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Bad year for anthrax in U.S. livestock

Laura Hatfield, contributing writer
CIDRAP News

Weather and soil conditions in several areas of the United States are leading to record livestock losses from anthrax. This summer, approximately 400 animals have died in North and South Dakota, Texas and Minnesota.

Anthrax is endemic in grazing animals in some regions where previous cases have occurred. If animals that die from anthrax are not properly buried or incinerated,

the bacterium that causes anthrax, *Bacillus anthracis*, can contaminate the ground under and around the carcass. The spores formed by *B. anthracis* survive

in the soil for decades, and heavy rain or construction can bring them to the surface where grazing animals inhale or ingest them. The disease can be rapidly fatal in infected animals before significant signs of illness are noticed.

South Dakota State Veterinarian Sam Holland said his state's outbreak began with a 660-head cattle and bison herd in Sully County. Since July 20, 155 animals from that herd have died, according to a July 29 press release. Anthrax has been confirmed in five additional herds, and laboratory test results are pending for another four. Affected counties are located in the central and northeast parts of the state. Brown, Hyde, Marshall, Potter and Sully counties all have confirmed or suspected infections. Nearly 200 animals have died in South Dakota.

Mortality is more than double the rate from a severe outbreak in 2002, when 53

animals in three South Dakota counties died. A veterinarian who examined an infected carcass came down with a rare case of cutaneous anthrax that year. His infection responded to antibiotic treatment.

Though transmission to humans is uncommon, Susan Keller, state veterinarian in North Dakota, said, "We recommend wearing long sleeves, gloves and a face mask when handling carcasses."

North Dakota producers are also experiencing livestock losses from the disease.

So far this year, approximately 200 grazing animals have died in 10 southeastern counties, according to an August 3 ProMED posting from Keller. A severe outbreak in North Dakota in

2000 killed nearly 150 animals.

In Texas, the anthrax outbreak this year is notable because of the location. Anthrax is reported nearly every year in the southwest region, but a recent discovery of the disease at two Sutton County ranches marks the first case in west-central Texas in more than 20 years.

The state has experienced heavy losses of deer and cattle, including severe outbreaks in 1997 and 2001, according to the World Organization for Animal Health.

Thurman Fancher, Area 6 director for the Texas Animal Health Commission, said in a press release, "Anthrax is under-reported because many ranchers in this area automatically dispose of carcasses and vaccinate livestock. . . . Anthrax is a reportable disease, however, and it's important to know when an outbreak occurs, so other ranchers can be notified to vaccinate."

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Watch for vesicular stomatitis

Larry Hollis
Extension Beef Veterinarian

Vesicular stomatitis (VS) has been diagnosed in several states this summer, and could be transported into Kansas with livestock movements to fairs and rodeos, as well as conventional livestock movements. The viral disease has been confirmed in New Mexico, Arizona, Utah, Colorado, Wyoming and Montana. The disease primarily affects horses, cattle, swine and sometimes sheep and goats. VS causes fever and blisters in the mouth, nostrils, hooves and teats, and looks like Foot and Mouth Disease (FMD). If you have a case of vesicular disease you should contact Kansas Animal Health Department immediately (785-296-2326) so the animals can be evaluated and FMD ruled out. Infected animals typically recover in a few days.

Since July 20, 155 animals from a herd in South Dakota have died from anthrax.

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Thank you to the Pfizer Animal Health Group, Livestock Division, Cattle Products Group, for financial assistance in publishing this newsletter.

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Minnesota has also lost animals to anthrax this year. The outbreak includes an unusual case of apparent co-infection of a herd with bovine tuberculosis. Though not confirmed by the veterinary lab at North Dakota State University, anthrax is suspected in a herd of cattle quarantined for infection with bovine tuberculosis, according to Terry Bolding, district veterinarian for the Minnesota Board of Animal Health. Bovine tuberculosis has not been seen in Minnesota since 1971.

Animal health officials urge farmers to report suspected cases of anthrax and adhere to quarantine restrictions. “Producers should consult their veterinarians as soon

as possible about having their animals vaccinated against the disease,” Holland urged in the South Dakota press release. Keller from North Dakota reminded farmers, “Burning carcasses and burying both the burned remains and the soil immediately under the carcass is the best means to clean a site. Texas Animal Health Commission regulations call for burning of an infected animal’s bedding and nearby manure in addition to the carcass itself.

Anthrax outbreaks among grazing animals present little threat to human health. “Visitors to the area should not be alarmed by anthrax,” said Fancher. “Just leave dead animals alone, and don’t pick up shed antlers or old animal bones.” Standard pro-

TECTIVE equipment is sufficient to protect persons handling infected carcasses.

