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Biosecurity and Equine Infectious Diseases

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Glossary

Biosecurity All hygienic practices (measures) designed to prevent the occurrence of infectious disease.

Equine herpesvirus 1 neurological disease (Equine herpes myeloencephalopathy, EHM) It is rare in the horse population but apparently increasing in incidence now occurring as outbreaks rather than as individual cases.

Infectious disease Pathogens spread easily from subject to subject either directly or indirectly; in general the index subject has the disease but may be a carrier.

Methicillin-resistant *Staphylococcus aureus* (MRSA) An emerging pathogen of the horse particularly associated with wounds and surgical site infections.

Neurological disease A condition affecting the nervous system control over motor and sensory functions producing a deficit.

Strangles A condition caused by *Streptococcus equi* subspecies *equi*, an unusual inhabitant of the horse's upper respiratory tract, can occur as outbreaks or affect individuals in closed herds through contact with carriers.

Zoonotic disease A disease that can be spread directly from animals to humans or from humans to animals.

Introduction

Infectious diseases are a constant threat to the health and welfare of horses. Several diseases have assumed greater importance as the performance and pleasure horse populations and equine activities have increased and there are new owners who do not understand the implications of equine infectious disease outbreaks to their animals. In addition, emerging diseases have beset the equine species in recent years in North America. Firstly, the introduction of the Old World West Nile virus to a susceptible population has made the virus to be considered as endemic. Secondly, the change in the antigenicity of equine herpesvirus-1 (EHV-1) to induce a more aggressive form of neurological disease may affect clusters of horses than the field strain encountered previously. Fortunately, North America has been spared the infection caused by the paramyxovirus responsible for Hendra virus infection spread by flying foxes (bats), which is a zoonotic disease and a deadly infection of horses in Australia. However, other infectious diseases are more likely to emerge among horses in North America in the next decade as pathogens and vectors move into and become accustomed to new climate zones. The threat of emergent diseases is apparent in the UK, where a warming climate could precipitate a northward spread of those *Culicoides* spp. responsible for African horse sickness among a highly susceptible population.

Vaccination and preventive management procedures are critical to disease prevention, but these need to be more widely adopted, complied with, and promulgated. The [National Animal Health Monitoring System \(1998\)](#) survey raised the specter that a significant number of horses throughout the United States do not receive regular vaccinations and preventive health is not part of the owners' protocol. Therefore, much work remains to be done in communicating the message before a devastating outbreak of infectious disease among horses takes hold. Unfortunately, vaccines are unavailable or are inefficient for several infectious diseases in the United States. Successful vaccines to protect against equine herpes

myeloencephalopathy (EHM) and equine strangles are awaited.

The list of infectious diseases, and the pathogens involved, considered as real or potential threats or concerns in the United States by veterinarians, technicians, and horse owners continues to expand. Some are emerging or emergent diseases (E). Other infectious diseases are potential zoonotic diseases (Z), spread from horses to humans or vice versa ([Table 1](#)).

This article provides an overview of biosecurity measures that apply to all potential pathogens with an emphasis on the principles learned from the threat of EHV-1 myeloencephalopathy (an emergent disease), MRSA (a zoonotic disease), and strangles, an infectious disease of historical record that remains a scourge to susceptible horses. The emphasis is on examples from the United States, although the principles of biosecurity are applicable elsewhere.

Much has been written in the veterinary, equine, and lay press on biosecurity matters and the threat of equine infectious diseases getting out of hand. The threat is still real. The human race lurches from one crisis to the next having eluded the bullet without learning from its errors. Biosecurity is much cheaper than dealing with actual infectious disease in any species. The importance of hand washing, the preeminent biosecurity measure, is a major factor why nosocomial infections with MRSA and *C. difficile* are such hazards to patients in human hospitals. The healthcare workers do not routinely wash their hands between patients. What has one learned?

Biosecurity

Biosecurity represents an important part of the veterinarian's responsibilities in the clinic or practice facility, at farms, stables, barns, and at shows and events, where horses commingle from multiple locations. Technicians, owners, and caretakers must understand the implications, share the responsibilities, and implement specific management procedures. Communication with and education of all personnel

