EQUINE DISEASE QUARTERLY

FUNDED BY UNDERWRITERS AT LLOYD'S, LONDON, BROKERS AND THEIR KENTUCKY AGENTS

April 2005
Volume 14, Number 2

IN THIS ISSUE

UK
UNIVERSITY OF KENTUCKY
College of Agriculture
Department of Veterinary Science

LLOYD'S

COMMENTARY

WEST NILE VIRUS INFECTION AND MARE REproductive Loss Syndrome are recent examples of well publicized "emerging" diseases in the equine population. The emergence of several bacterial species isolated from the reproductive, digestive, and respiratory tracts of sick and healthy horses has received less attention.

During the 1998 and 1999 foaling seasons, the Livestock Disease Diagnostic Center, UK College of Agriculture, Lexington, reported an increase in cases of nocardioform placentitis. The term describes a distinct placentitis based on location of lesions and presence of unclassified Gram-positive filamentous bacteria. (1) Taxonomic studies have proposed the names *Crossiella equi* (2) and *Amycolatopsis kentuckyensis* and *Amycolatopsis lexingtonensis*. (3) During the 2002 and 2003 foaling seasons, *Cellulosimicrobium (Cellumonas) cellulans* was the principal organism isolated from fetal tissues and placentae of abortions, premature births, and full-term pregnancies. (4) The changes observed were similar to those recorded for nocardioform placentitis, suggesting there may be more than one causal agent. Cellumonas is widely distributed in soils and decaying vegetable matter and was isolated from an aborted equine fetus in Australia in 1982. (5) Cellumonas and other similar Corynebacterium species have been isolated with increasing frequency from immune-compromised human patients and those with a history of broad-spectrum antibiotic therapy.

Both *Clostridium difficile* and C. *perfringens* types A, B, C, and D have been increasingly isolated from sporadic cases of diarrhea, antibiotic-associated enterocolitis, and nosocomial enterocolitis. (6) Disruption of the intestinal microflora predisposes to overgrowth by toxigenic bacteria. Difficulty exists in confirming a diagnosis of clostridial infection, as the organisms and toxins may be present in asymptomatic foals. Predisposing factors, including transportation, hospitalization, surgery, and antibiotic therapy have been recognized, similar to those responsible for the increasing incidence of human clostridial enterocolitis. A proliferative enteritis recognized in a variety of species, particularly swine, caused by L. *intracellularis* produces weight loss, colic, and diarrhea in older foals. (7)

A new proposed member of the Pasteurellaeae family—*Nicoletella semolina*—has been isolated from horses with respiratory disease. (8) Although considered a human pathogen, *Streptococcus pneumoniae* capsule type 3 plays an important role in the pathogenesis of inflammatory airway disease in young horses. (9) Methicillin-resistant *Staphylococcus aureus* isolated from the respiratory tract and wounds of horses has been reported with increasing frequency. (10)

It remains to be determined whether several of these organisms represent a new emerging species or result from the increased sophistication of isolation techniques. Whether these organisms are primary pathogens, part of the normal flora that occasionally give rise to disease, or coincidentally happen to be present when disease arises requires diligent scientific investigation.

A recurring theme is the routine use of antibiotic therapy. There is concern in human and veterinary medicine regarding the influence of antibiotic usage on the emergence of antibiotic-resistant and other bacteria not previously associated with disease. Insufficient dosages administered for an inadequate period of time have exacerbated the problem. Judicious use and selection of antibiotics with greater emphasis on surveillance and infection control measures are necessary to limit the veterinary and public health significance associated with these bacteria. (References, page 2.)

CONTACT: Dr. David G. Powell (859) 257-4757, dgpowe2@uky.edu, Maxwell H. Gluck Equine Research Center, University of Kentucky, Lexington, Kentucky.

INTERNATIONAL

Fourth Quarter 2004

THE INTERNATIONAL COLLATING CENTRE, Newmarket, reported the following disease outbreaks:

Atypical Myoglobinuria was suspected in France among a widely distributed population of 55 horses, 42 of which died. Cases were also reported from Switzerland.

Equine herpes virus abortion (EHV-1) was diagnosed among pregnant polo mares on two premises in Argentina. On one farm, losses were significant in mares that had been impregnated by embryo transfer. Sporadic EHV-1 abortions were also confirmed in France, Ireland, Japan, the United Kingdom, and the United States. Cases of EHV-1 neurological disease were reported on two premises in the United Kingdom, and there were two reports of Coital Exanthema (EHV-3) from Switzerland. Respiratory disease caused by EHV-4 was reported from France, Ireland, Japan, and the United Kingdom.

