African Horse Sickness

Perdesiekte,
Pestis Equorum,
Peste Equina,
Peste Equina Africana

Last Updated: May 2024

Importance

African horse sickness (AHS) is a serious arthropod-borne viral disease of equids, with a mortality rate that can reach 50-95% during outbreaks among naive horses. This disease is only endemic in sub-Saharan Africa at present; however, suitable vectors exist outside this area, and infected animals or vectors have occasionally carried the virus into AHS-free regions, most recently to Thailand and Malaysia in 2020. Some of these outbreaks were contained quickly and affected relatively few animals, but others lasted for a few years, with one extensive outbreak in the Middle East and Asia estimated to have resulted in the death of 300,000 equids in 1959-1962. The potential for dissemination is particularly high in animals that tend to develop mild or subclinical infections, such as zebras and donkeys, or horses with partial immunity.

As well as being an important trade issue, African horse sickness can be a significant economic burden in parts of Africa where draft horses are still common. In recent decades, new viruses have spread to some regions where only a single serotype was previously found, sometimes causing outbreaks when the locally used vaccines were not protective against the newly introduced serotype.

Etiology

African horse sickness can be caused by any of the nine serotypes of African horse sickness virus (AHSV), which belongs to the genus *Orbivirus* in the family Reoviridae. While some serotypes are cross-protective (e.g., serotypes 6 and 9), others are not.

Species Affected

African horse sickness mainly affects equids (horses, donkeys, mules, zebras), which also act as its maintenance hosts. However, this disease is known to occur in dogs, and antibodies to AHSV have been reported in other canids and wild carnivores, such as hyenas (*Crocuta crocuta*), jackals (various *Canis* spp.), African wild dogs (*Lycaon pictus*), cheetahs (*Acinonyx jubutus*), lions (*Panthera leo*) and large-spotted genets (*Genetta maculata*), though there are currently no reports of clinical cases in any of these species. Some authors have reported that wild carnivores may have antibodies to AHSV serotypes (e.g., serotype 4) that are not necessarily common among equids in the area. There are also some reports of seropositive herbivores including dromedary camels (*Camelus dromedarius*), sheep, goats, African elephants (*Loxodonta africana*), black rhinocerus (*Diceros bicornis*) and white rhinocerus (*Ceratotherium simum*). Attempts to establish experimental infections resulted in seroconversion with no evidence of virus replication in African elephants, and seroconversion in hyenas, while mink (*Mustela vison*) did not seroconvert or replicate virus. The significance of seropositive animals is still unclear, and no animals other than equids are thought to be important in maintaining or amplifying AHSV.

Zoonotic potential

AHSV is not zoonotic in its natural form; however, one neurotropic, mouse-adapted vaccine strain caused illness after accidental exposure in the laboratory.

Geographic Distribution

African horse sickness is endemic in sub-Saharan Africa, with the greatest virus diversity in the southern and eastern regions. Serotype 9 is widespread, while serotypes 1 to 8 occur in more limited areas. In recent decades, some of the latter viruses have spread to countries where they were not previously found. In particular, serotypes such as 2, 4, 6, 7 and 8 have been detected in Central Africa, where only serotype 9 was once common.

African horse sickness outbreaks have occurred sporadically outside Africa in the Middle East, the Mediterranean region of Europe and parts of Asia (e.g., the Indian subcontinent, Thailand, Malaysia). All outbreaks, to date, have been eradicated.

Transmission

Biting midges in the genus *Culicoides* are the biological vectors for AHSV. *Culicoides imicola* and *C. bolitinos* are known to transmit this virus in Africa, but other *Culicoides* species might also act as vectors there or on other continents. Laboratory

studies have identified at least one competent vector, *C. sonorensis* (formerly *C. variipennis*), in North America. How far *Culicoides* usually disperse from their breeding sites is unclear, but some species have been reported to fly up to several kilometers, and wind was implicated in the long-distance transport of these vectors in some AHS outbreaks. Adult *Culicoides* can survive mild winters, which may contribute to virus overwintering in some climates. Other arthropods such as mosquitoes, ticks or biting flies in the genera *Stomoxys* and *Tabanus* might also be capable of acting as mechanical or biological vectors, but they are thought to be, at most, only minor sources of infection.