Table 1 List of infectious diseases of horses

Viral
<ul style="list-style-type: none"> ● Equine coronavirus ● EHV-1 respiratory disease ● EHV-1 neurological disease (EHM) (E) ● EHV-4 respiratory disease ● Equine influenza ● West Nile virus (Z not by horses) (E) ● Vesicular stomatitis virus (multiple species) ● Equine viral arteritis ● Rabies (Z) ● Eastern/Western equine encephalitis (Z not by horse) ● Equine infectious anemia
Foreign animal diseases (viral)
<ul style="list-style-type: none"> ● Venezuelan equine encephalitis (Z) ● Japanese encephalitis (Z not by horse) ● African horse sickness ● Hendra virus (Z)
Bacterial
<ul style="list-style-type: none"> ● <i>Salmonella</i> sp. (Z) ● Strangles (<i>Streptococcus equi</i> var <i>equi</i>) ● Contagious equine metritis (<i>Taylorella equigenitalis</i>) ● Methicillin-resistant <i>Staphylococcus aureus</i> (MRSA) (Z) (E) ● <i>Leptospira</i> sp. (some serovars are Z) ● <i>Clostridium difficile</i> (Z) ● <i>Clostridium perfringens</i> ● <i>Lawsonia intracellularis</i> ● <i>Rhodococcus equi</i>
Foreign animal disease (bacterial)
<ul style="list-style-type: none"> ● Glanders (<i>Pseudomonas mallei</i>) (Z)
Protozoal (Tick borne)
<ul style="list-style-type: none"> ● Equine piroplasmiasis (babesiosis) ● Potomac horse fever (<i>Neorickettsia risticii</i>) ● Lyme Disease (<i>Borrelia burgdorferi</i>)

on infectious disease control measures is vital. This is before an outbreak happens and subsequently at regular intervals. Technicians must be empowered to enforce the codes of practice. They are a constant presence at the facility and integral to sustaining biosecurity procedures.

Biosecurity includes all hygienic practices designed to prevent the occurrence of infectious diseases, for example, preventing the introduction of infectious agents, controlling the spread within populations or facilities, and the containment or disinfection of infectious materials. Biosecurity is affected by the ecology of animal and human populations, the biological nature of infectious agents, and the management actions affecting interaction between host and agent. Rigorous hand hygiene is an important part of effective biosecurity and is emphasized throughout this article.

Preparedness is the Key to Biosecurity

A plan of action to manage equine infectious disease outbreaks should be in place before the onset of clinical disease

based on the evaluations of risk assessment, resource management, and horse management. All personnel must be familiar with and preferably certified on the infectious disease protocol for the premises. Risk assessment is the knowledge of ongoing disease outbreaks elsewhere and the potential for their spread. Resource management includes devising and establishing a chain of command with defined responsibilities, for example, to identify isolation and biosecurity capabilities both on- and off-site; to insure the availability of diagnostic sampling materials, disinfectants, and biocontainment materials; and to inspect facilities recently vacated after another equine or livestock event for adequate sanitation (decontamination) and waste removal before animals are readmitted (this includes transport vehicles and trailers/vans). Horse management involves publicizing and enforcing health requirements for access to the facility and implementing an equine identification and tracking system.

If an infectious disease is suspected, the veterinarian must make decisions and assume responsibilities according to the devised plan. The clinician should communicate to all individuals (key personnel, technicians, and owners at the premises) the established plan and indicate that diagnostic testing is valuable but can take time. The clinician must think biosecurity by not making the situation worse, for example, avoid rushing into a barn/stall without having a plan to leave it and must respond to the 'worst-case scenario' until a diagnosis is made.

In the case of a positive (suspect) diagnosis, management procedures based on disease-specific guidelines must be adopted. If there is no diagnosis, biosecurity measures must be maintained for a minimum of 21–28 days after the last case had developed, the differential diagnosis list must be expanded, and infectious disease experts and state health officials must be contacted.

Biosecurity measures have assumed a much greater level of significance based on the experiences gained by many veterinarians and support personnel engaged in managing outbreaks of life-threatening equine salmonellosis mainly in referral hospitals and, more recently, neurological disease associated with EHM at racetracks, showgrounds with large horse populations, and tertiary referral hospitals. Most tertiary referral hospitals, particularly those at universities, have infectious disease control policies and procedures for horses and other species that are updated regularly. Infectious disease control strategies (policies and procedures) should be developed for private practices (and individual equine operations) to cover on-site horse health admissions, accommodation, patterns of personnel and animal traffic on-site, disinfection, waste management, and off-site farm/stable/barn precautions by veterinarians and other personnel.

Biosecurity is only as effective as the acceptance of and compliance with regulations by all personnel, for example, the equine influenza outbreak that emanated from a quarantine center in Australia. Moreover, biosecurity measures however well developed and accepted cannot be taken for granted, for example, spread of foot-and-mouth disease virus from Pirbright, England (foot-and-mouth disease virus World Reference Laboratory) on the tires of contractors' vehicles.

