

Herpes viruses

About herpes viruses

Herpes viruses belong to an enormous family of DNA viruses – there are over 100 different strains and they affect most species (although it is rare for them to pass between one species and another). These viruses once contracted are life-long, and result in latent disease, meaning the virus hides in the body and manages to escape from the immune system. It does this by hiding in nerve cells – if anyone reading this is unfortunate enough to have experienced shingles, you will know how the patterns follow the nerve endings. Virus is shed at times of stress when a recrudescence of clinical signs can be seen. If the virus is passed to a species that is non-host adapted, the resulting clinical infection can be severe.

Route of transmission – how the viruses are spread

Herpes viruses are very varied, but in all cases, transmission is horizontal, i.e. the virus does not pass to the young during pregnancy. Direct contact is required, usually involving saliva, faeces, discharges from the eyes and nose, and respiratory secretions from coughs and sneezes. Tiny droplets of mucus can carry millions of viral cells and are easily dispersed with even the lightest of breezes, so particular care should be paid to ventilation in these cases.

Patients infected with herpes viruses will develop a small degree of immunity after the first infection, but viruses mutate frequently and the virus remaining in the nerve cells can and will remain infectious when released because of physiological or psychological stress.

Clinical signs (symptoms)

With so many different types of virus around, it is unsurprising that the clinical signs can vary and depend on the host species (i.e. the species affected), the particular strain of virus, and the virus location.

In general, herpes viruses have a preference for the nerves, lymphatic tissue (e.g. the glands in our necks), and epithelial cells (these line the gastrointestinal tract, the eyes, the skin and the respiratory tract). These viruses fully occupy the patient's immune system, so secondary infections with bacteria or other viruses are common. These secondary infections are usually the cause of any clinical signs appearing, so again the type of clinical signs seen depends on the type of secondary infection.

- ❖ **Tortoises** usually display necrotising lesions, those where the tissues are dying. Clinical signs may therefore include ulceration of the upper respiratory tract, causing mucoid to purulent discharges from the eyes and nose. Affected tortoises may have difficulty breathing, with rhinitis, conjunctivitis, inflammation of the tongue, abscesses in the back of the throat, or pneumonia may develop. Ulceration of the gastrointestinal tract may cause stomatitis (mouth rot) or regurgitation. These patients are invariably lethargic and anorexic, and may also have uncoordinated movements because of the effects on the virus on their nerves.
- ❖ **Terrapins**, although physiologically sharing many characteristics with tortoises, are often affected more severely. These patients are invariably lethargic and anorexic, but also develop generalized weakness resulting in very little activity or swimming. They may develop oedema (swelling of the tissues because of fluid infiltration) under the skin, or may develop bleeding disorders causing a speckled red appearance as if they have been dabbed with a paint brush.

- ❖ **Indian cobras, banded krates, and Mojave rattlesnakes** tend to develop swollen venom glands, producing very thick venom.
- ❖ **Iguanans** are, as with many of their conditions, usually anorexic and lethargic when infected with herpes viruses. They may turn a brilliant green colour, and are prone to spontaneous haemorrhages (bleeding)

Unfortunately, there are a number of clinical conditions that may develop after any species has been infected with herpes virus, again depending on the type of virus, the species affected, the severity of the infection, and the concurrent secondary infections. The possible consequences of an untreated infection with herpes viruses include the development of cancers, profuse bleeding (usually internally), death of the affected tissues (which can be fatal if the gastrointestinal tract ruptures), or death of the patient itself.

Diagnosis

The diagnosis of herpes viruses is complicated by the need to sample the virus whilst it is actively being shed, which may not be when the clinical signs are at their worst – usually shedding has much reduced by this time. Repeated blood tests to check the changes in levels of antibody are best for chronic infections, but acute or chronic infections may also be diagnosed with swabs from affected tissues using PCR or reverse PCR technology (where the viral DNA is reproduced and expanded from the tissues submitted).

Treatment of herpes viruses

Viral replication within the body is not regulated like normal replication, allowing viruses to continually mutate into something slightly different. It is therefore extremely difficult to develop targeted treatments, and the mainstay of treatment of affected patients remains in supportive care. This may include fluid and nutritional support, often with the aid of a feeding tube that is placed surgically, antibiotic courses to control the secondary infections, and surgical removal of abscesses and dead tissues as required (particularly those in the mouth).

There is a single medication that can act on herpes viruses called acyclovir. Its efficacy during clinical infection is good, but it must be administered relatively early in an infection course and for a long enough time. Even with the use of this drug, some of the virus will hide within the body to recrudescence at a later point in time. However, with effective supportive care – which may sometimes be intensive – some patients make excellent recoveries and continue with normal lives in between viral outbreaks.