Equine Disease Quarterly

April 1993
Volume 1
Number 3

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

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In this issue

Commentary

Despite the use of equine influenza vaccines for almost 30 years, the disease continues to disrupt equestrian competitions, including racing, as evidenced by the recent epidemic in Hong Kong.

Two types of equine influenza are recognized, equine-1 and 2. Equine-1 infection was last reported in 1980 from Yugoslavia; its absence over an extended period raises the question as to whether it is still circulating in the equine population. Equine-2 is extremely prevalent, and in recent years has become endemic in North America, Europe, and Scandinavia. Major epidemics among horse populations previously unexposed to equine-2 influenza occurred in South Africa during 1986, India 1987, China 1989, and Hong Kong 1992. Evidence from three of these outbreaks suggests virus was introduced following transportation of infected horses by air from either North America or Europe.

The ability of influenza viruses to undergo change (referred to as "antigenic drift") is well recognized. It is likely that antigenic drift has compromised the efficacy of current vaccines; as a consequence there is a need to update the strains contained in these vaccines. Licensing requirements imposed by federal authorities in the United States and Europe caused biologics manufacturers to be hesitant in introducing new strains.

The need to improve equine influenza vaccines is recognized, although additional precautions would reduce the spread of the disease. Veterinary authorities responsible for certifying the health status of horses prior to shipment must ensure health inspections are carried out in a thorough manner. Increased surveillance is necessary to isolate and characterize influenza viruses, thereby providing data for vaccine strain selection.

The risk of future outbreaks will be reduced when only healthy horses are permitted to travel and the equine population is immunized regularly with effective vaccines.

International

Fourth Quarter 1992

The International Collating Centre, Newmarket, England, and other reporting agencies confirm the following disease outbreaks:

Equine-2 influenza was reported from Denmark, France, Hong Kong, Italy, Sweden, and the United Kingdom. Finland reported contagious equine metritis (CEM) for the first time, among Standardbred mares artificially inseminated with semen from a temporarily imported stallion. CEM occurred among non-Thoroughbreds in Japan, Sweden, and the Netherlands. African horse sickness and dourine were reported from South Africa.

Cases of strangles were reported from Denmark, Ireland, Italy, New Zealand, Norway, Sweden, Switzerland, and the United Kingdom and *Salmonella typhimurium* from Australia, New Zealand, and the United Kingdom. Equine rhinopneumonitis (EHV-4) was reported from Australia, Ireland, and Switzerland.

Hong Kong Flu

Influenza A equine-2 virus was isolated during the recent epidemic that affected approximately 400 of the 958 Thoroughbred horses stabled at the Sha Tin racing and training complex in Hong Kong.

Signs of influenza, previously unrecorded among horses in Hong Kong, were first observed in mid-November 1992 and persisted for one month; this caused the cancellation of seven race meetings. The majority of horses had received an influenza booster vaccination in June 1992. Horses and ponies at one of

continued

the territory's nine riding schools were also affected.

Restrictions on movement and the cancellation of equestrian events were imposed and no further outbreaks were reported. The source of the epidemic was probably an infected horse(s) imported from England and Ireland during October.

Clinical signs including fever, nasal discharge, congested mucous membranes, and a frequent dry cough were of a mild nature. Racing resumed on December 23 and no adverse affects have been reported.

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Equine Protozoal Myelitis: Greater Diagnostic Capability

