**EQUINE VIRAL DISEASES**

(5 CE Hours)

**Learning objectives**

- List the kinds of viral diseases that affect equines.
- Describe an initial diagnostic overview.
- List the epidemiological background and considerations for diagnosis.
- Describe overall concepts of the body’s defense mechanisms.
- Describe how humans and animals defend themselves against infectious diseases.
- Explain how to diagnose and monitor the progress of infectious diseases.
- List the contraindications and differential diagnostic considerations.

**Introduction**

With an overall, direct and indirect, impact of $112 billion on the United States economy, the horse industry plays a major role in the economic health of the country. There are about 2 million horse owners and nearly 1.5 million paying jobs created by the industry. Altogether, there are approximately 7 million Americans involved. The horse industry contributes nearly $40 billion to the gross domestic product of the United States, with thoroughbred racing amounting for one-half of it. Texas, with almost a million horses, and California, with approximately 700,000, are the two leading states in the industry. Compared to the racehorse population of 800,000-900,000, the recreational horse population is nearly 4 million. The total horse population of the United States is 9 million.

Proper management and care for the horse is a function of the interest and funds invested by its owner. It must include an understanding of the infectious diseases threatening its health and welfare and how to protect it from such diseases.

Worldwide, the World Organization for Animal Health (OIE: Office International des Epizooties) by international agreement has decided that the following infectious equine diseases must be reported to the OIE: African horse sickness, western equine encephalomyelitis, eastern equine encephalomyelitis, equine rhinopneumonitis, equine influenza, equine infectious anemia, equine viral arteritis, dourine (Trypanosoma equiperdum), Surra (Trypanosoma evansi), equine piroplasmosis, Glanders (Burkholderia mallei), and contagious equine metritis (Taylorella equigenitalis). The equine viral diseases discussed here will concentrate on those of import in the United States (see chart below):

### **Equine Viral Diseases (United States)**

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*Streptococcus equi and rhodococcus equi are included as part of the equine respiratory complex Hendra virus as one of the new emerging diseases (Australia)*

**Japanese encephalitis virus (subtropical/tropical Asia, including Indonesia, Australia, Papua New Guinea and Pakistan)**

**Virus diseases**

The equine respiratory disease complex represents a group of the most common and most important diseases in horses. A number of viruses are involved: equine herpes virus types 1 and 4, equine influenza virus, equine viral arteritis, equine rhinitis virus, equine reovirus and equine adeno virus. In addition, there are two bacterial pathogens that are part of the equine respiratory disease complex: Streptococcus equi equi, causing strangles, and Rhodococcus equi, causing disease of the lower respiratory tract.

**Equine herpes viruses** are double-stranded DNA viruses, spherical to pleomorphic with icosahedral symmetry, 150 to 200 nm in diameter. There are 12 types of herpes viruses. Types 1, 3 and 4 are the ones producing diseases in equids.

**Equine herpes virus type 1** causes respiratory disease, neurological symptoms such as myeloencephalopathy, abortion and neonatal death. The virus is highly infectious. The rhinopneumonitis it produces starts with a mild fever, cough, nasal discharge and lymph node enlargement. It usually prefers the young horses, the weakened elderly and the immuno-compromised horses. It does cause abortions in pregnant mares.

The first indications of the disease usually show up within a week (a range of two to 10 days) after exposure. Neurological complications will develop as a result of thrombotic, ischemic inflammation of small blood vessels. They may show as lack of coordination, ataxia, and horses that seem shaky and wobbly and have difficulty standing up, with hindleg weakness and recumbency.

Equine herpes virus myeloencephalopathy is a degenerative disease of brain, medulla and spinal cord, which is irreversible. Like most herpes viruses, the virus may become latent and persist for the life of the horse. Common stress situations, such as shipping, showing, exercise and racing, injury and other diseases may trigger the dormant virus and release it to infect other horses. Outbreaks can often be linked to the aggregation of horses, staging of shows, racing and market places. The carrier animal is usually asymptomatic and cannot be identified as being infectious. The virus can be spread by aerosol, nasal secretions, aborted fetuses, placental fluids and afterbirths. Prevention from infection rests on thoughtful barn management practices and, of course, vaccination. There are both inactivated and modified live virus vaccines.

When traveling, keep contact and exposure to other animals to a minimum, and make sure that stalls and gear have been thoroughly disinfected before moving the horse in. Do not borrow watering or feed buckets, blankets, or ropes from others. Keep traffic to other stalls to a minimum because the virus can be carried by clothing, boots, gloves, riding implements, and so on.

Muzzle the horse to keep it from contact with likely infectious matter. Reduce stress and ensure quality nutrition. New horses entering the home barn should be quarantined for at least six weeks in isolated quarters before admission.

The diseased animal should be isolated so it cannot contact and infect others. It should be given rest and treated to deal with the overt symptoms. Occasionally, it may be necessary to provide medication to break up the mucus accumulation in the horse’s airways. Bacterial complications should be treated with the appropriate antibiotic chemotherapy.

When uncomplicated, the disease will be overcome within about four weeks. Virus shedding, as a rule, disappears within two weeks after the disease has run its course.

The barn that housed the infected animal should be thoroughly disinfected and kept out of bounds at least for a month.

**Equine herpes virus type 4** is genetically and antigenically distinct from equine herpes virus type 1. It produces an acute febrile infection of the upper respiratory tract in foals. Occasionally, it may cause abortion and sometimes neurological complications to the brain and spinal cord. By year 2, most horses have had an infection with herpes virus. The infection does not produce long-term immunity, although there seems to be some short-term resistance to re-infection.

The most significant economic losses of herpes virus infections lie in the not inconsiderable numbers of abortions. Obviously, the persistent presence of the virus and the stresses of pregnancy are ominous. The virus is present to high titers and dispersed throughout the body of the fetus. Fetal death and abortion occurs during the last four month of the pregnancy. There is no permanent effect on the dam. Mares that have aborted can conceive and foal normally after the abortion.

In newborn foals that appear sick or become sick shortly after birth, the outlook is poor; despite all care, they will not nurse, become feverish, lethargic, and oxygen deprived. There will be pneumonia, respiratory failure and death.

Aside from sound management practices, discussed earlier, vaccination must be the main effort for the prevention of outbreaks. Modified live virus vaccines and inactivated vaccines are
available commercially. While these vaccines will reduce the severity of the disease, reduce virus shedding and limit abortion incidence and severity, none of them seems to be able to protect against the neurological aspects of the disease. Annual re-vaccination is necessary.

In general, herd management should consider breaking the herd down to small groups, with minimum contact or travel between groups; minimizing stress situations and excessive handling and moving; reducing crowding; providing adequate and quality nutrition; and ongoing internal and external parasite control.

**Equine influenza virus** is an enveloped, spherical virus 80-120 nm in diameter. It may appear filamentous at times. It is highly infectious, causing upper respiratory tract disease. There are two subtypes: subtype A1 (H7N7) and subtype A2 (H3N8). Subtype 2 is the one most frequently occurring.

The highly contagious nature of this disease guarantees that any outbreak will spread rapidly through a susceptible population of equids. The virus travels via aerosol from the infected animal or by contaminated equipment, watering buckets, feed buckets, and grooming equipment. Within a day or two, it infects the surface cells of the entire respiratory tract, causing high fever, cough, serous nasal discharge and swelling of the regional, draining lymph nodes. Occasionally, it will produce epizootic cellulitis (swelling of the horse’s limb and trunk).

The damage to the respiratory tract epithelium predisposes the infected animal to bacterial superinfection, including bacterial pneumonia as symptomized by thick, purulent, yellowish-green nasal discharge. Without appropriate antimicrobial chemotherapy, this bacterial pneumonia will often be fatal. An infected horse won’t always show symptoms, but it will still be shedding the virus to infect other horses.

The similarity of symptoms to those from other causes of upper respiratory disease makes a diagnosis difficult. Influenza occurs mostly in 1- to 5-year-old horses, the ones that come in frequent contact with other horses.

The treatment of an influenza-diseased horse is the same as for other viral diseases: isolation, supportive care, and antibiotics against bacterial complications. The uncomplicated disease will reduce the severity of the disease, reduce virus shedding and limit abortion incidence and severity. None of them seems to be able to protect against the neurological aspects of the disease. Annual re-vaccination is necessary.

**Equine viral arteritis** is a spherical, enveloped RNA virus about 50 to 60 nm in diameter. The virus exists worldwide in horse populations everywhere except for Japan and Iceland. It produces respiratory disease, generalized viremia and abortion.

Infection is by aerosol droplets; the virus is taken up by macrophages and carried to the local lymph nodes. From the lymph nodes it is disseminated via the blood stream throughout the body, ending up in the endothelial cells of small blood vessels and arterioles of various organs, the adrenals, testes, thyroid and liver. It produces vasculitis, inflammatory cell infiltration, endothelial swelling and necrosis of the tunica media leading to edema and hemorrhaging in the affected organs. The triggering of abortions may be based on the same pathogenesis involving the myometrium of the dam. Some stallions may remain carriers by localizing virus reproduction in the ampulla of their vas deferens. Carrier stallions pass the virus on to their semen and are the main source for infecting the world’s horse populations.

Because the virus infects most equines without producing symptoms, it is distributed widely through breeding establishments, racetracks and show places. Aside from droplets, transmission can occur through venereal contact (natural or artificial insemination), vertically in utero, and via contaminated gear, hands or instruments.

The few infected horses that do exhibit symptoms may show fever, leukopenia, loss of appetite, and edema of limb, prepuce and scrotum. There may be conjunctivitis, tearing of the eyes and photophobia, edema around the eyes, rhinitis and nasal discharge, edema on the ventral body and mammary glands, urticaria around neck and head, discoordination of gait, dyspnea, diarrhea and icterus.

Occasionally it is fatal for young foals. Because it is a virus disease, treatment must remain symptomatic and supportive in the few more serious cases. Most cases will recover completely within a few weeks without treatment.

There is no way at this time to correct the carrier situation with stallions. Modified live virus vaccines are in use and safe and effective for most horses. They should not be given to pregnant mares or foals less than 6 weeks old. Breeding programs must consider the immune status of the animals to be vaccinated and the selection of confirmed non-carrier stallions.

Carrier stallions must be kept separate from susceptible and nonvaccinated animals. They should be allowed to service only vaccinated and immune mares. The same reservations, of course, are valid for the semen from carrier stallions. All semen not from a known carrier animal should be tested for the virus.

