
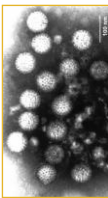


S l i d e 1	<h2 style="margin: 0;">African Horse Sickness</h2> <p style="font-size: small; margin: 0;"><i>Perdesiekte, Pestis Equorum, La Peste Equina, Peste Equina Africana</i></p>	<p><i>Last reviewed: October 2011 (TechFS 2006-11)</i></p> <p>African horse sickness (AHS), also known as Perdesiekte, Pestis Equorum, La Peste Equina, and Peste Equina Africana, is a serious, often fatal, disease that affects members of the Equidae family. The disease is spread by arthropod vectors (primarily <i>Culicoides</i> species–biting midges), with mortality in horses as high as 95%. Currently the disease is endemic in Africa, however infected animals or vectors may carry the virus into AHS-free regions. AHS is considered as one of the most lethal of horse diseases and is an OIE reportable disease (http://www.oie.int/animal-health-in-the-world/oie-listed-diseases-2011/).</p>
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S l i d e 2	<div style="text-align: center;"> <h3 style="margin: 0;">Overview</h3>  </div> <ul style="list-style-type: none"> • Etiology • Species Affected • Epidemiology • Economic Importance • Clinical Signs • Diagnosis and Treatment • Prevention and Control • Actions to Take 	<p>This presentation will discuss African horse sickness, its etiology, species affected, and transmission. It will also cover the epidemiology (e.g., geographic distribution, morbidity, mortality) of the disease as well as outline the economic impact the disease has had in the past and could have in the future. Additionally, we will overview the clinical signs and necropsy findings, as well as the diagnosis and treatment of the disease. Finally, we will address prevention and control measures for the disease, as well as actions to take if AHS is suspected.</p>
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S l i d e 3	<h2 style="margin: 0;">ETIOLOGY</h2>	
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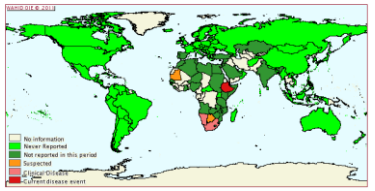
S l i d e 4	<div style="text-align: center;"> <h3 style="margin: 0;">African Horse Sickness Virus</h3>  </div> <ul style="list-style-type: none"> • Non-enveloped RNA • Family Reoviridae <ul style="list-style-type: none"> – Genus <i>Orbivirus</i> • Nine serotypes (1-9) <ul style="list-style-type: none"> – All viscerotropic – Serotype 9 <ul style="list-style-type: none"> • Endemic areas • Outbreaks outside of Africa – Serotypes 1-8 <ul style="list-style-type: none"> • Limited geographical areas 	<p>African horse sickness (AHS) is caused by the African horse sickness virus (AHSV), a non-enveloped, double-stranded RNA virus from the family Reoviridae, genus <i>Orbivirus</i>. The AHS virus is comparable in morphology and molecular structure to bluetongue virus (which is considered the prototype virus of the Genus <i>Orbivirus</i>). There are nine serotypes of the AHS virus, all of which are viscerotropic (having a predilection for the abdominal and thoracic viscera). Serotype 9 is widespread in endemic regions, while serotypes 1-8 are found only in limited geographic areas. Serotype 9 has also been responsible for the majority of AHS outbreaks outside of Africa. However serotype 4 caused one outbreak in Spain and Portugal between 1987 and 1990. [Photo is electron micrograph of Reoviridae from The National Center for Biotechnology Information at http://www.ncbi.nlm.nih.gov/ICTVdb/ICTVdB/60000000.htm]</p>
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S I d e 5	<p>African Horse Sickness Virus</p> <ul style="list-style-type: none"> • Inactivated by <ul style="list-style-type: none"> - Heat (temps greater than 140°F) - pH less than 6, or 12 or greater - Acidic disinfectants • Rapidly destroyed in carcasses that have undergone rigor mortis 	<p>The AHSV can be inactivated in the laboratory with formalin, β-propiolactone, acetyleneimine derivatives or radiation. It is also destroyed at a pH less than 6, or pH 12 or greater. Acidic disinfectants such as 2% acetic or citric acid have been recommended for decontamination when it is warranted. AHSV can survive in frozen meat, but is inactivated at temperatures greater than 60°C) 140°F. Optimum pH for virus survival is pH 7-8.5. The virus is destroyed at a pH less than 6, or pH 12 or greater; the virus is rapidly destroyed in carcasses that have undergone rigor mortis due to pH fluctuations.</p>
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S I d e 6	<p>EPIDEMIOLOGY</p>
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S I d e 7	<p style="text-align: center;">Species Affected</p> <ul style="list-style-type: none"> • Equidae <ul style="list-style-type: none"> - Horses, donkeys, mules - Zebras • Other <ul style="list-style-type: none"> - Camels - Dogs 	<p>The African horse sickness virus can infect horses, donkeys, mules, zebras, camels and dogs. The most serious infections occur in horses and mules, which appear to be accidental hosts. Zebras and donkeys rarely develop serious clinical signs. Zebras, which are often asymptomatic, are thought to be the natural reservoir hosts in most regions of Africa. Infections in camels have been reported, but appear to be uncommon and asymptomatic. [Photos: Top: Horse, by P.Futoma, Iowa State University; Middle: Mule. By J.Lascorz, http://commons.wikimedia.org/wiki/File:09.Moriles_Mula.JPG; Bottom: Zebra, by M.Dezemery at http://www.flickr.com/photos/mdezemery/2461520029/]</p>
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S I d e 8	<p style="text-align: center;">Geographic Distribution</p> <ul style="list-style-type: none"> • Endemic in sub-Saharan Africa • Outbreaks <ul style="list-style-type: none"> - Southern and northern Africa - Near and Middle East - Spain and Portugal 	<p>African horse sickness is endemic to sub-Saharan central and east Africa. The disease sometimes spreads to southern Africa and occasionally to northern Africa. AHS epizootics became less common in southern Africa during the latter half of the 20th century, possibly due to decreases in free-ranging zebra populations. Outbreaks have been reported outside Africa, in Egypt and other parts of the Middle East, as well as in Spain, Portugal, Morocco, Pakistan and India. [Photo: Distribution of most AHS cases from Government of United Arab Emirates at http://www.uae.gov.ae/uaeagricent/livestock/img/horse_Sickness1.jpg]* *Link defunct as of 2010</p>
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S I d e 9	<p style="text-align: center;">OIE Disease Distribution Map</p> 	<p>This map shows the reported disease distribution map for African horse sickness for July-December 2010. [Photo: World Animal Health Information Database, World Organization for Animal Health (OIE). http://web.oie.int/wahis/public.php?page=disease_status_map]</p>
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Incidence/Prevalence	
• Seasonal	- Late summer - early autumn
• Cyclic	- Drought followed by heavy rains - Influences insect breeding
• Epizootics halted by	- Frost - Lack of long-term vertebrate reservoir - Reduced numbers of vectors - Control measures <ul style="list-style-type: none"> • Vaccination, vector abatement