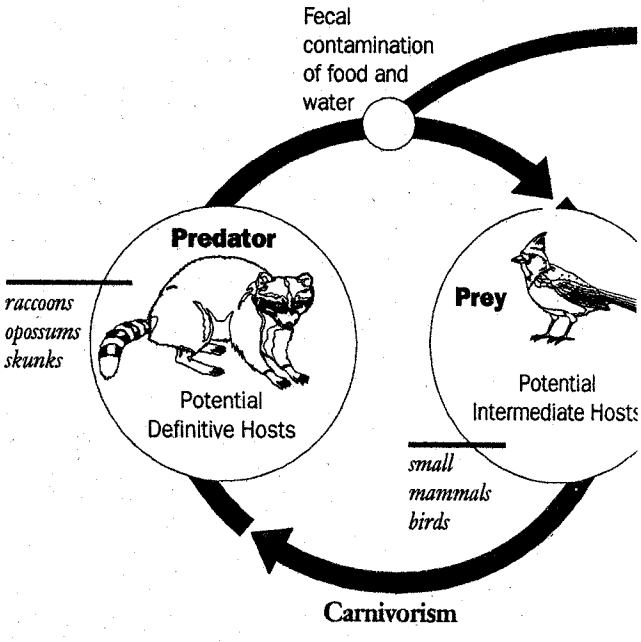
Equine protozoal myelitis (EPM) is a sporadic disease of horses which most commonly results in asymmetric incoordination and weakness, although it may mimic almost any neurologic condition. The causative organism is *Sarcocystis neurona*, a protozoal parasite.

The life cycle and mode of transmission of this parasite to horses has not yet been determined; however, several conclusions can be made based on our knowledge of related organisms. *Sarcocystis* spp. have a 2-host predator-prey life cycle, as illustrated in **Figure 1.**

Horses represent an aberrant host of *S. neurona.* Sporocysts are ingested and migrate to the central nervous system causing pathological lesions. The disease is not contagious; horses do not transmit *S. neurona* to other horses. Transmission from the horse to a carnivorous definitive host does not occur.

The definitive host is unknown, but must be an animal which has access to grain, hay, or pasture and sheds sporocysts into the feed which is subsequently eaten by the horse. Domestic animals have not been implicated and the source of equine exposure is most probably wildlife, including raccoons, skunks, or opossums.

Figure 1
Possible life cycle of
Sarcocystis neurona



EQUINE DISEASE QUARTERLY

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Using the technique of western blot analysis, a diagnostic test has been developed at the University of Kentucky Department of Veterinary Science which is highly specific for *S. neurona*. It is being used to evaluate the presence or absence of *S. neurona* antibodies in serum and cerebrospinal fluid. A positive test on serum reflects exposure of the horse to the organism, but not necessarily clinical disease.

Results suggest that as many as 20% of horses sampled in Ohio and Kentucky have been exposed to *S. neurona* without developing EPM. It is likely these horses developed an immune response, eliminating the organism before it reached the central nervous system. A positive test on serum alone is of limited value. Rare cases of EPM may be negative for *S. neurona* antibodies in serum, but positive in cerebrospinal fluid.

A positive test on a clean, non-blood contaminated sample of cerebrospinal fluid reflects active production of *S. neurona* antibodies, and is highly correlated to clinical disease. This is currently the most sensitive antemortem test available for EPM. The majority of EPM-affected horses should have a positive test on cerebrospinal fluid and serum. The comparison of serum and cerebrospinal fluid antibodies facilitates interpretation of the test, and both samples should be collected whenever possible.

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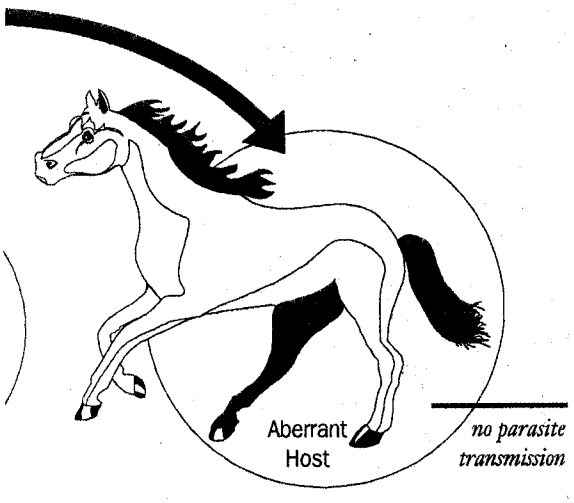


Equine Encephalomyelitis: Fewer Cases in 1992

Eastern and western encephalomyelitis (EEE, WEE) are caused by insect-transmitted arboviruses which damage the nervous system. Clinical signs include fever, anorexia, depression, incoordination, blindness, convulsions, and frequently death. The disease is confirmed by isolation of the virus from the brain of a dead horse or a rise in antibody titer in blood samples taken several weeks apart from a live horse.

