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# *African Horse Sickness Backgrounder*

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## Causative agent

African Horse Sickness (AHS) is caused by an orbivirus member of the Reoviridae family. Reoviridae are 60 to 80 nanometers (nm) in diameter, unenveloped, double-stranded, RNA viruses. They have an icosahedral shape, indicating that the nucleocapsid has 20 symmetrical faces. Reoviruses are unique in that the capsid has 3 layers. The bluetongue and equine encephalosis viruses are also orbiviruses. Rotaviruses also belong to the Reoviridae family. There are 9 immunologically distinct serotypes of the AHS virus.

## Natural distribution

AHS is also referred to as Perdesiekte, Pestis Equorum, Peste Equina, and Peste Equina Africana. The disease affects equids (horses, mules, donkeys, zebras) and occasionally dogs. Camels and elephants may also be infected with AHS virus; however, infection does not cause clinical disease.

AHS is endemic in the central tropical regions of Africa, but occasionally spreads to the southern or northern areas. The Sahara desert appears to impede northward expansion of the virus, but occasional spread along the Nile Valley or the west coast of Africa has occurred. Outbreaks of AHS outside of Africa occurred in the Near and Middle East (1959 to 1963; more than 300,000 animals died or were destroyed), Spain (1966, 1987 to 1990), and Portugal (1989). An outbreak in South Africa in 1996 resulted in 500 equine deaths.

The disease develops more commonly under moist, mild conditions and with warm temperatures. These conditions favor the presence and activity of the insect vectors. Persistence of the virus through the winter season (overwintering) occurs in an as yet undetermined manner and reservoir. Zebras have been implicated as that reservoir, but their role has not been confirmed.

The World Organization for Animal Health (OIE) classifies AHS as a notifiable disease because of its potential for rapid spread and important impact on the international trade of animals and animal products. The United States Agricultural Bioterrorism Act of 2002 recognized AHS as an agent that could pose a severe threat to animal health, human health, and animal products in the United States.

## Transmission

AHS is an infectious disease, but it is not contagious; that is, the disease is due to an infectious agent (a virus), but it is not spread from an infected animal to a susceptible animal by direct contact. The insect vectors of AHS are members of the *Culicoides* family, or midges. *Culicoides imicola* appears to be the primary vector, although other species are also capable of transmitting the disease. Mosquitoes and ticks may play a minor role in transmission. Biting flies, such as *Stomoxys* and *Tabanus* species, may mechanically transmit the virus, but the virus does not mature or replicate in these insects. Windborne vectors may facilitate virus movement over long distances. Climatic factors that increase vector activity, such as warm weather, humidity, and rainfall, will increase the likelihood of disease transmission.

Dogs can be experimentally infected with AHS; the most likely source of natural exposure is through the consumption of infected blood or horsemeat. Dogs are not believed to be capable of transmitting the disease.

The incubation period for AHS ranges from three to 14 days. Viremia may persist for up to 18 days in horses, and up to 28 days in zebras and donkeys. During this time, insect vectors can transmit the virus from infected equids to susceptible animals.

## Clinical Signs

Horses are most susceptible to AHS, followed by mules and European and Asian donkeys. African donkeys and zebras are more resistant to the virus.

Clinical signs of AHS most commonly occur five to seven days after infection with the AHS virus. Four forms of AHS have been identified: the peracute or pulmonary form (dunkop), the subacute edematous or cardiac form (dikkop), the acute or mixed form, and Horsesickness Fever.

Horses that develop the peracute form of the disease exhibit fever (104 to 106° F, 40 to 41 C) followed by a sudden onset of progressive respiratory distress. Increased respiratory rate and effort are observed, and the horse may stand with its forelimbs spread apart, head extended, and nostrils fully flared in its struggle to breathe. Spasmodic coughing and profuse sweating may also be observed. A frothy nasal discharge, resulting from severe pulmonary edema, is frequently observed before death. Death often occurs within a few hours of onset of respiratory signs.

The subacute form of AHS begins with a fever (102 to 106° F, 39 to 41 C) of three to six days' duration. The supraorbital fossae (the concavities above the eyes) and eyelids become edematous, followed by the cheeks, lips, tongue, intermandibular space (the hollow area on the underside of the head, between the jaws), laryngeal region (throatlatch), neck, shoulders, and chest. Edema of the lower limbs is not observed. Severe depression, colic, and pinpoint hemorrhages on the underside of the tongue and on the conjunctivae (the tissue around the eye) are observed prior to death. Death most often occurs because of cardiac (heart) failure and within 7 days.

Horses with the mixed form of AHS exhibit signs of both the peracute and subacute forms. The cardiac (subacute) form is often undetected, and death occurs following severe respiratory distress. On occasion, mild respiratory signs may precede edema and death from cardiac failure. This form is rarely diagnosed before death.

