WITH the growing occurrence and unpredictable nature of natural disasters, many horse owners are looking for ways to protect their animals. In addition to disasters, horse theft also is growing because owners are looking for guaranteed methods of identifying their horses. Microchip identification was created to improve the traceability of horses in disease outbreak scenarios and allow for the rapid and efficient management of investigations to minimize spread of contagious diseases in horses. Diseases such as equine hemorrhage shock, strangles, influenza, adenovirus, and others can spread rapidly and the ability to quickly identify animals aids veterinarians, farm managers, and other animal health professionals in developing the most appropriate action plan to protect them.

Microchip implantation is safe, simple, and inexpensive and usually will last a horse’s entire lifetime. The cost is generally about $50 to $75 and the chips currently being manufactured are function- ing for 25 years or longer. The tiny, non-migratory chip is the size of a grain of rice and takes only seconds to implant with a small syringe by a veterinarian or other trained person. The chip is implanted halfway between the horse’s poll and withers, just below the mane in the nuchal ligament on the horse’s near (left) side. The injection site is cleaned and disinfected prior to injection and sometimes shaved, ensuring little to no occurrence of an adverse reaction.

Microchip implantation is simple and not a concern for the owner. The accredited veterinarian will use the unique microchip number to record on official health papers and medical records. It is up to the owner to have their unique code maintained in personal medical records or registered with a commercially available and searchable database. A special handheld scanner is used to read the microchip through the skin of the animal. The scanner reads the number on the chip through radio frequency identification technology. Although there are several different companies manufacturing these microchips, most scanners are now considered universal as they are engineered to read a common frequency.

In the 1990s, Louisiana became the first state to require mandatory unique identification for all horses and annual Coggins testing. Microchips are a unique identifier superior to lip tattoos or brands since brands are not unique per horse and both tattoos and brands can be altered and/or difficult to read. Many breed organizations are now requiring microchipping for registration. Microchipping became especially important in the aftermath of hurricanes Katrina, Rita, Gustave, Ike, and Isaac in Louisiana when many horses were separated from their owners and needed to be identified in order to be reunited. Veterinarians working with affected horses in the coastal parishes and across the state of Louisiana in the recent aftermath of Hurricane Harvey are now finding microchipping invaluable with the massive ongoing sheltering operations. There is really no downside and no reason that a horse should not have microchip identification.

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

Outbreaks of African horse sickness were reported in all provinces in South Africa as the Western Cape. Occurrence was as expected for the time of the year.

Equine influenza was reported by the UK and the USA. The UK confirmed one outbreak. Influenza was recorded in 14 states in the USA, where it is considered endemic.

France, Germany, South Africa, Switzerland, and the USA recorded outbreaks of encephalitis. The number of confirmed outbreaks included those in Switzerland, four in Germany, and 11 in France.

Where the disease was reported as sporadic in South Africa, it is considered endemic in the USA. High clinical attack rates were seen in ponies and horses on a premises in one western state and in correlation with fumigations in two other states.

Equine herpesvirus (EHV-1) related disease was confirmed on a total of 13 premises. France confirmed one outbreak, Germany, Switzerland and the USA recorded one or two outbreaks and the USA reported that the disease was present in various states. Cases of abortion were diagnosed in France (two outbreaks), Germany (three outbreaks), Japan (one outbreak), the UK (two outbreaks), and the USA (one outbreak). EHV-1 related neurological disease was confirmed in France (one case), the UK (isolated cases on two premises), and the USA (four outbreaks on facilities in California, Colorado, Maryland, and New Jersey, each involving a single case of the disease).

Equine herpesvirus 4 was reported by France, Germany, Ireland, and the UK. The number of outbreaks varied from one in Ireland, three in Germany, four in the UK, and 14 in France. The USA recorded multiple cases of equine herpesvirus 2 and 5 infection, some associated with respiratory disease, in several states.

Equine infection anemia was confirmed in Germany, Switzerland, and the USA. The disease was identified on three premises in Germany, one in Switzerland, and five in the USA, both epidemiologically linked, with three cases on each premises.

France and Switzerland reported the presence of equine piroplasmosis. France confirmed the infection endemic and Switzerland recorded a single case of the disease. Contiguous equine encephalitis was reported in Germany. Two Icelandic stallions and fine mares were confirmed positive on a total of 13 premises.

France diagnosed a case of equine viral encephalitis (equine herpesvirus 4 in two premises as well as one case of leptospirosis infection).

Neurological placitis was confirmed in Kentucky, USA, with five cases caused by photomyiasis (lipomyas) and two by Cordyceps gypseus.

Twenty-three cases of leptospirosis were reported by the USA during the second quarter of 2017; reported serogroups included B (15), C1 (2), and C2 (6).

