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IN THIS ISSUE

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LLOYD'S

Commentary

Many different *Salmonella* serotypes have the ability to not only cause disease, but also to establish residence in the gut of an equine host, where they can exist without any clinical signs. These carriers continue to shed the organism to the detriment of others in the herd.

Historically *Salmonella* was considered to be present in many horses, although it was difficult to detect. Authors had, until recently, considered the prevalence of *Salmonella* organisms in normal horses to be between 10% and 30% of the population. In 1998, a National Animal Health Monitoring System survey of 8,417 horses indicated that only 0.8% of all horses actively shed *Salmonella* in their feces. In contrast, samples taken from horses arriving at necropsy showed a 34% prevalence of *Salmonella*.

University equine hospitals have recorded a *Salmonella* infection rate of 1.4% on admission, rising alarmingly after hospitalization to between 38% and 68%. Stress plays a role in such infections, and animals with large bowel obstruction seem to be particularly at risk. This post-hospitalization increase in infection rate could be explained by a recrudescence of the *Salmonella* already present in the gut of the horse, but this hypothesis is not in accord with the findings.

The consistency with which hospitals report the *Salmonella* found to be of one serotype is startling. In one report *S. infantis* was implicated, in others, *S. anatum*, *S. agona*, and *S. heidelberg* as well as the more common *S. krefeld* and *S. typhimurium*. The way in which these nosocomial infections are usually restricted to one variety of *Salmonella* would virtually rule out the possibility of recrudescence, underscored by the fact that horses arriving at the clinic at a later date become infected with the resident serotype.

Once a *Salmonella* infection has established itself within a hospital, great efforts are usually taken to remove all flooring, and a massive disinfection program is instituted. Total eradication is unlikely to be successful, and the organism often becomes established in the clinic. Samples taken within the hospital have consistently failed to identify the source, which is often a cause of frustration. High pressure water sprayers may be responsible for the aerosolized dissemination of the bacteria. The organism may remain in the drainage system, under rubber matting, and especially in the dust on window ledges and rafters.

All too often, hygiene protocols within the hospital are either ignored or nonexistent. Clinicians and technicians should wash their hands between cases. Rubber boots should be worn and thoroughly cleansed on exiting each stall. Protective clothing should be worn and disinfected after treating animals in isolation. Disposable gloves and gowns need to be discarded carefully. Stalls should be thoroughly cleaned and disinfected after each occupant.

Equine clinicians must learn to control and contain diseases such as salmonellosis. Standards of care and hygiene need to be established prior to an outbreak, and the need for constant vigilance is of paramount importance. This disease can be devastating to the horses under treatment, to the staff who care for them, and to the reputation of the hospital. ■

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Equine Disease Quarterly

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International

Second Quarter 2002

The International Collating Center, Newmarket, reported the following disease outbreaks:

- A single Thoroughbred was reported with Contagious Equine Metritis (CEM) from Hokkaido, Japan.
- Abortions attributable to equine herpes virus (EHV-1) were reported from France, Germany, Ireland, Japan, Sweden, Switzerland, and the United Kingdom. Twenty-five cases were confirmed among Thoroughbred mares in Central Kentucky. Three farms had three cases; one farm had two cases, and the remaining 14 farms had a single case. Cases attributable to EHV-4 were reported from Ireland and the United Kingdom. Coital exanthema (EHV-3) was diagnosed among animals on one premise in Ireland. Respiratory disease attributable to equine herpes virus was reported from France and Ireland. The paralytic form of equine herpes virus was reported from a single premise in France, Ireland, and Virginia, U.S.A.
- Of 75,000 blood samples tested for Equine Infectious Anemia (EIA) in Canada during 2001, 161 tested positive. Ten cases of equine rabies were confirmed in Canada during the same period—five in Ontario, three in Saskatchewan, and one in Manitoba.
- Equine influenza was reported from France, the United Kingdom, and Colorado, U.S.A. during the second quarter of 2002, and rotavirus was diagnosed on 15 premises in Ireland.
- Strangles was reported from Queensland, Australia, a Thoroughbred racetrack in Fort Erie, Ontario, Canada, and several premises in Ireland, Sweden, and Switzerland. ■

