



EQUINE DISEASE QUARTERLY

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COMMENTARY

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As equine practitioners, we are charged with the health and welfare of our client's horses, including dealing with infectious and non-infectious causes of neurologic disease. The veterinary industry has rapidly changed in complexity, with a new understanding of disease mechanisms, diagnostic testing, and treatments. Many challenges remain including how to make an accurate diagnosis, how to establish neuroanatomic localization, and how most effectively to treat affected horses. If a horse is suspected to have cervical vertebral stenotic myelopathy (wobbler syndrome), then cervical radiographs are called for, often followed by a myelogram, CT or MRI scan to help pinpoint the site of compression. Horses with multifocal, asymmetric signs affecting brain, brainstem and cranial nerves along with weakness and ataxia are likely to have equine protozoal myeloencephalitis (EPM). Accurate diagnosis of this condition requires an evaluation of antibodies in blood and CSF. Although EPM is most commonly caused by *Sarcocystis neurona*, clinical signs in some horses are a result of *Neospora hughesi* infection. The exposure rate for *N. hughesi* appears much lower than for *S. neurona*. Risk factors for developing EPM include age (< 5 and > 13 years), time of year (summer and spring), previous cases on a farm, and presence of woodland and opossums on the farm. EPM prevalence is less on farms where wildlife have little or no access to feed and if a creek or river is on the premises. Stressful events like concurrent disease, general anesthesia, or travel can increase the incidence of EPM. There are three FDA approved drugs for the treatment of horses suspected to have EPM.

Another important infectious cause of neurologic disease is equine herpesvirus 1 (EHV-1), which is addressed in this issue of the Equine Disease Quarterly by Dr. Peter Timoney. EHV-1 is problematic in that it can affect several body systems (respiratory, reproductive, and central

nervous system). The neurologic form of EHV-1 infection is termed equine herpesvirus myeloencephalopathy (EHM) and is caused by damage to blood vessels supplying the central nervous system. These vascular insults result in stroke-like injuries. Clinical signs often include ascending paralysis with urine dribbling, loss of tail and anal tone and pelvic limb weakness.

A diagnosis of EHM is made easier when several horses on the same premise present with fever, followed by ataxia, urine dribbling and in some cases abortion. Furthermore, diagnosis can be established by recognition of clinical signs in combination with a positive virus detection test (PCR or virus isolation) on a nasal swab and unclotted blood sample. Following confirmation of a diagnosis of EHM, affected animals or test positive animals should be isolated, and the premises quarantined; movement of horses off an affected facility increases the risk of spread of infection.

Herpesvirus can affect horses of all ages but is rarely seen in yearlings. The virus initially replicates in the mucosa of the upper respiratory tract, spreads to the lymphoreticular system, followed by development of a viremia and infection of other sites in the body. Although currently available vaccines do not reliably prevent infection, the development of viremia, or the establishment of latency, vaccination remains important in preventing and/or reducing the incidence of respiratory infections and abortions and is considered the best recommendation for owners to keep their horses healthy. Other infectious neurologic diseases that need to be considered include West Nile virus infection, rabies, and tetanus, all of which should be a part of the annual vaccination schedule for horses.

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First Quarter 2021

The International Thoroughbred Breeders Federation, International Collating Centre, Newmarket, United Kingdom, and other sources reported the following equine disease events.

African horse sickness was reported as endemic in the Republic of South Africa (RSA), except in the controlled area in the Western Cape Province. Sporadic cases (38 total) were recorded in six of the nine provinces.

France, Germany, the Netherlands, the UK, and the USA confirmed outbreaks of equine influenza. The number ranged from eight (France), five (Germany and the UK), and three in the Netherlands to at least 13, in as many states, in the USA, where the disease is endemic. Washington State reported a case of co-infection with equine herpesvirus 1. Many outbreaks involved only single cases.

Strangles is considered endemic in many countries with outbreaks recorded by Belgium (one), Canada (two), France (13), the Netherlands (19), Switzerland (five), and the USA (27). The majority of outbreaks were comprised of a single case.

Equine herpesvirus 1 (EHV-1) respiratory disease is endemic in most countries. Outbreaks were diagnosed in Belgium (eight), France (20), Germany and Sweden (12 apiece), Qatar and Switzerland (one each), Spain (two), the UK (five), and the USA (three). Belgium confirmed a case of co-infection with equine herpesvirus 4 and 5.