Efforts to reduce, if not eliminate, the virus reservoir must include the vaccination of all young animals between the ages of 6 months to 1 year before they have the opportunity to become naturally infected.

**Equine rhinitis virus** is ubiquitous as well. There are two types of equine rhinitis virus, both belonging to the *Picornaviridae* family. They are small, single-stranded RNA viruses, spherical, about 30 nm in diameter. Nonenveloped, they consist of an RNA genome surrounded by a protein shell.

The two types are equine rhinitis A (ERAV: genus Aphthovirus) and equine rhinitis B (ERBV: genus Erbovirus). The type A virus has only one subtype, while type B consists of three serologically distinct serotypes. Both types affect the upper respiratory tract and cause upper respiratory disease in horses worldwide.

Equine rhinitis A virus produces laryngitis, viremia and large amounts of initially serous, thick, discolored nasal discharge. Although an Aphthovirus, ERAV is not a foot-and-mouth-disease virus. It infects young adult horses when training for racing. It induces a good immune response, limiting virus growth and spread. It will occasionally infect man.

Equine rhinitis B virus, in contrast, infects the younger horse, mostly foals and weanlings, producing a much less impressive immune response. It also produces upper respiratory infections of varying degrees of severity worldwide. The lower antibody response following the equine rhinitis B virus infection results in more frequent, usually seasonal, re-infection. Once infected, some horses have shed the virus for up to two years.

**Equine reovirus** (respiratory enteric orphan virus) is a nonenveloped, spheroid virus with two capsids surrounding the double-stranded RNA genome. Reovirus exists worldwide and has been isolated from foals with diarrhea and race horses with upper respiratory disease. Little is reported about the pathogenicity of the virus. Its presence does not necessarily mean that it has produced a clinical disease.

The name of the virus – *Reo*, from respiratory enteric orphan – was coined by Sabin in 1959 “because the virus was not connected to any of the diseases it was associated with.” Indeed, serological surveys show hemagglutination inhibiting antibody to the virus in as many as 60 percent of the horses tested. Yet, only about 16 percent could be implicated in the etiology of respiratory disease when examining convalescent serum samples.

Inoculation of susceptible horses with reo virus isolates sometimes causes conjunctivitis and mild rhinitis lasting about two weeks, and the virus can be isolated from the feces, nasal and conjunctival swabs.

Evidently, reovirus is present in some respiratory events, occasionally producing mild upper respiratory tract disease, mild conjunctivitis with slight serous ocular and nasal discharge and nasal hyperemia, and infrequent coughing. While it will pass within a week or two, it is likely to interfere with the horse’s full performance. Performance stress may also lead to an enhanced severity of the disease, with follicular pharyngitis, upper and lower tract respiratory disease and systemic symptoms, including elevated temperature, labored breathing and dyspnea.

In Europe, a formalin inactivated reo virus type 1 and type 3 component with equine herpes type 1 and influenza/equine A 1 and 2 vaccine combination is used, eliciting effective antibody levels lasting for seven months.
Equine adenovirus is a non-enveloped, icosahedral, double-stranded DNA virus, 90 to 100 nm in size. There are two equine adenovirus types: Adenovirus type 1, which produces pneumonia, destruction of pancreatic and salivary gland tissue. It is fatal in Arabian foals with severe combined immunodeficiency disease, a genetic disorder of some Arabian horses that are born without an immune system (humoral or cellular) and usually die within the first four or six months of life from any kind of opportunistic infection. It is carried by a recessive gene that can be identified by DNA testing and excluded from breeding, or at least from breeding with another recessive gene-carrying mate.

Equine adenovirus Type 2 is the cause of gastrointestinal disease. Adenovirus type 1 has been isolated from the upper respiratory tract of horses with minor respiratory disease. The presence of the virus is usually asymptomatic and not a major problem. It probably does have some effect on racing performance.

Hendra virus is included here because this virus represents one of the new emerging diseases. Also called equine morbillivirus, it is an enveloped, spherical, single-stranded RNA virus about 150 nm in size. It is a member of the family Paramyxoviridae, and has been isolated initially from horses with respiratory and neurologic disease. It is limited to Australia and its natural reservoir is fruit bats, the flying fox (Pteropus). Transmission likely occurs by contamination of pasture or feed through infected birthing fluids or fetal tissues from bats. Hendra virus can survive for days in flying fox urine at 22 degrees C (72 degrees F).

The highest incidence of outbreaks is between August and January, the months when most species of the flying fox give birth.

Hendra virus is a zoonotic virus, causing disease in man. Animal handlers and veterinarians coming in contact with the body fluids and excretions of the infected horse can develop a respiratory disease with severe influenza-like symptoms, often followed by progressive encephalitis and death. In horses, the disease picture ranges from an asymptomatic to severe respiratory disease and neurological syndrome with a fatality rate as high as 75 percent.

Supportive and symptomatic care is the only treatment available. It is important to recognize the disease early because it takes only one to two weeks for the disease to show up in man. The incubation period in horses ranges from five days to two weeks, and the infected animal becomes often infective a day or two before the first expression of the disease.

Prevention begins with removal of access for the reservoir bats, such as open stalls, food and water bins, thorough cleaning, disinfection of horse stalls, and proper sanitation and hygiene. Outbreaks must be limited by immediate quarantine of facilities and animals, culling of infected horses and proper disposal of carcasses (burial, incineration).

Education of individuals involved in direct contact work with infected animals is absolutely essential: they must wear protective clothing, gloves, protective eyewear and facemasks. Feed and water troughs must be protected from bat access. The virus is susceptible to soaps, detergents and many common disinfectants, including hypochlorite, iodophors, biguanidines (e.g. chlorhexidine) and quaternary ammonium compounds, desiccation or heat, but resists inactivation by acids or alkalies; it can survive a wide pH range from 4 to 11.

The clinical signs in horses are characterized by vasculitis, respiratory disease and disease of the central nervous system. The respiratory disease is acute and rapidly progressive, often leading to death within days. Early signs are high fever and an elevated heart rate, anorexia, sweating and respiratory distress, including rapid, shallow, labored breathing. The mucous membranes may become congested and jaundiced. There is frequently ataxia and other neurological symptoms and subcutaneous edema. At death there is frothy, thick, often bloody nasal discharge. Death follows the initial symptoms in one to three days.

The few horses that survive may develop neurological aftereffects. The disease of the central nervous system consists of discoordination: ataxia, altered gait (high stepping, wobbling), altered consciousness, aimless walking, circling, apparent blindness on one or both eyes, unnatural head tilt, muscle twitches, tremors, facial paralysis, lockjaw, jaw spasms and involuntary chomping. There may be colic, straining to urinate and defecate and dribbling urine.

The virus can be isolated from oronasal secretions, the urine and various tissues as many as two days before the disease shows up, i.e., the animal may be shedding contagious virus before the first appearance of symptoms of the disease. Horse-to-horse transmission is possible in crowded conditions because of the close contact of the animals.

Postmortem pathology includes pulmonary edema, congestion and consolidation, and pulmonary lymphatic dilatation in obstructive anomalous drainage of pulmonary veins. Much of these symptoms are influenced or modified if not caused by virus-induced vasculitis: there are patchy petechial hemorrhages on pleura and lung parenchyma, generalized and visceral edema, bloody froth from airways, swollen and congested lymphatic system, petechiae and ecchymoses throughout the gastrointestinal tract and kidneys, and yellow discoloration of subcutaneous tissue. There is no treatment other than supportive care.

Surviving horses must be checked for virus persistence and continued shedding, and they should be held isolated; strict control of contact and traffic from and to the suspect animal should be instituted; and careful and thorough disinfection enforced. Persistent carriers should be euthanized. Flying fox debris should be removed safely and buried or burned. While the incidence is not large, three quarters of the horses that get the disease will die.

Bacterial infections also play a role in the equine respiratory disease complex.

Strangles, also called “distemper,” is an upper respiratory tract infection that is produced by the highly infectious Streptococcus equi equi. It can appear without the prodromal viral infection that is a prerequisite for many of the bacterial secondary complications. Main areas of infection are throat, local lymph nodes, the guttural pouch and the mucosae of the upper respiratory tract. Occasionally it will also involve the lungs.

The initial symptoms of strangles consist of fever above 39 degrees C, loss of appetite, stridor, thick yellowish nasal discharge and swollen, painful and hot lymph nodes of head, neck and mandible. The lymph nodes will become abscessed and break open, releasing thick, yellowish, evil-smelling pus. Strangles affects horses of all ages, but is more likely to occur in younger animals, weanlings and yearlings.

Infected animals must be placed in isolation, and separate equipment and strict control of traffic must be maintained. The infection is commonly spread by direct contact with an infected horse, via the infectious material from nasal discharge and abscessed lymph nodes, by use of common equipment (water troughs, hoses, feed bunks, pastures, stalls, trailers, tack, grooming equipment, nose wipe cloths or sponges), and by attendants’ hands and clothing. Thorough cleaning and disinfection of equipment is important to keep the disease from spreading.

Flies, birds and rodents may carry the pathogen and infect other animals. But outbreaks are usually started in aggregation points for horses: horse shows and riding events, racetracks, stud services, boarding stables and rental pastures.

After the disease has run its course, as many as 10 percent of the recovering animals may become carriers and continue to shed the pathogen on an intermittent basis without showing evidence of the disease. Usually, however, the disease and recovery from it confers lifetime immunity.

When strangles is a persistent endemic problem, vaccination may be called for. There are reports of hypersensitive reactions to vaccination, such as purpura hemorrhagica, which is caused by immune-mediated, generalized vasculitis (immune complexes of antibody and streptococcal antigen in vascular basement membranes). Two to four weeks after vaccination the horse may develop urticaria, pitting dependent edema (lower parts of body and limbs) and subcutaneous petechial hemorrhages. There may be swelling of the head and concomitant breathing difficulties. Purpura hemorrhagica also happens naturally after streptococcal infection. Not being avoidable either way, the question remains whether vaccination would tend to mitigate or exacerbate this occurrence.

Another primary bacterial pathogen producing equine respiratory disease without necessarily prodromal viral infection is Rhodococcus equi. It
produces lower respiratory tract disease in young foals (less than 5 months old) with pulmonary consolidation and abscess formation. It does not affect adult horses with a mature functioning immune system.