National

Equine Salmonellosis in the United States and Kentucky

Salmonella spp. cause a multitude of diseases in horses, including diarrhea, abscesses, septicemia, and other ailments. Over 2,200 serotypes of salmonellae are known and can be identified at the National Veterinary Services Laboratories (NVSL) in Ames, Iowa. With the exception of *S. typhi*, which only affects humans, all other salmonellae are zoonotic, posing possible transmission from animals to people.

In a NVSL report for isolates serotyped from July 1, 2000 - June 30, 2001, the most frequently isolated serotypes from the clinical cases, herd and flock monitoring, and meat inspection of all species were *S. typhimurium, heidelberg, newport, agona,* and *kentucky*; all have also been isolated from horses. From equine clinical cases reporting a primary or secondary *Salmonella* infection during this same time period, the most frequent serotypes recorded were S. *agona* (213), *typhimurium* (207), *typhimurium var copenhagen* (52), *newport* (211), and *newington* (54). Another 287 samples accounted for 44 serotypes.

The vast majority of horses that have died in Central Kentucky are submitted for necropsy at the University of Kentucky Livestock Disease Diagnostic Center. Any salmonellae isolates cultured are forwarded to NVSL for serotyping.

This surveillance has been ongoing since 1985, with the last report in the October 1996 *Equine Disease Quarterly*. From January 1990-June 1996, *S. typhimurium* was the predominant postmortem isolate, *newport* and *thompson* were the next most common serotypes, and 29 other serotypes were cultured.

Figure 1 illustrates the trend in serotypes isolated from 1996-2000. There was a leveling off of *S. typhimurium* from previous years (a high of 34 post mortem cases in 1994) and a steady increase in *S. typhimurium var copenhagen*. The most prominent feature is the significant increase in the isolation of *S. agona*, which emerged in 1999 and is ongoing to date. These isolates are from both primary and secondary infections. Previous to 1999, *S. agona*

had not been a common serotype isolated from post mortem or clinical cases since 1984.

During the period 1997-2000, 21 other serotypes were identified, and, as in previous years, the occasional new serotype appears that has not been seen in Central Kentucky. For example, in 2000, *S. othmarschen* was isolated from a newborn foal with a primary infection.

The emergence of a new serotype of significant equine importance emphasizes the need for veterinarians and horse owners to be aware of the problem and take preventive measures. Animals coming onto a farm should be isolated for a minimum of two weeks to prevent the introduction of infections. Routine, rigorous disinfection of stalls with chemicals known to be effective against salmonellae in the presence of organic matter is essential, both in hospitals and on farms. Since no commercially available vaccine exists against samonellae, disinfection and biosecurity are the primary preventive measures that *must* be undertaken. Horse owners need to be aware of the zoonotic potential of any *Salmonella*-positive horse and take proper precautions (isolation techniques, use of protective clothing, washing hands, etc.).

These measures are critical because of the zoonotic potential for any salmonellae, which can be deadly in immunocompromised individuals and dangerous to pregnant women. Of considerable concern to the human and veterinary medical professions is the emergence of multidrug-resistant strains of bacteria. In a report from the Centers for Disease Control and Prevention (MMWR, June 28, 2002), multidrug-resistant *S. newport* isolates were reported from New York, Michigan, Pennsylvania, Ohio, and Connecticut. The implicated cause of the outbreak was exposure to raw or undercooked ground beef, but the report also emphasizes the need for *Salmonella* surveillance, the judicious use of antibiotics by the veterinary and medical professions, and the continuous education of veterinarians and horse owners concerning biosecurity issues. ■

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Tying-Up in Horses

Tying-up, the most common muscle problem in horses, has also been called *azoturia*, *paralytic myoglobinuria*, and *chronic exertional rhabdomyolysis (ER)*. Clinical signs include sweating, stiffness, and reluctance to move forward. Tying-up is not a single disease, but a collection of clinical signs that may have different causes in each horse. Specific causes for tying-up have recently been identified, and more are on the research horizon.

