zebra and African donkeys are considered reservoir hosts. A vertebrate or invertebrate carrier has not been identified to date.

Transmission

Arthropod vectors, primarily some species of *Culicoides* biting midges, are biological vectors. Other insects have been suggested as possible vectors but none have been shown to play a role under natural conditions. Mechanical transmission is possible but inefficient.

Persistence in the Environment

AHS virus is relatively resilient and heat stable. Susceptible to changes in pH, especially below 6.0. Can remain infective for long periods of time in blood but is quickly inactivated in muscle during rigor mortis (due to lowered pH). AHS is an infectious disease and requires a vector or syringe – infection does not occur through the environment.

1.1 Introduction

African horse sickness (AHS) has been prevalent in sub-Saharan Africa and parts of the Middle East for centuries. This vector-borne, viral disease affects all members of the equidae family. Infection with AHS virus (AHSV) results in varying disease forms: subclinical, subacute or cardiac, acute respiratory or pulmonary, and mixed. The cardiac, pulmonary, and mixed forms are regularly fatal. In recognition of its impact, AHS is currently a World Organization for Animal Health (OIE) listed disease. While AHS has never occurred in the United States, its geographic distribution has grown and there is potentially a competent vector in North America.

1.1.1 Further Information

This document is intended to be an overview of AHS. Unless otherwise noted, this document is based primarily on information from “African horse sickness” (Mellor, 2004) and the OIE Technical Disease Card (2013). Additional resources, as well as references cited in this SOP, are listed in Attachment 1.A. Surveillance information and laboratory criteria are available for OIE *Terrestrial Animal Health Code* (2013) and OIE *Manual of Diagnostic Test and Vaccines for Terrestrial Animals* (2012), respectively.

These documents are on the Animal and Plant Health Inspection (APHIS) Intranet (<http://inside.aphis.usda.gov/vs/em/fadprep.shtml> for APHIS employees). Strategic documents and response plans are also available here: (http://www.aphis.usda.gov/animal_health/emergency_management/).

1.1.2 Goals

As a preparedness goal, APHIS will provide etiology and ecology summaries for AHS and update the summaries at regular intervals.

As a response goal, the Unified Command and stakeholders will have a common set of etiology and ecology definitions and descriptions to ensure proper understanding of AHS when establishing or revising goals, objectives, strategies, and procedures.

1.2 Purpose

The purpose of this document is to provide responders and stakeholders with a common understanding of the disease agent.

1.3 Etiology

1.3.1 Name

AHS is also known by perdesiekte, pestis equorum, peste equine, peste equina Africana.¹

1.3.2 Virus Characteristics

According to the International Committee on Taxonomy of Viruses,² AHSV is characterized as follows:

¹ Center for Food Security and Public Health, Iowa State University (CFSPH). 2006. “African Horse Sickness” *Technical Factsheet*. www.cfsph.iastate.edu.

- Family: *Reoviridae*
 - Subfamily: *Sedoreovirinae*
 - Genus: *Orbivirus*.
- Genome characteristics: double-stranded ribonucleic acid (dsRNA).

There are currently nine serotypes identified, with the most recent serotype discovered in 1960.

1.3.3 Morphology

Like bluetongue virus, another member of the *Orbivirus* family, AHSV is a non-enveloped virus with a segmented, dsRNA genome. The 10 segments of the genome are enclosed within a two-layer icosahedral (having 20 identical equilateral triangular faces) capsid. The 10 segments code for 7 structural proteins (VP1–VP7) and 4 non-structural proteins (NS1–NS3A). Two structural proteins, VP3 and VP7, which comprise the inner layer of the capsid, are conserved across all serotypes. VP2 and VP5 make up the outer layer; VP2 varies antigenically among the serotypes. Some cross-reaction among serotypes has been observed.

1.4 Ecology

1.4.1 Susceptible Species

Susceptible species include members of the equidae family: horses, mules, donkeys, and zebra. Antibodies to AHSV have been found in camels, African elephants, and black and white rhinoceroses; these animals are not believed to be epidemiologically significant. Dogs may also become infected after consuming infected horsemeat but are not thought to play a role in transmission.

