BLUEGRASS EQUINE DIGEST

Brought to you by the UK Equine Initiative and Gluck Equine Research Center

Welcome to the Bluegrass Equine Digest

Welcome to the first issue of *Bluegrass Equine Digest*, a new monthly electronic newsletter dedicated to providing free up-to-date information on equine research from the University of Kentucky's College of Agriculture.

Bluegrass Equine Digest brings together several entities, as content is brought to you by the UK Equine Initiative and Gluck Equine Research Center and the e-newsletter is brought to you by TheHorse.com and sponsor Pfizer Animal Health.

This newsletter will feature equine news, tips, and research findings from UK's College of Agriculture, including the Gluck Equine Research Center, the Livestock Disease Diagnostic Center, and a wide range of departments at UK that conduct equine research.

The *Bluegrass Equine Digest* will include short news items that will bring you up-to-date information on happenings in the research labs and from the field, plus in-depth articles from world-renowned experts to help you understand current "hot" topics. There will also be tips to help you better care for your horses and your property, and business advice from a team of experts.

Each month's newsletter, which will be e-mailed at the end of the month, will include updates on some or all of the following: people, places and things, research and lab highlights, infectious disease and immunology, reproduction, pathology, musculoskeletal, pharmacology/therapeutics/toxicology, genetics and genomics, parasitology, agricultural economics, nutrition and animal science, biosystems and agricultural engineering, entomology, landscape architecture, plant and soil sciences, community and leadership development, human environmental sciences, and a calendar of events.

TheHorse.com will also produce a printable PDF each month to accompany the monthly e-newsletter.

For more information on Gluck Equine Research Center visit www.ca.uky.edu/gluck, and for more information on UK's Equine Initiative visit www.ca.uky.edu/equine.

If you received this newsletter from a friend you can sign up now at TheHorse.com/Enewsletter/ by checking the box for *Bluegrass Equine Digest* at the right side of the page and enter your name and e-mail address to receive this monthly electronic newsletter for FREE.

ARTICLES OF INTEREST

Early Embryonic Loss and Genetics

TERI LEAR

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

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

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

Chromosomal translocations in horses have been rarely documented and all have caused repeated early embryonic loss (REEL). REEL can be quite costly due to additional costs for veterinary care, boarding, and transportation. Often an entire breeding season is lost. Until recently, only two equine chromosomal translocations had been described in literature. The first case was a mare that produced only two foals in seven years. The second was a stallion with a high incidence of early embryonic loss in the mares to which he was bred.

Recently, we identified different translocations in four mares experiencing REEL. While the reproductive history for each mare is somewhat different, all four mares lost embryos multiple times over multiple years and always prior to Day 65 of gestation. All four mares had normal reproductive tracts and estrous cycles. Once in foal, the mares

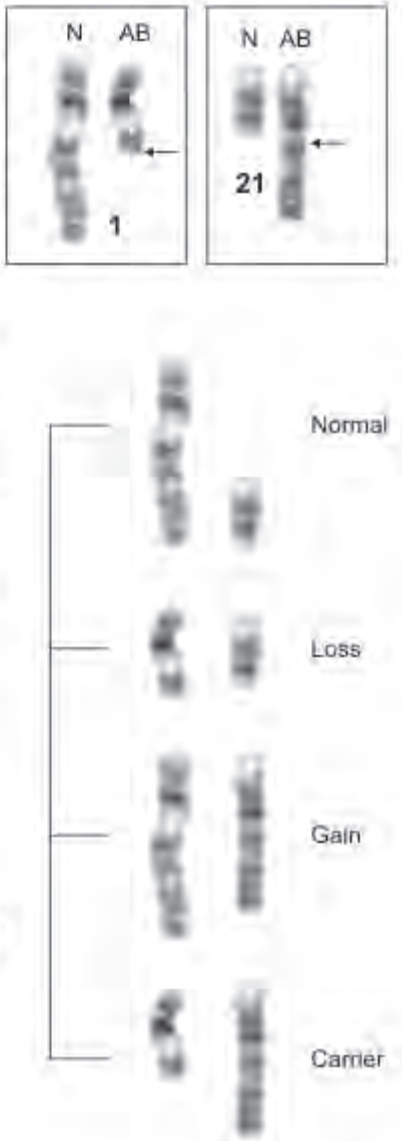

Figure 1. A chromosome translocation involving horse chromosomes 1 and 21 that causes repeated early embryonic loss. The normal chromosomes are on the left (N), and the abnormal chromosomes are on the right (AB) of each pair (boxes at top). Chromosome 1 has broken (arrow) and reattached to the bottom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

(EARLY EMBRYONIC...)

had normal ultrasound exams between Days 14 and 21. However, in some mares the embryo's heartbeat was lost by Day 28.

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

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

Samples may only be submitted by veterinarians. An equine practitioner may contact Lear at equigene@email.uky.edu; or 859/257-4757, ext. 81108. UK

Teri Lear, PhD, is a research associate professor in molecular cytogenetics at the Gluck Equine Research Center.

Reprinted from Equine Disease Quarterly April 2009, University of Kentucky, College of Agriculture, Department of Veterinary Science.

Visiting Scientist Builds Parasitology Bridges

JENNY BLANDFORD AND MARTIN NIELSEN

For six months, Martin K. Nielsen, DVM, was a visiting scientist at the Gluck Equine Research Center from the University of Copenhagen in Denmark through the Albert and Lorraine Clay Research Fellowship award. Nielsen, an assistant professor in the Department of Large Animal Sciences at the University of Copenhagen, began his stint at the Gluck Center in November 2008 and returned home in May 2009, but he built a bridge of cooperation between the two universities.

Nielsen

"The Clay Fellowship plays an important role in bringing scientists together, which allows for generating new ideas and building networks," Nielsen said. "I am deeply grateful for the opportunities I was given because of the Fellowship.

"Upon my return to Denmark, it has become clear that my time in Kentucky has been exceptionally fruitful," Nielsen added. "My own project is only a part of this. What strikes me is the wonderful research environment at the Gluck, where research ideas easily arise and develop."

After returning to Denmark in May, Nielsen submitted an article highlighting the research projects he conducted during his time at Gluck, including a two-part project on polymerase chain reaction (PCR) detection of DNA from the bloodworm *Strongylus vulgaris* in the bloodstream of horses and an on-farm survey of *S. vulgaris* and other strongyles in horses in Denmark and the United States. This research, designed to simplify detection of bloodworms via a blood test, was performed in collaboration with UK's Dan Howe, PhD.

PCR Detection of *S. Vulgaris* DNA in the Bloodstream

S. vulgaris spends about four months in the bloodstream of horses, and the intention was to attempt to detect DNA from this parasite in blood samples from naturally infected horses.

Necropsies were performed on two horses from the herd of Gene Lyons, PhD; this herd, kept on the university farm, has not been treated for parasites. Both of these horses had high numbers of arterial larvae of *S. vulgaris*. In addition, blood samples were collected from other horses where the *S. vulgaris* prevalence was known to be 100%.

DNA was extracted from serum and whole blood using a Qiagen blood and tissue kit.

Straight PCRs did not yield positive signals from any of the horses tested. Therefore, a nested PCR approach was attempted. For that, strongyle DNA was first amplified using a broad primer set targeting the whole ITS-2 region. In the second round, real-time PCR was performed with a *S. vulgaris* specific primer/probe set previously developed. With this approach, *S. vulgaris* DNA could be detected from heavily infected animals, but unfortunately, repeated analyses from the same horse were not consistent and other horses in the herd remained negative on testing.