AHSV does not spread between equids during casual contact, though it may be transmitted iatrogenically in blood during medical procedures. Zebras, horses, mules and donkeys can all develop viremia sufficient to infect *Culicoides*. The virus has been recovered from the blood of some experimentally infected horses for up to 21 days, though 4-8 days is more typical; from the blood of some experimentally infected zebras for up to 40 days (and the spleen at 48 days); and from donkeys for as long as 28 days, with viral RNA sometimes found up to 47 days or more.

Dogs can be infected by eating meat from infected equids, but there is increasing evidence that some dogs acquire the virus from biting midges. AHSV can persist for some time in frozen meat, and it is very stable at 4ºC (39ºF), particularly in the presence of stabilizing agents such as serum. It was reported to survive for up to 6 months at 4ºC in medium containing serum, and for more than 2 years in putrid blood. While dogs have transmitted AHSV to other dogs or horses via arthropods (including ticks) in laboratory experiments, viremia is usually low and transient, and dogs are not thought to be significant in the epidemiology of this disease.

Disinfection

AHSV is destroyed by acid (pH < 6) or basic (pH ≥ 12) conditions, and it can be inactivated with agents such as 2% acetic acid. Other disinfectants reported to be effective include formalin, β-propiolactone, acetylethyleneimine derivatives, Virkon® S and sodium hypochlorite (3%); however, this virus is resistant to lipid solvents such as ether. It is relatively heat stable, but can be inactivated by heating at72ºC (162ºF) for 2 hours.

Incubation Period

The incubation period for African horse sickness ranges from 2 days to 2 weeks (usually < 9 days), though experimental infections suggest that some cases might appear as late as 21 days. The pulmonary form has a shorter incubation period than the cardiac form.

Clinical Signs

Four different forms of African horse sickness have been recognized: the peracute (pulmonary) form, the subacute edematous (cardiac) form, the acute (mixed) form and horsesickness fever. Sudden death is also reported. Severe illnesses are seen most often in horses and mules, with the pulmonary and mixed forms usually predominating in previously unexposed horses, including foals that have lost their maternal antibodies. The mildest form, horsesickness fever, tends to develop in resistant species such as donkeys, or in other animals that have partial immunity. This form can also occur in zebras, although most infections in this species are asymptomatic.

The peracute or pulmonary form

The pulmonary form of African horse sickness usually begins with an acute fever, followed within a day or two by the sudden onset of severe respiratory distress. Animals with this form often stand with forelegs spread, head extended, and nostrils fully dilated. Common clinical signs 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.

The subacute edematous or cardiac form

The cardiac form begins with a period of fever and nonspecific signs of illness, usually lasting less than a week. Shortly before the fever starts to subside, edematous swellings appear in the supraorbital fossae and eyelids. These swellings later spread to involve the face, tongue, intermandibular space, laryngeal region, and sometimes the neck, shoulders and chest. Edema of the lower legs or ventral abdomen is rare. Other clinical signs, usually seen in the terminal stages of the disease, can include severe depression, colic, petechiae or ecchymoses on the ventral surface of the tongue, and petechiae in the conjunctivae. Death often occurs from cardiac failure. If the animal recovers, the swellings gradually subside over a few days to a week.

The acute or mixed form

The mixed form, which includes both pulmonary and cardiac signs, is the most common form of African horse sickness. However, the involvement of both organ systems may be difficult to recognize except at necropsy. In most cases, the cardiac form is subclinical but is followed by severe respiratory distress indistinguishable from pulmonary AHS. Occasionally, mild respiratory signs may be followed by edema and death from cardiac failure.

Horsesickness fever

Horsesickness fever is characterized mainly by fever, often with morning remissions and afternoon exacerbations. Other signs are generally minor and may include mild anorexia or depression, edema of the supraorbital fossae, congested mucous membranes and an increased heart rate. Affected animals are expected to recover without issues.

