

October 1993

Volume 2 Number 1

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

University of Kentucky

College of Agriculture

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International



The recently reported outbreaks of EVA in North America and England pose the question as to whether the incidence of this disease is increasing.

Over the years it has been recognized that the prevalence of EVA infection is higher among various breeds of non-Thoroughbreds as compared to Thoroughbreds. The outbreak among Thoroughbred race horses at Arlington is suspected to have originated as a result of cross infection between horses of different breeds stabled together. The suspicion in England is that their outbreak originated with an imported eventing horse.

The ever-increasing movement of horses nationally and across country and continental boundaries allows for greater contact within and between various breeds. As a consequence, the incidence of disease attributable to viral agents such as influenza, equine rhinopneumonitis or herpesvirus and equine arteritis virus has increased. Although restrictions on movement will reduce the spread of disease, it goes against the current trend of encouraging the free movement of horses for racing, competition, breeding, and sale. The alternative is to establish an immunized population through the use of safe and efficacious vaccines.

The recent EVA outbreak in North America has highlighted the importance of communicating up-to-date and accurate information particularly to state veterinarians and racetrack officials across the country. This would assist them in making objective decisions, thereby reducing the spread of equine infectious disease following interstate movement. The provision of a clearing house or collating center as presently exists for the reporting of equine disease outbreaks at an international level would achieve that goal.

Second Quarter '93

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

Equine-2 influenza was reported from Denmark, France, Norway, Sweden, Switzerland, United Kingdom, and the United States. From March through June cases of equine viral arteritis (EVA) were confirmed among approximately 100 horses on 6 premises in England. The outbreak is believed to have originated with an Anglo-Arab stallion imported as an event horse from Poland in 1992. This horse covered non-Thoroughbred mares in March 1993 and semen from the stallion was used to inseminate mares on 4 other farms.

Mares developed fever, conjunctivitis, and edema of the limbs. Clinical signs were also observed among foals and two other stallions at the farm. Equine arteritis virus was isolated from the respiratory tract of mares and the semen of 2 stallions including the Anglo-Arab. The last clinical case was reported on June 6.

An inactivated or dead vaccine ARTERVAC® (Fort Dodge Laboratories, IA) developed in the United States has been granted a provisional license enabling trials to be undertaken among mares and stallions on farms in the United Kingdom where there is a recognized risk of infection.

Venezuelan Equine Encephalitis (VEE)

During July the USDA confirmed an outbreak of VEE among horses in the state of Chiapas, Mexico, close to the border with Guatemala. Early reports indicate there were approximately 70 cases with 56 deaths. Restrictions on horse movement have been imposed, and a vaccination program using the TC-83 live vaccine began on July 17. By July 28, over 15,000 horses had been vaccinated. Horses entering the United States from Mexico are now required to undergo a 7-day quarantine in vector proof facilities.



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Racetrack Outbreak of EVA

During the middle of July, a number of Thoroughbred racehorses stabled at Arlington racetrack in Illinois developed fever, swollen legs, and a skin rash. The signs persisted for 7-10 days with affected animals making an uneventful recovery and returning to training.

Samples obtained from sick horses and sent to the Maxwell H. Gluck Equine Research Center, University of Kentucky, Lexington and the National Veterinary Services Laboratory, Ames, Iowa, confirmed serological evidence of equine viral arteritis infection and the presence of the virus on July 30. As of August 31, 190 cases had been reported in 9 barns.

The outbreak is suspected, but not proven, to have originated in early July with horses that became sick after they had visited a local equine hospital. In response to the outbreak, movement of horses from affected barns at Arlington was restricted and vaccination of 1,700 healthy horses with the modified live vaccine ARVAC® (Fort Dodge Laboratories, Iowa) was completed by August 13.

Toward the end of July clinical signs consistent with EVA were observed among horses at Churchill Downs, Kentucky and Ak-Sar-Ben, Nebraska. Samples obtained from sick horses confirmed the presence of EVA infection among 4 horses at Ak-Sar-Ben and 8 horses at Churchill Downs. To date there has been no further evidence of spread of infection at these racetracks. Both outbreaks originated following the movement of horses from Illinois in early July. Clinical cases have also been reported at Prairie Meadows, Iowa, and serological evidence suggests they have been exposed to EVA.

At a meeting convened by Arlington management on August 11 it was recommended that horses visiting the racetrack should be vaccinated 14 days prior to arrival and undergo a minimum period of isolation for 14 days, either in Illinois or in their home state, after racing. Since vaccinated horses will test serologically positive for EVA it is recommended that vaccinated horses, particularly colts, should have a pre-vaccine blood sample drawn. The serum sample can then be

tested to confirm the animal's negative EVA status prior to vaccination.

Stallions which become infected with equine arteritis virus have the potential to become shedders of the virus in their semen and can infect susceptible mares. The ratio of infected stallions which become shedders ranges from one in three to two in three. A stallion can shed the virus in semen for weeks or for his entire lifetime.



