



EQ GUINE DISEASE QUARTERLY

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COMMENTARY

"Epidemiology is the study of the distribution and determinants of health-related states or events in specific populations and the application of this study to control health problems."

- J.M. Last: A Dictionary of Epidemiology, ed 2. New York, Oxford University Press, 1988.

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WHILE A DEFINITION IS A GOOD PLACE to start when thinking about the role of epidemiology in equine health, for some people it is a bit formal and vague. Perhaps a more useful approach would be to consider the current applications of epidemiology to animal health and the benefits to be gained through epidemiological studies. To paraphrase a recent television advertising campaign, what can epi do for you?

While public health practice has currently placed an emphasis on determining risk factors and interventions for chronic conditions such as cardiovascular disease, diabetes, and cancer, the epidemiological focus in the veterinary community is directed at infectious/communicable diseases. There are exceptions, such as examining the risks for injury in performance horses or tracing of animal feed contaminated with melamine, but for the most part, infectious agents such as bacteria, viruses, and prions rule the day. This focus is a reflection of the population-based approach that veterinary medicine uses in dealing with infectious disease conditions and the recognition of the financial burden to both producer and consumer when infectious agents gain access to commercial agricultural systems.

Epidemiologic methods are used to address issues of equine health. The investigation of encephalitis attributed to West Nile Virus in horses residing on Long Island, New York, in 1999 identified risk factors for exposure: stabling near water sources and areas of dense foliage. Studies also quantified the morbidity and mortality among horses residing in areas

known to harbor infected mosquitoes (a clinical attack rate of 32% [25/79] with a corresponding case fatality rate of 36% [9/25] in exposed, unvaccinated horses), and predicted the geographic and temporal spread to other states—to the West Coast over a four-to-five year period. These epidemiologic studies provided critical information for equine and public health for this foreign animal disease, which is now endemic to North America.

More recently, the diagnosis and epidemiological follow-up to the occurrence of equine arteritis virus in Quarter Horses in the western United States demonstrated the ease with which a venereal disease can be dispersed across a large geographic area. The use of shipped, cooled semen demonstrated the need for industry-based interventions, such as the establishment of a code of practice for managing the health of equines used in commercial breeding.

For most purposes, simply tracing equines in commerce in order to evaluate their disease status (for example, with a certificate of veterinary inspection) or the status of other commingled animals (through testing for equine infectious anemia) may be all that is desired. For others, quantifying the risk for exposure to specific diseases, either emerging or domestic, is paramount. The important concept to remember is that the utility of any epidemiologically derived information will reside with the end user. What can epi do for you?

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INTERNATIONAL

Fourth Quarter 2008

THE INTERNATIONAL COLLATING CENTRE, Newmarket, England, and other sources reported the following disease outbreaks.

Two cases of Contagious Equine Metritis (CEM) were confirmed in non-Thoroughbred horses in France. As of February 23 the USDA reported 11 non-Thoroughbred stallions positive for (CEM) during the recent outbreak—four in Kentucky, three in Indiana, three in Wisconsin, and one in Texas. Three non-Thoroughbred mares have been identified as CEM positive in Wisconsin, Illinois, and California following natural breeding or via semen from stallions recently identified as CEM positive.

During 2008 the USDA reported 168 cases of Eastern Equine Encephalitis throughout the USA, with 86 cases in Florida, 22 in Georgia, and 21 in Alabama.

Respiratory disease attributable to equine herpesvirus (EHV) was diagnosed in a horse in France. Neurological disease attributable to EHV-1 was diagnosed in a horse in France and three polo ponies in the United Kingdom. Clinical signs exhibited by the polo ponies were not typical of EHV despite evidence of seroconversion and isolation of EHV-1 from the nasopharynx of one animal. Sporadic cases of EHV-associated neurological disease were reported from several parts of the USA. A case in a 7-year-old pony in Kentucky during November was caused by the non-neuropathogenic strain. During December two horses were euthanized on separate farms in Delaware resulting from the non-neuropathogenic strain, and a 2-year-old Thoroughbred filly at Laurel Park racetrack, Maryland, was euthanized following EHV-1 infection. At the end of December a Thoroughbred horse at Fair Grounds racetrack, Louisiana, developed signs of paralysis caused by EHV-1. Japan and the United Kingdom each reported a single case of EHV-1 abortion, and three cases were reported in central Kentucky.

A single case of Equine Infectious Anemia (EIA) was confirmed in South Germany. Switzerland reported the results of a serological survey undertaken during 2007 and 2008

among 666 domestic and imported horses, all of which tested negative for EIA.