Equine Arteritis Virus (EAV) was isolated from the semen of a non-Thoroughbred stallion while in quarantine in the United Kingdom. The animal had recently arrived from the Netherlands, to where it was returned, and was undergoing pre-export screening prior to shipment to New Zealand. Sporadic outbreaks of equine influenza were reported from Argentina, France, Ireland, and the United Kingdom.

Several cases of Leptospira abortion were confirmed among pregnant mares in Central Kentucky. *Salmonella abortus equi* was isolated from nine non-Thoroughbreds on two premises in Hokkaido, Japan, and cases of strangles were diagnosed on premises in Ireland and Switzerland. Outbreaks of rotavirus infection causing diarrhea occurred on four premises in Argentina.

The USDA reported 1,341 equine cases of West Nile virus infection in the United States during 2004 as compared to 4,500 cases in 2003. Highest incidence was in California (536), Texas (115), and Arizona (113), emphasizing the westward spread of the disease and the reduced incidence in the eastern states.

Equine Disease Quarterly

Editors
Roberta Dwyer
David Powell
Neil Williams

Staff
Diane Furry

Correspondence should be addressed to the editors, Department of Veterinary Science, Maxwell H. Gluck Equine Research Center, University of Kentucky, Lexington, Kentucky USA, 40546-0099
Telephone (859) 257-4757
Fax (859) 257-8542

Internet address:
http://www.uky.edu/Agriculture/VetScience/gluck1.htm

Reference List *for* Commentary, *page 1*

1. Donahue J.M. and Williams N.M., 2000. Emergent Causes of Placentitis and Abortion. In Timoney P.J., editor. Veterinary Clinics of North America, Equine Practice, 16: 443-456.

2. Donahue J. M. et al., 2002. *Crossiella equi* sp. nov., Isolated from equine placentas. International Journal of Systematic and Evolutionary Microbiology, 52: 2169-2173.

3. Labeda D.P. et al., 2003. *Amycolatopsis kentuckyensis* sp. nov., *Amycolatopsis lexingtonensis* sp. nov., and *Amycolatopsis pretoriensis* sp. nov., Isolated from equine placentas. International Journal of Systematic and Evolutionary Microbiology, 53: 1601-1605.

4. Bolin D.C. et al., 2004. Equine abortion and premature birth associated with *Cellulosimicrobium cellulans* infection. Journal of Veterinary Diagnostic Investigation, 16: 333-336.

5. Thomas R.J. and Gibson J.A., 1982. Isolation of *Oerskovia xanthineolytica* from an aborted equine foetus. Australian Veterinary Journal, 58: 166-167.

6. Jones R.L., 2000. *Clostridial enterocolitis.* In Timoney P.J., editor. Veterinary Clinics of North America, Equine Practice, 16: 471-485.

7. Lavoie J.P. et al., 2000. Equine proliferative enteropathy: a cause of weight loss, colic, diarrhea, and hypoproteinaemia in foals on three breeding farms in Canada. Equine Veterinary Journal 32: 418-425.

8. Kuhnert P. et al., 2004. *Nicoletella semolina* gen. nov., sp.nov., a New Member of *Pasteurellaceae* Isolated from Horses with Airway Disease. Journal of Clinical Microbiology, 42: 5542-5548.

9. Wood J.L.N. et al., 2005. Association between Respiratory Disease and Bacterial and Viral Infections in British Racehorses. Journal of Clinical Microbiology, 43: 120-126.

10. Weese J.S., 2004. Methicillin-resistant *Staphylococcus aureus* in horses and horse personnel. In Baln F.T. and Weese J.S., editors. Veterinary Clinics of North America, Equine Practice, 20: 601-613.

NATIONAL

Genetic Disease in the Horse

CONFUSION OFTEN SURROUNDS THE TERminology used to describe birth defects in horses and other animals. The terms "congenital" and "genetic" are used interchangeably by many. Actually, "congenital" merely refers to any defect present at birth. It may indeed be due to a genetic or inherited problem, but it also describes defects due to non-genetic causes, such as developmental problems caused by toxins, infection, poor uterine environment, or malposition. Genetic defects include anything that is due to a change in the DNA that affects development so that the foal is not normal when born. Sometimes a new mutation occurs in the embryo's DNA that causes a problem but is not inherited from either parent. Other genetic defects are indeed inherited from one or both parents.

