

Eastampton Veterinary Service, LLC

Matthew C. Edson
620 Powell Road, Eastampton, NJ 08060
Office: (609) 261-7280

Adult Equine Vaccination Recommendations

Below are the general vaccination guidelines we recommend for healthy adult horses. Since each horse has individual needs, we may adjust these recommendations based upon each animal's history, health, travel schedule, etc. The below recommendations are not intended to be a substitute for consultation with a veterinarian.

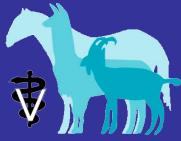
Vaccination schedule	
CORE VACCINES	
Tetanus	Initial: One dose followed by a booster in 3-4 weeks. Booster administered once yearly in the Fall.
Rabies	Initial: Single dose. Booster administered once yearly in the Fall.
EEE/WEE	Initial: One dose followed by a booster in 3-4 weeks. Booster administered once yearly in the Spring.
West Nile	Initial: One dose followed by a booster in 3-4 weeks. Booster administered once yearly in the Spring.
NON-CORE VACCINES	
Botulism	Initial: One dose followed by two boosters 2-4 weeks apart. Boosters administered yearly thereafter.
Strangles	Initial: One dose followed by a booster 3-4 weeks later. Boosters administered yearly thereafter. (IN or IM)
Potomac	Initial: One dose followed by a booster 3-4 weeks later. Boosters are administered every 6-12 months.
Equine Herpesvirus	Initial: One dose followed by a booster 3-4 weeks later. Boosters administered yearly thereafter.
Equine Influenza	Initial: One dose followed by a booster 3-4 weeks later. Boosters are administered every 6 months. (IN or IM)
Rotavirus	Pregnant mares only. Given during the 8 th , 9 th , and 10 th months of gestation.
Equine Viral Arteritis	Non-pregnant mares and breeding stallions three weeks prior to breeding (see further details below).

Core vaccines (recommended for all horses)

1. Tetanus

- **Initial vaccination consists of one dose followed by a booster in 3-4 weeks. After that, a booster is administered once yearly (Fall).**

All horses are at risk of development of tetanus, an often fatal disease caused by a potent



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neurotoxin elaborated by the anaerobic, spore-forming bacterium, *Clostridium tetani*. Tetanus toxoid is a core equine vaccine and is indicated in the immunization program for all horses.

Clostridium tetani organisms are present in the intestinal tract and feces of horses, other animals and humans, and are abundant as well as ubiquitous in soil. Spores of *Cl. tetani* survive in the environment for many years, resulting in an ever-present risk of exposure of horses and people on equine facilities. Tetanus is not a contagious disease but is the result of *Cl. tetani* infection of puncture wounds (particularly those involving the foot or muscle), open lacerations, surgical incisions, exposed tissues such as the umbilicus of foals and reproductive tract of the postpartum mare (especially in the event of trauma or retained placenta).

2. Rabies

- **Administered once annually (Fall)**

Rabies is an infrequently encountered neurologic disease of equids. While the incidence of rabies in horses is low, the disease is invariably fatal and has considerable public health significance. It is recommended that rabies vaccine be a core vaccine for all equids.

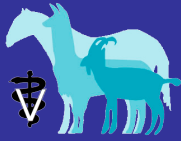
Exposure occurs through the bite of an infected (rabid) animal, typically a wildlife source such as raccoon, fox, skunk, or bat. Bites to horses occur most often on the muzzle, face, and lower limbs. The virus migrates via nerves to the brain where it initiates rapidly progressive, invariably fatal encephalitis.

3. Eastern/Western Equine Encephalomyelitis

- **Initial vaccination consists of one dose followed by a booster in 3-4 weeks. After that, a booster is administered once yearly (Spring).**

The distribution of EEE has historically been restricted to the eastern, southeastern and some southern states while outbreaks of WEE have been recorded in the western and mid-western states. Variants of WEE have caused sporadic cases in the northeast and southeast, most notably Florida. VEE occurs in South and Central America but has not been diagnosed in the United States for more than 20 years (and vaccination for this variant is not recommended in our area for various reasons). The availability of licensed vaccine products combined with an inability to completely eliminate risk of exposure justifies immunization against EEE and WEE as core prophylaxis for all horses residing in or traveling to North America and any other geographic areas where EEE and/or WEE is endemic.

