

# Equine Disease



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## Commentary



## International



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In this issue the significance of several bacterial diseases to the equine population is considered. Each year salmonellosis and *Rhodococcus equi* infections contribute directly and indirectly to the deaths of between 50 and 100 foals born on farms in central Kentucky.

Both salmonella and *R. equi* can contaminate the environment for long periods. Traditionally, *R. equi* has been associated with pneumonia among young foals exposed to dry dusty paddocks in the hot climates of California and Australia. *R. equi* multiplies in the digestive tract of young foals, resulting in fecal contamination of pastures and paddocks. The recognition that the organism has a wider geographic distribution in North America and Europe has led to increased scientific interest.

The risk of botulism has increased as new methods have evolved for the preservation of hay, including the production of large compact bales. Extreme compression of plant material during baling creates an anaerobic environment conducive to the production of clostridial neurotoxins, particularly if the bale contains soil or dead animal remains.

The California botulism outbreak discussed emphasizes the need to produce a vaccine with a broader spectrum of activity if the feeding of processed grass products to horses continues to develop.

## Third Quarter '92

The International Collating Centre, Newmarket, England confirmed the following disease outbreaks:

Equine influenza subtype 2 was reported from Italy, Denmark, France, England, and Sweden. Strangles was reported from eight countries and contagious equine metritis from Japan.

A condition referred to as hepatic encephalopathy caused the death of an undetermined number of horses in the west of France during the summer. The clinical signs included jaundice, incoordination, blindness, salivation and coma terminating in death. The condition was not contagious and a provisional diagnosis of toxicity linked to the ingestion of clover is still under investigation.

The veterinary regulations regarding the movement of horses from the U.S. to the United Kingdom imposed following the outbreak of disease at three racetracks in the Northeast were lifted on October 19.

## African Horse Sickness in Spain

Spain resumed vaccination against this disease in July to prevent its spread from Morocco, where cases were reported in 1991. The last reported case of the disease in Spain occurred in October 1990. The European Community is expected to adopt new measures regarding the control of African horse sickness in Spain.

The region of Andalucia, where the last cases occurred, would be declared an infected zone allowing horses to be moved out of the area only between February 1 and April 30, 1993 after undergoing 40 days' quarantine. Should no further outbreaks of the disease occur, these restrictions would be lifted at the end of 1993. Restrictions on the movement of horses from Portugal and other regions of Spain would end.



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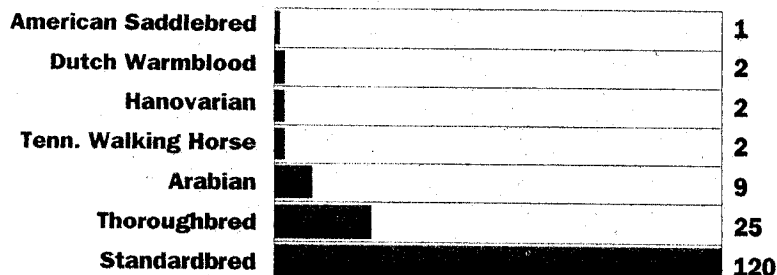
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## Equine Arteritis Virus: Significance for the Stallion

**Figure 1**

Breed distribution of North American stallions confirmed as semen shedders of EAV—1984-'92



Subsequent investigations revealed that although frequency of the carrier state can vary widely among different groups, it can occur in as high as 30 to 60% of stallions following natural infection with equine arteritis virus (EAV). Most carrier stallions carry moderate to high antibody titers while infected with the virus. Although the majority of long-term infected stallions continue to harbor EAV, there has been a limited number of persistently infected stallions spontaneously ceasing to shed virus after periods of several years, with no evidence of reversion to a shedding state at a later date.

Carrier stallions shed EAV constantly in the semen and transmit the infection solely by the venereal route either at time of natural breeding or artificial insemination. Equine arteritis virus is shed in the sperm-rich fraction of the ejaculate and is not present in the pre-ejaculatory fluids. There is no evidence of intermittency of virus shedding by carrier stallions nor of the existence of latent infection. No detectable impairment of fertility associated with the carrier state has been reported in any of the carrier stallions confirmed to date.

Carrier stallions would appear to play a major role in the epidemiology of EAV, providing an effective means of maintaining this virus in horse populations throughout the world. Studies have confirmed the EAV carrier state in stallions of various breeds in other countries beside the United States and Canada;

these include Sweden, Norway, Germany, Italy, South Africa, Australia, and New Zealand.

Results of virological examination of semen samples from stallions submitted to the University of Kentucky Maxwell H. Gluck Equine Research Center over a 9-year period since 1984 have revealed a significant number of stallions which shed EAV in the semen. The majority of these samples were submitted as part of a pre-export testing program.