African horse sickness has both a seasonal and an epizootic cyclical incidence. The disease most commonly occurs in late summer to early autumn, and after periods of drought followed by heavy rains. The warm, moist conditions are optimal for vector breeding. Some authors speculate that global warming could increase the risk for spread of arthropod-borne diseases such as AHS. Epizootics of AHS outside the enzootic sub-Saharan zone do not appear to be maintained for more than 2-3 consecutive years. Factors such as the absence of a long-term vertebrate reservoir, reduced numbers of vectors, and efficient control measures (vaccination and vector abatement) may play a role in preventing the disease from becoming endemic in these areas. Outbreaks are abruptly curtailed by severe frost. During the harsh winter months, the virus must survive in an appropriate reservoir.

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Morbidity/Mortality	
• Varies with species, previous immunity, form of disease	
• Mortality based on species	- Horse particularly susceptible
Species	Mortality
Horses	50-95%
Mules	50%
European and Asian donkeys	5-10%
African donkeys and zebras	Rare

Morbidity and mortality vary with the species of animal, previous immunity, and the form of the disease. Horses are particularly susceptible to the more severe forms of African horse sickness. The mixed and pulmonary forms tend to predominate in susceptible horse populations, and the mortality rate is usually 50% to 95%. In other species of Equidae, African horse sickness is generally less severe. In mules, the mortality rate is approximately 50%, and in European and Asian donkeys, 5% to 10%. Death is rare in African donkeys and zebra. Animals that recover from African horse sickness develop good immunity to the infecting serotype and partial immunity to other serotypes.

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Morbidity/Mortality	
• Mortality based on form of disease	
Disease Form	Mortality
Pulmonary form	Up to 95%
Cardiac form	50% or more
Mixed form	70-80%
Horsesickness fever	Typically recover

The pulmonary form is nearly always fatal. The cardiac form of the disease results in a mortality rate of 50% or higher. In the mixed form, mortality ranges from 70% to greater than 80%. Horsesickness fever rarely results in death.