Restrictions on the movement of horses from areas affected with the disease have been introduced by many states. Remington Park in Oklahoma, which commenced racing on September 17, required all horses to be vaccinated prior to entering their facility. It is recommended that prior to shipping horses any restrictions that are in place are determined by contacting the office of the state veterinarian or the racetrack veterinarian's office in the state of destination. In addition, it is



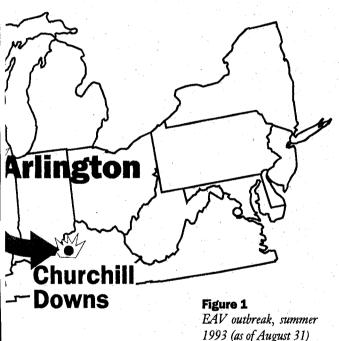
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strongly recommended that horses moved to farms from the racetrack are kept completely separate from pregnant mares, foals, and yearlings for at least a month. Pregnant mares that become infected with EVA have been known to abort.

Racetrack veterinarians are requested to be particularly alert to cases of fever, swollen legs, and skin rash occurring among horses grouped together, and to suspect EVA—particularly if there is a history of recent introduction of a horse(s) from a racetrack at which EVA has been confirmed. The incubation period appears to range from 6 to 11 days.



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Blood Typing and Identification

Blood typing has been used by horse breed registries for many years as a means of identification and parentage verification. Due to the number of gene marker systems tested, blood typing is an extremely powerful means of identification. This power also has been used in a variety of other circumstances that are not so well known.

One of these areas is drug testing. If a horse tests positive for a prohibited drug after a race, owners or trainers may claim that the blood or urine sample with the positive test was not taken from their horse. Blood typing methods can be used to compare the urine or blood samples that tested positive for the drug with another sample drawn from the suspected horse.

From a blood sample 10 or more gene marker systems can be tested. From a urine sample only five marker systems can be typed and this requires intensive concentration of proteins in the urine. A single difference in the types is proof that the samples came from different individuals. However, if the two samples are the same, this is not proof that the samples are from the same individual.

It is possible to give an estimate of the probability of two horses having identical types based upon the frequency of the genetic markers within the breed. For Thoroughbreds, the probability of two individuals having identical types at the five systems detectable from urine is 1 in 15 for the most common markers. If less common markers are found, the probabilities become very small. When all the genetic markers normally tested by blood typing can be used, the probability that two Thoroughbred horses will have the same type is no more than 1 in 25,000. For breeds such as Quarter horses, the probabilities exceed 1 in a million.

The Equine Blood Typing Research Laboratory of the University of Kentucky has performed several such comparisons of drug positive samples with blood samples of the horses provided by the owners. In all cases, the types have been the same.

Another case where the laboratory was requested to use the identification power of blood typing involved a horse that had a positive Coggins test for equine infectious anemia (EIA). When a horse tests positive for EIA, restrictions imposed by states differ considerably; but the horse is usually quarantined or destroyed. In this



instance, an individual was attempting to sell a horse that was suspected of being EIA positive. The state veterinarian provided the sample used for the Coggins test and a blood sample freshly drawn from the horse in question. The blood types of the two samples were identical and the owner of the horse was fined.

The power of blood typing as a tool for identification has many potential uses. However, its primary use remains parentage verification to maintain the integrity of the stud books of horse registries.

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Yew Poisoning in Horses

apanese and English yew (*Taxus*) are ornamental evergreens often seen around homes and driveways as well as in landscaping adjacent to barns. This shrub is one of the most poisonous plants to horses; ingestion of as little as 3 oz. causes sudden death.

Poisoning occurs when horses are fed trimmings of shrubs by owners or well-intentioned neighbors and when horses escape enclosures and eat the plant. Death often occurs while the animal is eating the leaves or twigs. Otherwise horses show muscle tremors, difficulty in breathing, collapse, convulsions and death within minutes.

Taxine is the toxic compound and causes rapid heart failure for which there is no treatment. Diag-

mosis is made post mortem by finding leaves and twigs in the mouth and stomach.

Horse owners are advised

not to plant these shrubs on farms and should inform neighbors not to feed horses clippings of any plant material. Likewise, horse show and event organizers should avoid using these plants as decoration in the show ring or on the show grounds.

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How Ivermectin Affects Strongyle Transmission

nformation on effects of an antiparasitic compound on reproduction and transmission of internal parasites is important. Passage of parasites out of the gastrointestinal tract of horses after treatment with an effective compound usually takes several days. However, very little is known about how soon parasite eggs are affected by the drug. Knowledge about this is critical in deciding how long to quarantine or isolate newly-introduced horses on a farm or horses to be placed on pasture free of parasitic larvae, such as newly-renovated areas.

Recently, two tests (A and B) were completed to determine how long horses should be isolated after ivermectin treatment to minimize transmission of small strongyles. Effect of treatment was determined by reducing egg and larval counts in fecal samples at posttreatment intervals. Larval counts are derived by culturing feces in order for worm eggs present to develop into larvae.

