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COMMENTARY

IN THIS ISSUE

Since its inception in 1992, the *Equine Disease Quarterly* has been distributed worldwide in a printed format. The funding to make this possible was provided through the generosity of Lloyd's of London, Underwriters and Brokers and their Kentucky Agents, and in later years, by Underwriters at Lloyd's, London. Circulation of the *Quarterly* went from several hundred copies in October 1992, to approximately 17,000 copies in 2021, and reached readers in over 87 countries.

In late 2020, Lloyd's Underwriters confirmed that because of changing circumstances, it could no longer sustain support of the *Equine Disease Quarterly* beyond 2021. Thankfully, a new sponsor, Equus Standardbred Station, Inc., recently came forward and generously agreed to help the continued distribution of this worthwhile publication.

This recent development provides a long overdue opportunity to bring about a change in how the *Quarterly* can best be made available to its readership. Considered opinion is to transition from mailing print copies to an electronic format, where each issue is available on the internet. This will have the important advantage that once each issue is posted, it will be immediately available to the readership with a link to a pdf for downloading or printing. In time, this will likely give rise to an increase in circulation of the *Equine Disease Quarterly* among veterinarians and equine stakeholders, especially since the publication is available at no charge and material published is not subject to copyright.

While the distribution of future issues of the *Quarterly* will change to an electronic format, the purpose of the publication will remain the same as was stated in the Commentary of the first issue, October 1992. It is to provide information on the incidence of particular diseases/syndromes and the emergence or re-emergence of previously unreported diseases. This will apply to outbreaks occurring in Kentucky, the USA, and other parts of the world. It will also serve as a reference source of the latest information on disease prevention and control strategies.

In preparation for the upcoming changes in how the *Quarterly* will transition into an electronic format, those currently receiving printed versions of the publication are strongly encouraged to review the information provided as follows and return their responses by no later than November 15, 2021.

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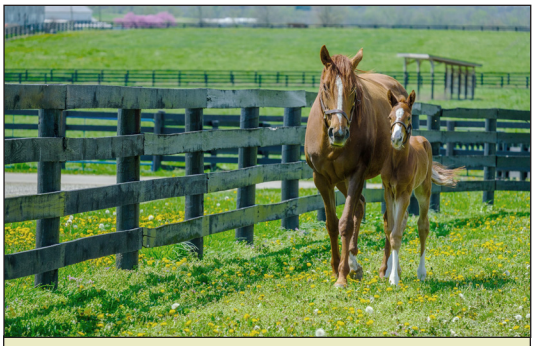

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https://tinyurl.com/EDQemail

In a brief survey, you'll be asked to provide your e-mail address, as well as your name and mailing address as printed on the *Quarterly*.

Please reply by **November 15, 2021**, to continue receiving your issue without interruption.

Thank you for your ongoing interest in receiving the *Equine Disease Quarterly*.

INTERNATIONAL

Second Quarter 2021

The International Thoroughbred Breeders Federation, International Collating Centre, Newmarket, United Kingdom, and other sources reported the following equine disease outbreaks.

African horse sickness was recorded throughout the endemic area in the Republic of South Africa (RSA). Cases were confirmed in all nine provinces including the controlled Protection Zone in the Western Cape Province, where the disease was identified on four premises.

Nepal reported an outbreak of glanders in 26 of 87 horses working at brick kilns and as "tanga-pullers." Of these, 16 horses died.

France, Germany, Tunisia, the UK, and the USA confirmed outbreaks of equine influenza. The number of outbreaks included two each in France and Tunisia, three in Germany, five in the UK, and ten in the USA, where the disease is endemic. With the exception of Tunisia, where 158 out of 861 horses were affected, the majority involved one case.

Outbreaks of strangles were recorded by Belgium (14), Germany (one), the Netherlands (19), Switzerland (seven), and the USA (35), where the disease is endemic. The majority involved one case.

Equine herpesvirus 1 (EHV-1) infection is endemic in most countries. Outbreaks of respiratory disease were reported by Belgium (three), France (10), Germany (one), Italy (11), the Netherlands, the UK and the USA (four each), and Sweden (11), one of which was a case of co-infection with *Streptococcus zooepidemicus*.

Outbreaks of EHV-1 abortion were confirmed by Belgium, Ireland, Japan and the UK (three apiece), Canada (one), France (four), Germany and the Netherlands (two apiece), Sweden (five), and the USA (eight). The majority involved one case.