Human inhalational anthrax from spores in the soil is extremely rare. Consumption of anthrax-contaminated meat is a potential source of gastrointestinal infection, but the Centers for Disease Control and Prevention reports there has never been a documented case of gastrointestinal anthrax in the United States. An article in *Morbidity Mortality Weekly Report* says this is because, “Livestock are vaccinated for anthrax in areas where the disease is endemic; animals routinely are inspected by federal and state meat inspectors before, during, and after slaughter; and raw meat is eaten infrequently.”

Biosecurity, biocontainment programs beneficial

*Michael W. Sanderson DVM, MS,
DACVPM, DACT*

Larry Hollis DVM, MAG

Biosecurity is preventing the introduction of pathogens or toxins that can damage cattle health or the safety and quality of a food product. Biocontainment is the control disease or intoxication within a herd. Biosecurity and biocontainment are important issues for food animal producers and veterinarians. With an increasingly global marketplace and increasing pressure on producers and veterinarians to minimize disease risk, biosecurity is an important component of an integrated production management program. Biosecurity programs may be efficiently integrated with Hazard Analysis Critical Control Point type programs to control food quality and safety, and minimize antimicrobial use and disease risk in cattle.

Biosecurity program development involves an assessment of the management practices of the farm that put it at increased risk of disease import or transmission within the herd. Once the risky practices are identified, a management plan can be implemented to control the risk of import or transmission.

We have several tools for the implementation of biosecurity and biocontainment programs. The implementation and importance of each depends on the individual farm and the importance of the individual disease agents for that farm. They include quarantine, testing, vaccination, traffic control, population density

control, environmental control and risk group segregation.

The application of these tools is specific to the specific disease agents, however, for agents that share a common transmission route, the methods of controlling disease are common. Management factors that help control disease associated with one agent are generally helpful against a number of agents.

The Voluntary Bovine Johne’s Disease Control Program is one way to implement biosecurity programs for clients. While the program is focused on prevention or control of Johne’s disease on the farm or ranch, the principles are useful for biosecurity and biocontainment of enteric disease in general. A well-done risk assessment and herd management plan will produce broader benefits to your clients than just Johne’s disease prevention. The risk assessment is a questionnaire covering management practices that affect Johne’s disease (enteric disease) risk. The results summarize the herd’s risk for Johne’s disease. The herd management plan is implemented based on the results of the risk assessment. Implementing the herd management plan benefits clients beyond Johne’s disease prevention and increases your involvement with herd management and consultation.

The USDA and the Kansas Animal Health Department will pay Kansas Joh-

ne’s certified veterinarians to conduct herd risk assessments and implement herd management plans for beef and dairy herds. The program will pay for the initial herd testing with a blood ELISA test if the producer wants to enter the testing program.

Kansas pays Johne’s certified veterinarians \$200 for completing a risk assessment

and herd management plan for beef herds and \$350 for dairy herds. Kansas will pay for 30 Johne’s ELISA tests from the herd through the KSU Diagnostic Lab.

To become a Johne’s certified veterinarian, attend a Kansas on-site training workshop (the last one was at June conference 2005, so watch for future dates), or complete online training at the University of Wisconsin Web site. Kansas has contracted with Wisconsin to provide free training to the first 100 Kansas veterinarians who sign up. If you are interested in online training, contact Mike Sanderson at sandersn@vet.k-state.edu for the Web site and access code.

For more information about the Voluntary Bovine Johne’s Disease Control Program and how it can help you implement biosecurity and biocontainment programs in your clients herds contact: Mike Sanderson, sandersn@vet.k-state.edu 785-532-5700

A well-done risk assessment and herd management plan will produce broader benefits to your clients than just Johne’s disease prevention.

Epidemiology and transmission of West Nile Virus disease

(Hayes EB, Komar N, Nasci RS, et al. *Emerging Infectious Diseases* 2005;11:1167-1173. The following was edited for the *Kansas Veterinary Quarterly*. The entire article along with one on the pathology and clinical manifestations of West Nile Virus in humans is available online at www.cdc.gov/eid)

West Nile virus (WNV) was first identified in the Western Hemisphere in 1999 as the cause of an outbreak of encephalitis in humans in New York City. The virus has spread across the continental United States into Canada and Mexico, and to the Caribbean Islands and Latin America. In the Western Hemisphere, most human cases of WNV have occurred in the United States, and 16,706 cases have been reported to the Centers for Disease Control through 2004. Of these cases, 7,096 (42.5 percent) were neuroinvasive, 9,268 (55.5 percent) were classified as West Nile fever and 342 (2.0 percent) had other or unspecified manifestations. The percentage of neuroinvasive infections is probably artificially high because the other forms are less likely to be identified. In the eastern United States, WNV disease has become endemic because it has occurred for six consecutive years. In the temperate zones of North America, the peak incidence is July through October. In the southern United States, cases have occurred as early as April and as late as December.