Equine arteritis virus infection with clinical signs was diagnosed among non-Thoroughbred horses on three premises in Denmark.

Twelve months following the last confirmed case of equine influenza in Australia on December 27, 2007, the World Organization for Animal Health (OIE) formally acknowledged the country's status as free of equine influenza. Sweden reported that performance and breeding Thoroughbreds as well as non-Thoroughbreds were diagnosed with equine influenza on approximately 50 premises. Six trotting tracks were temporarily placed in isolation, but as of January 8, only one track remained isolated.

Two cases of equine piroplasmiasis affecting non-Thoroughbred horses on two premises were reported from Switzerland.

Salmonella abortus equi infection affecting three non-Thoroughbred horses was diagnosed on one premise in Japan. Cases of strangles were confirmed from France, Ireland, Switzerland, and the United Kingdom.

West Nile Virus (WNV) infection was reported affecting 474 horses on 235 premises in central-east Italy during the last six months of 2008. Performance horses, breeding animals, and non-Thoroughbreds were involved. The USDA reported 162 cases of WNV infection among horses in the USA during 2008, with 32 in California and 41 in the state of Washington. This compares with 1,370 human cases with 37 fatalities reported by the Centers for Disease Control throughout the USA over the same period, with 411 cases in California and 99 in Mississippi.

South Korea reported the results of a serological survey conducted among 1,085 Thoroughbred horses including stallions, broodmares, young horses, and racehorses during 2008. All samples were negative for African Horse Sickness, EIA, Vesicular Stomatitis, and WNV. Ninety-five percent of samples were positive for Japanese Encephalitis as a result of vaccination, infection, or both.



Equine Disease Quarterly

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NATIONAL

Early Embryonic Loss and Genetics

CHROMOSOMES ARE LARGE SEGMENTS OF DNA wound around special proteins within each cell's nucleus. They contain nearly all of the body's genetic material. Horses have 64 chromosomes (or 32 pairs) including the two sex chromosomes (XY in males and XX in females). The sire and dam each contribute 32 chromosomes to their offspring. Modern cytogenetic methods enable the identification of all 32 pairs of chromosomes based on their unique features. The features of each chromosome pair are compared in a chart, or karyotype, to identify abnormalities. All chromosomal abnormalities affecting horses involve the sex chromosomes.

At cell division, chromosomes replicate and segregate into daughter cells. Sometimes during egg or sperm formation, chromosome replication and segregation can go awry, resulting in an abnormal chromosome complement being transmitted to the offspring. If the chromosomal abnormality is severe, it can result in early embryonic loss.

One type of chromosomal abnormality that causes early embryonic loss is a translocation (Figure 1). A translocation occurs when there is an interchange or transfer of chromosomal segments between two or more different chromosomes. Translocation carriers can be balanced or unbalanced. In a balanced translocation, all the necessary genetic material is present, and the individual appears normal. In an unbalanced translocation, extra genetic material may be present or genetic material may be missing, and the individual is abnormal.

Chromosomal translocations in horses have been rarely documented and all have caused repeated early embryonic loss (REEL). REEL can be quite costly due to additional costs for veterinary care, boarding, and transportation. Oftentimes an entire breeding season is lost. Until recently, only two equine chromosomal translocations had been described in literature. The first case was a mare that produced only

two foals in seven years. The second was a stallion with a high incidence of early embryonic loss in the mares to which he was bred.

Recently, we identified different translocations in four mares experiencing REEL. While the reproductive history for each mare is somewhat different, all four mares lost embryos multiple times over multiple years and always prior to day 65 of gestation. All four mares had normal reproductive tracts and estrous cycles. Once in foal the mares had normal ultrasound exams between days 14 and 21. However, in some mares the embryo's heartbeat was lost by day 28.

One mare always lost the embryo between days 45 and 65. Hormonal treatments were ineffective. Combined, all four of the mares produced only six foals in 30 breeding seasons. One mare produced two foals over the last 10 years. The second mare produced three foals over eight years. Blood samples from two of her foals were submitted for karyotyping. The results showed that one foal had a normal karyotype, and the other foal carried the same translocation as the dam. The third mare had been bred for six years but never produced a foal. The fourth mare produced only one foal in six years.

The results of this study suggest that chromosomal translocations causing REEL may be present in horses at a higher frequency than previously known. Early embryonic loss can be caused by numerous other factors besides a chromosomal translocation. However, for a mare that repeatedly loses embryos prior to day 65 of gestation for two or more years in a row, it may be advisable for veterinarians and breeders to send samples for karyotyping from the mare in order to rule out a chromosomal translocation.

