University of Kentucky

Bluegrass Equine DIGEST

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What's the Socioeconomic Status of Most Equestrian Participants?

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Nearly all responders identified as middle-class, middle-aged females.

Socioeconomic status (SES) is a complex assessment of an individual or group's economic and sociological standing that has strong impacts on people's lifestyle choices.

Equestrian participants have multiple roles and interests in the horse industry over time, and many will participate for decades. The complexity of SES takes into account education, income, occupation, residence location, heritage, and other cultural factors that affect this ability to participate.

However, what is the SES of most equestrian participants? What are the demographics of riders compared to nonriders? To try to answer these questions, University of Kentucky researchers Karin Pekarchik (College of Agriculture, Food, and Environment) and Kimberly Tumlin, PhD, MS, MPH, (College of Public Health) developed a survey distributed earlier in 2019.

They delivered "Portrait of an Equestrian: Characterizing Active Participants in Horse Activities and Horse Sports," internationally through multiple outlets. The survey addressed the socioeconomic status of recreational, amateur, and professional participants in the horse industry. Questions included type of equestrian participation, rider and handler income, education, residence, and social support structures.

The five states with the greatest number of respondents were Kentucky, California, Virginia, Pennsylvania, and Washington. The five countries with the most respondents were the United States, Canada, the United Kingdom, Australia, and New Zealand. Response demographics included more than half participating in English disciplines. Seventy percent of respondents categorized themselves as either recreational or amateur riders, not professionals. Most described themselves primarily as riders or trainers, having been involved with the sport for more than 20 years. Two-thirds of the respondents listed riding frequency as "most days."

Nearly all responders identified as middle-class, middle-aged females. Only 10% of respondents reported making more than $100,000 in personal income. That figure increased 250% when reporting a household income greater than $100,000. In contrast, only 14% of responders reported less than $35,000 in annual income. These economic factors can be barriers or boons to participation in equestrian sport, although 46% reported that their salary is sufficient to afford their horse habit.

Nearly three-quarters of respondents said they feel emotionally supported by friends and family. While more than half reported currently living in a rural setting, they did not necessarily grow up in the country or on a farm. At least half reported childhood residences in either suburban or urban locations.

Sport participation in youth programs and having a parent interested in a similar sport often influence participation over one's life course. However, more than two-thirds of the survey respondents did not participate in a youth or an adult club (such as 4-H, Pony Club, British Horse Society).

In This Issue

SOCIOECONOMIC STATUS

One of the researchers' most surprising findings was that most respondents reported that their families were not involved in horses. These findings suggest that recruiting new participants to the horse industry might be key to its sustainability. Understanding the complex nature of the SES of people involved in the horse industry could help maintain and grow participation rates in coming years. UK

>Karin Pekarchik, senior extension associate for distance learning, Department of Biosystems and Agricultural Engineering, and Kimberly Tumlin, PhD, MS, MPH, assistant professor, University of Kentucky Preventive Medicine and Environmental Health, Athletic Training and Clinical Nutrition, College of Health Sciences and College of Public Health, provided this information.

Stonestreet Farm Launches Yearling Warranty

Leading commercial Thoroughbred breeder Stonestreet Farm has announced the development of an innovative blood sampling, testing, and secure storage protocol in partnership with The University of Kentucky's Gluck Equine Research Center. The program was developed in response to reports of off-label bisphosphonate use in growing horses. Combined with newly available post-sale testing offered by Thoroughbred auction houses, buyers of a Stonestreet-bred and -raised yearling will have an opportunity to review a blood-health window of at least six months prior to their purchase.

"Last year we raised and sold nearly $20,000,000 of yearlings, but our focus has always been to raise racehorses, not sale horses," said Barbara Banke, owner of Stonestreet Farm. "We are proud of what we do, and I think transparency in raising a racehorse is so important. We want our buyers to have the utmost confidence in our yearlings."

The buyer of any Stonestreet yearling in 2019 can request a report stating the bisphosphonate and anabolic steroid test results for that horse.

PHOTOS COURTESY STONESTREET FARM

During 2019, blood samples were drawn from each Stonestreet yearling on a regular schedule as recommended by the Gluck Equine Research Center using current bisphosphonate detection periods. A third-party veterinarian hired by the Gluck Equine Research Center and experienced in handling samples within a regulatory environment drew the samples.

Scott Stanley, PhD, professor of analytical chemistry at the Gluck Equine Research Center, supervised sample collection, ensured a strict chain of custody, and authored documentation connecting each sample to the yearling from which it was taken. Samples were stored and frozen in accordance with the normal regulatory laboratory standards established for pre- and post-race blood samples in a dedicated freezer.

Gluck staff tested the samples for bisphosphonates and anabolic steroids. The buyer of any Stonestreet-bred and -raised yearling in 2019 can request a report stating the test results for the horse they purchased. Further, at their own expense, they can request testing for anabolic steroids and/or bisphosphonates on the blood samples that remain in secure

Masthead

University of Kentucky Ag Equine Programs

Holly Wiemers, MA, APR, managing editor and communications director of UK Ag Equine Programs, holly.wiemers@uky.edu

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STONESTREET YEARLING WARRANTY

storage at the Gluck Equine Research Center. Purchasers can request any additional testing during the seven days following the fall of the hammer by completing the Request to Test form available at Stonestreetfarms.com. Following completion of the secure storage period, the blood samples will be donated to Gluck and used in research projects.

"We applaud Stonestreet's efforts to employ an approach driven by transparency and good science as part of their sales operation," said Nancy Cox, PhD, dean of UK's College of Agriculture, Food and Environment. "We look forward to further results of this project as time goes on. This kind of project is what we do best, to merge our college's scientific capacity with a worthy industry goal. It also displays our commitment to safety in all aspects of the equine industry." UK

Source: Modified news release by Gemma Freeman of Stonestreet Farm

Blue-Green Algae: Dangerous to Pets and Livestock

Blue-green algae, also called cyanobacteria, are microscopic organisms normally present in aquatic ecosystems, including lakes and ponds. Scientists have identified thousands of species of blue-green algae; at least 80 are known to produce toxins that can cause illness and death in animals as well as humans.