Biosecurity Measures to Limit Infectious Disease Spread

Biosecurity for the technician at a veterinary practice and for the horse owner or caretaker is critical to managing the introduction of an infectious agent. The most common means of spread is through the introduction of a new horse, particularly a horse that is returned from a hospital to a property. New horses should be isolated from the resident population for a minimum of 14 days, ideally for 30 days. Clinical condition and temperature should be monitored daily. Supplies should be separated. The recent arrival should be attended to last. Manure and bedding should not be spread on the fields. Hand washing in running water with soap after handling horses (waterless hand foams or gels if no running water) should be mandatory even if gloves have been worn.

Vaccination is not 100% preventive but is critical to control an infectious disease as exposure is unlikely to be prevented. Vaccination decreases the severity of clinical disease and serves to increase resistance against certain diseases in individual horses and in horse populations.

Quarantine is an important aspect of implementing biosecurity measures. Ideally, there should be a separate facility used for any horse that has left the premises for showing or breeding (commingling) and not just for sick or new horses.

Technicians and handlers should initiate a traffic pattern, wear disposable gloves, booties, and barrier clothing with at-risk and sick horses, and practice the procedure for removing protective gear (without further contamination). Cleaning and disinfection frequency should be enhanced during an outbreak and hand hygiene is crucial.

Visitors should be restricted to one entrance and exit. Vehicle tires and visitor's shoes should be disinfected and footbaths and overboots must be provided. Records of visitors and their horse contact must be kept.

It is critical to be vigilant away from the horse premises (e.g., avoid direct contact with a diseased horse at an event or activity and do not touch horses' noses going down the line from stall to stall) and follow a specific biosecurity procedure on returning.

Biosecurity requires common sense and the ability to communicate clearly. The mantra is to adopt precautionary measures to include waste management, cleaning and disinfection, environmental control, and storage and use of equipment and supplies. Biosecurity plans for any horse facility are never completed. They may be patterned by a particular outbreak and should be reevaluated and updated regularly.

Equine Herpesvirus-1 Neurological Disease

EHV-1, a ubiquitous virus among horses, was occasionally responsible for neurological disease. However, when groups of horses became affected by a neurological disease at the same time, diagnostic results showed the agent to be a variant of EHV-1, prompting the adoption of strict biosecurity measures. Much was learned in a short time frame. Increased risk for exposure in recent US outbreaks included commercial shipping with horses collected from multiple sites and venues, such as quarantine stations, rest farms and stops, veterinary

clinics, race tracks, polo events, and hunter/jumper dressage shows, and movement of animals before the disease is recognized. Other important risk factors involved recent exposure to sick horses, including those having increased rectal temperature without evidence of recent surgery, vaccination, or other medical condition associated with fever, and recent exposure to horses that have acute neurological signs, especially if a noninfectious cause has been ruled out.

EHVs are major equine pathogens. There are nine EHV, not all are associated with overt clinical problems. EHV-1 and EHV-4 are α -herpes viruses transmitted by the respiratory route, infect the upper respiratory tract epithelium, and are the most common.

EHV-1 myeloencephalopathy (EHM) is not a new disease; however, neurological disease associated with EHV-1 (causing EHM) appears to be increasing in incidence in the United States and Europe, although it remains a relatively rare disease in the horse population. Outbreaks of EHM have occurred among pleasure horses and racehorses, show jumpers, horses referred to tertiary clinics, and quarter horses at a cutting horse show. This disease has had significant economic and equine activity impacts. Some racetracks ordered quarantine for several weeks for thousands of horses sent to compete after multiple cases of EHV-1 causing neurological disease occurred, resulting in deaths of several horses. Shows were canceled in many Western states and interstate movement of horses was restricted after the cutting horse event. Some tertiary referral centers have imposed strict quarantine protocols after clinical cases of EHM were confirmed at their facilities.

EHV-1 is highly contagious and is a serious concern and threat to those involved with horse shows, events, farms, auctions, and clinics with a high volume of horse traffic. Certain strains of EHV-1 that affect the central nervous system have resulted in outbreaks of EHM with high morbidity and case fatality. Horses with neurological signs have large viral loads in their blood and nasal secretions. The incubation period is highly variable depending on the host, pathogen, and the environment (including stress) and ranges from 1 to 12 days but often 1 to 3 days. Once sick, the horse can shed the virus and be a source of infection for 7–10 days and for up to 28 days in some instances. Horses can be clinically normal and still shed the virus.

The virus may be disseminated by nasal and respiratory secretions for up to 11 m (35 feet) (animals should be separated by 300 feet) and can survive a wide temperature range (surviving 3–4 weeks in the environment without a host). Transmission can occur directly by nose-to-nose contact and indirectly by vectors, contaminated hands, shoes, clothing and equipment, inanimate objects, such as water buckets, brushes, bits, clippers, etc., and aborted placenta and fetal tissues.