Infections in dogs

Cases described in naturally infected dogs, to date, have appeared as an acute respiratory illness, with a brief period of nonspecific signs (e.g., fever, anorexia, lethargy) followed by pulmonary involvement with excessive salivation, severe dyspnea, tachypnea, and crackles on lung auscultation. Affected animals usually deteriorate rapidly and, in the

terminal stage, sometimes have copious serous oronasal discharge or foam around the nostrils. Almost all reported cases have been fatal. However, antibodies have sometimes been found in dogs living in endemic regions, suggesting that milder cases or subclinical infections are also possible, and AHSV has been isolated from feral dogs not reported to be ill. In early experiments, which used crude preparations of virus (e.g., filtered blood from infected horses), some dogs had no apparent signs of illness, while others developed febrile reactions and survived, or died with evidence of severe pulmonary disease.

Post Mortem Lesions

Click to view images

Equids

The characteristic lesions of the pulmonary form of African horse sickness include interlobular edema of the lungs and hydrothorax. The lungs are typically mottled red, non-collapsed and heavy, with distended interlobular septae. Some animals may have frothy fluid in the trachea and pulmonary airways, which can flow from the nostrils and the cut surface of the lungs. In more prolonged cases, there may be extensive interstitial and subpleural edema, and hyperemia of the lungs may be less apparent. There are also occasional reports of extensive fluid accumulation in the thoracic cavity with near normal appearance of the lungs. The lymph nodes are usually enlarged and edematous, particularly in the thoracic and abdominal cavities. Additional lesions in some animals may include fluid in the abdominal cavity; subcapsular hemorrhages in the spleen; congestion of the renal cortex, edematous infiltration around the aorta and trachea; petechial hemorrhages on the pleura and various serosal surfaces; and hyperemia and petechiae in the small and large intestines, with hyperemia of the gastric fundus. Cardiac lesions are not prominent, although petechiae may be found on the pericardium and there may be increased pericardial fluid.

In the cardiac form, there is usually a yellow gelatinous infiltrate in the subcutaneous and intermuscular fascia of the head, neck and shoulders, and occasionally the brisket, ventral abdomen and rump. Hydropericardium is common, and the epicardium and endocardium often contain petechial and ecchymotic hemorrhages. Other lesions can include ascites or gastrointestinal lesions that resemble those in the pulmonary form, as well as prominent submucosal edema in the cecum, large colon and rectum. The lungs are usually normal or slightly edematous/engorged, and the thoracic cavity rarely contains excess fluid. Animals with the mixed form have a mixture of lesions from both the cardiac and pulmonary forms.

Dogs

The gross lesions reported in dogs have been consistent with pulmonary disease, and include hydrothorax, mediastinal edema, and pulmonary congestion and edema, as well as areas of emphysema and/or areas of hepatization in the lungs of some animals. In some cases, the fluid in the lungs (clear and straw-colored) gelled on exposure to the air. Red-tinged foam has been noted sometimes in the airways. Some naturally or experimentally infected dogs also had hyperemia of the intestinal mucosa, petechiae and ecchymoses on the endocardium, and congestion of the liver and other internal organs.

Diagnostic Tests

African horse sickness is often diagnosed by virological methods, as many animals do not survive until antibodies become detectable, usually around 8-14 days after infection. AHSV, its nucleic acids and antigens can be found in the blood of live animals or in various tissues, especially the spleen, lung and lymph nodes, collected at necropsy. Virus isolation from the blood is most likely to be successful if samples are collected early in the febrile stage.

Reverse-transcription polymerase chain reaction (RT-PCR) assays are often used to diagnose clinical cases. They can also recognize subclinical infections during import testing. Some tests detect all serotypes, while others are serotype-specific. The latter can be used for rapid serotyping of field isolates, and RT-PCR with sequencing can distinguish vaccine strains from field strains in clinical samples. Loop-mediated isothermal amplification (LAMP_ assays have also been published. AHSV antigens can be detected in the blood or tissues with ELISAs.

AHSV can be isolated in various mammalian cell lines such as BHK-21 or Vero cells, in *Culicoides* or mosquito cell lines (e.g., KC cells), and in embryonated eggs. If necessary, the virus can also be recovered by intracerebral inoculation of newborn mice. Isolates can be serotyped (e.g., for vaccine selection) by virus neutralization or RT-PCR.