Heavy growth ("blooms") of these toxin-producing algae can cause high concentrations of toxins in the water. In North America, *Anabaena, Aphanizomenon, Oscillatoria,* and *Microcystis* are the blue-green algae species most commonly associated with poisoning.

In Central Kentucky, blooms are most common in late summer and early fall, during hot, sunny weather. Contamination of water with excess nutrients, particularly nitrogen and phosphorus, further encourages algal growth. Common sources of excess nutrients include fertilizer runoff from fields, lawns, and gardens and direct manure and urine contamination from livestock.

Blooms can produce a blue-green sheen on the water surface, or they can be pea-green and thick, like spilled paint. Blooms can also be brown or white. They can form scums, slimes, or mats. It is impossible to tell if a bloom is toxic just by its appearance; consider all blooms potentially toxic.

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Because it's impossible to tell if a bloom is toxic just by its appearance, consider all blooms potentially toxic.

Blue-green algae can produce neurotoxins (affecting the nervous system) or hepatotoxins (causing liver damage), and some species can produce both types. Neurotoxins can cause muscle tremors, seizures, excessive salivation, diarrhea, difficulty breathing, and death within hours or even minutes of exposure. Hepatotoxins cause vomiting, diarrhea, bloody or dark stool, and pale or jaundiced (yellow) mucous membranes. Animals can die quickly, or they can develop liver failure over several days.

There are no antidotes for blue-green algae toxins, so early decontamination and supportive care can mean the difference between life and death for an exposed animal. If your pet develops these or any other signs after recent exposure to water—even water with no obvious algal blooms--seek immediate veterinary care. Toxins can persist in the water for more than a week after the bloom itself has collapsed.

To prevent blue-green algae poisoning in pets and livestock:

- Provide plentiful clean, clear, fresh water for your animals. Keep water bowls, buckets, and troughs clean and well-maintained.
- Never let your pets (or children) swim in, play in, or drink discolored, slimy, scummy, or otherwise suspicious water. Assume any bloom is toxic.
- Pay attention to local health and water advisories, and respect water body closures. Water that appears clean can still contain high concentrations of toxins.
- Fence off farm ponds, creeks, and other natural water sources to prevent livestock from contaminating them as well as drinking from them.
- Fence off backyard ponds and other natural water sources to keep pets from accessing them.
- Prevent fertilizer and/or manure from running off into water sources.
- If your pet does access suspicious water, wash him thoroughly with clean, fresh water, and prevent him from licking his fur. Wash your own hands and arms after washing your pet, as exposure to blue-green algae can cause skin, eye, nose, and throat irritation in humans.
- If animals become ill after exposure to a pond, lake, or other natural water source, seek immediate veterinary care—even if the water appeared clean, toxins can still be present. Tell your veterinarian if your animal might have been exposed to blue-green algae. This can help direct treatment, as many other illnesses can have similar signs. UK

>Megan C. Romano, PhD, Veterinary Toxicology Resident, University of Kentucky Veterinary Diagnostic Laboratory, provided this information.

Weed Management Plans for Horse Pastures

Fall is a good time to evaluate the quality of your horse pastures, because it is easy to see which weeds were most prevalent and uncontrolled during the summer and are now large and seed-producing. It is also a good time to develop a weed management plan for pastures in the coming year. When creating an effective weed management plan, consider the pasture's purpose, weed species and abundance, which weeds should be controlled and how, and sources of information.

The pasture's purpose. If pasture is a significant portion of your horses' diet, you'll want a high-quality, nearly weed-free forage. Conversely, a "pasture" maintained as a drylot for feeding horses will contain many weeds, but there is little reason to control these weeds since there are few, if any, desirable forages in the drylot. Kentucky horse pastures usually are maintained between these two extremes. Property owners often ask why these weeds are in their pastures, followed by what they should do about them. Forages grown with adequate fertility and not overgrazed will limit weed occurrence but not prevent all weeds from growing.

Weed species, abundance, and distribution. Plants we call weeds grow in ecological niches—environments that allow for germination, vegetative growth, and maturation. Horse pastures provide several of these ecological niches that allow some weeds to thrive. Kentucky is located in the temperate transition zone in which both warm-season and cool-season plants grow.

Warm-season weeds germinate in spring or early summer, grow, and produce seeds before frost. Cool-season weeds germinate and produce some growth in the fall and seeds the following spring or summer.

When pastures house many weed species, horse pasture managers face the challenge of determining which weeds, if any, they should control. The most abundant weeds in horse pastures are usually annual species that produce thousands of seeds. Spiny pigweed, also known as spiny amaranth, for instance, produces more than 100,000 seeds per plant. This weed is widespread and occurs most often in compacted areas along fences and around feeding and watering areas of pastures. Spiny pigweed is also a good example of the "patchiness" of weeds; they often grow only in certain portions of the pasture where their ecological niche occurs.

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Weedy species to treat with herbicides in the fall include (from top left, clockwise) purple deadnettle, thistles, buckhorn plantain, and common chickweed.

Which weeds to control, when, and how. Generally, you should remove all poisonous weeds and weeds that inhibit grazing from a pasture.

Poison hemlock occurs widely across Kentucky and is toxic to horses and other animals. Although horses rarely eat it, you should remove it 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. Many small, tender "weeds" are nutritious and readily consumed when small but rarely consumed as large plants.

Methods of removing horse pasture weeds are limited to hand removal, mowing, and herbicides, and each has its advantages and disadvantages.

Hand-weeding can be very effective and is particularly useful for removing poisonous plants, such as poison hemlock, from a pasture. You should not only control poisonous plants but remove them from the pasture to prevent animals from consuming them.

The downside of hand-weeding is that the process is slow and inefficient for large areas. Mowing is rarely effective at killing weeds in pastures—mowing low enough to kill the weeds (2 inches or less) removes valuable forage. Mowing heights of about 6 inches will keep some large weeds from producing seeds but does not control smaller weeds.

Herbicides are efficient and provide excellent control, but in transition zone areas such as Kentucky, one herbicide will not control all the weeds with one treatment.