The USA confirmed three cases of Crimean-Congo hemorrhagic fever in Kentucky, one case of Tickborne disease (E. chaffeensis), one case of T. equi, and one case of T. equi (E. phagocytophilum) and isolated cases of C. mouth and C. felis infection.

Three outbreaks of vesicular diarrhea were recorded in France. The USA reported a case of pulmonary attherosclerosis (Leuconostomus) in a feed.

Rhodococcus related disease was reported as endemic in the USA, with 22 cases recorded during the review period.

Switzerland diagnosed two cases of arthropod myopathy and four cases of distemper, the latter involving three premises.

West Nile encephalitis was reported by South Africa (34 cases, three cases, and the USA (one case). Five of the cases in South Africa represented co-infection with equine piroplasmosis and six with the USA (one case). The disease was reported in all provinces in South Africa as the Western Cape.

Equine metritis was diagnosed in France (44 cases, lineage 2 virus) and the USA (one case). The USA confirmed three cases of Crimean-Congo hemorrhagic fever in Kentucky, one case of T. equi (E. phagocytophilum), and isolated cases of C. mouth and C. felis infection.

Three outbreaks of rotaviral diarrhea were confirmed in Switzerland. The USA confirmed three cases of rotaviral diarrhea in California, Colorado, and New Jersey, each involving a single case of the disease.

The USA confirmed one outbreak of anthrax, New York (one case). The disease was reported in all provinces in South Africa as the Western Cape.

The USA confirmed one outbreak of anaplasmosis, New York (one case). Anaplasma marginale was identified on three premises in Germany, and two by Cordyceps gypseus.

The USA confirmed one outbreak of pyopneumothorax, New York (one case). The disease was reported in all provinces in South Africa as the Western Cape.

The USA confirmed one outbreak of equine piroplasmosis, New York (one case). Piroplasmosis was confirmed in Switzerland (equine piroplasmosis) on two premises as well as one case of leptospirosis infection.

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Equine Colic Update

There is a prophecy about equine colic. As long as there are horses, they will have colic. Based on reports in the literature over the last 50 years, the incidence of all colic cases has not changed over time and the incidence in a normal horse population ranges from 1 to 10 episodes of colic per 100 horses per year. However, horses that have had one or more previous episodes of colic have an incidence rate up to 5 times higher. Survival varies with the severity of the disease, but the overall mortality rate for all types of colic in horses is over 75%. Mortality is highest for cases related to mesenteric ischemia due to mesenteric infarction. Incidence from horses in Southern California, with a higher frequency expected in specific populations of horses such as breeding mare mares, horses with small numbers of foals, or those exposed to abrupt changes in management. Women being treated for dysmenorrhea and those hospitalized after general anesthesia for elective procedures have an increased incidence of colic.

The causes of these acute, mesenteric intestinal disorders include colic. Simple colic, commonly diagnosed as “gas colic,” “spasmodic colic,” or “spasm,” makes up approximately 85% of all colic episodes, yet the cause or mechanism for these gastrointestinal abnormalities remains unknown. Similarly, the cause of cases with a higher mortality and mortality such as intestinal strangulations have yet to be known. Epidemiological research over the past three decades has identified risk factors appearing to have a causal relationship to specific mechanisms such as dietary intake and transection on the necessity to be elucidated. Although gastrointestinal function is most likely multifactorial, with combined current risk factors and increased risk.

Numerous factors are reported to increase colic risk including some basic husbandry and feeding practices (Table 1). Some of these factors are specific, but still are significantly related to increased colic risk.

Over the last few years, survival has improved for the most life-threatening types of colic. During the 1960s to 70s, the survival rate for severe obstructions and strangulations was near zero. Over the last few years, survival has increased to less than 50%. Although improvements in surgical expertise, anesthesiology, and perioperative management have contributed to improved surgical outcomes, the continued development of new antifibrinolytic medications and an increased understanding of the role of endotoxemia and its ability to improve colic treatment and survival.

The causes of colic are multifactorial and the ability to improve colic treatment and survival.

Still, how intestinal dysfunction and systemic responses are initiated remain to be discovered. More research is needed if we are to change the colic prophecy.

Equine Influenza Beyond Equines

The human “influenza season” in North America is now about to begin again, while in South America a spring is approaching and their influenza season is almost over. The influenza season happens every year during autumn, winter, and early spring months, and the influenza viruses that circulate each season tend to be the same except for influenza A/H1N1, A/H5N1, and influenza B viruses.