In conclusion, DNA of *S. vulgaris* cannot reliably be detected by PCR in blood samples. The reason for this is that *S. vulgaris* is not a circulating pathogen and DNA released by this parasite becomes dilute in the large blood volume of horses.

Based on the experience generated from this endeavor, a new research approach was decided with Howe. The new project involves creation of cDNA libraries and immunoscreening for identification of candidates for diagnostic targets in an antigen-capture approach. At present, RNA has been extracted from both migrating L4 and adult stages of both *S. vulgaris* and *S. edentatus*. These preparations are kept on stock in Howe's laboratory for further processing.

"The intention is to recruit a graduate student for this project," Nielsen said. "Separate funding will be applied for. The student will be based at the University of Copenhagen, but will spend a longer period in Dr. Howe's laboratory to receive the training needed." Several publications are expected to result from the research project.

Survey of Worms on U.S., Danish, and Swedish Farms

The second part of Nielsen's project was to collect fecal samples from horses on farms around Lexington, Ky. The criteria for inclusion were that horses had not been treated for at least six weeks prior to sampling. Fecal egg counts were generated from every horse, and eggs were isolated for subsequent DNA extraction and PCR analysis. In addition, information about the deworming schedule was obtained on each farm. With help from Lyons, about 500 samples were collected. Similar numbers of samples will be collected in Denmark and Sweden.

All samples will be shipped to Denmark, where they will be analyzed with a real-time PCR assay Nielsen previously developed and validated for detection and quantification of *S. vulgaris* DNA in fecal samples.

The end results of the two-part project will be published in scientific journals.

Future projects also arose during Nielsen's visit with researchers at the Gluck Center and other universities. Research projects include generating a spreadsheet of data on the relationship between worm and egg counts, analyzing data evaluating cytokine expression and fecal egg counts in response to anthelmintic treatment, a study evaluating the potential genetic background for horses consistently shedding low parasite egg numbers, and the use of novel DNA/RNA sequencing techniques for studying mechanisms of anthelmintic resistance in equine nematodes. UK

Martin K. Nielsen, DVM, collaborated with the Gluck Center while he was visiting from the University of Copenhagen. Jenny Blandford is the Gluck Equine Research Foundation Assistant at the Gluck Center.

Articular Cartilage Maturation in Foals

JAMIE MACLEOD

During a three-year study on articular (joint) cartilage maturation, researchers at the Gluck Equine Research Center used new genetic techniques to investigate how joint cartilage changes between newborn foals and young adult horses.

It is well-understood that bone structure remodels during the first few years of life and as a horse initiates strenuous exercise. However, similar types of changes in the structure and function of joint cartilage are not widely appreciated and were the focus of this study led by Michael Mienaltowski, DVM, and Jamie MacLeod, VMD, PhD.

Transcriptional profiling was used to evaluate changing patterns of gene expression reflecting cartilage growth and development. Transcriptional profiling is a new technique in

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biomedical research that allows scientists to look at the expression of all the genes concurrently as a group. For an analogy, transcriptional profiling would be like looking at the whole forest, or all of the genes, before focusing on a single tree, or a single gene.

In previous studies, evaluating many thousands of equine genes concurrently was not possible due to a lack of the critical technical resources for horses. By looking at the entire gene expression profile, scientists are now able to get a broader perspective of functional changes in cartilage or any other horse sample.

Seven newborn foals and nine 4- to 5-year-old adult horses were compared in the study. The results demonstrated substantial changes in the function of cartilage cells between newborn foals and young adult horses. Research from other scientists has indicated that normal early activity in a foal, such as running around in a paddock, is important to the cartilage maturation process. When observing joint cartilage tissue in foals and young adult horses, it looks very similar on the outside. However, patterns of gene expression within cartilage cells and the structural organization of cartilage proteins indicate that important changes occur during the first few months after birth.

Articular cartilage is a critical tissue in synovial joints, essential for joint movement, and necessary for the dissipation of mechanical forces placed on a horse's joints during physical activity. As knowledge is accumulated about the substantial changes that joint cartilage goes through early in life, scientists will need to focus on how these changes impact joint health in adult horses, including susceptibility to important diseases like osteoarthritis. Joint disease is the most common cause of lameness and shortened careers in equine athletes. UK

Jamie MacLeod, VMD, PhD, is the director of UK Equine Initiative and Dickson Professor of Equine Science and Management and the John S. and Elizabeth A. Knight Chair at the Gluck Center.

ENVIRONMENTAL IMPACT OF EQUINE CARCASS DISPOSAL STUDY UNDER WAY

EMILY PLANT

As with all large animals, disposal of horse carcasses in an environmentally safe manner requires planning and resources. Since horse slaughter facilities have been outlawed in the U.S., the problem of disposal is in the public eye.

In June 2008 the United States Department of Agriculture held a national forum to discuss the issue of the unwanted horse. Unwanted horses may be sick, injured, old, unmanageable or dangerous. They may be horses the owner is no longer economically able to care for or horses that no longer meet the owner's expectations. Owners may try to find a new home for the animal. This might not be an option, however, and euthanasia may be the chosen method of disposal. In addition to the animals owners have to euthanize, owners must also handle animals that die from disease or old age. Each year, more than 200,000 equine carcasses must be disposed of in the United States. The question becomes how to dispose of the carcasses?

Research at the University of Kentucky's Von Allmen Center for Green Marketing will first consider the six predominant methods currently available for disposing of livestock carcasses: incineration, burial, rendering, landfill, composting, and alkaline hydrolysis. Each method considered presents a different level of environmental impact. If the animal is contaminated with a disease that might infect other animals or humans, that carcass poses serious concerns for the risk of contamination.

The problem of carcass disposal is not limited only to equines—all commercial animal productions must deal with the decision of how they will dispose of carcasses. Human burials also have an enormous impact on the environment. Each year in the United States, approximately 30 million board feet of hardwoods (much of which is sourced from rainforests), 104,272 tons of steel, 2,700 tons of copper and bronze, and 1.636 million tons of reinforced concrete are buried in the 2.2 million acres occupied by cemeteries. Additionally, 827,000 gallons of embalming fluid (primarily formaldehyde, which is a highly toxic substance) are used to prepare bodies for burial. Binders, glues, stains and varnishes are used to produce and finish caskets, all of which are placed permanently into the ground. These

(CARCASS DISPOSAL...)

materials are detrimental to the environment, leaching dangerous chemicals into the soil and water.

The problem of how to dispose of potentially harmful materials extends far beyond the area of dealing with living creatures at the end of their lives. The average household generates 100 pounds of chemical waste every year, including fertilizers and pesticides, flammables, paint, old batteries, light bulbs, and aerosols. These chemicals can have a devastating impact on the environment, poisoning wildlife and polluting the soil so plant life cannot grow.

By examining the problem of equine carcass disposal, this research will provide insight into how marketers can deal with the problem of lessening the impact of harmful materials on the environment by exploring how more environmentally sound disposal methods can be encouraged.

The author acknowledges the support of University of Kentucky's Von Allmen Center for Green Marketing. For more information on the center, visit http://gattongreen.uky.edu.