The breed distribution of stallions from which virus was isolated is illustrated in **Figure 1**. EAV was isolated from 161 stallions of 7 different breeds; the majority of virus isolations (120) were obtained from Standardbred stallions. The high percentage of Standardbred stallions shedding virus in the semen is related to the prevalence of natural infection occurring within this breed.

Occurrence of the carrier state in stallions can be prevented by annual vaccination of seronegative stallions with the modified EVA vaccine (ARVAC, Fort Dodge Laboratories, Ames, IA). Annual vaccination of the Thoroughbred stallion population has been successfully carried out in the states of Kentucky and New York since 1985.

If implemented, a similar strategy would effect a major reduction in frequency of the carrier state in the Standardbred breed. Currently, the high frequency of carrier stallions has reduced the export market for Standardbred stallions and their semen, particularly to countries which require horses and semen to be free of the virus.

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## Botulism: A Threat to Foals

**B**otulism, also known as Shaker Foal Syndrome, is caused by a neurotoxin formed by the bacteria *Clostridium botulinum* type B. This anaerobic bacterium is commonly found in soils of several states including Kentucky, New York, New Jersey, Delaware, Maryland, Pennsylvania, and Virginia.

Botulism caused by *Clostridium botulinum* type C has been reported in California and Florida. A 1989 outbreak in California resulted in the deaths of 31 horses following the ingestion of processed alfalfa

### EQUINE DISEASE QUARTERLY

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continued



hay cubes in which the remains of fur and flesh of small animals were found.

Exposure occurs when horses ingest the preformed neurotoxin from feed contaminated with decomposing animal or plant material. The bacteria can also invade a pre-existing lesion in the body, such as a gastric ulcer, intestinal lesion, navel abscess, or skin wound, resulting in production of the neurotoxin.

Clinical signs include muscular weakness and tremors causing recumbency, depression, difficulty in swallowing, and eventually death. Following rapid onset of symptoms, mortality is 80-100%. Typically, foals affected with botulism are 3 weeks old. Adult horses can develop botulism, although this is rare.

Botulism neurotoxin does not cause characteristic pathologic lesions. A definitive diagnosis at post mortem requires the isolation of the bacteria from lesions or identifying the neurotoxin in tissues and serum, which is very difficult. Because of this, post mortem reports often give a presumptive diagnosis of botulism. The exact number of cases seen at the Kentucky Livestock Disease Diagnostic Center is therefore hard to determine, but it is estimated that 10-12 cases occur each year.

Treatment of affected foals includes administering botulism type B antitoxin (costing approximately \$1,000), antibiotics, and intensive care. Antitoxin must be administered early in the disease before the toxin becomes bound at the junction of nerve and muscle. The average cost of treating affected foals can range from \$2,000-\$2,500.

Prevention is simple and inexpensive using the vaccine Bot Tox-B (Neogen Corporation, Lansing, MI) which is effective only against type B and not type C. Mares which are vaccinated prepartum will pass on protection to the foal via colostrum. Foals two months of age and adults are immunized with an initial series of three doses of vaccine followed by a single annual booster. All pregnant mares in the above listed states and those which will be shipped to those states for foaling and breeding should be vaccinated.

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## Contracted Foal Syndrome: A Common Congenital Defect

**C**ontracted foal syndrome (CFS) was the most common congenital defect seen in fetuses and newborn foals submitted to the Livestock Disease Diagnostic Center between 1986 and 1991.

CFS is characterized by foals born with rigid contracture defects involving the tendons of the limbs, spine, and face. It accounted for more than half (188/348) the birth defects seen during the 6-year period. The number of contracted foals seen each year was fairly consistent and the incidence varied between 28 and 34. Overall, congenital malformations comprised nearly 10% (348/3569) of the fetuses and newborn foals submitted for post mortem during the 6-year period.

CFS may be a cause of dystocia in the mare and in many instances, deformed foals cannot be delivered by natural means due to rigid deformity of the limbs and must be removed by Caesarean section or fetotomy. The deformity was seen primarily in Thoroughbreds, although the condition was also observed in other breeds (Figure 2).