Clinical signs of EHV-1 infection vary from mild respiratory disease to severe EHM. Neurological signs are generally, although not exclusively associated with a recent history of pyrexia, respiratory disease, or abortion in the affected animal or more likely a herd mate. Respiratory signs may be minimal and of short duration. Increased rectal temperature may be the only clinical sign. There may be two fever spikes – an initial temperature rise that is usually mild (101.5–102.5), and transient for up to 4–5 days, and may be missed; the horse may remain clinically normal or develop respiratory disease

with signs of nasal discharge, an increased temperature (> 102.5), and coughing. The second wave starts between days 5 and 7 and can continue until day 10 coinciding with the viremia. Infected pregnant mares may spontaneously abort within a week to several months of being exposed, although the mares may have shown only limited initial signs. Other signs include scrotal edema and loss of libido in stallions, uveitis, dependent and facial edema in adults, nasal discharge, and pneumonia in young foals.

Neurological Signs

Expression of clinical signs in the relatively small number of EHM cases is a consequence of vasculitis followed by hemorrhage, thrombosis, hypoxia, and secondary ischemic degeneration in the central nervous system.

Signs include progressive or acute onset ataxia (incoordination) or recumbency approximately 1 week after viral exposure that affects a single animal or group of at least 2 years of age. Signs may progress rapidly over 24–48 h. Pyrexia and depression are usually noted 3–4 days before the onset of neurological signs, which include pelvic limb ataxia, paresis, or paralysis and may affect all four limbs. Signs are usually more severe in the pelvic limbs than in the thoracic limbs and may lateralize. There may be signs consistent with lower motor neuron dysfunction, urinary incontinence, reduced tail tone, penile or vulval flaccidity, and loss of perineal sensation. Tetraplegia and death may supervene. Paralysis and recumbency carry an increasingly poor prognosis. Horses rarely show abnormal mentation or develop cranial nerve signs. Most horses exhibit mild to moderate neurological signs and stabilize rapidly becoming normal 3–6 months after onset of clinical signs, although residual deficits may persist.

EHV infections are ubiquitous. Most animals are infected during the first year of life. The pathogen is latent. Up to 80% of horses (possibly more) are purported to be latently infected with EHV-1 or EHV-4, cannot easily be identified, and serve as important reservoirs.

EHM outbreaks have occurred in late fall through late spring, possibly coinciding with pregnancy and lactation associated with abortigenic EHV-1 in mares and respiratory EHV-4 transmission among foals. EHM outbreaks may be dependent on the reactivation of latent EHV-1, EHV-4, or both viruses on introducing a new animal into a population and associated stress. EHV-1 (and possibly EHV-4) can remain latent for the horse's entire life. Under stress conditions, the virus may replicate and is shed in nasal secretions. Other horses can be exposed and infected without necessarily the shedder becoming ill. Outbreaks may represent new infections with a virulent strain that occurs in a large number of horses over a short period of time.

Immunology

High virus-neutralizing titers (humoral immune response) are detected within a few days of the onset of neurological signs. However, there is no correlation between the level of serum antibody titers and virus protection. Viremia occurs in the presence of neutralizing antibodies, as apparent in vaccinated or naturally immune horses. Cellular immunity is critical and

complex and plays an important role in clearing EHV-1 leukocyte-associated viremia. EHV-1 exhibits similarities with many herpes viruses and may be capable of modulating (evading) the horse's immune response.

Therapy

Supportive therapy may be needed for several weeks to months and includes slinging the horse if it is weak in the pelvic limbs or is recumbent. Ataxic horses that remain standing may be better on a (firm) ground surface than in a stall. Use of nonsteroidal anti-inflammatory drugs, such as flunixin meglumine, makes the patient feel better, and the antiherpesvirus drugs widely used in human patients, acyclovir and valacyclovir, may be beneficial early in the course of the disease or prophylactically.

Prognosis

Many horses stabilize and recover completely in weeks to months. Some retain residual neurological deficits. The prognosis is worse if the horse is recumbent in the first 24 h.

Prevention

Efficacy of EHV-1 vaccines is questionable and randomized controlled studies are lacking. Vaccine products provide varying levels of protection against respiratory disease (modified live virus (MLV) and killed) and abortion (killed and some MLV). Anecdotal evidence suggests that vaccination may offer reasonable protection against a flare up of respiratory disease (although few data exist). Protection against abortion is less clear as the prevalence is extremely low. Current EHV-1, EHV-4, or EHV-1/4 vaccines are not effective against the neurological disease. At best the vaccinated horse that develops neurological disease will shed fewer virus particles and be less likely to spread the disease to other horses. There is evidence of an association between neurological disease and recent and regular vaccinations with mono- or bivalent killed vaccines.