This research is under way and will be a complete draft by December. Emily Plant is a third-year doctoral candidate in the Gatton College of Business and Economics, Marketing Department. She is a member of the Equine Initiative student working group and active in Equine Initiative activities. UK

ASC-173 BOTULISM: A DEADLY DISEASE THAT CAN AFFECT YOUR HORSE

FERNANDA C. CAMARGO, BOB COLEMAN, LAURIE LAWRENCE, DEPARTMENT OF ANIMAL SCIENCES

Botulism is a deadly disease caused by the toxins produced by the bacterium *Clostridium botulinum*. The botulinum toxin is a potent neurotoxin that impairs nerve function, including those of the diaphragm, leading to paralysis. When the nerves to the diaphragm are paralyzed, the affected animal stops breathing and will die as a result.

C. botulinum is an anaerobic (lives without oxygen), Gram-positive, spore-forming bacterium. The toxin produced by *C. botulinum* is one of the most potent toxins known to science. The *Clostridium* family is known for causing rapid, severe, and deadly diseases, including botulism, tetanus, blackleg, malignant edema, etc. Sometimes the onset of clostridial disease is so rapid that no clinical signs are noted, and the owner simply finds the animal dead.

The botulism toxin blocks the release of acetylcholine in the neuromuscular junction. Acetylcholine is a neurotransmitter, which transmits information from the nerve cell to the muscle cell. It is released in the neuromuscular junctions (the space where nerves communicate and stimulate muscles), and it enables muscle contraction; therefore, without acetylcholine, the muscles will not contract. A very important muscle in any mammal's survival is the diaphragm, which, when contracted, enables breathing. Ultimately, without acetylcholine, a horse infected with botulism will die of respiratory failure and asphyxia, due to the paralysis of the diaphragm.

There are seven types of botulism recognized (A, B, Ca, Cb, D, E, F, and G), based on the antigenic specificity of the toxin produced by each strain. Types A, B, E, and F cause human botulism. Types A, B, C, and D cause most cases of botulism in animals. In horses, Type B botulism is responsible for more than 80% of the cases.

The bacterium and its spores are widely distributed in nature. They are found in soil, sediments of streams and lakes, and in the intestinal tracts of fish and mammals. The bacteria will produce toxins under conditions of decaying plants and animals.

A horse can get botulism in three ways:

1) By consuming forage or feed containing the bacteria, which will then produce the toxins in the intestinal tract (more commonly found in foals, known as shaker foal syndrome, or toxicoinfectious botulism).

2) By consuming feed or forage containing the preformed toxins of *C. botulinum* (known as forage poisoning).

3) Through wounds contaminated with the bacteria, generally puncture wounds. Wound borders will close, providing an anaerobic environment, which is a favorable condition for the bacteria to produce the toxins.

Although the incidence of the disease is low,

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it is of considerable concern because of its high mortality rate if not addressed and treated immediately and properly. The mid-Atlantic region of the eastern United States and especially Kentucky is where botulism is most commonly found, although the disease is reported worldwide. The spores of *C. botulinum* Type B can be found in the soil of most regions of the United States, although they are more frequently found in the northeastern and Appalachian regions. The western region is more abundant with *C. botulinum* Type A, and Type C occurs mainly in Florida.

Vaccination Chart: Adult Horses			
Broodmares		**Other Adult Horses** *(> 1 year of age)*	
Previously vaccinated	**Previously unvaccinated or having unknown vaccination history**	**Previously vaccinated**	**Previously unvaccinated or having unknown vaccination history**
1 dose, annually, 4 to 6 weeks prepartum	3-dose series: 1st dose: at 8 months' gestation 2nd dose: 4 weeks after 1st dose 3rd dose: 4 weeks after 2nd dose	Annual revaccination	3-dose series: 2nd dose: 4 weeks after 1st dose 3rd dose: 4 weeks after 2nd dose Annual revaccination

The Different Faces of Botulism

Forage poisoning can occur when horses eat spoiled forage or any type of feed contaminated by a decaying animal. Spoilage is common in hay that was baled with a high moisture content. In cases associated with hay, horses eating round-baled hay are at higher risk of developing the disease. Also, horses consuming hay that is spilled on the ground and then allowed to be mixed with soil and feces are at increased risk for botulism. Moreover, botulism can occur when dead animals accidentally get baled in hay during harvest. The decomposing carcass is an excellent anaerobic incubator for botulism spores present in the intestinal tract of the dead animal or bird. Once toxin is formed in the carcass, it leaches out and contaminates the hay or other feed material. Improperly ensiled silage and haylage with a high moisture content and alkaline pH (pH >4.5) provide optimal conditions for the production of botulinum toxin. Improperly fermented silage (pH >4.5) should not be fed to horses because it predictably causes botulism. In contrast, cattle are more resistant to botulism and may not develop the disease as readily as horses when fed spoiled silage, although there have been several reports of cases of botulism in cattle.

Botulism in foals, also known as shaker foal syndrome, happens when the foal starts to nibble at hay and grass, between 2 and 12 weeks of age, and ingests the bacteria directly from the soil. The bacteria will form spores in the intestinal tract of the foal and produce toxins. One interesting fact is that the normal flora of adult horses inhibit the intraintestinal growth of botulinum spores, limiting the occurrence of toxicoinfectious botulism to neonates. The toxins are absorbed into the bloodstream and cause the blockage in the neuromuscular junction, which keeps the muscles from contracting. The foal will start to show signs of difficulty in standing, shaking (thus, the name of the syndrome), and then he will fall down over and over again. The foal will not be able to suckle and will, if not diagnosed and treated in time, eventually die of respiratory failure.

Wound botulism occurs when *C. botulinum* contaminates a wound, such as a deep puncture wound, and the wound borders close, creating an anaerobic environment. The bacteria then produce the deadly toxins anaerobically. It has been seen after castration and been associated with injection abscesses, puncture wounds, trauma, and surgery in adult horses and in foals with umbilical hernias treated with clamps. This mechanism is similar to tetanus infection.

Regardless of the port of entry, the sequence of events that follow is the same for every horse. Once the toxin enters the body, it

Vaccination Chart: Foals and Weanlings *(<12 months of age)*	
Mares vaccinated in the prepartum period	**Unvaccinated mares**
3-dose series: 1st dose: 2 to 3 months of age 2nd dose: 4 weeks after 1st dose 3rd dose: 4 weeks after 2nd dose	3-dose series: 1st dose: 1 to 3 months of age 2nd dose: 4 weeks after 1st dose 3rd dose: 4 weeks after 2nd dose
Maternal antibody does not interfere with vaccination; foals at high risk may be vaccinated as early as 2 weeks of age.	

reaches the bloodstream and is distributed to nerves throughout the body. The toxin then prevents the transmission of impulses from the nerve to the muscle, impeding muscle contraction and leading to paralysis. The course of the disease is very rapid, taking only one to two days to cause death, depending on the dose of toxin or bacteria ingested.

Clinical Signs and Diagnosis

Clinical signs of botulism in horses are weakness; decreased muscle tone of the tail, eyelids, and tongue; trembling; dilated pupils; lying down; difficulty in swallowing; drooling; and green or milky nasal discharge. Foals will nurse for about one minute, start to shake, and collapse on the floor. They will not be able to swallow the milk, and the owner will notice the milk all around the muzzle of the foal or the foal drooling the milk. In the same way, adult horses may drop the grain, push the grain around the feed bucket, cover it in saliva, and not be able to actually eat it. Eventually, the horse will be lying down more often, be unable to stand, or stand with an "elephant-on-a-ball" posture, with all four feet placed close together under the body. As the horse gets weaker and cannot stand anymore, it may collapse instead of deliberately folding its legs under itself as a normal horse would do. Finally, as the chest muscles and diaphragm get involved, respiratory failure occurs, and the horse dies of asphyxia.

