EQUINE DISEASE QUARTERLY

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

JULY 2006

Volume 15, Number 3

IN THIS ISSUE

UK UNIVERSITY OF KENTUCKY
College of Agriculture
Department of Veterinary Science

LLOYD'S

COMMENTARY

DISEASE OUTBREAKS AFFECTING EQUINE populations in various parts of the United States evoke genuine concern for the welfare of the animals. This is apparent not just among individuals and groups within the industry, but in the general public as well. Recent examples of disease problems receiving considerable publicity include the outbreaks of paralytic disease caused by equine herpesvirus-1 (EHV-1) at racetracks in the Northeast during the early part of 2006, the considerable losses attributable to West Nile virus infection throughout the United States over the last six years, and the large number of fetal losses in Kentucky during 2001 and 2002 as a consequence of Mare Reproductive Loss Syndrome (MRLS).

Inevitably, bad news travels fast via television, radio, newspapers, journals, and the widely accessible Internet. In highlighting problems, the media often act as an additional stimulus to identify the cause and source as well as providing an educational role in publicizing measures for prevention and control as an outbreak progresses. The rapid introduction, within two years, of an equine vaccine against West Nile virus infection is a case in point, contributing to a reduction in the number of horses that have died from this disease following widespread publicity exhorting horse owners to vaccinate their animals.

Other diseases that hitherto have been of concern have not been in the headlines recently. The last major epidemic of equine influenza in the United States occurred in 1965, although equine influenza type 2 continues to circulate in the equine population. By a combination of natural immunity through exposure and induced immunity through vaccination, outbreaks are limited in geographical distribution and severity of clinical signs. Twenty-three horses—16 Warmblood stallions and seven mares imported to the United States from Europe since 1997—have been identified as positive for Contagious Equine Metritis (CEM) when tested in quarantine prior to their release. Failure to identify these animals would have undoubtedly resulted in re-introduction of CEM into the equine population.

The number of cases of EHV-1 abortion among the expanding pregnant mare population of Central Kentucky is at an all-time low. Over the last three foaling seasons there have been 58 confirmed EHV-1 abortions among Thoroughbred mares—less than two EHV-1 abortions per 1,000 pregnant mares. Similarly, the incidence of Equine Infectious Anemia (EIA) has dropped significantly within the state, from 20 cases in 1995 to zero in 2005. Within a similar time frame, the percentage of serological positives for Equine Viral Arteritis (EVA) based on the annual screening of yearlings and breeding stock going through the Kentucky sales has remained at six per 1,000 animals tested, testament to the efficacy of annual state-mandated live vaccination of Thoroughbred stallions.

These examples of "good news" do not, however, guarantee that disease outbreaks attributable to these pathogens will emerge in the future. Should this occur, pathogens will be quickly identified, and the inevitable publicity will contribute to their control and education of the horse-owning public.

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

INTERNATIONAL

First Quarter 2006

THE INTERNATIONAL COLLATING CENTRE, Newmarket, England, and other sources reported the following disease outbreaks:

African Horse Sickness affecting Thoroughbreds and non-Thoroughbreds was reported in several locations in South Africa. Identification of *Taylorella equigenitalis*, the bacteria causing Contagious Equine Metritis (CEM), was reported by four countries. In Ireland semen from a Dutch Warmblood stallion imported in June 2005 was found to be positive based on PCR (Polymerase Chain Reaction) testing. The stallion was only used for artificial insemination, and no natural matings have taken place. Mares inseminated with semen from the horse were negative for CEM. In Switzerland, six stallions—two Warmblood, and four Franches-Montagnes—involved in a research project at the Swiss National Stud were identified positive for the streptomycin sensitive strain of *T. equigenitalis* during a routine pre-season health screening in February 2006. Three Standardbred stallions imported to Sweden from Italy earlier in 2006 were positive for CEM when routinely tested prior to collection of semen for artificial insemination. A Warmblood mare in the United Kingdom imported from Germany was identified as CEM-positive following routine screening.

Equine influenza was widely reported in France, on a single premise in Ireland, three premises in Sweden, and a single premise in the United Kingdom. The respiratory form of equine herpesvirus (EHV) was reported among Thoroughbred weanlings on two premises in Argentina, on numerous premises involving different breeds of horses in France, and among yearlings in Ireland.

