GV-SOLAS
Gesellschaft für Versuchstierkunde
Society for Laboratory Animal Science

Expert Information

From the Working Group on Hygiene

Implication of infectious agents on results of animal experiments

Murine Respirovirus

Status November 2019

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Contents

Murine Respirovirus

Background

- Also known as Murine Parainfluenzavirus or Sendai Virus (SeV)

Host species

- Mouse, rat, hamster, (guinea pig)

Organotropism

- Respiratory tract

Clinical disease

- Usually inapparent.
- Severe clinical disease with complicating infections (*M. pulmonis*, CAR bacillus).

Pathology

- Focal/segmental necrotizing inflammation of respiratory epithelium.
- Suppurative or necrotizing bronchitis and bronchiolitis.
- Foci of interstitial pneumonia.

Morbidity and mortality

- Up to 100% of a colony infected.
- Morbidity and mortality depending on host strain[1-4]

Interference with research

Physiology

- Murine Respirovirus infection in guinea pigs and rats enhances airway responsiveness to acetylcholine and substance P.[5,6]
- Murine Respirovirus infection aggravates the airway damage in rat lung allografts with
- chronic rejection.[7]
- Murine Respirovirus infection reduces the life span of the H-2d and H-2b genotypes B10 congenic mice.[8]

Pathology

- increased number of mitotic cells in bronchial epithelium and in lung parenchyma[9]
- increase in bronchiolar mast cells persists for months after infection[10]
- Murine Respirovirus nucleoprotein gene is detectable in the olfactory bulbs of intranasally infected mice for at least 168 days post-infection (p.i.) by PCR[11]

Immunology

- increase in natural killer cell mediated cytotoxicity[12]
- induction of tumor necrosis factor and other cytokines[13-16]

- long term effect on the immune system (55 out of 63 parameters are affected)[17]
- Murine Respirovirus infection of C57BL/6 mice elicits a strong CD4+ and CD8+ T-cell response in the respiratory tract.[18]
- infected mice have enhanced numbers of cytotoxic T-lymphocyte precursors (> 20x background) for life.[19]
- impairment of macrophage function causing delay in wound healing[20]

Infectiology

- decrease of pulmonary bacterial clearance[21]
- interaction with bacterial pathogens[22]

Oncology

- production of polyploid variants of tumor cells with increased chromosome numbers and reduced tumourigenicity[23]
- reduced transplantability of hamster tumor cells in combination with augmented cell-mediated immunity[24,25]
- altered host responce to transplantable tumors[26-29]
- strong influence on chemically induced carcinogenesis[30]

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