Mostly, however, bacterial complications follow a predisposing viral disease. Bacteria such as streptococcus equi zoopidemicus, actinobacillus equuli, bordetella bronchiseptica, Escherichia coli, pasteurella spp. and pseudomonas aeruginos, that participate in equine respiratory disease are usually opportunistic bacteria, the resident microflora of the upper respiratory tract normally present in the healthy body. These bacteria take advantage of the virally caused surface damage of the respiratory tract and the mucociliary apparatus as well as the viral destruction of the bronchi-pulmonary lymphatic tissue and weakening of the immune response.

Indications of complicating secondary bacterial infections are persistent fever, depression, thick, mucopurulent nasal discharge and abnormal breathing sounds and symptoms ranging from rhinitis and tracheitis to acute pneumonia, pleuropneumonia and other serious invasive disease.

When reviewing the equine respiratory disease complex, we must include non-infectious causes, such as inflammatory and reactive airway disease (excessive mucus production, constriction of bronchioli and alveoli, mucosal hyper reactivity and dyspnea) that may be caused by dust, organic or otherwise, and toxic fumes affecting the performance of the afflicted horse.

Aside from antibacterial chemotherapy to control the bacterial aspects of equine respiratory disease and the supportive treatment of its symptoms, the environmental factors surrounding the horse must be controlled. Stables should be clean, dust free, free of the smell of ammonia and other chemicals that might irritate the eyes and upper respiratory apparatus, have good feed quality (absence of mold, rodent debris, bat and bird access), ambient moisture and temperature control, and, of course, control of internal and external parasitic infestations.

**Equine encephalitis complex** covers illnesses of the central nervous system, often deadly, reaching mortality rates of 90 percent.

There are four types of equine encephalomyelitis virus: eastern equine encephalitis, common in the Eastern regions of the United States and Canada, the Caribbean, and Central and South America, including along the Gulf Coast. Western equine encephalomyelitis is found west of the Mississippi River in the United States, in western Canada, Mexico and parts of South America. St. Louis equine encephalomyelitis is common throughout the United States and Venezuelan. Equine encephalomyelitis is usually found in Florida, South and Central America and Trinidad.

Affecting the central nervous system, the syndrome affects all muscular activity of the body. There is no treatment other than supportive care, anti-inflammatory and anti-seizure medication.

The disease outbreaks are a result of the flying season of mosquitoes, which are the carrier and transmitting vectors. The incubation period, from the time of mosquito bite to the first symptom, is one to three weeks.

The first symptoms usually reported are depression, apparent blindness, going off feed, high fever, hanging lip, loss of coordination and dyspnea, and may reach complete paralysis. Some horses may take six weeks to recover, and they may have permanent brain damage and may not be safe to use for riding or breeding.

Eastern, western and Venezuelan equine encephalitis belong to the family of Togaviridae, serocomplex alphavirus. They are single-stranded RNA viruses, spherical about 65 nm to 70 nm in diameter, enveloped with an icosahedral capsid within. The primary reservoir of alphaviruses is birds. Horses and man are dead-end hosts. High infection rates in birds are followed by infections in horses and man, with mosquitoes responsible for passing on the virus.

Alphaviruses are among the most serious pathogens for horses. There are many varieties of alphavirus species throughout the world, many with like symptomatology, but only a few are active in the United States.

**Eastern equine encephalitis** consists of two antigenic variants:
- The more pathogenic and more antigenically homogeneous one is prevalent in the United States and Canada.
- A somewhat less pathogenic and less antigenically homogeneous one is found predominantly in South America.

Like all alphaviruses, these are transmitted by mosquitoes (Culiseta melanura, Culiseta morsitans).

The virus enters the lymphatic system and lymph nodes, replicating in neutrophils and macrophages, and destroying them in the process. As a result, there will be fever, lymphopenia and leucopenia. As the virus becomes viremic, it will spread, enter other organs and the central nervous system.

Early symptoms appear within about five days after infection. They are neurological in nature: The horse becomes quiet and depressed, exhibits head pressing, aimless wandering, circling, ataxic gait, asymmetric paralysis, paresis and convulsions. Mortality rates reach up to 75 to 90 percent. Death occurs usually within three days after the onset of the disease.

Alphaviruses cause disease in man. Like the horse, man is a dead-end host and is not an essential part of the transmission process. Birds, rodents and mosquitoes are the main reservoir for these viruses.

The symptoms in man, as a rule, include fever, rash, encephalitis and arthritis. Following the infected mosquito bite, the virus enters the lymphatic system and the blood stream and disseminates throughout the body. It will enter the central nervous system to multiply in and destroy neurons, causing encephalitis and often death. After entering the lymphatic system, alphaviruses will stimulate T cells and induce the production of interferon and antibody to limit virus progress.

There are no alphavirus vaccines. Removal of mosquito breeding grounds, stagnant surface water, public spraying and use of protective clothing are presently the best means of dealing with the disease.

*Culiseta melanura* is the main vector for eastern equine encephalitis. Its importance in the transmission process is highlighted by the regional and seasonal occurrence of the disease. In Florida, for instance, where the mosquito season covers all four seasons, transmission...
occurs year-round with the summer as the peak, while in the more temperate climates, transmission occurs during the late summer and fall until the first frost.

By contrast, the western equine encephalitis virus is transmitted mostly by Culex tarsalis and a tick, Dermacentor andersoni, to horses and human beings. Dependent on the presence of infected vectors, its incidence increases with early spring rains and a warm summer. Only female Culex mosquitoes will pass on the disease. They bite animals and man as well as birds, and suck blood from the infected viremic host and spread the virus by passing it on directly to its next meal donor.

Infected birds, of course, are an excellent means of spread over wide areas. The female Culex passes the virus on vertically through its eggs. It needs repeated feedings for the development of a batch of eggs. Infected Culex eggs survive for years in dry areas and will arise during rainy periods and floods.

The disease process in horses is like that for other alphaviruses, although there may be less of a viremia for this virus. Its main course of operation is in the central nervous system, the brain and spinal cord. Immunologically active cells, such as macrophages and neutrophils, invade the brain tissue and perivascular regions, leading to neuronal destruction, spotty demyelination, focal necrosis, thrombosis and endothelial proliferation.

Mortality rate in infected horses is 20 to 50 percent.

Western equine encephalomyelitis in man is not very common: there were fewer than 700 cases in the United States during the past 46 years. Fewer than five cases are seen annually in the United States, usually in early summer.

The reason for this may be that open symptomatic infections are rarely seen. When there are such, they appear within about a week to 10 days after the mosquito bite. They bracket the spectrum of symptoms, from a light influenza-like picture, possibly a headache, a light fever to an abrupt high fever, nausea and vomiting, encephalitis, coma and death.

More infants and children get sick than adults, and about one out of three develop neurological consequences, including behavioral disorders, spasticity, seizures and retardation. Mortality rate is about 4 percent, and it is mostly the elderly that will die.

There is no treatment other than supportive care. The control of mosquitoes and mosquito breeding grounds and the avoidance of mosquito bites (insect repellent, protective garb) are essential for prevention.

Venezuelan equine encephalitis affects all equines and man, but does not occur very often in the United States. In 1971, there was a major epidemic in horses in Texas, but only about 100 people were affected. Data from international outbreaks suggest that many more subclinical infections may have occurred.

The diseased animal may exhibit progressive disease of the central nervous system or it may die without any prodromal symptoms. The mortality rates reported reach as high as eight of 10 infected animals.

The virus will infect and produce disease in man, especially when in weakened condition or immuno-compromised, the very young and the elderly. It causes influenza-like symptoms, fever, headache, hypotension, stiff neck, altered behavior, coma and death. During the course of three epidemics in Venezuela and Columbia in the last half of last century, more than 300,000 people were infected, more than 12,000 had severe neurological complications, and more than 2,000 died. In patients developing encephalitis, as many as one out of five may die.

Transmission again is via mosquito bite, feeding on an infected viremic animal then passing it on to a new host. Thus the effectiveness of spread is a direct function of the number of mosquitoes present, the viremic character of the virus and the access to a virus reservoir. The control of mosquitoes in the subtropics in large swamp-like regions is all but impossible.

St. Louis encephalitis virus is transmitted like all the other mosquito-borne viruses, during the mosquito season. Birds appear as intermediate hosts, amplifying the virus population in their blood without showing signs of the disease themselves. There have been several St. Louis encephalitis virus epidemics in the past. In 2008, the Centers for Disease Control and Prevention reported eight “confirmed and probable St. Louis encephalitis virus neuroinvasive disease cases,” and five “confirmed and probable St. Louis encephalitis virus non-neuroinvasive disease cases.”

It is often asymptomatic, but when illness does occur, it is more severe and more frequent in older people than in the young. Fatality rates in individuals over 50 years of age are 7 to 24 per 100 people, while they are less than 5 in 100 in those younger. Individuals surviving the more severe disease may show long-term damage to the central nervous system as well as paralysis, memory loss, and loss of fine motor skills. There is no vaccine and no cure available for this disease.

Prevention depends on mosquito control:

Eliminate standing water, keep mosquitoes out of stalls, and use mosquito traps and insect repellent. The infected horse should be isolated and kept in a screened area to keep mosquitoes from feeding off him.

West Nile virus is a mosquito-borne virus that infects horses. It belongs to the family Flaviviridae. It infects and can be deadly for humans, geese, wild birds, squirrels, alligators and dogs. Birds and mosquitoes are the natural reservoir, and maintain the virus by transmission from one to the other. Both play the essential role in the spread of the virus.

Viral transmission depends on the mosquito-bird life cycle, usually from mid-July to October in temperate regions, and year-round in semi-tropical regions of the country. Man and horse are considered “dead-end hosts,” but it is a reportable disease.

The incubation period can range from three days to two weeks. While more than 60 percent of unvaccinated infected horses do not get sick, some show signs of encephalomyelitis expressed as listlessness; somnolence and hyperreactivity; central and peripheral nervous system dysfunction with fever; ataxia including stumbling, staggering, wobbly gait, circling, weakness or paralysis of the hind limbs; impaired vision; lack of coordination; head pressing; convulsions; drooping lip, teeth...
grinding and inability to swallow; colic; and coma. The mortality rate of infected horses is about 30 to 40 percent.

