

atency is the biological mechanism by which all herpesviruses permanently survive in nature. Latency provides a natural reservoir for herpesvirus. Some viruses have a reservoir in insects, others have a reservoir in birds (such as West Nile virus). For herpes, the biological reservoir is the latently infected host animal.

When a herpesvirus is latent, only its genetic material (viral DNA) resides in the cells of the host animal. When that animal becomes stressed, the virus recrudesces (reactivates) and begins to multiply, sometimes causing clinical illness in the host. The virus isn't spread in its latent form; only when it reactivates. However, the host animal does not have to exhibit clinical signs to be spreading virus. This type of animal is called a "silent carrier" host.

Mares and Foals

The continuous cycle of reactivation from latency and subsequent spread of EHV among horses is the least understood epidemiological factor in the spread of herpesvirus in horse populations.

A pregnant mare can pass EHV-1 through the uterus, but that usually results in abortion. Sometimes a newborn death is caused by EHV-1. Some people think it is from an in utero (within the uterus) infection. The foal was already infected when it was born and couldn't overcome it.

A newborn foal gets maternal antibodies against herpesvirus from colostrum. However, when a mare is stressed by foaling, breeding, etc., the latent herpesvirus can reactivate and be spread through the mare's respiratory tract to the foal. Since the foal is protected by maternal antibodies, it might not show clinical signs. This silent transfer is the most common means for mare-to-foal spread of EHV.

Foals over a couple of months old infected with herpesvirus might have overt respiratory disease with a snotty nose and fever, but it's not a killer at that age.

Herpesvirus can become active and

be shed during any stressful event that occurs subsequent to the initial transfer of virus. That usually occurs during the post-weaning period in young foals, and during the stress of training, racing, and performing in young adult horses.

Testing

The initial clinical signs of EHV-1 or EHV-4 are similar to equine influenza. So how do you know what causes snots? Only by laboratory diagnosis. There are some subtle differences; flu spreads more rapidly, involves more horses, causes more coughing, and generally is a more severe respiratory disease. It would take an astute clinician to make the call without lab help.

If your veterinarian recommends a nasal swab for a sick horse, have it done in order to know what illness you are dealing with.

George Allen, PhD, of the University of Kentucky's Gluck Equine Research Center, has an ongoing research project concerning the latency and passing of EHV-1 in broodmares. The aims of the project are:

- 1. To characterize the natural annual pattern of reactivation and transmission of EHV-1 from latently infected, chronic carrier mares to their foals and susceptible herdmates; and,
- 2. To determine the effect of stressinducing broodmare farm management practices on altering the natural patterns of EHV-1 reactivation and transmission and the role of such changes in virus reactivation/transmission patterns as risk factors for herpesvirus abortion.

EHV-1 and -4 become latent in the trigeminal ganglia nerve and lymph nodes of the respiratory tract. These two sites of latency are equally important.

After transport, you can tell if the virus has reactivated by testing nasal swab samples for virus.

It is important to test horses with respiratory signs to determine what is causing the problem. If it's EHV-1, then you have to consider the possibility of the more serious neurologic form of the virus.

Respiratory Outbreaks

Most herpes respiratory outbreaks are caused by EHV-1 or -4. If a young horse brings herpes to an older horse population, the older horses might become infected, but usually don't show signs.

There is a lot of subclinical EHV-4 infection in older horses. They've seen the herpesvirus, and re-exposure is a natural boost to their immune system.

Neurologic Latency?

There is a gene mutation in the strain of EHV-1 that causes the neurologic form of the disease. No one knows if reactivation of the latent mutated virus is the source of all neurologic outbreaks, or whether the "wild" EHV-1 mutates anew during each neurologic outbreak.

Is it possible that the mutated gene resides in a latent form in some animals in which it doesn't cause any problems, then is passed to other horses when that "silent carrier" is stressed?

The answer is still unknown.

Any animal could carry the (neurologic) mutated form of EHV-1, but that mutation is not causing any problems in that animal. Only when the virus reactivates does it cause problems.

For example, a young horse might be exposed to the neurologic strain of EHV-1. It becomes latent and doesn't cause problems in that young horse. Then when the horse is stressed with weaning or going into training, the virus recrudesces and it is spread.

Alternatively, the horse might carry the wild strain (non-neurologic) EHV-1 in a latent form, stress causeses it to reactivate, and it mutates during reactivation to the neurologic form.

Editor's note: George Allen, PhD, head of the OIE Herpesvirus Reference Laboratory at the Gluck Equine Research Center, acted as a scientific resource in this article.