However, there are many other influenza viruses in the world. The ‘H’ and ‘N’ varieties (called “subtypes”) of influenza A viruses now go up to H30 and N11 with the recent discovery of new subtypes in pigs. Most of these subtypes are rare in mammalian hosts but common in wild animals. The only influenza subtypes that have been confirmed to infect horses recently are A/H3N2 and A/H7N7, and the horse-adapted H5N1 virus appears to have disappeared from horses nearly 40 years ago.

Does this mean that horses cannot be infected by influenza viruses from other species of animals? The answer is no, possibly they can be. Transmission of influenza viruses between different species definitely occurs. Humans, mice, dogs, cats, horses, seals, and sometimes other mammals

Table 1. Factors predisposing to colic

<table>
<thead>
<tr>
<th>Factor</th>
<th>Description</th>
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<tr>
<td>Breed</td>
<td>Older age</td>
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<tr>
<td></td>
<td>Inactivity following injury</td>
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<td></td>
<td>Diet change</td>
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<td>Increased time in a stall</td>
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<td>Absence of deworming</td>
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<td>Recent transport</td>
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<td>High concentrate feeds</td>
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<td>Decreased potable water</td>
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<td>Change in weather</td>
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<td></td>
<td>Round bale hay</td>
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<tr>
<td></td>
<td>Cribbing/crib-biting</td>
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<tr>
<td></td>
<td>History of colic</td>
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<tr>
<td></td>
<td>Tapeworm infection</td>
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<td>Absence of dental exams</td>
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such as mink have occasionally been infected by influenza viruses from birds. This was long thought to happen only rarely, but since 1997 in southeast Asia there have been annual occurrences of humans contracting bird flu subtypes such as H5N1 or H7N9 and these cases are often lethal. Almost all of these cases have been dead-end transmissions, meaning that each case appears to be a separate event with very little evidence they are capable of spreading from human to human.

Can bird flu viruses infect horses? The answer is most likely yes. One piece of evidence is that the H3N8 subtype was not always circulating in horses; it first appeared in 1963, and its genetic ancestors seem to have been bird flu viruses. In 1989 in northern China a strain of bird flu was positively confirmed to cause a large-scale disease outbreak in horses. Its subtype was also H3N8. Was that coincidence or is there some unique characteristic of the H3N8 subtype that makes it more apt to infect horses? Those questions remain unanswered. It is known that the molecular receptors present on the surface of horse tracheal respiratory cells are a little different from other species, which could be a contributing factor to the specificity of the influenza viruses that infect horses. Work in the author’s laboratory suggests that this is not the entire explanation and other factors must be involved.

A related question is whether influenza viruses can be transmitted from horses to other mammals. That answer is definitely yes. About 15-20 years ago, H3N8 horse flu infected dogs in the USA and has persisted in dogs ever since. Can it jump from dogs back into horses?—maybe, although none of the dogs that have adapted to dogs have become much less adapted to horses.

Are humans at risk of infection from equine influenza viruses? Experimental infection of humans has been conducted 50 years ago suggesting that infection can happen but the route is mostly not well understood. Humans with horse exposure do sometimes develop antibodies against equine influenza viruses. The closely related virus of equine influenza has actually demonstrated the presence of equine influenza virus in that patient’s mucosa. The message for readers is: Transmission of influenza viruses from one species to another can happen and sometimes does happen. If your horse is sick with the flu, take elementary biosafety precautions such as washing hands and equipment that came in contact with that horse, as soap will kill influenza viruses. And if you are sick with the flu, it is probably best not to cuddle your pets.

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Equine Identification in the United States

The need for horse identification spans back to the 1800s, when branding cattle and horses was commonplace for tracing these animals to specific ranches or owners. Historically, horses were identified by physical description. With many solid horses without unique identifying markings, this identification modality had limited usefulness. For example, it would be challenging to individually describe a group of yearling Friesian fillies in a field. The transition to utilization of individual animal brands and lip tattoos aided in the identification of equine in the United States. Unfortunately, the recognized disadvantages of these identification modalities include inducing pain and stress in the animal, potential transmission of disease agents, and inherent safety issues associated with the procedures. Additionally, brands cannot be canceled and uniquely identified. Over the years, the industry has researched innovative ways to uniquely identify horses. Iris scanning was developed by Japanese researchers in 2000, but the expense of this process and limited access to reading equipment made this a non-viable option. The recent advances in microchip technology have made this procedure the desired identification modality of the future. Today, the international standards for acceptable microchips are:

- ISO 11784/11785 compliant and ICAR certified
- 15-digit numeric, no letter format
- 134.2 kHz frequency

National
Concerns have been raised regarding impact of the invasive procedure. However, subsequent microchip implantation studies have proven that microchip administration yields minor transient pain and inflammation at the injection site and minimal microchip migration following the correct implementation in horses.