Outbreaks of EHV-1 neurologic disease were diagnosed by Belgium, France and the UK (one each), Canada (two), Italy (10), and the USA (seven). Most outbreaks involved one case.

Equine herpesvirus 4 (EHV-4) respiratory outbreaks were reported by Belgium (two), France (21), Italy (two), the Netherlands and Switzerland (one apiece), and the USA (reported in multiple states).

Single cases of EHV-4 abortion were recorded by Belgium and Ireland. Italy diagnosed a case of EHV-4 neurologic disease.

The USA reported cases of equine herpesvirus 2 and/or 5, some associated with respiratory disease.

A case of equine adenovirus was diagnosed by the USA. France confirmed two cases of equine arteritis virus infection in stallions.

The USA reported 17 cases of *Rhodococcus equi* infection.

Equine infectious anemia was confirmed by Canada (single cases on four premises), Italy (nine outbreaks, each involving one or two cases), and the USA (seven outbreaks, four involving two to seven cases).

The RSA, Switzerland, and the USA reported cases of equine piroplasmosis. The disease is endemic in RSA, with outbreaks recorded in eight of nine provinces, the majority in Gauteng. Switzerland confirmed two outbreaks, each involving a single case, and the USA one outbreak, involving two Quarter Horses.

Contagious equine metritis was diagnosed by Germany (10 outbreaks, all Icelandic horses) and the UK (one case in an imported stallion). Belgium reported one case of *S. zooepidemicus* abortion.

A range of enteric pathogens were recorded by various countries. The USA diagnosed outbreaks of salmonellosis, two involving serogroup B, one serogroup C1 and one untyped *Salmonella*. Outbreaks of rotavirus infection were confirmed by France (eight), Switzerland (one), and the USA (five involving rotavirus Type A and one Type B). One outbreak of clostridial enterocolitis was reported by Switzerland. The USA confirmed 129 PCR positive samples for *Clostridium perfringens* and 107 for *Clostridiodes difficile*, the majority in Kentucky. Outbreaks of equine coronavirus were diagnosed by Germany (two), Switzerland (one), and the USA (three cases). Single cases of *Lawsonia* enteropathy and Potomac Horse fever were also recorded by the USA.

Several neurological diseases were recorded during the second quarter. Two cases of the dumb form of rabies were confirmed in the USA, one in Colorado and the other in Florida. The USA also reported five outbreaks of Eastern equine encepha-

Equine Disease Quarterly

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lomyelitis and two of West Nile encephalitis. The RSA recorded equine encephalosis as endemic in the country, occurring in eight of nine provinces, with the majority of cases in Gauteng (33) and the Northern Cape (31). Single cases of anaplasmosis were diagnosed by Canada and Switzerland. Switzerland also reported a case involving co-infection with anaplasmosis and ehrlichiosis. Outbreaks of leptospirosis were confirmed by Germany (one) and Switzerland (two).

Exhaustion in Horses

Exhausted horse syndrome refers to a range of metabolic and physiologic conditions that may occur when horses become fatigued. Affected horses may display a decrease in energy, appetite, or appear stiff and weak. In serious cases, cardiac arrhythmias, shock, muscle damage, colic, and diarrhea may develop. Horses that are under-conditioned, performing in endurance events, or exercised in hot or humid environments are at higher risk for exhaustion. If exercise is not immediately halted and treatment initiated, then life-threatening complications may occur.

Exhaustion may develop with any prolonged period of exercise such as endurance rides, three-day events, and extended trail rides. Many factors contribute to the potential for exhaustion. Different breeds are better suited to prolonged exercise while others excel at shorter, more high intensity work. The animal's training and fitness ideally should be suited for the event they are participating in, although even highly prepared animals may develop exhaustion. Any underlying disease, including lameness, anemia, and respiratory disease will increase the risk of fatigue.

Exhaustion is a multifactorial condition. Heat, electrolyte imbalance and energy stores may contribute. A large amount of heat is produced while exercising. Heat needs to be appropriately regulated and removed from an exercising animal via sweat and air movement. If heat is not removed, then the core body temperature steadily increases. Approximately 65% of heat is lost via sweat, 25% via respiratory evaporation, and the remaining 10% via other mechanisms. This is made more difficult when the conditions include high heat and humidity or when the animal is dehydrated. Sweat contains important electrolytes such as sodium, potassium, calcium and chloride which are lost as the horse sweats. If electrolyte imbalances are not corrected, then serious derangements occur leading to shifts in blood pH and cellular stability. The primary energy source for muscles is stored glycogen, which is a finite resource. Glycogen stores can be increased with training and exercise, but once depleted the muscle lacks a primary energy source and exercise will slow or stop.