Outbreaks of EHV-1 abortion were reported by Belgium and Sweden (six), Canada (two), France and the Netherlands (three), Germany (11), Ireland (seven), Japan (eight), the UK (eight), and the USA (ten). With very few exceptions, all were represented by single cases of abortion. Outbreaks of EHV-1 neurologic disease were diagnosed by Belgium (eight, one with four cases, another with eight and a third with 24), Canada (seven outbreaks), France (five, one with four cases and another with five cases), Germany (two), and Italy (four).

Equine herpesvirus 4 (EHV-4) respiratory outbreaks were reported by Belgium (six, one extensive), France (53, one extensive), the Netherlands and the USA (two apiece), Belgium (five), France (two reported outbreaks of EHV-4 abortion, all single cases), and Switzerland (one case). Germany

and the USA recorded cases of equine herpesvirus 2 and/or 5, some associated with clinical respiratory disease.

Equine infectious anemia was confirmed in Canada and Romania (two outbreaks in each country, each comprising single cases), and the USA (five outbreaks, all involving single cases).

The RSA regards equine piroplasmiasis as endemic with a variable number of cases in eight of the nine provinces, the majority were in Gauteng Province.

Single cases of contagious equine metritis were confirmed on individual premises in Germany. Coital exanthema (equine herpesvirus 3) was diagnosed on two premises (single cases) in France. Four cases of leptospiral abortion were recorded in Kentucky, USA, as were eight cases of nocardiform placentitis/abortion. Japan reported a case of *Salmonella Abortusequi* in a non-Thoroughbred, and Belgium diagnosed three cases of *Streptococcus zooepidemicus* abortion on individual premises.

The USA reported salmonellosis (two cases involving serogroup B) and clostridial enterocolitis (eight cases of *Clostridium perfringens* Type A, beta-2 toxin; 99 cases of *C. perfringens*, toxin untyped; 63 cases of *Clostridioides difficile*), many in Kentucky. Of four outbreaks of equine coronavirus confirmed by Switzerland, three were single cases. Numerous outbreaks of neonatal diarrhea were reported by Kentucky, some infected by a novel rotavirus. Kentucky also recorded two cases of *Lawsonia* enteropathy and four cases of Tyzzer's disease (*Clostridium piliforme*). Germany and the RSA confirmed one and two cases of West Nile virus infection, respectively.

A single case of Eastern equine encephalomyelitis was recorded in an unvaccinated horse in Florida, USA. The USA also confirmed a case of rabies in an unvaccinated mule in California.

The RSA reported that equine encephalosis was endemic in the country, with a variable number of cases diagnosed in eight of the nine provinces, mostly in Gauteng Province.

A case of "Pigeon fever," (*Corynebacterium pseudotuberculosis*) was diagnosed in Washington State, USA.



Equine Disease Quarterly

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Equine Herpesvirus 1 Revisited: Significance and Control Strategies

Equine herpesvirus 1 (EHV-1) is the most significant equine herpesvirus in terms of equine health and economic impact to equine industries worldwide. EHV-1 is believed to have co-evolved with horses over millions of years. This co-evolutionary relationship resulted in the development of a life-long carrier state in a high percentage of infected horses. This involves viral latency (silent infection) of various sites (trigeminal ganglia in the central nervous system, respiratory lymphoid tissues, and CD3+ T lymphocytes in the blood). Latency ensures perpetuation of EHV-1 by serving as a virus reservoir for infection and dissemination in susceptible populations. It is no wonder that EHV-1 is ubiquitous in horse populations worldwide.

A wide range of clinico-pathological syndromes are attributed to EHV-1 infection. EHV-1 infections can result in respiratory disease in foals and two to three-year-old horses in training; contagious abortion in mares; congenital disease and death in foals infected *in utero*; and neurologic disease (myeloencephalopathy) in horses of variable ages, especially older animals. Less frequently encountered EHV-1 diseases include: retinouveitis in foals; fatal generalized peracute disease (pulmonary viscerotropic infection) in young adult to older adult horses; intestinal ganglionitis and impaction; and scrotal edema and loss of libido in stallions.

EHV-1 was implicated as the agent responsible for annual occurrences of “abortion storms” in the Thoroughbred breeding population in Kentucky at least as far back as 1933 and is widely acknowledged as the most significant cause of equine contagious abortion in many countries. Implementation of sound management practices and prophylactic vaccination of pregnant mares have, where practiced, greatly reduced the frequency of EHV-1 abortion storms. Susceptible mares exposed to EHV-1 late in pregnancy may carry to term, but give birth to a diseased foal that invariably succumbs from fulminant viral pneumonitis. Unless appropriate biosecurity measures are taken, there is a high risk that affected foals can serve as a source of infection through direct or indirect means for healthy foals and pregnant mares.

