

This article was downloaded by: [North Carolina State University]

On: 10 August 2009

Access details: Access Details: [subscription number 907216899]

Publisher Taylor & Francis

Informa Ltd Registered in England and Wales Registered Number: 1072954 Registered office: Mortimer House, 37-41 Mortimer Street, London W1T 3JH, UK



Journal of Agromedicine

Publication details, including instructions for authors and subscription information:

<http://www.informaworld.com/smpp/title~content=t792303961>

That Horse Bit Me: Zoonotic Infections of Equines to Consider after Exposure Through the Bite or the Oral/Nasal Secretions

Ricky Langley ^a; Thomas Morris ^a

^a North Carolina Department of Health and Human Services, Occupational and Environmental Epidemiology Branch, Raleigh, North Carolina, USA

Online Publication Date: 01 July 2009

To cite this Article Langley, Ricky and Morris, Thomas(2009)'That Horse Bit Me: Zoonotic Infections of Equines to Consider after Exposure Through the Bite or the Oral/Nasal Secretions',Journal of Agromedicine,14:3,370 — 381

To link to this Article: DOI: 10.1080/10599240903058087

URL: <http://dx.doi.org/10.1080/10599240903058087>

PLEASE SCROLL DOWN FOR ARTICLE

Full terms and conditions of use: <http://www.informaworld.com/terms-and-conditions-of-access.pdf>

This article may be used for research, teaching and private study purposes. Any substantial or systematic reproduction, re-distribution, re-selling, loan or sub-licensing, systematic supply or distribution in any form to anyone is expressly forbidden.

The publisher does not give any warranty express or implied or make any representation that the contents will be complete or accurate or up to date. The accuracy of any instructions, formulae and drug doses should be independently verified with primary sources. The publisher shall not be liable for any loss, actions, claims, proceedings, demand or costs or damages whatsoever or howsoever caused arising directly or indirectly in connection with or arising out of the use of this material.

REVIEW

That Horse Bit Me: Zoonotic Infections of Equines to Consider after Exposure Through the Bite or the Oral/Nasal Secretions

Ricky Langley, MD, MPH
Thomas Morris, MD, MPH

ABSTRACT. Millions of individuals are in contact with horses through occupational or recreational activities. Injuries from horses are responsible for over 100,000 emergency room visits each year in the United States. Although various types of traumatic injuries related to direct contact with horses are well described, roughly 3% to 4.5% of all reported injuries are due to bites by equines. The immediate injuries are commonly either blunt or penetrating trauma to local tissue; however, the bite exposure may also transmit a microbial agent of equine origin that can lead to a zoonotic infection. In almost all jurisdictions in the United States, animal bites are considered public health events and should be reported to the local health departments. Many animals can harbor many unusual zoonotic pathogens that both the individual health provider and public health officials much consider as they can adversely impact both the patient and the community health. This review focuses on those zoonoses that have been reported in the literature, including those that may in theory be transmitted from equine to human by direct inoculation or exposure to oral/nasal secretions from horses and other equine species.

KEYWORDS. Equine, horse, Zoonoses, infection, bite, secretions

CASE REPORT

In October, 2007, the North Carolina State Laboratory for Public Health (NCSLPH) reported to the NC Division of Public Health's Veterinary Public Health (VPH) program that a horse

specimen submitted by a large animal veterinarian in a central NC county had tested positive for rabies virus. The local health department and the NC Department of Agriculture and Consumer Services (NCDA) Veterinary Division were notified to initiate an investigation.

Ricky Langley and Thomas Morris are affiliated with the North Carolina Department of Health and Human Services, Occupational and Environmental Epidemiology Branch, Raleigh, North Carolina, USA.

The authors would like to thank Dr. Kristi Pierce with Southridge Veterinary Hospital in Kannapolis, NC, for contributing the case report.

Address correspondence to: Ricky Langley, MD, MPH, North Carolina Division of Public Health, 1912 Mail Service Center, Raleigh, NC 27699-1923, USA (E-mail: rick.langley@ncmail.net).

Case History

In mid-October, 2007, a large animal veterinarian was called to see a 6-year-old Rocky Mountain horse gelding because of signs of colic, depression, and anorexia following the normal morning feeding. Its last vaccination for rabies was reported to have been in 2005; however, this could not be confirmed. On day 1 the horse was bright, alert, and responsive (BAR), with normal vital signs and a capillary refill time (CRT) of approximately 3 seconds. Physical exam showed decreased bowel sounds in all four quadrants; rectal exam revealed a moderate sized firm impaction in the dorsal colon. The horse was given 200 mg of xylazine IV (intravenous) without much effect. It was started on IV fluid therapy and water and mineral oil were administered via nasogastric tube (NGT). Additionally, 600 mg of flunixin meglumine was given IV. On day 2 the owner noted that the horse was acting frantic, and now biting at his sides and both front fetlocks. Administration of IV flunixin meglumine calmed the horse. The veterinarian's exam noted the horse was still BAR, and that the heart rate (HR), respiratory rate (RR), and temperature were within normal limits. Bowel sounds were still decreased in all four quadrants. The new finding was that the horse was very sensitive to palpation of his abdomen; however, there was no impaction detected on rectal exam. Minimal feces without mineral oil was found. Again, the horse was given IV sedation with minimal effect, a NGT was placed, and additional sedation given. Because of several small fire ant mounds noted inside the stall, it was thought the horse's abnormal signs of biting his fetlocks and sides were potentially due to insect bites. A repeat dosing of water and mineral oil were given, plus 100 mg prednisolone. The owner was instructed to continue IV fluid therapy. The owner later reported that the horse had responded well to treatment. On day 3 the horse showed clinical signs of improvement, and a small pile of oil-covered feces was noted in the stall. The veterinarian's findings included a normal HR, RR, and temperature. Prior to the rectal exam it was again sedated with detomidine and butorphenol IV, with little effect. Despite repeated

