GUIDELINES FOR VACCINATION OF HORSES

Guidelines for use of vaccines in horses are intended to be a point-of-reference, or a framework, to direct specific activities of veterinarians as they employ vaccines in their respective practices. These guidelines are neither regulations nor directives for all situations and should not be interpreted as such. It is the responsibility of attending veterinarians, through an appropriate veterinarian-client-patient relationship, to utilize this information coupled with available products to determine the best professional care for their patients. It is impractical to recommend a “standard” vaccination program for all horses because each individual situation must be evaluated based on risk of disease (anticipated exposure, environmental factors, geographic factors, age, breed, use, and sex of the horse), potential for adverse reactions to a vaccine(s), anticipated efficacy of the selected product(s), and cost. Cost should include expenses incurred due to time out of competition, labor and medication if or when horses develop clinical disease and need treatment, as well as expenses of time, labor and vaccine(s) to properly immunize the horses.

Programs for the control of infectious diseases are important components of good managerial practices directed toward maximizing the health, productivity, and performance of horses. Infectious disease in an individual horse or outbreaks of infection within a group of horses occur when sufficient quantity of an infectious agent overcomes the resistance acquired through prior natural exposure to the disease or through vaccination. Thus, programs for the control of infectious diseases should be directed toward 1) reducing the exposure to infectious agents in the horses’ environment (i.e., challenge), 2) minimizing factors that diminish resistance, and 3) enhancing resistance to those diseases by vaccination. The incidence of infectious diseases in populations of horses tends to increase with increased number and concentration of susceptible horses at a facility, movement of horses on and off the facility, or with external environmental and managerial influences.

Conditions on breeding farms, in sales or boarding facilities, in barns of performance and show horses, or at racetracks are often ideal for introduction and transmission of infectious diseases, particularly infections of the respiratory tract. On breeding farms, introduction of horses from various origins, commingling of horses of different ages, and the high proportion of susceptible horses pose special problems and serve to demonstrate some important considerations in the practice of disease control. Managerial practices that reduce risks of exposure to infectious diseases, coupled with appropriate use of vaccines will, in time, lower the incidence or severity of infectious diseases. Different degrees of risk will result in different recommendations for vaccination. If managerial changes can not or will not be implemented to optimize control of infectious disease, vaccination alone can not be expected to be successful.

The client should have realistic expectations and understand that 1) vaccination serves to minimize the risk of infection but does not prevent disease in all circumstances; 2) the primary series of vaccines and booster doses should be appropriately administered prior to likely exposure; 3) each horse in a population is not protected to an equal degree nor for an equal duration following vaccination; and 4) all horses in a herd should be appropriately vaccinated, and whenever possible, the same schedule should be followed. This practice will simplify record keeping, minimize replication and transmission of infectious agents in the herd, and optimize herd-immunity by protecting those horses in the herd that responded poorly to vaccination. A properly administered, licensed product should not be assumed to provide absolute effective protection during any given field epidemic. Copies of the vaccination and health maintenance records should accompany horses entering or
leaving sales, training, or breeding facilities. Similarly, owners of equine facilities should establish prerequisites for vaccination of all horses entering the facility and request that copies of their vaccinal records accompany those horses. Horses should be appropriately vaccinated at least one month before entering or leaving such a facility in order to produce adequate antibodies before the anticipated exposure.

Only federally licensed products should be used and strict attention should be afforded the manufacturer’s recommendations regarding storage, handling, and routes of administration of the vaccine to maximize efficacy and safety. However, results of research or clinical experience may support alternate protocols for vaccination that will improve the efficacy of a vaccine without increasing adverse effects. Protection is not afforded the patient immediately after administration of a vaccine that is designed to induce active immunity. In most instances a series of multiple doses of an inactivated vaccine must be administered initially for that vaccine to induce protective active immunity. Two to three weeks are required to produce adequate concentrations of antibodies and before booster revaccinations can be protective. Foals with residual maternal antibodies generally produce a greater serologic response when an initial series of 3 doses is administered rather than the 2-dose series recommended by most manufacturers of vaccines for older horses without residual maternal antibodies.

It is important to vaccinate broodmares 4 to 6 weeks before foaling for their own protection, as well as to maximize concentrations of immunoglobulins in their colostrum to be passively transferred to their foals. Simply vaccinating the mare is not sufficient. The foal must receive adequate amounts of high quality colostrum and absorb adequate amounts of specific colostral immunoglobulins before absorption of macromolecules ceases (generally 24 to 48 hours). Specific colostral immunoglobulins provide protection against field infections for several months but also may interfere with vaccinal antigens and prevent the active immunologic response by the foal; a phenomenon termed “maternal antibody interference.” Although protective concentrations of antibody decline with time, vaccination of a foal while these colostral antibodies are present - even at concentrations less than those considered to be protective - is of minimal to no value because of maternal antibody interference. Consequently, that foal may be susceptible to infection before the primary vaccinal series can be completed.

After receiving a vaccine(s) intramuscularly, some horses experience local muscular swelling and soreness or transient, self-limiting signs including fever, anorexia and lethargy. Severe reactions at sites of injection can be particularly troublesome requiring prolonged treatment and convalescence. These adverse reactions are not always predictable but are part of the inherent risks of vaccination. It is, therefore, recommended that horses not be vaccinated within 2 weeks before shows, performance events, sales or domestic shipment. Some veterinarians may elect not to vaccinate horses within 3 weeks of international shipment. That allowance of time should be sufficient for 1) production of antibodies to the vaccine prior to the time of major susceptibility; and 2) recovery from unexpected adverse reactions that might otherwise interfere with the horse’s performance or health prior to, or during, shipment.

Though uncommon, the possibility always exists for adverse reactions (including anaphylaxis) associated with administration of a vaccine; therefore, vaccines should be administered by, or under the direct supervision of a veterinarian. Adverse reactions should be reported to the vaccine’s manufacturer, and may also be reported to the USDA (1-800-752-6255) or the USP Veterinary Practitioners Reporting Program (Forms may be obtained or reports submitted by calling USP at 1-800-487-7776).
Vaccines are currently available in North America to aid in the prevention of the following equine infectious diseases: tetanus; eastern, western, and Venezuelan equine encephalomyelitis; equine influenza; equine herpesvirus-1 and equine herpesvirus-4 infection (rhinopneumonitis); strep (Streptococcus equi) infection; rabies; equine monocytic ehrlichiosis (Potomac horse fever); toxicoinfectious botulism; equine viral arteritis; anthrax; or rotaviral diarrhea. General guidelines for use of the most frequently indicated equine vaccines under various managerial conditions and in various geographic locations are provided in this report.