The clinical syndrome that has attracted the most concern in recent years is equine herpesvirus myeloencephalopathy (EHM). This syndrome has been recorded with increasing frequency in North America and Europe over the past 20 years (Figure 1). In 2007, the USDA designated EHM caused by a hypervirulent strain of EHV-1, a potentially emergent disease of the horse. EHM tends to be seasonal with increased case numbers in winter and spring.

A comprehensive biosecurity plan is critical to prevent and control outbreaks of EHM. Optimally, its aim should be to prevent the introduction of an equine pathogen viz. EHV-1 onto a premises, be it a farm or event venue (equestrian, racetrack, horse show), by whatever measures are considered appropriate and necessary. This is especially important when dealing with an EHM outbreak. In such a situation, the primary aim should restrict the spread of infection at the index premises/facility by quarantine of infected and exposed horses. Furthermore, every effort should be made to ensure that the outbreak is effectively contained and eliminate the possibility of virus spread to other premises/facilities. Restriction of movement of exposed horses off an affected premises is crucially important, and failure to observe this precautionary measure carries a considerable risk of EHV-1 spread that can have very significant consequences. This was exemplified at the NCHA Western National Championship at Ogden, Utah

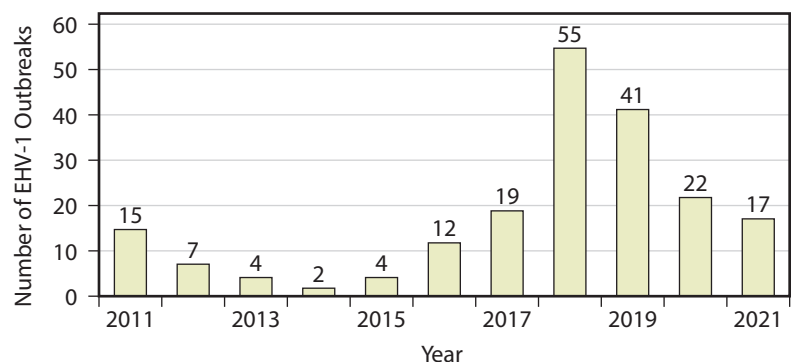
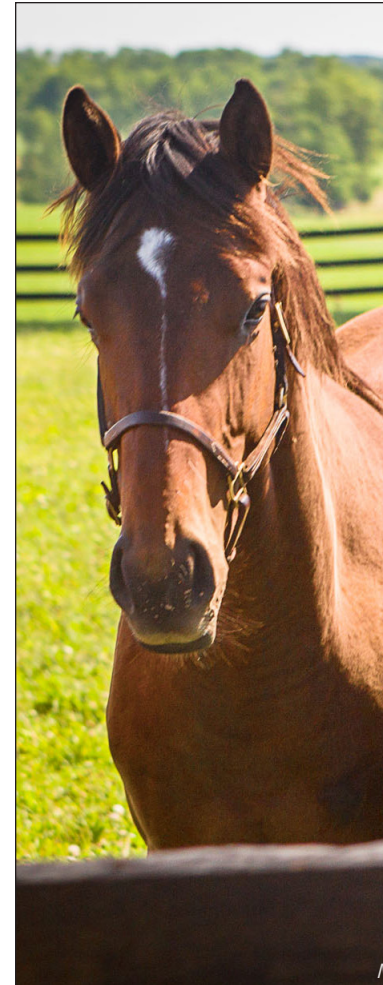


Figure 1. Recorded Outbreaks / Cases of Equine Herpesvirus 1 Myeloencephalopathy in the USA, 2011- May 10, 2021.

4 in May 2011, when exposed horses departed the event and subsequently spread EHV-1 to 12 US states and two Canadian provinces. An analogous situation occurred at the CES Spring Tour in Valencia, Spain in February 2021. During the extensive EHM outbreak at this event, a significant number of exposed horses were transported back to their countries of origin. The outcome was calamitous with multiple horses developing neurologic disease, some of which died or were euthanized.

Equine herpesvirus 1 remains a highly significant pathogen that has the potential to cause a range of clinical syndromes in the horse and can have considerable economic consequences for equine industries.

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KENTUCKY

Common Equine Hepatotoxins in Central Kentucky

Liver disease in horses can occur due to many causes, including infectious agents, neoplasia, and toxicants. Clinical signs are often nonspecific, and can include inappetence, depression, colic, weight loss, weakness, icterus (jaundice), yawning, head-pressing, abnormal behavior, and coagulation abnormalities. Toxic agents can vary tremendously depending on an animal's environment and geographic location. This article describes some of the hepatotoxicants (liver toxins) that affect horses in central Kentucky.