dosing sedation was still considered suboptimal. The rectal exam revealed a small amount of oil-covered feces. This time, the horse was very resistant to NGT placement, and no reflux was obtained; it was given water and mineral oil as before, and the owner was instructed to continue IV fluids and start to graze the horse for 15 to 20 minutes every 2 to 3 hours. On day 4 the horse exhibited aggressive behavior and was charging the owner and other horses. Approaching the horse would elicit a biting response. Additionally, the horse would throw itself on the ground and begin to self mutilate, bite at his sides and front fetlocks. When the veterinarian arrived to recheck, the horse's behavior created an unsafe situation to conduct the physical exam. On observation, in addition to its aggressive and self-mutilating behavior, the horse was also vocalizing loudly, kicking uncontrollably, and biting the stall boards. Its head had to be tied down for IV sedation with detomidine and butorphenol, and the medications had to be repeated in 20-minute intervals to keep the horse calm. Though the horse's initial presentation was consistent with colic, the assessment of these new clinical signs suggested strongly of a neurological component, and there was consideration that this abnormal behavior was consistent with the furious form of rabies. The owner stated that there had been no known bite wounds found on the horse nor any rabid animals seen in the area. The horse was euthanized, and specimens were sent to the NC SLPH for rabies testing. Results were positive.

The health department's investigation for human exposures identified the two veterinarians who cared for the horse, the owner and a family member. No bites were reported but each had extensive and repeated close contact with the horse and its secretions. After consultation with VPH, each were offered and received rabies postexposure prophylaxis per the Centers for Disease Control and Prevention (CDC) guidelines.⁶⁶ The NCDA's veterinary medical officer assessed the exposure likelihood of the other horses and did not put them in quarantine. There have been no further incidents of rabies on the farm since.

INTRODUCTION

Horses, ponies, mules, donkeys, burros, and zebras are equine mammals with which most people are familiar. The use of equines has been a traditional source of labor on farms to pull equipment for activities such as plowing, and as the primary means of transportation for hauling people and materials in wagons. Horses are also used for controlling or "rounding up" animal herds such as cattle. They are involved in recreational activities such as rodeos, horse races, and equestrian competitions. Although mechanization, especially tractors and trucks, has largely displaced these beasts of burden on the farms and roads of the United States, they are still used in most other regions of the world in activities generally associated with agriculture. The United States Department of Agriculture periodic census of agriculture estimated in 2002 there were 3,644,000 horses and ponies and an estimated 105,300 mules/burros/donkeys on farms and ranches in the United States.¹

The American Horse Council Foundation estimated 9.2 million horses in the United States (US) in 2005² and that 4.6 million people are involved in the industry as owners, service providers, employees and volunteers. It is estimated that two million persons own a horse in the United States.²

Horses can on occasion show aggressive behavior, and because of their large size (a typical full-grown horse may weigh as much as 2000 pounds), there is the potential for serious injury or death when handling these animals.³⁻⁵ Serious injuries to humans may occur from a variety of actions by or interactions with an equine. Injuries previously reported include being bitten, kicked, trampled, thrown off or falling from atop of an equine. Other types of events have occurred from being dragged, or crushed when an equine has either fallen, rolled upon, or pinned against another object. Other injuries, and death, can occur due to collisions with inanimate objects such as motor vehicles, hanging signs, or tree limbs. Drowning incidents of horse riders have been reported when crossing rivers. A recent study estimated that 102,900 people are treated yearly in emergency rooms in the US due to nonfatal horse related injuries.⁶

One study covering 27 states from 1976 to 1987 found that approximately 17 people per year died from horse-related injuries, and a higher incidence of nonfatal events that resulted in serious traumatic brain injuries.^{7,8}

Although considered uncommon, the equine may occasionally inflict a bite on humans. Of all reported injuries involving horses, approximately 3% to 4.5% are related to bites.⁹⁻¹¹ Because an equine can exert a great deal of force in closing its jaws, the severity of injuries may range from mild superficial pressure trauma, cutaneous breaks of the skin, deep lacerations with loss of tissue, to amputations of digits and even the nose.^{5,12} Though anecdotal, anaphylaxis has been reported subsequent to an equine bite.¹³ Though the acute trauma is readily apparent from a biting event, there is also the risk of exposure to various microbes in the oral secretions of equines. Transmission of such agents with zoonotic potential can also occur from the nonbite exposures to the oral and respiratory secretions. In 2007, the North Carolina Division of Public Health was notified of four horses with confirmed rabies, and evaluated several individuals who were exposed to the oral secretions. A review of the literature was initiated to identify which zoonoses were known to be associated with direct contact with horses and other equine species by either bite or exposure to oral or nasal secretions.

METHODS

A query was made of PubMed¹⁴ using the following keyword terms: horse, pony, donkey, mule, burro, equine, and also bite, injury, or infection. All articles or abstracts in English identified and retrieved were also examined for additional sources in their bibliography.

RESULTS

Normal Flora of Nose and Mouth of Horses

The mouth and nose of the normal horse harbor a large variety of microbes. Most are

harmless saprophytes.¹⁵ In a study of 12 normal horses, 270 bacterial isolates were obtained from the pharyngeal tonsillar surface and 98 obligatory anaerobic bacteria were characterized.¹⁶ Rarely, some of these microorganisms may lead to infections of the mouth or respiratory tract in horses. These organisms may be transmitted to other animals or even humans causing infections that may be serious or even fatal (Table 1).