On premises with confirmed EHM, exposed horses should not receive booster vaccinations. However, the immune status of nonexposed horses and those that must enter the premises if not vaccinated against EHV-1 within the past 60 days should be enhanced by booster vaccination. The reduced nasal shedding of infectious EHV-1 by recently vaccinated horses may indirectly help to protect other horses by reducing the dose of exposed virus.

Regulations

State animal health officials provide information on enhanced requirements in the face of an outbreak.

Several states require reporting of EHV-1 (known as EHM) and quarantine of affected and in-contact horses.

Clinical Outcomes

Clinical outcomes in EHM outbreaks are modulated by many risk factors, including the host immune system (vaccination,

cytotoxic T-lymphocytes, and major histocompatibility complex), stress (lactation, double infections, and racing), and the genetic characteristics of virus isolates. The proportion of animals showing neurological signs when infected with the neuropathic strain varies with each outbreak. Neurological diseases outbreaks are relatively rare compared with non-neurological disease outbreaks, suggesting that the majority of EHV-1 strains encode the nonneuropathic strain.

Diagnostic Testing

Identification of the pathogen requires nasal swab (shedding may be of short duration) and whole blood sample (for buffy coat) at the onset of clinical signs (temperature). Polymerase chain reaction provides the most rapid testing. Real-time polymerase chain reaction (RT-PCR) assays are used to detect and differentiate neuropathic from nonneuropathic EHV-1 strains.

RT-PCR assays detect only viral genomic deoxyribonucleic acid in the specimens tested and are unable to distinguish between lytic, dead, or latent virus and do not predict clinical outcome. Interpretation of PCR viral detection for EHV-1 should be done only in the context of the presenting signs for disease in the horse being tested. The significance of a positive PCR in an asymptomatic horse is unknown.

Horses with high fevers and signs of coughing or mild nasal discharge, with or without neurological deficits, should be tested for EHV-1 by PCR diagnostics if there are no other explanations for their signs of disease. Detection of a positive PCR for EHV-1 in such instances should warrant isolation and limited movement of exposed horses.

Management of Outbreaks of Neurological Disease Associated with EHV-1

If the horse is at high risk for exposure, primary security measures must be implemented to include stopping any horse movement at the event or hospital, confining the horse to a stall or moving immediately to a separate facility and placing all horses with clinical signs in this area. Disease surveillance must be introduced by taking and recording rectal temperature twice daily, having the owner/caretaker/technician contact the veterinarian immediately if findings are abnormal, and having the veterinarian perform a physical examination on suspect horses and initiating clinical and diagnostic testing. Access must be limited to essential personnel, veterinarians, technicians, and caretakers who should receive training in and follow biosecurity protocols.

Once a positive horse is identified, either state or voluntary quarantine must be imposed for 21 days after the last clinical sign (usually the temperature rise), although quarantine may last at least 28 days. Recommended exit (discharge) requirements include testing negative for EHV-1 shedding from the nasal cavity and no virus detected in blood.

It is vital to identify and implement a secondary perimeter to allow horses to be exercised and enable limited continuation of internal organized activities. This could be the entire facility, venue, or township. All animals within the secondary

perimeter are considered free of infection but at increased risk of exposure and development of disease. Enhanced surveillance is critical. Horses can travel only from outside of this facility and are under the regulation of the veterinarian in charge. Arrival and departure of any horse must be recorded. Every horse should have its temperature taken twice daily and be physically inspected for disease daily. All horses admitted must have a current health certificate and should be vaccinated at the recommendation of the veterinarian in charge. To exit, a horse must have a health certificate endorsed 'free of EHV-1' signed by the veterinarian in charge and with vaccination requirements specified by the receiving venue. Of particular note, the PCR test does not provide a diagnosis in the absence of clinical disease and should not be used as a screening tool in clinically normal nonexposed animals.

Methicillin-Resistant *Staphylococcus aureus*

Staphylococcus aureus is a common bacterium that colonizes the skin and is associated with disease in many species. MRSA is one of the most important nosocomial pathogens and is associated with increased morbidity, mortality, duration of hospitalization, and treatment costs in humans. MRSA is an important emerging pathogen of the horse. MRSA is typically resistant to all β -lactam antibiotics (penicillin and cephalosporin families) and to many other antibiotics. *Staphylococcus intermedius*, another coagulase-positive *Staphylococcus* spp., occurs in horses and can be methicillin resistant. MRSA in horses is associated with wound and surgical site infections, cellulitis, catheter site infections, pneumonia, septic arthritis, skin infections, endometritis, and peritonitis.