Horses with exhaustion will have an increased heart rate, temperature, and respiratory rate. They may appear depressed, unwilling to eat and drink, and, in some cases, develop colic, shock, or laminitis. Horses that move with a stiff gait may have significant muscle damage and/or laminitis. These animals should not be forced to move if treatment can be provided on site. Affected horses are typically dehydrated and blood work shows evidence of stress and electrolyte imbalances. Muscle enzyme values will be increased and often continue to increase as muscle damage continues. Kidney values, which reflect both dehydration and renal damage, may be elevated and urine may be significantly decreased in volume and dark brown/red in color. Genetic testing, muscle biopsy or other diagnostic testing may be needed to determine an underlying cause in cases of repeated myopathy or suspected exhaustion.

Treatment includes immediately stopping exercise and initiating assisted cooling. This can be done with electric fans and running large volumes of cold water over the entire body. Intravenous fluids can be given to restore hydration and electrolyte status. Once there is evidence that the intestinal tract is functioning, then oral fluids may be added to aid hydration; however, these should not be administered until the horse has good gut sounds and no signs of colic. Horses should not be transported following exhaustion until cleared by their medical team.

Exhaustion is best prevented by acclimatization, proper training, nutrition, and supplementation of electrolytes. Electrolyte products require adequate water intake to be effective. Horses given concentrated electrolytes without appropriate water consumption will actually increase dehydration. Acclimatization to a climate with excessive heat, altitude, or humidity may take up to two weeks. Conditioning will create larger muscle glycogen stores, improve efficiency of heat elimination, and train horses to eat and drink during prolonged work. It is also important to allow horses enough time to recover and rehydrate from transport prior to beginning competition.

Event organizers will assess the safety associated with heat and humidity, and events may need to be cancelled if environmental conditions are considered dangerous. A rider may be the first to recognize that their horse is displaying abnormal behavior or unwillingness to work, which is often an early sign of exhaustion. Vet checks throughout endurance events are designed to identify horses showing early signs of fatigue. These checks may require horses be rested or removed from competition before serious complications occur.

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NATIONAL

Polyuria and Polydipsia in Horses

Polyuria (PU), defined as an increased volume of urine, is a rare, but significant, indicator of a variety of diseases. Polydipsia (PD), excessive thirst and fluid intake, is the natural companion to PU as these two clinical signs nearly always occur together. A healthy, average-sized horse weighing 1,100 lb (500 kg) will typically drink ~13 gallons (50 L) of water each day, although this can vary based on environmental factors, exercise, pregnancy status, illness, and more. Urine production in healthy, adult horses is ~2.5-4 gallons (10-15 L) per day for an average horse. An adult horse with PD will have a daily fluid intake that is double or more than normal, and horses with PU will often have urine production in excess of 2-3 times normal. Once it has been determined that a horse is PU or PD, a veterinarian should be called to investigate the cause.

Psychogenic Polydipsia: Primary or psychogenic PD (PPD) occurs when a horse drinks excessively in the absence of a physiologic reason and is one of the most common causes of PD and subsequent PU. Diet, management, and housing have been implicated as factors contributing to PPD. This condition, sometimes considered a vice, can be seen in horses housed in stalls and can occur with other behavioral abnormalities such as chewing on doors or eating bedding. Stressors such as changes in housing, management, and environment have also been reported to incite PPD. Dietary factors such as high dry matter intake or compulsive salt consumption may lead to excessive water consumption. Horses with PPD will produce copious volumes of urine, more than those with Cushing's disease or chronic renal failure. Owners often report that stalls or paddocks are soaking wet and the horse drinks more than other horses. Urine will be dilute, but these animals have normal kidney function and are usually in good body condition.

Cushing's Disease (Pituitary Pars Intermedia Dysfunction—PPID): The incidence of PU in horses with PPID is highly variable, but has been estimated to be around 33%.

Chronic Renal Failure (CRF): The most common causes of chronic renal failure (CRF) and end-stage kidney disease in horses are drugs, toxins, myopathy, and developmental abnormalities. Although PU is a relatively common sign of CRF, it sometimes goes unnoticed by owners, because it is mild to moderate. This is especially true if an animal is housed with other horses or is on pasture, making it hard to know how much urine an individual horse is producing. Additional clinical signs are dependent on the stage of CRF, but can include weight loss, ventral edema, rough or dull hair coat, decreased appetite, dental tartar and oral ulcers, blood in the feces, and poor performance.

