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IN THIS ISSUE

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COMMENTARY

Although *Salmonella* was first identified in the late 1800s, it has been a major cause of disease and mortality for humans and animals for hundreds of years. More than 2,200 serotypes of this bacteria exist, with humans being susceptible to virtually every one. In horses of all ages a wide variety of serotypes has been documented as causing diarrhea, septicemia, joint infections and abscesses.

Many people consider salmonella infections as "out of sight, out of mind" until potential disaster strikes. Two large animal hospitals in the United States were closed in 1996 for several weeks due to nosocomial salmonella infections among their equine patients. "Nosocomial" means that the animal (or person) is infected with the disease while in the hospital. The August 1, 1996 issue of the *Journal of the American Veterinary Medical Association* featured papers detailing equine cases of *S. krefeld* in an intensive care facility and *S. anatum* in another veterinary teaching hospital causing temporary closure. Private practices and breeding establishments are not immune from economically devastating outbreaks.

The horse-owning public might view any closure of a veterinary hospital due to a nosocomial infection as reflecting negligent management and care for its patients. Administrators do not close hospitals for disease control measures unless they have a vested interest in the future health of their patients and confidence of clients. Consider the costs of such a closure for disinfection and disease control: labor, disinfection supplies, extra sterilization of equipment, discard of potentially contaminated materials, time for client communications, and loss of revenue. A one-month closure can easily cost $500,000 or more.

Although major advances have been made in the rapid diagnosis of salmonellosis with polymerase chain reaction testing, no vaccine is available. Prevention and control of salmonellosis depend on decreasing stress, avoiding overcrowded conditions, routine and thorough disinfection of equine facilities and equipment, and responsible use of antibiotics. Instead of "out of sight, out of mind" the mindset should be "an ounce of prevention is worth a pound of cure."

INTERNATIONAL

Second Quarter 1996

The International Collating Centre, Newmarket, confirmed the following disease outbreaks.

Equine-2 influenza was reported from Denmark, France, Sweden and the United Kingdom. Cases of EHV-1 abortion were confirmed from Denmark, France, Germany, Ireland, Switzerland and Sweden. Multiple cases on the same farm were reported from Italy, Japan and the United States. Strangles as usual was widespread, reported from Denmark, Ireland, Italy, New Zealand, Sweden, United Kingdom and the United States. Singapore reported a case of a horse in quarantine recently arrived from Australia.

Salmonellosis was reported among horses in Ireland, Japan, New Zealand and Switzerland involving serotypes *typhimurium, dublin, infantis, hindmarsh* and *abortus equi.*

African Horse Sickness was reported from South Africa involving serotypes 1, 2, 4 and 7.

The United Arab Emirates reported a case of surra caused by *Trypanosoma evansi* in an Arab horse. This was the first recorded case in the country; the affected animal also tested positive for equine piroplasmosis.

Endophyte Toxicity: A Cause of Foal Mortality

A Thoroughbred stud farm in Argentina experienced losses among newborn foals in 1995.

Fatalities commenced at the end of July and continued until November with 26 foal deaths from 71 mares that foaled. Two thirds of the foals were born with signs of dysmaturity including weakness, lack of the suck reflex, lying down for extended periods and inability to stand without assistance. Within 24 hours many foals developed jaundiced mucous membranes and retention of meconium. Blood profiles revealed low IgG levels, neutropenia, uremia and elevated bilirubin levels.

Despite intensive supportive therapy including intravenous fluids, serum and plasma transfusions, hormones and antibiotics, many foals failed to respond; they became comatose, exhibiting occasional seizures, and died within 24 hours and eight days of birth. Postmortem examinations did not reveal a consistent pattern with some fatalities attributable to pneumonia, polyarthritis and bone fractures.

Histories of the mares indicated little or no udder development at foaling. Most mares exhibited extreme difficulty at foaling with profound dystokia. Premature separation of the chorion was common and many mares had retained placentas. Examination of the placentas indicated they were heavy and thickened, reminiscent of the "red bag syndrome" although not excessively edematous. Abortion and prolonged gestation were not a feature.