Humans can transmit MRSA to horses. Approximately 25% healthy children and adults can carry the organism in their nose or on the skin. Bacterial carriage causes no ill effects. However, a wound or an illness requiring hospitalization can result in an active infection. Approximately 0–5% horses carry MRSA in their nasal passages with a far lower percentage on the skin or in the intestinal tract. Carrier rates of up to 50% were found on some farms with a history of MRSA in horses. Carrier horses can transmit MRSA to other horses and may develop active infections under certain conditions. Carrier horses can transmit MRSA to humans. There is an equine-adapted strain that is uncommon in humans unless they have contact with horses. Studies of equine veterinarians have reported colonization rates of 10–14% with the equine strain of the bacterium.

Eighty percent of horses with MRSA infections survived in a multicenter study. Specific complication included prolonged hospital stays and additional surgeries, particularly if pneumonia or wound infections supervened. Adequate antibiotic options (topical and systemic) exist in most cases and culture and sensitivity will guide selection. The antimicrobial resistance pattern from many studies shows that MRSA isolates are frequently resistant to gentamicin, tetracycline, and trimethoprim-sulfa. Early detection of the pathogen and treatment are crucial to the outcome. Drugs, such as vancomycin, used in stubborn human multiple-drug-resistant pathogens, such as MRSA cases, should be avoided in horses and other animals.

Control of Methicillin-Resistant *Staphylococcus aureus*

Control involves implementation of all the elements of the aforementioned biosecurity measures. Ideally, horses infected or colonized with MRSA should be isolated. This may be difficult to achieve on farms. However, prevention of nose-to-nose contact and transmission by people and to people is of paramount importance. Hand hygiene is crucial even after wearing disposable gloves to touch a horse. Tissues can be quickly contaminated. Bleach solution (1:10) or quaternary ammonium compounds can be used to decontaminate the treatment area or stall. Stringent cleaning protocols must be effected for common equipment items, such as an ultrasound unit and treatment carts. MRSA organisms survive well in dust.

Standard operating procedures should be adopted for wounds, for example, at this teaching hospital, all surgical wound infections must have a sample obtained for culture and sensitivity before specific therapy is started. Furthermore, a patient who has received previous antibiotic therapy for a wound or infection that remains unresolved must have a sample obtained for culture and sensitivity at the initial examination.

Strangles in Horses

This is not an emerging disease of horses, although it remains an important infectious disease for susceptible individuals or populations. The causative agent is *S. equi* subspecies *equi*, (Lancefield Group C, Gram positive β -hemolytic bacterium). There is a close genetic relationship between *Streptococcus equi* subspecies *equi* and *Streptococcus zooepidemicus*, the former being a clone of the more genetically diverse *S. zooepidemicus* and isolates of these two organisms show at least 92% homology. Immunity is species specific; immunization with *S. zooepidemicus* does not protect against challenge by *S. equi equi*. *Streptococcus equi* (as subsequently described) is not a normal inhabitant of the equine upper respiratory tract. PCR may aid in confirming *S. equi* isolates but not active infection.

Epidemiology

Infection occurs primarily in horses 1–5 years of age and in geriatric and immune-compromised horses. In susceptible populations, morbidity is high (up to 100%) and mortality low (10%) with appropriate treatment.

Approximately 75% horses are immune for 5 years or longer if infected and not treated. Immunity is not lifelong. Most animals recover from the disease and eliminate *S. equi* over a 4–6-week period. Up to 10% affected animals continue to shed *S. equi* intermittently for prolonged periods after resolution of clinical signs.

The organism is transmitted by direct contact with nasal secretions or lymph node discharges from infected horses or by exposure to fomites. Highly concentrated or transient populations are at greater risk of contracting the disease. A strangles outbreak suggests a recent addition to a stable (premise) and a recovering horse may shed *S. equi* for several weeks. In a closed herd there is most likely to be exposure to an asymptomatic chronic carrier of the organism in the guttural pouch and paranasal sinuses. The organism survives for long periods in the environment depending on temperature and substrate.

Pathogenesis

Streptococcus zooepidemicus usually infects young horses through the nose or the mouth and colonizes the mucosal surface and tonsillar tissues of the nasopharynx. By contrast, *S. equi* is a poor colonizer. Rather than colonizing the epithelial surface, *S. equi* quickly invades tonsillar tissues of the oro- and nasopharynx and multiplies rapidly producing many extracellular microcolonies. Virulence factors contribute to pathogenicity. At the onset of fever, tonsillar tissues and one or more mandibular and retropharyngeal lymph nodes are heavily infiltrated by neutrophils and long chains of extracellular *S. equi* organisms. Mutant *S. equi* lacking virulence factors are not seen in draining lymph nodes.

Clinical Signs

The incubation period is 2–6 days, typically with fever (>103 F), lethargy, depression, and serous nasal discharge that becomes mucopurulent. Submandibular and retropharyngeal lymph nodes initially firm become fluctuant and rupture after 7–10 days (retropharyngeal lymph node abscesses may rupture internally into a guttural pouch).