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TRANSMISSION	

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Transmission

- Not contagious
- Vector-borne: *Culicoides* spp.
 - *Culicoides imicola* – principal vector
 - *C. bolitinos*
 - *C. variipennis*
 - Other potential arthropods
- Viremia in Equidae
 - Horses: 12 to 40 days
 - Zebras, African donkeys: up to 6 weeks




African horse sickness is not a contagious disease; that is, it does not spread directly from horse to horse. The virus is transmitted by the arthropod-vector *Culicoides* (biting midges). The principal vector species is *Culicoides imicola* ; however other *Culicoides* species (*C. bolitinos* or *C. variipennis*, which is common in many parts of the United States) should also be considered potential vectors. Transmission of AHS by other arthropods is thought to be a very minor source of infection. Mosquitoes may be potential biological vectors, while biting flies (e.g., *Stomoxys* and *Tabanus*) may potentially serve as mechanical vectors. The viremia that develops in Equidae species is high enough to infect competent vectors; however, the length of viremia varies, lasting 12-40 days in horses and up to 6 weeks in zebras and African donkeys. [Photo: A *Culicoides* biting midge from www.defra.gov.uk]

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***Culicoides* spp.**

- Biting midges, “punkies”, “no-see-ums”
 - Extremely small ~1/8”
 - Species identified by wing pattern
- Habitat
 - Margins of water sources
- Life cycle: 2-6 weeks
 - Eggs hatch in 2-10 days
- Females are bloodsucking
 - Greatest biting activity dusk to dawn



Culicoides species, or biting midges, are flies in the family Ceratopogonidae [Class: Insecta, Order: Diptera, Family: Ceratopogonidae]. They have also been called “punkies”, and “no-see-ums”. They are extremely small insects, typically measuring less than 1/8 inch and have distinctive wing patterns. Margins of streams and lakes, mud holes, tree holes, salt marshes, tide pools, swamps, rice fields, and runoff from dairy and feedlot pens are all habitats for *Culicoides* spp. The insect’s life cycle ranges from 2-6 weeks, depending on the species and environmental conditions. Eggs are usually deposited in masses of 25-300 in water, and hatch in about 3 days at 80°F. Only females feed on blood, with the greatest time of biting activity occurring near dusk and around dawn. Environmental temperature and moisture are the main factors determining their prevalence in the environment. [Photo: *Culicoides* (biting midge) from USDA]


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ECONOMIC IMPORTANCE

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History

- 1600: First recorded
 - Horses to southern Africa
- 1921: Sir Arnold Theiler
 - Described 7 major epizootics in South Africa from 1780-1918
- 1959-61: Middle East
 - 1st outbreak outside Africa
- 1987-91: Spain, Portugal
 - Imported zebra reservoirs
 - New *Culicoides* species




Historically, African horse sickness (AHS) was first recorded south of the Sahara Desert in the mid-1600s with the introduction of horses to southern Africa. In 1921, Sir Arnold Theiler (considered to be the father of veterinary science in South Africa) described seven major epizootics (a disease suddenly and temporarily affecting a large number of animals over a large area) of AHS that occurred in South Africa between 1780-1918. [Note: He also was responsible for developing an early vaccine for the disease, which provided a foundation for future research leading to effective preventive measures for this fatal disease of Equids]. In 1959-61, the first documented outbreak of AHS out of its traditional enzootic region of Africa occurred in the Middle East (Israel, Iran, Pakistan, Afghanistan, India, Turkey, Iraq, Syria, Lebanon, Jordan, and Cyprus). During this outbreak, as many as 300,000 animals died or were destroyed. India reported a 90% mortality in Equidae involved. In 1987 Spain had an outbreak of AHS, which later spread to Portugal. It was suspected that the

disease reached the country from subclinically infected zebras imported from Namibia, Africa. [Note: Zebras show no clinical signs when infected with the virus but can have viremia, and therefore be infectious for the arthropod vector, for as long as 6 weeks]. During this outbreak, it was found the virus was effectively spread by a non-traditional *Culicoides* species, increasing the list of potential vectors capable of transmitting the virus. [Photo: Sir Arnold Theiler. From Gluck Equine Research Center, Department of Veterinary Science, University of Kentucky. <http://www.ca.uky.edu/gluck/aboutthetheiler.asp>; Source: <http://www.cidrap.umn.edu/cidrap/content/biosecurity/ag-biosec/anim-disease/ahs.html>]

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Economic Impact

- 1989: Portugal
 - 137 outbreaks
 - 104 farms
 - 206 equines destroyed
 - 170,000 equines vaccinated
 - Cost \$1.9 million



As an example of the economic impact the disease has had, especially in naïve populations, lets look at the 1989 outbreak in Portugal. This was the first time the disease was reported in this country, and followed outbreaks occurring in Spain. Of the 137 outbreaks (on 104 farms), 206 equines died (14%) or were destroyed (16%); 81.5% were horses, 10.7% were donkeys and 7.8% were mules. An eradication campaign was initiated, including the vaccination of all Portuguese equines (170,000 animals). Among those animals vaccinated, 82 died or were euthanized due to suspected or confirmed AHS. One year after ending vaccination, Portugal was declared free of AHS. The estimated cost of the eradication campaign was US \$1.9 million. [Map from <http://www.globalorganizationplanning.com/images/spain-map.gif>; Data source: Protas M, Boinas FS, Oliveira E, Sousa J, Rawlings F. African horse sickness in Portugal: a successful eradication programme. *Epidemiol Infect.* 1999 Oct; 123(2):337-46.]