Sporadic tying-up is seen in horses that have always exercised normally but suddenly exhibit signs of this muscle problem. It can be due to: exercise in excess of training level, exhaustive exercise, respiratory infections, lack of dietary selenium/vitamin E, or lack of dietary electrolytes and minerals. These horses usually recover with rest, adjustment of the diet treatment, and gradual return to exercise, going on to perform successfully.

Other horses have a chronic form of tying-up from a very young age with continual problems even when exercised lightly. This syndrome has been described in many breeds and can have different causes. A thorough investigation into the cause of tying-up in these cases is necessary and requires the cooperation of the horse owner/trainer and veterinarian, often in consultation with a veterinary medical specialist. A work-up for tying-up involves evaluation of urine and serum electrolytes and minerals, measurement of muscle enzymes released into the serum pre-exercise and four hours post-exercise, and evaluation of muscle biopsies.

One cause of chronic ER in Quarter Horse-related breeds, Draft horses, and Warmbloods is a metabolic defect called *polysaccharide storage myopathy (PSSM)*. This defect is the most common cause of ER in Quarter Horse- and Draft-related breeds and appears to be inherited. Horses with PSSM store an excess of glycogen in their muscle. Muscle sections stained with Periodic Acid Schiff's Stain show that a proportion of the glycogen is stored in an abnormal fashion and is not available for energy production. Horses with PSSM have a greater sensitivity to insulin, which likely increases transport of

Figure 1.
Salmonellae isolated from necrop
University of Kentucky Livestock
1996-2000.

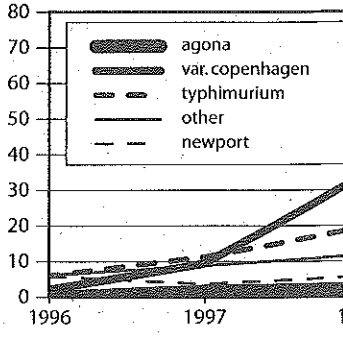
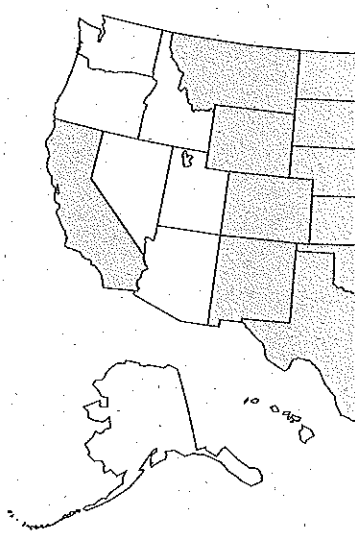
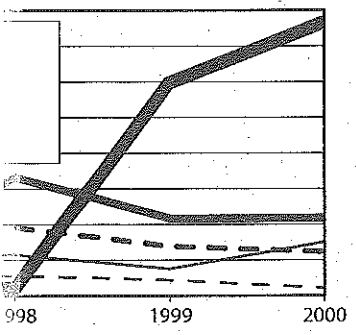


Figure 2.
Cases of West Nile Virus—2002.



material submitted to the
Disease Diagnostic Center,



sugar into skeletal muscle after a carbohydrate meal. The exact link to muscle necrosis is not clear but may be related to disruption of the balance of energy metabolism by excessive muscle glucose 6 phosphate. The lactic acid level in these horses when they are tied up is actually very low. Treatment of horses with polysaccharide storage myopathy involves supplying them with feed that maintains low blood sugar and low blood insulin concentrations (no grain, with a fat source such as rice bran) combined with regular daily exercise. Stall rest or irregular exercise may cause another episode of tying-up. Over 90% of horses will improve dramatically and return to full athletic performance if the recommended changes in diet and exercise are followed.