Lymphadenopathy, if severe, may lead to dysphagia and respiratory distress, hence the term 'strangles.'

Pus in the guttural pouches may become inspissated, leading to chondroid formation enabling *S. equi* to persist for up to several years and be transmitted to naïve horses. There is a soft moist cough occasionally. The average course of the disease is 3 weeks. Atypical infection is associated with mild signs and minimal lymph node abscess development.

Pyrexia is the consequence of the acute phase inappropriate immune response generated by *S. equi* and can be monitored and detected in the absence of bacterial shedding. Once a strangles outbreak is confirmed, presumptively infected pyrexic horses may be identified and isolated before the organism is passed on to in contacts.

Diagnosis

Strangles diagnosis is based on clinical signs and identification (culture) or detection (PCR) of *S. equi* from a lymph node, nasopharyngeal swab, or lavage fluid from a guttural pouch. Chronic asymptomatic carriers pose the greatest difficulty in identification. Culture of lavage samples collected endoscopically is considered the gold standard for carrier detection. Serology is becoming widely used. The enzyme-linked immunosorbent assay (ELISA) for SeM protein is useful to detect recent but not current infection, assess the need for vaccination, identify horses predisposed to purpura hemorrhagica (PH), and to diagnose *S. equi*-associated PH and metastatic abscessation.

Treatment

Treatment is contentious and is a function of the stage of the disease. Penicillin is the drug of choice, although the organism is sensitive to many antimicrobials. Treatment plans have been developed, for example, (1) Horse exposed: penicillin helps to prevent seeding of the pharyngeal lymph nodes. It should be

continued for as long as the horse is exposed to the organism. Once stopped, there is a risk of the horse developing strangles. (2) Horse exhibiting signs without abscessation: penicillin may arrest disease progression. The horse should be isolated. (3) Horse exhibiting signs of lymph node abscessation: penicillin slows progression of lymph node abscessation; thus, the abscess should be hot packed, lanced, and flushed. The horse should be treated in isolation, and (4) horse systemically ill or developed complications: supportive care and penicillin should be provided.

Complications

The major complications are:

1. 'Bastard strangles,' in which metastatic (internal) abscesses occur in the mesentery or parenchymatous organs and even in the brain promulgated by inadequate antimicrobial therapy, although abscesses can occur naturally at those sites following infection. Clinical signs include intermittent colic, periodic pyrexia, anorexia, depression, weight loss, and neurological dysfunction.
2. PH – an aseptic vasculitis following reexposure to *S. equi* by natural infection or vaccination. Affected animals have higher IgA titers to SeM and to nonspecific proteins in the *S. equi* culture supernatant and immune complexes of IgA and M-like proteins. There is an association with the attenuated live intranasal *S. equi* vaccine. Clinical signs include a mild transient reaction to a severe and fatal form, with pitting edema of limbs, trunk, and head; petechiation and ecchymoses of mucosae; and colic. Vasculitis may lead to skin sloughing and infarcts of skeletal muscle. Death can result from pneumonia, cardiac arrhythmias, renal failure, or gastrointestinal disorders.

Other complications of strangles include guttural pouch empyema and chondroid formation; septicemia, development of infectious arthritis, pneumonia, and encephalitis; retropharyngeal abscesses; laryngeal hemiplegia; tracheal compression following abscesses in the cranial mediastinal lymph nodes; endocarditis or myocarditis following abscess formation; suppurative necrotic bronchopneumonia; and two types of myopathies (myositis) – one a vasculitis with infarction of skeletal muscle and pulmonary and gastrointestinal tissues characterized by unrelenting pain; the second affecting quarter horses showing malaise, significant muscle atrophy, and chronic active rhabdomyolysis.

Control During an Outbreak

Control follows the detailed biosecurity measures that are integral to the sensible management of an infectious disease. Specifically, prevent the spread of infection to horses on other premises and to new arrivals. Ideally, all movement of horses on and off the premises must be stopped and new horses should be isolated for 3 weeks. Identify symptomatic and asymptomatic carriers by sampling nasopharyngeal or guttural pouch regions at weekly intervals and test for *S. equi* by culture and PCR. Infectious horses should be isolated from those screened negative for *S. equi*. Rectal temperature should be

taken twice daily. The isolation area should be cordoned off and staff should be dedicated to this area with protective clothing and footwear. The area and contents should be disinfected. After clinical signs disappear, perform at least three consecutive nasopharyngeal swabs or lavage for *S. equi* at approximately weekly intervals and test by culture and PCR to detect carriers.

Prevention

Less immunity is provided following vaccination than that following recovery from natural disease.