People of all ages appear to be equally susceptible, but the incidence of encephalitis and death increases with age, especially in people 60 to 89 years old. In 2002, the median age of people who developed encephalitis was 64 years (range 1 month to 99 years) compared with a median age of 49 years (range 1 to 97 years). Of the 2,942 cases of encephalitis reported in 2002, 276 (9 percent) were fatal. From 2002 through 2004 there were 1,051 reported cases of WNV in children less than 19 years old; 317 (30%) of these children developed meningitis and two with meningitis died.

Transmission

By far the most important source of infection is mosquitoes. West Nile virus is primarily transmitted by *Culex* mosquitoes, but other genera can be vectors. In North America, WNV has been identified in 59 species of mosquitoes. However, less than 10 of the species are important vectors. In the northeast, *Cx. pipiens*, the northern house mosquito, is the principal vector. In the south, *Cx. quinquefasciatus*, the southern house mosquito, is the principal vector, and west of the Mississippi River it is *Cx. tarsalis*. Vertical transmission has been demonstrated in all three species and the virus has been isolated from hibernating mosquitoes, which provides methods for the virus to overwinter in cold climates. Both hard and soft ticks can become infected, but they are not important in WNV transmission.

It appears that the level of virus in the blood of humans and other mammals is not high enough to infect mosquitoes. Thus, it is unlikely that humans or other mammals play a role in transmission.

Birds are the most important amplifying hosts for WNV. Experimentally, species in the orders Passeriformes (song birds), Charadriiformes (shorebirds), Strigiformes (owls) and Falconiformes (hawks) have sufficient virus in their blood to infect mosquitoes. Certain passerines, including common grackles, crows, jays, magpies, house finches and house sparrows are highly infectious to mosquitoes and have mortality rates of more than 40 percent. Species of Columbiformes (pigeons), Piciformes (woodpeckers) and Anseriformes (ducks) do not appear to be infectious for mosquitoes. Sufficient viremia to infect mosquitoes was found in experimentally infected alligators, but not in red-eared sliders, garter

snakes, green iguanas or bullfrogs. Lake frogs appear to be a competent reservoir for WNV in Russia.

Nonmosquitoborne Transmission

In 2002, a case of intrauterine transmission of WNV was identified. In 2003, the CDC reported WNV infection in 74 pregnant women. Most of the women that were followed to the end of pregnancy delivered healthy babies. One case of probable transmission through breast milk was also identified in 2002.

Transmission by blood transfusion was first documented in 2002, and in June 2003 the United States and Canada began testing donated blood for WNV and disposing of potentially infected blood components. Six cases of infection by blood transfusion were identified in 2003 and one case in 2004. Transmission by organ transplantation was identified in 2002. In addition, transplant patients are at increased risk of developing severe WNV disease. In Toronto in 2002 the estimated incidence of WNV encephalitis in organ transplant patients was about 40 times higher than the general population. It is unknown whether other groups of immunocompromised people are at increased risk.

WNV has been contracted by laboratory workers by accidental percutaneous inoculation and possibly by aerosol. There is a case of WNV infection in workers at a turkey farm that might have been by aerosol transmission. Nonmosquitoborne transmission has been shown or strongly suspected in farmed alligators, domestic turkeys and domestic geese, and transmission by close contact has been demonstrated experimentally in birds and alligators.

In spite of these nonmosquitoborne methods of transmission, mosquitoes are by far the most important vector. The cornerstones of control and prevention of WNV infection are mosquito control and prevention of WNV-infected mosquitoes feeding on humans.

West Nile virus is primarily transmitted by *Culex* mosquitoes, but other genera can be vectors.

In the Western Hemisphere, most human cases of West Nile Virus have occurred in the United States, and 16,706 cases have been reported to the Centers for Disease Control through 2004.

Lawsuit claims veterinarians misrepresent vaccination need

You may be a party to a potential class-action lawsuit arising from an alleged misrepresentation of the need for vaccinations for pets.