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U.S. Economic Impact

- U.S. Horse Industry (2007)
 - Inventory: 4 million horses
 - Sales: \$2.0 billion
 - Employment: 4.6 million Americans
- Risk factors
 - Disease not in U.S. - naïve population
 - Arthropod vector is in U.S.
 - Outbreak would result in movement and trade restrictions

The potential impact of AHS in the U.S. could be substantial. In 2007, the U.S. equine industry had an inventory of 4.0 million horses and 283 thousand mules, burros and donkeys, with a total value of sales of 2.0 billion dollars (USDA, National Agricultural Statistics Service, 2007 Census of Agriculture Database). Additionally, an estimated 4.6 million Americans are involved in the industry (i.e., horse owners, service providers, employees or volunteers). Since the disease has never occurred in the Americas, our Equidae species are naïve and highly susceptible to the virus. The U.S. has arthropod vectors potentially able to transmit African horse sickness. Additionally, in the event of an outbreak of AHS in the U.S., an emergency disease declaration and immediate ban on the movement or trade of all Equidae species would be enacted. These factors demonstrate the potential economic impact this disease could have if introduced into the U.S.

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AFRICAN HORSE SICKNESS IN ANIMALS

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Incubation Period	
• Experimental: 2-21 days	
• Natural infection: 3-14 days	
Disease Form	Incubation Period
Peracute (pulmonary) form	3-5 days
Subacute (edematous or cardiac) form	7-14 days
Acute (mixed) form	5-7 days
Horsesickness fever	5-14 days


In experimental infections, the incubation period of AHS in Equidae has been found to range from 2 to 21 days. In natural infections, the incubation period appears to be approximately 3 to 5 days for the peracute (pulmonary) form, 7 to 14 days for the subacute (edematous or cardiac) form, 5 to 7 days for the acute (mixed) form and 5 to 14 days for horsesickness fever.

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Clinical Signs	
• Four forms of the disease	
– Peracute (pulmonary)	
– Subacute edematous (cardiac)	
– Acute (mixed)	
– Horsesickness fever	
• Symptomatic infections most common in horse and mules	
• Zebras typically asymptomatic	


As just mentioned, there are four different manifestations of African horse sickness: the peracute (pulmonary) form, the subacute edematous (cardiac) form, the acute (mixed) form, and horsesickness fever. Clinical signs are characterized by damage to the respiratory and circulatory system as a result of increased vascular permeability. Symptomatic infections occur most often in horses and mules. The pulmonary and mixed forms usually predominate in susceptible populations of horses. The pulmonary form is also the most common form in dogs. The mildest form, horsesickness fever, tends to be seen in horses with partial immunity, mules and donkeys. This form can also occur in zebras, although most cases in this species are asymptomatic.

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Peracute - Pulmonary Form	
• Acute fever	
• Sudden, severe respiratory distress	
• Dyspnea, tachypnea	
• Profuse sweating	
• Spasmodic coughing	
• Frothy serofibrinous nasal exudate	
• Rapid death (few hours)	

The peracute or pulmonary form of African horse sickness usually begins with an acute fever, followed by the sudden onset of severe respiratory distress. Infected animals often stand with forelegs spread, head extended, and nostrils fully dilated. Other clinical signs may include tachypnea, forced expiration, profuse sweating, spasmodic coughing, and a frothy serofibrinous nasal exudate. Dyspnea usually progresses rapidly, and the animal often dies within a few hours after the respiratory signs appear. Mortality for this form is nearly always fatal (near 95%). [Photo: Abundant froth draining from the nostrils reflects severe pulmonary edema. Source: Plum Island Animal Disease Center]

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Subacute Edematous - Cardiac Form	
• Edema	
– Supraorbital fossae, eyelids	
– Cheeks, lips, tongue, intermandibular space	
– Neck, thorax, chest	
– Not in lower legs	
• If animal recovers, swellings subside over 3-8 days	

The subacute or cardiac form of African horse sickness usually begins with a fever (102-106°F) that lasts for 3 to 6 days. Shortly before the fever starts to subside, edematous swellings appear in the supraorbital fossae (top photo) and eyelids (bottom photo). Swelling then spreads to involve the cheeks, lips, tongue, intermandibular space, laryngeal region, and sometimes the neck, shoulders and chest. It is important to note that no edema of the lower legs is observed. If the animal recovers, the swellings gradually subside over the next 3 to 8 days. [Photo: (top) edematous swelling of the supraorbital fossae; (bottom) palpebral conjunctivae, Source: USAHA 'The Grey Book'.]