The disease can hit suddenly and result in the death of horses that were perfectly healthy the day before. Botulism causes a flaccid paralysis, unlike tetanus that causes a rigid paralysis.

One of the worst aspects of botulism is that only the motor nerves are affected, the ones that are responsible for muscle movement. As such, the sensory function is left unimpaired. This means that victims of botulism will continue to experience hunger, thirst, fear, distended bladder, pain, and all other sensations, but they simply cannot move in response.

Clinical signs, history, and environmental observation are used to make a tentative diagnosis. If one horse in a group shows signs of botulism, others may quickly follow, if they have been fed from the same source of contaminated hay or feed.

Botulism is clinically diagnosed and more challenging to diagnose with laboratory tests. It is difficult to detect botulinum toxin in animals or to isolate the toxin from feedstuffs and feces of an affected horse. Routine blood tests are found normal (suspect botulism if the blood work is normal, yet the horse is showing signs of weakness!). Therefore, there may be more actual cases of botulism than the ones that are diagnosed. If clinical signs are compatible with botulism and the hay being fed is spoiled and contains areas of moist, decomposing material or a dead animal, or if a foal is involved, one should suspect botulism.

Treatment

Treating a horse with botulism can be very costly, difficult, and often too late. It is better to prevent the disease than to treat it. Recommended treatment for botulism includes early administration of hyperimmune plasma containing antitoxin. The antitoxin binds to the toxin molecules that are free floating in the bloodstream and neutralizes them before they bind to nerve cells, but it cannot reverse the effects of bound toxin. The bond that forms between the toxin and the nerve cell is irreversible.

The horse's body can make new neuromuscular junctions to replace the ones that are affected by the toxins; however, this process requires 7 to 10 days. It is a challenge to keep a horse alive that is recumbent and cannot eat or drink. In adult horses, being recumbent for a few days poses a problem in itself. They can develop pressure sores, colic, muscle damage, etc. Moreover, the horse will need to be mechanically ventilated and administered supportive therapy. However, it is very difficult to keep an adult horse on a ventilator for days, as the available machines are not designed to support this workload. If the paralysis has extended to the breathing muscles of an adult horse, it is humane to euthanize it.

Prevention

Horse owners should be cautious about feeding hay that has been rained on during the harvesting phases. Round-baled hay is particularly a risk factor when baled at excessive moisture content. Any hay with rotten or decaying material should not be fed to horses. Since the spoiled material is

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most likely to be internal in round hay bales, it may be impossible to visually determine this condition unless the bales are opened. If the exterior of the bale is rotten with dark discoloration and moldy or if the bales feel warm, they should not be fed to horses. An unspoiled round bale, put out for a group of horses, is generally not a problem.

There is also a risk for botulism if horses are being fed silage or haylage, especially if the fermentation process was inadequate to lower the pH to inhibit the growth of the bacteria and toxin production. Haylage, silage, and high-moisture hay are more prone to spoilage. For people who own horses and cattle and, thus, feed silage to all their animals, it is important to mention that cattle are not as sensitive to botulism as horses, but they do die from this disease.

Vaccination

There is a USDA-approved vaccine available to prevent botulism. The vaccine can be purchased from your veterinarian. Talk with your veterinarian about the best vaccination schedule for your herd. The following vaccination schedule is the proposed schedule of the American Association of Equine Practitioners (AAEP).

Educational programs of Kentucky Cooperative Extension serve all people regardless of race, color, age, sex, religion, disability, or national origin. Issued in furtherance of Cooperative Extension work, Acts of May 8 and June 30, 1914, in cooperation with the U.S. Department of Agriculture, M. Scott Smith, Director of Cooperative Extension Service, University of Kentucky College of Agriculture, Lexington, and Kentucky State University, Frankfort. UK

WEED OF THE MONTH

Common name: Musk thistle
Other names: Nodding thistle
Scientific name: *Carduus nutans L.*
Life Cycle: Biennial; sometimes annual
Origin: Eurasia
Poisonous: No

Musk Thistle Growth Stages
Seedling, Rosette, and Mature Flowering Plant

Musk thistle, also known as nodding thistle in some areas, is distributed across the United States and is listed as noxious in many states. This invasive species can reach heights of 6 feet and is found in pastures, rangeland, and along roadsides. Possibly the only redeeming value of this plant is the bright red to purple flowers that bloom from May to September.

Its light, windborne seeds can move great distances to infest adjacent areas. Seeds germinate in the fall or spring and form rosettes. Generally the flowering plants are 2 years old, although some plants act as an annual and produce seeds after one year of growth.

Controlling musk thistle in pastures is relatively easy with herbicides that kill the thistle and do not harm pasture grasses. Consult your local Cooperative Extension Service agent for a list of herbicidal control in your area. Mowing as a removal method must be timely for it to be effective. Mow after the stem elongates, but before seed heads are produced. Thistlehead weevil larvae eat the seeds of this thistle and are an effective biological control in some areas of the United States.

Bill Witt, PhD, provided this information. He is a researcher in Plant and Soil Sciences.

Acknowledgments

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Fernanda C. Camargo, PhD, Bob Coleman, PhD, and Laurie Lawrence, PhD, are from the Department of Animal and Food Science

Tall Fescue Control in Horse Pastures

WILLIAM W. WITT

Managers on Kentucky horse farms prefer pastures used for grazing pregnant mares to be composed of Kentucky bluegrass and orchard grass and little, if any, tall fescue. Most of the tall fescue in Kentucky pastures is "KY 31," and essentially all of that species contains the natural race of the endophytic fungus *Neotyphodium coenophialum*. This fungus is implicated in late-term pregnancy problems, such as an increase in gestation, dystocia (difficult birth), and agalactia (failure to produce milk). Horse farm managers prefer to remove tall fescue and other problematic and unsightly weeds from the pasture.

Research objectives Determine the control of tall fescue and troublesome weeds from Kentucky bluegrass/orchard grass pastures by 1) selectively killing tall fescue with imazapic herbicide and not harming the desired grasses; or 2) killing all vegetation with glyphosate herbicide and seeding desirable grasses.

Methods and materials A horse pasture composed of about 50% Kentucky bluegrass and 50% tall fescue was selected in 2005 to compare different methods to reduce tall fescue population. Treatments of imazapic and glyphosate, used alone or in combination, were applied at different times in 2005 and 2006. Imazapic treatments were applied with methylated seed oil at 1 qt/A (quart per acre) as per instructions on the label. The selective removal treatments contained only imazapic. Total renovation treatments included glyphosate.

All treatments were applied at a volume of 15 gallons of water per acre. Individual plots were 40 feet wide and 80 feet in length, and each treatment was replicated four times. The entire experimental site was over-seeded with a mixture composed of 60% Kentucky bluegrass and 40% orchard grass on Sept. 7, 2005. Dates of individual treatments are listed in the table on page 10.

The experimental site was managed along with the rest of the horse farm and included mowing every seven to 10 days to a height of about 6 inches and fertilization as per soil test recommendation in September. Relative percentage of tall fescue, Kentucky bluegrass, and orchard grass was determined by the point intercept method. Imazapic is sold in Kentucky under the trade names of Panoramic and Plateau. Glyphosate is sold under numerous trade names. The product used in these experiments was Roundup WeatherMax.