Animals that survive longer or have mild illnesses can be diagnosed by serology. Serological tests include ELISAs, immunoblotting, virus neutralization and complement fixation, though the last test is now infrequently used. Other assays (e.g., immunodiffusion, hemagglutination inhibition) have also been described. Paired samples are recommended, particularly where the disease is endemic, as previous exposures or vaccination can complicate test interpretation. AHSV does not cross-react with any other known orbivirus, and cross-reactivity between AHSV serotypes is variable.

Treatment

Treatment is symptomatic and supportive.

Control

Disease reporting

A quick response is important for containing outbreaks in AHS-free regions, as the virus is more difficult to control once it has entered a vector population. Veterinarians who encounter or suspect African horse sickness should follow their national and/or local guidelines for disease reporting. In the U.S., state or federal veterinary authorities should be informed immediately.

Prevention

Live attenuated vaccines containing various serotypes are routinely used to control African horse sickness in endemic regions. They must be matched to the serotype(s) of the circulating viruses (or to a cross-protective serotype) and do not necessarily provide sterile immunity. Live attenuated AHS vaccines have some drawbacks, including potential transmission to other animals by *Culicoides* vectors, reassortment with field viruses, and teratogenicity in pregnant mares. No other types of vaccines are commercially available at present, though inactivated vaccines were sometimes used during outbreaks outside Africa in the past.

Reduction of exposure to the *Culicoides* vectors is also likely to be helpful, though it is unlikely to be effective as the sole control measure. Some possibilities include avoiding environments where midges are more prevalent (e.g., low-lying, damp pastures), using insect repellents or barriers such as insecticide-impregnated nets, and stabling animals from late afternoon to after dawn to prevent exposure to peak *Culicoides* feeding activity, which occurs around sunset and sunrise in the summer. However, it should be noted that some species of *Culicoides* will enter barns and stables, especially late in the season, and that outdoor feeding activity also begins earlier in the evening as the weather cools. One study suggested that open stables might provide some degree of protection from *C. imicola*, but did not protect horses from *C. bolitinos*.

Insecticide-treated netting can also help protect AHSV-free horses being transported through endemic areas, though its efficacy is not absolute. Effective control of *Culicoides* in the environment with insecticides or other measures is challenging and generally considered impractical, due to their extensive breeding sites and large populations, as well as the current poor understanding of the degree of control needed to actually reduce disease incidence. Surveillance of sentinel animals might provide early warnings of outbreaks in endemic regions or detect the introduction of a new serotype.

Testing and quarantine can help prevent imported equids from introducing AHSV into countries free of this virus. If an outbreak occurs in an AHS-free country, control measures include the establishment of quarantines and movement controls, vaccination campaigns in some instances, and possibly the euthanasia of infected animals, depending on the situation. Stabling equids in insect-proof housing, at least overnight, can provide some protection to uninfected animals, and may also reduce the risk that infected animals will transmit the virus to vectors.

Morbidity and Mortality

African horse sickness is a common disease in sub-Saharan Africa, and significant numbers of equids (e.g., 34% of equids and up to 50% of donkeys in Ethiopia) have been exposed to this virus in some regions. Outbreaks tend to occur in late summer and autumn, with cycles sometimes recurring at irregular intervals. Climatic conditions reported to favor epidemics are heavy rain alternating with hot, dry periods. The onset of cold temperatures has ended some AHS outbreaks outside Africa, but the virus has persisted over multiple years in other cases, including in parts of Europe.

Clinical cases in equids vary in severity, depending on the virulence of the viral strain and species of animal, as well as its level of immunity. The mixed and pulmonary forms of African horse sickness tend to predominate in previously unexposed horse populations, where mortality is often 50-95% during outbreaks. Animals that develop the pulmonary form almost always die, while the cardiac form has a case fatality rate of 50% or more in horses. Estimates of the case fatality rate in the mixed form range from 70% to greater than 80%.