Figure 2 shows the geographical distribution of cases of EEE and WEE in the United States during 1992 as reported by the National Veterinary Services Laboratories, the Centers for Disease Control (CDC), and state veterinary diagnostic laboratories. (Fourth quarter figures for 1992 were not available from CDC.)

The cases of equine EEE (88) and WEE (8) are a significant decrease compared to 1991 (**Figure 3**) when more than 150 cases of EEE occurred in Florida alone. Although no cases were diagnosed in some states, this does not indicate that encephalomyelitis cases did not occur, only that none were confirmed.

Infected mosquitoes can transmit the disease to humans. During 1992 one human case of EEE was reported from Massachusetts and one fatal case from Florida. No human WEE cases were reported.

Birds serve as the reservoir host; during 1992 pheasants in South Carolina were diagnosed with EEE, and emus in Georgia, Oklahoma, and Texas with EEE and WEE. Emus are short flightless birds related to ostriches.

The single case of EEE in Kentucky was a horse from Owen County which showed clinical symptoms of encephalomyelitis and subsequently recovered. During 1991, a mixed breed yearling died of EEE in Laurel County.

Figure 3 shows a dramatic increase in the number of equine EEE cases during 1991. This is due in part to weather factors, which were favorable to the mosquito vectors. Studies have shown that increased precipitation in the fall of the preceding year is associated with EEE outbreaks. The decline in cases in 1992 may be explained not only by differing weather conditions, but also public awareness of the disease and increased vaccination of horses.

Effective EEE and WEE vaccines are available. All horses should be vaccinated, even in states where the diseases are sporadically reported. Horses along

Figure 2
Cases of equine encephalomyelitis, 1992

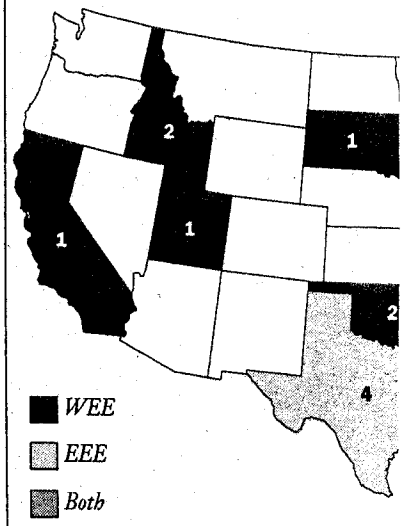
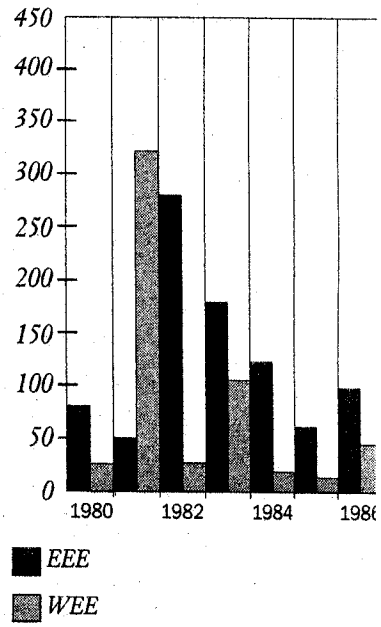
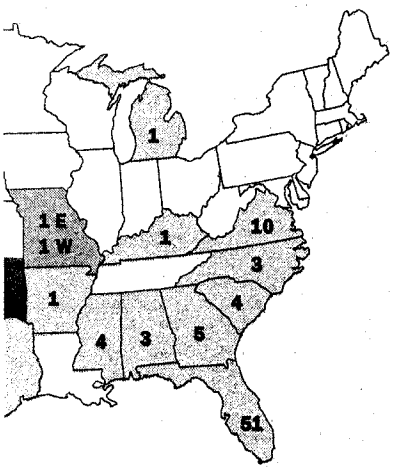