Abortion attributable to EHV-1 was diagnosed among various breeds on several premises in France and on premises in Germany, Ireland, Japan, the United Kingdom, and Central Kentucky in the United States. Reports of the neurological form of EHV-1 were received from Japan, United Kingdom, and the United States. The outbreaks in the United States occurred on three Thoroughbred racetracks and at two training facilities in Maryland and Pennsylvania.

A small number of cases of late fetal loss attributable to Mare Reproductive Loss Syndrome (MRLS) were diagnosed on two premises in Florida in April.

Strangles was widely reported in Ireland, South Africa, Sweden, and Switzerland.

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.ca.uky.edu/gluck/index.htm

Strangles Vaccines and Immunity to *Streptococcus equi*

EQUINE STRANGLES IS CAUSED BY *STREPTOCOCCUS EQUI*, a biovar, or clonal descendent, of an ancestral *S. zooepidemicus*. Recovery from the disease is accompanied by onset of acquired resistance to the disease in approximately 75% of horses, an immunity that persists for five years or longer—hence the greater incidence of strangles in younger horses. The immunologic basis of acquired resistance is not well understood but appears to function at the level of the tonsil-resistant horses that exhibit rapid tonsillar clearance of *S. equi* following experimental intranasal challenge. Although most horses are colonized by the closely related *S. zooepidemicus*, which shares many cross-reactive immunogenic proteins with the clonal *S. equi*, these horses are not protected against strangles. Conversely, strangles vaccines do not protect against respiratory or uterine disease caused by *S. zooepidemicus*. Thus, current vaccine research is heavily focused on immunogens expressed by *S. equi* but not by *S. zooepidemicus*.

Vaccination was first attempted over 100 years ago with a variety of live and killed preparations of *S. equi*. Studies in Australia in

the early 1940s indicated that useful protection could be stimulated using formalinized suspensions of gently-heat-inactivated, early-growth-phase bacteria. A similar vaccine marketed in the United States in the 1960s was associated with a high frequency of local and systemic reactions and later replaced by vaccines prepared from protein-rich acid and enzyme extracts. These modifications resulted in fewer adverse reactions and stimulation of high levels of serum antibody to the antiphagocytic SeM protein. However, although potent, their use in the field provided only marginal levels of protection. Since injected vaccines failed to induce the local mucosal immune responses that develop in convalescent protected animals, a non-encapsulated, attenuated *S. equi* was developed as an intranasal vaccine. A modification of this concept involving the injection of a live aroA$^-$ mutant of *S. equi* into the upper lip has been marketed in Europe. These live vaccines have the potential to cause abscesses following inadvertent entry into needle puncture sites. Other safety concerns include reversion to virulence and the occasional occurrence of purpura hemorrhagica and bastard strangles. Moreover, not all vaccinates make protective responses, which may indicate faulty administration or a non-responder horse.

Vaccines in Current Use

ACID AND ENZYMATIC EXTRACT VACCINES (United States, Australia): Naïve horses and foals require a schedule of two or three doses at an interval of two weeks. Booster doses may be given once annually. Mares boostered a month before foaling will have enhanced levels of colostral antibody. Animals with titers of 1:1600 or greater in the SeM ELISA should not be vaccinated because of the risk of purpura and because this level of antibody may signify an existing protected state. Also, horses known to have had strangles within the previous year need not be vaccinated.

INTRANASAL VACCINE (United States): Only healthy, nonfebrile animals free of nasal discharges should be vaccinated. Two doses are given at a two-to-three week interval and must be applied so that the vaccine reaches the naso and oropharyngeal tonsillar tissues. Foals may be vaccinated at 1 to 2 months of age, although there is an enhanced risk of mandibular abscessation in this age group. Use of live vaccine during an outbreak is controversial and perhaps should be restricted to animals with no exposure to known cases or exposed animals. It should be kept in mind that protective immunity will not develop until at least two weeks after vaccination and that the process of vaccination itself increases the risk of transmission of wild virulent *S. equi* should an infected horse unknowingly be handled during the procedure. However, numerous anecdotal experiences suggest that use of intranasal vaccination during an outbreak has been effective in stopping an outbreak.

MUCOSAL VACCINE (Europe): The vaccine is injected submucosally on the inside of the upper lip in a dose volume of 0.2 ml. Horses older than 4 months are vaccinated twice at an interval of four weeks to prime and subsequently every three months to maintain immunity. If continuous immunity is not required, a single dose will restore immunity in a primed horse at risk of exposure to *S. equi*.