1.4.2 Reservoir and Carriers

Zebras are believed to be the reservoirs for AHSV because they typically experience a subclinical course of infection. The fact that AHSV has not become established outside of sub-Saharan Africa (the native habitat of zebra) provides further evidence. Additionally, the extremely high rates of mortality exhibited by AHSV infected horses and mules are consistent with these animals being accidental hosts. Although *Culicoides* are active year-round through much of the geographical disease range, AHSV can nevertheless be maintained in biting midges during their low activity periods and/or when temperatures are too low to allow for viral replication in the vector; thus the disease can “overwinter” or be maintained in midges for up to 11.5 months.³

1.4.3 Distribution

AHS is limited to the tropical and sub-tropical areas of sub-Saharan Africa (Figure 1). From 2008 to 2012, outbreaks of AHS have been reported in Angola, Botswana, Eritrea, Ethiopia,

² International Committee on Taxonomy of Viruses. 2011. <http://ictvonline.org>.

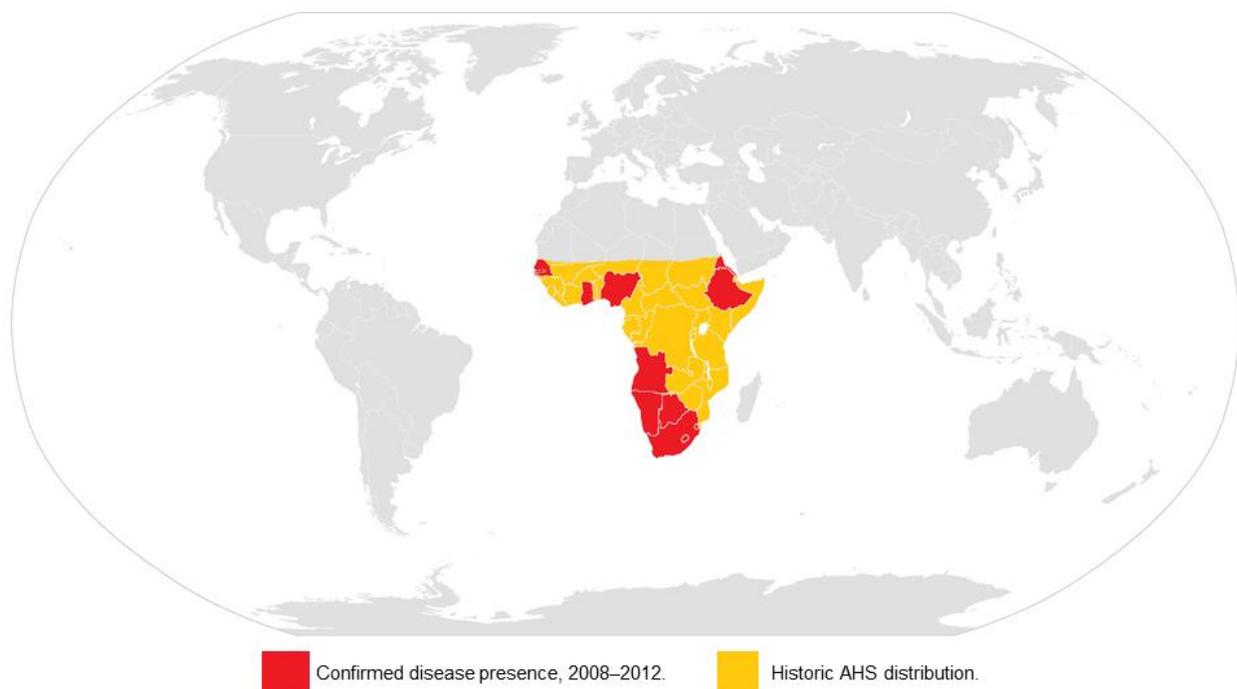
³ Wilson A, Mellor PS, Szmargd C, Mertens PPC. 2009. “Adaptive strategies of African horse sickness virus to facilitate vector transmission.” *Vet. Res.* 40: 16.