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**Figure 2**  
Incidence of CFS among post mortem cases—1988-'91

Morgan	1
American Saddlebred	3
Standardbred	4
Thoroughbred	116

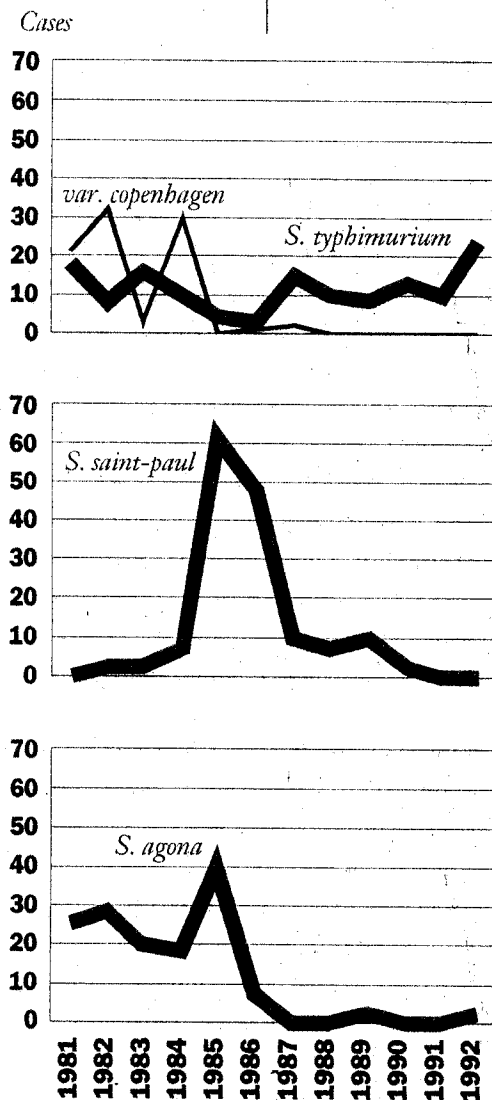
## Salmonellosis: A Cyclical Disease

**S**almonella is a bacterium which causes disease in all animals and humans. More than 1,500 serotypes are recognized and certain types are considered to be more pathogenic for horses.

Animals are primarily exposed to the bacteria by ingestion or *in utero*. Not all exposed horses develop disease, such as diarrhea, septicemia, and arthritis, but they can become carriers and shed the bacteria in feces. Fecal shedding, while not affecting the health of the horse, poses a potential source of infection to other horses, especially foals.

**Figure 3**

Major stereotypes of salmonellae isolated from horses in central Kentucky—1981-'92



Weak and premature foals and those suffering from concurrent disease and injury are particularly susceptible to salmonella infection. Recent transportation and antibiotic therapy also increase the risk of acquiring infection.

All salmonellae isolated by the Livestock Disease Diagnostic Center over the past 12 years were forwarded to the National Veterinary Services Laboratories in Ames, Iowa, for serotyping. Using this data, epidemiologic information has been obtained to evaluate salmonellosis among the equine population of central Kentucky.

Figure 3 outlines the predominant salmonellae serotypes isolated between 1981 and 1992. *Salmonella typhimurium* was the most consistent serotype isolated throughout this period. A subtype of *S. typhimurium*, variety *copenhagen*, was commonly cultured in the early 1980s but is now only rarely isolated; a similar pattern was seen with *S. agona*.

Two-thirds of *S. saint-paul* isolates during 1985-1986 were obtained shortly after horses, particularly foals, were admitted to a medical or surgical facility, with very few multiple cases occurring on farms. The data illustrate the cyclical nature of salmonellosis, and how serotyping can pinpoint a source of the problem and lead to solutions.

Besides the predominant serotypes, more than 30 other salmonellae types have been cultured from horses over the 12-year span. Occasionally one serotype causes significant disease problems for only one foaling season. During 1989, 22 cases of foal enteritis caused by *S. infantis* were confirmed on farms in central Kentucky.

In California during 1989, a single farm outbreak of salmonellosis in newborn foals caused fatalities and multiple cases of diarrhea and septic arthritis. The mares were discovered to be carriers of *S. ohio* and shedding the organism at the time of parturition. This serotype is rarely associated with disease in horses.

The control and prevention of salmonellosis in all animal species is complicated by many problems. Environmental hygiene is a high priority since salmonellae can readily survive in barns, stalls, and hospital facilities.

Salmonella can remain viable in moist environments and even freezing temperatures for long periods of time, but is susceptible to drying, heat >140°F, and disinfectants such as phenols. No equine vaccine is commercially available to protect against the many serotypes of salmonellae; however, bacterins can be made against specific serotypes when an outbreak affects a farm.

Because asymptomatic shedding horses exist, a period of quarantine for incoming horses on the farm is essential in addition to establishing a thorough disinfection program.

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## Rhodococcus equi: A Summer Problem

**R***hodococcus equi* is known for its ability to cause a highly fatal abscessating pneumonia in foals. Other disease entities caused by *R. equi* include enterocolitis and diarrhea, skin and lymph node abscesses, osteomyelitis, meningitis, abdominal abscessation and peritonitis.