Veterinarians working on small animals are targeted for lawsuits. The solicitation for participation in the lawsuit against veterinarians who recommend allegedly excessive vaccination programs can be found online at www.dogsadversereactions.com/classaction.html. The Chicago law firm of Childress Duffy Goldblatt, Ltd. initiated this class-action law suit and has prepared an e-mail response site for pet owners to access if they want to participate in the suit. This lawsuit has been advertised in several pet magazines. The solicitation to pet owners reads:

If, within the last four years, you have paid for any of the following pet vaccinations without receiving adequate disclosure, you may have a claim for damages. The vaccines include the following:

1) Annual vaccination for canine distemper, parvovirus, and feline distemper, rhinotracheitis, calcivirus (Scientific studies indicate that repeat administration of these vaccines provides no beneficial effect.)

2) Corona virus vaccination (Scientific studies indicate dogs over 8 weeks old are not susceptible to this disease.)

3) Leptospirosis or Lyme disease vaccination (Research indicates these diseases are rare to non-existent in Texas and many other parts of the country.)

4) Feline Aids vaccine, Feline Infectious Peritonitis vaccine, or Giardia vaccine (Scientific studies have shown these vaccines to be ineffective.)

If you have paid for any of the above vaccinations in the last four years and would like information concerning your rights, please send an e-mail to:

*Roy R. Brandys or John Sawin
Childress Duffy Goldblatt, Ltd.
petvaccine@childresslaw.net
312-494-0200*

The implications of this lawsuit are obvious. It reminds us to follow sound science in vaccination recommendations.

Baytril banned for use in chickens, manufacturer won't appeal

(From "Deadline for Baytril Looms" by Schuff Sally in Feedstuffs, Aug. 8, 2005)

On September 12, Dr. Lester Crawford issued a decision to withdraw FDA approval for the fluoroquinolone-class antibiotic, Baytril 3.23% Concentrate Antimicrobial Solution, which had been approved for use in the treatment of poultry diseases. Crawford's decision upholds an earlier decision by an FDA administrative law judge that had been appealed. Crawford said that his decision was based on finding resistance to fluoroquinolone antibiotics in isolates of *Campylobacter* spp. He also cited research that reported that resistance of *Campylobacter jejuni* to enrofloxacin (Baytril) and ciprofloxacin increased seven-fold within 24 hours in broiler chickens treated with Baytril according to label directions. The resistance

reportedly persisted throughout the lifespan of the flock. According to a 1999 report by the CDC, campylobacter causes 2.4 million cases of human illness yearly in the U.S. and 80 percent of the cases originate with food. Poultry products are recognized as an important source of campylobacter infection, and campylobacter contamination is much more prevalent in poultry products than in other types of meat. Bayer Animal Health, the manufacturer of Baytril, chose not to appeal.

Editor's note: This announcement does not currently affect Baytril 100 Injectable for cattle. Use of enrofloxacin by parenteral injection has not been shown to produce antimicrobial resistance when used in cattle to treat bovine respiratory disease. But large animal practitioners should remember that the FDA has declared extra-label use of

any fluoroquinolone in any dosage form in food animals to be illegal. Thus, treating calfhood diarrhea with Baytril tablets or injectable is illegal. One justification for this illegal classification is that enrofloxacin has the same potential to produce resistant intestinal microbes when used to treat intestinal infections in other species similar to the resistance reported in poultry.

Small animal practitioners using oral dosage forms of enrofloxacin should realize that the same potential to produce resistance also occurs in the intestinal microflora of pets. Because of proximity of pets to humans, we should always remind pet owners whose animals are being treated with any antimicrobial to use excellent sanitation to help ensure that they do not acquire resistant organisms from their pets.

Pig disease spreads to humans with fatal consequences

Jerome Nietfeld, DVM PhD
Veterinary Diagnostic Laboratory

On July 22, 2005, Chinese officials reported to the World Health Organization 20 cases and 9 deaths from a mysterious disease affecting people in Sichuan Province. By August 3, there were at least 206 cases with 38 deaths and 18 critically ill patients. Symptoms included high fever, malaise, nausea and vomiting, followed by meningitis, subcutaneous hemorrhage, toxic shock, and, in severe cases, coma. Chinese investigators determined that affected people were predominately adult male farmers who had slaughtered, handled or eaten infected pigs. Simultaneous with this outbreak, Chinese animal health officials investigated an outbreak of *Streptococcus suis* type II that was causing illness and death of pigs in the same province. They also found that at least 19 of the affected individuals were infected with *S. suis*, and that none of the infected people had any contact with other infected individuals. Because *S. suis* typically causes sporadic infection and not outbreaks in humans, some individuals (not in China) theorized that a second pathogen might be involved in the outbreak, and that the disease might be spreading person-to-person. However, Chinese health authorities insist that no evidence of infectious agents other than *S. suis* has been found in the initial or

subsequent investigations. They also stress that there is no evidence of human-to-human spread of the disease.