Other equids are less susceptible. The mortality rate is often around 50% in mules during epidemics, and 5-10% in donkeys imported from Asia or Europe. Native African donkeys rarely have serious clinical signs, though they may sometimes develop horsesickness fever. This mild illness can also be seen in horses or mules that have some pre-existing immunity and/or are infected with less virulent viruses, and occasionally in zebras. Asymptomatic infections are common in zebras, and can also be seen in other equids, including vaccinated horses. Equids that recover from African horse sickness develop good immunity to the infecting serotype and partial immunity to other serotypes.

At least 50 cases of AHS, almost all fatal, have been reported in dogs. Treatment attempts have generally been unsuccessful, and in one outbreak, 15 of 17 dogs died after being fed meat from a sick horse. Nevertheless, one study of experimentally infected dogs, as well as evidence for antibodies or viruses in healthy dogs, suggest that milder cases or subclinical infections may also occur. Seroprevalence among dogs in endemic regions generally appears to be < 10%.

Public Health

Humans are not natural hosts for the African horse sickness virus, and no cases have ever been seen after contact with field strains. However, facial (nasal) exposure to broken vials containing a neurotropic vaccine strain adapted to mice caused encephalitis and retinitis after a laboratory accident.

Internet Resources

American Association of Equine Practitioners (AAEP). Resource Library

Animal Health Australia. African Horse Sickness. AUSVETPLAN Response Strategy

Department of Agriculture, Land Reform and Rural Development, Government of South Africa. Recommendations for Protecting Equines from AHS in the AHS Infected Zone

EFSA Panel on Animal Health and Welfare. Scientific Opinion on the Assessment of the Control Measures of the Category A Diseases of Animal Health Law: African Horse Sickness

National Horseracing Authority, South Africa. African Horse Sickness

The Merck Veterinary Manual

United States Animal Health Association. Foreign Animal Diseases

United States Department of Agriculture (USDA) Animal and Plant Health Inspection Service (APHIS). Disease Alert: African Horse Sickness

USDA APHIS. Foreign Animal Disease Preparedness and Response Plan. African Horse Sickness Standard Operating Procedures

World Organization for Animal Health (WOAH)

WOAH. African Horse Sickness (including information about AHS-free countries)

WOAH Regional Representation for Asia and the Pacific. Asia and the Pacific. African Horse Sickness: Communication and Preparedness Materials

WOAH Manual of Diagnostic Tests and Vaccines for Terrestrial Animals

WOAH Terrestrial Animal Health Code

Acknowledgements

This factsheet was written by Anna Rovid Spickler, DVM, PhD, Veterinary Specialist from the Center for Food Security and Public Health. The U.S. Department of Agriculture Animal and Plant Health Inspection Service (USDA APHIS) provided funding for this factsheet through a series of cooperative agreements related to the development of resources for initial accreditation training.

The following format can be used to cite this factsheet. Spickler, Anna Rovid. 2024. *African Horse Sickness*. Retrieved from http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.php.

References

Agriculture and Resource Management Council of Australia and New Zealand. Australian veterinary emergency plan operational procedures manual [online]. Disease strategy: African horse sickness; 1996. Available at: http://www.animalhealthaustralia.com.au/fms/Animal Health Australia/AUSVETPLAN/decfnl2.pdf.* Accessed 4 Dec 2006.

Aklilu N, Batten C, Gelaye E, Jenberie S, Ayelet G, Wilson A, Belay A, Asfaw Y, Oura C, Maan S, Bachanek-Bankowska K, Mertens PP. African horse sickness outbreaks caused by multiple virus types in Ethiopia. Transbound Emerg Dis. 2014;61(2):185-92.

Alexander KA, Kat PW, House J, House C, O'Brien SJ, Laurenson MK, McNutt JW, Osburn BI. African horse sickness and African carnivores. Vet Microbiol. 1995;47(1-2):133-40.

Awad FI, Amin MM, Salama SA, Aly MM. The incidence of African horse sickness antibodies in animals of various species in Egypt. Bull Anim Health Prod Afr. 1981;29(3):285-7.