Between 1986 and 1991 *R. equi* was isolated from 119 post mortem cases submitted to the Livestock Disease Diagnostic Center. The seasonal distribution of these cases is shown in Figure 4, confirming the high prevalence during May, June, and July. The vast majority of cases, 92, occurred among Thoroughbreds, although cases were also recorded among American Saddlebreds, Standardbreds, Quarter Horses, Arabians and ponies. Cases were equally distributed among males and females.

The age distribution of horses from which *R. equi* was isolated at post mortem is shown in Figure 5; it indicates that of 110 cases, exactly half occurred among foals less than 8 weeks of age.

In recent years the number of cases identified at post mortem has fallen from just over 20 per year during the 1980s to 15 in 1990 and 14 in 1991. This may well reflect the progress in being able to make an early diagnosis and successfully treat clinical cases.

Historically, penicillin and gentamicin was the antibiotic combination of choice but foal survival despite treatment was rare. Recently the combination of erythromycin and the anti-tuberculosis drug, rifampin, has significantly increased survival rate. Early detection using a

blood test has enhanced survival, as previously a clinical diagnosis was often delayed until *R. equi* was well established and caused extensive damage.

Other diagnostic procedures to locate lesions and evaluate the response to treatment include thoracic and abdominal ultrasound examination and chest radiography.

Why is there increased concern if improved detection and treatment of *R. equi* infection is available? Rare but severe and sometimes fatal complications following medication have been experienced. Prevention is undoubtedly preferable to treatment.

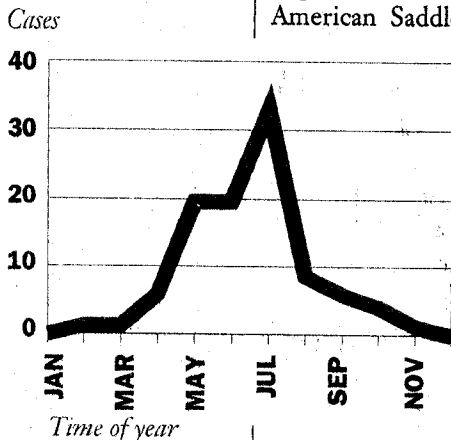
*R. equi* does not pose a threat to foals with a mature immune system, which develops by 5 to 6 months of age. The prophylactic use of transfused plasma obtained from adult horses immunized with *R. equi* organisms has been shown to protect young foals on farms with a known history of the disease.

Plasma may do more than just provide antibodies, since other components in plasma may enhance the immune system. Recent studies from Texas A&M and the University of California, Davis, demonstrated that plasma administration was very effective. Intravenous plasma administration was not effective as a method of treatment once infection was established.

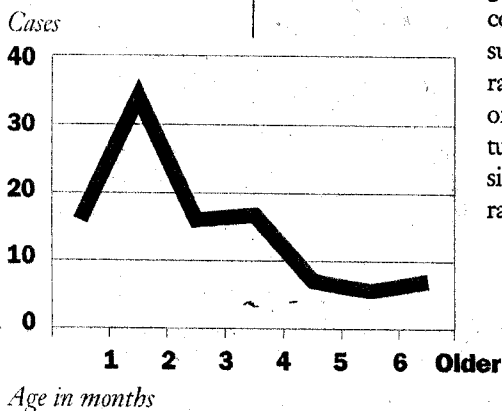
Hyperimmune plasma joins the improved diagnostic techniques and newer antibiotic regimes available to prevent and treat *R. equi* infections. It is unlikely that this worldwide infection will be eliminated, but what was once a fatal condition has responded to clinical investigation, tilting the chances of survival in favor of the foal.

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**Figure 4**  
Seasonal distribution  
of post mortem cases of  
*R. equi*—1986-'91



**Figure 5**  
Age distribution of  
post mortem cases of  
*R. equi*—1986-'91



## Kentucky's Equine Embargo: The Consequences

**O**n July 16, 1992 the Kentucky Commissioner of Agriculture issued an embargo prohibiting the movement to Kentucky of any equines from the six New England states in response to an outbreak of disease at three racetracks in New Hampshire and Massachusetts.

Road checks were set up on the northern and eastern borders of Kentucky. Inspection of horses at equine events within the state was also undertaken to ensure that owners of competing horses were complying with the embargo.

During the period of the embargo, which lasted until September 2, 903 vehicles were stopped and inspected. Two trailers containing horses from the northeastern states en route to Georgia and Florida were denied permission to travel through Kentucky. Three horses were ordered to be removed having entered Kentucky from the embargoed states.

Show horse owners were contacted and requested not to bring their horses to Kentucky. These included 70 horses registered to participate in the Pony Club National Finals at the Kentucky Horse Park in Lexington, and 30 horses entered for the World's Championship Horse Show in Louisville.

Ninety-three warnings and citations were issued during the embargo involving horses traveling within or entering the state that did not have the appropriate certification for the equine infectious anemia Coggins test.

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