Results All treatments decreased the percentage of tall fescue compared to the untreated areas, which contained 57% tall fescue (Table 1). All combinations of glyphosate and two imazapic treatments in one year reduced tall fescue composition to less than 3%. While the selective removal treatments (imazapic only) and the total renovation treatments (those with glyphosate) greatly reduced tall fescue, both methods have drawbacks. The selective removal technique removes the tall fescue, but other weeds occupy the sites where tall fescue plants died. This can result in weedy

TREATMENT	AMOUNT PER ACRE	DATE APPLIED	% KENTUCKY BLUEGRASS	% ORCHARD GRASS	% TALL FESCUE	% NIMBLEWILL
GLYPHOSATE	3 QT	7-21-05	15 B	85 A	0 C	0.4 C
GLYPHOSATE + GLYPHOSATE	2 QT +2 QT	7-21-05, 8-25-05	19 B	81 A	0 C	0 C
GLYPHOSATE +IMAZAPIC	2 QT,10 OZ	7-21-05, 6-19-06	13 B	86 A	0 C	0 C
IMAZAPIC	12 OZ	5-18-05	28 AB	40 A	27 B	10.7 B
IMAZAPIC +IMAZAPIC	10 OZ +10 OZ	5-18-05, 6-21-05	16 B	76 A	3 C	22.5 A
UNTREATED			36 A	7 C	57 A	2.9 C

Table 1. Grass composition in a Kentucky bluegrass and orchard grass horse pasture after treatment with herbicides, Woodford County, Ky. Data was collected in October 2006, one year after renovation. Data presented represents the percentage of each grass present at the time of data collection.

(TALL FESCUE...)

pastures in those pastures that contain 50% or more of tall fescue at the time of treatment. The total renovation technique results in an excellent pasture, but it cannot be grazed for several months during the renovation process.

An important factor to consider is what weeds will appear when tall fescue is removed. An example was found in this study. Nimblewill (*Muhlenbergia schreberi*) is a native, warm-season grass that reproduces by seeds and numerous fine stolons and is not impacted by the weekly mowing regimes. The problem with nimblewill is aesthetic as well as it not being palatable to horses. In heavily grazed pastures, the nimblewill has a competitive advantage over Kentucky bluegrass and orchard grass. Nimblewill rarely grows with tall fescue, but when tall fescue was removed with imazapic, nimblewill emerged and became a serious weed in the pasture and occupied 10-25% of the pastures treated with imazapic. UK

Listing of products implies no endorsement by the University of Kentucky or its representatives. Criticism of products not listed is neither implied nor intended.

Acknowledgement. The author appreciates the cooperation of WinStar Farm, LLC, providing the pastures for this research.

William W. Witt, MS, PhD, is a professor and researcher in UK's Plant and Soil Science Department.

Using Soil-Cement on Horse and Livestock Farms

STEVE F. HIGGINS, D. SPENCER GUINN, AND DONNIE STAMPER, BIOSYSTEMS AND AGRICULTURAL ENGINEERING

Most farmers in Kentucky can identify with myriad problems associated with mud forming around high-traffic areas, including areas around horse and cattle waterers, feed bunks, round bale feeders, walk paths, and gate entrances. Mud is usually a result of animals congregating in and around these areas, but increased traffic can enhance the problem. In many cases, finding solutions to mud problems on farms is not the issue—the issue is determining how to make solutions economical.

Concrete and heavy traffic pads are traditional remedies for reducing mud on horse and livestock farms. A nontraditional option is soil-cement, which is about one-third the cost of concrete. Soil-cement is a highly compacted mixture of soil, Type 1 Portland cement, and water. Soil-cement was originally used as a stabilizing agent for riverbanks, and it has since been used as a subgrade and subbase modifier for roadways, water retention structures, and dam construction.

Agricultural uses of soil-cement in Kentucky have been in smaller areas, such as around automatic waterers and feed bunks, horse paths, and gate entrances. In those areas, soil-cement has been beneficial in stabilizing the soil and preventing mud.

Laboratory tests show completed soil-cement mixture is suitable as a livestock heavy traffic pad solution because it will form a hardened, stiff, durable material.

To demonstrate its use, soil-cement was used in an area around an automatic waterer (Figure 1). In the past, soil around this waterer had to be replaced every three years with approximately 6 to 9 inches of soil. The success of soil-cement as a heavy traffic pad material is illustrated in Figure 2, which shows the stability of the watering area after two years of exposure to the horses and the elements. This document describes the process of how to install soil cement areas.

The amount of cement needed to construct a pad varies, depending on the type of soil with which it is mixed and its intended use. As a base treatment for highways and roads, a 4% cement mixture of soil-cement has been used successfully. However, as a surface treatment (as on most farms), the cement percentage should be increased.

Proper mixing when preparing soil-cement is crucial. Improper mix amounts can lead to inferior soil-cement and can cost extra. Research shows that a mix with 6-15% cement by weight will provide an adequate ratio to construct a proper pad. A more in-depth method for determining mixture content can be found by using the tables below.

(USING SOIL-CEMENT...)

To properly use these tables, consult a soil survey book for your county, or use the Web Soil Survey (http://websoilsurvey.nrcs.usda.gov). Soil survey books are compiled for specific counties and contain soil survey maps and engineering properties tables for those areas. For example, the University of Kentucky has a farm in Woodford County and the soil survey map shows a MiB soil (Maury silt loam on 2-6% slopes) for one area of the farm. The engineering properties table contains the classification of this soil, which is located under the label AASHTO (American Association of State Highway and Transportation Officials). This particular MiB soil is an A-4. Table 1 demonstrates the amount of cement needed.

Figure 1. A typical waterer and concrete pad used in agriculture.

Figure 2. Shows the stability and wear of a watering pad area that was treated with soil cement two years prior.

For the A-4 soil, cement requirement typically ranges between 7-12%. The 7-12% ranges are guidelines for cement use. A 7% mixture might be sufficient for a light-traffic pad, but for our example we expected our pad to receive a high volume of traffic. Therefore, 12% was used.

To calculate the number of bags of cement needed, use Equation 1 (see page 13). Assume your lengths and widths are in feet, your depths are in inches, and you have a cement density of 97 lbs/cu ft.

An example of how to calculate the number of cement bags needed to construct a soil cement pad that measures 10 feet by 15 feet by 5 inches deep is provided below. Assuming cement bags weighing 94 pounds will be purchased, the written equation would look like this:

$$10 \times 15 \times \frac{5}{12} \times \frac{12}{100} \times 97 \div 94 = 7.7 \cong 8 \textit{ bags}$$

For a long-term pad on a general farm, 6-15% cement by dry weight is needed. Adding a small size aggregate to the soil before adding the cement is a good way to strengthen the soil-cement while adding traction. Class I sands, or limestone sand, are a good size that can still be incorporated into the soil.

To make soil-cement effective, some guidelines must be followed. The area planned for improvement, regardless of its history, should be free of all organic matter. All grass, weeds, sticks, stumps, and manure must be scraped off and replaced with fresh soil, if needed) (Figure 3 on page 13). Rocks can be a beneficial additive to soil-cement. It is not required to completely rid the area of rocks, as rocks can add to the strength of the soil-cement. However, rock that is too large can lead to poor compaction. A recommended rock size is found in dense grade aggregate (DGA).