Gambia, Ghana, Lesotho, Namibia, Nigeria, Senegal, South Africa, and Swaziland. The disease is suspected in a number of other African countries but has not been confirmed.⁴

Though currently confined to the African subcontinent, from 1959 to 1961 AHS serotype 9 spread out of Africa into Saudi Arabia, Syria, Lebanon, Jordan, Iraq, Turkey, Cyprus, Iran, Afghanistan, Pakistan, and India. A massive vaccination and stamping-out campaign resulted in eradication of AHS in the Middle East in 1961. A few years later in 1966, serotype 9 again moved north and then into Spain where it was eradicated within 3 weeks. Appearances of AHS in North Africa have been attributed to the movement of nomads and their animals.

Subsequent outbreaks have occurred sporadically in North Africa, Spain, and Portugal with stamping-out and vaccination efforts eradicating the disease.

Figure 1: Distribution of AHS and areas of confirmed disease presence, 2008–2012.



1.4.4 Transmission

AHS is an arthropod-borne disease, primarily transmitted between hosts by some species of *Culicoides* biting midges. To a lesser degree *Culex*, *Anopheles*, and *Aedes* mosquito species as well as *Hyalomma* and *Rhipicephalus* tick species serve as biological vectors.⁵ Out of over 1500 identified species of *Culicoides*, there is evidence that approximately 30 are capable of

⁴ OIE. 2013. World Animal Health Information Database. *Disease Timelines*. www.oie.int.

⁵ Mellor PS. 2004. "Epizootiology and vectors of African horse sickness virus." *Comp Immun. Microbiol. Infect. Dis.* 17(3/4): 287–296.

transmitting orbiviruses.⁶ Biting flies may play a minor role in mechanical transmission, but this method is inefficient. AHS is not transmitted by casual contact.

In Africa where AHS is endemic, the primary vector is *C. imicola*, which can be found throughout Africa, much of Southeast Asia, Portugal, Spain, Italy, Greece, and other Mediterranean countries. *C. bolitinos* was recently identified as an important vector in the cooler highland areas of southern Africa.

C. sonorensis, which is native to North America and a vector of bluetongue virus, is a competent vector for AHSV in the laboratory. *C. sonorensis* is found primarily throughout the western and southern United States.

Correlations between temperature and infection rates have been observed for AHSV infection of *Culicoides*. As temperatures rise the infection rates of *Culicoides* midges increase and transmission can occur sooner than in cooler temperatures. Interestingly, midge survival rates decrease as temperature increases. Therefore the rate of transmission results from the interaction of these two variables. Transmission is also affected by weather patterns. Cyclical epidemics occur in South Africa after drought followed by heavy rains brought on by the El Niño-Southern Oscillation phenomenon.

1.4.5 Incubation & Infective Period

The incubation period ranges from 2 to 21 days but is usually 7–14 days long. According to the OIE *Terrestrial Animal Health Code* (2013),⁷ the infective period, or when animals are a source of infection for *Culicoides* midges, is 40 days for domestic horses.

1.4.6 Morbidity and Mortality

AHS takes on four different clinical forms in infected equids: subclinical (horse sickness fever), subacute or cardiac, acute respiratory or pulmonary, and mixed. Morbidity and mortality are dependent on the species of animal, previous immunity acquired, and the form of the disease. Horses are more susceptible to the severe (pulmonary and mixed) forms of disease than other equids. Likewise, horses experience higher rates of mortality (50–95 percent) than mules (about 50 percent), European and Asian donkeys (5–10 percent), or African donkeys and zebras (near 0 percent).

Horse sickness fever is rarely, if ever, fatal. This form is often the result of infection with less virulent strains of AHSV or the existence of previous immunity. This is the only form of disease experienced by African donkeys and zebras.

The subacute or cardiac form exhibits mortality rates of 50 percent and higher, the mixed form has mortality rates at 70–80 percent or higher, and the pulmonary form is almost always fatal.