Diabetes: Two types of diabetes exist and can cause PU/PD in horses, but both types are rare. Diabetes insipidus occurs due to either decreased or absent production of vasopressin, a hormone responsible for absorption of water by the kidneys or insensitivity of the kidneys to vasopressin. Clinical signs include enormous volumes of dilute urine. In contrast, diabetes mellitus (DM) occurs due to insulin deficiency (type 1) or insulin resistance (type 2). PU, PD, and weight loss are common signs in horses with type 2 DM. Differentiation between the two types of diabetes and their respective subtypes is necessary for treatment and prognosis.

Other Causes of Polyuria: Drugs such as diuretics, sedatives, and corticosteroids can cause PU in horses, and other drugs can induce PU by inciting

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acute or chronic kidney injury. Aminoglycosides (gentamicin, amikacin), oxytetracycline, nonsteroidal anti-inflammatories (phenylbutazone, flunixin meglumine), polymyxin B, amphotericin B, imidocarb dipropionate, and bisphosphonates are all potentially toxic to the kidneys and have documented and/or anecdotal reports of renal disease following their use. Additional diseases associated with PU include but are not limited to liver failure, neoplasia, toxicities, inflammatory conditions, and parasitism.

Diagnosis: Testing to determine the cause of PU/PD should start with a chemistry profile and urine analysis for the presence of elevations in kidney enzymes, changes in electrolyte levels, high blood glucose levels, and measurement of urine specific gravity to determine the concentration of the urine. Additional bloodwork may be necessary, and urinalysis and additional urine testing may be useful for identifying early kidney disease. Occasionally, advanced testing is needed to diagnosis the underlying cause of PU.

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Equine Rotavirus B and Neonatal Foal Diarrhea

Group A and B rotaviruses are significant enteric pathogens that cause diarrhea of variable severity in humans and domestic animals. Despite no difference in clinical signs or symptoms, it has been well documented that there is no antibody-mediated cross-protection between groups A and B. The horse is a unique species in which only group A rotaviruses typically cause foal diarrhea outbreaks.

During the 2021 foaling season (February and March) in central Kentucky, increased frequencies of diarrhea were noted in neonatal foals between 1 and 4 days of age. Mares had been previously vaccinated with an inactivated monovalent equine rotavirus A vaccine during their pregnancy. Foals developed diarrhea at approximately 24 h of age and diarrheic episodes typically lasted 3-4 days. Some farms experienced up to 100% morbidity. Newly born foals on affected farms quickly developed diarrhea, suggesting a highly contagious disease. Clinical signs included inappetence, weakness, dehydration, severe electrolyte imbalance, and watery yellow and foul-smelling diarrhea. Hemorrhagic watery diarrhea was noticed in some cases. None of the foal's dams developed diarrhea.

Diagnostic investigation of fecal samples from diseased foals, which included PCR and microbial culture, failed to detect diarrhea-causing pathogens, including equine rotavirus A. Interestingly, metagenomic sequencing data identified an abundant amount of novel rotavirus B genome in fecal specimens from affected foals and absence of other known enteric pathogens, suggesting an etiological role of this rotavirus B in neonatal foal diarrhea. Further evidence linking rotavirus B to this outbreak of highly contagious foal diarrhea is supported by the poor responses of the affected foals to antibiotic treatment as well as seemingly ineffective equine rotavirus A-specific passive maternal antibodies in affected foals against this new virus. Subsequent analysis of 33 foal diarrheic samples by RT-qPCR identified 23 rotavirus B-positive cases (69.69%). Despite a small sample size, the prevalence of equine rotavirus B in diseased foals was similar to that reported in equine rotavirus A-associated foal diarrhea outbreaks.

Bioinformatic analysis of the viral genome data showed that the protein sequence of all 11 viral segments had greater than 96% homology (relatedness) with group B rotaviruses previously found in ruminants. Furthermore, evolutionary studies demonstrated clustering of the equine virus with group B rotaviruses of caprine and bovine strains that circulated in the USA. These results indicate the possibility that the equine rotavirus B was of ruminant origin.

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In summary, evidence suggests that a novel, ruminant-origin, group B rotavirus has emerged in horses and was associated with outbreaks of neonatal foal diarrhea in the 2021 foaling season in Kentucky. Emergence of the ruminant-like group B rotavirus in foals clearly warrants further investigation due to the significant impact of the disease in neonatal foals and its economic impact on the equine industry.

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