**Tetanus**

Tetanus is an often fatal disease caused by a potent neurotoxin elaborated by anaerobic, spore-forming bacteria, *Clostridium tetani*. These organisms are present in the intestinal tract and feces of horses, other animals, and human beings, and are abundant in soil. Spores of *Cl. tetani* can live in the environment for many years, resulting in frequent exposure of horses and people on equine farms. The disease is not contagious but occurs when *Cl. tetani* infects puncture wounds (particularly those involving a foot or muscle), open lacerations, surgical incisions, or exposed tissues such as the umbilicus of foals. All horses should be vaccinated against tetanus by use of tetanus toxoid to induce active immunity. Available vaccines are formalin-inactivated, adjuvanted toxoids that are safe, and induce long-lasting immunity.

Adult nonvaccinated horses respond well to an initial series of 2 doses of tetanus toxoid administered 3 to 6 weeks apart, followed by an annual booster. Protective concentrations of immunoglobulin are usually attained within 14 days of the second injection and may persist for up to 5 years. Concentrations of anti-tetanus antibodies in colostrum from non-vaccinated mares are not predictable. For that reason, the annual booster for pregnant mares should be administered 4 to 6 weeks before foaling to protect the mare if she sustains foaling-induced trauma, and to enhance concentrations of immunoglobulins in her colostrum. Colostrum-derived antibodies significantly interfere with the immune response by the foal if it is vaccinated with tetanus toxoid before it is about 6 months-of-age. If a foal received appropriate transfer of colostral antibodies from a vaccinated mare, that foal should receive its primary series of three (3) doses of tetanus toxoid, at 4- to 6-week intervals, beginning at 6-months-of-age or older. Foals born to nonvaccinated mares or mares with unknown vaccinal history, should receive this initial 3-dose series of toxoid, at 3- to 6-week intervals, starting at 3 to 4 months-of-age followed by an annual booster. The 3-dose initial series produced a more consistent anamnestic response and is currently recommended in preference to the 2-dose series previously used. Extension of the annual interval for revaccination is not currently recommended pending supporting documentation of duration of immunity. Vaccinated horses that sustain a wound or undergo surgery more than 6 months after their previous tetanus booster should be revaccinated with tetanus toxoid.

Tetanus antitoxin is produced by hyperimmunization of donor horses with tetanus toxoid. Administration of one vial of antitoxin (1,500 IU) to a nonvaccinated horse induces immediate passive protection that usually lasts only 2 to 3 weeks. Protection lasting up to 6 weeks can be accomplished with higher doses. A small, but significant, number of horses experience serum sickness and fatal hepatic failure (serum hepatitis) several weeks after receiving tetanus antitoxin. Indications for administration of tetanus antitoxin include the nonvaccinated horse that has sustained an injury or the foal born to a nonvaccinated mare. In those instances, tetanus antitoxin and tetanus toxoid should be administered concurrently, with separate syringes in different sites. Second and third (if
the patient is a young horse from a nonvaccinated mare) doses of toxoid should be administered at 4- to 6-week intervals to complete the primary series. Thereafter, annual boosters with tetanus toxoid are recommended.

**Equine Encephalomyelitis (Sleeping Sickness)**

In the United States, equine encephalitides for which vaccines are available include eastern equine encephalomyelitis (EEE), western equine encephalomyelitis (WEE) and Venezuela equine encephalomyelitis (VEE). Outbreaks of WEE have been recorded in the western and mid-western states, with sporadic cases reported in the northeast and southeast. The distribution of EEE has historically been restricted to the eastern, southeastern and some southern states. Venezuelan equine encephalomyelitis occurs in South and Central America but has not been diagnosed in the United States for more than 20 years. An outbreak of VEE in southern Mexico in 1993 prompted recommendations to vaccinate horses residing in Mexico and in the USA within 40 miles of the US-Mexican border (California, Texas, New Mexico, and Arizona).

The viruses that cause encephalomyelitis are transmitted by mosquitoes, and infrequently by other bloodsucking insects, to horses from wild birds or rodents, which serve as natural reservoirs for these viruses. Human beings are also susceptible to these diseases when the virus is transmitted to them by infected mosquitoes; however, horse-to-horse or horse-to-human transmission by mosquitoes is highly unlikely, because the amount of virus in the blood of horses affected by EEE or WEE is small. The viremia that occurs with VEE is higher and direct horse-to-horse or horse-to-human transmission is possible. Of these 3 encephalidities, WEE has the lowest mortality (approx. 50%). Eastern equine encephalomyelitis is the most virulent for horses, with mortality approaching 90%. Venezuelan equine encephalomyelitis is also lethal, but some horses apparently develop subclinical infection, resulting in lasting immunity. Risk of exposure and geographic distribution of these encephalomyelitides vary from year-to-year with changes in distribution of insect vectors and reservoirs of the respective viruses. It is recommended that all horses in North America be immunized against WEE and EEE, because of the unpredictable nature of those factors and the effects of the disease. Horses should be properly vaccinated against VEE when outbreaks approach or when residing in areas where there is a risk of exposure to the disease. Horses traveling to areas where VEE occurs should be appropriately vaccinated before departure.

Vaccination with one of several available inactivated bivalent (WEE/EEE) or trivalent (WEE/EEE/VEE) vaccines provides effective control of these diseases. While adult horses may respond well to two doses of vaccine, primary vaccination of foals involves administration of 3 doses of vaccine. Annual revaccination is appropriately completed in the spring, prior to the peak season of the insect vector. Booster vaccination of pregnant mares 4 to 6 weeks before foaling enhances colostral concentrations of immunoglobulins that generally protect their foals against these diseases for 6 to 7 months. Primary vaccination of foals born late in the foaling season may be delayed until the following spring in climates where mosquitoes die during the winter; otherwise, foals should be vaccinated starting at 6 months-of-age in order to avoid maternal antibody interference. Foals should receive an initial series of three (3) doses of killed-virus vaccine administered 4 to 6 weeks apart and should be revaccinated at 1 year-of-age to ensure adequate protection. Because of the high mortality associated with EEE, foals born in areas where there is a high risk of exposure to EEE should be vaccinated beginning at 4-months-of-age. An initial series of three doses of vaccine against EEE should be administered at 4- to 6-week intervals, followed by a fourth dose at one year-of-age. In
states where mosquitoes are active year-round, many veterinarians prefer to vaccinate horses semiannually (at 6-month intervals) to ensure uniform protection throughout the year, although this practice is not specifically recommended by manufacturers of vaccines. Annual vaccination against encephalomyelitis can be combined with tetanus toxoid or other inactivated antigens, where appropriate, using polyvalent vaccines.