⁶ Wilson A, Mellor PS, Szmargd C, Mertens PPC. 2009. “Adaptive strategies of African horse sickness virus to facilitate vector transmission.” *Vet. Res.* 40: 16.

⁷ World Organization for Animal Health (OIE). 2013. “Article 12.1.1, Chapter 12.1.” *Terrestrial Animal Health Code*. www.oie.int.

1.4.6.1 Clinical Signs

Equids with horse sickness fever experience fever (104°F) and general malaise for 1 to 2 days. Shortness of breath and other pulmonary signs may occur. Animals suffering from the cardiac form develop fever (102–106°F) along with swelling of the supraorbital fossa, eyelids, face, neck, thorax, brisket and shoulders; death usually occurs within one week. Those affected with the pulmonary form also develop fever (104–106°F), shortness of breath, coughing, dilated nostrils with oozing fluids, conjunctivitis, and death due to anorexia within one week. The mixed form takes its name from its characteristic cardiac and pulmonary signs. Equids with the mixed form of AHS experience mild pulmonary signs, fluid-filled swellings, and death from cardiac arrest within one week.

1.5 Environmental Persistence

AHSV, while resilient and fairly stable, is susceptible to fluctuations in pH brought on intentionally or through rigor mortis. Table 1-1 provides an overview of the temperatures and chemicals/disinfectants to which AHSV is vulnerable.

Table 1-1. Physical and Chemical Resistance Characteristics of AHSV

Action	Resistance
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 (158°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 (OCG): 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°C (-4°F) and -30°C (-22°F).
pH	Survives pH 6.0 – 12.0. Readily inactivated below 6.0. Optimal pH (for survival) 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).

Source: OIE. 2013. "African Horse Sickness." *Technical Disease Card*. www.oie.int.

1.6 Risk of Introduction to the United States

According to a 2005 study⁸ conducted by the American Horse Council Foundation, there are 9.2 million horses in the United States and 4.6 million Americans are directly involved in the equine industry. The U.S. horse industry had a direct economic impact of \$39 billion in 2005 and an indirect impact of \$102 billion. The horse industry is a vital component of the U.S. economy; concern about an AHS incursion is warranted.

⁸ American Horse Council Foundation. 2005. "National Economic Impact of the U.S. Horse Industry." <http://www.horsecouncil.org/national-economic-impact-us-horse-industry>.

There are 119 species of *Culicoides* midges in the United States, including *C. sonorensis* which has been shown to be an efficient vector for AHSV in a laboratory setting. Given that throughout the last 50 years, AHS has made incursions into naïve areas more than a few times and survived for multiple years due to its ability to overwinter within the midges, there is cause for vigilance and caution.

The APHIS (for equines) and the U.S. Fish and Wildlife Service (for wild equids)⁹ have import requirements set in place to reduce the likelihood of AHSV introduction. Per APHIS regulation, imported horses undergo inspection prior to export. Horses imported from AHS-affected countries¹⁰ are required to undergo a 60-day quarantine upon arrival in the United States. Given the relatively quick course of disease it is unlikely that an infected horse would be imported to the United States and later serve as a source of infection.

⁹ U.S. Fish and Wildlife Service. “Importing and Exporting Your Commercial Wildlife Shipment.” <http://www.fws.gov/le/commercial-wildlife-shipment.html>.

¹⁰ Oman, Saudi Arabia, the Yemen Arab Republic, and all countries in African except Morocco. See “Protocol for the Importation of Equines.” http://www.aphis.usda.gov/import_export/animals/animal_import/equine/equine_import_quarantine.shtml.

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Attachment 1.B Abbreviations

AHS	African horse sickness
AHSV	African horse sickness virus
APHIS	Animal and Plant Health Inspection Service
CFSPH	Center for Food Security and Public Health
dsRNA	double-stranded ribonucleic acid
FAD PReP	Foreign Animal Disease Preparedness and Response Plan
OIE	World Organization for Animal health
SOP	standard operating procedure
USDA	United States Department of Agriculture
TDD	telecommunication device for the deaf