In an effort to control the disease, the Chinese have distributed 2 million notices that warn farmers not to slaughter pigs or eat their meat. They have set up roadside quarantine stations to prevent dead pigs

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from going to slaughter and to make sure that the pig carcasses are incinerated. Large quantities of pork from the affected areas have been quarantined. The Chinese feel that the outbreak is coming under control. Chinese animal health authori-

ties announced that they are distributing a vaccine for *S. suis* to be administered to pigs in the affected area. Ultimately, the plan is to vaccinate 10 million pigs.

Streptococcus suis occurs worldwide and has been isolated from a wide variety of animals, but it is best adapted to pigs, which commonly carry low numbers of the organism in their tonsils. Carrier pigs are asymptomatic, but under certain conditions some serotypes of *S. suis* can multiply and cause septicemia, meningitis, pneumonia, pleuritis, peritonitis and arthritis in pigs.

Human infection with *S. suis* was first recognized in the 1960s. According to the WHO, *S. suis* is a not uncommon cause of bacterial meningitis in people in some parts of southeastern Asia. Human infec-

tion appears to be rare in other parts of the world. Transmission is usually through skin wounds, but infection by ingestion or through mucous membranes has been suspected. The most important risk factor for human infection is contact with pigs or uncooked pork products. Typical clinical signs in humans include fever and signs of meningitis (headache, neck pain, vomiting, light intolerance and decreased consciousness). Approximately 50 percent of affected individuals experience some hearing loss, which is usually permanent. Arthritis and pneumonia are possible. Toxic shock syndrome (TSS), which can be caused by other *Streptococcus* species and by *Staphylococcus* species, occurs in some individuals. TSS can lead to severe damage to many vital organs, including the liver, kidneys, and the circulatory system. TSS is only partially responsive to antibiotic treatment and it carries a graver prognosis than other forms of the disease.

Because healthy pigs carry very low numbers of *S. suis* in their tonsils, they represent very little, if any, threat to humans. However, sick pigs with bacteremia, pneumonia and/or polyserositis have very large numbers of *S. suis* in their tissues and are a potential source of human infection. To prevent infection, avoid contact with sick or dead pigs and their excreta and body fluids. If it is necessary to handle pigs or raw pork, people should wear protective gloves, wash their hands after handling pigs or raw pork, and clean and cover all wounds.

Activists upset by farrowing exhibit

(From "Pork Alert" published by *Pork Magazine*, Aug. 23, 2005, volume 6, issue 34)

Animal rights activists are targeting a two-sow farrowing exhibit at Blank Park Zoo in Des Moines, Iowa. A coalition of groups, including the Sierra Club, says the exhibit represents factory-farm-style pork production. Proponents say the exhibit, including farrowing crates, shows how producers give sows and their piglets individual care.

The zoo's chief executive officer, Terry Rich, listened to the groups' complaints, but says he won't change the exhibit before it closes next month. Rich says the zoo looked at the facts of modern pork production and talked with Iowa State University and Iowa Department of Agriculture personnel.

The exhibit is the first phase of AgZoo, a program aimed at bridging the knowledge gap about agriculture for Iowa's growing urban population.

Featured foreign animal disease

African horse sickness (*Pestis equorum*; Equine plague)

Alexandra Eckhoff, DVM, PhD
College of Veterinary Medicine

Definition

African horse sickness (AHS) is an acute or subacute, arthropod-borne, noncontagious viral disease of the family Equidae that is endemic to Africa. The clinical signs and pathologic lesions are caused by an increase in vascular permeability and are characterized by respiratory and cardiovascular disease. This disease is one of the most lethal horse diseases and carries with it potential economic losses and disruption of international activities. Currently, there is a two-year embargo of equines from South Africa imposed by the European Union because of an outbreak of African horse sickness last year.

Etiology

African horse sickness is caused by a double-stranded, 55 to 70 nm, RNA virus of the genus *Orbivirus* in the family *Reoviridae*. The virus is stable at pH 6.0 to 12.0, and it is relatively heat stable, especially if in the presence of protein. In blood in OCG preservative (Edington medium) AHS virus remains infective for at least twenty years when refrigerated and for months at ambient temperature (20 to 25 °C). AHS virus is inactivated by many disinfectants.

Nine distinct serotypes of AHS virus have been isolated, with the last isolate in 1960. This indicates that the virus is genetically stable and that the nine serotypes probably developed over many centuries.

