Twenty years ago the Lloyd’s Equine Disease Quarterly was born. The inaugural Commentary stated: “The purpose of the Equine Disease Quarterly is to provide accurate information highlighting an increase or decrease in the incidence of a particular equine disease or syndrome. It will also document the emergence of new or unfamiliar conditions.”

In the first edition, the International Collating Centre report was 44 words long; now it fills an entire page at 600+ words. Why should the average horse owner even want to know about equine diseases on other continents? As was repeatedly emphasized at the October 2012 9th International Conference on Equine Infectious Diseases in Lexington, KY, horses are worldwide travelers, and only through constant disease surveillance, testing, and communication can we further the understanding of disease incidence and spread. Abstracts of the conference lectures are available at http://www.eidc2012.com/uploads/EID_IX_Proceedings_reduced_size.pdf.

As an example, prior to 1999 the average horse owner had never heard of West Nile Virus (WNV). In a few short years after its diagnosis in the USA, the first equine WNV vaccine was produced and now several vaccines are commercially available. Twenty years ago, Hendra virus was unknown. While this disease is still foreign to North America, we can all learn valuable lessons on how the Australians have dealt with the disease since its emergence. In contrast, grass sickness has been a recognized equine disease for nearly a century, yet remains mysterious despite research efforts. Learn more about these diseases in this edition.

The EDQ has reported on diseases of importance in North America via national database data and a cadre of generous guest authors, leaders in their fields. Documenting numbers of cases of diseases and conditions in Kentucky has been accomplished through combing the records of the University of Kentucky Livestock Disease Diagnostic Center (now the UK Veterinary Diagnostic Laboratory). From necropsy cases with diagnoses of cardiovascular disease (1997) to Salmonella isolates (2002) and leptospiral abortion updates (2011), this valuable information provides a glimpse into disease incidence and prevalence not easily obtained anywhere else in the world. Other Kentucky-specific data is generously provided from the Office of the Kentucky State Veterinarian, the Kentucky Public Health Veterinarian, and others.

Unlike most other things in life, the EDQ remains free in print copy due to the generous support of Lloyd’s of London and is mailed to individuals in more than 100 countries. All editions are archived and available online at http://www.ca.uky.edu/gluck/Q_articles.asp, and articles are widely disseminated by reprinting in veterinary publications, lay equine magazines, and newsletters.

Since 1986, Lloyd’s has donated more than $1 million to support various activities undertaken within the Department of Veterinary Science, including funds for the Equine Disease Quarterly. The outcome of this partnership has provided tangible benefits to the equine industry, not just locally, but at national and international levels. We look forward to an ongoing relationship to benefit the health of horses around the world.

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The International Collating Center, Newmarket, United Kingdom (UK), and other sources reported the following disease outbreaks.

Contagious equine metritis was reported from Germany. Infection with the causal bacteria, *Taylorella equigenitalis*, was confirmed in non-Thoroughbreds: two stallions and three mares on one premises and a stallion on a second premises. One case of equine coital exanthema (equine herpesvirus-3) was diagnosed in Kentucky, USA.

Strangles was recorded in Germany, Ireland, Switzerland, and the USA. Ten cases were reported from Ireland. Outbreaks involving 17 horses on two premises occurred in Switzerland. Single cases and outbreaks of strangles were reported from Maine, Kentucky, and Pennsylvania in the USA, where the disease is endemic.

Outbreaks of equine influenza were reported from Argentina, the UK, and the USA. The disease was confirmed at three Thoroughbred racetracks in Buenos Aires; the virus was a strain of Florida, clade 1 sublineage. The UK diagnosed influenza on two premises. Several cases of influenza were confirmed in Kentucky and Wisconsin in the USA.

Equine herpesvirus (EHV)-related diseases were reported from Argentina, France, the UK, and the USA. Argentina reported five cases of EHV-1 abortion (non-neuropathogenic strains) in 2 of 10 non-vaccinated Thoroughbreds and 3 of 70 vaccinated Thoroughbreds. In France, EHV-1 and -4 were implicated in a respiratory outbreak in Thoroughbreds at a training center; also reported was a single case of EHV-1 neurologic disease. The UK noted outbreaks of EHV-4 respiratory disease on 10 premises and one case of EHV-1 neurologic disease. EHV respiratory disease was also reported in the USA. Several cases of infection with EHV-2 or -5 were diagnosed in California, Kentucky, and Ohio in the USA.

Ireland reported cases of *Pneumocystis carinii* infection in a group of foals.

Equine infectious anemia (EIA) was confirmed in Canada, France, Germany, and the UK. Single cases were diagnosed on two premises in Alberta and one in the Yukon. France reported the infection in three Camargue horses. Germany reported several outbreaks: four in one state, two in another, and single premises in two other states. The index case on one premises was in a foal that received plasma therapy; the donor mare was positive. Upon investigation, six other plasma recipients were found to be EIA positive.

Equine piroplasmosis was reported from France (endemic), Spain (endemic), Switzerland, United Arab Emirates (endemic), and the USA. Switzerland confirmed one case based on clinical evidence and serologic findings. The USA reported testing more than 190,000 horses since November 2009; no new cases of infection were detected in the third quarter.