Compared to other species such as the dog, the horse has a comparatively low incidence of inherited disorders. New technologies have resulted in identification of the actual mutations, which permits breeders to screen their animals and identify carriers through DNA analysis.

One of the first recognized inherited disorders in the horse was Severe Combined Immunodeficiency (SCID) in the Arabian horse. Affected foals die within the first few months of age due to the inability to fight infection. It is estimated that about 3% of Arabian foals were born with this condition. Inherited as an autosomal recessive, both parents must be carriers. Since carriers appear healthy and normal, the development of a DNA test that detects the mutation allows the breeder to avoid carrier-to-carrier matings.

Another inherited disorder, which affects extensive numbers of Quarter horses descended from the stallion Impressive, is hyperkalemic periodic paralysis (HYPP). Unlike SCID, this is a dominant disorder. Only one affected parent is needed to pass the gene on to the foal. The mutation causes a defect in a sodium channel gene that regulates the movement of sodium into and out of muscle tissue. Horses with HYPP experience muscle tremors and can become temporarily paralyzed. Severe attacks can lead to heart and respiratory failure, resulting in death. Horses with two copies of the defective gene (homozygous) usually do not survive very long. Horses with one copy of the mutation can lead functional lives if fed a controlled diet. In fact, the disease causes enlargement of the muscles in these horses, which gives them an advantage in halter classes at shows. Again, a DNA test is available to determine which horses carry this mutation, and matings that would produce a foal homozygous for the mutation can be avoided.

A third heritable disease for which a DNA test has been developed also occurs in Quarter horses and Paint horses. Glycogen Branching Enzyme Deficiency (GBED) is a fatal disease affecting foals. The mutation causes a lack of the functional form of the enzyme needed to properly store sugar molecules as glycogen. Tissues that require glycogen as an energy source, such as heart muscle, skeletal muscle, and brain, cannot function properly. Foals are stillborn or die at a very young age.

The Overo Lethal White Syndrome was the bane of Paint horse breeders. When two carriers are mated, a white foal, or nearly all-white foal, might be produced that dies or needs to be euthanized within days of birth. These foals have abnormalities of the intestine that prevent them from passing feces. A DNA test is also available for this disorder so breeders can determine which horses are carriers.

A DNA test is not yet available for Equine Hyperelastosis Cutis, which occurs in Quarter horses descended from the Poco Bueno/King bloodline. It does not occur in all horses descended from these sires, and its expression is variable, with some horses being more severely affected than others. Skin layers in affected horses are not attached normally, creating loose areas of skin that are easily stretched away from the body. This makes the skin very susceptible to trauma, and the condition is often initially diagnosed when the horse goes into training and is subjected to saddle pressure. There is no treatment, and currently there is no genetic test.

Fortunately, the technology exists to eventually identify the mutation for this disorder as well as other genetic diseases. While it may never be possible to prevent congenital defects that are due to novel mutations that occur during development or due to environmentally induced defects, we do have the tools to assist breeders in preventing the predominant inherited diseases that are recognized in horses.

CONTACT: Dr. Kathryn T. Graves, (859) 257-2510, ktgraves@uky.edu, Equine Parentage Verification and Research Laboratory, University of Kentucky, Lexington, Kentucky.

Risk Reduction

INFECTIOUS DISEASES ARE A CONSTANT risk to the health and welfare of horses. The threat of strangles, influenza, equine herpesvirus, rotavirus, Salmonella, Rhodococcus, etc. are only some of the reasons for a comprehensive vaccination program. However, no vaccine is 100% effective, and vaccines are not commercially available for many diseases, including the dreaded salmonellosis.

However, multiple farm management techniques can have a significant influence on reducing the risk of disease outbreaks. Not all are as labor intensive as completely disinfecting stalls and aisleways. These techniques include:

- Group horses of similar uses. Show horses, yearlings, broodmares, riding horses, should not be commingled.

- Plan a traffic pattern to take farriers, veterinarians, and other personnel to barns and pastures with at-risk horses (e.g., pregnant mares or mares and foals) first, and work toward horses that have multiple exposures to pathogens (show and trail riding horses).

- Isolate any new horses to the farm for a minimum of 14 days, and ideally 21 days. The horse can be monitored for infectious diseases, and any necessary vaccinations and deworming can be completed at that time.

- Isolate horses returning from a hospital stay for similar periods of time. The stress of transportation and medical procedures can lower horses' immunity, and they may come in contact with other equine patients, some of which may be shedding pathogens.