AASHTO SOIL CLASSIFICATION	ASTM (AMERICAN SOCIETY FOR TESTING AND MATERIALS) SOIL CLASSIFICATION	TYPICAL RANGE OF CEMENT REQUIREMENT (% BY WEIGHT)
A-1-a	GW,GP,GM,SW,SP,SM	3-5
A-1-b	GM,GP,SM,SP	5-8
A-2	GM,GC,SM,SC	5-9
A-3	SP	7-11
A-4	CL,ML	7-12
A-5	ML,MH,CH	8-13
A-6	CL,CH	9-15
A-7	MH,CH	10-16

Table 1. Cement requirements for different soils.

Equipment needed to complete site preparation:

EQUATION 1

l = length (feet)
w = width (feet)
d = depth (feet)
% = percent cement

p_c = *density of cement* $\left(97 \frac{lb}{cu\ ft}\right)$

s = Cement bag size (in lb)
 # = Number of bags needed

• A garden tiller (self-propelled or tractor power take-off driven)
• A tractor with a loader
• A vibratory rolling compactor
• A water source

1. Use the tiller to pulverize the soil. More than one pass of the tiller might be required to reach the consistency of a seed bed. Use caution; do not till beyond desired depth. Loose soil makes it easier to add rock, if desired.

2. Add a smooth layer of rock over the loosened soil on a one-to-one (or slightly less) basis by volume. Use Equation 2 to estimate the amount of rock to be applied. Once rock has been spread, make a couple more passes with the tiller to sufficiently combine the two ingredients.

3. Once a uniform and solid foundation for the soil-cement has been prepared, spread cement over the soil and rock mixture. The cement must be more evenly spread than the rock. If bags of Portland cement will be used, space the bags evenly before emptying them. Once the cement has been spread, make one or two slow passes of the tiller to mix the materials together to form a desired blend.

Note: Limestone rock (common in Kentucky) is estimated to have a density of 100 lb/cu ft.

Figure 3. Rock, soil, and cement have been mixed. Notice the remaining organic matter is being removed.

4. Add water to the soil-cement mixture. To ensure uniform water content throughout the soil-cement, water should be added to the mixture while it is being tilled. There are two quick field tests to determine if the correct water content has been reached. One is to pick up a small amount of the mix and form it into a ball in your hand. The resulting ball of mix should hold its shape and not stain your hand, as well as be able to be pulled apart without disintegrating. The second test is to form a small amount of mix into a ball

EQUATION 2

$l \times w \times \frac{d}{12} \times p_r = R$

p_r = *density of rock* $\left(100 \frac{lbs}{cu\ ft}\right)$

R = Rock needed (in lbs)

and drop the ball from about 10 inches onto the tilled ground. If the ball of mix can be picked up again, the water content is appropriate.

5. Compact the soil-cement mixture. To ensure a completed soil-cement layer with the highest strength and durability possible, compact with a vibratory sheeps foot roller. Compact the soil-cement mixture until the surface no longer pumps, ruts, shoves, mars, or otherwise deforms under the weight of the roller. If a smooth surface is desired, a smooth drum roller can be run over the surface after the sheeps foot roller. At this point, the soil-cement is ready to be cured.

To ensure that the soil-cement does not dry too quickly from high temperatures or wind, it is important to keep the surface continuously moist until it has fully cured (Figure 4). Although precipitation before all ingredients are mixed can be detrimental, rainfall on a compacted soil-cement layer will serve to aid in the curing process. Be careful not

(USING SOIL-CEMENT...)

to use a sprinkler with too much force to avoid disturbing the uncured surface. Plastic covering can be used on small areas to inhibit the soil-cement from curing too quickly. Soil-cement should only be constructed when the outside air temperature is 40°F or above and should never be constructed with muddy or frozen materials.

Figure 4. A 360-degree sprinkler is one way to keep the soil-cement wet.

Figure 5. Horses on a soil-cement pad top dressed with Class I sand.

After a typical curing period of two or three days has passed, the soil-cement can be top-dressed with 57s (course aggregate with a 100% passing through 1.5-inch screen), Dense Grade Aggregate (DGA), Class I sand (crusher fines), or it can be left exposed. Top dressing depends on the operator's judgment. At this point, the soil-cement should be sufficiently cured to support animal and equipment traffic (Figure 5). If the area is small enough to top-dress without equipment moving onto freshly compacted (uncured) soil-cement, feel free to do so. Top-dressing will help keep moisture in the soil-cement during the curing period. UK

Further Reading

High Traffic Area Pads for Horses (ID-164) at www.ca.uky.edu/agc/pubs/id/id164/id164.pdf

Using Dry Lots to Conserve Pastures and Reduce Pollution Potential (ID-171) at www.ca.uky.edu/agc/pubs/id/id171/id171.pdf

Steve F. Higgins, PhD, is an agriculture research specialist in Biosystems and Agricultural Engineering.

KENTUCKY DIAGNOSTIC CENTER DIRECTOR HONORED AT ARMY RETIREMENT CEREMONY

Craig Carter, DVM, PhD, Dipl. ACVPM, director of the University of Kentucky Livestock Disease Diagnostic Center, was recognized during a retirement ceremony March 22 at the Army Reserve Medical Command in Pinellas Park, Fla., for his 42 years of military service.

During the ceremony, Carter, who enlisted in the U.S. Air Force in June 1967, was awarded the Legion of Merit and Joint Service Commendation Medal for his service.

Carter

The Legion of Merit was awarded to Carter for exceptionally meritorious service in positions of increasing responsibility, culminating in a 30-year career as Veterinary Readiness Advisor, Army Reserve Medial Command. The Joint Service Commendation Medal was awarded to Carter for distinguishing himself by exceptionally meritorious service as Veterinarian, Task Force Ramadi, Iraq, from April through July 2008.

During periods of inactive status with the army, Carter received his master's degree in

(CARTER HONORED...)

epidemiology and a doctorate in veterinary medicine from Texas A&M University.

After graduation, Carter opened a general veterinary practice in Texas. In 2005 Carter accepted a position as professor of epidemiology with the University of Kentucky and joined the 332nd Medical Brigade in Nashville, Tenn., as the brigade veterinary staff officer. Soon he was selected to succeed Colonel Bill Brown as the veterinary readiness advisor at AR-MEDCOM, Pinellas Park, Fla., where he assisted and advised several veterinary units in preparation for overseas deployments.

In early 2008 Carter deployed to Camp Ramadi, Anbar Province, Iraq, as the Joint Forces Command Senior Veterinary Advisor. In this assignment, Carter traveled throughout Iraq evaluating veterinary laboratories and writing funding proposals for infrastructure capacity building.

In civilian life, Carter has been very engaged internationally as a consultant to the Department of Defense, U.S. Department of Agriculture, and US AID. His travels have included Iraq, Kuwait, Jordan, China, Thailand, Australia, Uruguay, Nicaragua, and Ethiopia, where he delivered lectures and participated in nation-building activities.

In spite of heavy professional commitments and extensive travel requirements, Carter has remained committed to his community and has been active in community service. A high point of his community service occurred when he chaired the committee that constructed the Brazos Valley All-Wars Veterans Memorial in College Station, Texas. The memorial was dedicated by President George H.W. Bush on Nov. 22, 2002.

In 2007 Carter accepted the position of director of the Livestock Disease Diagnostic Center at the University of Kentucky. The center provides diagnostic support for more than 3,000 animal hospitals in Kentucky and throughout the United States. The LDDC has the highest equine case load worldwide. UK

NEW COLLEGE OF AGRICULTURE ONLINE GIVING SITE LAUNCHED

JENNY BLANDFORD

A new University of Kentucky College of Agriculture online giving site was launched June 1 to help secure donations for equine research.