**Equine Influenza**

Equine influenza is one of the most common infectious diseases of the respiratory tract of horses. It is endemic in the equine population of the United States and throughout much of the world, with the notable exception of Australia and New Zealand. Rapid national and international transportation of horses facilitates the spread of the virus. The density and movement of horses at breeding farms, racetracks, training facilities, boarding stables, shows, or similar athletic events increases the risk of infection. Equine influenza virus does not constantly circulate, even in large groups of horses, but is sporadically introduced by an infected horse. This epidemiologic finding and the rapid elimination of the virus by the equine immune response suggest that infection can be avoided by preventing entry of the virus into an equine population (ie. by the quarantine of newly arriving horses for at least 14 days), and by appropriate vaccination before exposure.

A number of factors increase the risk of infection by equine influenza virus. The most important factors identified to date are age (horses 1 to 5 years-of-age are more susceptible), serumal concentration of influenza virus-specific antibody, and frequent contact with large numbers of horses. The importance of local mucosal protection is difficult to quantitate by methods currently available. Older horses are generally less susceptible to infection but that protection can be overwhelmed in horses frequently exposed at shows or similar athletic events. Although the disease is endemic in many countries and infection cycles continuously, explosive outbreaks occur at intervals of several years when the immunity of the equine population wanes and sufficient antigenic drift in the virus has occurred to generate a new viral strain.

Equine influenza is highly contagious and the virus spreads rapidly through groups of horses in aerosolized droplets dispersed by coughing. Contaminated buckets, grooming or feeding equipment and tack may serve as fomites because the virus can survive for hours on such objects. Clinical signs of influenza include nasal discharge, fever, lethargy, anorexia, cough, and myalgia. Severity of those signs depends on the degree of existing immunity among other factors. The incubation period may be as short as 1 to 3 days, and infected horses can shed virus for up to 10 days in their nasal secretions. Horses that are partially immune can become subclinically infected and shed virus. Immunity to the same (homologous) strain of virus following natural infection persists for approximately one year, but unlike immunity following vaccination with inactivated vaccines, is not well correlated with results of hemagglutination inhibition (HI) or single radial immunodiffusion (SRH) tests. Those tests measure serumal antibodies against specific strains of equine influenza virus. Immunity following vaccination with inactivated influenza vaccines is short-lived, allowing recently vaccinated horses to become infected and shed virus, thereby contributing to maintenance and spread of infection within the equine population.

It is worthwhile to attempt to definitively diagnose equine influenza viral infection because specific measures can then be initiated to contain the spread of the disease by isolation of infected horses and their immediate contacts. In addition, vaccination to boost immunity in the face of an outbreak may be a practical strategy. Frustrations with conventional techniques for viral isolation can be over-
come by use of a capture ELISA technique that is currently marketed for human use, (Directigen 
FluATM, from Becton Dickinson, Franklin Lakes, NJ; or FLU OIATM, from BioStar, Boulder, CO). 
Those diagnostic tests can be used directly for equine patients and provide results within 15 min-
utes, with a sensitivity similar to that of viral isolation. Those diagnostic procedures are indicated 
when presented with a horse that exhibits compatible clinical signs, particularly if the horse meets 
other high-risk criteria.

Equine influenza is caused by the orthomyxovirus equine influenza A type 2 (A/equine 2). Equine 
influenza A type 1 has not been identified as a cause of natural infection for many years and its in-
clusion in vaccines is no longer considered necessary. Antigenic drift of the A/equine 2 subtype has 
resulted from point mutations in the genes encoding the amino acid sequences of the hemagglutinin 
(HA) and neuraminidase (NA) glycoprotein antigens on the surface of the virus. The result was emer-
gence of viral strains representing two antigenically heterologous lineages, the American and 
Eurasian (H3N8) viral strains. Like the prototypic strain A/equine 2/Miami 63, variants are named 
according to the location and year that they were first isolated. Antigenic drift, by generating anti-
genically heterologous viruses, reduces the degree and duration of protection conferred by previous 
infecion or vaccination. Although antigenic drift of equine influenza virus is slower than that of 
human influenza virus, it is still recommended that equine vaccines contain killed viral antigens 
from isolates obtained within the most recent 5 years, and ideally, representatives of each (American 
and Eurasian) lineage should be included. However, the American lineage (A/equine 2/91) contained 
in the modified live-virus (MLV) intranasal vaccine provided protection when vaccinated horses were 
challenged with Eurasian and American (A/equine 2/98) strains.

In the USA, for manufacturers of vaccines to comply with federal regulations for licensing and mar-
keting of vaccines, any change of a vaccine, such as including the most recently isolated influenza 
virus, usually leads to costly and time-consuming evaluation of the revised product. Consequently, 
viral antigens in all commercially available inactivated equine influenza vaccines were chronologi-
cally behind the antigenic drift of field viruses, resulting in suboptimal protection. In previous years, 
the short-lived immunity following vaccination with inactivated equine influenza vaccines and the 
lag-time for antigenically new strains of virus to be incorporated into vaccines were the impetus for 
recommendations of frequent revaccination (intervals of 2 to 4 months). However, frequent revac-
cination did not necessarily reduce the incidence of the disease and serologic response varied among 
the different vaccines administered.

All horses should be vaccinated against equine influenza unless they live in an isolated facility that 
is totally closed. Adult horses may adequately respond to two doses of killed-virus vaccine. In younger 
horses a primary series with 3 doses of the killed-virus vaccine, administered 3 to 6 weeks apart, 
induced higher and more persistent antibody titers than those induced by use of the previously rec-
ommended 2-dose initial series. Subsequent revaccination should be at intervals of 3 to 12 months, 
depending on the age of the horse as well as the degree and duration of risk of acquiring infection. 
For young horses in competition, revaccination is recommended at intervals of 3 to 4 months until 
they are 2 years-of-age. On breeding farms, all mature horses should be revaccinated on the basis of 
their risk of exposure. Boosters for pregnant broodmares should be administered 4 to 6 weeks before 
foaling using the killed-virus vaccine to maximize concentrations of immunoglobulins in colostrum. 
Foals from vaccinated mares may be protected for several months by antibodies passively transferred 
via colostrum. Those antibodies may also lessen that foal’s immunologic response to any killed-virus 
influenza vaccine administered before 9 months-of-age. Thus, when a killed-virus vaccine is used, 
the program for primary vaccination of foals against influenza, depends on the vaccinal status of the
dam as well as the risk of acquiring infection. Primary vaccination for foals born to vaccinated mares
and kept isolated from exposure to horses from other premises may be delayed until those foals are
9 months-of-age and should consist of a series of 3 doses of killed-virus vaccine administered at
intervals of 4 to 6 weeks. It is currently recommended that primary vaccination of foals, born to non-
vaccinated mares, begins when the foal is 6 months-of-age, using a series of 3 doses of killed-virus
vaccine, administered at intervals of 4 to 6 weeks. Mature performance, show, or pleasure horses
constantly at risk of exposure should be revaccinated at 3- to 6-month intervals.