Transmission of EEE/WEE/VEE is 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



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and direct horse-to-horse or horse-to-human transmission is possible. Of these 3 encephalidites, WEE has the lowest mortality (approx. 50%). Eastern equine encephalomyelitis is the most virulent for horses, with mortality approaching 90%. Epidemiological evidence indicates that young horses are particularly susceptible to disease caused by EEE. Venezuelan equine encephalomyelitis can also be lethal, however some horses develop subclinical infections which result in lasting immunity.

The risk of exposure and geographic distribution of EEE and WEE vary from year-to-year with changes in distribution of insect vectors and reservoirs important in the natural ecology of the virus. EEE activity in mosquito and birds, and resultant disease in humans and equids, continues to cause concern along the East Coast and demonstrates northward encroachment. WEE has caused minimal disease in horses in the last two decades; however, the virus continues to be detected in mosquitoes and birds throughout the Western states. In addition, variants that cause clinical disease in equids have been detected in the eastern U.S.

4. West Nile Virus

- **Initial vaccination consists of one dose followed by a booster in 3-4 weeks. After that, a booster is administered once yearly (Spring).**

West Nile virus (WNV) is the leading cause of arbovirus encephalitis in horses and humans in the United States. Since 1999, over 24,000 cases of WNV encephalitis have been reported in U.S. horses, with 1,069 cases reported in 2006. In 2006, there was a 14% increase in human cases and new expansion of WNV into 52 U.S. counties. The occurrence of over 2,500 human cases in 2007 indicates widespread viral activity in the environment. 1,086 equine cases were reported in the U.S. in 2006. As of October 2007, 250 equine cases were reported. This decline likely reflects both vaccination and naturally acquired immunity. Nonetheless, horses represent 96.9% of all non-human mammalian cases of WNV disease.

This virus has been identified in all of the continental United States, most of Canada and Mexico. Several Central and South American countries have also identified WNV within their borders. The virus is transmitted from avian reservoir hosts by mosquitoes (and infrequently by other bloodsucking insects) to horses, humans and a number of other mammals. West Nile virus is transmitted by many different mosquito species and this varies geographically. The virus and mosquito host interactions result in regional change in virulence of the virus and no prediction can be made regarding future trends in local activity of the viruses. Horses and humans are considered to be dead-end hosts for WNV; the virus is not directly contagious from horse to horse or horse to human. Indirect transmission via mosquitoes from infected horses is highly unlikely as these horses do not circulate a significant amount of virus in their blood.

Non-Core Vaccines (recommended based upon risk)

Botulism

- **First time vaccination consists of three doses each administered 2-4 weeks apart. After the initial series, a booster is given once yearly.**



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While Botulism infection is not particularly common, infection can be devastating. Treatment consists of *Equine Origin Botulism Antitoxin* which is quite expensive (\$1500+ per dose). Horses that progress to recumbency have less than a 25% chance of survival. Those that do survive the initial stage of infection will likely require two or more costly weeks of hospitalization and a long rehabilitation period.

Botulism has been observed in horses as a result of the action of potent toxins produced by the soil-borne, spore-forming bacteria, *Clostridium botulinum*:

- *Wound botulism* results from vegetation of spores of *Cl. botulinum* and subsequent production of toxin in contaminated wounds.
- *Shaker Foal Syndrome (toxicoinfectious)* results from toxin produced by vegetation of ingested spores in the intestinal tract.
- *Forage poisoning* results from ingestion of preformed toxin produced by decaying plant material, including improperly preserved hay or haylage, or animal carcass remnants present in feed.
- *Equine Grass Sickness (Equine Dysautonomia)* is considered a form of botulism resulting from the overgrowth of *Cl. botulinum* type C in the intestinal tract, especially the ileum. There are reports of isolated cases of the disease occurring in the U.S.

Botulinum toxin is the most potent biological toxin known and acts by blocking transmission of impulses in nerves, resulting in weakness progressing to paralysis, inability to swallow, and frequently, death. Of the 8 distinct toxins produced by sub-types of *Cl. botulinum*, types B and C are associated with most outbreaks of botulism in horses.