Initially an infectious etiology was suspected but attempts to isolate equine herpesvirus-1 (EHV-1) and a variety of bacteria produced negative results. By October the possibility that mares had been exposed to endophyte toxins as a result of grazing fungal contaminated pasture was suspected. At that time nine mares were left to foal. They were administered daily oral doses of domperidone 1 mg/kg of body weight until they foaled. Within days of initiating treatment udder development was observed and seven mares produced healthy foals under normal conditions.

Domperidone is a dopamine antagonist that facilitates release of the pituitary hormone prolactin. Dopamine and like-acting substances including the ergot alkaloids are considered the potent toxins ingested by horses when they graze tall fescue contaminated with the fungal endophyte *Acremonium*.

The exact identity and source of the endophyte has proved difficult to establish. The majority of paddocks, many of which had been reseeded within the last five years, were composed predominantly of annual or perennial ryegrass along with other grasses. Tall fescue was only present in one paddock grazed by mares in May. Laboratory examination of pasture samples and seeds indicated the presence of an endophyte in ryegrass and fescue.

Traditionally, endophyte positive ryegrass has been associated with a syndrome referred to as "ryegrass staggers" and contaminated tall fescue with reproductive problems among horses. During this outbreak none of the neurological signs associated with ryegrass staggers were observed. However toxicological analyses of ryegrass pasture revealed the presence of "tremogenic" neurotoxins considered responsible for ryegrass staggers. Further investigation is obviously necessary to elucidate these somewhat confusing observations.

Other factors which considerably exacerbated the problem in 1995 as compared to previous years were the severe drought conditions experienced on the farm from May through September and the high stocking density of mares.

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Import/Export of Horses For the Summer Olympics

The importation, monitoring and subsequent exportation of approximately 230 horses was a major undertaking accomplished by USDA-APHIS and Georgia Department of Agriculture officials in June through August of this year. Most of the equine competitors (210) coming to the summer games arrived in Atlanta through direct flights from Europe.

Since Atlanta is not a permanently approved import center, bringing horses directly into Atlanta required special approval from USDA and Georgia Department of Agriculture. This temporary approval is routinely given to various ports for special events.

Normal USDA import requirements for horses depend on the origin of the animals. For example, horses coming from Europe require a short quarantine period, on average three days, so that serological

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tests can be completed for Equine Infectious Anemia, Equine Piroplasmosis, Dourine and Glanders. USDA allowed only horses qualified for the three-day quarantine to come directly into Atlanta.

Most of the horses coming to the Games arrived on charter flights flown from one of three cities in Europe with one flight originating in Monterrey, Mexico. Arrival procedures were developed to safely and efficiently get the animals off the plane and into their individual stalls in the Atlanta Equine Complex (AEC) at Hartsfield International Airport. Even on larger shipments (45-50 horses), we were able to get all animals into their stalls within two hours after flight arrival. At no time did any animal spend more than a few minutes without air conditioning.

Once unloaded from the jet-stalls, the horses were immediately brought into the AEC where they were identified, their hooves picked and disinfected, and their blankets and leg wraps removed. Then they were taken directly to their stalls and a Veterinary Medical Officer collected blood samples, conducted a physical examination, and sprayed each animal with an acaricide (tick spray). Blood samples were processed and immediately hand-carried to the National Veterinary Services Laboratories in Ames, Iowa. The serological tests were immediately set up, results were telephoned within 36 hours to release the horses.

To expedite exportation, arrangements were made to process the horses at the Georgia International Horse Park (GIHP). First, each animal was given an examination by an accredited veterinarian followed by an inspection by a Veterinary Medical Officer. The international certificates were completed, signed and endorsed by USDA. Horses were loaded on horse vans at the GIHP and taken directly to the airport where they were transferred to jet-stalls. The horses departed on five charter flights.

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VIRUS ALERT

Insect surveillance in Rhode Island during September has indicated the presence of Eastern Equine Encephalitis (EEE). This emphasizes the need to routinely vaccinate horses against this disease, particularly in the northeastern United States.

Re-Emergence in Mexico of Venezuelan Equine Encephalomyelitis (VEE)

On July 22, 1996, the government of Mexico notified the US Department of Agriculture of an outbreak of VEE in equines in the state of Oaxaca in southern Mexico (**Figure 1**). VEE is a zoonotic mosquito-borne viral disease affecting all equine species and humans.