A modified-live-equine influenza /A2 vaccine is now available for intranasal administration. Studies
have shown the MLV vaccine to be safe and that a single administration to naive horses was protec-
tive for at least 6 months. Circulating antibodies alone did not correlate with protection, suggesting
that local protection at the nasal mucosa may also be enhanced by that vaccine. The product is
licensed for vaccination of non-pregnant animals over 11 months-of-age using a single dose of vac-
cine, followed by boosters at 6-month intervals. There may be an advantage to using a primary series
of two doses at an interval of 3 months. Generally, horses shed vaccinal virus for less than 1 week
after vaccination. But the amount and duration of shed vaccinal virus is so minimal that other hors-
es in contact with them will not be vaccinated. Incorporation of the MLV vaccine into a program rely-
ing on the inactivated vaccine can occur when routine boosters of inactivated vaccine are scheduled.

Preliminary evidence supports the safety of the MLV intranasal vaccine when administered to foals
less than 11 months-of-age. However, data are pending to support efficacy of the product when used
in foals less than 11 months-of-age; therefore, if it is used in younger foals, it is recommended that
a second dose be administered when the foal is 11 months-of-age or older. Maternal antibody inter-
ference and mucosal interference against the MLV vaccine need to be studied further.

**Equine Herpesvirus** (Rhinopneumonitis)

Equine herpesvirus type 1 (EHV-1) and equine herpesvirus type 4 (EHV-4) can each infect the res-
piratory tract causing disease that varies in severity from mild to severe and characterized by fever,
lethargy, anorexia, nasal discharge, and cough. Infection of the respiratory tract with EHV-1 and EHV-
4 is most common in weanlings, yearlings, and young horses entering training, especially when hors-
es from different sources are commingled. Equine herpesvirus 1 causes abortion of virus-infected
fetuses from infected mares, the birth of weak nonviable foals, or a paralytic neurologic disease
(myeloencephalopathy) secondary to vasculitis of the spinal cord and brain. The virus is spread via
aerosolized secretions from infected coughing horses, by direct and indirect (fomite) contact with
nasal secretions, and, in the case of EHV-1, by aborted fetuses, fetal fluids, and placentae associated
with abortions. Like herpesviruses in other species, these viruses can remain latent in the majority
of horses, which do not show clinical signs but may experience recrudescence of infection and shed-
ding of the virus when stressed. Those epidemiologic factors seriously compromise efforts to control
these diseases and explain why outbreaks of EHV-1 or EHV-4 can occur in closed populations of hors-
es. Because both viruses are endemic in most equine populations, most mature horses have devel-
oped some immunity through repeated natural exposure; thus, most mature horses do not develop
serious respiratory disease when they become infected but may be a source of exposure for other
susceptible horses. In contrast, horses are not protected against the abortigenic or neurologic forms
of the disease, even after repeated exposure.

Primary indications for use of equine herpesvirus vaccines include prevention of EHV-1-induced
abortion in pregnant mares, and prevention of respiratory tract disease (rhinopneumonitis) in foals,
weanlings, yearlings, young performance and show horses that are at high risk for exposure. All horses do not produce postvaccinal antibodies against EHV, but the presence of those antibodies does not, in itself, ensure complete protection. Consistent vaccination appears to reduce the frequency and severity of disease and limit the occurrence of abortion storms but unambiguously compelling evidence is lacking. Managerial changes for care of pregnant mares are of primary importance for control of clinical disease caused by EHV. It is recommended that all pregnant mares be vaccinated during the fifth, seventh, and ninth months of gestation using a licensed inactivated EHV-1 vaccine. Many veterinarians also recommend a dose during the third month of gestation and some recommend a dose at the time of breeding. Vaccination of mares with an inactivated EHV-1/EHV-4 vaccine 4 to 6 weeks before foaling is commonly practiced to enhance concentrations of colostral immunoglobulins for transfer to the foal. Barren mares and stallions should be revaccinated before the start of the breeding season and thereafter based on risk of exposure.

Primary vaccination of foals involves administering 3 doses of inactivated EHV-1/EHV-4 vaccine or modified-live EHV-1 vaccine, 3 to 4 weeks apart, beginning at 4 to 6 months-of-age. Even with this regimen, a substantial number of foals fail to seroconvert, presumably due to maternal antibody interference. Immunity following vaccination appears to be short-lived and it is recommended that foals, young horses, and performance or show horses at high risk be revaccinated at 3- to 4-month intervals, as with influenza. The benefit of intensive vaccination programs directed against EHV-1 and EHV-4 in foals and young horses is not clearly defined because, despite frequent vaccination, infection and clinical disease continue to occur. Maternal antibody passively transferred to foals from vaccinated mares may decrease the incidence of respiratory disease in foals, but disease can still occur in those foals. Frequent vaccination of nonpregnant mature horses, except those on breeding farms, with EHV vaccines is generally not indicated. Available vaccines make no labeled claim to prevent the myeloencephalitic form of EHV-1 infection. Vaccines containing EHV-1 appear to offer some protection against infection with EHV-4.

**Streptococcus equi infection** (Strangles)

Strangles (also known as “distemper”) is a highly contagious disease caused by the bacterium *Streptococcus equi* subspecies *equi* (*S. equi*). Strangles primarily affects young horses (weanlings and yearlings), although horses of any age can be infected. The organism is transmitted by direct contact with infected horses or sub-clinical carriers, or indirectly by contact with water troughs, feed bunks, pastures, stalls, trailers, tack, or grooming equipment contaminated with nasal discharge or pus draining from lymph nodes of infected horses. *Streptococcus equi* can survive in the environment for at least 3 months, when it is protected from exposure to direct sunlight and disinfectants, and it can be a source of infection for new additions to the herd. After inhalation or ingestion, *S. equi* induces a severe inflammatory response, which causes upper respiratory discomfort, anorexia, and copious mucopurulent nasal discharge. The bacteria then spread to the lymph nodes around the head, jaw, and throat, causing them to enlarge, abscess, rupture and drain pus. Enlarged lymph nodes may compress the pharynx and trachea, causing swallowing or breathing difficulty, hence the name strangles. Affected horses generally have a fever (102 to 106 F) and are lethargic and anorectic, particularly at the height of fever shortly before abscesses drain.