Strangles

- **First-time vaccination consists of one vaccine followed by a booster 3-4 weeks later. After the initial series is completed, a booster is given once yearly.**
- **Available in intranasal and intramuscular forms**

Streptococcus equisubspecies equi (S. equi var. equi) is the bacterium which causes the highly contagious disease strangles (also known as “distemper”). Strangles commonly affects young horses (weanlings and yearlings), but horses of any age can be infected. Vaccination against *S. equi* is recommended on premises where strangles is a persistent endemic problem or for horses that are expected to be at high risk of exposure. Following natural infection, a carrier state of variable duration may develop and intermittent shedding may occur. The influence of vaccination on intermittent shedding of *S. equi* has not been adequately studied.

The organism is transmitted by direct contact with infected horses or sub-clinical shedders, or indirectly by contact with: water troughs, hoses, feed bunks, pastures, stalls, trailers, tack, grooming equipment, nose wipe cloths or sponges, attendants’ hands and clothing, or insects contaminated with nasal discharge or pus draining from lymph nodes of infected horses. *Streptococcus equi* has demonstrated environmental survivability particularly in water sources and when protected from exposure to direct sunlight and disinfectants, and can be a source of infection for new additions to the herd.



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Infection by *S. equi* induces a profound inflammatory response. Clinical signs may include fever (102-106° F); dysphagia or anorexia; stridor; lymphadenopathy (+/- abscessation); and copious mucopurulent nasal discharge.

S. equi and *S. zooepidemicus* are antigenically similar organisms. However, exposure to, or vaccination against, one does not confer reliable immunity to the other.

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. Clinical signs may include urticaria with pitting edema of the limbs, ventral abdomen and head; subcutaneous and petechial hemorrhage; and sloughing 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.

Potomac

- **Initial vaccination consists of one dose followed by a booster 3-4 weeks later. Boosters are given 1-2 times yearly depending on risk.**

Equine monocytic ehrlichiosis is caused by *Neorickettsia risticii* (formerly *Ehrlichia risticii*). Originally described in 1979 as a sporadic disease affecting horses residing in the eastern United States near the Potomac River, the disease has since been identified in various other geographic locations in the United States and Canada. 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.

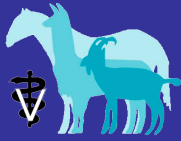
Clinical signs are variable but may include: fever, mild to severe diarrhea, laminitis, mild colic, and decreased abdominal sounds. Uncommonly, pregnant mares infected with *N. risticii* (usually in the middle trimester between 90 and 120 days) can abort due to fetal infection at 7 months of gestation.

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. Proposed explanations for this include lack of seroconversion and multiple field strains whereas only one strain is present in available vaccines.

Equine Herpesvirus (Rhinopneumonitis)

- **Initial vaccination consists of one dose followed by a booster in 3-4 weeks. A booster is then given once yearly.**

Equine herpesvirus type 1 (EHV-1) and equine herpesvirus type 4 (EHV-4) can each infect the respiratory tract, causing disease that varies in severity from sub-clinical to severe and is characterized by fever, lethargy, anorexia, nasal discharge, and cough. Infection of the respiratory tract with EHV-1 and EHV-4 typically first occurs in foals in the first weeks or months of life, but recurrent or recrudescent clinically apparent infections are seen in weanlings,



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yearlings, and young horses entering training, especially when horses from different sources are commingled. Equine herpesvirus type 1 causes epidemic abortion in mares, the birth of weak nonviable foals, or a sporadic paralytic neurologic disease (myeloencephalopathy) secondary to vasculitis of the spinal cord and brain.

Both EHV-1 and EHV-4 spread via aerosolized secretions from infected coughing horses, by direct and indirect (fomite) contact with nasal secretions, and, in the case of EHV-1, contact with aborted fetuses, fetal fluids, and placentae associated with abortions. Like herpesviruses in other species, these viruses establish latent infection in the majority of horses, which do not show clinical signs but may experience reactivation of infection and shedding 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 horses.

Primary indications for use of equine herpesvirus vaccines include prevention of EHV-1-induced abortion in pregnant mares, and reduction of signs and spread respiratory tract disease (rhinopneumonitis) in foals, weanlings, yearlings, young performance and show horses that are at high risk for exposure.