Pyrrrolizidine alkaloids (PAs)—PAs are found in a variety of plant species and can cause chronic liver disease and eventual liver failure with prolonged ingestion. Due to the liver's immense reserve and regenerative capacity, horses can appear normal until greater than 80% of the liver is affected. Severe clinical signs can then develop rapidly, giving the appearance of acute disease. PA-producing plants found in Kentucky include ragwort, butterweed, groundsel, and rattlebox (*Senecio*, *Packera*, and *Crotalaria* spp.). Most PA-producing plants are unpalatable when fresh, although horses may eat them when other forage is lacking. The plants become palatable when dried and can pose a greater threat in hay. Poisoning can be prevented by providing high-quality forage and checking hay for weeds.

Cocklebur (*Xanthium strumarium*)—Cocklebur seedlings (2-leaf stage) contain large quantities of the toxin carboxyatractyloside. Seedlings emerge in early spring when other forage is limited and are readily consumed by horses. High concentrations of the toxin are present in the seeds, and horses have been poisoned by cocklebur seed contamination of hay or grain. Cocklebur poisoning can be

prevented by providing high-quality forage and inspecting hay and grain for cocklebur seeds.

Aflatoxin—A mycotoxin produced by species of *Aspergillus* molds under certain conditions. Aflatoxin can contaminate corn, oats, or any other high-energy feedstuff. Mold can contaminate crops growing in fields, particularly if the plants have been stressed by drought, frost, insects, or other harmful elements. Warm and damp storage conditions and pests also facilitate mold growth. Aflatoxin can be present in toxic concentrations with or without visible mold. To avoid the risk of mold and mycotoxins, feed should be purchased from reputable sources; stored in a cool, dry location; and adequately protected from rodents and other pests.

Blue-green algae (cyanobacteria)—Under the right environmental conditions, these microscopic organisms normally present in ponds, lakes, streams, oceans, and other natural water sources can undergo rapid growth (harmful algal blooms, HABs). Certain species produce toxins, including hepatotoxins (e.g., microcystins and nodularins). Horses can die rapidly after ingestion, and animals are often found dead in or near water sources. Excess nitrogen and phosphorus from animal waste or fertilizer runoff can facilitate HABs. Horses should be provided with fresh, clean water, and ponds or other natural water sources should be fenced off to prevent blue-green algae poisoning.

Amanitin—Toxin found in certain species of mushrooms, including *Amanita*, *Galerina*, and *Lepiota* spp. Mushrooms can appear suddenly in pastures and occasionally in stalls. Horses are less likely to ingest mushrooms than other animal

5 Equine Hepatotoxins, continued from page 4.

species, but the best way to prevent poisoning is to monitor pastures and remove mushrooms.

Certain medications (e.g., phenylbutazone, flunixin, acetaminophen, salicylates, antifungals, and many others) can cause liver damage, especially if administered in high doses, for prolonged periods of time, or to animals with pre-existing conditions. Accurate dosing and careful consideration of medication risks versus benefits can help minimize chances of adverse effects.

Many other substances can cause liver damage and may be more common in different geographic areas. Consultation with a clinical veterinary toxicologist should be considered when toxicosis is suspected.

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Twenty Years' Experience with West Nile Virus, 2001-2020

Until 1999, West Nile (WN) virus was unknown in the Western Hemisphere, much less the USA. In August of that year, the virus was identified in New York City and caused the deaths of seven persons from viral encephalitis. Coincidentally, the virus was also implicated in the death of birds in New York's Bronx Zoo and crows in the precincts of the zoo.

West Nile virus is a mosquito-transmitted arbovirus, member of the Japanese encephalitis virus serocomplex, family Flaviviridae, known for many years to be a highly adaptable virus, as evidenced by its extensive distribution throughout the Old World. As such, it was no surprise that WN virus spread rapidly from its presumed point of introduction in New York. It initially extended southwards along the eastern seaboard, while also migrating westwards into the hinterland of the USA. With the exception of Alaska and Hawaii, WN virus had reached the remaining 48 states by 2004. The virus was later confirmed in Alaska in 2018.

Kentucky reported its first cases of WN virus in birds and horses in 2001 (Figure 1). Devastating losses were recorded in crows and other members of the corvid family, and eight cases were diagnosed in horses in counties in the north-central part of the state. In 2002, the virus dramatically spread throughout much of Kentucky with the exception of the eastern part of the state. To date, 2002 was witness to the greatest number of cases recorded in horses (513), humans (75), and birds in Kentucky. Case numbers in horses (102) and humans (14) declined significantly the following year. With the exception of minor surges in horse cases in 2006, 2012, 2013, 2017, and 2018, the annual number of reported cases of infection remained in single digits. With the exception of 2006 and 2013, the same years also saw corresponding minor surges in human cases (CDC, ArboNET). Cases of WN virus infection have been confirmed in horses every year since 2001 and, with the exception of 2020, also in humans.