Baker T, Carpenter S, Gubbins S, Newton R, Lo Iacono G, Wood J, Harrup LE. Can insecticide-treated netting provide protection for equids from *Culicoides* biting midges in the United Kingdom? Parasit Vectors. 2015;8:604.

Barnard BJ. Epidemiology of African horse sickness and the role of the zebra in South Africa. Arch Virol Suppl. 1998;14:13-9.

Barnard BJ, Bengis RG, Keet DF, Dekker EH. Epidemiology of African horsesickness: antibodies in free-living elephants (*Loxodonta africans*) and their response to experimental infection. Onderstepoort J Vet Res. 1995;62(4):271-5.

Barnard BJ, Bengis R, Keet D, Dekker EH. E pidemiology of African horsesickness: duration of viraemia in zebra (*Equus burchelli*). Onderstepoort J Vet Res. 1994;61(4):391-3.

Bevan LEW. The transmission of African horse-sickness to the dog by feeding. Vet J. 1911; 67:402-8.

Binepal VS, Wariru BN, Davies FG, Soi R, Olubayo R. An attempt to define the host range for African horse sickness virus (Orbivirus, Reoviridae) in East Africa, by a serological survey in some Equidae, Camelidae, Loxodontidae and Carnivore. Vet Microbiol. 1992;31(1):19-23.

Braverman Y1, Chizov-Ginzburg A. Role of dogs (*Canis domesticus*) as hosts for African horse sickness virus. Vet Microbiol. 1996;51(1-2):19-25.

Bunpapong N, Charoenkul K, Nasamran C, Chamsai E, Udom K, Boonyapisitsopa S, Tantilertcharoen R, Kesdangsakonwut S, Techakriengkrai N, Suradhat S, Thanawongnuwech R, Amonsin A. African horse sickness virus serotype 1 on horse farm, Thailand, 2020. Emerg Infect Dis. 2021;27(8):2208-11.

Carpenter S, Mellor PS, Fall AG, Garros C, Venter GJ. African horse sickness virus: history, transmission, and current status. Annu Rev Entomol. 2017;62:343-58.

Crafford JE, Lourens CW, Smit TK, Gardner IA, MacLachlan NJ, Guthrie AJ. Serological response of foals to polyvalent and monovalent live-attenuated African horse sickness virus vaccines. Vaccine. 2014;32(29):3611-6.

Dennis SJ, Meyers AE, Hitzeroth II, Rybicki EP. African horse sickness: a review of current understanding and vaccine development. Viruses. 2019;11(9):844.

Diouf ND, Etter E, Lo MM, Lo M, Akakpo AJ. Outbreaks of African horse sickness in Senegal, and methods of control of the 2007 epidemic. Vet Rec. 2013;172(6):152.

Durán-Ferrer M, Villalba R, Fernández-Pacheco P, Tena-Tomás C, Jiménez-Clavero MÁ, Bouzada JA, Ruano MJ, Fernández-Pinero J, Arias M, Castillo-Olivares J, Agüero M. Clinical, virological and immunological responses after experimental infection with African horse sickness virus serotype 9 in immunologically naïve and vaccinated horses. Viruses. 2022;14(7):1545.

Durr PA, Graham K, van Klinken RD. Sellers' revisited: a big data reassessment of historical outbreaks of bluetongue and African horse sickness due to the long-distance wind dispersion of *Culicoides* midges. Front Vet Sci. 2017;4:98.

EFSA Panel on Animal Health and Welfare; Nielsen SS, Alvarez J, Bicout DJ, Calistri P, Depner K, et al. Scientific opinion on the assessment of the control measures of the category A diseases of Animal Health Law: African horse sickness. EFSA J. 2021;19(2):e06403.

el Hasnaoui H1, el Harrak M, Zientara S, Laviada M, Hamblin C. Serological and virological responses in mules and donkeys following inoculation with African horse sickness virus serotype 4. Arch Virol Suppl. 1998;14:29-36.

Erasmus BJ, Guthrie AJ. African horse sickness. In: Foreign animal diseases. Richmond, VA: United States Animal Health Association; 1998. Available at: http://www.vet.uga.edu/vpp/gray_book02/fad/ahs.php.* Accessed 29 Nov 2006.