There are optimum times to control weeds with herbicides. The following months are the preferred time for herbicide treatment of several weedy species in Kentucky:

- **October-November:** Common chickweed, henbit, purple deadnettle, dandelion, buckhorn plantain, musk thistle, bull thistle, Canada thistle, poison hemlock
- **February-March:** Buttercups, curly dock, broadleaf dock, chicory
- **May-July:** Spiny pigweed, white clover, hemp dogbane, goldenrod, cocklebur, perilla mint, common ragweed, jimsonweed

WEED MANAGEMENT

Sources of information. Consult your local Cooperative Extension Service agricultural agent for specific information on herbicides in your area. Remember, not all herbicides are registered for use in all states and countries, so read the label carefully, and follow all directions. Many Cooperative Extension Services have publications regarding weed control in pastures, including:

Broadleaf Weeds of Kentucky Pastures. AGR-207. ca.uky.edu/agc/pubs/AGR/AGR207/AGR207.pdf

Weed Management in Grass Pastures, Hayfields, and Other Farmstead Sites. AGR-172. ca.uky.edu/agc/pubs/agr/agr172/agr172.pdf UK

>William W. Witt, PhD, emeritus professor and weed scientist with the University of Kentucky's Department of Plant and Soil Sciences, provided this information. Email: wwitt@uky.edu

Legendary Equine Disease Outbreaks

Outbreak. The word itself can be terrifying, sounding like some kind of prison escape involving dangerous inmates. And in a way, that's exactly what it is—but on a much larger scale. Billions of pathogens escape their hosts and slip into farms, barns, stalls, tack, equipment, and, inevitably, horses. Worse, they reproduce, sending out more of their destructive kind, wreaking havoc at alarming rates and causing illness, death, and financial losses.

Getting outbreaks under control can be tricky, costly, and frustrating. Some happen because of biosecurity oversights; others occur due to a lack of understanding of how diseases function. Some even occur as a result of malicious intent.

The history of equine science is full of stories about devastating outbreaks. While their trails of destruction are tragic, their tales of management and resolution are heroic. Join us as we look back at some of the world's most legendary equine outbreaks and what we've learned from them.

S. zooepidemicus in Iceland

When it comes to keeping diseases out, Iceland leads the planet. The isolated country of fewer than 500,000 inhabitants benefits from hundreds of miles of surrounding ocean to keep it free of diseases that are endemic elsewhere. And its import rules are strict, clear, and simple: No horses enter. Ever. That's it. No horses have been allowed into Iceland in the past 1,000 years. The country has one breed of horse—the Icelandic—and it's a product of historical local development, with no outside influences from any other breed. In fact, the import rule is so strict, even horses leaving Iceland for an international competition can never come home.

In addition to this rigid closed-door import policy, the government has set up tight control measures to keep diseases out. If you've been on a farm before traveling to Iceland, you must disinfect any clothes or equipment you bring with you. Better yet, leave it at the farm and buy new ones that have never touched a horse. Iceland doesn't have many of the most common equine infectious diseases. And they aim to never get them.

But 2010 was a strange year for Iceland. In mid-April, the Eyjafjallajokull volcano erupted, spreading ash and gases across the island. Researchers warned that hydrogen sulfide and ash could provoke bronchial constriction, cough, and bronchitis among the country's horses. So when horses started coughing and having snotty noses, some owners suspected volcanic ash was to blame.

But the signs didn't add up. Stabled horses were more seriously affected than horses living outdoors. And horses beyond the ash cloud's path were getting

In 2010 Iceland's population of 77,000 horses faced exposure to a pathogen they'd never encountered.

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sick. By early May competitions were being canceled to contain what was clearly an infectious disease.

"Initially, the Icelandic vets thought that a virus, such as equine herpesvirus (EHV), was causing the disease," says Andrew Waller, PhD, head of bacteriology at the Animal Health Trust (AHT), in Newmarket, U.K. "Samples were sent to labs all over the world to screen for different viral pathogens (disease-causing organisms), but none matched up with cases of disease."

Clinical signs of Iceland's "mystery disease" included a dry cough, weakness, shortness of breath, nasal discharge, and, rarely, fever, all lasting up to six weeks. Only a few cases were fatal.

It wasn't until Waller's team received isolates from which they were able to finally confirm a diagnosis. The bacteria were about 97% identical to *Streptococcus equi*, which causes strangles. But Iceland's sick horses didn't have strangles. They had "strep zoo," or *Streptococcus equi* subsp *zooepidemicus*.

Worse, they were spreading it among each other at epic rates because they were stabled in close quarters and working in indoor arenas over the country's long, dark winter.

"Full genome sequencing of 257 isolates of Iceland's *S. zooepidemicus*, in collaboration with the Wellcome (Trust) Sanger Institute, showed very few changes in the pathogen across the country," Waller says. "This close identity confirmed there'd been rapid spread of a single strain," which they named ST209.

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In 1975 Irish Thoroughbred mares began falling ill with what veterinarians eventually discovered to be contagious equine metritis.

"While it's impossible to know how ST209 got past Iceland's tough biosecurity rules, chances are it traveled in with an unwitting passenger," Waller says.

Researchers have also identified ST209 in Scandinavian horses, a person in Finland, an Icelandic cat, and a woman in Iceland who miscarried. "The pathogen could have come into Iceland inside a human," he says. "But the most likely scenario is that it traveled on clothing or riding equipment that hadn't been properly cleaned according to the biosecurity rules."

Scientists used the information from laboratory testing to manage the epidemic, which was "extremely interesting from a scientific perspective," says Waller. They were particularly curious about how one pathogen could quickly spread across an entire country of naive (never previously exposed) horses. But the experience also reminded officials about Iceland's vulnerability to such diseases and the importance of upholding its stringent biosecurity policies.

"This epidemic highlighted just how susceptible the special population of horses in Iceland are to new diseases that they have not encountered before," he says. "The import regulations in Iceland were already tough, but these measures have been further tightened to avoid more dangerous diseases, such as strangles, from making the trip to this incredible land of fire and ice."

Flu in Australia

Three years before Iceland's strep zoo outbreak, another island country full of naive unvaccinated horses fell victim to a devastating epidemic. This time it was Australia, when equine influenza slipped past its tough biosecurity firewalls.