A total of 32 cases of the disease were diagnosed among some 2,500 horses in four municipalities within the state of Oaxaca. Cases were reported in rural areas often flooded during rainy periods. The first case was diagnosed on June 16, and the last case was recorded on July 23, 1996. Official reports confirm a total of 12 deaths in horses but no illness in humans.

VEE virus was isolated from two horses. The identity of both isolates was independently confirmed by the USDA's National Veterinary Services Laboratories, Ames, Iowa, and Dr. Rebecca Rico-Hesse, Department of Epidemiology and Public Health, Yale University, School of Medicine, as subtype 1, variant E. This is an enzootic variant of the virus whose known distribution includes the coastal areas of southeastern Mexico and central America (Weaver, Bellew and Rico-Hesse, *Virology*, Vol. 191, pp. 282-290, 1992). The virus is maintained in nature via a transmission cycle involving small animals and mosquitoes. It has a low pathogenicity for equids and may cause illness in humans.

The Mexican government implemented emergency measures in Oaxaca and neighboring states to control the spread of VEE outside of the areas initially affected with the disease. Movement of horses to other parts of the state of Oaxaca and to other states was prohibited. As of August 9, a total of 16,700 horses in affected and adjacent municipalities in this state of Oaxaca had been vaccinated against VEE using the live attenuated TC/83 Vaccine. A spraying program for the mosquito vector was carried out in urban areas in the affected region. Increased epidemiologic surveillance and public awareness campaigns were instituted throughout Mexico.

On July 24, 1996, USDA APHIS tightened the import requirements on equines from Mexico by increasing the quarantine period from 3 to 7 days. This measure involved importations both at border crossings and the animal import centers in Miami and New York.

This year's outbreak marks the second occurrence

Figure 1

of VEE in Mexico in the recent past. In 1993, there was a similar outbreak of the disease affecting equines in the state of Chiapas, a neighboring state to Oaxaca in southwestern Mexico. The 1993 outbreak was also caused by VEE virus, subtype 1-E variant. These two outbreaks of VEE in southern Mexico underscore the potential for future outbreaks of the disease to occur in this and other regions in which the 1-E variant is known to occur. It is worth bearing in mind that in the event of future global climatic changes, *e.g.*, global warming, the natural nidality of enzootic variants of VEE virus and their known geographic distribution could change significantly.

The 1993 and 1996 outbreaks in Mexico and the widespread epidemic involving humans and equines in Venezuela and Colombia in 1995 confirm the re-emergence of VEE as a disease of major importance in an era of ever increasing international movement of horses.

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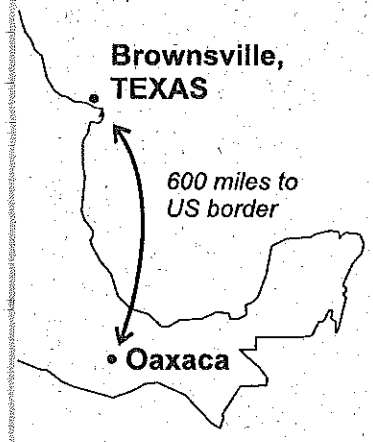
N A T I O N A L

Potomac Horse Fever: An Update

Equine monocytic ehrlichiosis (Potomac horse fever) was first recognized as a cause of diarrhea in 1979. The disease is an acute systemic disease of horses caused by *Ehrlichia risticii*. *E. risticii* is an obligatory intracellular bacterium in the family *Rickettsiaceae*. The disease has been serologically identified in horses from the United States, Canada and Europe. Culture of the organism has been successful from infected horses in Ohio, Maryland and Kentucky.

The routes of transmission of natural infections are unknown. The idea of the disease being vector borne is still important. Recently work by Dr. Y. Rikihisa at Ohio State has demonstrated that the organism is genetically very similar to *Neorickettsia helminthica*, which is the causative organism of salmon poisoning in dogs. It is possible that we have been searching in the wrong place for the source of transmission of natural infection in horses.