Safety and efficacy issues are driving efforts to develop improved strangles vaccines in a number of laboratories around the world. Comparisons of the genomic sequences of *S. equi* and *S. zooepidemicus* have the potential to greatly accelerate discovery of vaccine components. However, since strangles is a disease with a complex pathogenesis involving several virulence factors that operate at different stages of infection, discovery of an effective combination of protective antigens and appropriate mode of delivery and presentation will require expensive and time-consuming experimentation in the natural host.

CONTACT: Dr John F. Timoney, (859) 257-4757, jtimoney@uky.edu, Maxwell H. Gluck Equine Research Center, University of Kentucky, Lexington, Kentucky.

NATIONAL

Drug Resistance of Equine Internal Parasites

THE MAIN INTERNAL PARASITES TRADITIONALLY considered to be important in horses are bots, ascarids, large and small strongyles, and pinworms. Other species, such as tapeworms, stomach worms, and intestinal threadworms also can be of clinical importance. Of the approximately 100 species of internal parasites in horses, about one-half belong to the small strongyle group. The following discussion concentrates on the strongyles and ascarids.

Strongyles live as adults in the large intestine and lay eggs that pass in the feces of horses. In the environment, the egg embryonates into a first-stage larva, which hatches. It then undergoes further development into a second and then into a third-stage larva, which is the infective stage. Grazing horses ingest the third-stage larva after it crawls up on pasture vegetation, hay, or other food. In the horse, the third stage develops through two more stages (the fourth and then the fifth, which is the adult). Large strongyles are more pathogenic than the small strongyles because the infective third stage migrates outside the large intestine into blood vessels and may go into internal organs. This may result in blockage of blood vessels and/or other problems that can result in debilitation or even death of the horse. Small strongyles in the infective stage migrate into the wall of the large intestine, where they encyst. These parasites can cause clinical problems (larval cyathostomiasis) and at times even result in killing the horse. During migration of large strongyles and encystment of the small strongyles, there is development to the fourth stage and, in some instances, fifth stage. During these latter stages, they return to the lumen of the large intestine, mature to egg-laying adults, and the life cycle continues.

Ascarid eggs that are passed in horse feces have a thick shell. Embryonation occurs in the egg in the environment to the infective larval stage; however, the larva does not hatch from the egg until ingested by a horse. These parasites migrate in blood vessels through the liver and then to the lungs, where they undergo further development before being swallowed again. They mature in the small intestine, where they can cause problems, including blockage or rupture of the wall. Ascarids can be the cause of death in young horses.

Over the years, several dramatic changes have occurred pertaining to chemical control of internal parasites. These are:

- Compounds have become much safer, and smaller amounts need to be used.
- Paste formulations have been developed, which are much easier to administer than the earlier liquid compounds that were given via stomach tube.
- Drug-resistance has become evident for several species.
- The number of commercially-available compounds, including entire classes, has decreased to a very low level.

New classes of compounds have not been developed for more than 20 years.

Fortunately, at this time the most pathogenic parasite species (the large strongyles) have been controlled so well that they are virtually absent in horses on farms with good parasite control programs. There has been no apparent resistance exhibited by these parasites; however, surveillance should be maintained to insure their continued control by antiparasitic products.

Resistance among small strongyle species currently has not been found for ivermectin and moxidectin but has been documented for all other commercially-available products, i.e., the benzimidazoles (fenbendazole, oxfendazole, and oxibendazole), the tetrapyrimadines (pyrantel pamoate), and piperazine. Recently, there have been indications that ivermectin and possibly moxidectin are much less effective against ascarids in foals than found previously.

Typically, drug-resistance in the small strongyles occurs after a period of usage of anthelmintics. Even though the macrocyclic lactones (ivermectin and moxidectin) still are highly effective on these parasites, it is possible resistance will develop. It is suggested that these compounds be used sparingly to prolong their effectiveness on small strongyles.

Various treatment recommendations are used for parasite control, including daily feeding of pyrantel tartrate and therapeutic administration of compounds. The latter involves periodic treatment—e.g., monthly, bimonthly, and seasonal (spring and summer). One of the most practical and cost-saving measures is to do counts of strongyle eggs per gram of

feces (EPGs). Then, only treat horses with EPG counts above a certain level. Some farms use a value of 40 or 100 before administering a dewormer. To determine the efficacy and possibility of resistance to a certain drug, an EPG count should be done on feces from a horse on the day of treatment and again two weeks later to be sure the drug effectively removed the adult egg-laying worms.