In early August 2007 imported horses from the U.S., Japan, the U.K., and Ireland arrived in two quarantine stations in Australia for their mandatory two-week stay. Per protocol, they were all tested for specific antibodies (blood proteins horses

Hendra: A Horse and Human Risk

Veterinarians identified the deadly zoonotic (can spread between animals and humans) Hendra virus in Australia in 1994. Since then 102 infected horses and four humans in Queensland and New South Wales have died. Biosecurity and quarantine efforts in Queensland, in 2011, are featured on the cover this month.

Flying foxes, a type of fruit bat that lives in Australia, transmit the virus. Horses get it by inadvertently ingesting droppings or bodily fluids of infected flying foxes, and humans contract it when handling infected equine patients. Infected horses can display a variety of clinical signs, including lethargy, respiratory distress, frothy nasal discharge, and elevated body temperature and heart rate. Authorities caution, however, that Hendra does not cause specific signs.

In 2012 a vaccine against Hendra hit the Australian market, and it remains the most effective way to minimize the risk of contracting the virus. Veterinarians continue to urge horse owners in, around, or traveling to high-risk Hendra areas along Australia's east coast to vaccinate their animals—particularly during the cooler months.

"Vaccination of horses provides a public health and workplace health and safety benefit by reducing the risk of Hendra virus transmission to humans and other susceptible animals and helps to ensure high standards of animal health and welfare," says Ben Poole, BVSc, MANZCVS, president of the Australian Veterinary Association's equine group.

"Every one of these horses that has died because of Hendra represents one more compelling reason for horse owners to vaccinate their horses," he adds.

DISEASE OUTBREAKS

produce in response to vaccination and exposure that the immune system uses to fight disease), including those against equine influenza—which is critical because Australia is one of only three officially equine-flu-free countries (the other two are Iceland and New Zealand). One of the imported horses at the Eastern Creek Quarantine Facility, in Sydney, New South Wales (NSW), had no evidence of antibodies against equine influenza, even though he'd been properly vaccinated prior to arrival (which is required for incoming horses).

Within days the horse fell sick with fever and respiratory signs, and testing showed he'd seroconverted to positive (developed antibodies) for flu while in the quarantine station. That meant he'd picked up the disease from another imported horse. Two other horses in the same quarantine stable were also sick, says Martyn Jeggo, PhD, director of the Australian Animal Health Laboratory, in Newcomb, Victoria.

Then the flu virus popped up a few days later at an equestrian event 100 miles away in Maitland, New South Wales. With spring's emergence in the Southern Hemisphere, riders and horses were heading into the competition season in enthusiastic droves. And the virus took full advantage of that movement. Horses that had attended the Maitland event were falling sick in their home stables, spreading the virus to their stablemates, which were transported to other events before owners realized the risk. Equine influenza spread through NSW and south into Victoria like a bushfire.

What happened? How could this country in the middle of an ocean that hadn't encountered equine influenza in recorded history suddenly have such widespread disease in less than two weeks? Where did it come from? And how did it get out of quarantine?

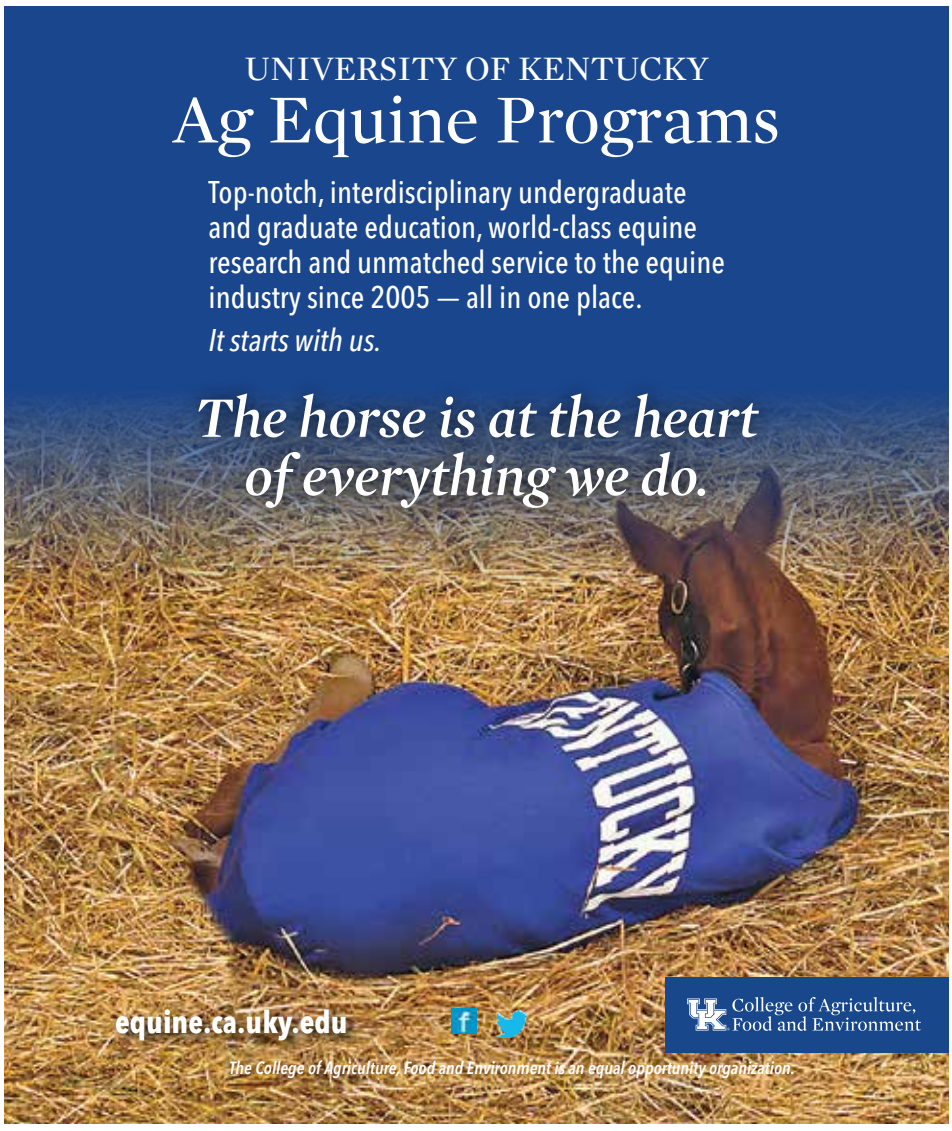
Those are questions researchers investigated for years. Their best guess is the virus was imported with Japanese horses—since Japan had its own epidemic (its first since 1977) at the same time. If the horses imported from Japan in August 2007 were infected, they would have picked up the virus about a week before Japanese officials discovered the epidemic. But it's also possible the virus came into Australia with American horses, because the Japanese flu epidemic came from horses imported from the U.S. into Japan. Either way, even if the Japanese and U.S. horses were vaccinated according to protocol, they could have picked up a strain the vaccine didn't prevent, Jeggo says.