Other breeds of horses may tie up for completely different reasons. Current research indicates another cause of tying-up exists in Arabian, Standardbred, and Thoroughbred horses that is related to an abnormality in the way the muscle cells regulate intracellular calcium during a muscle contraction. It may involve a genetic predisposition. This form of tying-up, called recurrent exertional rhabdomyolysis (RER), is not related to dietary calcium intake. Five per cent of racing Thoroughbreds, especially young, nervous fillies, are affected. Energy metabolism and glycogen storage appears to be normal in RER horses. Muscle stiffness usually occurs when exercise and excitement combine, such as at a horse show, after a steeplechase, or when horses are being held back to a slower pace than they desire. In Standardbreds, tying-up often occurs after 15 minutes of jogging. The approach to treating these horses is to minimize excitement and stress and substitute fat supplements containing rice bran for part of the grain ration.

Research continues in order to develop the best diagnostic tests and treatments for the variety of forms of tying-up in these breeds. ■

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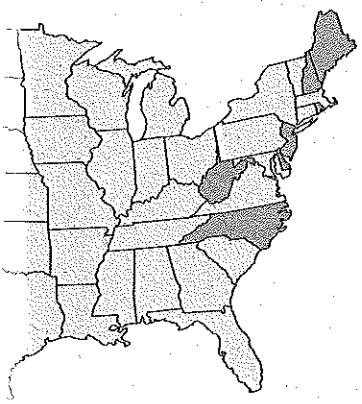


☐ humans or horses
▩ mosquitoes and birds only

As of 9/10/02

West Nile Virus Update

West Nile Virus (WNV) during 2002 has been detected within the United States of America in birds, mosquitoes, humans, and horses in 41 states and the District of Columbia. It has also been identified in four provinces of Canada—Manitoba, Ontario, Quebec, and Saskatchewan. The virus has spread geographically westward in each of the four years since it was initially confirmed in North America. It has expanded in 2002 to 14 states that had previously not reported the presence WNV: California, Colorado, Kansas, Minnesota, Montana, Nebraska, New Mexico, North Dakota, Oklahoma, South Carolina, South Dakota, Texas, West Virginia, and Wyoming (see **Figure 2**.) As of the beginning of September, approximately 3,500 equine cases had been reported in 32 states and over 1,000 human cases in 30 states plus the District of Columbia. ■

Insulin Resistance in the Horse: Significance and Pharmacologic Management

Obesity and insulin resistance: Insulin resistance can be defined simply as an inability of the body to remove blood sugar or glucose from the circulation. In an effort to decrease blood sugar levels, pancreatic insulin output increases, but continued hypersecretion eventually leads to complete failure of insulin secretion, and type-2 diabetes prevails. While insulin resistance is normally associated with obesity, it is has been recognized for many years that temporary insulin resistance may also occur in conditions of inflammation, infection, injury, and several non-pathological conditions, including pregnancy and puberty. As with obese humans, excess bodyweight may predispose the horse to develop insulin resistance and lead to the development of several related diseases, most notably equine Cushing's disease and laminitis. Additionally in the pre-pubertal animal, insulin resistance has been associated and perhaps implicated as a causal factor in the development of osteochondritis dessicans (OCD) lesions.

