

Equine Viral Arteritis

Fernanda C. Camargo and K. Amy Lawyer, Animal and Food Sciences, and Peter Timoney, Veterinary Sciences

Equine viral arteritis (EVA) is a contagious disease of horses and other equine species caused by equine arteritis virus (EAV) that is found in horse populations in many countries. It was first isolated and identified in 1953 from the lung of an aborted fetus with characteristic pathologic changes in the smaller arteries, which is how the disease got its name.

EVA was differentiated from influenza and equine rhinopneumonitis caused by equine herpesviruses 1 and 4, both of which can cause clinically similar respiratory disease in horses. Although the majority of cases of primary infection with EAV are asymptomatic, the virus can cause signs of respiratory and systemic illness, abortion in pregnant mares, and infrequently, life-threatening respiratory or enteric illness in young foals. A significant outcome of EAV infection in the sexually mature colt or stallion is establishment of a long-term carrier state in a variable percentage of infected animals.

An extensive outbreak of EVA on a large number of Thoroughbred breeding farms in Kentucky in 1984 resulted in widespread national and international concerns over the disease. That was the first occasion of EVA diagnosis in Thoroughbreds in North America. The occurrence led to major restrictions being placed on movement of horses from the United States, many of which are still in place today.

Transmission

Equine arteritis virus is most frequently transmitted by direct physical contact with acutely infected horses via virus-containing secretions or excretions. Transmission can also occur through breeding mares naturally or by artificial insemination with the semen of an acutely or chronically infected stallion.

Additionally, EAV can be spread through indirect contact with objects contaminated with virus in urine or other body secretions/excretions of acutely infected horses, aborted fetuses, and placental membranes and fluids. Exposure in such instances is by the respiratory route. There is evidence that EAV can also be transmitted via embryo transfer.

The prevalence of EAV infection—that is, frequency of EAV antibodies in a group or population of horses—varies from country to country and from breed to breed. Higher seropositivity rates often occur in Standardbreds and warmbloods. EAV infection is considered endemic in Standardbreds in the United States, with up to 85% of adult Standardbreds frequently being seropositive to the virus. The seroprevalence of EAV infection in warmblood stallions and mares in some European countries can be very high, with up to 93% of Austrian Warmblood stallions found antibody positive in one study.

Persistently infected carrier stallions are the primary natural reservoir of EAV and are majorly responsible for perpetuating and maintaining the virus in equine populations. Equine arteritis virus is restricted to the reproductive tract in the carrier stallion, where it persists in certain of the accessory sex glands. Long after the animal has recovered from the acute phase of the infection, virus continues to be shed, sometimes for many years, in the semen of such stallions. Studies have shown that persistence of EAV is a testosterone-dependent carrier state. Neither mares, geldings, nor sexually immature colts can become carriers of the virus.

When mares without detectable antibodies to the virus are bred to a carrier stallion, the transmission rate can be as high as 85-100%. Bred mares will seroconvert (produce EAV antibodies) within 7-28 days. EAV can remain viable in fresh,

cooled, and frozen semen for varying periods of time—years in the case of frozen semen. Once mares are infected with EAV, they can then transmit the virus to other horses, primarily via the respiratory route but also venereally, for 6-10 days.

Development of the Disease

After respiratory exposure, EAV rapidly spreads from the lungs to the regional bronchial lymph nodes, where it multiplies and is released into the bloodstream and lymphatics. The virus is dispersed throughout the body principally by way of a cell-associated viremia. In the vast majority of cases, EAV is cleared from the body fluids and tissues of infected horses within 28 days, the exception being the carrier stallion. The vascular lesions start to diminish after 10-12 days and resolve completely in a matter of a few weeks.

Clinical Outcome

If clinical signs occur, they develop within 3-13 days after exposure, normally a week if transmission is via the venereal route.

Acutely infected animals may present any or all of the following signs:

- fever, loss of appetite, depression, lymphocytopenia
- dependent edema or fluid accumulation in and swelling of limbs, scrotum, sheath, and mammary glands
- respiratory signs, including nasal and/or ocular discharge
- conjunctivitis or “pink-eye”
- skin rash
- abortion in the pregnant mare
- interstitial pneumonia and enteritis in young foals
- persistent infection in the stallion

Some clinical signs are pictured in Figure 1.

Treatment

No specific anti-viral treatment currently exists for this disease. Non-steroidal anti-inflammatories are recommended in severe clinical cases of EVA to reduce the severity of illness. These non-steroidal anti-inflammatories are especially indicated in affected stallions to minimize the chances of a period of temporary sub-fertility that can last for up to four months. The vast majority of horses that develop EVA will experience a full and uneventful recovery with no adverse side effects, even without the intervention of any symptomatic treatment.