TABLE 1. Microbial Agents Reported to Cause Infections in Humans from Equine Bites or Exposure to Equine Oral/Nasal Secretions

Organism	Equine	Reference
<i>Streptococcus anginosus</i> , <i>Streptococcus mutans</i>	Horse	Marrie, 1979 [44]
<i>Actinobacillus lignieresii</i> , <i>E. coli</i>	Horse	Dibb, 1981 [18]
<i>Actinobacillus suis</i>	Horse	Ruddy, 1986 [20]
<i>Yersinia</i> sp.	Horse	Raisanen, 1989 [45]
<i>A. lignieresii</i> , <i>Actinobacillus equuli</i> -like bacterium, <i>Streptococcus</i> spp., <i>Staphylococcus aureus</i> , <i>Neisseria</i> spp.	Horse	Peel, 1991 [17]
<i>Pasteurella multocida</i> , <i>S. aureus</i> , <i>E. coli</i> , <i>A. suis</i> , <i>Prevotella melaninogenica</i>	Horse	Peel, 1991 [17]
<i>A. lignieresii</i>	Horse	Benaoudia, 1994 [19]
<i>Staphylococcus hyicus</i> subsp. <i>hyicus</i>	Donkey	Osterlund, 1997 [32]
<i>Streptococcus equi</i> subsp. <i>zooepidemicus</i>	Horse	Downar, 2001 [41]
<i>Pasteurella caballi</i>	Horse	Escande, 1997 [25]
<i>Streptococcus</i> group B, <i>Pseudomonas aeruginosa</i>	Zebra	Toovey, 2004 [43]
<i>Burkholderia mallei</i>	Horse/donkey/ mule	Bender, 2004 [29]
<i>Listeria monocytogenes</i>	Horse	Bender, 2004 [29]
<i>Rhodococcus equi</i>	Horse	Bender, 2004 [29]
Hendra virus	Horse	Hanna, 2006 [46]
Vesicular stomatitis virus	Horse	Letchworth, 1994 [51]

Bacteria Associated with Human Infections

Actinobacillus

There have been several cases in the literature of infection due to *Actinobacillus* spp. arising after a bite of a horse.¹⁷⁻²¹ *Actinobacillus* spp. are gram-negative coccobacilli that are part of the normal oral flora of horses, rabbits, sheep, cattle, and pigs. At least 11 species are known. In infections associated with horse bites, the majority of cases present with purulent, foul smelling pus from the area of the bite. *Actinobacillus lignieresii* is the most frequent species of *Actinobacillus* spp. found in wounds associated with horse bites. *A. lignieresii* is an opportunistic pathogen in animals causing chronic granulomatous lesions of the tongue in cattle and skin and mammary lesions in sheep.¹⁷

Peel et al. report a case of a person bitten on the hand by a horse.¹⁷ The wound predominantly grew *A. lignieresii* and an *Actinobacillus equuli*-like bacterium along with a light mixed population of *Staphylococcus aureus*, *Neisseria* spp., and *Streptococcus* spp. Other *Actinobacillus* spp. have rarely been reported to cause infection.²⁰ *Actinobacillus suis* were isolated from a finger wound after a horse bite. The culture grew viridans *Streptococci* and a gram-negative bacterium identified as *A. suis*. Ruddy et al. note that the CDC has identified *A. suis* from a donkey, a zebra and hamster bite.²⁰ In a second case reported by Peel et al., a man bitten by a horse presented with a compound fracture of the left radius and ulna and a macerated wound on the arm associated with muscle loss.¹⁷ Over the course of three months, the patient underwent several débridements and grafting procedures but had repeated infections with purulent discharge from which cultures grew *S. aureus*, *Prevotella melaninogenica*, *Escherichia coli*, and *Pasteurella multocida*. The patient underwent a variety of antibiotic treatments. A bone biopsy specimen from the injured arm was then obtained, and *A. suis* were isolated along with *E. coli*. At this point antibiotic therapy was changed. A bone graft and flap operation was performed one month later. The procedure was uncomplicated by bacterial infection and cultures of subsequent specimens yielded no significant growth.

Because *Actinobacillus* and *Pasteurella* spp. are closely related organisms, if extensive biochemical or molecular testing is not done, the *Actinobacillus* spp. can often be misidentified as a *Pasteurella* spp. Dibb et al. initially identified an infection that occurred after a horse bite as due to *Pasteurella pneumotropica*; however, a confirmation testing performed at the CDC identified it as *A. lignieresii*.²²

Pasteurella

Pasteurella multocida is one of the most frequent organisms isolated from wounds secondary to cat or dog bites. These cases may be severe and occasionally cause death.²³ *P. multocida* has been isolated from a horse-bite wound;¹⁷ in the same case report, *A. suis* was cultured from a bone biopsy later.

Two cases of horse bites causing a wound infection with *Pasteurella caballi* have been reported. It was isolated from a horse-bite wound in a veterinary surgeon.²⁴ Later, Escande et al. reported a case of infection in a 56-year-old man bitten by a horse from which it was isolated.²⁵ *P. caballi* has been associated with endocarditis and lower respiratory infections in horses.²⁶

Burkholderia

Infection with *Burkholderia mallei* (a small gram-negative bacillus, formerly known as *Pseudomonas mallei*) can cause a subcutaneous infection known as "farcy" or can disseminate to a condition known as Glanders.²⁷ It is primarily a disease affecting horses, donkeys and mules. Glanders has not been reported in the United States since the 1940s; however, it is endemic in Africa, Asia, the Middle East, and Central and South America. Secretions from the horse's respiratory tract and skin lesions are highly infectious. The incubation period can be from days to months, and the disease may also recrudescence years later. In humans, Glanders can produce four types of disease: localized form, pulmonary form, septicemia, and chronic form. If the exposure occurred by inhalation, a febrile pneumonia usually develops with necrosis of the tracheobronchial tree. If *B. mallei* is exposed onto the skin, then pustular lesions characteristically appear. Both routes could

lead to multiple abscesses, sepsis, and death. Glanders has low contagious potential, but because of the efficacy of aerosolized dissemination and the lethal nature of the disease, *B. mallei* was studied as biological weapon for warfare²⁸ and is a category B agent on the CDC list of biological agents that could potentially be used in terror events.

Rhodococcus

Rhodococcus equi, a gram-positive, weakly acid-fast coccobacillus, initially isolated from horses, is becoming increasingly recognized as an important pathogen for immunosuppressed human hosts.²⁹ *Rhodococcus equi* causes severe pyogranulomatous pneumonia in foals and pulmonary infections in immunocompromised humans. Cases of meningitis in humans have been noted.³⁰ Previously, many cases may have been missed because the organism resembles oropharyngeal commensal diphtheroids. In many cases, contact with horses can be recalled from patients. The mode of spread from horses to humans is not understood but *Rhodococcus* has been isolated from tracheobronchial secretions in foals.³¹

Staphylococcus

Osterlund and Nordland report a case of a donkey bite to the thumb of a woman.³² The patient sought medical help five days later, and a culture of the wound grew *Staphylococcus hyicus* subsp. *hyicus*. The patient was given cephadroxil and the infection resolved 12 days later when seen at follow-up. *S. hyicus* is associated with skin infections and mastitis in horses, cattle, sheep, goats, and pigs.