- If a horse is observed as being sick (cough, runny nose/eyes, diarrhea, fever, etc.), it should be isolated immediately and protective clothing utilized by everyone working with the animal: disposable gloves and booties and coveralls should be re-used only with that horse. Be sure to provide alternative gloves for employees who are allergic to latex, such as gloves made of nitrile or vinyl.

- Stalls of sick horses should be mucked out last, and preferably separately, using pitchforks, shovels, and other tools that are properly disinfected prior to their next use. Alternatively, use separate tools for healthy horses' stalls and a different set for sick animals' stalls.

- Manure and bedding from stalls housing sick animals, including those experiencing abortions, should not be spread on fields. This material should be composted away from all animals or disposed of in a manner approved by local ordinances.

- In every barn provide running water, liquid hand soap (pump-style container), and disposable paper towels for handwashing. All employees should wash their hands prior to leaving at the end of their shift, and in the midst of a disease problem, they should thoroughly wash their hands after working with sick animals, whether or not they were wearing disposable gloves. During an outbreak or when running water is not available, have waterless hand foams or gels (at least 62% ethyl alcohol) to use after handling horses. Remind employees that these products are flammable!

- Rodent control is paramount year-round! One barn mouse can ingest Salmonella and be a multiplication factory better than any petri dish. The mouse droppings contain enormous amounts of bacteria that can effectively seed the horse's environment and feed supply with infectious bacteria. Insect, bird, and bat control are also important. Remove standing water, bird nests, and other habitats. Hire professionals for removal of bat roosts and also for difficult rodent or wildlife control.

- Clean and disinfect stalls, water buckets, grooming tools, pitchforks, and other items routinely, and increase the frequency during an outbreak situation.

- Most importantly, communicate and educate employees and enforce biosecurity procedures on the farm.

CONTACT: Dr. Roberta Dwyer, (859) 257-4757, rmdwyer@uky.edu, Maxwell H. Gluck Equine Research Center, University of Kentucky, Lexington, Kentucky.

KENTUCKY

Characteristics of the Equine Placenta

THE PLACENTA PROVIDES PROTECTION for the fetus and regulates the fetal environment. Previous work in the United Kingdom has examined placental parameters associated with normal births. A study was undertaken in the Central Kentucky area by veterinarians from the Hagyard Equine Medical Institute to examine placental parameters associated with foals making a normal transition to extra-uterine life. This study comprised observations from 168 foalings during a two-year period on three farms. Information was collected on factors related to the mare, the foal, and the placenta, as illustrated in Table 1.

To gain insight into the normal parameters, researchers in the past have used a set of exclusion criteria to eliminate potentially abnormal placentas. These were mostly associated with abnormal foaling events (i.e., abnormal delivery, more than two hours for the foal to stand, or more than four hours for the foal to nurse). The study found that there were not significant differences in the placental parameters between those classified as normal and abnormal based on foaling events. It suggests this arbitrary method of classification may not in fact separate normal and abnormal placentas. Different methods of classification based on placental characteristics, such as the presence of placentitis, should be examined in the future to separate normal and abnormal placentas.

The average times to stand and nurse, 66.4 and 112.7 minutes respectively, were similar to results recorded in previous studies. There was not a significant correlation between foal weight and the time to stand, agreeing with previous results. However, foal weight was significantly correlated with gestational age, allantochorionic weight, and the age of the dam.

Long umbilical cords have been associated with three outcomes: strangulation, excessive cord twisting, and necrosis of the cervical pole of the allantochorion. These outcomes lead to increased risks of abortions and stillbirths. One of the aims of the current study was to examine factors that influence umbilical cord length. The influences of 49 different factors on total umbilical cord length were examined; two were found to be significant after controlling for other variables. As mare age increased, the length of the umbilical cord also increased. Also, as the length of the nonpregnant horn increased, umbilical cord length also increased. This could be due to an increased ability of the foal to move within the uterine environment because of the parity of the dam (i.e., greater fetal kinesis).

Mares were divided into three age categories: less than 8 years of age, 8 to 14 years of age, and more than 14 years of age. There were significant differences in some of the parameters by mare age. Mares more than 14 years of age had longer gestational lengths, heavier allantochorionic portions of the placenta, and shorter time to a foal's first nursing. Foal weights were lowest among those mares less than 8 years of age.