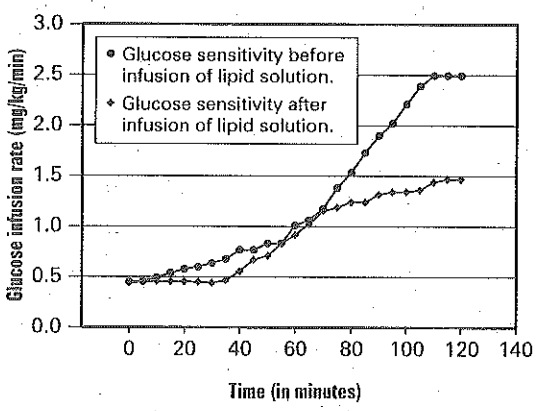
Identification of insulin resistance: Two methods are currently employed by physicians to identify insulin resistance in humans. The first approach is to measure blood glucose and insulin levels following a period of fasting. The second test involves oral administration of a glucose solution and identifies the clearance rate from the circulation (glucose tolerance test). Both tests—elevated levels of glucose and insulin and prolonged insulin responses to glucose administration—are indicative of insulin resistance. While each test offers an inexpensive and practical assessment of insulin sensitivity, a more accurate test may frequently be employed, namely the euglycemic, hyperinsulinemic clamp procedure. The clamp procedure utilizes a basic principle of maintaining a steady-state blood level of insulin and glucose via constant infusion. The total amount of glucose infused over time is an index of insulin action on glucose metabolism. Because insulin actively causes glucose uptake by peripheral tissues, primarily muscle and fat cells, an insulin-sensitive individual requires glucose to be infused at a high rate to maintain constant blood levels. In contrast, the insulin-resistant patient requires much less glucose to maintain basal plasma glucose levels, since peripheral tissues fail to utilize glucose as rapidly as occurs in sensitive individuals.

The mechanism whereby insulin resistance leads to certain disease states in horses remains largely unknown. However, lessons learned from human studies suggest that insulin resistance represents a condition of low-grade inflammation. As a result, insulin resistance may be accompanied by elevated blood levels of several chemical signals (cytokines) related to inflammation. In turn, some cytokines may predispose the individual to changes in local blood flow of tissues and release of inflammatory agents that lead to tissue injury. The exact mode of this relationship is unclear.

Development of a model to induce transient insulin resistance—what can we learn? To further understand the relationship between insulin resistance and some disease conditions, our laboratory has recently developed a simple method to induce a transient insulin resistance condition that may provide an opportunity to investigate changes in the release of cytokines potentially involved in the etiology of some diseases, including laminitis. This method utilizes infusion of a 20% lipid emulsion containing heparin (to facilitate enzymatic production of free fatty acids) for four hours. The change in sensitivity to insulin before and after infusion of a lipid solution is illustrated in **Figure 3.** Prior to infusion of the lipid solution, glucose infusion rates were higher, indicating a more insulin-sensitive state. Immediately following infusion of the solution, glucose infusion rates were lower, indicating a decreased response to insulin.

Potential methods to block insulin resistance—physical and pharmacological: In humans physical exercise can improve insulin sensitivity through different mechanisms, including increased glucose uptake in muscle cells and improved insulin action on target tissues. A forthcoming report (VI International Conference on Equine Exercise Physiology, Lexington, Ky., Sept. 2002) demonstrates that increased insulin sensitivity occurs without an alteration in body weight or condition in horses subjected to short-term, low intensity exercise (1 week at 30 minutes per day). This finding is useful because many obese, insulin-resistant horses are older, and high intensity exercise may be too demanding.

Figure 3.
Change in sensitivity to insulin before and after infusion of a lipid solution.



Currently, many therapeutic drugs effectively improve insulin sensitivity in humans, including two families of drugs, thiazolidinediones and biguanides. These drugs function like exercise, by reducing glucose production in the liver and increasing muscle glucose utilization. An investigation is currently under way to determine whether one of these therapeutic drugs is effective in the horse. ■

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Bibliographies

New bibliographies are available via the Morris Library Web site at <http://www.uky.edu/Agriculture/VetScience/morris>. Topics covered are Contagious Equine Metritis and West Nile Virus. Unlike other bibliographies on the Web site, dates and languages of publication are comprehensive. ■

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