Figure 3
Annual cases of equine encephalomyelitis





the Atlantic Coast and southeastern states should be vaccinated regularly, as well as horses traveling to these areas for show, race, or breeding purposes.

The recommended vaccination schedule against EEE/WEE is:

Foals: Vaccinate at 3, 4 and 6 months; then every 6 months.

Broodmares: Schedule revaccination 3-4 weeks prior to foaling. Revaccinate in 6 months.

Adults: Start with 2 doses 3-4 weeks apart. Revaccinate every 6 months.

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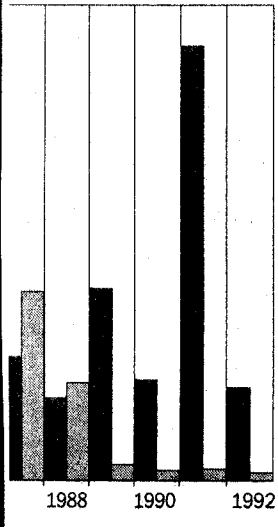
Kentucky

Equine Placentitis

Placentitis is one of the most important causes of equine abortion and stillbirth. During the 1988 and 1989 foaling seasons it accounted for 236 of 954 instances of reproductive loss in mares based on submissions to the Livestock Disease Diagnostic Center. The total comprised 714 Thoroughbred cases, 92 Standardbred, 30 American Saddlebred, and 118 other breeds.

Microorganisms associated with placentitis were isolated or demonstrated from 162 cases. Major pathogens identified in decreasing order were *Streptococcus zooepidemicus* (39), *Leptospira* spp. (37), *Escherichia coli* (33), nocardioform actinomycete (16), fungi (15), *Pseudomonas aeruginosa* (11), *Streptococcus equisimilis* (11), *Enterobacter agglomerans* (5), *Klebsiella pneumoniae* (5), and alpha-hemolytic *Streptococcus* (5). Pathogens were not recovered from 64 cases and overgrowth by saprophytic bacteria was recorded for 10 cases. In this study, 27 cases had mixed bacterial growth and 93 cases had bacteria cultured both from placenta and fetal organs.



Leptospira spp. and nocardioform actinomycete are two important, newly-emerging bacteria associated with equine placentitis. Regardless of gestational age, *Leptospira* spp. induced a diffuse placentitis with the presence of a large number of spirochetes in the placental tissues. The nocardioform actinomycete, a gram-positive, filamentous and branching bacillus, induced a unique chronic-active, focally extensive placentitis located at the base of the horn or at the junction between the body and horn of the allantochorion.

With the exception of *Leptospira* spp., most cases of bacterial or mycotic placentitis are ascending infections. In this study, the bacteria isolated from the placenta were comparable to those isolated from the outer genital tract. The lesional distribution also supported this ascending infection hypothesis, especially lesions caused by fungi and those of chronic placentitis, which frequently affected the cervical star area. However, some bacteria, including *Leptospira* spp., were able to reach the placenta via the bloodstream; especially those cases in which abortion occurred during the early stage of gestation.

Gestational age plays a major role in the development of placental lesions, although microbes also dictate the reaction of the placenta. Placentitis associated with fetuses expelled early in the stage of gestation was frequently acute and bacteremic. In contrast, placentitis observed in late gestation was usually focal and chronic with a severe tissue reaction.