The seasonality of equine WN infections, based on the weekly onset of illness, was consistent with other arboviral encephalomyelitides (Figure 2). Apropos of the 765 equine cases of infection recorded in Kentucky since 2001, the onset of virus activity ranged from early June to mid-July, depending largely on the level of mosquito activ-

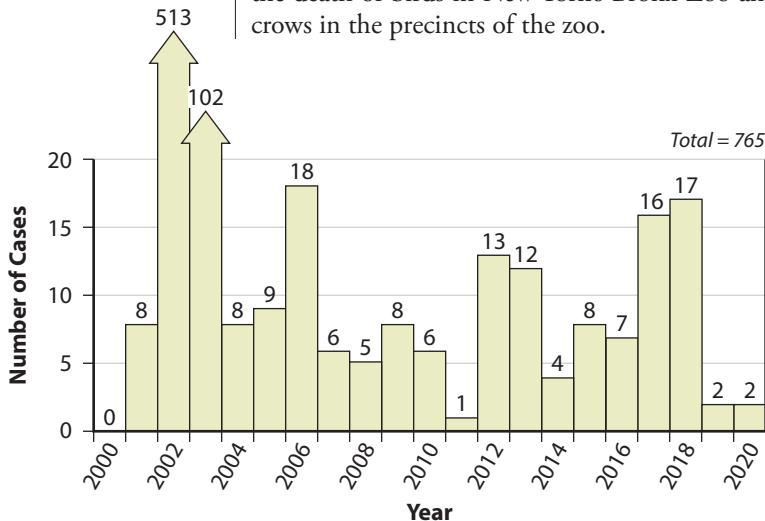


Figure 1. Annual number of equine cases of WN virus infection in Kentucky, 2000-2020.

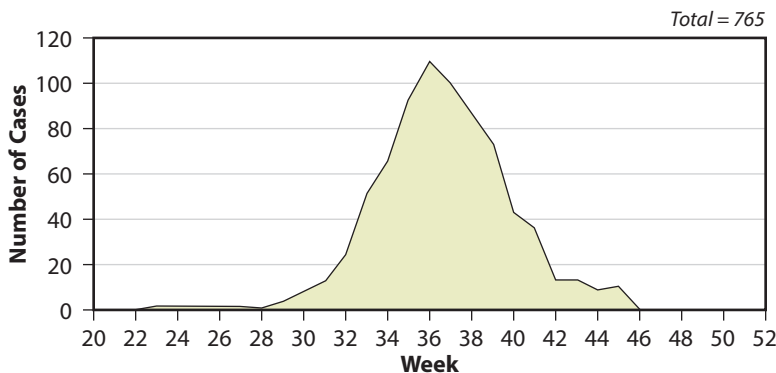


Figure 2. Cumulative equine WN virus cases of infection by weekly onset of illness in Kentucky, 2000-2020.

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ity in any particular year. The peak of infections occurred approximately mid-September, and virus activity ceased by mid-November.

A breakdown of the 20-year total of WN virus infections in Kentucky revealed that the most common horse breeds/categories included American Quarter horses (AQH), Tennessee Walking horses (TWH), Thoroughbreds, and pleasure horses, of which AQH and TWH's comprised almost 50% of the overall number. Females outnumbered males 57% to 43%. Ages of confirmed WN cases ranged from three months to 39 years, with a median of eight years. Of the 765 total cases, 541 (71%) survived and 224 (29%) died or were euthanized.

The first equine vaccines against WN virus-related disease received conditional licensure from the USDA in 2001. Even though vaccination is effective in protecting against disease, the great majority (86%) of WN cases recorded in Kentucky had never been vaccinated or were only partially vaccinated (10%). Only approximately 4% of cases had current vaccination histories. While this is not totally surprising, it is disappointing considering that WN virus is one of five core equine vaccines strongly recommended by the American Associa-

tion of Equine Practitioners. Of the 656 cases of WN infection without prior vaccination history, approximately 30% died or were euthanized. Greater efforts are therefore needed to encourage horse owners to take advantage of vaccination if continued losses from WN virus infection are to be reduced or even eliminated.

This review confirms that WN virus has become endemic in Kentucky and will likely continue to cause horse and human disease. Its broad host and vector range, capability for transovarial transmission in mosquitoes, and horizontal transmission in birds and a diversity of mammals has ensured its perpetuation.

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