Administration of ivermectin, liquid or paste formulation, was directly into the mouth of treated horses. Four horses were treated with each formulation and 2 horses were nontreated controls. Ivermectin was given once at the therapeutic dose rate (200 µg/kg) to each treated horse. Fecal samples for small strongyle egg and larval counts were collected at treatment and every 4 hours up to 60 hours, continuing at longer intervals.

For horses treated with the liquid formulation, negative egg counts were found at 50 (A) or 60 (B) hours posttreatment and negative larval counts by 28 (A) or 56 (B) hours posttreatment. Horses treated with the paste formulation had egg counts of 10 at 50 hours and 0 by 72 hours posttreatment. Larval counts for paste-treated horses were negative at 32 (A) or 48 (B) hours after treatment. There was evidence based on incomplete development of larval stages in some instances that treatment adversely affected the small strongyle larvae even before counts were negative.

Data from this research, although only from a few horses, indicate that horses should be isolated for about 3 days after treatment with ivermectin to minimize contamination of pasture with small strongyle eggs.

This information is derived from:

D.B. Berry II, E.T. Lyons, J.H. Drudge, S.C. Tolliver, D.E. Granstrom, and K.J. McDowell. 1993. "Observations in Horses on the Effects of Ivermectin Treatment on Strongyle Egg Production and Larval Development," Journal of the Helminthological Society of Washington, 60:89-92.

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Figure 2. Yew. Taxus spp.

Post Partum Deaths of Mares

healthy foal is delivered during an uncomplicated foaling and all appears normal until the mare starts to tremble, then collapses and dies. An analysis of records at the Livestock Disease Diagnostic Center for the 1992 and 1993 foaling seasons provided the following interesting observations about mares that died shortly after foaling. Mares that died varied in age from 3 to 24 years with the majority being in the 11-15 year range, mirroring the general population of mares in central Kentucky. The month of death is shown in Table 1.

Table 1 *Monthly distribution*of mare deaths

	Jan	Feb	Mar	Apr	May	June	July
1992	2	13	14	7	12	2	1
1993	2	6	15	8	14	1	1

Of 98 records reviewed, reproductive complications caused deaths in 57 mares (Figure 1), with uterine artery rupture accounting for 40 cases. This often occurred immediately after foaling and since hemorrhage was internal, no bleeding was observed from the vagina and death was rapid. One mare survived 45 minutes before dying of a ruptured uterine artery. Rupture of the right uterine artery was twice as common as the left, although the significance of this is not

known. Other complications included uterine perforation and prolapse, vaginal tears, and uterine hemorrhaging. One mare died 16 days post partum from uterine abscesses, hemorrhage, and a vaginal tear.

Gastrointestinal disorders were the second most common cause of death with 36 cases, 19 caused by perforation of the cecum. The reason why the cecum is affected with such frequency is not known. Rupture of the stomach, small colon, and rectum also occurred. Most histories indicated mares had a normal foaling but then developed post partum colic, necessitating surgery at which time the ruptured organs were discovered. Any major rupture of the gastrointestinal tract usually requires euthanasia as horses are susceptible to fatal peritonitis.

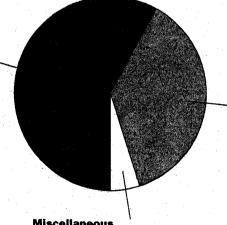
There is no way to predict if mares will develop post foaling complications, especially rupture of the uterine artery. Minor post partum hemorrhages can be treated successfully and uterine ruptures and prolapses corrected surgically. Farm managers should carefully observe the mare after foaling and obtain veterinary advice promptly if complications arise.

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Figure 3
Causes of post-partum death in 98 mares (1992-93)

Reproductive Complications	57
Ruptured Uterine Artery	40
Perforated Uterus	6
Pelvic/Uterine Hemorrhage	4
Prolapsed Uterus	
Vaginal Tear	2
Prolapsed Small Intestine	1
Ruptured Uterus & Rectum	



Gastrointestinal Complications 36 Cecal Perforation 19 Ruptured Stomach 5 Ruptured Small Colon 5 Torsion of Large Colon 4 Colic 1 Peritonitis 1 Ruptured Rectum 1

Miscellaliguus	2
Cardiac Dysfunction	2
Fractured Pelvis	
Ruptured Abdominal Aorta	
Ruptured Bladder	

EIA Surveillance

During 1992 a total of 65,634 equine blood samples were tested for equine infectious anemia (EIA) by the Coggins test. The majority of samples, 47,653 (private tests), were submitted by horse owners to comply with state regulations. An additional 14,938 (market tests) were obtained from horses going through markets and stockyards; 361 tests were a result of tracing incontact horses associated with positive cases.

Thirty five horses tested positive, 16 by private tests, 12 by market tests, and 7 as a result of tracing.

For the first six months of 1993, a total of 43,273 samples were tested. Fourteen horses tested positive, 6 as a result of private tests and 8 from market tests.

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