In 2010, there were 114 cases of West Nile virus reported in the United States. The symptoms in man include fever, headache, nuchal rigidity and body aches, often skin rash and swollen lymph nodes. There may be stupor, disorientation, coma, tremors, convulsions, paralysis, and possibly death.

Cats acquire the virus from consuming infected birds or rodents. The virus exists worldwide. The similarity to other diseases of the central nervous system requires laboratory diagnostic procedures for a definitive diagnosis (virus isolation and serology). Treatment consists of supportive care. Recovered horses will be immune for years.

Preventive measures are vaccination and mosquito control (removing standing waters, etc.). There is no cross protection between eastern, western and Venezuelan equine encephalitis and the West Nile virus.

Vaccines are available for these equine encephalitis viruses as well as for West Nile virus. They are killed virus vaccines containing adjuvants to enhance their antigenicity. Dual vaccine combinations are in use for eastern and western equine encephalitis, and are also available with a tetanus toxoid component. These inactivated virus vaccines must be given twice initially, two to four weeks apart, and then boosters should be given once a year at least two weeks before the mosquito flying season starts. Some recommend repeat vaccinations every four months or at least a second dose of the vaccine in the fall. Foals should be revaccinated after six months.

Vaccination must be complemented by mosquito control, including use of insect repellent and elimination of sources of standing water.

Two vaccines for Venezuelan equine encephalitis have been used: one, an attenuated strain of the virus (TC-83), passed 83 times in guinea pig heart cells, and two, an inactivated vaccine (C-84) produced from the TC-83 vaccine strain.

The attenuated vaccine has a history of mild to moderate side effects in man, including stiff neck, confusion and seizures, and there have been reports of fatal abnormalities and deaths. The attenuated vaccine is not used in the United States. However, C-84 is accepted for use in horses.

La Crosse encephalitis belongs to the family of Bunyaviridae, serogroup California. It consists of single-stranded RNA, is spherical or oval, surrounded by an envelope and 90nm to 100nm in diameter. La Crosse encephalitis virus is transmitted by the eastern tree hole mosquito (Aedes triseriatus), which in addition to the chipmunk, squirrel and other small mammals, serves as a reservoir. Habitat for both hosts is in deciduous forests.

The virus ending up in man or horse is more by accident than by design, and both are considered “dead-end hosts.” There is no open disease in horses.

Viremia is not long enough and virus levels not high enough for mosquitoes to pick up the virus when feeding and to pass it on. The mosquito passes the virus on vertically to its eggs, allowing it to over-winter to infect again in subsequent years. About 80 to 100 human La Crosse encephalitis cases are reported yearly.

Powassan virus is a single-stranded RNA virus and belongs to the family Flaviviridae. It is tick-borne (Ixodes cookie) and produces a non-suppurative meningoencephalitis in horses with focal necrosis. It has been found in Ontario and the eastern United States.

Symptoms consist of tremors of neck and head, discoordination, excessive mastication producing foaming saliva, wobbly gait, and staggering and recumbency. The pathology shows aseptic encephalomyelitis, neuronal necrosis and necrotizing areas in the brain tissue.

Powassan virus is zoonotic and will produce severe encephalitis in man. The virus will pass to its new host within the first 15 minutes of attachment of the carrier tick. Symptoms usually develop one to three weeks later.

The virus reservoir is maintained in a cycles of tick-roden, and tick-small mammals (skunks, squirrels, ground hogs). The reservoir hosts do not develop noticeable disease.

Japanese encephalitis virus is a member of the Flaviviridae family. It is spherical, enveloped with icosahedral symmetry about 50nm in diameter. It is an arthropod-borne (mostly Culex, but also Aedes and Anopheles) and infects horses, donkeys, pigs, birds and man. It also infects a variety of other species, however, all of them asymptptomatically. Birds, again, are part of the arthropod-bird virus-maintenance reservoir cycle.

A number of epidemics in the past produced high mortalities in man (4,000 in 1924 in Japan, 2,500 in 1949 in South Korea) and in horses (3,700 in 1949 in Japan). It is prevalent in the subtropical and tropical regions of Asia, including Indonesia, Australia, Papua New Guinea and Pakistan.

Worldwide, 20,000-50,000 cases of viral encephalitis in humans are reported every year. The incidence varies, depending on the season and the presence of mosquitoes.

The disease is usually much more severe in the elderly and the very young. Fortunately, the virus is asymptomatic in most people it infects; less than 1 percent of infected individuals will become sick. However, there are occasional reports of morbidity rates as high as 4 percent.

After an incubation of one to two weeks, there may be fever, chills, myalgia, severe headache and vomiting. In children, there are symptoms of gastrointestinal disease, abdominal pain, nausea and vomiting. Aftereffects may consist of reduced levels of alertness and consciousness, nuchal rigidity, convulsions, flaccid paralysis of upper extremities, tremors, rigidity, epileptic seizures and diminished cognitive and language capacity.

In horses, the incubation period is one or two weeks. A few horses will exhibit minor evidence of a disease, including transient fever, anorexia, lethargy, swollen and jaundiced mucosae for maybe two to three days and then recover without complications. Other horses will show signs of transient central nervous system involvement: sullessness, lack of coordination, anorexia, fluctuating fever, difficulty swallowing, nuchal rigidity, impaired vision and radial paralysis. This will pass within a week or so.

The most severe form of the disease is often followed by collapse and death within a day or two. It is also called the “hyperexcitable form,” with high fever, circling and aimless wandering, apparent blindness, profuse sweating, violent reactions, excitable and muscle tremors. Only about 5 percent of horses that show symptoms will show the latter form. Some neurological damage, such as ataxia, may persist in animals recovering from the disease.

Post-mortem, there are no gross lesions, but may be diffuse encephalomyelitis, perivascular cuffing and focal gliosis, dilated blood vessels and mononuclear infiltration.

In the pig, the virus interferes with the normal reproductive process, producing abortion, stillbirths and mummification of fetuses, and piglets born alive develop tremors and convulsions and may die soon after birth. Their pathology may show hydroencephalus, cerebellar hypoplasia and spinal hypomyelogenesis. Nonpregnant pigs may show no symptoms at all, or they may develop a mild transient fever or encephalitis. Japanese encephalitis season goes from spring to fall concomitant with the mosquito flying season, only modified by changes in the rainy season.

The diagnosis suggested in the infected horse by fever and neurological symptoms and in pigs by the high number of stillbirths, mummified or weak newborns must be confirmed in the laboratory. Japanese encephalitis must be reported to the local authorities as soon as it is suspected.

Disease prevention includes both vaccination and mosquito control. An inactivated vaccine produced in Japan has been successful in reducing morbidity and mortality significantly. All animals imported from one of the regions suspected to have the virus should be vaccinated without fail and so should foals born from imported mares.

Vaccination will not break the cycle of transmission because birds are involved and bound to pass on the virus infection. Mosquito control is helpful in controlling transmission, including
removal of breeding grounds, insect repellent and spraying walls with insecticides, fans producing a strong side wind, and mosquito netting.

Rabies virus is part of the Rhadoviridae family, serocomplex Lyssavirus. The family of Rhadoviridae are bulletshaped, enveloped single-stranded RNA viruses (75 by 150 nm in size). Rabies will affect any mammal, but particularly carnivores and bats, producing acute viral encephalomyelitis. Once clinical symptoms have appeared, death is certain.

Reservoir animals for rabies, which occurs worldwide, vary by regionality. In the United States in 2008, 93 percent of all reported cases came from wildlife animals: raccoons (34.9 percent); bats (26.4 percent); skunks (23.2 percent); foxes (6.6 percent); and other animals, including rodents, hares and rabbits. Domestic animals represent only 7 percent of all rabid animals reported.

Rabies in man in the U.S. is usually caused by bat rabies, and cats seem to be most often affected. Virus is found in rabid cat saliva, and rabid cat bites have produced rabies in man. In the U.S., more cats are reported with the disease than dogs. Transmission may occur via skin wounds and mucosa, but this is unusual, as animal bites seem to be the predominant course of infection. High-dose aerosols in bat caves have occasionally produced rabies. An extended incubation period will improve chances of postexposure effectiveness of rabies immune globulin. Annual death rates in man have declined from the hundreds in the early last century to two or three a year today thanks to effective animal and human vaccines and the use of immunoglobulin.

The early signs of rabies are not very descriptive: influenza-like, headache, general discomfort, malaise and fever. At the bite site, an itching sensation will be followed by progression to central nervous system disease such as agitation, confusion, anxiety and hallucinations, behavior changes, insomnia and delirium. The prognosis after the appearance of clinical signs of rabies is not good; the disease is almost always fatal. It is therefore crucial that the rabies virus infection be identified early and treatment in the form of human immune globulin, along with a course of rabies vaccination, be commenced as soon as possible.

Rabies in horses is uncommon; there are about 40 cases per year. The horse can contract rabies through the skin, a bite from a rabid animal or by infected saliva entering through a break in the skin. The incubation time can be two to six to 12 weeks, even up to one year.

The early clinical signs are varied: there are behavioral changes, sullenness, dullness, depression, nervousness, irritability, difficulty swallowing, drooling, going off feed, abdominal pain, colic, problems urinating and defecating, fever and convulsions, and sensitivity of the injured area, including biting and favoring it. After neurological symptoms appear, there is quick progress to recumbency and death within about a week’s time.

If there is no sign of a rapid continuing progression by day five, it is probably not rabies, and other causes should be investigated. Such causes might be equine herpes virus, any one of the viruses of the equine encephalomyelitis complex, botulism, tetanus, protozoal myelitis, moldy corn poisoning, and trauma of brain or spinal cord. The intact head should be sent on wet ice to the local public health unit for definitive diagnosis. Fluorescent antibody staining can be performed quickly and results returned immediately. If positive, the rabies treatment and vaccination course should be commenced immediately for anyone exposed to the rabid animal. Virus isolation is possible in mice and cell cultures.

Treatment of an infected horse will be too late because it would only be started after the appearance of symptoms. On the other hand, if the horse has been vaccinated for rabies, a booster given immediately plus a thorough cleaning out of the wound with ample soap and water is recommended. Quarantine and observation of the surviving horse for clinical symptoms for at least six months are mandatory. Upon appearance of symptoms, the horse should be euthanized and its head submitted for laboratory confirmation.