The intramuscular killed (*S. equi* extract) vaccine with an adjuvant is used more widely than the intranasal attenuated *S. equi* live culture, which should not be administered with parenteral injections or at the time of invasive procedures. No current vaccine guarantees prevention of strangles in vaccinated horses.

Vaccination of infected animals during an outbreak may be associated with PH, and vaccination during an outbreak is controversial.

The 2005 American College of Veterinary Internal Medicine Consensus Statement recommends measuring SeM-specific antibody titers by ELISA before vaccination and to not vaccinate if titers $\geq 1:3200$. Horses previously given the intranasal vaccine, nonthoroughbreds and nonwarmbloods, are significantly more likely to have higher titers ($> 1:1600$).

Generation of an immune response to multiple protective epitopes may be required to achieve the greatest protection. Development of a strangles vaccine (ideally with differential diagnostic potential) benefitting from an improved immunogenicity and safety profile is a priority to achieve widespread protection against strangles.

References

- Ainsworth, D.M., Hackett, R.P., 2004. *Streptococcus equi* infections (strangles). In: Reed, S.M., Bayly, W.M., Sellon, D.C. (Eds.), *Equine Internal Medicine*, second ed. St. Louis, MO: Saunders, pp. 308–312.
- Anderson, M.E.C., Lefebvre, S.L., Rankin, S.C., et al., 2009. Retrospective multicentre study of methicillin-resistant *Staphylococcus aureus* infections in 115 horses. *Equine Veterinary Journal* 41, 401–405.
- Axon, J.E., Carrick, J.B., Barton, M.D., et al., 2011. Methicillin-resistant *Staphylococcus aureus* in a population of horses in Australia. *Australian Veterinary Journal* 89, 221–225.
- Boyle, A.G., 2011. *Streptococcus equi* subspecies *equi* infection (strangles) in horses. *Compendium Continuing Education for Veterinarians* 33, E1–E8.
- Dunowska, M., Morley, P.S., Traub-Dargatz, J.L., Van Metre, D.C., 2007. Biosecurity. In: Sellon, D.C., Long, M.T. (Eds.), *Equine Infectious Diseases*. St. Louis, MO: Elsevier, pp. 528–539.
- Harrington, D.J., Sutcliffe, I.C., Chanter, N., 2002. The molecular basis of *Streptococcus equi* infection and disease. *Microbes and Infection* 4, 501–510.
- Jordan, D., Simon, J., Fury, S., et al., 2011. Carriage of methicillin-resistant *Staphylococcus aureus* by veterinarians in Australia. *Australian Veterinary Journal* 89, 152–159.
- National Animal Health Monitoring System (NAHMS) USDA-APHIS; VS, 1998. Part 1: Baseline Reference of 1998 Equine Health and Management, 29.
- Sweeney, C.R., Timoney, J.F., Newton, J.R., et al., 2005. *Streptococcus equi* infections in horses: Guidelines for treatment, control and prevention of strangles. *Journal of Veterinary Internal Medicine* 19, 123–134.
- Timoney, J.F., Kumar, P., 2008. Early pathogenesis of equine *Streptococcus equi* infection (strangles). *Equine Veterinary Journal* 40, 637–642.

- Waller, A.S., Jolley, K.A., 2007. Getting a grip on strangles: Recent progress towards improved diagnostics and vaccines. *Veterinary Journal* 173, 492–501.
- Waller, A.S., Paillot, R., Timoney, J.F., 2011. *Streptococcus equi*: A pathogen restricted to one host. *Journal of Medical Microbiology* 60, 1231–1240.
- Weese, J.S., 2007. Staphylococcal infections. In: Sellon, D.C., Long, M.T. (Eds.), *Equine Infectious Diseases*. St. Louis, MO: Elsevier, pp. 257–263.

Relevant Websites

www.cahfs.ucdavis.edu

California Animal Health and Food Safety Laboratory.

<http://www.ca.uky.edu/gluck/BiblioEHV1.asp>

Gluck Equine Research Center, Lexington, KY — OIE reference laboratory for EHV-1.

www.vetmed.ucdavis.edu/vme/taqmanservice/diagnostics.html
Real-Time Research and Diagnostic Core Laboratory.

www.vetmed.ucdavis.edu/ceh/ehv1_general.cfm

UC Davis Center for Equine Health.

http://www.vetmed.ufl.edu/extension/documents/MRSAand_horses.pdf
University of Florida MRSA and horses.

www.aphis.usda.gov/animal_health/animal_dis_spec/horses/
USDA-APHIS Center for Epidemiology and Animal Health.

www.aphis.usda.gov/animal_health/animal_dis_spec/horses/
USDA-APHIS Veterinary Services.