"Grievous" biosecurity breeches—such as a farrier not washing his equipment between horses—could have led to the disease's spread within the quarantine facility and beyond, says Debra C. Sellon, DVM, PhD, Dipl. ACVIM, director of the Veterinary Teaching Hospital at Washington State University, in Pullman.

Australian authorities did blame biosecurity oversights within the Eastern Creek facility for the outbreak after comparing that facility to the country's other quarantine station, where the flu virus was contained. Horses there had been exposed to the virus, as testing showed, but did not fall ill or spread the disease. However, researchers were never able to determine exactly how the virus got out of the Eastern Creek quarantine station.

What followed was mobilization of the largest animal health emergency effort in Australian history. As soon as veterinarians diagnosed the first horses from the Maitland show, authorities shut down all equine movement. Wherever horses were, they stayed—even if they were away from home. Absolutely zero movement was allowed, with strict enforcement.

"Extraordinary efforts were taken to contain the outbreak ... and to prevent it from reaching the wild Brumby population," says Anne Jackson, MA, VetMB, PhD, MRCVS, of the Australian Veterinary Association. "Drivers of horse floats or small trucks who seemed to think that the movement restrictions either didn't



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apply to them or that a quick trip home into Victoria wouldn't be noticed, were highly surprised to find that all 32 border crossings at the Murray River between NSW and Victoria were manned 24/7 with armed guards. Nothing equine was going to cross that river."

Meanwhile, the country initiated a vaccine campaign, inoculating 140,000 horses in an emergency effort. As the world's horse industry watched, wondering if Australia would ever recover its flu-free status, veterinarians and authorities kept going full force—quarantining, enforcing movement restrictions, vaccinating, tracing, keeping records, washing and sterilizing, and educating.

Their program worked. By December 2007, just four months after the first case diagnosis, disease spread had ceased. In February 2008 the World Organization for Animal Health (OIE) officially declared Australia once again free of equine influenza. In all, the disease had hit 10,000 properties, infecting around 50,000 horses and covering 100,000 square miles.

"This epidemic led to real lessons in biosecurity," Sellon says. "People weren't paying attention to the details. It's phenomenal to see how just little mistakes in biosecurity can impact so many horses in the entire country. And the tiniest little lapse can cause this kind of economic devastation to an industry. When they were in the middle of that epidemic, I honestly had my doubts as to whether they would get it eradicated again, but they did. That was a pretty amazing effort."

Equine Viral Arteritis in North America

Scientists at the University of Kentucky's (UK) Department of Veterinary Science, in Lexington, first isolated the equine arteritis virus (EAV) in 1953 after an extensive outbreak in Ohio that caused mares on a Standardbred breeding farm to abort and other horses to develop respiratory disease. The disease—equine viral arteritis (EVA)—subsequently spread to farms in Pennsylvania, California, and Kentucky. However, it attracted little attention nationally or internationally for 31 years, says Peter Timoney, PhD, FRCVS, professor and former department chair and director of UK's Gluck Equine Research Center and an OIE-recognized expert on EVA.

In 1984 EVA mysteriously resurfaced, this time in Central Kentucky. The virus appeared on a Thoroughbred breeding farm housing 17 stallions, though no one has ever figured out where it came from. All 17 stallions became infected—some with severe clinical signs—but none died. However, the disease spread via the stallions' semen to 41 Thoroughbred breeding farms throughout the region. The industry was hit hard, with hundreds of sick horses and aborted foals that season.

"This outbreak resulted in an unprecedented response from the international horse community and national veterinary authorities around the world," says Timoney. "The uncertainty over how far this infection would spread led to a unique action, taken by France, the U.K., and Ireland, to impose an embargo on the import of all horses from the U.S. into their countries, until the industry in Kentucky had the opportunity to bring the situation under control."

One month later the countries lifted the embargo. "But that was succeeded by some of the most stringent import requirements seen yet in the international breeding industry, and other countries followed suit," he adds.

Until then, the only country that required EAV testing was Japan, as it had tested its horse population in the 1960s and found it to be entirely seronegative for the virus. Timoney says it took the 1984 outbreak to make international breeders impose strict requirements for EAV testing in imported horses.

This outbreak was a landmark event for another, equally significant reason. It was the first time researchers understood that stallions could become carriers of the disease.

"We tested a significant number of Standardbred stallions between 1984 and 1998 and found that 69% were seropositive for antibodies to the virus," Timoney says. "Where it was possible to obtain sequential semen samples over time, it was found that the majority were long-term carriers. Some were likely lifelong carriers."

There's no treatment for the carrier status, aside from castration.

"This event helped us understand the significance of the carrier state and importance of the carrier stallion as the reservoir of EAV," he says. "We're talking about the ability of the long-term carrier animal, showing no clinical signs of disease, to serve as a highly effective means of dissemination of the virus during a breeding season, and also to ensure its perpetuation for years and years."

Infectious Agents as Weapons of War

Horses provided critical transportation and force in human battles in prehistoric times up until the past 100 years. Their importance to the outcome of war made them prime targets for enemy sabotage. In 20th-century warfare, in addition to sinking transport vessels filled with horses, opposing armies tried to obliterate equine "soldiers" by purposefully infecting them with disease. The most popular agent for this infectious destruction was *Burkholderia mallei*, the bacterium responsible for the upper respiratory disease glanders.