Exposure to or vaccination against *S. equi* does not provide protection against *Streptococcus zooepidemicus* and vice versa. Inactivated sub-unit (M-protein) vaccines and whole-cell bacterins are available for intramuscular injection as an adjunct to the prevention of strangles. Given the clon-
al nature of the vaccinal bacteria, autogenous bacterins are not advocated. The inactivated products are not completely effective in preventing the disease, but they appear to reduce severity of the disease and reduce its incidence by as much as 50% during outbreaks. The influence of vaccination on intermittent shedding of *S. equi* has not been adequately studied. All injectable, inactivated strangles vaccines, particularly the whole-cell bacterin, tend to cause reactions at the site of injection more often than do most other equine vaccines, particularly when administered in the neck. Injection in the gluteal muscles is not recommended because gravitational drainage along fascial planes can be obscure, can damage considerable tissue and can erupt in undesirable locations resulting in lesions that require prolonged time to heal. Maternal antibody interference is not known to occur when injectable, M-protein vaccines are administered, and mucosal interference needs to be studied further.

A modified live bacterial vaccine for intranasal administration is available. In order to avoid inadvertent contamination of other vaccines, syringes and needles, it is advisable and considered a good practice to administer all parenteral vaccines before handling and administering intranasally the modified live vaccine against *S. equi*.

Vaccination against *S. equi* is not routinely recommended except on premises where strangles is a persistent endemic problem or for horses that are expected to be at high risk of exposure. Vaccination is not routinely recommended for pleasure or performance horses kept in low-risk situations. Manufacturers’ recommendations for use of inactivated injectable vaccines call for primary vaccination with a series of 2 or 3 doses administered at intervals of 2 to 4 weeks, depending on the product used, followed by annual revaccination. Efficacy may be improved by using a primary series of 3-doses with boosters at 6-month intervals, regardless of the injectable product used. On breeding farms, efforts should be concentrated on preventing infection of foals and weanlings by vaccinating brood-mares with approved products that contain inactivated M-protein, 4 to 6 weeks before foaling. When an inactivated M-protein vaccine is used for primary vaccination of foals at high-risk for exposure to strangles, it is recommended that the initial series begin at 4 to 6 months-of-age, using 3 doses administered at 4- to 6-week intervals.

When the modified live bacterial vaccine is used for primary vaccination of foals, two doses may be administered intranasally at a 3-week interval, beginning at 6 to 9 months-of-age. This modified live vaccine has been safely administered to foals as young as 6 weeks-of-age when there is a high risk of infection such as occurs during an outbreak, but the efficacy of its use in very young foals has not been adequately studied. If administered to young foals in this manner, a third dose of the modified live vaccine should be administered 2 to 4 weeks before the foal is weaned to optimize protection during that time of high risk of infection. Semiannual (6-month intervals) or annual revaccination is recommended.

Following natural or vaccinal exposure to streptococcal antigens, certain individuals may unpredictably develop purpura hemorrhagica, an acute, non-contagious syndrome caused by immune-mediated, generalized vasculitis. Clinical signs develop within 2 to 4 weeks following natural or vaccinal exposure to streptococcal antigens and those signs include urticaria with pitting edema of the limbs, ventral abdomen and head. Subcutaneous and petechial hemorrhage may occur as may sloughing and exudation of involved tissues. Severe edema of the head may compromise breathing. Immediate medical attention should be sought for individual horses suspected of having purpura hemorrhagica.
Rabies

Rabies is an infrequently encountered neurologic disease of horses, which occurs when horses are inoculated with the rabies virus through the bite of infected (rabid) wildlife. Even though the incidence of rabies in horses is low, the disease is invariably fatal and has considerable public health significance. Wildlife species that serve as the natural reservoirs for infection with this rhabdovirus differ among regions of North America. Bites on horses are most often on the muzzle, face, and lower limbs. The virus then migrates via nerves to the brain where it initiates rapidly progressive, invariably fatal encephalitis. Clinical signs are highly variable and include fever, lethargy, anorexia, altered behavior, hyper-responsiveness to touch, weakness, incoordination, apparent lameness, colic, inability to swallow, blindness, hyperactivity, and convulsions.

All horses kept in areas where rabies is endemic in the wildlife population are at risk and should be vaccinated. Several vaccines containing inactivated (killed) rabies virus are licensed for intramuscular injection to horses and appear to be safe. Foals born to nonvaccinated mares may be vaccinated according to the manufacturers’ recommendations which call for primary vaccination of foals of at least 3 months-of-age with one dose, followed by a second dose at 1 year-of-age and annual boosters thereafter. Colostrum-derived antibodies interfere with active immunization of foals. Therefore, foals born to vaccinated mares should receive the first dose of vaccine no earlier than 6 months-of-age, a second dose 1 month after the first, a third dose at 1 year-of-age, followed by annual revaccination. None of the licensed vaccines are labeled for administration to pregnant mares; therefore, it is recommended that mares be vaccinated before breeding. However, it should be recognized that some veterinarians administer the killed-virus vaccine to pregnant mares without reports of adversity. Modified-live rabies vaccines are not licensed for use in horses and should not be used.

Equine Monocytic Ehrlichiosis (Potomac Horse Fever)

Equine monocytic ehrlichiosis is caused by *Ehrlichia risticii* and was originally described in 1979 as a sporadic disease affecting horses residing in northeastern United States near the Potomac River. Although the disease remains most prevalent in those eastern states, particularly near water-ways, it has been identified in several regions of the United States and Canada. The disease does not appear to be directly contagious and it now appears that trematode parasites of freshwater snails are the primary vector involved in its transmission. The disease is seasonal, occurring between late spring and early fall in temperate areas, with most cases in July, August, and September at the onset of hot weather. The disease may affect individual horses sporadically or cause outbreaks involving multiple horses. Clinical signs vary greatly among horses but include fever (102 to 107 F), lethargy, anorexia, reduced or absent borborygmi, mild to profuse diarrhea, colic, dehydration, and laminitis. A relatively large proportion of affected horses die or are euthanatized because of serious complications such as laminitis.