Staphylococcus aureus is a well-described bacterial pathogen that has affected humans and various mammals. Animals known to either harbor or be infected by *S. aureus* include cattle, dogs,^{33,34} cats,³⁵ and swine.³⁶ The first report of methicillin-resistant *S. aureus* (MRSA) in an equine was 1997,³⁴ and the first series of MRSA infections in an equine hospital was 1999.³⁷ Unlike almost all other equine-associated zoonoses, the evidence is strong that transmission of MRSA is bidirectional: colonization and exposure may occur from horse to human,³⁸

and from human to horses³⁷; this epidemiologic phenomenon is not limited to equines only.³⁹ The earlier reports of MRSA involving equines were found to have a phenotypic profile similar to MRSA strains detected in hospital and health care settings. More recently, however, community-acquired MRSA (Ca-MRSA) has been described in the veterinary literature, particularly as a nosocomial pathogen in equine veterinary hospitals. Ca-MRSA is notable for two features: the first is that clinical infections of Ca-MRSA in humans are usually skin and soft tissue, and most Ca-MRSA strains have the Pantin-Valentine Leukocidin (PVL) gene, which may be a virulence factor for infection (this is a point of controversy in the scientific literature and beyond the scope of this paper). PVL has been detected in strains collected in several species; however, PVL is almost never present in Ca-MRSA isolated from horses. MRSA has been isolated from the nares of horses and in areas in equine hospitals associated with horses' nostrils such as twitches, muzzles, stall walls, feed bins, and water buckets.⁴⁰

Streptococcus

Streptococcus equi subsp. *zooepidemicus* (Lancefield group C) is a normal commensal of the skin and upper respiratory mucosa of horses. It also causes wound and respiratory infections of young horses and foals, including purulent nasal discharge and abscesses of submandibular nodes in some cases (strangles). There are several reports in the literature of humans developing serious infection from this organism. Downar et al. report a case of a 49-year-old female who cared for eight horses and one donkey.⁴¹ All the animals had been well except for a new colt with oral and nasal secretions. Two weeks prior to the woman's admission to the hospital, she had been kicked in the face with his knee. Two days later, she developed a sore throat, myalgia, and neck swelling. She later developed photophobia, and decreased level of consciousness and meningitis was diagnosed. *S. equi* subsp. *zooepidemicus* was cultured from her blood and cerebrospinal fluid. On further investigation of the case, the patient's husband and two

children as well as the horses and donkey were tested by oropharyngeal swab for the bacteria. The family members tested negative, but two of the eight horses (including the colt) and the donkey tested positive for the bacteria.

Another case of group C streptococcal meningitis due to *S. equi* subsp. *zooepidemicus* was reported to be due to a pet horse.⁴² The organism was isolated from the patient's cerebrospinal fluid (CSF) and the horse's pharynx.

An unprovoked attack by a zebra on a South African female resulted in a bite of the lower leg with substantial loss of tissue.⁴³ In this case report, a *Streptococcus* group B bacterium and *Pseudomonas aeruginosa* were cultured; however, these pathogens were detected approximately one month after the bite event, with a complicated course involving an initial débridement and later reexploration, débridement, and drainage with skin grafting of the wound site. It wasn't made clear whether these two organisms were the result of the bite or acquired during the post-operative stage.

Marrie et al. report a case of a horse bite to the forearm.⁴⁴ After the initial debridement, complications ensued, and *Streptococcus angiosus* and *Streptococcus mutans* were then isolated from the wound.

Yersinia

There has been a case of *Yersinia* spp. infection reported in a human resulting from a horse bite.⁴⁵

Viral Pathogens and Other Microbes of Potential Concern

There are many agents, primarily viral, that are of potential or theoretical concern to humans from exposure to equine secretions (Table 2). Fortunately, few or no cases in humans have been reported, but with increasing numbers of immunocompromised individuals and newly emerging infectious agents being discovered, humans may be exposed to these agents and may be at risk to develop illness. Although the various routes of transmission of the agent from equines to humans are not known, for many, spread via oral/nasal secretions is suspected.

TABLE 2. Equine Zoonotic Pathogens Acquired by Bites or Exposure to Oral/Nasal Secretions

Bite infections	Nonbite infections	Potential agents of concern
<i>Actinobacillus lignieresii</i>	<i>Burkholderia mallei</i>	Borna virus [†]
<i>Actinobacillus suis</i>	<i>Rhodococcus equi</i>	Nipah virus
<i>Pasteurella caballi</i>	<i>Streptococcus equi</i>	Rabies virus [‡]
<i>Pasteurella multocida</i>	<i>Staphylococcus aureus</i> [§]	<i>Mycobacterium bovis</i> [‡]
<i>Pasteurella pneumotropica</i>	Equine influenza virus	Equine Foamy virus
<i>Staphylococcus aureus</i> [§]	Hendra virus	
<i>Staphylococcus hyicus</i>	Vesicular stomatitis virus	
Streptococcus group B		
<i>Streptococcus angiosus</i>		
<i>Streptococcus mutans</i>		
<i>Yersinia</i> spp.		

[†]Exposure route is uncertain.

[‡]No equine associated human cases yet reported or confirmed.

[§]Including methicillin-resistant *S. aureus* (MRSA).

^{||}H7N7 & H3N8 are the only viruses associated with horses.

Hendra Virus

Hendra virus is a recently described virus in the genus Henipavirus within the family Paramyxoviridae.^{46,47} It was formerly known as equine morbillivirus and first described after an outbreak of severe respiratory illness in horses, causing 14 deaths in horses, and the death of a horse trainer in 1994. Fortunately few outbreaks have been reported as it has a high mortality rate. There have been six known human cases, with fatalities reported in three of the cases. Severe encephalitis in humans may occur. The evidence indicates that transmission from horses to humans appears to be via physical contact with oral and nasal secretions from very ill dying or dead horses. The disease has not been reported in horses in the United States yet. The reservoir for the virus appears to be fruit bats (*Pteropus* spp.), commonly known as flying foxes.