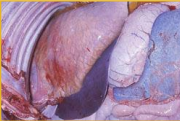
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Subacute - Cardiac Form	
• Terminal stages	
– Severe depression, colic, petechiae of conjunctivae and ventral tongue	
• Death from cardiac failure	
• Mortality 50% or higher	
• Death within 4-8 days	

Clinical signs seen in the terminal stage of the subacute/cardiac form of the disease can include severe depression, colic, and petechiae under the ventral surface of the tongue and in the conjunctivae. Death is typically due to cardiac failure. The mortality rate for the subacute edematous or cardiac form of AHS is usually 50% or higher, with death occurring 4 to 8 days after the onset of clinical signs.

S I i d e 2 6	<h3>Acute - Mixed Form</h3> <ul style="list-style-type: none"> • Pulmonary and cardiac forms • Cardiac signs usually subclinical <ul style="list-style-type: none"> - Followed by severe respiratory distress • Mild respiratory signs <ul style="list-style-type: none"> - Followed by edema and death • Diagnosed by necropsy • Mortality 70-80% 	<p>In the acute or mixed form of African horse sickness, symptoms of both the pulmonary and cardiac forms are seen. In most cases, the cardiac form is subclinical and is followed by severe respiratory distress. Occasionally, mild respiratory signs may be followed by edema and death from cardiac failure. The mixed form of African horse sickness is rarely diagnosed clinically, but is often seen at necropsy in horses and mules. The mortality rate for the acute-mixed form varies from about 70% to greater than 80%.</p>
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S I i d e 2 7	<h3>Horsesickness Fever</h3> <ul style="list-style-type: none"> • Mild clinical signs • Characteristic fever (3 to 8 days) <ul style="list-style-type: none"> - Morning remission (undetectable) - Afternoon exacerbation • Other signs <ul style="list-style-type: none"> - Mild anorexia or depression - Congested mucous membranes - Increased heart rate • Rarely fatal 	<p>In horsesickness fever, the clinical signs are mild. The characteristic fever usually lasts for 3 to 8 days; morning remissions and afternoon exacerbations are often seen. Other symptoms are generally mild and may include mild anorexia or depression, edema of the supraorbital fossae, congested mucous membranes and an increased heart rate. Animals almost always recover from horse-sickness fever. This form of the disease is rarely fatal.</p>
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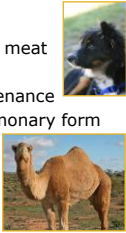
S I i d e 2 8	<h3>Post Mortem Lesions</h3> <ul style="list-style-type: none"> • Pulmonary form <ul style="list-style-type: none"> - Severe, diffuse pulmonary edema - Hydrothorax - Fluid in abdominal and thoracic cavity - Enlarged endematous lymph nodes - Hyperemia and petechial hemorrhages in intestines 	<p>In the pulmonary form of African horse sickness, interlobular edema of the lungs and hydrothorax are the characteristic lesions. In the most acute cases, frothy fluid flows from the nostrils and the cut surface of the lungs, which are mottled red, noncollapsed and heavy. In more prolonged cases, there may be extensive interstitial and subpleural edema, and hyperemia may be less apparent. Fluid accumulation can occur in the abdominal and thoracic cavities. Occasionally, extensive fluid accumulation may be noted in the thoracic cavity (hydrothorax), with near normal appearance of the lungs. The lymph nodes, particularly the nodes in the thoracic and abdominal cavities, are usually enlarged and edematous. Less often, there may be subcapsular hemorrhages in the spleen, congestion in the renal cortex or gastric fundus, and edematous infiltration around the aorta and trachea. The stomach mucosa may be hyperemic and edematous. Hyperemia and petechial hemorrhages may also be apparent in the small and large intestines and the pericardium may contain petechiae. [Photo: Horse lung exhibiting severe interlobular edema. There are petechiae on the pulmonary pleura and the splenic capsule. Source: Plum Island Animal Disease Center]</p>
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S I i d e 2 9	<h3>Post Mortem Lesions</h3> <ul style="list-style-type: none"> • Cardiac form <ul style="list-style-type: none"> - Yellow gelatinous infiltrate <ul style="list-style-type: none"> • Head, neck, shoulders • Brisket, ventral abdomen, rump - Hydropericardium - Submucosal edema of cecum, large colon, rectum • Mixed form <ul style="list-style-type: none"> - Mixture of above findings 	<p>In the cardiac form, a yellow gelatinous infiltrate can be seen in the subcutaneous and intermuscular fascia of the head, neck and shoulders, and occasionally the brisket, ventral abdomen and rump. Hydropericardium is common. The epicardium and endocardium often contain petechial and ecchymotic hemorrhages. Lesions may also be found in the gastrointestinal tract, resembling the pulmonary form. In addition, prominent submucosal edema may be noted in the cecum, large colon and rectum. Ascites can also be seen. In the cardiac form, the lungs are usually normal or slightly engorged, and the thoracic cavity rarely contains excess fluid. In the mixed form, the post-mortem lesions are a mixture of typical findings from both the cardiac and pulmonary forms.</p>
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AHS in Other Species