If Potomac Horse Fever has been confirmed on a farm or in a particular geographic area, it is likely that additional cases will occur in future years. Foals appear to have a low risk of contracting the disease. Vaccination against this disease has been questioned because field evidence of benefit is lacking. Explanations include lack of seroconversion and multiple strains in field cases whereas there is only one strain in available vaccines. If vaccination is elected, a primary series of 2-doses, administered 3 to 4 weeks apart, results in peak protection 3 to 4 weeks after the second dose. Manufacturers recommend revaccination at 6- to 12-month intervals, although some veterinarians
encourage an interval of 3 to 4 months for horses in endemic areas because protection following vacc-
ination is incomplete and short-lived. Vaccination should be timed to precede the anticipated peak
challenge during the summer months or fall. Based on current information, available vaccines are
licensed for use in stallions and pregnant mares and can be administered to gestating mares, 4
weeks before foaling. Colostral antibodies may block the primary vaccinal response of foals born to
immune mares when foals are vaccinated before 5 to 6 months-of-age. In about 33% of foals, mater-
nal antibody is detectable beyond 3 months and at least up to 5 months-of-age. Due to the low risk
of clinical disease in young foals and the possible interference by colostral antibodies, primary
immunization for most foals can begin after 5 months-of-age. If the primary series of vaccinations
is initiated when foals are less than 5 months-of-age, additional doses should be administered at
monthly intervals up to 5 months-of-age to ensure that an immunologic response is achieved.

Botulism
Three forms of botulism, namely toxicoi nfectious botulism (shaker foal syndrome), forage poi-
soning, and wound botulism have been observed in horses as a result of the action of potent toxins
produced by the soil-borne, spore-forming bacteria, \textit{Clostridium botulinum}. “Wound botulism”
results from vegetation of spores of \textit{Cl. botulinum} and subsequent production of toxin in contami-
nated wounds. Shaker foal syndrome results from toxin produced by vegetation of ingested spores in
the intestinal tract. Forage poisoning results from ingestion of preformed toxin produced by decay-
ing plant material or animal carcasses present in feed. Botulinum toxin is the most potent biologi-
cal toxin known and acts by blocking transmission of impulses in nerves, resulting in weakness pro-
going to paralysis, inability to swallow, and frequently, death. Of the 8 distinct toxins produced by
sub-types of \textit{Cl. botulinum}, types B and C are associated with most outbreaks of botulism in horses.
Almost all cases of shaker foal syndrome are caused by type B. Shaker foal syndrome is a significant
problem in Kentucky and in the mid-Atlantic seaboard states in foals between 2 weeks and 8 months-
of-age. A vaccine (toxoid) directed against \textit{Cl. botulinum} type B only is licensed for use in horses in
the United States. Its primary indication is for prevention of the shaker foal syndrome by colostral
transfer of antibodies produced by vaccination of the pregnant mare. Limited information suggests
that foals vaccinated with the toxoid at 2 weeks, 4 weeks and at 8 weeks-of-age developed adequate
serologic response, even in the presence of passive maternal antibodies. If at high risk of the disease
foals born to nonvaccinated mares may benefit from transfusion of plasma from a vaccinated horse
or from antitoxin to \textit{Cl. botulinum} type B. Vaccination with the toxoid as described above may also
be protective. The efficacy of these latter practices needs further study.

For primary vaccination, mares should be vaccinated during gestation with a series of 3 doses
administered 4 weeks apart, scheduled so that the last dose will be administered 4 to 6 weeks before
foaling to enhance concentrations of immunoglobulin in colostrum (ie. months 8, 9, 10 of gesta-
tion). Subsequently, mares should be vaccinated annually with a single dose 4 to 6 weeks prior to
foaling. After passively derived colostral antibodies wane, foals in endemic areas should receive a pri-
mary series of 3 doses of vaccine, administered 4 weeks apart, starting when the foal is 2 to 3 months
of age. Other horses can be immunized using a primary series of 3 doses of vaccine administered at
4-week intervals followed by annual revaccination. Currently, there are no licensed vaccines avail-
able for preventing botulism due to \textit{Cl. botulinum} type C or other subtypes of toxins and cross-pro-
tection between the B and C subtypes does not occur; thus routine vaccination against \textit{Cl. botulinum}
type C is not currently practiced.
Horses and foals with clinical botulism may be treated with botulinum antitoxin administered intravenously. Antitoxin is not effective against toxin that has been translocated to motor end-plates. Therefore, clinical signs may progress for 12 to 24 hours after administration of the antitoxin or until all internalized toxin has attached to motor end-plates. The dose of antitoxin to botulinum type B that is recommended for treating a foal is 30,000 IU and for an adult is 70,000 IU.

**Equine Viral Arteritis**

Equine viral arteritis (EVA) is a contagious disease of equidae caused by equine arteritis virus (EAV) and is found throughout the world. All breeds appear to be susceptible to the virus but the prevalence of infection, as determined by seroconversion, is much higher in some breeds, notably Standardbreds, than in others. Many horses infected with the virus develop no signs of disease. Although there is a high seroprevalence of infection in Standardbreds, clinical disease is rarely observed in this breed, indicating that subclinical infection is common. Conversely, Thoroughbreds and most other breeds have a low seroprevalence of infection, but show fulminant clinical signs when they become infected. Equine viral arteritis is of special concern because the virus can cause abortion in pregnant mares, death of young foals and establish a carrier state in stallions. Outbreaks of EVA are infrequent and sometimes difficult to diagnose because of clinical similarity to several other diseases (eg. equine rhinopneumonitis, influenza, equine infectious anemia or purpura hemorrhagica). Clinical signs vary in severity and may include some or all of the following: fever, anorexia, depression, edematous swelling of the eyelids, face, limbs, trunk, mammary glands, and genitals; lacrimation, conjunctivitis, rhinitis, nasal discharge, skin rash, and uncommonly, pneumonia and death of young foals. Aerosolized droplets of respiratory secretions containing virus can transmit the virus from horses with acute clinical disease, but perhaps of greater concern is transmission of the virus to mares in semen from sub-clinically infected carrier stallions by natural breeding or artificial insemination.

Following a large-scale outbreak of EVA in Kentucky in the mid-1980s, a modified-live-virus vaccine was developed and licensed for commercial use, primarily to 1) prevent infection and establishment of the carrier state in previously unexposed stallions, and 2) protect non-pregnant mares being bred to carrier stallions. Subsequently, the vaccine was shown to be effective in controlling outbreaks of the disease in concentrated populations of performance horses at racetracks. Only a few countries currently restrict the importation of horses that test serologically positive for neutralizing antibodies against EAV, whether by natural infection or vaccination, with the exception of seropositive carrier stallions. Because the seroconversion following vaccination cannot be distinguished from that resulting from natural infection, vaccination may complicate testing of horses for export. **Prior to vaccination, horses should be tested by a laboratory proficient and experienced in testing for antibodies to this virus to confirm that they are seronegative.** Vaccination of stallions and non-pregnant mares has been shown to be a safe and effective means of controlling the disease. Annual revaccination of all breeding stallions 28 days before the start of breeding season is highly recommended as a means of preventing establishment of the carrier state. Furthermore, in breeds or areas in which EAV is prevalent, vaccination of intact males between 6 and 12 months-of-age should be strongly encouraged after first receiving negative serologic results. Horses up to 6 months-of-age may be serologically positive due to maternal, colostrum-derived antibodies. Virus may be shed for 21 days from vaccinated stallions or from mares bred to carrier stallions; therefore, isolation of these individuals for this period of time is also recommended. Coordination of vaccina-
tion with state and/or federal regulatory officials, along with results of pre-vaccinal tests that provide evidence that the horse was seronegative prior to vaccination may be helpful in resolving disputes but do not guarantee that entry will be granted into foreign countries or onto breeding farms.