"The use of *B. mallei* was one of the first documented cases of biological warfare, and it was targeted against horses," says Debra C. Sellon, DVM, PhD, Dipl. ACVIM, director of the Veterinary Teaching Hospital at Washington State University, in Pullman.

The Germans first isolated the infectious agent in 1882, in hopes of finding a cure for the disease. But a little over 30 years later the country's military was allegedly using that scientific knowledge to spread glanders among Russian horses and mules on the Eastern front during World War I. Meanwhile, a U.S.-born German scientist, Anton Dilger, supposedly hired dockworkers to infect U.S. horses with the agent as they were being loaded into ships taking them overseas to war.

There's little information about these efforts' effects or how far the "epidemic" stretched—if it even resulted in an epidemic. However, it apparently inspired similar attacks. The Japanese were believed to have purposefully infected horses (as well as human prisoners of war) with *B. mallei* at the Pinfang Institute, in China, during World War II.

"This kind of epidemic is just an entire class apart from other epidemics, because it's intentional and targeted at an innocent animal pulled into the work of humans in their battles against each other," Sellon says. "It's just a horrible thing to do. And worse, it's propagated by medical scientists. How could someone trained in medicine have so little respect for life?

Because of this discovery, the OIE made EVA "one of its most regulated diseases," Timoney says.

A subsequent outbreak at the Arlington Park racetrack, in Chicago, in 1993 exposed more than 1,600 horses to the disease, resulting in at least 200 clinical cases, he says. "What was significant about that outbreak was that, unlike the 1984 outbreak that was spread via the venereal route, this outbreak spread almost exclusively via the respiratory route."

Equine viral arteritis hit the North American breeding industry on a large scale once again in 2006, when one of the most sought-after Quarter Horse sires contracted the disease in New Mexico. "Within two to three weeks, the virus had spread through shipped fresh-cooled semen to 18 American states, as well as Canadian provinces," Timoney says. "That event was associated with clinical outbreaks of EVA, outbreaks of abortion, deaths in neonatal foals, and exposure and infection of stallions, a percentage of which became long-term carriers. It was an event of major importance and significant economic impact."

Contagious Equine Metritis in Ireland

In the 1970s in Ireland, the Thoroughbred horse racing industry was in full swing, having developed out of a long and rich cultural history of gallop and hunt racing that began more than 2,000 years ago. But starting in 1975, Thoroughbred mares on an Irish stud farm developed signs of a venereally transmitted disease. They had purulent (containing pus) vaginal discharge, reproductive tract inflammation, and temporary infertility.

Researchers sought an explanation for a disease that seemed very different from other known infections of the mare's reproductive tract. They collected endometrial swabs and cultured them in standard laboratory conditions. But nothing revealing grew out of those cultures. For two years the industry struggled with this curious infection, which became known as contagious equine metritis (CEM), all the while spreading it unknowingly within Ireland and the U.K. and introducing it into Australia.

"The main infectious agent that showed up on cultures was *Proteus mirabilis*, a very common environmental contaminant," Timoney says. *Proteus* is an opportunistic bacterium that can cause urinary tract infections in humans and horses. But it likely wouldn't cause the discharge and infertility these breeders were dealing with.

It took the insight of a sexually transmitted disease specialist (Dr. Eddie Taylor, in Cambridge, U.K.) to recognize the similarities between the mares' signs and those of women suffering from gonorrhea. "What was especially telling was that it was the mare that was infected and became clinical (showed signs of disease) following exposure to the bacterium, but not the stallion," Timoney says. "The stallion was an asymptomatic carrier of whatever the causal agent was."

So Taylor tried a different culture technique, using "more enriched media" for the genital swabs and an environment that included 5 to 10% carbon dioxide, in hopes of bringing out a different bacterial growth. The outcome was a game changer, says Timoney. Under such conditions a small, delicate bacterium grew on cultures from clinically affected mares in three to four days. "The true cause of CEM was identified and subsequently classified as *Taylorella equigenitalis*, in recognition of the person who first discovered it," says Timoney.

Targeted antibiotic treatment cured the mares easily. In 1978 the Thoroughbred breeding industries in the U.K., France, and Ireland developed a Code of Practice to prevent and control CEM, which was eliminated from those countries' Thoroughbred breeding populations within several years.

African Horse Sickness

Deadly African horse sickness (AHS) devastates working equids every year. Endemic in sub-Saharan Africa, it has mortality rates as high as 95% in naive populations. Vaccines exist; treatments don't.

The AHS virus spreads via biting midges that thrive in the sub-Saharan climate—a fact that, for a long time, reassured researchers that AHS was unlikely to become established in Europe. We now know that's not true due to factors such as climate change and the globalization of animal movement and trade. But even before this became apparent, a devastating AHS outbreak in southern Spain alerted scientists to a frightening reality: It's not just African midges that can carry the virus.

"The transmission did not just occur with its regular midges from Africa; it looks like there were other species, more common in Northern Europe, that were involved," says Simon Carpenter, PhD,

How to DNA Test Through a Breed Registry

Q I have a horse who is said to be registered but, unfortunately, during Hurricane Harvey those papers were lost due to flooding in our area. The previous owners do not remember the registered name given, nor the parents, but I was informed that I could further my knowledge about her bloodlines through a DNA test. Is this true? And if so, could you explain how this works and what I would need to do?

Lauren Castilaw
Via email

A I am sorry for your difficulties due to the hurricane. Many horse registries require DNA testing prior to registration to verify parentage before issuing papers. If this is the case for your horse's registry, then contact the registry and explain your situation. Each registry has a contract laboratory that does all their DNA testing and maintains a database of the DNA profiles of horses submitted to the lab. If the registry is not willing or able to reissue the papers based on ownership information, then they will likely require you to submit a hair sample from your horse to the laboratory to generate a DNA profile again. The lab can then search that registry's database for a matching DNA profile and thereby identify your horse. If your horse was not DNA-profiled prior to registration, then unfortunately there is little that can be done to verify the identity of your horse with complete certainty.