- Dogs
 - Ingestion of infected horse meat
 - Not usually by insect bites
 - No role in spread or maintenance
 - Dogs usually have the pulmonary form
- Camels, zebras
 - Inapparent infection



Experimentally, dogs have been found to be susceptible to the AHS virus. Infection typically occurs following the consumption of virus-infected horse meat. Canines are not thought to become naturally infected with AHS through vector bites, and are also not considered important in the spread or maintenance of the virus. It is generally accepted that dogs play no role in the spread or maintenance of AHS because they do not develop viremia sufficient to infect vectors. Camels and zebras can be inapparently infected with AHS virus. In an Egyptian survey, antibodies were found in sheep, goats, and buffaloes. [Top photo: Dog. Source: Megan Smith, Iowa State University; Bottom photo: Camel. Source: <http://a-z-animals.com/animals/camel/>]

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DIAGNOSIS AND TREATMENT

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Differential Diagnosis

- Equine viral arteritis
- Equine infectious anemia
- Hendra virus infection
- Purpura hemorrhagica
- Equine piroplasmiasis
- Equine encephalosis virus
- Anthrax
- Toxins

The differential diagnosis includes equine viral arteritis, equine infectious anemia, Hendra virus infection, purpura hemorrhagica and equine piroplasmiasis. In Africa, equine encephalosis virus, another orbivirus transmitted by *Culicoides*, causes a syndrome resembling horsesickness fever. Toxins, anthrax and other causes of sudden death, as well as diseases that result in severe respiratory distress, should also be considered.

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Diagnosis

- Clinical signs
 - Supraorbital swelling is characteristic
- History
 - Prevalence or exposure to competent vectors
 - Travel from enzootic area
- Laboratory tests - definitive diagnosis
- Serotype needed for control measures

African horse sickness should be suspected in Equidae demonstrating the previously mentioned clinical signs. Supraorbital swellings are particularly characteristic of this disease. Additionally, a history of prevalence or exposure to competent vectors or of travel from an enzootic area can be important factors. Tentative diagnosis of AHS may be obtained by serology if blood is taken during the febrile stage; however, laboratory confirmation is essential for definitive diagnosis, and serotype determination will be important for control measures. Virus isolation and identification may be done from a number of tissues; samples taken should include small (2-4 g) sections of the spleen, lung, and lymph nodes. Samples for virus isolation should be stored and transported at 4°C. AHS viral antigens can be detected with enzyme-linked immunosorbent assays (ELISAs), as well as with a reverse-transcription polymerase chain reaction (RT-PCR), which can be particularly useful for rapid serotyping of viral RNA. Currently there is no efficient treatment for African horse sickness. Surviving Equidae develop solid immunity to the homologous serotype but remain susceptible to heterologous serotypes. Vaccines have been developed for all 9 serotypes of the virus, but are not currently available in the U.S.