**Anthrax**

Anthrax is a serious and rapidly fatal septicemic disease caused by proliferation and spread of the vegetative form of *Bacillus anthracis* in the body, acquired though ingestion or contamination of wounds by soil-borne spores of the organism and is encountered only in limited geographic areas where alkaline soil conditions favor survival of the organism. Vaccination is indicated only for horses pastured in endemic areas. There is no vaccine licensed for use in horses, but the Sterne’s strain, nonencapsulated live spore vaccine that is licensed for use in cattle has been used to vaccinate horses. A primary series consisting of two doses of that vaccine should be administered subcutaneously 2 to 3 weeks apart followed by annual revaccination. Adverse systemic or local effects may occur occasionally. Little objective information is available regarding use of this vaccine in horses but clinical evidence suggests that it provides protection; however, vaccination of pregnant mares is not recommended. Because it is a live bacterial product, appropriate caution should be used during storage, handling and administration of the vaccine. Concurrent administration of antimicrobial drugs that are effective against anaerobes, and *B. anthracis* in particular, is contraindicated if the vaccine is to function as intended.

**Rotaviral diarrhea**

It has been estimated that as many as 70% of all foals will have at least one diarrheal episode prior to weaning. Foal diarrhea can be caused by any of several etiologies, but one of the major infectious causes is equine rotavirus which has been the cause of 50% or more of the cases of foal diarrhea in some areas. Equine rotavirus is transmitted via fecal-oral contamination, and damages the tips of villi in the small intestine resulting in cellular destruction, maldigestion, malabsorption, and diarrhea.

In a field study of an inactivated rotavirus A vaccine, a higher incidence of disease occurred in foals from the non-vaccinated control mares (18 out of 95; 19.0%) than in foals from the rotavirus-vaccinated mares (8 out of 85; 9.4%); but, the investigators concluded that the difference was not statistically significant (Fisher’s exact test; P=0.08). Results of a study in Japan showed that vaccination of pregnant mares with two doses of inactivated rotavirus-vaccine caused anti-rotavirus antibody to increase in colostrum and the mare’s milk, and reduced clinical signs in foals that suckled colostrum from those vaccinated mares, as compared to foals from non-vaccinated controls. The foals in the Japanese study were challenged with wild-type rotavirus to prove the efficacy of passive immunity for newborn foals against rotavirus infection. In that study the control foals were deprived of colostrum but were fed powdered cow’s milk to terminate intestinal absorption of macromolecules, and were placed back with their non-vaccinated mares after the colostrum was milked out of the mare. These results suggest the potential benefit of passively transferred maternal antibodies against equine rotavirus. Additional testing of the vaccine is needed to evaluate its efficacy.

The vaccine that is available in the USA contains rotavirus “A” and cross-protection against rotavirus “B” is not afforded with that vaccine. The inactivated rotavirus A vaccine is indicated for administration to pregnant mares, in endemic areas, to enhance concentrations of colostral immunoglobulins against equine rotavirus A. Data indicated that vaccination tended to reduce the severity of infection
rather than to prevent the signs. A three-dose series of the vaccine is required during each pregnancy at 8, 9 and 10 months of gestation. Some evidence suggests that after the initial three-dose series, one annual booster at 10 months of gestation may be sufficient to elevate maternal serumal antibodies. Although used in some instances, the latter protocol needs to be studied further. It is essential that the newborn foal receives an adequate amount of colostrum and absorbs sufficient antirotavirus A antibodies. For that reason, it is recommended that the concentration of total immunoglobulin in serum from newborn foals be checked, between 12 and 24 hours after foaling, to determine if failure of passive transfer (FPT) has occurred. If the foal has FPT it may be susceptible to rotaviral diarrhea even though the mare was vaccinated. An effective challenge-model has not been developed to test the efficacy of this vaccine in the U.S.A. There are no data to suggest that vaccination of the newborn foal with inactivated rotavirus A vaccine has any benefit for preventing or reducing the severity of infection. Further studies are needed to evaluate the efficacy of vaccination of foals against equine rotavirus before that practice can be recommended.
BIBLIOGRAPHY


1. A “standard” vaccination program does not exist.
2. Vaccination is an aid in prevention of infectious diseases.
3. Vaccination programs will not succeed without appropriate managerial practices.
4. Not all animals that receive a vaccine will mount an immunologic response; those that respond will not have equal responses.
5. Vaccines should be selected on the basis of
   a. demographics of the targeted disease
   b. effects of the disease, should it occur
   c. risks of exposure to the disease
   d. efficacy of vaccination program to reduce problems associated with the disease
   e. cost of appropriate vaccination
   f. potential adverse effects of the vaccination program
6. All animals in a group should be appropriately vaccinated according to their specific needs.
7. Expectations of the client for the vaccination program should be realistic.
8. Strict attention should be afforded the manufacturer’s recommendations for storage, handling, and route of administration of the vaccine.
9. For most inactivated vaccines, a series of multiple (generally 3) doses must be administered initially to induce protective immunity before booster vaccination can be productive.
10. In order to maximally protect foals during the first few months of life, broodmares should receive booster vaccinations during 4 to 6 weeks before foaling, and it is essential that foals receive an adequate amount of quality colostrum and absorb colostral antibodies.
11. Foals from appropriately vaccinated mares should receive their initial vaccination against most diseases no sooner than 6 months-of-age; in endemic areas, vaccination against Eastern Equine Encephalomyelitis should begin at 3 to 4 months-of-age until additional information indicates otherwise.
12. Adverse reactions should be reported to the manufacturer of the product involved.
13. Do not vaccinate within 2 to 3 weeks of shows, performance events, sales or shipment.
### TABLE 1. Suggested vaccination schedules for horses