Host range

The AHS virus infects members of the family *Equidae*, with mortality of 70 to 90 percent in horses, 50 percent in mules, 5 to 10 percent in donkeys, and 0 percent in zebras. Because of the high mortality rates, horses and mules are considered accidental hosts. Horses, mules and donkeys probably do not have an important role in maintenance of the virus in nature because the disease has not become established outside

of Africa (they die too acutely to transmit the disease to others). The role zebras in the persistence and spread of AHS virus is unknown.

Dogs can become infected experimentally or following ingestion of infected horse flesh, but they do not become infected by insect bites, and they play no role in the spread or persistence of AHS.

There is no evidence that humans can become naturally infected with AHS virus. However, certain neurotropic vaccine strains can cause encephalitis and retinitis in humans following transnasal infection.

Transmission

African horse sickness is primarily transmitted by *Culicoides spp.* (midges) that are most active at sunrise and just after sunset. The principal biological vector is *Culicoides imicola*, but other species, such as *C. variipennis* (the vector for bluetongue virus in the United States), should be considered as possible biological vectors. Large biting flies, like *Stomoxys spp.* and *Tabanus spp.*, ticks, and mosquitoes are possible, but uncommon, vectors. In temperate regions, AHS has a seasonal occurrence. The first cases occur towards midsummer, and the disease disappears in the autumn after a few frosts. The disease is most prevalent in warm, moist, low-lying valleys or marshes. The onset of the disease is preceded by seasons of heavy rain following periods of hot dry conditions.

Geographic distribution

African horse sickness appears to be endemic in central and southern Africa. The Sahara Desert is a natural barrier against spread of the disease to northern Africa. Occasionally the infection does spread along the Nile valley or along the West Coast of Africa and reaches northern Africa. The disease has also occurred outside Africa on a few occasions, the most notable being outbreaks in the Middle East and Spain.

Incubation period

The duration of the disease and the length of the incubation period might be linked to the virulence of the strain, because the highly fatal forms have the shortest incubation periods. Incubation can last from two to 21 days in experimental infection and as long as 16 days in the natural infection, with a thermic rise marking the end of the incubation period. Viremia generally lasts about four to eight days and typically is accompanied by intermittent, progressively increasing fever, with temperatures rising in the evening and dropping in the morning. The fever peaks after three to four days, when the clinical stage begins. High concentrations of virus are present in the blood, spleen, lung and lymph nodes, and smaller amounts are found in excretions and secretions. Viremia can last as long as 17 days in horses and 28 days in zebras and donkeys.

Clinical signs

Four clinical forms of AHS have been described.

Fever form (Horse sickness fever)

This is the mildest form (can be subclinical) and it can be overlooked in natural outbreaks. The incubation period is four to 14 days. Intermittent fever up to 104° F (40° C) in the afternoons followed by morning remission for three to eight days is common. Additional signs can include congested mucous membranes, anorexia and depression. The temperature spikes gradually decrease and the animal recovers in 10 to 11 days. This form of the disease may be observed in donkeys and zebras or in immune horses infected by a heterologous serotype of AHS virus.

Acute respiratory form (peracute form; pulmonary form)

This form of the disease is characterized by a two- to five-day incubation period and a dramatic and rapidly progressive respiratory compromise. An acute fever of about 104 to 107°F (40 to 42°C) occasionally can be the only clinical sign for a couple of days. This is followed by dyspnea, spasmodic coughing, congested conjunctiva and swollen supraorbital fos-

African horse sickness is primarily transmitted by *Culicoides spp.* (midges) that are most active at sunrise and just after sunset.

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sae. The animal tends to stand with its forelegs spread apart, its head extended and nostrils fully dilated. The respiratory rate becomes drastically elevated (60 to 75 bpm) with a marked abdominal component (heave lines). Profuse sweating is common and there can be frothy, serofibrous fluid exuding from the nostrils (Figure 1). Recovery from this form is rare. Death is caused by anoxia, which occurs within 30 minutes to a few hours after the animal becomes dyspneic.

Cardiac form (subacute form; edematous form)

This form of the disease has an incubation period of five days to two weeks. The onset of clinical signs is marked by a fever of 102 to 106°F (39 to 41°C) that progressively rises for three to five days, peaks at 10 to 12 days, and then decreases. Once the temperature drops, characteristic firm and non-painful edematous swellings appear. The edema usually involves the supraorbital fossae, the eyelids, lips, cheeks, tongue, intermandibular space and laryngeal region and can extend down the neck and chest, but does not affect the lower limbs. The earlier the appearance of edema, the more severe the disease. Terminally, petechial hemorrhages develop in the conjunctivae and under the ventral surface of the tongue. Depression is present and, in some cases, colic and recumbency are observed. With this form of AHS, coughing and foaming are not present. Some animals die from cardiac failure about four to eight days after the onset of the febrile reaction. In animals that survive, the edema, beginning with the temporal fossa, subsides over three to eight days.