S I d e 3 4	<p style="text-align: center;">Laboratory Diagnosis</p> <ul style="list-style-type: none"> • Laboratory tests <ul style="list-style-type: none"> – Virus isolation – ELISA, RT-PCR – Serology (tentative) – Necropsy: spleen, lung, lymph node • More than one test should be used • AHSV does not cross-react with other known orbiviruses 	<p>African horse sickness can be diagnosed by isolating the virus or detecting its nucleic acids or antigens. More than one test should be used to diagnose an outbreak (particularly the index case) whenever possible. AHSV antigens can be detected with enzyme-linked immunosorbent assays (ELISAs). A reverse-transcription polymerase chain reaction (RT-PCR) technique is used to detect viral RNA. A recently developed type-specific RT-PCR assay can be used for rapid serotyping. Serology can also be used to diagnose African horse sickness. Antibodies can be detected within 8 to 14 days after infection, and may persist for one to four years. The indirect ELISA and complement fixation tests are the prescribed tests for international trade. AHSV does not cross-react with other known orbiviruses.</p>
S I d e 3 5	<p style="text-align: center;">Sampling</p> <ul style="list-style-type: none"> • Before collecting or sending any samples, the proper authorities should be contacted. • Samples should only be sent under secure conditions and to authorized laboratories to prevent the spread of the disease. 	<p>Before collecting or sending any samples from animals with a suspected foreign animal disease, the proper authorities should be contacted. Samples should only be sent under secure conditions and to authorized laboratories to prevent the spread of the disease.</p>
S I d e 3 6	<p style="text-align: center;">Samples To Collect</p> <ul style="list-style-type: none"> • For virus isolation <ul style="list-style-type: none"> – Blood samples – Necropsy samples <ul style="list-style-type: none"> • Spleen, lung, lymph nodes • Paired serum samples are recommended • Store and transport samples at 39°F 	<p>In live animals, blood samples collected into anticoagulant should be taken for virus isolation. Success is most likely if these samples are collected early during the febrile stage. Necropsy samples for virus isolation should include samples of the spleen, lung and lymph nodes. The samples for virus isolation should be stored and transported at 4°C (39°F). Serum should also be taken for serology. Paired serum samples are recommended, and are particularly important in areas where the disease is endemic.</p>
S I d e 3 7	<p style="text-align: center;">AFRICAN HORSE SICKNESS IN HUMANS</p>	
S I d e 3 8	<p style="text-align: center;">AHS in Humans</p> <ul style="list-style-type: none"> • No natural infection in humans • Neurotropic vaccine strains <ul style="list-style-type: none"> – Transnasal infection can lead to encephalitis or retinitis • Handle modified live AHS vaccine strains with caution 	<p>There is no evidence that humans can become infected with field strains of AHS virus through contact with infected animals, nor from working in laboratories; however, it has been shown that certain neurotropic vaccine strains may cause encephalitis and retinitis in humans following transnasal infection. Modified live vaccine strains of AHS should be handled with caution.</p>

S I d e 3 9	<p>PREVENTION AND CONTROL</p>	
S I d e 4 0	<p style="text-align: center;">Recommended Actions</p> <ul style="list-style-type: none"> • IMMEDIATELY notify authorities <ul style="list-style-type: none"> – OIE reportable disease • In the U.S. notify <ul style="list-style-type: none"> – Federal Area Veterinarian in Charge (AVIC) <ul style="list-style-type: none"> www.aphis.usda.gov/animal_health/area_offices/ – State Veterinarian <ul style="list-style-type: none"> www.usaha.org/Portals/6/StateAnimalHealthOfficials.pdf • Quarantine premises 	<p>African horse sickness is reportable to the World Organization for Animal Health (OIE). Disease notification requirements for OIE member nations and import/export guidelines can be found in the OIE Terrestrial Animal Health Code [http://www.oie.int/eng/normes/mcode/A_summry.htm].</p> <p>If you suspect a case or outbreak of African horse sickness, contact your state and/or federal veterinarian immediately and quarantine the premises. If AHS is detected in a non-endemic country, a strict quarantine zone should be established. Individual veterinarians who encounter a case of African horse sickness should follow their national and/or local guidelines for disease reporting and diagnostic testing.</p>
S I d e 4 1	<p style="text-align: center;">Disinfection</p> <ul style="list-style-type: none"> • Disinfectants <ul style="list-style-type: none"> – Sodium hypochlorite (bleach) – 2% acetic or citric acid • Killed <ul style="list-style-type: none"> – pH less than 6 – pH 12 or greater • Rapidly destroyed in carcasses that have undergone rigor mortis 	<p>The AHS virus can be inactivated in the laboratory by formalin, β - propiolactone, acetyl-ethyleneimine derivatives, or radiation. Sodium hypochlorite (bleach) is an effective disinfectant against the virus. The virus is also destroyed at a pH less than 6 or greater than 12, so acidic disinfectants, such as 2% acetic or citric acid have been recommended for decontamination when warranted. Due to pH fluctuations, the AHS virus is rapidly destroyed in carcasses that have undergone rigor mortis; however the virus can survive in frozen meat, but is inactivated at temperatures greater than 60°C (140°F).</p>
S I d e 4 2	<p style="text-align: center;">Control</p> <ul style="list-style-type: none"> • Quarantine <ul style="list-style-type: none"> – Equidae from endemic areas <ul style="list-style-type: none"> • Asia, Africa, Mediterranean – Minimum 60 days at point of entry • Vector control and protection <ul style="list-style-type: none"> – Insect repellants – Stable in insect-proof housing from dusk to dawn 	<p>Horses cannot enter the US from an African horse sickness endemic country unless they have been in an AHS-free country or area of the world for at least 60 days. Upon entering the US, horses are then subject to the regular 3 or 7 day quarantine period at the point of entry. If African horse sickness is detected in a country where it is not endemic, a strict quarantine zone and movement controls should be established. Euthanasia of infected and exposed animals may be considered. Whenever possible, all Equidae should be stabled in insect-proof housing. At a minimum, stabling from dusk to dawn, the period when <i>Culicoides</i> are most active, is recommended. Vector control measures such as modifications of <i>Culicoides</i> breeding areas, insect repellents, and targeted applications of insecticides or larvicides may also be useful.</p>
S I d e 4 3	<p style="text-align: center;">Control</p> <ul style="list-style-type: none"> • Monitor temperature of all equids <ul style="list-style-type: none"> – If febrile <ul style="list-style-type: none"> • Euthanize or isolate in an insect-free stable until cause is determined • Vaccination <ul style="list-style-type: none"> – In endemic areas – Surrounding protection zone – Not available in the U.S. 	<p>Monitoring for fever may be helpful for the early detection of African horse sickness. Each susceptible animal should have its temperature taken regularly (optimally, twice daily). Those animals that develop a fever should be kept in insect-free stables until the cause of the fever has been established, or killed to prevent potential virus transmission to the vector. In endemic areas, vaccination is strongly recommended for susceptible Equidae. Additionally, areas around the affected area should vaccinate, as well, to produce a surrounding protection zone.</p>