Kathryn T. Graves, PhD
Associate Clinical Professor, Genetic Testing at Gluck Equine Research Center
University of Kentucky, Lexington, Kentucky

head of the Entomology Group at the U.K. Biotechnology and Biological Sciences Research Council Pirbright Laboratory, in Woking, Surrey.

It happened when a central Spanish zoo received a shipment of imported zebras from Africa in 1987. The zebras had likely been recently bitten by infected midges and were carrying the virus when they entered Spain. But unlike horses, zebras show no clinical signs of infection. They don't get sick, and they don't die. They just act as healthy reservoirs, with midges feeding on their blood, then transmitting the virus to susceptible horses, which do get sick and die.

Shortly after the zebras arrived, Spanish riding horses in the area started getting seriously ill and dying. Their owners were shocked and devastated, helplessly watching their horses struggle with painful clinical signs ranging from pulmonary distress to heart failure and die as foam discharged from swollen lungs through the horses' nostrils. Veterinarians were confused, distraught, and frustrated, unable to cure or even treat their clients' show horses and beloved companions.

"While the disease was identified relatively rapidly, most vets had no experience in combating the virus and had no idea what they were up against," Carpenter says. "The main hope was that AHS would die out over winter, as it had done in previous outbreaks, although there was also a concerted vaccination campaign."

Authorities managed as best they could

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In 1987 the African horse sickness outbreak in Spain was linked to a shipment of infected zebras.

under the circumstances, he says, holding out for when, theoretically, the midges would die or hibernate through the cold season.

Despite that outbreak being quite localized and more than 38,000 horses being vaccinated in 1987, AHS re-emerged in 1988 far to the south of the initial cases. "This may have been a consequence of a horse movement and then local transmission," Carpenter says. "It's a very visual disease, lots of hemorrhaging, very traumatic to see, and the first thing an owner wants to do is get his horse as far away from the disease as possible. But if their horse was already exposed and just wasn't symptomatic yet, he was carrying that virus to wherever his owner tried to take him, spreading it more."

African horse sickness reached Portugal and Morocco that year, bringing the total number of horses killed to approximately 2,000 by winter.

Despite vaccination, it took two more years—and cost the lives of 3,000 Spanish horses—but Spain finally eradicated AHS from its territories. "The real lesson of the outbreak was to realize that, contrary to what we thought, virus could overwinter consistently, over extended periods of time," says Carpenter. "Even with effective vaccines, these outbreaks were incredibly challenging to deal with.

"This outbreak gave the worldwide veterinary community a real lesson in the importance of recognizing diseases that aren't typical to your home area," he adds. "Certainly in 1987 the number of people in Europe with experience in AHS was limited, and that led to serious limitations in our efforts to control it."

Take-Home Message

Major equine disease outbreaks are extremely rare. But when they happen, they serve as unforgettable lessons in biosecurity and disease management. Prevention includes strict adherence to vaccination, screening, and disinfection protocols. Containing the outbreak requires good communication and education in local—as well as foreign—diseases.

"You don't want to be the veterinarian that saw that case and didn't recognize it for what it was," says Sellon. UK

>Christa Leste-Lasserre, MA is a freelance writer specializing in equine topics. A native Texan, she rides English, breeds Warmbloods, and raises three young equestrians in Paris, France.

Fall Nitrogen Benefits Pastures Year-Round

Fall is a critical time to invest in pastures to protect them throughout the winter and ensure good grazing in the spring. Most cool-season horse pastures should be fertilized with nitrogen in the fall to boost root reserves and extend the grazing season. Other fertilizers can also be added in the fall, as directed by a soil test.

Fall Nitrogen

Nitrogen applied in the spring or summer provides a significant boost to grass growth, but many farm owners can't use this additional growth and instead mow it down. Consider applying nitrogen in the fall to cool-season pastures instead. Fall nitrogen will not greatly increase grass growth, but it will boost the pasture grasses' root reserves, allowing plants to remain greener longer into winter, survive winter better, and green up sooner in the spring. This effectively prolongs the grazing season.

Additionally, a strong spring pasture will have better cover, thereby reducing annual weeds. Apply 40 to 50 pounds of nitrogen per acre to pastures once or twice in the fall. This equates to 85 to 105 pounds per acre if using urea (46-0-0). Applications can occur anytime between September and the first hard freeze (an overnight temp lower than 20 degrees Farhenheit) and should be at least six weeks apart. You can

COURTESY KRISTA LEA

Nitrogen and some other fertilizers can easily be spread using a cone seeder on the back of an ATV.

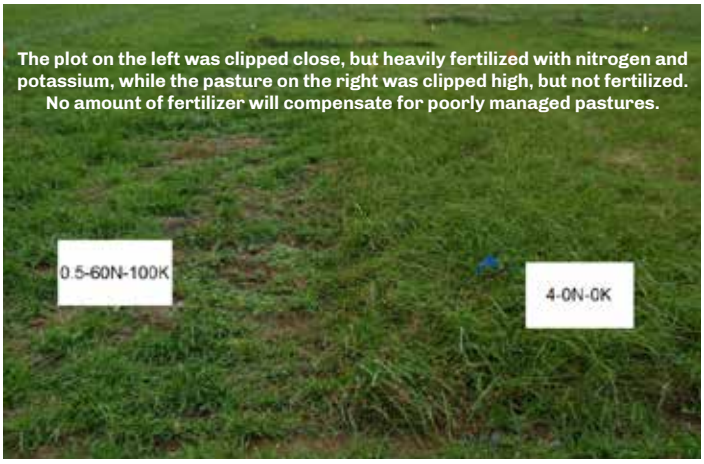
spread nitrogen fertilizers easily using a cone seeder on the back of an ATV or a tractor. You shouldn't have to remove horses from pastures if equipment is operating properly and not leaving large piles of pellets. Do not fertilize when grass leaves are wet, as fertilizer pellets can stick to the leaf surface and damage it.