Acute or mixed form

In this form, the pulmonary and cardiac symptoms appear either simultaneously or successively. Typically the animal dies of the cardiac form or of an exacerbation of the respiratory form. This form of AHS is seldom diagnosed clinically, yet it is commonly seen at necropsy in the cases of AHS in horses and mules. The manifestations of the disease include mild non-progressive pulmonary signs followed by edematous swellings and effusions, and death results from cardiac failure. In most cases, the cardiac form is acutely followed by the pulmonary form.

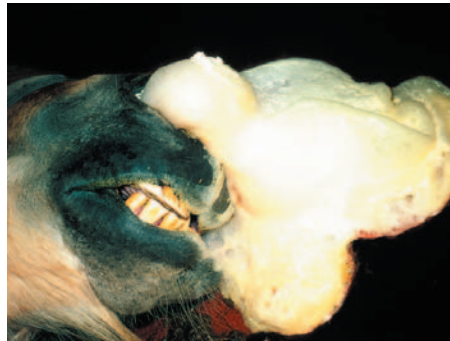


Figure 1. Froth exuding from the nose of a horse that died of African horse sickness.

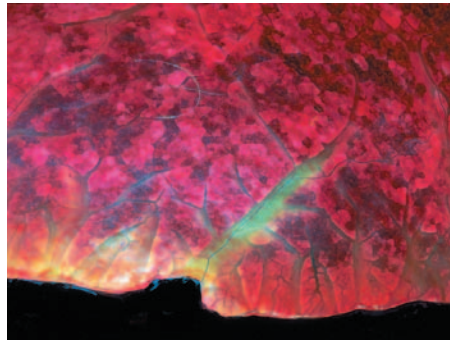


Figure 3. Severe interlobular and intersititial edema and multifocal hemorrhages in the lung of a horse that died of African horse sickness.

Pathologic lesions

Necropsy findings depend on the clinical form of disease responsible for death. For the acute respiratory form, lesions can include frothy exudate from nostrils and in bronchi and bronchioles, and extensive pulmonary edema, especially in the interlobular space (Figures 1 to 3). In some cases, the lungs can appear normal, but the thoracic cavity contains up to 8 L of fluid (Figure 4). Thoracic lymph nodes are edematous and petechiae are present in the pericardium, and there is pericardial effusion. Other less common lesions include periaortic and peritracheal edema, and congested abdominal viscera (gastric fundus, intestines, spleen, and renal cortex). Cardiac lesions are usually not present.

In the cardiac form, lesions include petechiae or ecchymoses on the epicardium and endocardium, hydropericardium, and myocarditis. The most prominent change is the presence of yellow gelatinous material in subcutaneous or intramuscular tissues along the jugular veins, ligamentum nuchae, and, in severe cases, the chest, ventral abdomen, and gluteal region (Figure 5). The lungs are usually normal

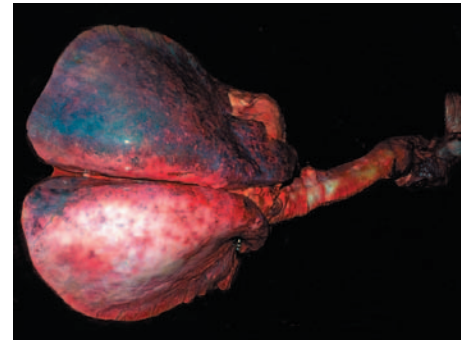


Figure 2. Pleural hemorrhages, congestion and edema in lungs from a horse that died of African horse sickness.



Figure 4. Fluid in the pleural cavity of a horse that died of African horse sickness.



Figure 5. Yellow gelatinous material between in the intermuscular fascia of the neck of a horse that died of African horse sickness.

or mildly distended, and the changes to the abdominal viscera are similar to those with the pulmonary form, but they can be more pronounced.

In the mixed form, the lesions are a combination of the pulmonary and cardiac forms.