In an area where there is a history of rabies and reports of rabies in wildlife, vaccination should be started, beginning with 3-month-old foals, and yearly boosters. Indeed, the American Association of Equine Practitioners (AAEP) included rabies vaccination in its list of core vaccinations. This horse vaccination program must be complemented by vaccinating pet cats and dogs, not touching or adopting wild animals and being on the lookout for stray animals in the area.

Considering that rabies is recognized usually late during the infection process, exposure to the saliva of an infected nonsymptomatic animal could have serious consequences. The best method of avoiding that is an up-to-date vaccination program.

Other nonrespiratory and nonencephalitis viruses include equine infectious anemia, which is a lentivirus, part of the family called Retroviridae. It is an RNA virus, enveloped and spherical to pleomorphic in shape and about 80 to 100 nm in diameter. The lentiviruses are a group of slow viruses causing immunodeficiency in man (HIV-1), simians (SIV), felines (FIV), sheep and goat, cattle (BIV) and horses (EIAV).

This virus delivers enough genetic information to the host cell to allow it to replicate even in nondividing cells. While viruses normally have to be extruded from their producer cell to infect another cell, immunodeficiency viruses can go directly from cell to cell by direct contact without having to complete the virion particle.

The equine infectious anemia symptomatology ranges from minimal clinical effects to high morbidity and mortality. Horses, ponies, mules and donkeys are natural hosts to the virus. The disease exists worldwide. In the United States fewer than half a percent of all the animals tested were positive to the virus. There are no vaccines available. Disease control programs must include serological testing.

Equine infectious anemia virus infection persists for life, with the virus circulating with the blood and being available to tabanids (horseflies, deer flies) to pick up with their blood meal and pass on to the next host. Because their bite is painful to the animal, they will be fought off by the host and sent on to the next animal, increasing the speed and range of virus spread. The virus does not replicate in the fly, suggesting that the horse is the only host and reservoir. Naturally occurring outbreaks coincide with the peak density of the blood-sucking horsefly population.

After infection, the incubation period is three to seven weeks. The disease may develop in three forms. The acute form consists of sudden onset, high fever (40 to 43 degrees C) severe depression, anorexia, rapid loss of physical condition, profound weakness and prostration, petechiae on the oronasal mucosa and bloody, yellowish nasal discharge. There may be dependent edema (edema of the ventral abdominal area and extremities), splenomegaly and jaundice and severe anemia shown by low hematocrit, low hemoglobin value and a low red blood cell count, occasionally ending with death.

Some horses develop the subacute form, with milder symptoms than for the acute form, occasionally recurring after stress events with concomitant loss of weight and weakness. The chronic form may follow the previous course of events, but there is no fever or anemia; the animal may appear normal but unthrifty and lack stamina, and will be consistently viremic for the horse fly to feed upon and to infect other horses. The longer it remains undetected, the more animals it will infect.

The final diagnosis must be confirmed by serology using the Coggins test (agar gel immunodiffusion) and ELISA. While the ELISA lends itself to a quick screen, taking only minutes to execute, it must be confirmed by the Coggins test (one to two days) to satisfy regulatory requirements.

Once infected, always shedding: The risk of keeping antibody positive animals around is considerable. Antibody levels produced by the infection do not reach high levels and are not very protective. This fact explains the persistent viremia in carrier animals. Foals born to infected mares will have maternal antibody for months and still be viremic.

Because there is no cure for the disease and infected horses will persistently be contagious, the U.S. Department of Agriculture and state animal health regulatory agencies require euthanasia or strict lifelong quarantine for horses testing positive for equine infectious anemia virus.

Vesicular stomatitis virus is part of the rhabdoviridae family. The virus is bullet shaped, enveloped, and 75 nm by 180 nm in size. Arthropods are implicated in transmission as indicated by the seasonal occurrence of the disease.
(late spring to early fall). Exposure to infective saliva or effluent from lesions of an infected horse, shared feeding and watering implements, and transfer of lesion matter from infected humans all can transmit the virus. Infection requires an open break in the skin to be successful.

After an incubation period of two days to one week, the horse develops fever, and blisters begin to form on oronasal mucosae and the hoof corona. The animal will go off feed when the vesicles burst and commence to ulcerate. The horse begins to froth and drool, and the open lesion become painful. It may stamp its feet, become lame and develop laminitis. Occasionally, the hoof will slough off.

Fatality is low, and the animal may recover within a week or two, but bacterial superinfection of the open lesions may delay recovery. It must be differentiated from diseases with similar picture: gum irritation, and phenylbutazone toxicity ingestion of blister beetles.

The horse should be isolated, and cross contact with feeding and watering utensils and other animal handling equipment should be eliminated. Limit access of arthropod vectors and rodents by using insect repellent and netting.

Equine herpesvirus type 3 is a member of the Herpesviridae family. It causes a venereal disease, called equine coital exanthema, a contagious genital infection of the male and female sex organs, including the vulva, vagina, penis, prepuce, perineum, scrotum, as well as on lips and teats. It produces numerous small blisters, which turn to pustules and will burst and ulcerate. They often become bacterially infected, requiring antibiotic ointments for treatment. The disease will not affect the fertility of the infected males and stallions, but mating must be stopped until animals have fully recovered, usually about two weeks. Immunity following infection does not last long, but protects stallions during the same mating season.

The main route of transmission is venereal, through sexual contact, but direct-contact infection following poor personal sanitation and use of contaminated equipment has been observed. There is usually vaginal discharge, and flying insects are likely to carry the virus as well. The incubation period can be as short as two days.

Like other herpes viruses, equine herpesvirus type 3 may lie latent for the life of the animal, only occasionally triggered by extreme stress to break out again.

Rotaviral diarrhea is caused by Rotavirus, a non-enveloped RNA virus belonging to the family of Reoviridae, subfamily sedoreovirinae. Rotaviral diarrhea is responsible for half or more of all foal diarrheas. While about one-half of all susceptible foals become infected and sick, the mortality of the disease is minimal; usually less than one in 100 infected foals will die, provided they received proper veterinary care. Transmission is via the fecal-oral route.

The virus destroys the intestinal epithelium of the small intestine and its villi and interferes with food absorption and digestion and causes severe diarrhea. A large proportion of foals in the United States will have had at least one bout of diarrhea by the time of weaning.

Pregnant mares can and should be immunized with an inactivated rotavirus Group A vaccine to raise levels of maternal and colostral antibody. Where vaccination has been practiced regularly, incidence and severity of the disease has significantly decreased.

### Diagnosis

There is no one procedure covering all possible events and circumstances. The commonality of symptoms with the various causative agents for the equine respiratory disease complex or the equine encephalitis complex requires laboratory diagnosis in all of them.

Diagnostic procedures in today’s laboratories have become very efficient and precise. Test reagents are tailor-made for every specific need and circumstance, and most of them are available commercially. Every laboratory can perform the standard test with a minimum of variation from test to test.

Polymerase chain reaction tests, enzyme-linked immunosorbent assay, fluorescein conjugated antibody and immunohistochemistry are procedures capable of clearly identifying the virus, the viral antigens and antibody stimulated by it. Essentially, specific antibody produced to a specific antigen, be it viral, bacterial or any other,
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Variations of these procedures are antibody to specific antigens that are being labeled antibody to identify and measure allow the bulk production of species-specific guinea pig, rabbit, sheep and so on. This would particular animal species – man, horse, cattle, manner by producing antibody directed at the antigen is present in the test sample or on the cytometry or fluorescence microscopy. When the antibody is present in the test sample or on the test surface, it will retain the labeled antibody and specifically and clearly identify it. This same system can be used also in an indirect manner by producing antibody directed at the immunoglobulins (IgG, IgM, IgA etc.) of a particular animal species – man, horse, cattle, guinea pig, rabbit, sheep and so on. This would allow the bulk production of species-specific labeled antibody to identify and measure antibody to specific antigens that are being sought. Variations of these procedures are used everywhere. These examples are meant to show the principle behind each test with the understanding that they might be modified from lab to lab to satisfy specific requirements.

Speed is of critical importance in the diagnosis of rabies; it may be life-saving. Once the virus has entered the nerve pathways, it is protected from the body’s immune response and, as a rule, becomes unstoppable. The mouse neutralization test and the rapid fluorescent focus inhibition test are the measurements of choice.

Vaccination

The American Association of Equine Practitioners (AAEP) has proposed a list of core vaccinations, “the immunizations that protect from diseases that are endemic to a region, those with potential public health significance, required by law, virulent/highly infectious, and/ or those posing a risk of severe disease.” These core vaccinations include tetanus, eastern equine encephalitis/western equine encephalitis, West Nile virus and rabies. In addition, the AAEP proposes risk-based vaccination guidelines for anthrax, botulism, equine herpes virus, equine viral arteritis, equine influenza, Potomac horse fever, rotaviral diarrhea and strangles.

Numerous vaccines are on the market. They may not necessarily stop all infectious disease, but they will certainly reduce the incidence, severity and outcome of an infection. All vaccination must be accompanied by good horse-keeping management practices to strengthen the horse’s constitution, reduce stress factors, maximize health, productivity and performance, and control infection and disease transmission.

Factors that increase risks of infection include stress; overcrowding; parasitism; poor nutrition; inadequate sanitation; contaminated water source or supply; concurrent disease; inadequate rodent, bird and insect control; and the movement of people, vehicles, and equipment on and off facilities during infectious disease outbreaks.

The USDA Animal and Plant Health Inspection Service reports on the following vaccination practices on U.S. equine operations for controlling infections in equids. Theoretically, all horses should be vaccinated with the basic vaccines, such as eastern and western equine encephalitis, West Nile virus and rabies (core vaccines). Of equine operations in 28 states surveyed in 2006, the following vaccination frequency was reported:

- Rabies (44.5 percent).
- Equine influenza (72.5 percent).
- Eastern and western equine encephalitis (75.6 percent).
- Tetanus (81.3 percent).
- West Nile virus (85.3 percent).

Other vaccines that were not part of that survey include strangles, rhinopneumonitis (herpesvirus) and equine viral arteritis (EVA).

The American Association for Equine Practitioners suggests the following considerations before vaccination:

- The likelihood of disease, anticipated exposure, environmental factors, geographic factors, age, breed, use, and sex of the horse.
- Consequences, morbidity, mortality, infectivity for man.
- Effectiveness of vaccine.
- Possible adverse reactions.
- Cost considerations (cost of immunization versus cost of the disease if not immunized).