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"Wobbler" Syndrome: No Change

Figures compiled between 1987 and 1991 from necropsies submitted to the Livestock Disease Diagnostic Center indicate that "wobbler" syndrome is the most common neurologic disease seen in the central Kentucky equine population.

Generally, the cases had a history of 2 or more months of incoordination, although a few cases were found dead with no prior history of illness. All 264 cases reported in this series had a demonstrable narrowing of the cervical vertebral canal and microscopic lesions consistent with impingement on the

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Figure 4
Wobbler syndrome breed distribution

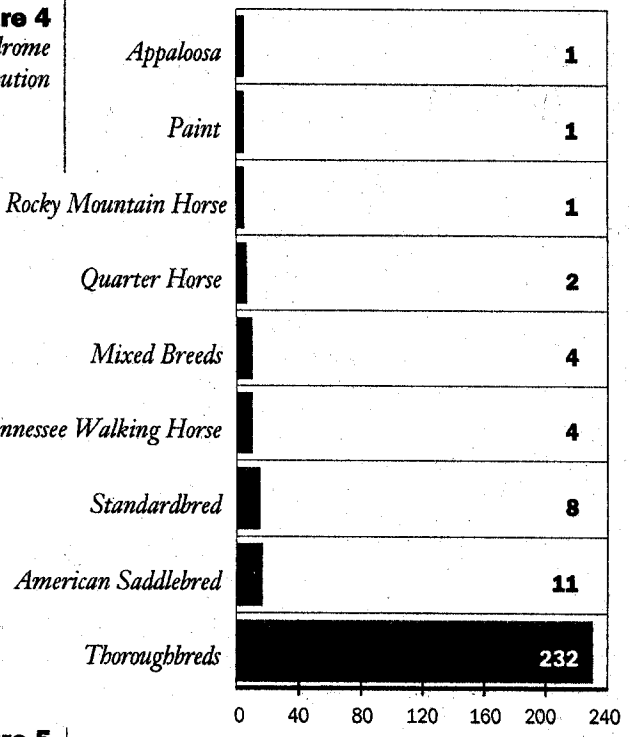
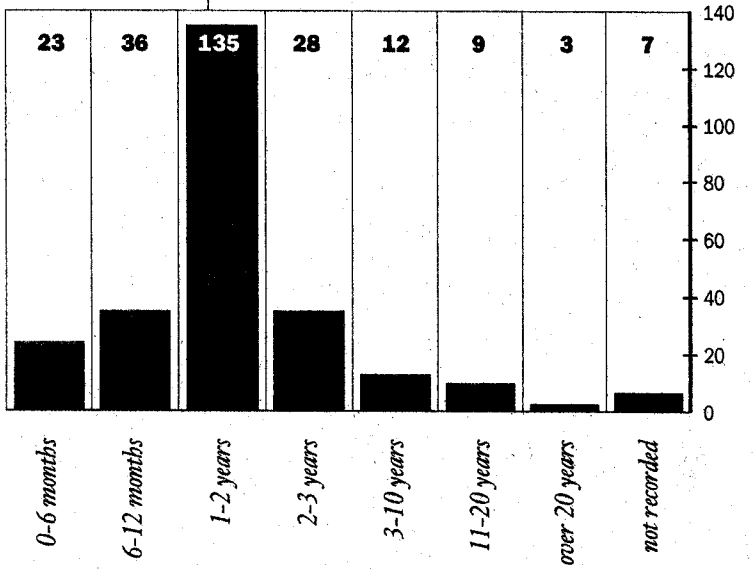


Figure 5
Wobbler syndrome age distribution



cervical spinal cord. Although most cases were Thoroughbreds, cases were identified in 7 other breeds and 4 were mixed breeds (**Figure 4**).

Horses less than 2 years of age comprised 77% of the cases (**Figure 5**). Historically, "wobbler" has been synonymous with ataxia of foals; and the current data continue to support the view that growth and skeletal tissue development are key factors in the cause and expression of this problem. Nearly 75% of the cases were male but females represented 62% of the cases greater than 2 years of age. Does this sex difference represent growth associated risks for each sex?

