Borna Disease Virus

This disease is not present in Australia at this time.

The classic hosts of BDV are horses, sheep and other farm animals, but the virus has been identified in animals as diverse as marsupials (the opossum) to rhesus monkeys. Lack of efficient detection techniques and frequent sub clinical infection inhibit good epidemiological studies, but currently natural infection has been reported only in Central Europe, North America, New Zealand, Japan, Iran, and Israel. Borna disease shows seasonal prevalence, occurring most often in spring and early summer.

The virus is assumed to be transmitted through salival, nasal, or conjunctival secretions because BDV-specific RNA has been found in these secretions. Animals become infected by direct contact with these secretions or by exposure to contaminated food or water. A minimum incubation period of 3 to 4 weeks is estimated for horses and sheep with nonspecific signs such as hyperthermia, anorexia, colic, and constipation in the initial phase of the disease

Borna Disease Virus has presented in animals with a range of neurological consequences, from fatal Borna disease to subtle behavioral changes. Some animals often show no clinical signs. Advanced age and immunodeficiency increase the probability of severe illness. Early infection seems to persist in the central nervous system to cause developmental and behavioral abnormalities. Recurrent episodes in surviving animals are possible and are frequently associated with stress.

In animals, acute Borna disease is associated with behavioral disturbances. Outcomes vary greatly across species, but in horses mortality rates are 80 to 100%. Symptoms include:

- Fatigue
- Hyperactivity
- Sensitivity to light and sound
- Dribbling and champing of jaws
- Paralysis of hind limbs, jaw and tongue
- Constipation

The ties between the human disease and BDV remain largely speculative, but many studies suggest ties to a variety of psychiatric disorders from depression to schizophrenia. As in animals, there is likely a range of consequences associated BDV infection. This is supported by some case studies that show disease progression following BDV infection similar to that shown in animals, involving muscle weakness, paresis and depressive apathetic behavior.

The antiviral drug amantadine sulfate may be a potential treatment for BDV. It has been demonstrated in vitro to inhibit wild-type BDV replication and spread of infection. Amantadine has recently been used to successfully improve clinical symptoms of depression in humans.

Experimental vaccines for BDV have had mixed results. The immunopathology of infection caused some vaccines to exacerbate disease, but recent evidence suggests there is a possibility of an effective vaccine being developed.

Dribbling and Champing of Jaws