Re-vaccination should be considered before events of stress, travel, and entering areas with high population density (breeding farms, boarding stables, show barns, and race tracks). Any time horses are moved, they should be accompanied by copies of a complete vaccination record and health maintenance history. No animal should be accepted without such records.

Boosters, if applicable, should be administered at least one month before movement to allow enough time for an appropriate immune response. Proper veterinary procedure calls for detailed record keeping of the veterinarian’s actions, not
### Western Blot to locate and identify antigen

<table>
<thead>
<tr>
<th>Step</th>
<th>Material</th>
<th>Process</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Virus or antigen</td>
<td>May require lysis of cells with RIPA buffer, NP-40, Triton X-100.</td>
</tr>
<tr>
<td>2</td>
<td>SDS-PAGE</td>
<td>Sort by size/molecular weight.</td>
</tr>
<tr>
<td>3</td>
<td>Copper stain (0.3 M CuCl2)</td>
<td>To visualize fractions and control SDS-PAGE process.</td>
</tr>
<tr>
<td>4</td>
<td>0.1-0.25 M Tris/0.25 M EDTA pH 8.0</td>
<td>To destain; add 1X Tris-glycine buffer + methanol 20 percent for transfer.</td>
</tr>
<tr>
<td>5</td>
<td>SDS-PAGE fractions</td>
<td>Apply electrical field to transfer onto nitrocellulose blotting paper.</td>
</tr>
<tr>
<td>6</td>
<td>Ponceau red (0.2 percent in TBST)</td>
<td>Visualize fractions and control effective transfer, destain with TBST.</td>
</tr>
<tr>
<td>7</td>
<td>THE BLOT: Blocking</td>
<td>Rinse with TBST plus non-fat milk protein or BSA to block sticky surface.</td>
</tr>
<tr>
<td>8</td>
<td>Specific primary antibody in TBST</td>
<td>Add, incubate overnight at 4 degrees C (may keep blocking agent in TBST or not).</td>
</tr>
<tr>
<td>9</td>
<td>TBST</td>
<td>Rinse repeatedly 5 min or more each time to remove unattached AB.</td>
</tr>
<tr>
<td>10</td>
<td>Enzyme linked secondary antibody</td>
<td>Add, incubate, shake at rt temperature for 1-2 hours (no blocking agent in TBST).</td>
</tr>
<tr>
<td>11</td>
<td>TBS-T</td>
<td>Rinse to remove unattached secondary antibody.</td>
</tr>
<tr>
<td>12</td>
<td>Add luminal substrate, ECL, ECL+</td>
<td>Read enhanced chemiluminescence, photograph (CCD camera), scan.</td>
</tr>
</tbody>
</table>

Sample material is separated by molecule size with SDS-PAGE, (sodium dodecyl sulfate polyacrylamide gel electrophoresis), separation products transferred to nitrocellulose blotting paper (the blot) and identified by specific antibody. TBS-T (tris buffered saline +0.1 percent Tween20); horseradish peroxidase-conjugated antibody against primary antigen-specific antibody; ECL+: enhanced chemiluminescence.

### Mouse Neutralization Test (MNT): Measuring Antibody Titers

<table>
<thead>
<tr>
<th>Step</th>
<th>Material</th>
<th>Process</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Challenge virus</td>
<td>Titrate intracerebrally in young adult mice.</td>
<td>Small effective 100 percent killing dose.</td>
</tr>
<tr>
<td>2</td>
<td>Test serum/negat., pos control sera</td>
<td>Dilute in five-fold steps.</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Serum dilution+small 100 percent kill dose</td>
<td>Mix: inoculate intracerebrally into young adult mice.</td>
<td>21-day survival.</td>
</tr>
<tr>
<td>4</td>
<td>Check mortality</td>
<td>Determine LD₅₀; Dilution that protects 50 percent of test mice (Reed and Muench).</td>
<td></td>
</tr>
</tbody>
</table>

### Rapid Fluorescent Focus Inhibition Test (RFFIT) Measuring Antibody Titers

<table>
<thead>
<tr>
<th>Step</th>
<th>Material</th>
<th>Process</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>BHK cells</td>
<td>Determine cell concentration to produce monolayers.</td>
<td>Desired cell suspension</td>
</tr>
<tr>
<td>2</td>
<td>Test Serum/negat., pos control sera</td>
<td>Dilute in three-fold steps in flat-bottom microtiter plates.</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Serum dilution + 80 percent CPE dose virus</td>
<td>Incubate 1 hour at 37 degrees C.</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Desired cell suspension</td>
<td>Add to serum dilution/virus mix, incubate 24 hrs at 37 degrees C.</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Aceton fixative</td>
<td>Fix cell layer.</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Fluorescent conjugated Rabies AB</td>
<td>Add and count fluorescent foci.</td>
<td>Non-neutralized virus</td>
</tr>
</tbody>
</table>

The vaccines available are inactivated vaccines and modified live virus vaccines. The modified live virus vaccines are developed by serial passage in cell cultures until the virus has lost its pathogenicity or virulence while still retaining its antigenic character or by passing it at low temperatures and selecting low temperature mutants. Because the modified live virus is still able to replicate and multiply, less virus is needed per dose, and through its natural amplification, a broader spectrum of antigenicity can be achieved. It will stimulate both cellular and humoral responses equally well. On the other hand, immune-compromised animals may not be able to limit the growth of the attenuated virus and may develop symptoms of disease. In pregnant mares, they occasionally produce illness and fetal disease or abortions.

**Avirulent variants** of the pathogen have been used successfully. Antigenically sufficiently similar to the pathogen, they can be used directly, like vaccinia virus that was used to protect against smallpox and succeeded in eradicating the disease. Other vaccines recently developed or in the process of development are hand-tailored vaccines produced by genetic manipulation. Virulence genes are removed and replaced with a specific bit of complementary DNA (cDNA) capable of producing the desired antigenic protein necessary for a specific immune response, chimeric vaccines or recombinant vaccines where the virulent virus gene has been replaced by closely related safe virus virus genes capable of producing the desired antigen. The process in fact splices together two different organisms, insert of such genetic matter into a vector virus, such as canarypox vector vaccine, that enters mammal but does not kill them.

Plasmids, lambda phages have been used as vectors as well. The combination of a plasmid vector combined with modified vaccinia virus will replicate in infected cells and stimulate the immunity for which the vector has been programmed (hepatitis B surface antigen, rabies, herpes simplex virus and others). Details and procedures of DNA vaccines and DNA recombination vaccines are beyond the scope of this course.

A number of chimera vaccines are now available (foot-and-mouth-disease, malaria, porcine circovirus type 2, tetravalent dengue virus vaccine chimerized onto yellow fever vaccine virus). The recombinant canarypox vectored vaccine undergoes abortive replication in nonavian cells and with only a small part of the genetic material of the canarypox introduced, it will remain harmless to the host. This has been proven in a number of different species of host animal. Canarypox-vectored vaccines include vaccines against rabies, equine influenza virus, West Nile virus, canine distemper and feline leukemia virus.
Elite

**Coggins Test (Agar-Gel Immunodiffusion: AGID)**

<table>
<thead>
<tr>
<th>Step</th>
<th>Material</th>
<th>Process</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Petri dish (100 mm ⊙)</td>
<td>Add 15-17 ml of 1 percent Noble agar in 0.145 M borate buffer, pH 8.6±0.2. Punch six wells 5.3 mm around same size center well, 2.4 mm apart.</td>
</tr>
<tr>
<td>2</td>
<td>Viral antigen</td>
<td>Place in center well</td>
</tr>
<tr>
<td>3</td>
<td>Serum samples, controls</td>
<td>Place serum sample and control serum in alternate well around center</td>
</tr>
<tr>
<td>4</td>
<td>Incubator</td>
<td>At room temperature 24 to 48 hours (humid environment)</td>
</tr>
<tr>
<td>5</td>
<td>Intense oblique light beam</td>
<td>Black background, observe and if positive, find precipitation lines between AG and AB.</td>
</tr>
</tbody>
</table>

*If strongly positive, precipitation line is thick/diffuse and visible in 24 hours; weak it may take two days.*

**Tentative response to imminent outbreak**

<table>
<thead>
<tr>
<th>Step</th>
<th>Observation/Process</th>
</tr>
</thead>
<tbody>
<tr>
<td>Suspicion of infectious respiratory disease</td>
<td>Fever, cough, lymphadenopathy, abnormal nasal discharge, abnormal respiratory sounds, urticaria, limb edema.</td>
</tr>
<tr>
<td>Establish primary biosecurity perimeter and place signs</td>
<td>From center point (infected animal) outward to first available physical barrier and effective separation or isolation from other noncontact animals within range of 50 ft (influenza aerosol spread); no traffic in or out; stop all horse movement.</td>
</tr>
<tr>
<td>Move infected animal to isolation pen</td>
<td>Re-assign isolation pen primary biosecurity perimeter; keep initial biosecurity perimeter as well until proven cleared.</td>
</tr>
<tr>
<td>Monitor disease progress</td>
<td>Rectal temperatures 2x per day; clinical signs and symptoms.</td>
</tr>
<tr>
<td>Diagnostic laboratory testing</td>
<td>Virus isolation; ELISA, FA testing; PCR; bacterial culture.</td>
</tr>
<tr>
<td>Establish secondary biosecurity perimeter</td>
<td>Antibody: neutralization; hemagglutination inhibition, ELISA.</td>
</tr>
<tr>
<td>Moving animals</td>
<td>Must provide detailed health certificate and diagnostic test results with every animal to be moved to satisfy recipient facilities and jurisdictions.</td>
</tr>
<tr>
<td>Facility control during and after outbreak</td>
<td>Barrier control, waste and rodent control; disinfection procedures.</td>
</tr>
<tr>
<td>Informing the public; groups of concern</td>
<td>Risk assessment; import/export requirements.</td>
</tr>
</tbody>
</table>

*NB: Make sure to have trained staff to deal with and examine suspect animals; bar access by outsiders or nonessential personnel; record animal and personnel movement before and during outbreak; keep records detailed and to the point.*

**Inactivated or killed vaccines** do not contain live contagious pathogens and cannot spread the virus. However, because the virus does not multiply and amplify the antigenic mass necessary to stimulate an effective immune response, it must often be concentrated and usually provided with adjuvants like aluminum salts, mineral oils, and mycobacterial products such as Freud’s adjuvants that will modulate, stimulate and reinforce the antigenicity of the vaccine.