Horsesickness Fever is characterized by a biphasic fever of up to 104°F (40 C) for three to eight days; the fever subsides in the morning, but recurs in the afternoon. Other nonspecific signs, such as mild anorexia, depression, increased heart rate, and congestion of the mucous membranes, may be observed. This form is most commonly seen in African donkeys and zebras or in partially immune animals, and is frequently unrecognized during natural outbreaks. A form of AHS that affects the nervous system has been reported, but is rare.

Other diseases with clinical signs similar to AHS include anthrax, equine viral arteritis, equine infectious anemia, Hendra virus infection, purpura hemorrhagica, equine encephalosis, trypanosomosis, and equine piroplasmiasis. In South Africa, AHS and equine encephalosis may occur simultaneously.

Humans do not become infected with AHS after contact with natural strains of the virus. However, encephalitis (inflammation of the brain), retinitis (inflammation of the retina of the eye), and clotting disorders have been reported in humans exposed to a neurotropic vaccine strain intranasally.

## Diagnosis

Edema of the supraorbital fossae is considered characteristic of AHS. In endemic areas, a preliminary diagnosis of AHS can be made based on this finding. Confirmation of AHS, however, requires virus isolation and identification.

Isolation of AHS is more successful when blood samples are obtained during febrile episodes and kept from coagulating. Virus isolation is performed using cell culture, intracerebral inoculation of suckling mice, or intravenous inoculation of embryonated eggs. Enzyme-linked immunosorbent assay (ELISA) or reverse-transcriptase polymerase chain reaction (RT-PCR) can be performed on blood or on spleen, lung, and lymph node tissue to detect the AHS virus. Serologic tests include complement fixation (CF), ELISA, immunoblotting, hemagglutination, and virus neutralization (VN). Paired serum samples, obtained 21 days apart, are preferred. Because the virus concentrates in the spleen, lung, and lymph nodes, necropsy samples of these organs can facilitate isolation of the virus.

The CF and ELISA are preferred for diagnosis when international trade is anticipated. The VN test is most often used for determining the serotype of AHS involved in the case or outbreak, but immunodiffusion and hemagglutination inhibition tests can also be used.

Before samples are submitted, the proper authorities should be contacted. Not all laboratories are capable of handling and testing the samples, and samples should only be submitted to authorized laboratories. All samples should be stored and transported at 4C.

### Treatment

**African Horse Sickness is a reportable disease.** State or Federal animal health officials should be notified immediately when clinical signs of AHS are observed. There is no effective treatment for AHS. Frequently, affected horses are euthanatized, and cadavers are destroyed.

### Morbidity and Mortality

The case fatality rate (the number of affected animals that die from the disease) in horses with AHS ranges from 50 to 95%, depending on the form of disease. The case fatality rate associated with the peracute (pulmonary) form approaches 100%. The subacute (cardiac) form results in a case fatality rate of approximately 50 to 70%, and the mixed form is associated with a case fatality rate greater than 80%. The Horsesickness Fever form of AHS is rarely fatal. Because mules are less susceptible, the overall case fatality rate is approximately 50%. The case fatality rate in Asian and European donkeys is 5 to 10%. African donkeys and zebras rarely die from AHS.

### Prevention and Control

Current U.S. importation laws restrict entry of equids from AHS-endemic unless they have been in a country or area designated as AHS-free for a minimum of 60 days. Following entry, animals are subject to an additional three to seven day quarantine period at an approved quarantine facility.

Cases or outbreaks of AHS in nonendemic areas require strict quarantine and isolation. Insect repellents should be applied, and animals must be stabled from dusk to dawn (the time of highest vector activity). Insect-proof housing is recommended. Rectal temperatures should be obtained twice daily on all equids. Any equids that develop a fever should be isolated from others and housed in an insect-free stable until the cause of the fever is determined. Alternatively, febrile animals may be euthanatized.

Animals that survive AHS develop good immunity to the specific serotype and partial immunity to other serotypes of the AHS virus. Immune mares will convey passive immunity to their foals for up to 6 months.

Attenuated and inactivated vaccines have been used for prevention of AHS. Monovalent vaccines, effective against only one serotype of the virus, are beneficial when the infecting strain has been identified. Polyvalent vaccines are effective against two or more serotypes of the virus. Administration of the vaccine is not permitted in surveillance zones because of the difficulty in differentiating vaccinated from infected animals.

The AHS virus can survive at room temperature for up to 37 days, and for up to 20 years at 4 C. The organism is susceptible to formalin, acetyleneimine derivatives, iodophors, phenol, radiation,  $\beta$ -propiolactone, or pH conditions less than 6 or greater than 12.