Recently, improved microchip temperature sensing technology has enabled accurate body temperature recording. Not only does this type of chip provide accurate individual identification, but it also provides a take-home health monitoring benefit. For example, should an equine hyperthermia myoglobinuria incident occur at a large breeding facility, management could easily and rapidly monitor temperature per the biothermal microchip and move horses with elevated temperature to isolation before the horses start shedding viruses.

In the last five years, the equine industry in the United States has embraced the use of microchips for equine identification. In 2008, the Jockey Club began offering microchips for sale to its members. The Jockey Club began mandating that thoroughbred foals have an ISO 11784/11785 compliant microchip implanted starting with the 2017 foal crop. Impressively, Jockey Club members embraced microchipping technology and two-thirds of the 2016 foal crop were voluntarily microchipped. Recognizing the need and benefit of microchipping, the United States Hunter Jumper Association (USHJA) passed a regulation change requiring microchips starting in the 2018 competition year. During that competition year, any horse entering the show in the USHJA young horse division must be microchipped. Recognizing the need for microchips, the Equine Identification Forum was established in January 2017, the industry recognized the great strides that have been taken and has agreed that additional efforts are necessary for industry wide acceptance of microchipping. For more information on the Forum discussions visit http://www.animalagriculture.org/proceedings/equineidforum.

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Clostridium Perfringens and Necrotizing Enterocolitis in Neonatal Foals

Necrotizing enterocolitis (damage and death of cells in small intestine and colon) is a serious disease of sudden onset in foals less than 4-6 days of age associated with a high death rate, despite therapeutic interventions. Clinical manifestations of enterocolitis in foals can vary from being found dead to a spectrum of clinical manifestations. The most common of these include failure to nurse, distention, peritonitis, and death. Although the association of Clostridium difficile, Neorickettsia risticii (Potomac horse fever) and Salmonella infection with enterocolitis in adult horses is clearly established, many cases of fatal necrotizing enterocolitis in foals have no known risk factors. Clostridium perfringens is the most commonly isolated clostridial species worldwide and is part of the normal intestinal flora. Following acquisition of this bacterium by the foal from the mare’s udder or the environment, the organism multiplies rapidly in the stomach and intestines. C. perfringens numbers are reduced fairly quickly, so that by the time foals are several months of age, the organism is found in relatively low numbers within the large intestine. The almost universal presence of C. perfringens, belonging to the type A group, in the intestine of healthy young animals has complicated the understanding of its role in enterocolitis. Although most strains don’t cause intestinal disease, there are two types that do so in foals. One of these, C. perfringens type C, is a well-established but relatively uncommon cause of necrotizing enterocolitis in foals. The other, C. perfringens type A, includes a subset that produces a novel pore-forming toxin called NetF (Necrotizing enteritis toxin, Foal). This novel toxin is related...
to the beta toxin of type C strains that causes severe enteric disease in foals, other species, and in humans. We found netF-positive C. perfringens in 74% (11/15) of foals with necrotizing enteritis but not in 11 foals with undifferentiated diarrheal disease. In another study, netF-positive C. perfringens was identified in 6 of 23 isolates from foals in Kentucky with severe enteritis. In adult horses with undifferentiated diarrhea, the detection rate of netF among C. perfringens isolates was low (4/58). This would suggest that netF is primarily associated with necrotizing enteritis in neonatal foals.

One explanation why type C and netF-positive type A C. perfringens cause necrotizing enteritis in very young foals is because of the trypsin inhibiting effect of colostrum. Trypsin is a protein-degrading enzyme secreted by the pancreas during digestion, and its inhibition by colostrum prevents the breakdown of protein toxins, such as NetF. The reservoir for NetF-producing C. perfringens is not yet known.

Real-time PCR can be used to rapidly diagnose necrotizing enteritis caused by netF-positive C. perfringens. An alternative but slower approach is to culture C. perfringens and confirm the presence of toxin genes (cph for type C and netF for type A) by PCR.

In terms of prevention, an autogenous bacterin-toxoid vaccine has been used for mare immunization in Kentucky in an attempt to prevent type A C. perfringens enteritis in foals. This vaccine likely includes NetF since colostrum is inhibited with the vaccine has antibodies to NetF. Hyperimmune plasma is commercially available for the prevention or treatment of necrotizing enteritis in foals caused by C. perfringens type A, including NetF, C, and D.

Although, the discovery of NetF has furthered our understanding of C. perfringens enterocolitis in foals, further work is required to fully understand how NetF-producing type A C. perfringens causes disease. Research studies are slowly chipping away at C. perfringens enterocolitis, and the discovery of NetF has been another important step forward.

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