These vaccines can consist of the whole organisms inactivated by formaldehyde, phenol, beta-propiolactone or similar. They can consist of the antigenic components of the organism, the broken-down or purified antigenic protein, with usually fewer side effects or local reactions, and subunit vaccines, consisting of defined and often highly purified subunit vaccines (foot-and-mouth-disease, influenza A and B, hepatitis B surface antigen) and toxoids (tetanus, diphtheria toxoids) and recombinant subunit vaccines, synthetically produced antigens (hepatitis B vaccine). Highly purified subunit vaccines may well stimulate a humoral antibody response, but unless provided with T-cell epitopes to stimulate T-helper cells, the cellular immunity may not develop.

Advantages of a subunit vaccine include that it can be produced without virus cultivation, that it is cleaner, has no other viral proteins or contaminants, i.e., produces fewer side reactions, and is neither contagious nor oncogenic. Disadvantages are lower immunogenicity, a need for adjuvants (likelihood for side-reactions there), a need for multiple doses and regular boosters, and a lack of cell-mediated immunity.

Anti-idiotyp antibodies are produced with antigens generated from the antigenic images in antibodies elicited by the original antigen. This is an effort to produce vaccines from antigens too hazardous or difficult to grow. Examples of vaccines produced by this procedure are hepatitis B virus, rabies, Newcastle disease virus and feline leukemia, reoviruses and poxviruses.

Any vaccination program must involve the considerations of which and how many animals of a herd to vaccinate, at what age, any particular season of the year, breeding status of the individual, and the effect on herd immunity. Suggestions for procedures to limit and control respiratory disease outbreaks are presented in the following table.

### Vaccines and vaccination schedules

The following procedures are based on recommendations provided by the American Association of Equine Practitioners about their core vaccines. They include eastern equine encephalitis, western equine encephalitis, West Nile virus, rabies and tetanus vaccines.

Vaccination for eastern and western equine encephalitis should be a must for horses travelling in or through endemic areas, especially in view of the high mortality rates for these diseases. EEE is deadly for close to 90 percent of the infected horses, while WEE kills one of every two infected horses. The vaccines available are formalin inactivated, containing an adjuvant to modulate and broaden antigenicity of the product. They produce excellent protection against intracranial challenge injections and have cross-protecting qualities against Venezuelan equine encephalitis. There also is an inactivated vaccine against VEE (The modified live virus vaccine for VEE has shown serious side effects and has been implicated in human fetal deaths). A three-way vaccine includes tetanus along with EEE and WEE. The “four-way-shot” consists of EEE, WEE, tetanus and Influenza.

Among the arbovirus encephalitic diseases, West Nile virus is important because in addition to horse encephalitis cases, it shows an increased incidence of human infections. It is present all over the United States, Canada, Mexico and parts of South America and is deadly for one out of three infected horses.

There are three licensed vaccines for West Nile virus:

- A whole virus vaccine, inactivated and adjuvanted. It requires two priming doses three to six weeks apart and yearly booster inoculations.
- A canarypox vector vaccine, a recombinant adjuvanted vaccine that acts by inserting antigenic properties into the host cell with viral replication. After two priming intramuscular injections three to six weeks apart, it requires yearly boosters. It can be given following the use of the other vaccines.
- A modified live chimera vaccine that consists of a live apathogenic virus carrier of the antigenic properties of West Nile virus. It requires only a single priming dose followed by yearly revaccination.
Rabies is another one of the core vaccines. Although the rabies incidence in horses is relatively low, it is always fatal. Once the virus reaches the local nerve endings, it is protected from the body’s immune system, will migrate through the nerves to the brain and cause rapidly progressive deadly encephalitis. That explains the need for quick action when a human being is involved.

There are three inactivated vaccines available. All are produced in rabies virus-infected vero cell lines and inactivated with beta-propiolacton. The vaccine is given via intramuscular inoculation and is highly immunogenic. A single dose is usually sufficient.

Post exposure treatment in man requires the thorough cleansing of the wound with ample soap and water, the administration of 20 IU/kg and 40 IU/kg bodyweight of anti-rabies serum or gammaglobulin along with five intramuscular doses of rabies vaccine given on days 0, 3, 7, 14, 30.

The forth core vaccine is tetanus, which is caused by the anaerobic Clostridium tetani. It is not a contagious disease but result of the fatal neurotoxin produced by that organism when embedded in deep wounds, removed from access to air. Tetanus toxoid, formalin-inactivated and

---

### Eastern/Western Equine Encephalitis Combination Vaccine (formalin inactivated)

<table>
<thead>
<tr>
<th>Animal</th>
<th>Vaccine</th>
<th>Initial vaccination</th>
<th>Booster vaccination</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adult (vaccinated)</td>
<td>Formalin inactivated</td>
<td></td>
<td>Yearly; before vector season¹</td>
</tr>
<tr>
<td>Adult (never vaccinated)</td>
<td>Formalin inactivated</td>
<td>2 doses, 4-6 weeks apart</td>
<td>Yearly; before vector season.</td>
</tr>
<tr>
<td>Pregnant mare (vaccinated)²</td>
<td>Formalin inactivated</td>
<td></td>
<td>4-6 weeks to foaling or before vector season.</td>
</tr>
<tr>
<td>Pregnant mare (unvaccinated)²</td>
<td>Formalin inactivated</td>
<td>2 doses, 4 weeks apart</td>
<td>4-6 weeks to foaling or before vector season.</td>
</tr>
<tr>
<td>Foal of vaccinated mare</td>
<td>Formalin inactivated</td>
<td>2 doses, 4-6 weeks apart</td>
<td>10-12 months of age or before vector season.</td>
</tr>
<tr>
<td>Foal of unvaccinated mare</td>
<td>Formalin inactivated</td>
<td>2 doses, 4-6 weeks apart</td>
<td>10-12 months of age or before vector season.</td>
</tr>
<tr>
<td>Foals (south): 2-3 months old³</td>
<td>Formalin inactivated</td>
<td>3 doses, 4 weeks apart</td>
<td>10-12 months of age or before vector season.</td>
</tr>
</tbody>
</table>

¹ Risk situations may require additional doses; ² Recommend to vaccinate mares while open; ³ earlier mosquito seasons; NB.: Horses recovered from disease are immune for life, unless immune-compromised.

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### West Nile Virus Vaccine (inactivated whole virus, canarypox vector, MLV chimera)

<table>
<thead>
<tr>
<th>Animal</th>
<th>Vaccine</th>
<th>Initial vaccination</th>
<th>Booster vaccination</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adult (vaccinated)</td>
<td>Inactivated whole virus</td>
<td>2 doses, 4-6 weeks apart</td>
<td>Yearly; before vector season.</td>
</tr>
<tr>
<td>Adult (less than 5 years of age)</td>
<td>Inactivated whole virus</td>
<td></td>
<td>Half-yearly to yearly; before vector season.</td>
</tr>
<tr>
<td>Adult (less than 15 years of age)</td>
<td>MLV Chimera</td>
<td>Single dose</td>
<td>Yearly; before vector season.</td>
</tr>
<tr>
<td>Adult (never vaccinated)</td>
<td>Canarypox vector</td>
<td>2 doses, 4-6 weeks apart</td>
<td>Yearly; before vector season.</td>
</tr>
<tr>
<td>Adult (less than 5 months of age)</td>
<td>MLV Chimera</td>
<td>Single dose</td>
<td>Yearly; before vector season.</td>
</tr>
<tr>
<td>Pregnant mare (vaccinated)</td>
<td>Inactivated whole virus</td>
<td>2 doses, 4-6 weeks apart</td>
<td>Yearly; before vector season.</td>
</tr>
<tr>
<td>Pregnant mare (unvaccinated)</td>
<td>Inactivated whole virus</td>
<td>2 doses, 4-6 weeks apart</td>
<td>Yearly; before vector season.</td>
</tr>
<tr>
<td>Pregnant mare (unvaccinated)</td>
<td>Canarypox vector</td>
<td>2 doses, 4-6 weeks apart</td>
<td>Yearly; before vector season.</td>
</tr>
<tr>
<td>Pregnant mare (vaccinated)</td>
<td>MLV Chimera</td>
<td>Single dose</td>
<td>Yearly; before vector season.</td>
</tr>
<tr>
<td>Foal of vaccinated mare</td>
<td>Inactivated whole virus</td>
<td>2 doses, 4-6 weeks apart</td>
<td>10-12 months of age; before vector season.</td>
</tr>
<tr>
<td>Foal of vaccinated mare</td>
<td>Canarypox vector</td>
<td>2 doses, 4-6 weeks apart</td>
<td>10-12 months of age; before vector season.</td>
</tr>
<tr>
<td>Foal of vaccinated mare</td>
<td>MLV Chimera</td>
<td>Single dose</td>
<td>10-12 months of age; before vector season.</td>
</tr>
<tr>
<td>Foal of unvaccinated mare</td>
<td>Inactivated whole virus</td>
<td>2 doses, 4-6 weeks apart</td>
<td>10-12 months of age; before vector season.</td>
</tr>
<tr>
<td>Foal of unvaccinated mare</td>
<td>Canarypox vector</td>
<td>2 doses, 4-6 weeks apart</td>
<td>10-12 months of age; before vector season.</td>
</tr>
<tr>
<td>Foal of unvaccinated mare</td>
<td>MLV Chimera</td>
<td>Single dose + second</td>
<td>10-12 months of age; before vector season.</td>
</tr>
</tbody>
</table>

Recommendation is to vaccinate mares while open; * or shorter if first dose during mosquito season (3-4 weeks). Horses recovered from disease are immune for life, unless immune-compromised; Risk situations may require additional doses

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### Rabies (inactive; cell culture product)

<table>
<thead>
<tr>
<th>Animal</th>
<th>Vaccine</th>
<th>Initial vaccination</th>
<th>Booster vaccination</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adult (vaccinated)</td>
<td>2 ml im or sc</td>
<td></td>
<td>Yearly</td>
</tr>
<tr>
<td>Adult (never vaccinated)</td>
<td>2 ml im or sc</td>
<td>Single dose</td>
<td>Yearly</td>
</tr>
<tr>
<td>Pregnant mare (vaccinated)¹</td>
<td>2 ml im or sc</td>
<td></td>
<td>4-6 weeks before foaling</td>
</tr>
<tr>
<td>Pregnant mare (unvaccinated)³</td>
<td>2 ml im or sc</td>
<td></td>
<td>4-6 weeks before foaling</td>
</tr>
<tr>
<td>Foal of vaccinated mare</td>
<td>2 ml im or sc</td>
<td>6 months of age</td>
<td>4-6 weeks later; then yearly</td>
</tr>
<tr>
<td>Foal of unvaccinated mare</td>
<td>2 ml im or sc</td>
<td>3-4 months of age</td>
<td>Yearly</td>
</tr>
<tr>
<td>Vaccinated horse exposed to the virus</td>
<td>2 ml im or sc</td>
<td>Revaccinate at once; observe for 45 days for symptoms</td>
<td></td>
</tr>
</tbody>
</table>

¹ recommend to vaccinate before breeding

Non-vaccinated horse exposed to the virus: euthanize immediately or veterinary supervision for six(6) months

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Rabies is another one of the core vaccines. Although the rabies incidence in horses is relatively low, it is always fatal. Once the virus reaches the local nerve endings, it is protected from the body’s immune system, will migrate through the nerves to the brain and cause rapidly progressive deadly encephalitis. That explains the need for quick action when a human being is involved.
combined with adjuvants will stimulate antibody production and protection from the neurotoxin. Different areas, climates, regions, different populations have different needs and requirements. While the core based vaccines should be given to all horses in the United States, risk-based vaccination guidelines include the following viral diseases: equine herpesvirus (rhinopneumonitis), equine viral arteritis, equine influenza and rotaviral diarrhea. Strangles, although being a bacterial disease, is included as an essential part of the equine respiratory disease complex.