Diagnosis

EVA cannot be diagnosed on clinical signs alone, as these signs can closely resemble those of a wide range of other infectious and non-infectious equine diseases. Laboratory confirmation of a provisional clinical diagnosis is required.

Diagnosis of the carrier state in the stallion is based upon detection of the virus in the semen. Carrier stallions are constant shedders of EAV in semen but not in any other secretion or excretion.

Currently, it is not possible to differentiate a vaccine-induced antibody response from that due to natural infection.

Economic Significance of EVA

EVA can economically impact both the breeding and performance sectors of the equine industry. Direct financial losses resulting from outbreaks on breeding farms include the following:

- loss of foals (abortion or death)
- decreased commercial value of persistently infected stallions
- reduced demand to breed to carrier stallions because of the expense and inconvenience involved in vaccinating and isolating the mares before and after breeding
- reduced export markets for horses that are seropositive

An EVA outbreak at the racetrack or show grounds has the potential for widespread transmission and financial losses as a result of disruption of training schedules, reduced race or show entries, or even cancellation of horse-related events.

Figure 1. Signs of EAV infection.



Depression



Swelling of scrotum



Swelling of limbs



Skin rash, urticaria, or hives



Swelling of eyes and conjunctivitis



Swelling of mammary glands



Ocular discharge and swelling of eyes

Prevention

EVA is a preventable and controllable disease. Integral to the success of current control programs against EVA is the availability of a safe and effective vaccine against the disease.

Vaccination will:

- provide clinical protection against EVA, especially in the face of a field outbreak of the disease

- protect stallions against development of the carrier state
- immunize seronegative mares before they are bred to an EAV-infected stallion or with EAV-infective semen
- prevention and control of outbreaks in non-breeding populations

Prevention involves minimizing or eliminating direct or indirect contact of susceptible horses with various secretions, excretions, or tissues of infected horses together with a strategic vaccination program of stallions and sexually mature colts to prevent establishment of the carrier state. Discuss with your veterinarian about the need to vaccinate your breeding and performance horses.

Current control programs focus primarily on:

- restricting spread of the virus in breeding horse populations to prevent outbreaks of EAV-related abortion and/or illness and death in very young foals
- minimizing risk of establishing the carrier state in stallions

Nationally, no ongoing programs exist for preventing introduction of EAV or curtailing its spread among performance horses at racetracks, equestrian events, or horse shows. The infrequency with which such disease events occur is not sufficient justification for such a program. Horses travelling internationally, however, need to be confirmed that they are not shedding the virus. They also need to be either seronegative or, if seropositive, be accompanied by certification that their serum antibody level is either stable or decreasing. Semen being exported needs to be accompanied by laboratory certification that it is negative for EAV.

The following are some management measures that can help you prevent or minimize the spread of EAV:

- Ideally, isolate all new horses and those returning from other farms, sales, or racetracks for three to four weeks.
- Segregate pregnant mares from other horses on the farm and maintain these mares in small groups until they have foaled, which makes it easier to contain an outbreak should it occur.
- Breeding farm operations should blood-test all new breeding stallions for the presence of antibodies to EAV before each breeding season and if found seropositive, confirm that their semen is negative for virus.
- When planning to breed your mare, make sure the stallion is either a non-carrier or has been vaccinated against EVA.
- Annually vaccinate all non-carrier breeding stallions at least four weeks before the start of each breeding season.
- If you intend to breed a seronegative mare to a carrier stallion, vaccinate the mare at least 21 days before breeding. Mares do not have to be revaccinated if they are to be rebred to the same or another carrier stallion.
- Isolate first-time vaccinated mares bred to a carrier stallion from all but known seropositive horses for three weeks. It is especially important to avoid any contact between these mares and pregnant mares to which they could spread the virus by the respiratory route.

Resources

- United States Department of Agriculture (USDA): http://www.aphis.usda.gov/animal_health/animal_diseases/eva/
- Balasuriya, U.B.R., MacLachlan, N.J. Equine Viral Arteritis. In: *Equine Infectious Diseases*. Editors: Debra C. Sellon and Maureen Long, Saunders-Elsevier, Missouri, USA, pp. 153-164, 2007.
- Timoney, P.J. and McCollum, W.H. Equine Viral Arteritis. In: *The Veterinary Clinics of North America: Equine Practice on Infectious Diseases*. Editor: Josie L. Traub-Dargatz, W.B. Saunders, Philadelphia, Pennsylvania, USA, pp. 295-309, 1993.
- Timoney, P.J. Equine Viral Arteritis. In: *Equine Respiratory Diseases*. Editor: P. Lekeux, International Veterinary Information Service (www.ivis.org), Ithaca, New York, USA, 2002.