Other Fertilizers

Unlike nitrogen, other soil amendments such as phosphorus, potassium, and lime should be applied only after a soil test. Apply only the recommended amounts, as additional inputs are expensive, do not benefit the pasture, and can run off into nearby bodies of water. While mixed fertilizer bags such as 10-10-10 or 19-19-19 are convenient, they might not allow full fertilization of one component without overfertilizing another. Contact your local county Extension agent for soil sampling.

Good Management

While maintaining good soil fertility is essential to having productive pastures, so is good management. No amount of fertilizer can compensate for overgrazed pastures. Fall is also a great time to assess how well pastures performed throughout the year and make plans for next year. Rotating horses between two or more pastures allows grasses to rest and will ultimately result in greater production and fewer weeds. UK



The plot on the left was clipped close, but heavily fertilized with nitrogen and potassium, while the pasture on the right was clipped high, but not fertilized. No amount of fertilizer will compensate for poorly managed pastures.

COURTESY KRISTA LEA

>Krista Lea, MS, research analyst and coordinator of UK's Horse Pasture Evaluation Program within the Department of Plant and Soil Sciences; and Ray Smith, PhD, professor and forage extension specialist within UK's Department of Plant and Soil Sciences, provided this information.

Clostridial Myositis

Clostridial myositis is a rare but serious bacterial infection, which causes inflammation and death of muscle and release of bacterial toxins into the bloodstream. This condition is also referred to as myonecrosis, malignant edema, and gas gangrene. It occurs most often in horses that have recently received an intramuscular injection. Clinical signs appear six to 72 hours following the injection, and horses initially exhibit acute swelling, heat, and pain of the affected area. The disease progresses rapidly and the horse's condition may decline within hours. The affected animal exhibits signs of systemic toxemia; death can supervene rapidly in severe cases. Clostridial bacteria produce gas that results in a characteristic emphysematous (bubbly) feel or crepitation of the region. Clostridial myositis is a true medical emergency, with survival linked to prompt intervention through aggressive antibiotic treatment and wound debridement.

Clostridial myositis is overrepresented in Quarter Horses, possibly due to their heavy muscling.

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The Clostridium genus consists of over 150 known species of Gram-positive, anaerobic, sporeforming bacteria. The spore-forming ability of these bacteria allows survival for long periods of time in the environment. When spores encounter a location without oxygen, such as damaged muscle, they are triggered to proliferate and produce exotoxins, which cause extensive tissue and vascular damage. The clostridial species that commonly cause myositis include *C. perfringens, C. septicum,* and *C. chauvoei*.

Clostridial myositis has been reported following intramuscular inoculations of vaccines, ivermectin, antihistamines, phenylbutazone, vitamins, prostaglandins, and most commonly, flunixin meglumine. Infrequently, cases occur following inadequate perivascular administration of compounds, foaling trauma, or puncture wounds. In a study by Peek et al. in 2003, stallions and Quarter horses were overrepresented, and the authors hypothesized this might be due to the heavy muscling of these groups.

The mechanism by which the bacterial spores arrive in the muscle of the horse is unknown. It is possible spores are introduced at the time of injection. Another theory is that bacteria are translocated from their normal environment in the intestine, in times of inflammation or colic and arrive in the muscle via the bloodstream. No association linking whether or not the injection site is cleaned prior to injection and the development of myositis has been established. A higher incidence of myositis with irritating substances such as nonsteroidal drugs and vitamins is reported, potentially due to increased tissue damage and

CLOSTRIDIAL MYOSITIS

creation of an oxygen-free environment.

Diagnosis involves aspirating a small amount of fluid for anaerobic culture and Gram-staining to look for the presence of Gram-positive rods. To treat the infection, large incisions are made into the muscle and fascia to expose the bacteria to oxygen and debride dead tissue. General supportive care is critical because these bacteria produce toxins that have secondary effects on the horse, including the potential to reduce the contractility of the heart. Clostridial toxins may also result in anemia, thrombocytopenia, and leukopenia. Horses are commonly treated with high doses of intravenous penicillin, intravenous fluids, cardiovascular support, and wound care. Hyperbaric therapy, where available, is suggested as an adjunct to routine treatment.

Survival has been reported to range from 31% to 73% and appears to be better for infections with *C. perfringens* compared with *C. septicum* or *C. chauvoei*. Horses that survive the initial toxemic stages of disease have an improved prognosis. The wounds created by a combination of infection and treatment are usually large, and may take weeks to months to heal entirely. Horses which do not survive show signs of intravascular coagulation and multi-organ failure.

There is no definitive prevention for clostridial myositis. When giving intramuscular injections, use large and well-vascularized muscle groups, and when possible avoid giving irritating substances in the muscle if there is an alternative route such as oral or intravenous administration. UK

CONTACT:
Rebecca Ruby, BVSc (dist), MSc, Dipl. ACVP
Rebecca.Ruby@uky.edu
(859) 257-8283
UK Veterinary Diagnostic Laboratory
University of Kentucky

This is an excerpt from Equine Disease Quarterly, funded by underwriters at Lloyd's, London.

UK Animal Scientists Bring Home Honors

Several faculty members and students in the University of Kentucky College of Agriculture, Food and Environment's Department of Animal and Food Sciences recently earned national awards from the American Society of Animal Sciences (ASAS).

"We are very proud of the animal and food sciences faculty and students we have at UK," said department chair Richard Coffey. "They continue to excel in research, teaching, and extension. Cutting-edge research and excellent creative teaching methods are the norm here, and our students and producers are benefiting from that."

One of those awards—the ASAS Distinguished Teacher Award—went to Bob Coleman, PhD, extension equine professor. Coleman has been at UK since 1998. He teaches and advises students in the equine science and management program and is the advisor for the UK Equestrian Team. The award, sponsored by Purina Animal Nutrition LLC/Land O' Lakes Inc., recognizes a dedicated individual's teaching accomplishments in animal sciences courses. ASAS said Coleman is genuinely invested in his students and shows interest in student motivation, mastery of subject matter, teaching improvement, extracurricular student activities, and service to the agricultural industry. UK

>Aimee Nielsen is an agricultural communications specialist in UK's College of Agriculture, Food and Environment.

PHOTO CREDIT

Dr. Bob Coleman won the ASAS Distinguished Teacher Award for his investment in his students and service to the agricultural industry.

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