Erasmus BJ, Young E, Pieterse LM, Boshoff ST. The susceptibility of zebra and elephants to African horsesickness virus. Proceedings of the Fourth International Conference on Equine Infectious Diseases, Lyon. Princeton, NJ: Veterinary Publications Inc.; 1976.

Fischer-Tenhagen C, Hamblin C, Quandt S, Frölich K. Serosurvey for selected infectious disease agents in free-ranging black and white rhinoceros in Africa. J Wildl Dis. 2000;36(2):316-23.

Fowler VL, Howson ELA, Flannery J, Romito M, Lubisi A, Agüero M, Mertens P, Batten CA, Warren HR, Castillo-Olivares J. Development of a novel reverse transcription loop-mediated isothermal amplification assay for the rapid detection of African horse sickness virus. Transbound Emerg Dis. 2017;64(5):1579-88.

Guthrie AJ. African horse sickness. In: Foreign animal diseases. Richmond, VA: United States Animal Health Association; 2008. p. 103-9.

Guthrie AJ, Maclachlan NJ, Joone C, Lourens CW, Weyer CT, Quan M, Monyai MS, Gardner IA. Diagnostic accuracy of a duplex real-time reverse transcription quantitative PCR assay for detection of African horse sickness virus. J Virol Methods. 2013;189(1):30-5.

Hanekom J, Lubisi BA, Leisewitz A, Guthrie A, Fosgate GT. The seroprevalence of African horse sickness virus, and risk factors to exposure, in domestic dogs in Tshwane, South Africa. Prev Vet Med. 2023;213:105868.

Hopley R, Toth B. Focus on: African horse sickness. Vet Rec. 2013;173(1):13-4.

King S, Rajko-Nenow P, Ashby M, Frost L, Carpenter S, Batten C. Outbreak of African horse sickness in Thailand, 2020. Transbound Emerg Dis. 2020;67(5):1764-7.

Maclachlan NJ, Guthrie AJ. Re-emergence of bluetongue, African horse sickness, and other orbivirus diseases. Vet Res. 2010;41(6):35.

Mayo CE, Weyer CT, Carpenter MJ, Reed KJ, Rodgers CP, Lovett KM, Guthrie AJ, Mullens BA, Barker CM, Reisen WK, MacLachlan NJ. Diagnostic applications of molecular and serological assays for bluetongue and African horse sickness. Rev Sci Tech. 2021;40(1):91-104.

Meiswinkel R1, Baylis M, Labuschagne K. Stabling and the protection of horses from *Culicoides bolitinos* (Diptera: Ceratopogonidae), a recently identified vector of African horse sickness. Bull Entomol Res. 2000;90(6):509-15.

Mellor PS, Hamblin C. African horse sickness. Vet Res. 2004;35:445-66.

M'Fadyean J. The susceptibility of the dog to African horse sickness. J Comp Pathol Ther. 1910; 23:27-33.

Miller M, Buss P, Joubert J, Maseko N, Hofmeyr M, Gerdes T. Serosurvey for selected viral agents in white rhinoceros (*Ceratotherium simum*) in Kruger National Park, 2007. J Zoo Wildl Med. 2011;42(1):29-32.

Mullens BA, McDermott EG, Gerry AC. Progress and knowledge gaps in *Culicoides* ecology and control. Vet Ital. 2015;51(4):313-23.

O'Dell N, Arnot L, Janisch CE, Steyl JC. Clinical presentation and pathology of suspected vector transmitted African horse sickness in South African domestic dogs from 2006 to 2017. Vet Rec. 2018;182(25):715.

Page PC, Labuschagne K, Venter GJ, Schoeman JP, Guthrie AJ. Efficacy of alphacypermethrin-treated high density polyethylene mesh applied to jet stalls housing horses against *Culicoides* biting midges in South Africa. Vet Parasitol. 2015;210(1-2):84-90.

Robin M, Page P, Archer D, Baylis M. African horse sickness: The potential for an outbreak in disease-free regions and current disease control and elimination techniques. Equine Vet J. 2016;48(5):659-69.