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Why Take Nasal Swabs?

INFLUENZA IS ONE OF THE MOST CONTAGIOUS diseases of horses and therefore requires a timely and accurate diagnosis to help prevent a major outbreak. This diagnosis can be achieved only by testing clinical specimens from nasal swabs.

Rapid diagnosis facilitates rapid intervention, including quarantine and vaccination—historically the most successful means to prevent the spread of influenza. Vaccination in the face of an influenza outbreak can work if the horse has already been primed by previous vaccination and if at least a seven-day interval between vaccination and exposure to the disease has occurred.

Influenza is a moving target, often changing its viral coat to confuse the immune system. The vaccines have to be periodically updated to keep up with the changing viruses. The only way to keep the vaccines in step with the circulating viruses is to obtain samples of those viruses for testing. The mechanism for updating the vaccines depends upon voluntary submission of nasal swabs from veterinarians to their state diagnostic laboratories or directly to the

OIE (World Organization for Animal Health) international reference laboratories for equine influenza in England, Germany, and in Kentucky at the Gluck Equine Research Center.

Same-day test results are now possible for equine influenza using commercial rapid detection kits. The limiting factors are (1) expense: these tests cost \$25-50 per swab and (2) the quality of the swab sample. Samples should be taken early in the disease process. Minimally use 6-inch Dacron swabs in pairs or alternatively uterine swabs (no 4-inch cotton swabs!).

See our Web site at <http://www.ca.uky.edu/gluck/ServFlu.asp> for further information about nasal swabbing and submission to the OIE reference laboratory at the Gluck Center.

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Potential Threat of African Horse Sickness to the United States

AFRICAN HORSE SICKNESS VIRUS (AHSV) and bluetongue virus (BTV) are both members of the genus *Orbivirus* of the family Reoviridae. Both cause serious, non-contagious but infectious, arthropod-borne diseases in equids and ruminants, respectively. AHSV infects all equids, causing asymptomatic infection in zebra and African donkeys, but it is the most lethal infectious disease of horses known, with mortality as high as 95%. BTV is thought to infect all known species of ruminants; however, severe disease usually occurs only in certain breeds of sheep and some species of deer. Zebra are thought to be the reservoir host of AHSV in equines and of BTV in bovines.

The distribution of both diseases reflects the

presence of their infected arthropod vectors, which are certain species of *Culicoides* biting midges, the temperature required for viral replication in these vectors, and transmission by these vectors. BTV unexpectedly entered Northern Europe in August of 2006, creating a rapidly spreading bluetongue epizootic in the Netherlands, Belgium, Germany, France, and Luxembourg, with over 2,000 cases. The virus overwintered by an unknown mechanism, although the 2006-2007 winter was the second mildest winter in Northern Europe on record. The epizootic continued into 2007, resulting in 45,000 cases. Because of the recent dramatic change in epidemiologic status of BTV and its *Culicoides* sp. vectors in Europe, both the

equine and ruminant industries have become concerned about the potential for entry for these diseases into the United States.

The feasible routes for entry of AHSV into the United States include importation of infected animals and introduction of infected vectors.

Federal regulations exist for the legal importation of domestic and wild equidae from countries that the USDA-APHIS considers to be affected with AHS. Further regulations are available on the USDA Web site about the minimum 60-day quarantine for all equines originating in AHS-affected countries.

(Go to http://www.aphis.usda.gov/import_export/animals/animal_import/equine/equine_import60day.shtml).

These animals can only be imported through the New York Animal Import Center. At this facility, only 16 horses and no zebra entered from AHS-affected countries during the last three years. Zebras are not generally imported because of the expense and presence of successful breeding programs in the United States.

The introduction of infected vectors is dependent upon several weather factors. Dispersion of *Culicoides spp.* over distances up to 400 miles (700 km) over water and 90 miles (150 km) over land has been postulated. However, the shortest distance from Africa to the United States is 3,000 miles (4,830 km). To cover such long distances, transport would need to be at high altitude (3.5 miles; 6,000 m), at which air temperature is far below 32° F (0° C), and *Culicoides spp.* would not survive.