Regarding control of ascarids, which commonly occur in young horses, the recent finding of apparent drug-resistance of ivermectin and possibly moxidectin is alarming. This means it is important to determine by fecal examination, as stated above with the small strongyles, if these or other products are still effective in foals. It is recommended to treat foals every six to eight weeks with an effective ascaridicide to remove the worms before they mature. If the ascarids are allowed to mature, which occurs about 10 to 12 weeks after initial infection, then at least two possible life-threatening situations may occur regarding the small intestine of the infected horse: the wall may be ruptured by the action of the ascarids or the lumen may be impacted with dead large ascarids after treatment.

Research on parasite control is a priority in the Classical Parasitology section in the UK Department of Veterinary Science. This involves monitoring the level of parasitism among horses, particularly young animals, on local farms.

CONTACT: Dr. Eugene Lyons, (859) 257-4757, elyons1@uky.edu, Maxwell H. Gluck Equine Research Center, University of Kentucky, Lexington, Kentucky.

KENTUCKY

Skull Fractures in Horses

HORSES OFTEN ARE VICTIMS OF ACCIDENtal injury. Their gregarious nature, social hierarchy, heightened flight response, and handling and confinement by humans puts them at increased risk of trauma. A fairly common and usually catastrophic injury of horses is trauma to the head resulting in fracture of the skull.

The head of a typical adult horse weighs in excess of 40 pounds. This, coupled with the long neck placing the head well outside the center of mass, causes the head to strike the ground with tremendous force during a fall. Also, the speed and strength of a horse can result in severe impact of the head against an object when rearing or running.

Over the past five years, the University of Kentucky Livestock Disease Diagnostic Center has diagnosed 34 cases of skull fracture, with a yearly average of 6.8 cases. These were diagnosed in several breeds, with Thoroughbreds predominating. The affected horses ranged in age from 2 days to 23 years. Most cases were adult horses, but yearling and younger horses were affected as well.

Horses suffering a skull fracture often had a history of being handled with the horse rearing and flipping over, striking its head on the ground, or hitting its head on an overhead structure such as a trailer or stall ceiling. In other instances the history indicated the horse running directly into an object, such as a tree, fence, or barn. Occasionally, the horse was simply found dead with the causative event not being observed. The clinical signs in horses suffering a skull fracture included ataxia, recumbancy, paralysis, blindness, nystagmus, seizures, coma, and death. Hemorrhage from the nose or an ear was sometimes observed. Medical treatment was usually attempted; however, the horses often died or the severity of the injury necessitated euthanasia.

The diagnosis was made or confirmed at

necropsy. The pathological findings among cases were similar. These sometimes included scrapes or excoriation of the skin on the head and bruising and hemorrhage of the subcutaneous tissue at the point of impact. Hemorrhage in an ear canal, nasal passages, or a gutteral pouch was not unusual. Commonly, there was epidural hemorrhage and tearing of the meninges, with hemorrhage into the brain and around the anterior cervical spinal cord. Fractures involved many of the different bones comprising the skull. The structure of the skull makes the occurrence of one fracture unlikely. The force resulting in a fracture and displacement in one area is transferred across the skull, fracturing another area. The occipital bone was most commonly fractured, with a concurrent fracture of the basisphenoid bone. These fractures sometimes involved the acoustic meatus, with bleeding into the ear. Fractures of this type are typical in a horse that flips over backward, striking its head. In contrast, running into an object or being kicked was associated with fracture of the frontal bone. Other bones fractured in this type of injury included the parietal, temporal, and zygomatic bones. Fractures of the frontal bone were often associated with hemorrhage into the frontal sinus and nosebleed.

It is important to recognize that horses are susceptible to this type of injury and how serious head injury can be. Special care is warranted when handling extremely nervous or "flighty" horses so that they are not put in situations or an environment where injury is more likely to occur if the horse becomes unruly or panics.

CONTACT: Dr. Neil Williams (859) 253-0571, nmwillia@uky.edu, Livestock Disease Diagnostic Center, University of Kentucky, Lexington, Kentucky.

Equine Disease Quarterly Newsletter

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

Address Service Requested