Equine Influenza

- **Initial vaccination consists of one dose followed by a booster in 3-4 weeks. A booster is then given every 6 months.**
- **Available in intranasal and intramuscular forms.**

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. Equine influenza is highly contagious and the virus spreads rapidly through groups of horses in aerosolized droplets dispersed by coughing. The severity of clinical signs depends on the degree of existing immunity, among other factors. Horses that are partially immune can become subclinically infected and shed virus.

Horses 1 to 5 years old are more susceptible. Older horses are generally less susceptible to infection, but immunity can be overwhelmed in horses frequently exposed at shows or similar athletic events. Horses in these age groups that have frequent contact with large numbers of horses should be vaccinated.

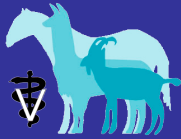
Rotavirus

- **Pregnant mares are vaccinated in their 8th, 9th, and 10th months of gestation.**

Rotavirus, a non-enveloped RNA virus, is a major infectious cause of foal diarrhea and has been documented to cause 50% or more of foal diarrhea cases in some areas.

While rotavirus diarrhea morbidity can be high (50% of susceptible foals), mortality is low (<1%) with veterinary intervention.

Equine rotavirus is transmitted via the fecal-oral route and damages the small intestinal villi resulting in cellular destruction, maldigestion, malabsorption, and diarrhea.



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As many as 70% of all foals in the United States will have at least one diarrheal episode prior to weaning. Mare owners need to be aware that strict biosecurity and disinfection during the foaling season also mitigates the morbidity associated with most types of infectious foal diarrheas and other contagious diseases.

Vaccination of mares results in a significant increase in foals' rotavirus antibody titers. Field trials of rotavirus vaccination in pregnant mares have shown a decrease in incidence and severity of foal diarrhea on farms that historically had annual rotaviral diarrhea cases. Other studies have shown increased rotavirus antibody in vaccinated mares' colostrum.

Equine Viral Arteritis

- **Non-pregnant mares and breeding stallions three weeks before breeding. Antibody testing prior to vaccination in stallions/mares to be exported is necessary.**

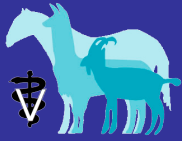
Equine viral arteritis (EVA) is a contagious disease of equids caused by equine arteritis virus (EAV), an RNA virus that is found in horse populations in many countries. While typically not life-threatening to otherwise healthy adult horses, EAV can cause abortion in pregnant mares; uncommonly, death in young foals; and establish a long-term carrier state in breeding stallions. While various horse breeds appear equally susceptible to EAV, the prevalence of infection can vary widely, with higher seropositivity rates occurring in Standardbreds and Warmbloods.

Historically, outbreaks of EVA have been relatively infrequent. However, the number of confirmed occurrences appears to be increasing, likely attributable to increases in:

- 1) Global movement of horses
- 2) Accessibility of carrier stallions
- 3) Utilization of shipped cooled or frozen virus-infective semen

Transmission most frequently occurs through direct contact with virus-infective respiratory secretions leading to widespread dissemination of the virus among susceptible horses in close proximity. Venereal transmission by infected stallions has a significant role in virus spread on or between breeding farms. Equine arteritis virus can be very efficiently spread through artificial insemination and the use of fresh-cooled or frozen semen. The virus has been shown to remain viable for considerable periods of time in raw, extended or frozen semen held at temperatures equal to or less than 4°C. Indirect transmission, though less significant, can occur through contact with virus-contaminated fomites.

The majority of primary EAV infections are subclinical or asymptomatic. EVA can vary in clinical severity both between and within outbreaks. EVA cannot be diagnosed based on clinical signs alone, as case presentation is similar to various other infectious and non-infectious equine diseases. Laboratory confirmation is required for diagnosis.



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Clinical signs, if they occur, typically develop 3-7 days post-infection and are variable but may include: fever; depression; anorexia; dependent edema (lower limbs, scrotum and prepuce or mammary glands); localized or generalized urticaria; supra or periorbital edema; conjunctivitis; and serous to mucoid nasal discharge. Abortion is a frequent sequel to infection in the unprotected, pregnant mare. Young foals exposed to EAV can develop a life-threatening pneumonia or pneumoenteritis.