Nipah Virus

This virus is a henipavirus, like Hendra virus, but is usually associated with infections in swine.⁴⁷ Infection with Nipah virus in humans during this swine outbreak was associated with an encephalitis (inflammation of the brain) characterized by fever and drowsiness and more serious central nervous system disease, such as coma, seizures, and inability to maintain breathing. Some patients have had a respiratory illness during the early part of their infections. Nipah virus infection has been associated with >40% mortality rate in humans.

In an outbreak in Bangladesh, Nipah virus was detected in a brain tissue sample from a horse and seroconversion in two horses was detected.⁴⁸ Nipah virus may possibly be transmitted via oral secretions from infected horses although no human cases associated with horses has been reported. The fruit bat is the reservoir for this virus.

Vesicular Stomatitis Virus

Vesicular stomatitis is a viral disease that primarily affects cattle, horses, and swine.^{49,50} Vesicular stomatitis virus is in the genus Vesiculovirus within the family Rhabdoviridae. It has a wide host range. In affected livestock, vesicular stomatitis causes blister-like lesions to form in the mouth and on the dental pad, tongue, lips, nostrils, hooves, and teats. These blisters swell and break, leaving raw tissue that is so painful that infected animals generally refuse to eat or drink and show signs of lameness.

The route of spread of vesicular stomatitis is not fully known; insect vectors, mechanical transmission, and movement of animals are probably responsible. Once introduced into a herd, the disease apparently moves from animal to animal by contact or exposure to saliva or fluid from ruptured lesions. In affected livestock, the incubation period for vesicular stomatitis ranges from 2 to 8 days. Often, excessive salivation is the first sign. Body temperature may rise immediately before or at the same time lesions first appear. Initially, close examination of the mouth reveals blanched and raised vesicles. In horses, these lesions generally occur on the upper surface of the tongue. When the

blisters break open, painful ulcers develop that cause horses to drool and froth from the mouth. This sign can be mistaken for biting problems, dental abnormalities, or colic. Infected animals generally go off feed and suffer mild to significant weight loss. Additionally, mild lameness may occur if lesions develop along the coronary band. Some cases of severe lameness have been reported when hooves were sloughed. The number of affected animals on a premise will vary. Five percent to 10% of horses within an infected herd typically show clinical signs. If there are no complications, such as secondary infections, affected horses may recover in as quickly as two weeks. However, in more severe cases, ulcers can take up to two months to heal and horses continue to be contagious while the lesions continue to heal. Fortunately, vesicular stomatitis usually does not cause death in the animal.

Humans can contract vesicular stomatitis when handling affected animals.⁵¹ The incidence of this disease in humans may be underreported, as it may often go undetected or be misdiagnosed. In people, vesicular stomatitis causes an acute influenza-like illness with symptoms such as fever, muscle aches, headache, and malaise. People who handle potentially infected horses should wear gloves and should not allow saliva and blister fluids to come in contact with open wounds or mucous membranes such as the eyes or mouth.

Equine Influenza Viruses

Horses can contract influenza and may possibly transfer it to other animals including humans. Two influenza-A virus subtypes have been associated with equines, H7N7, or equine-1, and H3N8, equine-2. (The equine-1 H7N7 subtype should not be mistaken for the avian influenza subtype H7N7, a highly pathogenic avian influenza strain that in 2003 during a poultry outbreak in the Netherlands infected 89 humans with one fatality of a veterinarian). H7N7 causes a more severe respiratory illness and is not as commonly isolated except in Central Asia. H3N8 has caused outbreaks in South America (due to introduction of infected horses from the United States) and in China.⁵² The outbreak in China was thought to originate

from an avian influenza virus, because it is distinctly different from the typical equine 1 virus. An outbreak of canine flu (H3N8) is thought to have originated from horses in Florida that introduced the infection to racing greyhounds. In Chile a possible human case in 1973 was described. This case of influenza and seroconversion occurred in a human associated with horses suffering a respiratory distress diagnosed as equine influenza; unfortunately the isolated virus was not typed.⁵³ In an experiment in 1965, five humans were deliberately inoculated with a H3N8 horse flu virus. The virus infected all of them, and one of them became ill.⁵⁴ It is therefore theoretically possible that equine influenza could be transmitted to humans.

Equine Foamy Virus

Foamy viruses are also called spuma- or spumaretroviruses and have been found in non-human primates, cows, cats, and horses. Zoo workers have seroconverted after working with simians infected with foamy virus. In several cases of seroconversion, the only known exposure was from a simian bite or exposure to simian saliva. Simian foamy virus is common in oral secretions of some nonhuman primates.⁵⁵ It is not known if equine foamy virus is found in equine saliva. Other potential exposures to equine foamy virus may occur from butchering or from eating horsemeat. The long-term risk is not known; however, children or the immunocompromised may be at higher risk after exposure to these retroviruses.⁵⁶

Borna Virus

Borna disease is a sporadically occurring, progressive viral polyencephalomyelitis that primarily affects horses and sheep and other animals.^{57,58} The etiological agent, Borna disease virus (BDV), is an enveloped, single-stranded RNA virus in the virus family Bornaviridae within the order Mononegavirales. The virus can induce severe clinical signs typically of viral encephalitis, with striking behavioral disturbances. After an incubation period lasting a few weeks to several months, BDV infection causes locomotor and sensory dysfunctions followed by paralysis and death in horses. However,

natural infections seem to be subclinical in most cases. There is some evidence that Borna disease (BD) virus, or a related agent, can infect man and may induce mental disorders. BD virus-specific antibodies could be demonstrated in 4% to 7% of sera (depending on origin) from more than 5000 psychiatric or neurological patients from Germany, United States, and Japan.⁵⁹ It is not known for certain if or how Borna virus can be transmitted to humans. A study in Japan found the seroprevalences (2.6% to 14.8%) of BDV were significantly higher in the blood donors from four regions where most horse farms are concentrated, compared with only 1% in the blood donors from Sapporo, the largest city in Hokkaido.⁶⁰

In a serological study in Finland, two veterinarians had antibodies to Borna virus detected.⁶¹ A study by Thomas et al. found those living or working on livestock farms had higher seroprevalence (2.6%) than those on mixed (2.3%) or arable (1.6%) farms; however, this was not statistically significant, and exposure to horses, sheep, and cats did not increase risk of seropositivity. Seropositives were no more likely to report symptoms of psychiatric morbidity.⁶² More research is needed to see if Borna virus can be transmitted from animals to humans and if Borna virus may be an etiologic agent for neuropsychiatric disease in humans.