The diagnosis of Potomac horse fever is by serologic testing utilizing the indirect fluorescent antibody (IFA) test. The test does not determine if the horse is currently infected or if antibodies remain from previous exposure to ehrlichial antigens. The IFA test cannot distinguish between infection and vaccination titers. Polymerase chain reaction (PCR) testing has been developed to detect the partial 16S rRNA sequence of *E. risticii* using blood and fecal samples.

The disease is characterized by fever, depression, loss of appetite, diarrhea, dehydration and often laminitis. Infected horses usually exhibit leukopenia and hemoconcentration at the time diarrhea is noted. Because these findings are similar to those found in horses with acute endotoxemia, diarrhea caused by *Salmonella* spp. is the primary differential diagnosis.

In 1994 blood specimens were obtained in Dr. Rikihisa's laboratory from 26 clinical cases of horses which were naturally infected in Ohio, Kentucky, Pennsylvania and Indiana. At least seven of these horses had been vaccinated. The use of culture isolation resulted in positive identification of the organism in 21 of 22 seropositive horses. Culture by isolation was more sensitive than IFA or PCR for the diagnosis of Potomac horse fever.

Killed vaccines are available from several commercial sources. Although efficacy of vaccines in the field is marginal, vaccination is very important. The marginal nature of the vaccines is based on the fact the organism can sometimes be isolated from sick horses which have been vaccinated. Even if complete protection is not achieved, vaccination may lessen the impact of the disease, diminish the chance of transmission by decreasing the number of circulating organisms, and/or reduce the time the organisms are in the bloodstream.

Some non-vaccinated horses may show an antibody titer in serum without evidence of clinical signs indicating that sub-clinical infection may occur. In a comparative study performed by examining split serum samples in several laboratories the presence of false positives was noted.

During the spring and summer of 1996 it appears that fewer horses were affected with Potomac horse fever despite an above average number of horses presented at our clinic for diarrhea. Reasons for this include more horses being vaccinated, early recognition and treatment without diagnostic confirmation by veterinarians in the field, and perhaps the organism being less prevalent than in years past.

Antigenic variations have been observed in organ-

isms isolated from clinical cases of Potomac horse fever in Ohio, Kentucky and elsewhere. It is possible that the virulence of the organism has lessened. Whatever the cause, Potomac horse fever remains an important differential diagnosis for horses presented to our clinic with acute fulminant diarrhea and laminitis.

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Equine Salmonellosis: Central Kentucky and US

Salmonella causes significant disease in horses with a variety of different serotypes. When a culture from a suspect animal has a positive result for *Salmonella*, some laboratories can further differentiate between different broad serogroups, designated by the letters A-I. Laboratories such as the National Veterinary Services Laboratories (NVSL) in Ames, Iowa can specifically identify the serotype (also known as serovars), such as *Salmonella typhimurium*, which is a member of serogroup B.

At the Livestock Disease Diagnostic Center, all *salmonellae* cultures isolated from equine postmortem cases, as well as positive samples submitted from clinical cases, have been forwarded to the NVSL for serotyping. From January 1990 to June 1996, 301 salmonella isolates representing 32 different serotypes were cultured from equine samples. *Salmonella* were recovered from 207 necropsy cases including 124 (60%) from animals less than two years of age, and 83 (40%) from adult horses. The annual distribution of these isolates is shown in **Figure 2.** A positive *salmonella* culture does not mean that the horse died from the effects of salmonellosis since horses can die for other reasons but still shed the bacteria.

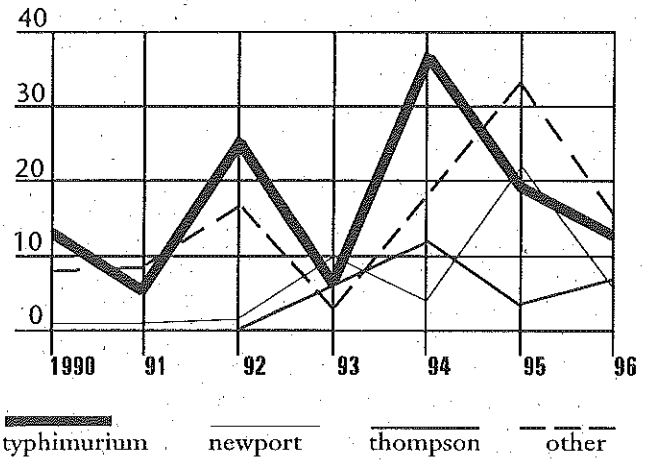

Figure 2. *Salmonella serotypes isolated from horses in Kentucky*

Of the 94 clinical cases, the majority of isolates were identified as *typhimurium* (34), *newport* (21), or *thompson* (14). For 14 other serotypes there were 5 or fewer isolations. The sources of these isolations were feces (89), joints (3) and abscesses (2).