This study provides information on the parameters associated with a normal foaling in the Central Kentucky area. In future, studies on correlations between *in utero* measurements and measurements at birth will provide veterinarians with valuable information for diagnosing problems earlier in fetal development.

TABLE 1.
Placental parameters associated with foals making normal transition to extra-uterine life.

Category	Variable	Number	Mean	Standard Deviation	Range
MARE	Age of Dam (yrs)	168	12.7	5.6	5-26
	Number of Registered Foals	155	6.0	4.1	0-15
	Time to Pass Placenta (min)	75	50.3	52.7	5.0-357.0
FOAL	Gestational Age (days)	167	342.0	8.3	312-362
	Foal Weight (kg)	157	54.1	6.3	29.5-74.5
	Time to Stand (min)	150	66.4	34.1	15.0-260.0
	Time to Nurse (min)	136	112.7	48.3	35.0-290.0
PLACENTA					
	Total Umbilical Length (cm)	165	65.0	19.0	30.5-183.0
	Amniotic Cord Length (cm)	85	32.6	9.0	15.5-65.0
	Chorionic Cord Length (cm)	85	30.9	11.5	9.0-62.0
	Number of Cord Twists	162	0.94	1.4	0-7
	Number of Amniotic Twists	82	0.54	0.84	0-5
	Number of Chorionic Twists	82	0.49	0.82	0-4
	Placental Weight (kg)	59	7.1	1.4	3.6-11.4
	Chorionic Weight (kg)	141	4.1	1.0	2.3-7.3

Source: Observations from 168 foalings during a two-year period on three Central Kentucky farms.

CONTACT: Karin Bosh, Graduate Research Assistant, (859) 257-4757, Maxwell H. Gluck Equine Research Center, University of Kentucky, Lexington, Kentucky.

Dr. Luke Fallon, (859) 255-8741, Hagyard Equine Medical Institute, Lexington, Kentucky.

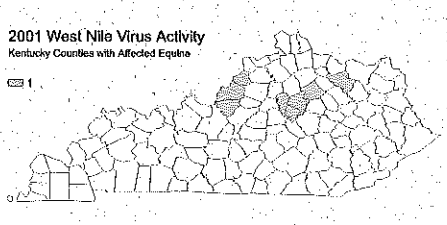
Kentucky's 2004 West Nile Surveillance Program

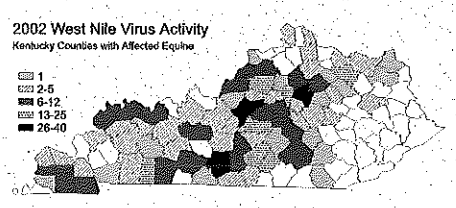
THE KENTUCKY DEPARTMENT OF Agriculture's Division of Animal Health again in 2004 investigated and reported to the state's Department of Public Health suspected cases of encephalitis affecting equines in Kentucky. A diagnosis of West Nile virus infection was confirmed in eight cases. Four survived the infection; three were euthanized; and one died. Two cases had received an initial West Nile vaccination but no booster; the remaining six had no vaccine administered during the previous 12 months.

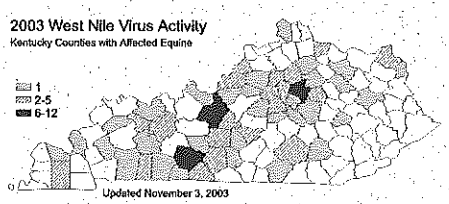
A complete and comprehensive three-year study of West Nile, its effect on Kentucky's equine population, and statistical groupings can be found on our Web site at http://www.kyagr.com/state_vet/ah/programs/equineprogs/index.htm.

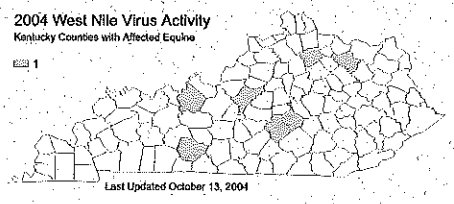
CONTACT: E.S. Rusty Ford, (502) 564-3956, Rusty.ford@ky.gov, Equine Programs Manager, Kentucky Department of Agriculture, Frankfort, Kentucky.

For more information on these or any of our other equine programs visit www.kyagr.com/state_vet/ah/index.htm.









Equine Disease Quarterly Newsletter

Department of Veterinary Science
Maxwell H. Gluck Equine Research Center
University of Kentucky
Lexington, Kentucky 40546-0099

Address Service Requested