<table>
<thead>
<tr>
<th>Disease/vaccine</th>
<th>Foals/weanlings</th>
<th>Yearlings</th>
<th>Performance Horses</th>
<th>Pleasure Horses</th>
<th>Broodmares</th>
<th>Comments</th>
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</thead>
<tbody>
<tr>
<td>Tetanus toxoid</td>
<td>From nonvaccinated mare:</td>
<td>Annual</td>
<td>Annual</td>
<td>Annual</td>
<td>Annual, 4 to 6 weeks prepartum</td>
<td>Booster at time of penetrating injury or surgery if last dose not administered within 6 months</td>
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<td></td>
<td>First dose: 3 to 4 months</td>
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<td>Second dose: 4 to 5 months</td>
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<td>Third dose: 5 to 6 months</td>
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<td></td>
<td>From vaccinated mare:</td>
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<td></td>
<td>First dose: 6 months</td>
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<td>Second dose: 7 months</td>
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<td></td>
<td>Third dose: 8 to 9 months</td>
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<td>Encephalomyelitis (EEE, WEE, VEE)</td>
<td>EEE: (in high-risk areas)</td>
<td>Annual, spring</td>
<td>Annual, spring</td>
<td>Annual, spring</td>
<td>Annual, 4 to 6 weeks prepartum</td>
<td>In endemic areas booster EEE and WEE every 6 months; VEE only needed when threat of exposure; VEE may only be available as a combination vaccine with EEE and WEE.</td>
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<td></td>
<td>First dose: 3 to 4 months</td>
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<td>Second dose: 4 to 5 months</td>
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<td></td>
<td>Third dose: 5 to 6 months</td>
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<td>WEE, EEE (in low-risk areas) and VEE:</td>
<td>Annual, spring</td>
<td>Annual, spring</td>
<td>Annual, spring</td>
<td>Annual, 4 to 6 weeks prepartum</td>
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<td></td>
<td>From nonvaccinated mare:</td>
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<td>First dose: 3 to 4 months</td>
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<td>Second dose: 4 to 5 months</td>
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<td>Third dose: 5 to 6 months</td>
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<td>From vaccinated mare:</td>
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<td>First dose: 6 months</td>
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<td>Second dose: 7 months</td>
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<td>Third dose: 8 months</td>
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<tr>
<td>Influenza</td>
<td>Inactivated injectable:</td>
<td>Every 3 to 4 months</td>
<td>Every 3 to 4 months</td>
<td>Annual with added boosters</td>
<td>At least semiannual, with 1 booster 4</td>
<td>A series of at least 3 doses is recommended for primary immunization</td>
</tr>
<tr>
<td>Condition</td>
<td>Vaccine Type</td>
<td>Dose 1</td>
<td>Dose 2</td>
<td>Dose 3</td>
<td>Booster 1</td>
<td>Booster 2</td>
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<tr>
<td>Rhinopneumonitis (EHV-1 and EHV-4)</td>
<td>First dose: 4 to 6 months Second dose: 5 to 7 months Third dose: 6 to 8 months Then at 3-month intervals</td>
<td>Every 3 to 4 months</td>
<td>Every 3 to 4 months</td>
<td>Optional: semiannual if elected</td>
<td>Fifth, seventh, ninth month of gestation (inactivated EHV-1 vaccine); optional dose at third month of gestation.</td>
<td>Semiannual with 1 dose of inactivated M-protein vaccine 4 to 6 weeks prepartum of foals.</td>
</tr>
<tr>
<td>Strangles</td>
<td>Injectable: First dose: 4 to 6 months Second dose: 5 to 7 months Third dose: 7 to 8 months</td>
<td>Semiannual</td>
<td>Optional: semiannual if risk is high</td>
<td>Optional: semiannual if risk is high</td>
<td>Vaccination of mares before breeding and 4 to 6 weeks prepartum is suggested. Breeding stallions should be vaccinated before the breeding season and semiannually.</td>
<td>Vaccines containing M-protein extract may be less reactive than whole-cell vaccines. Use when endemic conditions exist or risk is high.</td>
</tr>
<tr>
<td>Disease</td>
<td>Vaccine Information</td>
<td>Frequency</td>
<td>Dose Duration</td>
<td>Comments</td>
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<tr>
<td>Rabies</td>
<td>Fourth dose: 12 months. Intranasal: First dose: 6 to 9 months, second dose: 3 weeks later.</td>
<td>Annual</td>
<td></td>
<td>Foals as young as 6 weeks-of-age may safely receive the intranasal product but a 3rd dose should be administered before weaning.</td>
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<tr>
<td>Potomac horse fever</td>
<td>First dose: 6 months, second dose: 7 months. Foals born to non-vaccinated mares: First dose: 3 to 4 months, second dose: 12 months.</td>
<td>Semiannual</td>
<td></td>
<td>Semiannual with 1 dose 4 to 6 weeks prepartum. Booster during May to June in endemic areas.</td>
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<tr>
<td>Botulism</td>
<td>Foal from vaccinated mare: 3-dose series of toxoid at 30-day intervals starting at 2 to 3 months-of-age. Foal from nonvaccinated mare: see comments.</td>
<td>Not applicable</td>
<td></td>
<td>Initial 3-dose series at 30-day intervals with last dose 4 to 6 weeks prepartum. Only in endemic areas. A third dose administered 4 to 6 weeks after the second dose may improve the response of foals to primary immunization. Foal from nonvaccinated mare may benefit from: 1) toxoid at 2, 4 and 8 weeks-of-age; 2) transfusion of plasma from vaccinated horse; or 3) antitoxin. Efficacy needs further study.</td>
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<tr>
<td>Equine Viral Arteritis</td>
<td>Intact colts intended to be breeding stallions: One dose at 6 to 12 months-of-age</td>
<td>Annual for colts intended to be breeding stallions</td>
<td>Annual for colts intended to be breeding stallions</td>
<td>Annual for colts intended to be breeding stallions</td>
<td>Annual for seronegative, open mares before breeding to carrier stallions; isolate mares for 21 days after breeding to carrier stallion</td>
<td>Vaccinate mares at 8, 9, and 10 months of gestation, each pregnancy. Passive transfer of colostral antibodies aid in prevention of rotaviral diarrhea in foals.</td>
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<tr>
<td>Rotavirus A</td>
<td>Little value to vaccinate foal because insufficient time to develop antibodies to protect during susceptible age</td>
<td>Not applicable</td>
<td>Not applicable</td>
<td>Not applicable</td>
<td>Not applicable</td>
<td>Check concentrations of immunoglobulins in foal to be assured that there is no failure of passive transfer.</td>
</tr>
</tbody>
</table>

*As with the administration of all medications, the label and product insert should be read before administration of all vaccines.
†Schedules for stallions should be consistent with the vaccination program of the adult horse population on the farm and modified according to risk.
EEE = eastern equine encephalomyelitis; WEE = western equine encephalomyelitis; VEE = Venezuelan equine encephalomyelitis; EHV-1 = equine herpesvirus type 1; EHV-4 = equine herpesvirus type 4.