According to the latest NVSL annual report (10/94 to 9/95) which records serotypes by animal species from which they were isolated, the most common equine serotypes were *typhimurium* (149); *typhimurium* var. *copenhagen* (24); *newport* (37); *anatum* (28); *oranienburg* (21); *dublin* (11) and *krefeld* (10). Serotypes of 45 other *Salmonella* were found in horses. These data must be carefully interpreted as examples of *Salmonella* serotypes which can be found in horses, and not as prevalence of equine serotypes, since not all isolates from horses are serotyped by NVSL.

In looking at these serotypes in relation to their isolation from other species, it is readily apparent that cross contamination could easily occur. *S. typhimurium* and *S. typhimurium* (var. *copenhagen)* are commonly isolated from bovine, swine and avian species (including chickens and turkeys) with over 2,300 positive cultures; *S. anatum* and *S. oranienburg* from bovine and avian species; *S. dublin* from bovine; and *S. newport* from bovine, swine and avian.

Salmonellae of several different serotypes (including *ohio, infantis, krefeld, anatum, saint-paul* and *typhimurium)* have caused significant outbreaks of disease and mortality on horse farms and in veterinary hospitals. Tracking serotypes is important from an epidemiologic standpoint, because it can lead to insights into the origin of infection. This information will aid in the prevention and control of the disease.

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K E N T U C K Y

Alimentary Disease of Older Horses

Diagnosis of alimentary tract disease was made in 389 horses necropsied at the Livestock Disease Diagnostic Center between January 1, 1993 and December 31, 1995. Horses included in this tabulation were one year of age and older and accounted for 8% of the total equine necropsy cases. The total number of equine necropsies performed during this period was

4,757; Thoroughbreds accounted for 73% of the equine necropsies and 62% of the cases with alimentary tract disease.

Cases with alimentary tract lesions included Thoroughbred (240), Quarter Horse (29), Tennessee Walking horse (24), Standardbred (16), American Saddlebred (15), Arabian (10), Appaloosa (5), Belgian (4), Morgan (3), Rocky Mountain Saddlehorse (2), and one each of the following breeds: Hackney, Paint, Pinto, Percheron, Pasofina, Racking, Swedish warmblood. Also included in this group were ponies (8), miniature horses (5) and mix breed/grade or unknown breed (21).

Upper gastrointestinal system diseases were few. Conditions involving the oral cavity included two cases of gingivitis and two cases of ulcerative esophagitis. The most common conditions were gastric dilation and ingesta engorgement 143 (37%) resulting in rupture of the stomach (49), and rupture of a site in the small or large intestine (87), leading to massive peritonitis. Gastric dilation and engorgement without rupture was observed in seven cases.

A grain overload or enterotoxemia-like condition associated with heavy consumption of a carbohydrate rich diet was observed in 19 cases (5%). This subgroup represents acute deaths without convincing postmortem gastric dilatation. There were 34 cases (9%) of colonic impaction without rupture. Obstruction and/or strangulation of a segment of the small intestine numbered 29 cases (7%) including pedunculated lipoma (18), small intestine intussusception (3), entrapment of the small intestine within the epiploic foramen (5) and small intestine diverticuli (2). A single case of blockage of the small intestine by a hairball was identified.

Inflammation was observed in 30 cases (8%) involving a specific etiologic diagnosis including Potomac horse fever (1), salmonellosis (14), gastric trichostrongylosis (1) and severe small and large strongyle colonic parasitism (14). Lower gastrointestinal inflammation where no specific diagnosis was identified involved 128 cases (33%) including gastritis (13), enteritis (16) and colitis (99). Nine cases of rectal tears that resulted in massive fecal peritonitis were identified.

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