Rodriguez-Sanchez B, Sanchez-Vizcaino JM, Uttenthal A, Rasmussen TB, Hakhverdyan M, et al. Improved diagnosis for nine viral diseases considered as notifiable by the world organization for animal health. Transbound Emerg Dis. 2008;55(5-6):215-25.

Sahu SP, Dardiri AH. Susceptibility of mink to certain viral animal diseases foreign to the United States. J Wildl Dis. 1979;15(3):489-94.

Salama SA, Dardiri AH, Awad FI, Soliman AM, Amin MM Isolation and identification of African horsesickness virus from naturally infected dogs in Upper Egypt. Can J Comp Med. 1981;45(4):392-6.

Scacchia M, Molini U, Marruchella G, Maseke A, Bortone G, Cosseddu GM, Monacoo F, Savini G, Pini A. African horse sickness outbreaks in Namibia from 2006 to 2013: clinical, pathological and molecular findings. Vet Ital. 2015;51(2):123-30.

Skowronek AJ1, LaFranco L, Stone-Marschat MA, Burrage TG, Rebar AH, Laegreid WW. Clinical pathology and hemostatic abnormalities in experimental African horsesickness. Vet Pathol. 1995;32(2):112-21.

Stern AW. African horse sickness. Compend Contin Educ Vet. 2011;33(8):E1-5.

Theiler A. The susceptibility of the dog to African horse sickness. J Comp Pathol Ther. 1910; 23:315-25.

Thompson GM1, Jess S, Murchie AK. A review of African horse sickness and its implications for Ireland. Ir Vet J. 2012;65(1):9.

United States Department of Agriculture (USDA) Animal and Plant Health Inspection Service (APHIS). Foreign animal disease preparedness and response plan. African horse sickness standard operating procedures. USDA APHIS; 2013 Aug. Available at: https://www.aphis.usda.gov/sites/default/files/sop_1_ee_ahs.pdf. Accessed 28 May 2024.

Van Rensberg IB, De Clerk J, Groenewald HB, Botha WS. An outbreak of African horsesickness in dogs. J S Afr Vet Assoc. 1981;52(4):323-5.

van Schalkwyk A, Ferreira ML, Romito M. Using a new serotype-specific polymerase chain reaction (PCR) and sequencing to differentiate between field and vaccine-derived African horse sickness viruses submitted in 2016/2017. J Virol Methods. 2019;266:89-94.

van Vuuren M, Penzhorn BL. Geographic range of vector-borne infections and their vectors: the role of African wildlife. Rev Sci Tech. 2015;34(1):139-49.

Wernery U, Joseph S, Raghavan R, Dyer B, Spendrup S. African horse sickness fever in vaccinated horses: short communication.J Equine Vet Sci. 2020;88:102967.

Weyer C. African horse sickness. In: Winter AL, Moses MA, editors. The Merck veterinary manual.Rathway, NJ: Merck and Co; 2021. Available at: https://www.merckvetmanual.com/generalized-conditions/african-horse-sickness/african-horse-sickness. Accessed 28 May 2024.

Weyer CT, Joone C, Lourens CW, Monyai MS, Koekemoer O, Grewar JD, van Schalkwyk A, Majiwa PO, MacLachlan NJ, Guthrie AJ. Development of three triplex real-time reverse transcription PCR assays for the qualitative molecular typing of the nine serotypes of African horse sickness virus. J Virol Methods. 2015;223:69-74.

Weyer CT, Quan M, Joone C, Lourens CW, MacLachlan NJ, Guthrie AJ. African horse sickness in naturally infected, immunised horses. Equine Vet J. 2013;45(1):117-9.

World Organization for Animal Health [WOAH] . Manual of diagnostic tests and vaccines [online]. Paris: WOAH; 2019. African horse sickness. Available at: https://www.woah.org/fileadmin/Home/eng/Health_standards/tahm/3.06.01_AHS.pdf. Accessed 25 May 2024.

Zientara S, Weyer CT, Lecollinet S. African horse sickness. Rev Sci Tech. 2015;34(2):315-27.

*Link is defunct