Rabies

Rabies is a disease of warm-blooded mammals, and the one disease that most people will immediately associate with animal bites, regardless of the rarity of human cases in the United States. Rabies virus (genus *Lyssavirus*) is a neurotrophic RNA virus in the family *Rhabdoviridae* that can lead to an acute progressive encephalomyelitis. The condition is considered universally fatal; less than 10 people diagnosed with clinical rabies have survived the disease. Notably, there has not been a documented case of horse to human rabies reported in the US or in the medical literature. The domestic dog has traditionally been the source of exposure leading to human rabies cases; however, other carnivorous and omnivorous animals, including cats, are known to transmit rabies to humans.

Starting in the 1940s and 1950s, mandatory vaccination in most states led to the virtual elimination of companion animals (dogs and cats) being a primary reservoir for rabies and greatly reducing the incidence for rabies in domesticated animals—one of the more notable triumphs of human and veterinary public health.⁶³ Since the 1960s, human rabies cases from exposures inside the United States are almost always from bat-variant rabies virus. Presently the main reservoir and leading vectors of rabies are certain wild animal species (raccoon, skunk, and fox are the main terrestrial), and the bat species. Wild carnivores are the most likely animals to transmit the rabies virus to horses, although bats can as well. Although some species are more resistant to infection after exposure, the equine species is considered very susceptible to developing rabies disease. It is estimated that between 40 and 50 horses per year have died from rabies in the United States.⁶⁴

The mode of exposure and transmission of rabies virus is overwhelmingly by an animal bite that breaks the skin; however, not all bites from a proven rabid animal will lead to clinical rabies. The virus is usually present in saliva of infected mammals, and transmission from one mammal to another is almost always by a bite by the rabid animal onto a susceptible species. There are no data available whether an equine will shed rabies virus in their saliva prior to developing clinical rabies. Although there is no defined quarantine period for equines, unlike for dogs and cats, a 14-day observation period has been recommended by the Kentucky state public health veterinarian for over a decade without incident (M. Auslander, personal communication, 2008). This advisory has not been scientifically validated. Humans may potentially be exposed when treating an ill horse from their oral secretions.

In horses, once clinical symptoms begin, the mean survival time was 4 to 5 days.⁶⁵ There are United States Department of Agriculture (USDA)-approved equine vaccines for rabies prevention available; however, at this time there is no approved protocol for postexposure prophylaxis (PEP) for any animal. There is at present no treatment for any animal that develops clinical rabies. Current recommendations are that humans exposed to either saliva or nervous tissue of a

rabid equine animal should undergo rabies post-exposure prophylaxis.⁶⁶

Mycobacterium

Horses can become infected with *Mycobacterium bovis*. *M bovis* is usually associated with cattle but other mammals may become infected with this organism. In horses, lesions occur in the pharyngeal area, mesentery, lungs, liver, and spleen. Theoretically horses could transmit this to humans if they had oral lesions or a respiratory infection.⁶⁷ Symptoms and x-ray appearance in humans are similar to cases due to *M. tuberculosis*. There are no reported cases of *M. bovis* in humans related to exposure by equines.

Prevention of Bites/Exposure to Oral and Nasal Secretions

Infections in humans can be prevented by practicing good personal hygiene, especially hand washing with soap and water after any contact with horses. The use of personal protective equipment such as gloves and gown or lab coat should be worn when examining horses in the veterinary clinic or hospital. The use of masks and goggles should be considered if the animal is coughing or sneezing. Standard operating procedures should be developed on dealing with sick animals. Isolation protocols and stalls should be planned for to prevent transmission of pathogens from infected animals to other animals, humans, or the hospital environment.^{29,68} Weese⁴⁰ has developed a protocol to eradicate endemic MRSA colonization on horse farms.