Cervical vertebral stenosis and associated spinal cord lesions occurred at all intervertebral joints except C_1-C_2. Grossly evident compression of the spinal cord was most frequently and easily seen at C_3-C_4. Curiously, C_5-C_6 was the second most often recognized site associated with functional compression.

Multiple compression sites were observed in 89 cases, and these generally extended from C_3-C_4 through C_5-C_6. A variety of lesions involving the articular facets were observed; however, no pattern of cartilage/bone lesions was readily apparent.

The number of "wobbler" cases diagnosed each year in central Kentucky is remarkably consistent. This condition will probably remain a significant health problem related to raising foals until the cause is clearly defined.

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Mares Imported from CEM Countries

Permits issued by the Kentucky State Department of Agriculture provide data on the number of mares imported from countries which have reported contagious equine metritis (CEM).

Since 1982, 1,487 mares and fillies comprising 1,208 Thoroughbreds and 279 non-Thoroughbreds have been imported. The non-Thoroughbreds included competition horses, primarily polo and hunter/dressage fillies. Of the total, 87% originated from England, Ireland, and France; the remainder were from Australia, Denmark, Germany, Italy, Japan, Netherlands, Norway, and Sweden. They do not include fillies entering the United States and Canada to race and subsequently sent to Kentucky to be mated. The number of mares imported each year, as

shown in **Figure 6**, indicates a decrease from 234 in 1983 to 64 in 1990, although numbers have risen since then.

Mares and stallions from countries which have reported CEM must undergo a rigorous screening program in the country of origin and on arrival in the United States, as regulated by the USDA. Countries reporting CEM include Austria, Belgium, Czechoslovakia, Denmark, Ireland, Italy, Japan, Germany, France, Netherlands, Norway, Sweden, Switzerland, and the United Kingdom.

Stallions and mares imported from any foreign country must, according to Kentucky state regulations, undergo testing and treatment before entering the breeding shed. Stallions should be treated and mated with two test mares that are subsequently cultured and blood tested. Mares must be blood tested, cultured and treated before they are mated. The imported mare should be the last mare bred on the day, and the next three mares bred to the same stallion should be blood tested 15 to 40 days after they are mated. Precise details of these procedures can be obtained from Rusty Ford.

Signs of CEM range from a profuse vaginal discharge, developing several days after the mare is mated, to no obvious signs with mares coming into season between 8-15 days.

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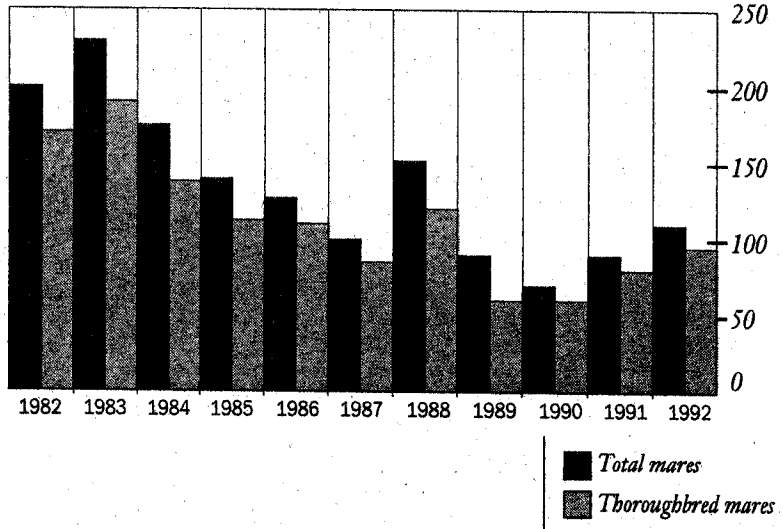
Figure 6
Mares imported from CEM countries



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