Diagnosis

In endemic areas, the clinical signs yield a presumptive diagnosis. Lab confirmation is based on virus identification and determination of serotype. Blood samples obtained at fever peak should be

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preserved in OCG solution (50 percent glycerol, 0.5 percent potassium oxalate, 0.5 percent phenol, q.s. with distilled water) and transported refrigerated to the lab. At necropsy, samples of organs with the highest viral titers (spleen, lymph nodes, and lungs) should be preserved in 10 percent buffered glycerin and transported refrigerated to the lab.

Virus isolation can be performed in infant mice or cell cultures. Specimens for virus isolation should be refrigerated, not frozen. Polymerase chain reaction assays are available and faster than virus isolation. Complement fixation tests are useful in establishing a diagnosis, and virus neutralization tests and hemagglutination inhibition tests are used to serotype the virus.

Horses that survive develop specific antibodies two weeks after infection. The antibodies peak about 10 days later and persist one to four years. As with other viral diseases, acute and convalescent phase serum samples are considered diagnostic.

Differential diagnosis

Various diseases have been cited as having similar clinical signs to one of the four forms of AHS. However, most of these differentials may typically be ruled out by necropsy. Among the list of differential diagnoses are anthrax (ruled out based on location and necropsy findings), equine influenza (limited to the respiratory system and ruled out based on its low lethality), infectious anemia (possible confusion during the hyperthermic phase, ruled out based on necropsy findings in horses that succumb acutely), heat stroke (ruled out based on necropsy), acute intoxication (ruled out based on necropsy), equine viral arteritis (ruled out based on the different location of the edema in the two diseases and the lower mortality rate of EVA), trypanosomiasis found in some countries of Europe and America (ruled out by analyzing blood samples microscopically), and purpura hemorrhagica (the hemorrhage and edema are more severe and extensive with purpura than with AHS and typically involve the limbs and lower abdomen).

Purpura usually occurs sporadically and AHS occurs as epizootics.

Preventive measures

The most common means of introduction of AHS into a disease-free country is by importation of infected animals. Because zebras and African donkeys do not manifest clinical signs, they are particularly high-risk carriers. In the United States, Equidae from African countries

are quarantined for 60 days in an insect-proof facility at the point of entry and tested for virus. The presence of antibodies does not interfere with importation of animals into

disease-free countries.

Recommendations for outbreaks of AHS are made by the International Office of Epizootics (OIE) and vary internationally and nationally. On an international level, AHS should be reported immediately to the OIE and all neighboring countries and the OIE should be made aware of its clinical manifestations. Direct contact is established between veterinary agencies in the affected and neighboring countries and animal transportation is restricted. An effective disinfection protocol must be put into practice starting in the affected area. Quarantine and import measures should be reinforced and activities that require grouping or assembly of animals should be prohibited. When the disease appears, affected equids are vaccinated with polyvalent vaccines and rested for two weeks. Once the serotype is available, animals should be vaccinated with the homologous serotype. Vaccinated horses should be kept in stables during the peak vector feeding times and vector control should be initiated via insect repellents and insecticides. Additionally, body temperatures should be monitored twice daily to detect viremic animals. All affected animals should be isolated or euthanized. Research of the causal agent, vectors and reservoirs is initiated but control measures should be implemented even before the final diagnosis has been made.

Immunization

Survivors develop a life-long immunity to homologous serotypes but remain sus-

ceptible to heterologous types. Foals born to immune dams have a passive immunity for six months.

Currently, there are vaccines for all nine serotypes of AHS. These vaccines are cell culture derived or mouse-brain attenuated and they provide long-lasting immunity. Inactivated vaccines also exist, but require two doses to provide adequate protection. These vaccines induce a local reaction at the site of inoculation and provide short-term protection.

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Newsletter Coordinators

Larry C. Hollis, Extension Beef Veterinarian
785-532-1246 • lhollis@oznet.ksu.edu

Jerome C. Nietfeld
785-532-4460 • niefeld@vet.ksu.edu

Contributors — K-State Research and Extension

Dale Blasi	Ron Hale	Twig Marston
Scott Beyer	Mike Brouk	Sandy Johnson
Joel DeRouchey	Mike Tokach	John Smith
Jim Nelssen	Bob Goodband	Cliff Spaeth

Contributors — Veterinary Diagnostic Laboratory

G.A. Andrews	R. Ganta	R. Pannbacker
M.M. Chengappa	S. Kapil	J.A. Pickrell
B. DeBey	K.S. Keeton	S.S. Dritz
D.A. Mosier	M.F. Spire	M.W. Dryden
T.G. Nagaraja	S. Stockham	B.W. Fenwick
M.J. Wilkerson	F.W. Oehme	

K-State Research and Extension
137 Call Hall
Manhattan, KS 66506

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