**Equine herpesvirus type 1 and type 4** both cause respiratory disease, but type 1 will also produce epidemic abortions, stillbirths and nonviable foals and, occasionally, myeloencephalopathy. Frequently latent, as expected from herpesviruses, it may lead to occasional recurrence following stress situations and infect other horses in the group. There are inactivated vaccines licensed for use against only the respiratory disease caused by the virus. They are somewhat less antigenic than vaccines licensed for both the respiratory disease and abortions. A modified live equine herpesvirus type 1 produces a quicker and more effective response. Vaccination does not guarantee protection from the abortive or neurological form of the disease.

**Equine viral arteritis** causes abortions and death of young foals. Breeding stallions may become carriers, with the virus persisting in the reproductive tract of the male, and pass on the virus. The apparent increase of the disease is blamed on greater global movement of horses, considered safe and effective for stallions and the nonpregnant mare, although there may be a mild fever following the initial vaccination. Vaccination is intended to protect stallions from infection and from becoming a carrier, to immunize seronegative mares and to stop short the occasional outbreak of disease. All animals to be vaccinated should be checked for antibody at or before vaccination to confirm that the antibody found after vaccination was not due to pre-existing disease. This is critically important for animals to be moved, sold or exported. To minimize or mitigate likely outbreaks of equine viral arteritis in a nonbreeding population, animals should be vaccinated yearly. Animals considered for future breeding or sale or exportation must be certified seronegative before or at the time of the initial vaccination. The **equine influenza** respiratory disease is common, indeed endemic in horse populations worldwide (except New Zealand and Iceland). It appears sporadically in a group of horses, introduced by an infected animal, and disappears with the developing immune response in the herd. Two-week quarantine of new entries into the herd helps prevent the occurrence. So does preventive vaccination. Frequent exposure to other horses at horse shows, the racetrack and other aggregation of horses are likely sources for contact and infection. The high infectiousness of the virus and dissemination by aerosol droplets results in a very efficient system of transmission. Partially immune animals may not exhibit symptoms but still shed the virus and infect other horses. Herd immunity will wane as virus exposure decreases due to the prevalent herd immunity. Simultaneously, antigenic drift will make sure that new influenza virus strains will be selected and move in adapting to the prevalent herd immunity. Equine influenza vaccines, therefore, should be updated regularly to include the more recent strains of virus developing in the Americas and Eurasia. There are inactivated vaccines, representing a combination of circulating strains. As a rule, these vaccines require two or three initial doses about four to six weeks apart and a booster six months later or at 10 or 12 months of age. A booster four to six weeks before foaling would
Equine Influenza (Inactivated, Modified Live Virus, Canarypox Vector Vaccine)

<table>
<thead>
<tr>
<th>Animal</th>
<th>Vaccine</th>
<th>Initial vaccination</th>
<th>Booster vaccination</th>
</tr>
</thead>
<tbody>
<tr>
<td>Show, performance, traveling horse</td>
<td></td>
<td></td>
<td>Every 6 months.</td>
</tr>
<tr>
<td>Adult (regular, vaccinated)</td>
<td></td>
<td></td>
<td>Yearly.</td>
</tr>
<tr>
<td>Adult (non-vaccinated)</td>
<td>MLV</td>
<td>1 dose intranasal</td>
<td>Half-yearly to yearly (depends on risk).</td>
</tr>
<tr>
<td></td>
<td>Inactivated</td>
<td>2 doses: 3-4 weeks apart</td>
<td>3rd 3-6 months later; then half-yearly to yearly.</td>
</tr>
<tr>
<td></td>
<td>Canarypox</td>
<td>2 doses: 4-6 weeks apart</td>
<td>Every 6 months.</td>
</tr>
<tr>
<td>Pregnant mares (vaccinated)</td>
<td>Inactivated</td>
<td></td>
<td>4 to 6 weeks before foaling.</td>
</tr>
<tr>
<td></td>
<td>Canarypox</td>
<td></td>
<td>4 to 6 weeks before foaling.</td>
</tr>
<tr>
<td>Pregnant mares (non-vaccinated)</td>
<td>Inactivated</td>
<td>2 doses: 4-6 weeks apart</td>
<td>3rd dose 4 to 6 weeks before foaling.</td>
</tr>
<tr>
<td></td>
<td>Canarypox</td>
<td>2 doses: 4-6 weeks apart</td>
<td>2nd dose not later than 4 weeks before foaling.</td>
</tr>
<tr>
<td>Foals of vaccinated mare</td>
<td>MLV</td>
<td>1 dose: intranasal</td>
<td>10-12 months of age and yearly.</td>
</tr>
<tr>
<td></td>
<td>Inactivated</td>
<td>2 doses: 4-6 weeks apart</td>
<td>10-12 months of age and yearly.</td>
</tr>
<tr>
<td>Foals of non-vaccinated mare</td>
<td>MLV</td>
<td>1 dose: intranasal</td>
<td>10-12 months of age and yearly.</td>
</tr>
<tr>
<td></td>
<td>Inactivated</td>
<td>2 doses: 4-6 weeks apart</td>
<td>3rd dose at 6 months of age; then yearly.</td>
</tr>
<tr>
<td>Outbreak in vaccinated horses</td>
<td>Any</td>
<td>1 dose each.</td>
<td></td>
</tr>
<tr>
<td>Outbreak in nonvaccinated horses</td>
<td>MLV</td>
<td>1 dose, intranasal each.</td>
<td></td>
</tr>
</tbody>
</table>

tend to increase maternal antibody protecting the newborn foal.

A modified live, cold-adapted virus vaccine, which can be given intranasally, provides a relatively quick immune response within seven days and lasts for six months to a year. It should not be given to pregnant mares. It can be given following primary vaccination with inactivated vaccine to deepen and widen immunity. A Canarypox vector vaccine is also available, which after two initial doses requires six-monthly boosting. It seems to boost antibody responses even in younger foals that may still be with maternal antibody.

Rotaviral diarrhoea is caused by a rotavirus (family: reoviridae, subfamily: Sedoreovirinae) and is responsible for more than half of all foal diarrhoea in many areas. More than two-thirds of the United States foal population will have had at least one episode of diarrhoea by the time they are weaned. Vaccination of mares will result in the production of maternal antibody and significant reduction of rotavirus diarrhoea incidence and severity. The rotavirus vaccine is inactivated Group A virus. Only pregnant mares require vaccination to protect the newborn foal with its maternal and colostral antibody.

References and suggested reading

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Rotaviral Diarrhea (inactivated Group A Virus) Vaccine

<table>
<thead>
<tr>
<th>Animal</th>
<th>Vaccination</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pregnant mares</td>
<td>3 doses, im</td>
<td>8th, 9th, 10th month of gestation.</td>
</tr>
<tr>
<td>Newborn foals</td>
<td>Not necessary.</td>
<td></td>
</tr>
</tbody>
</table>

When colostral antibody wanes, foal may still become sick but less so and for shorter period.


EQUINE VIRAL DISEASES
Final Examination Questions
Choose True or False for questions 1-15 then complete your test online at www.elitecme.com.

1. The equine respiratory disease complex represents a group of the most common and most important diseases in horses.
   True    False

2. Equine herpes virus type 1 causes respiratory disease, neurological symptoms such myeloencephalopathy, abortion and neonatal death.
   True    False

3. Infection with equine herpes virus type 4 will produce long-term immunity in a horse.
   True    False

4. The highly contagious nature of equine influenza guarantees that any outbreak will spread rapidly through a susceptible population of equids.
   True    False

5. A horse infected by the equine influenza virus is protected from bacterial complications.
   True    False

6. The lower antibody response following the equine rhinitis B virus infection results in more frequent, usually seasonal, re-infection.
   True    False

7. An emerging disease, Hendra virus is a zoonotic virus that causes disease in man, developing into a respiratory disease with severe influenza-like symptoms, often followed by progressive encephalitis and death.
   True    False

8. Animals that survive strangles usually have only a few months of immunity from the condition after recovery.
   True    False

9. Death of a horse after infection from the eastern equine encephalitis usually occurs three weeks after onset.
   True    False

10. A number of vaccines exist for alphaviruses.
    True    False

11. An animal with the Venezuelan equine encephalitis virus may exhibit progressive disease of the central nervous system or it may die without any prodromal symptoms.
    True    False

12. In pigs, the Japanese encephalitis virus interferes with normal reproductive processes, producing abortion, stillbirth and mummification of fetuses.
    True    False

13. Rotaviral diarrhea is responsible for half or more of all foal diarrheas.
    True    False

14. Re-vaccination is discouraged before events of stress, travel, and entering areas with high population density (breeding farms, boarding stables, show barns and race tracks).
    True    False

15. Vaccination for West Nile virus is important because in addition to horse encephalitis cases, it shows an increased incidence of human infections, and is deadly for one out of three infected horses.
    True    False