

Sendai Virus

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Background: Sendai virus (SV) is one of the most important respiratory pathogens of rats and mice. Hamsters can also be infected, but they do not show symptoms of disease. SV is an ssRNA virus from the family *Paramyxoviridae*, and multiple strains have been described.

Transmission: SV is extremely contagious. Transmission is via direct contact and aerosol infection of the respiratory tract.

Clinical Signs: In rats, the infection is asymptomatic with minor effects on reproduction and growth of pups. In mice, the infection can be clinical or subclinical.

When the virus is introduced into a naïve colony, clinical signs may include teeth chattering, difficulty breathing, prolonged gestation, poor growth, and death of young mice. After the virus becomes established in a breeding colony, the subclinical state is seen. In this situation, mice are infected shortly after weaning, as their maternal antibodies decrease, and they show few clinical signs. There is no carrier state, so if there are no naïve animals (young pups or outside replacement stock), the infection will clear itself from the colony.

Diagnosis: Diagnosis is usually based on serology, via ELISA or IFA or both. The diagnosis can be strengthened by demonstration of typical lesions in clinically ill animals.

Effects on Research: SV causes reversible necrosis of the respiratory tract. The lesions can be more severe if concurrent infection with pathogens such as *Mycoplasma pulmonis* are present. Aged and immunodeficient mice and rats infected with SV develop a severe form of pneumonia.

SV also has significant effects on the immune systems of infected animals. Immunity to SV is both cell and antibody mediated. Reported effects include:

- Interference with early embryonic development and fetal growth.
- Alterations of macrophage, natural killer (NK) cell, and T- and B-cell function.
- Alterations of cytokine and chemokine production.
- Alterations of bronchiolar mast cell populations.
- Alterations of pulmonary hypersensitivity.

- Isograft rejection.
- Alterations in respiratory physiology
- Alterations in response to transplantable tumors and lung allografts.
- Alterations in the neoplastic response to carcinogens.
- Alterations in apoptosis rates.
- Alterations in wound healing.

Prevention: To prevent this disease, obtain replacement stocks from sources that are known to be free of disease. Tumor lines should be assessed for infection using MAP tests or other appropriate tests. Personnel working with infected animals should not enter rooms that contain naïve animals.

All animals should be placed in microisolator caging environments that are handled with the aid of a laminar flow hood using sterile techniques during handling and observation of the animals.

Eradication: The most effective way to eradicate SV infections is to cull the colony and obtain clean replacement stock. However, this is not always a feasible option when working with valuable mice.

Caesarian rederivation or embryo transfer can be used to produce offspring that have not been exposed to the virus. Repeated serological evaluations should be performed prior to reintroduction of the mice into a naïve population.

A breeding moratorium of at least 8 weeks can also be used to prevent the spread of the virus from young weanling animals to younger naïve animals. The animals should be housed in microisolator caging and handled with standard microisolator techniques. This method requires repeated serologic testing and strict adherence to a zero-tolerance for breeding policy. It is important to note that transgenic and knockout mice often have altered immune systems that may allow them to sustain the infection for longer periods of time or to develop a carrier state. In these cases, the breeding moratorium would not be the appropriate means of eradication.

References:

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