In the USA, the number of equine cases of Eastern equine encephalomyelitis during the third quarter was 35; the national total is 171 cases in 2012. The number of affected states went from 6 to 16 with the greatest increase in disease cases confirmed in Louisiana, Mississippi, North Carolina, and Alabama. The number of equine cases of West Nile encephalitis rose dramatically in the third quarter in the USA; a total of 427 cases were reported from 40 states, with maximal increases in Texas, Pennsylvania, California, Indiana, and Iowa. Single cases of Hendra virus infection were confirmed on three premises in Queensland, Australia, all fatal.

Vesicular stomatitis infection (New Jersey strain) was diagnosed in 25 equine (on 18 premises) in New Mexico and Colorado.

Salmonellosis was reported by Ireland (three cases) and the USA. *S. agona* and *Salmonella* spp. belonging to Groups B, C1, and C2 were involved. Argentina recorded limited outbreaks of rotavirus Group A infection in foals. Equine monocytic ehrlichiosis was reported in the USA, with multiple cases diagnosed in Ohio and Kentucky.

An isolated case of *Anaplasma phagocytophilum* infection was recorded in Switzerland.

*Second Quarter Report for Australia*
A marked increase in the incidence of Hendra virus cases (termed “spillover events”) in horses over the last 18 months has again brought this disease to prominence. Hendra virus was first recognized in 1994 following the death of a popular horse trainer and 20 horses in Hendra, a suburb of Brisbane (Queensland), Australia. A member of the Paramyxoviridae, Hendra virus has been categorized in the Henipah virus genus due to its similarity to Nipah virus, another lethal, bat-borne zoonotic agent.

Between the 1994 outbreak and June 2011, 14 Hendra virus spillover events had been confirmed that resulted in seven human infections (four fatal, including two veterinarians). The publicity following these events resulted in widespread testing of suspect or ill horses and more stringent use of personal protective equipment (PPE). Since June 2011, at least 25 outbreaks have occurred, nearly double the total number of cases prior to then. A welcome difference in the recent spate of Hendra cases is that no human infections have resulted from personnel handling or treating infected horses. Extensive testing of in-contact humans by health authorities is routine following every confirmed equine Hendra case. Both heightened awareness of veterinarians and horse owners, together with a greater acceptance of PPE practices are considered to be largely responsible for the lack of human infections during the recent cases.

Equipment now in use by eastern Australian equine practitioners includes: disposable splash-proof overalls, disposable respirators (N95 rated), safety eyewear or full-face shield, rubber boots or disposable shoe covers, and disposable gloves (often used in duplicate or triplicate). Pre-packaged PPE kits are now widely available. Following intensive lobbying by veterinary associations, costs for these items and other biohazard equipment are subsidized for Queensland practitioners dealing with suspect cases.

In addition to these measures, the Australian Veterinary Association and Veterinary Licensing Boards have insisted that all veterinary students be certified in PPE application and be aware of basic facts regarding Hendra virus infection prior to observing equine veterinary practice. Although Hendra virus in horses was originally restricted to the northeastern seaboard of Queensland and northern New South Wales, recent outbreaks have extended farther to the south and east. The natural hosts of Hendra virus are bats (flying foxes) that most likely shed virus through urine, feces, and other bodily fluids. Areas beneath fruiting trees such as fig trees are considered particularly high risk for horses as this is where bats tend to congregate. Human infection results from close contact with the body fluids and tissues of infected or dead horses.

Although Hendra virus has instilled fear in the Australian horse community, there have been some recent developments: a vaccine for horses has been fast-tracked for release in 2013, and a stall-side rapid diagnostic test is also under development.

1 N95 facemasks are rated by National Institute for Occupational Safety and Health (USA) and filters at least 95% of airborne particles (equivalent to European standard P2).
More than 100 years ago, an outbreak of grass sickness was recognized in Scotland, and this devastating disease remains one of the great unsolved mysteries in equine medicine. Still not knowing its cause, and therefore lacking any means for causal therapy, the disease is a continuing threat.

As its name suggests, grass sickness is strongly associated with grazing and occurs in acute, subacute, and chronic forms with a considerable overlap in clinical signs. The major symptoms relate to partial or complete paralysis of the digestive tract. Considering the nature of the damage to the nervous system, combined with epidemiological factors, a type of toxin is suggested as the causative agent.

Many potential causes have been examined but none has survived scientific scrutiny despite almost 90 years of investigation. However, the cause was associated with exposure to grazing or the ground right from the beginning and therefore it was also considered that grass sickness might be a form of botulism. The first reports of botulism in horses in connection with feedstuffs were reported in the United States and additional cases also were associated with grazing. Beside this, some symptoms of these two diseases are comparable.