The new secure site, https://giveto.uky.edu/AG_p/ag.htm, allows potential donors to choose to donate from a list of about 40 programs, including the Gluck Equine Research Enrichment Fund, Equine Initiative, and Livestock Diagnostic Disease Center Enrichment.

The online giving form provides donors the opportunity to make a contribution to the College of Agriculture to be fulfilled over a period of time. Donors can designate gifts to benefit a specific school, department, program, or project by filling out the appropriate fields on the online form.

"This site allows donors to support their particular interests of programs in the UK Equine Initiative," said Nancy M. Cox, PhD, associate dean for research in the College of Agriculture. "Also, we appreciate hearing from our stakeholders; together, we can support Kentucky's signature equine industry."

An online pledge card is also available at www.uky.edu/Development/pledges/AGpledge.htm. By filling out the online pledge card, UK's Office of Development will mail donors a monthly, quarterly, or yearly billing reminder. The options are selected online. A pledge card does not require credit card information to be submitted online.

Donors also have the opportunity to make a contribution "in honor of" or "in memory of" a horse, person, or organization. These options are available through the online secure site and online pledge card.

Previously, the College of Agriculture did not have a separate online donation site and was linked with all other colleges through the University of Kentucky's Office of Development.

All donations to the College of Agriculture are tax-deductible. UK

Jenny Blandford is the Gluck Equine Research Foundation Assistant at the Gluck Center. She can be reached at jenny.blandford@uky.edu or 859/257-4757, ext. 81089.

Changing Faces in UK's Equine Programs

JENNY BLANDFORD, HOLLY WIEMERS

The past few years saw many faculty changes at UK's Gluck Equine Research Center and Equine Initiative. Additions to Gluck included a new Department of Veterinary Science chair and director, a new Gluck Equine Research Foundation executive director, and the loss of a longtime faculty member. Three new faces joined the Livestock Disease Diagnostic Center (LDDC). In the Equine Initiative, the past year saw the addition of Jamie MacLeod, VMD, PhD, Director of the UK Equine Initiative and Dickson Professor of Equine Science and Management. Additionally, new equine-specific faculty were hired in Agricultural Economics and Animal and Food Sciences.

Changes in the Gluck Center

In summer 2008, Mats Troedsson, DVM, PhD, Dipl. ACT, became the new director of the Gluck Equine Research Center and chair of the Department of Veterinary Science. Troedsson replaced Peter Timoney, FRCVS, PhD, who was chair of the Department of Veterinary Science from 1989 to 1999 and 2002 to 2008. However, Timoney did not leave Gluck and is now a full-time research scientist in the infectious diseases and immunology program.

Troedsson

Troedsson's previous position at the University of Florida from 2002 to 2008 was as professor and service chief for reproduction in the Department of Large Animal Clinical Sciences in the College of Veterinary Medicine. Prior to his tenure in Florida, Troedsson was in the University of Minnesota's Department of Clinical and Population Sciences.

Troedsson, who joined the reproduction program at Gluck, received his DVM in Sweden and his PhD at the University of California, Davis.

A few months later, another faculty member joined the reproduction program as a research scientist. Ed Squires, MS, PhD, Hon. Dipl. ACT, who spent 33 years as faculty at Colorado State University, came on board in November 2008 as Executive Director of the Gluck Equine Research Foundation and Director of Development and Industry Relations.

Squires

A native of West Virginia and a graduate of West Virginia University, Squires was inducted into UK's Equine Research Hall of Fame in 2007. He is the editor of the *Journal of Equine Veterinary Science* and has published more than 260 papers, written 30 book chapters, and published 14 textbooks.

In April 2008 longtime faculty member George Allen, PhD, died. Allen had been a professor in the Department of Veterinary Science since 1979.

Allen was known to the scientific community as one of the world's foremost authorities on equine herpesvirus infections in equines.

LDDC Faculty Additions

Cynthia Gaskill, DVM, PhD, joined the LDDC as an analytical toxicologist, associate professor, and veterinary clinical diagnostic toxicologist. Gaskill's primary duty is to provide an analytical and diagnostic toxicology service for the animal industry in Kentucky. She is also responsible for the establishment and direction of a toxicology laboratory and development of a top-class program in analytical toxicology at the Diagnostic Center.

Gaskill, who worked on several cattle ranches and horse farms in the western United States during her youth, obtained her DVM from Colorado State University and her PhD from Atlantic Veterinary College. After obtaining her PhD, she accepted a tenure track faculty position at Atlantic Veterinary College as a clinical toxicologist.

Alan Loynachan, DVM, PhD, Dipl. ACVP, is an anatomic veterinary diagnostic pathologist and assistant professor at the LDDC. He works with other faculty, developing a nationally recognized veterinary pathology program that aids in the reduction of animal morbidity (illness) and mortality (death).

Prior to coming to Kentucky, Loynachan was an assistant professor and diagnostic pathologist at Iowa State University, Department of Veterinary Diagnostic and Production Animal Medicine. He

(CHANGING FACES...)

was also a pathology consultant for Sirrah-Bios in Ames, Iowa. Loynachan earned his DVM in 2003 and his PhD in veterinary microbiology in 2005 from Iowa State University.

As a veterinary diagnostic pathologist and assistant professor at LDDC, Lynne Cassone, DVM, is responsible for helping to reduce loss from disease in Kentucky's livestock and other animal industries by providing competent and timely diagnostic pathology at the department's diagnostic center. Duties also include diagnostic gross pathology and histopathology of biopsy and necropsy specimens, mammalian, avian, and reptilian species (approximately 80% of her caseload is equine) and regular participation in resident training.

Cassone earned her DVM in 1999 at Texas A&M University. Prior to coming to Lexington in September 2008, Cassone was a staff pathologist at Texas Veterinary Medical Diagnostic Laboratory.

Other College of Agriculture Staff Changes

MacLeod

MacLeod, Director of UK Equine Initiative and Dickson Professor of Equine Science and Management and the John S. and Elizabeth A. Knight Chair at the Gluck Center, is a Professor of Veterinary Science. He became the Director of the Equine Initiative in a 20% capacity, with the other 80% of his time comprised of his distinguished research with the Gluck Center. In his Equine Initiative role, MacLeod focuses primarily on advancing the Equine Initiative through productive working groups made up of faculty in subject areas of interest as well as through engaging in active faculty relations throughout the college. He's been involved in developing the Equine Initiative since its formation in 2005.

Bob Coleman, PhD, Associate Director for Undergraduate Education in Equine Science and Management and Extension Horse Specialist, is already a familiar face to many across Kentucky. Coleman has taken on a different role in the past few years, warranting a reintroduction to many who knew him in his former roles in Horse College and equine youth programs. Coleman now heads UK's new Equine Science and Management undergraduate degree program. He is continuing his work as an Extension Horse Specialist and maintains his involvement in Horse College programming and equine adult education. In addition, Coleman teaches several courses in the program and serves as faculty advisor for UK's Equestrian Team.

Jill Stowe, PhD, assistant professor in the Department of Agricultural Economics with a joint appointment with UK's Department of Economics, teaches and conducts research on issues related to the equine industry. Originally from New Mexico, Stowe received a bachelor's degree in mathematics from Texas Tech University and a doctorate in Economics from Texas A&M. She was employed at Duke University's Fuqua School of Business from 2002 to 2008. She started at UK in July 2008 and will conduct research 60% of the time and teach the other 40%. Her professional areas of interest include economic research of the equine industry, decision making under risk and ambiguity, other-regarding behavior, game theory, neuroeconomics, optimal contracting, and sports economics.