REFERENCES

1. U.S. Department of Agriculture National Statistical Service. Available at http://www.nass.usda.gov/QuickStats/indexbysubject.jsp?Pass_group=Livestock+%26+Animals. Accessed September 5, 2008.
2. American Horse Council Foundation. National economic impact of the horse industry, 2005. Available at www.horsecouncil.org/statistics.htm. Accessed September 17, 2008.
3. Santiago V, Barcala L, Tovar JA. Horse bite injury. *Eur J Dermatol*. 1998;8:437–438.
4. Lathrop SL. Animal-caused fatalities in New Mexico, 1993–2004. *Wilderness Environ Med*. 2007;18:288–292.
5. Shipkov CD. Nasal amputation due to donkey bite: immediate and late reconstruction with a forehead flap. *Injury Extra*. 2004;35:85–90.
6. Thomas KE, Annest JL, Gilchrist J, Bixby-Hammett DM. Non-fatal horse related injuries treated in emergency departments in the United States, 2001–2003. *Br J Sports Med*. 2006;40:619–626.
7. Bixby-Hammett D, Brooks WH. Common injuries in horseback riding. *Sports Med*. 1990;9:36–47.
8. Centers for Disease Control. Alcohol use and horseback-riding-associated fatalities—North Carolina, 1979–1989. *MMWR*. 1992;41:341–342.
9. Fox SE, Ridgway EB, Slavin SA, Upton J, Lee BT. Equestrian-related injuries: Implications for treatment in plastic surgery. 2008;123:826–832.
10. Kiss K, Swatek P, Lenart I, Mayr J, Schmidt B, Pinter A, Hollwarth M. Analysis of horse-related injuries in children. *Pediatr Surg Int*. 2008;10:1165–1169.
11. Craven JA. Paediatric and adolescent horse-related injuries: does the mechanism of injury justify a trauma response? *Emerg Med Australas*. 2008;20:357–362.
12. Ludolph E, Niezold D. Traumatic upper arm amputation due to a bite by a horse. *Unfallchirurgie*. 1992;181:48–89.
13. Rolla G. Anaphylaxis after a horse bite. *Allergy*. 2005;60:1088–1089.
14. PubMed. Available at www.ncbi.nlm.nih.gov/pubmed.
15. Boyer EEH. Studies on the bacterial flora of the mouth and nose of the normal horse. *J Bacteriol*. 1919;4:61–63.
16. Bailey GD, Love DN. Oral associated bacterial infection in horses: studies on the normal anaerobic flora from the pharyngeal tonsillar surface and its association with lower respiratory tract and paraoral infections. *Vet Microbiol*. 1991;26:367–379.
17. Peel MM, Hornidge KA, Luppino M, Stacpoole AM, Weaver RE. Actinobacillus spp. and related bacteria in infected wounds of humans bitten by horses and sheep. *J Clin Microbiol*. 1991;29:2535–2538.
18. Dibb WL, Digranes A, Tonjum S. *Actinobacillus lignieresii* infection after a horse bite. *Br Med J (Clin Res Ed)*. 1981;283:583–584.
19. Benaoudia F, Escande F, Simonet M. Infection due to *Actinobacillus lignieresii* after a horse bite. *Eur J Clin Microbiol Infect Dis*. 1994;13:439–440.
20. Ruddy A, Hughes J, Bourbeau P. *Actinobacillus suis*: Finger isolate following a horse bite. *Clin Microbiol Newslett*. 1986;8:187–188.
21. Ashhurst-Smith C, Norton R, Thoreau W, Peel MM. *Actinobacillus equuli* septicemia: an unusual zoonotic infection. *J Clin Microbiol*. 1998;36:2789–2790.
22. Dibb WL, Digranes A. Characteristics of 20 human *Pasteurella* isolates from animal bite wounds. *Acta Path Microbiol Scand Sect B*. 1981;89:137–141.

23. Griego RD, Rosen T, Orengo IF, Wolf JE. Dog, cat, and human bites: a review. *J Am Acad Dermatol*. 1995;33:1019–1029.
24. Bisgaard M, Heltberg O, Frederiksen W. Isolation of *Pasteurella caballi* from an infected wound on a veterinary surgeon. *APMIS*. 1991;99:291–294.
25. Escande F, Vallee E, Aubart F. *Pasteurella caballi* infection following a horse bite. *Zentralbl Bakteriol*. 1997;285:440–444.
26. Christensen H, Hommez J, Olsen JE, Bisgaard M. *Pasteurella caballi* infection not limited to horses—a closer look at taxon 42 of Bisgaard. *Lett Appl Microbiol*. 2006; 43:424–429.
27. Currie BJ. *Burkholderia pseudomallei* and *Burkholderia mallei*: melioidosis and Glanders. In: *Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases*. 6th ed. Mandell GL, Bennett JE, and Dolin R (eds.). Philadelphia: Elsevier Churchill Livingstone; 2005:2622–2632.
28. Srinivasan A, Kraus CN, DeShazer D, Becker PM, Dick JD, Spacek L, Bartlett JG, Byrne WR, Thomas DL. Glanders in a military microbiologist. *N Engl J Med*. 2001;345:256–258.
29. Bender JB, Tsukayama DT. Horses and the risk of zoonotic infections. *Vet Clin Equine*. 2004;20:643–653.
30. DeMarais PL, Kocka FE. Rhodococcus meningitis in an immunocompetent host. *Clin Infect Dis*. 1995;20:167–169.
31. Venner M, Heyers P, Strutzberg-Minder K, Lorenz N, Verspohl J, Klug E. Detection of *Rhodococcus equi* by microbiological culture and by polymerase chain reaction in samples of tracheobronchial secretions of foals. *Berl Munch Tierarztl Wochenschr*. 2007;120:126–133.
32. Osterlund A, Nordlund E. Wound infection caused by *Staphylococcus hyicus* subspecies *hyicus* after a donkey bite. *Scand J Infect Dis*. 1997;29:95.
33. Baptiste KE, Williams K, Willams NJ, Wattret A, Clegg PD, Dawson S, Corkill JE, O'Neill T, Hart CA. Methicillin-resistant staphylococci in companion animals. *Emerg Infect Dis*. 2005;11:1942–1944.
34. Weese JS, Archambault M, Willey BM, Dick H, Hearn P, Kreiswirth BN, Said-Salim B, McGeer A, Likhosvay Y, Prescott JF, Low DE. Methicillin-resistant *Staphylococcus aureus* in horses and horse personnel, 2000–2002. *Emerg Infect Dis*. 2005;11:430–435.
35. Sing A, Tuschak C, Hörmansdorfer S. Methicillin-resistant *Staphylococcus aureus* in a family and its pet cat. *N Engl J Med*. 2008;358:1200–1201.
36. Smith TC, Male MJ, Harper AL, Kroeger JS, Tinkler GP, Moritz ED, Capuano AW, Herwaldt LA, Diekema DJ. Methicillin-resistant *Staphylococcus aureus* (MRSA) strain ST398 is present in midwestern U.S. swine and swine workers. *PLoS ONE*. 2008;4:e4258.
37. Seguin JC, Walker RD, Caron JP, Kloos WE, George CG, Hollis RJ, Jones RN, Pfaller MA. Methicillin-resistant *Staphylococcus aureus* outbreak in a veterinary teaching hospital: potential human-to-animal transmission. *J Clin Microbiol*. 1999;37:1459–1463.
38. Weese JS, Caldwell F, Willey BM, Kreiswirth BN, McGeer A, Rousseau J, Low DE. An outbreak of methicillin-resistant *Staphylococcus aureus* skin infections resulting from horse to human transmission in a veterinary hospital. *Vet Microbiol*. 2006;114:160–164.
39. Van Loo I, Huijsdens X, Tiemersma E, de Neeling A, van de Sande-Bruinsma N, Beaujean D, Voss A, Kluytmans J. Emergence of methicillin-resistant *Staphylococcus aureus* of animal origin in humans. *Emerg Infect Dis*. 2007;13:1834–1839.
40. Weese JS. Methicillin-resistant *Staphylococcus aureus* in horses and horse personnel. *Vet Clin Equine*. 2004;20:601–613.
41. Downar J, Willey BM, Sutherland JW, Matthew K, Low DE. Streptococcal meningitis resulting from contact with an infected horse. *J Clin Microbiol*. 2001;39:2358–2359.
42. Low AE, Young MR, Harding GK. Group C streptococci meningitis in an adult. Probable acquisition from a horse. *Arch Intern Med*. 1980;140:977–978.
43. Toovey S, Annandale Z, Jamieson A, Schoeman J. Zebra bite to a South African tourist. *J Travel Med*. 2004;11:122–124.
44. Marrie TJ, Bent JM, West AB, Roberts TM, Haldane EV. Extensive gas in tissues of the forearm after horse bite. *South Med J*. 1979;72:1473–1474.
45. Raisanen S, Alavaikko A. Yersinia infection from a horse bite. *Duodecim*. 1989;105:1496–1497.
46. Hanna JN, McBride WJ, Brookes DL, Shield J, Taylor CT, Smith IL, Craig SB, Smith GA. Hendra virus infection in a veterinarian. *Med J Aust*. 2006; 185:562–564.
47. Hooper PT, Williamson MM. Hendra and Nipah virus infections. *Vet Clin North Am Equine Pract*. 2000; 16:597–603.
48. Lam SK. Nipah virus—a potential agent of bioterrorism. *Antiviral Res*. 2003;57:113–119.
49. University of Nebraska-Lincoln Extension, Institute of Agriculture and Natural Resources. Vesicular Stomatitis in Horses. Available at <http://www.ianrpubs.unl.edu/epublic/pages/publicationD.jsp?publicationId=169>. Accessed September 20, 2008.
50. U.S. Department of Agriculture. Vesicular Stomatitis Questions and Answers. Available at http://www.aphis.usda.gov/lpa/pubs/fsheet_fa_notice/faq_ahvs.html. Accessed September 21, 2008.
51. Letchworth GJ. Vesicular stomatitis. In: *Viral Infections of Equines*. 1994. Ed Studdert M. Available at http://books.google.com/books?id=LKjfsHLvngcC&pg=PA265&lpg=PA265&dq=vesicular+stomatitis+virus+in+horses&source=web&ots=L290StLtBk&sig=ViX7AJuJtmUe-yziSfV1he5uiLo&hl=en&sa=X&oi=book_result&resnum=2&ct=result#PPP1,M1. Accessed September 20, 2008.
52. Can Avian flu infect our pets. Available at www.akvma.org/can_avian_flu_infect_our_pets.htm. Accessed September 25, 2008.