That grass sickness might be caused by Clostridium botulinum was first proposed almost 90 years ago in 1923. However, these investigations failed to demonstrate clear evidence and the idea lost favor. Moreover, doubts persisted because of the anaerobic and ubiquitous nature of C. botulinum. In 1999, new interest in the organism began. Researchers showed that green grass blades can contain botulinum toxin. On a European study where grass sickness occurred twice within eight months, grass and soil samples and necropsy specimens were tested for the presence of bacterial forms and toxin of C. botulinum. Different types of the germ (A-E) and neurotoxin (BoNT) were found and showed that growing grass contained free BoNT. Bacterial forms in soil were numerous in May, but were not found in August and September. It is speculated that biofilms adhere to lower parts of the grass and allow C. botulinum to survive within a bacterial consortium while being trapped and protected from dehydration.
Other indications for the involvement of clostridia are some similarities with the equine atypical myopathy, with *Clostridium sordellii* as a suspected causative agent. Here, we also encounter a regional, seasonal, and pasture-associated disorder that is characterized by degenerative alterations. Also, environmental factors seem to influence the incidence of both diseases as well as access to pasture, certain weather conditions and the degree of resistance in older horses. The exact role the toxins of *C. sordellii* or *C. botulinum* play in the respective diseases has yet to be defined. Researchers in 1924 demonstrated statistically highly significant protection against grass sickness by vaccinating horses with a mixture of botulinum toxin and antitoxin, and today work continues in the United Kingdom toward demonstrating efficacy of a botulinum toxoid against equine grass sickness. Such progress will take more time, and we must make sure to use other preventive measures to the best of our knowledge. Careful husbandry and meticulous pasture management must be of the highest standards.

It brings to mind the saying of the French physiologist Claude Bernard (1813-1878): “It is not the germ that causes disease but the terrain in which the germ is found”—an opinion that is older than the grass sickness itself.

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**KENTUCKY Pastures**

What grows in your pasture? Ideally, forage that is nutritious to horses is abundant. However, a perusal of most Kentucky horse pastures will uncover 20 plant species, many of which are weeds. The abundance of weedy species depends greatly on the management of the pasture. Overgrazing of pasture grasses and soil compaction are primary causes of weed occurrence.

A high quality, nearly weed-free forage is desired if the pasture is a significant portion of the diet. Conversely, a pasture maintained as a dry lot for feeding horses will contain many weeds but there is little reason to control these weeds since there are few, if any, desirable forages present. My experience indicates that Kentucky horse pastures are maintained between these two extremes. Frequent concerns of farm managers are plant identification in pastures and weed control.

Kentucky is located in the transition zone between warm-season and cool-season weeds, meaning weeds grow well in summer and winter. This provides horse pasture managers with the challenge of what weeds, if any, should be controlled in a pasture. Generally, poisonous weeds and weeds that inhibit grazing should be removed from a pasture. Poison hemlock occurs widely across Kentucky and is toxic to horses and other animals. Although rarely eaten by horses, it should be removed from the pasture. Musk thistle and bull thistle are found throughout Kentucky and inhibit grazing. Canada thistle occurs less frequently but also inhibits grazing and is more difficult to control. Large crabgrass and yellow foxtail are warm-season grasses of summer. Horses graze the large crabgrass but not yellow foxtail. Buckhorn plantain is a cool-season plant that horses consume when pasture grass is limited. Other common weeds are listed in Table 1.

No one, simple weed control solution exists for all these species. First, determine if there is a need for removing the weeds. A poisonous plant such as poison hemlock is controlled by hand weeding, mowing at the proper time, or by applying herbicides in late fall or early spring. Regardless of the method, do not allow animals to graze dying or decaying hemlock plants. Hand weeding and removal of the plants from the pasture is the safest
method. Thistles generally are too numerous to hand weed, and herbicides are needed.

For most weeds, mowing is not an effective control technique. Mowing may prevent seed production of some weeds; however, to kill many weeds, the mower must cut at about two inches or lower. While this may control some weeds, it also reduces grass production. If mowing is not the best answer, consult your county extension agent for weed identification and proper use of herbicides to achieve weed control. The paper Response of Pasture Weeds to Herbicides or Mowing (Green and Witt, 2012) is available from the local Kentucky County Extension Agent for Agriculture and Natural Resources. This publication contains photographs of commonly occurring pasture weeds and specific control tactics.

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### Table 1. Common Kentucky Weeds.

<table>
<thead>
<tr>
<th>Cool-Season Weeds</th>
<th>Warm-Season Weeds</th>
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<tbody>
<tr>
<td>Musk thistle</td>
<td>Large crabgrass</td>
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<tr>
<td>Bull thistle</td>
<td>Yellow foxtail</td>
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<tr>
<td>Canada thistle</td>
<td>Goosegrass</td>
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<tr>
<td>Buckhorn</td>
<td>Johnsongrass</td>
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<tr>
<td>Broadleaf plantain</td>
<td>Nimblewill</td>
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<tr>
<td>Broadleaf dock</td>
<td>Spiny amaranth</td>
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<tr>
<td>Purple deadnettle</td>
<td>Buttercups</td>
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<tr>
<td>Chickweed</td>
<td>Common ragweed</td>
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<tr>
<td>Wild garlic</td>
<td>Asters</td>
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<tr>
<td>Star of Bethlehem</td>
<td>Perilla mint</td>
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<tr>
<td>Cocklebur</td>
<td>Hemp dogbane</td>
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