Fernanda Camargo, DVM, PhD, is an assistant professor in the Department of Animal and Food Sciences and equine extension specialist focusing on youth. In 2001 her passion for horses brought her to Lexington, where she completed her doctorate at UK in equine pharmacology and toxicology. She was specifically involved in the development, creation, and validation of several analytical methods for the detection of pharmacological compounds in equine biological fluids.

She is now an equine extension professor at UK with a major emphasis in equine youth extension through the 4-H Horse Program, which touches more than 6,000 youth and is one of the country's largest 4-H horse programs. She develops and delivers educational materials and activities for county extension agents for 4-H youth development, horse owners, volunteer leaders, parents, and 4-H youth. In addition, she contributes to the adult equine extension program alongside Coleman.

Mary Rossano, MS, PhD, assistant professor in the Department of Animal and Food Sciences, is also conducting research in epidemiology. Rossano currently teaches in the new undergraduate program and is slated to teach 70% of the time and conduct research the other 30%. She currently teaches classes that include Equine Management (both a hands-on version and an online version through distance learning), Horse

(CHANGING FACES...)

Behavior and Handling, and Domestic Animal Biology. Her research interests are in equine epidemiology via surveys and observational studies of outcomes of equine management practices and factors associated with health, performance, profitability, and environmental impact.

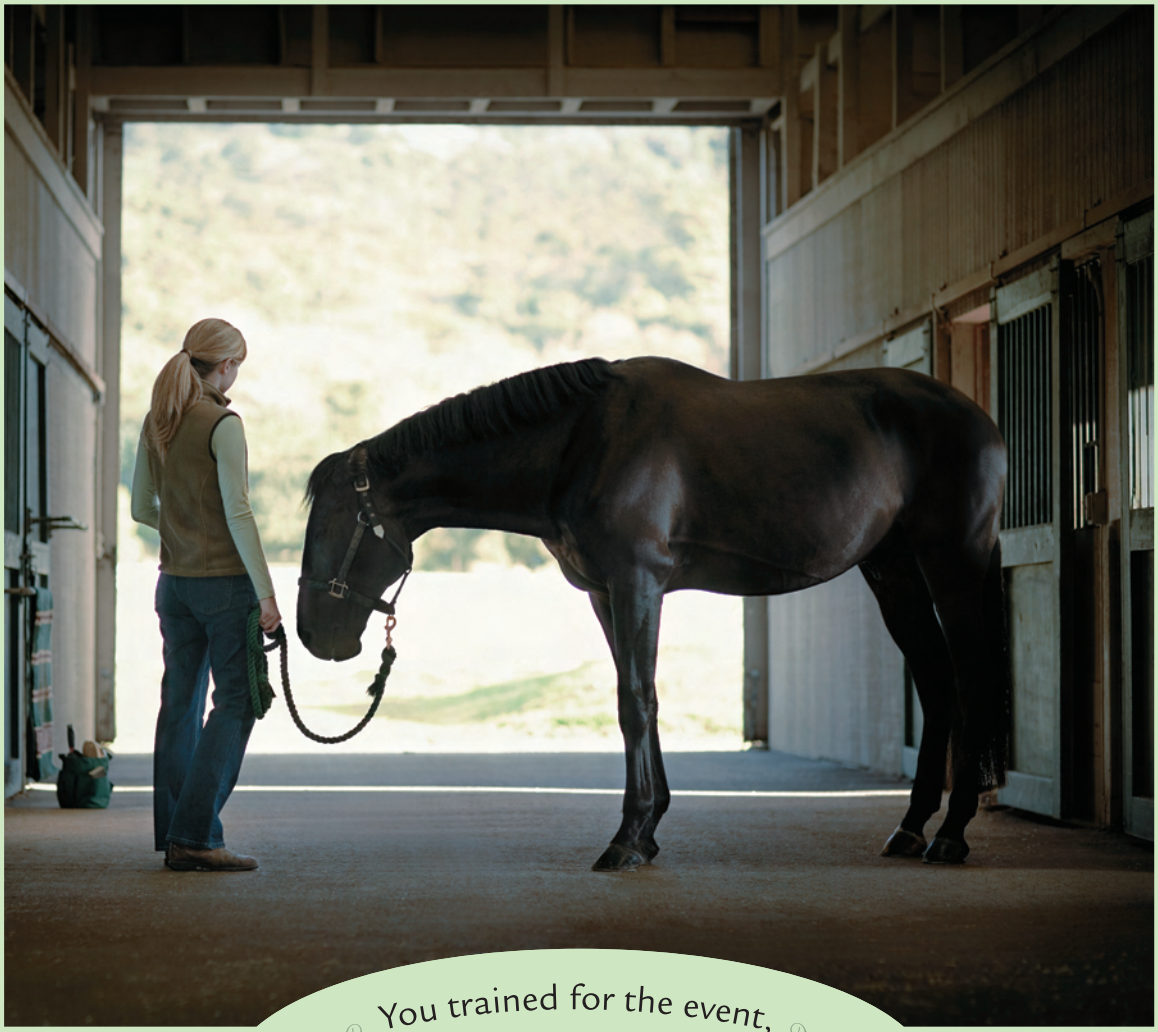
Kristine Urschel, PhD, assistant professor in the Department of Animal and Food Sciences, is also conducting research in nutrition. Urschel hails from Edmonton, Alberta, Canada. She received her bachelor's and doctoral degrees at the University of Alberta. She completed a post-doctoral fellowship in equine nutrition at Virginia Tech, where she worked from April 2007 to July 2008. Her research at Virginia Tech was twofold: first, to investigate the factors regulating protein synthesis and metabolism in healthy, adult horses; and second, to develop and validate stable isotope techniques that could be used to study protein metabolism and requirements in horses of all ages. She plans to build on this initial research at UK. Urschel's research will work toward an improved understanding of how the equine body uses and metabolizes dietary protein as well as the knowledge of how protein and amino acid requirements change with age, ultimately helping horse owners optimize the protein composition of horse diets to support better performance and/or health. UK

Jenny Blandford is the Gluck Equine Research Foundation Assistant. She can be reached at jenny.blandford@uky.edu or 859/257-4757, ext. 81089. Holly Wiemers is the communication director of the UK Equine Initiative. She can be reached at holly.wiemers@uky.edu or 859/257-2226.

CALENDAR OF EVENTS

July 23, Princeton Field day (all commodity), Princeton, Ky., 8 a.m.- 3 p.m., http://ces.ca.uky.edu/wkrec/

July 25, Airway inflammation seminar, Department of Veterinary Science Equine Diagnostic and Research Seminar, LDDC Conference Room, www.ca.uky.edu/gluck/NewsSeminar.asp



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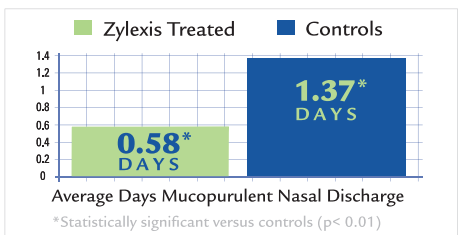

Demonstrated clinical reduction of nasal discharge following commingling challenge with EHV-1 and/or 4.[1]

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1 Data on File, Study Report No. Equine 1-98, Pfizer Inc.