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Sendai Virus

Classification

RNA virus, enveloped

Family

Paramyxoviridae

Note: Parainfluenzaviruses are subdivided into 3 groups, Pl-1, Pl-2, and Pl-3. Sendai is a member, but not the only member of Pl-1.

Affected species

Mice, rats, hamsters, and guinea pigs have been reported to be serologically positive, i.e., to have antibodies that react with the Sendai antigen commonly used in serology assays. Whether or not guinea pigs and some other rodents are truly susceptible to Sendai virus is controversial.

Frequency

Very rare in modern animal facilities; common in pet and wild rats and mice.

Transmission

Sendai virus (SV) is transmitted by aerosol and contact with respiratory secretions, but is not transmitted well by soiled bedding. The virus is highly contagious, but the infection does not persist in immunocompetent animals.

Clinical signs and Lesions

SV is one of the few viruses that may cause clinical signs in immunocompetent rodents. Clinical signs seen in mice are signs of pneumonia, such as dyspnea, chattering teeth, and death in young mice. On necropsy, dark, consolidated foci may be seen in the lungs. Microscopic changes are dependent on the extent of lung infection and concurrent immune response. SV infects mucociliary epithelium, and the changes to the epithelium may last for weeks, predisposing animals to secondary infections. Alveolar spaces contain white blood cells, fibrin, and necrotic cellular debris. Syncytia and intracytoplasmic eosinophilic viral inclusion bodies may be seen in the respiratory epithelium. Immunodeficient animals may present with a wasting syndrome associated with pulmonary consolidation.

In rats, SV is generally asymptomatic, but infected rats may have problems with reproduction, and microscopic changes include rhinitis, bronchitis, and bronchiolitis.

Susceptibility to severe disease is strain-dependent, with DBA/2 mice and Brown Norway rats very susceptible and C57BL/6 mice and F344 rats resistant.

Guinea pigs and hamsters are also occasionally seropositive on assays using SV antigen. The significance of this is unclear.

Diagnosis

SV infection is usually diagnosed in mice and rats by serology (ELISA, IFA, MFIA™) using SV antigen. Antibody titers rise rapidly, and serologic diagnosis may be made 8-12 days after infection. Diagnosis may also be made by PCR, and is recommended on symptomatic animals, as these animals would probably not have seroconverted to SV. A colony of mice suddenly presenting with widespread clinical signs of pneumonia such as dyspnea and chattering teeth should have SV on the list of possible infections. The microscopic changes associated with SV infection are characteristic and may also help confirm diagnosis.

In rats, guinea pigs, and rodents other than rats and mice, positive serology using SV antigen can be due to exposure to PI-2 or PI-3 virus. Positive MFIA™, ELISA, or IFA should be confirmed by the strain-specific HAI, which will discriminate between PI-1, PI-2, and PI-3. Further determination of whether PI-1 antibodies in guinea pigs indicate SV infection can be made using sentinel mice (which are susceptible to SV but not to other PI-1 viruses) or by PCR and sequencing of the amplified product. This is especially important since guinea pigs are susceptible to human-borne parainfluenzavirus infections.

Interference with Research

In addition to the lung changes, SV may cause infertility, predispose to secondary bacterial infection, and result in death of susceptible strains. Animals infected with SV are unsuitable for research purposes.

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Prevention and Treatment

Wild or pet mice and rats may serve as a reservoir of SV infection and access of wild rodents to the animal facility should be controlled. Regular serologic testing of resident animals, albeit at a low frequency due to the rarity of the virus, and quarantine of suspect incoming animals is advised. SV may contaminate animal biological products, therefore cell lines, transplantable tumors, and other biological products should be tested with PCR or by the MAP test (mouse antibody production) before being inoculated into animals. The handling of infected animals will depend on their value and the possibility of replacing them. Humans commonly serve as a source of non-Sendai parainfluenzavirus infections in laboratory rodents.

The virus is labile in the environment, and special measures are not required for disinfection. However, due to its high contagiousness, very strict measures are required to prevent the spread of the virus. In general, total depopulation, thorough cleaning of all aspects of the animal room, and restocking are recommended. Hysterectomy rederivation and embryo transfer have proven successful in eradication of SV. In immunocompetent animals, the cessation of breeding and deliberate spread of infection ("burn-out") has also been effective, but is not recommended. The transmission of SV can be limited by the use of cages with filter covers, reduction of staff movements, and by strict measures of housing and care.

References

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