53. Berríos P. Equine influenza in Chile (1963–1992): a possible human case. *Rev Chilena Infectol.* 2005;22:47–50.
54. Presiding Officers' Science Series Note No. 1—March 2008 Equine Influenza (Horse Flu). Available at www.atse.org.au/uploads/0803note1.pdf. Accessed September 25, 2008.
55. Blewett E, Black D, Lerche N, White G, Eberle R. Simian foamy virus infections in a baboon breeding colony. *Virology.* 2000;278:183–193.
56. Bastone P, Truyen U, Lochelt M. Potential of zoonotic transmission of non-primate foamy viruses to humans. *J Vet Med B.* 2003;50:417–423.
57. Richt JA, Rott R. Borna disease virus: a mystery as an emerging zoonotic pathogen. *Vet J.* 2001;161:24–40.
58. Richt JA, Grabner A, Herzog S. Borna disease in horses. *Vet Clin North Am Equine Pract.* 2000;16:579–595.
59. Rott R, Herzog S, Bechter K, Frese K. Borna disease, a possible hazard for man? *Arch Virol.* 1991;118:143–149.
60. Takahashi H, Nakaya T, Nakamura Y, Asahi S, Onishi Y, Ikebuchi K, Takahashi TA, Katoh T, Sekiguchi S, Takazawa M, Tanaka H, Ikuta K. Higher prevalence of Borna disease virus infection in blood donors living near thoroughbred horse farms. *J Med Virol.* 1997;52:330–335.
61. Kinnunen PM, Billich C, Ek-Kommonen C, Henttonen H, Kallio RK, Niemimaa J, Palva A, Staeheli P, Vaheri A, Vapalahti O. Serological evidence for Borna disease virus infection in humans, wild rodents and other vertebrates in Finland. *J Clin Virol.* 2007;38:64–69.
62. Thomas DR, Chalmers RM, Crook B, Stagg S, Thomas HV, Lewis G, Salmon RL, Caul EO, Morgan KL, Coleman TJ, Morgan-Capner P, Sillist M, Kench SM, Meadows D, Softley P. Borna disease virus and mental health: a cross-sectional study. *Q J Med.* 2005;98:247–254.
63. Centers for Disease Control and Prevention. Compendium of Animal Rabies Prevention and Control, 2008. *MMWR Mort Morb Wkly Rep.* 2008;57(RR-2):1–9.
64. Cooperative Extension Service. Rabies in horses. University of Kentucky. ASC-125. 1990.
65. Green SL, Smith LL, Vernau W, Beacock SM. Rabies in horses: 21 cases (1970–1990). *J Am Vet Med Assoc.* 1992;200:1133–37.
66. Centers for Disease Control and Prevention. Human rabies prevention—United States, 2008 Recommendations of the Advisory Committee on Immunization Practices. *MMWR Mort Morb Wkly Rep.* 2008;57(RR-3):1–28.
67. American Public Health Association. *Control of Communicable Diseases Manual.* 19th ed. Washington, DC: American Public Health Association; 2008:639–659.
68. Weese JS. Barrier precautions, isolation protocols, and personal hygiene in veterinary hospitals. *Vet Clin Equine.* 2004;20:543–559.