Temperatures at 80-86° F (27-30° C) are optimal for AHSV transmission in the laboratory, while temperatures below 59° F (15° C) inhibit virus replication within the midge. As temperatures increase, midge infection rates increase and virus replication quickens, but midge survival rates decrease. At cooler temperatures, AHSV within the *Culicoides spp.* vector becomes "latent," but replication begins rapidly as temperatures warm. Midges are most active around dusk and night with light wind speeds in areas of minimal to no precipitation and a relative humidity (RH) of 75-85%. The midge can become desiccated at low RH and oversaturated at high levels.

There are no references available describing *Culicoides spp.* in cargo, including imported flowers or plants. There are almost no data recording the presence of *Culicoides spp.* on aircraft.

Outbreak Scenario in the United States

The United States has multiple components that would support at least a focal outbreak of AHS: the presence of susceptible horses, areas with suitable weather conditions that would encourage viability of an introduced vector, and the presence of a capable vector. A highly competent experimental laboratory vector for AHSV is *Culicoides sonorensis*. This vector has a wide U.S. distribution (absent only from the northeastern states) and is the biological vector for BTV. If a foreign midge vector were to successfully invade the *C. sonorensis* eco-niche and begin an AHSV epizootic, *C. sonorensis* would soon become infected and the likely primary vector.

The only component missing for establishment of enzootic areas in the United States is a sufficiently large zebra population or another yet unknown reservoir host and the virus.

AHS is currently on the list of diseases for which a response plan is soon to be written by USDA-APHIS VS National Center for Animal Health Emergency Management. This plan will replace the outdated AHS Red Book previously published by the USDA.

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KENTUCKY

Anatomy of a Disaster

A WINTER WEATHER WARNING FOR KENTUCKY had been issued, and all the horses were in the barn for the night as a precaution. After all, how often have the weather forecasters been overenthusiastic about “catastrophic” storms?

Overnight the electricity went out; both landline and cell phones were dead. Trees, limbs and telephone poles were down, blocking roads and causing major damage to buildings, vehicles, and equipment.

Throughout the state 760,000 electrical customers were without power; some counties had water supply issues due to the power outage and broken water lines. People could not communicate with first responders; emergency rescue teams were unable to respond due to blocked roads. People learned that satellite telephones or ham radios (both battery-powered) were the only means of communication.

The average temperature was 20° F, and ½ to 1 inch of ice glazed everything in sight, including the underlying snow. On several nights, the wind chill was below zero.

This was the situation in late January, with the western half of the state hardest hit. Fence lines were down because of fallen trees or power poles. Farms dependent on electrical pumps for well water had no water for livestock. Farmers with generators could get their water flowing again, but they didn't know for

how long. First responders' priorities were primarily human health and safety, with animal health and safety and preservation of property running a distant second and third. Resources went to highly populated areas first.

Of the 120 Kentucky counties, 95 declared states of disaster, resulting in state and federal disaster declarations and qualifying the state for federal aid. More than 30 people in Kentucky died due to the storm; no firm data are yet available about the number of livestock and equine deaths and losses.

After one week, 300,000 electrical customers still had no power despite a tremendous response of electrical workers coming in from more than 20 surrounding states. In some areas, people were advised it would be three to four weeks before power could be restored.

A disaster can happen at any time and can last hours, days, or weeks.

Failing to plan is planning to fail.

(For information on a disaster plan for your family, go to <http://www.redcross.org>. For your horses, go to http://www.aaep.org/emergency_prep.htm).

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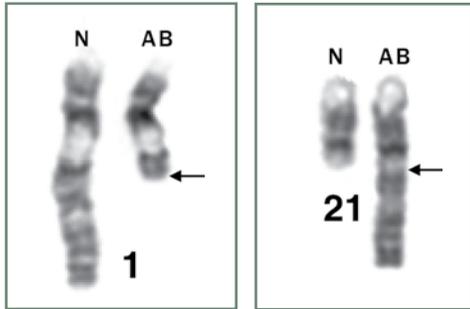


FIGURE 1.
A chromosome translocation involving horse chromosomes 1 and 21 that causes repeated early embryonic loss.

The normal chromosomes are on the left (N), and the abnormal chromosomes are on the right (AB) of each pair (boxes at top). Chromosome 1 has broken (arrow) and reattached to the bottom of chromosome 21 (arrow). If the normal chromosomes segregate together to a sperm or egg, the foal will be normal. If the abnormal chromosome 1 segregates with a normal chromosome 21, genetic material will be lost; if the abnormal chromosome 21 segregates with a normal chromosome 1, genetic material will be gained. Both cases cause death of the embryo. If both abnormal chromosomes segregate together, the embryo will have a complete genetic complement but be a carrier of the same translocation.