<p>S I d e 4 4</p>	<h3>Vaccination</h3> <ul style="list-style-type: none"> • Attenuated live vaccine available <ul style="list-style-type: none"> – Horses, mules, donkeys – Not in U.S. – Reassortment possible – Teratogenic • No killed or subunit vaccine available • Recovering animals <ul style="list-style-type: none"> – Lifelong immunity post-infection to the infecting serotype 	<p>Vaccines have been developed for the 9 serotypes of AHS virus. Attenuated (monovalent and polyvalent) live (Vero cell) vaccines for use in horses, mules, and donkeys are currently available in some countries (but not in the U.S. at this time). These vaccines result in viremia, and the viruses could theoretically reassort with an outbreak virus. Attenuated vaccines may not be safe in AHS-free countries. They are also teratogenic. No killed or subunit vaccines are currently manufactured commercially. Animals that recover from the disease develop solid life-long immunity against the infecting viral serotype.</p>
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<p>S I d e 4 5</p>	<h3>Additional Resources</h3> <ul style="list-style-type: none"> • World Organization for Animal Health (OIE) <ul style="list-style-type: none"> – www.oie.int • Center for Food Security and Public Health <ul style="list-style-type: none"> – www.cfsph.iastate.edu • USAHA Foreign Animal Diseases (“The Gray Book”) <ul style="list-style-type: none"> – www.aphis.usda.gov/emergency_response/downloads/nahems/fad.pdf • Center for Infectious Disease Research and Policy <ul style="list-style-type: none"> – www.cidrap.umn.edu/cidrap/content/biosecurity/ag-biosec/anim-disease/ahs.htm • African Horse Sickness Trust <ul style="list-style-type: none"> – www.africanhorsesickness.co.za 	<p>For more information, see these listed resources. References used for this presentation:</p> <ul style="list-style-type: none"> • Center for Food Security and Public Health (CFSPH). African horse sickness. Technical Fact Sheet. 30 Nov 2006. http://www.cfsph.iastate.edu/Factsheets/pdfs/african_horse_sickness.pdf • U.S. Animal Health Association (USAHA). Foreign Animal Diseases, 7th edition. 2008. Guthrie, AJ. African Horse Sickness. p103-109. www.aphis.usda.gov/emergency_response/downloads/nahems/fad.pdf • Center for Infectious Disease Research and Policy (CIDRAP). African horse sickness. 26 Sept 2004. http://www.cidrap.umn.edu/cidrap/content/biosecurity/ag-biosec/anim-disease/ahs.html • U.S. Department of Agriculture, Animal and Plant Health Inspection Service (USDA APHIS). Horse Reports and Highlights – African Horsesickness. http://www.aphis.usda.gov/animal_health/emergingissues/byspeciescategory/horse.shtml
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<p>S I d e 4 6</p>	<h3>Acknowledgments</h3> <p><i>Development of this presentation was funded by grants from the Centers for Disease Control and Prevention, the Iowa Homeland Security and Emergency Management Division, and the Iowa Department of Agriculture and Land Stewardship to the Center for Food Security and Public Health at Iowa State University.</i></p> <p><small>Authors: Glenda Dvorak, DVM, MPH, DACVPM; Anna Rowid Spickler, DVM, PhD Reviewers: James A. Roth, DVM, PhD; Sindy Coritto, BA; Katie Spaulding, BS; Meghan Blankenship, BS; Kerry Leedom Larson, DVM, MPH, PhD, DACVPM</small></p> <p style="text-align: center; font-size: small;">Center for Food Security and Public Health, Iowa State University, 2011</p>	
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