Sendai Virus

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 epithilium
- 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 (Brownstein and Winkler, 1986, Parker et al., 1978, Percy et al., 1994, Steward and Tucker, 1978)

Interference with research

Physiology

- Sendai virus infection in guinea pigs and rats enhances airway responsiveness to acetylcholine and substance P (Elwood et al., 1993; Yamawaki et al., 1995)
- Sendai virus infection aggravates the airway damage in rat lung allografts with chronic rejection (Winter et al., 1994)
- Sendai virus infection reduces the life span of the H-2d and H-2b genotypes B10 congenic mice (Yunis and Salazar, 1993)

Pathology

- increased number of mitotic cells in bronchial epithelium and in lung parenchyma (Richter, 1970)
- increase in bronchiolar mast cells persists for months after infection (Sorden and Castleman, 1995)
- Sendai virus nucleoprotein gene is detectable in the olfactory bulbs of intranasally infected mice for at least 168 days post-infection (p.i.) by PCR (Mori et al., 1995)

Immunology

- increase in natural killer cell mediated cytotoxicity (Clark et al., 1979)
- induction of tumor necrosis factor and other cytokines (Aderka et al., 1986; Costas et al., 1993; Mo et al.,1995; Uhl et al., 1996)
- long term effect on the immune system (55 out of 63 parameters are affected (Kay, 1978)
- Sendai virus infection of C57BL/6 mice elicits a strong CD4+ and CD8+ T-cell response in the respiratory tract (Cole et al., 1994)
- infected mice have enhanced numbers of cytotoxic T-lymphocyte precursors (> 20x background) for life (Doherty et al., 1994)
- impairment of macrophage function causing delay in wound healing (Kenyon, 1983)

Infectiology

- decrease of pulmonary bacterial clearance (Degre and Solberg, 1971)
- interaction with bacterial pathogens (Jakab, 1981)

Oncology

- production of polyploid variants of tumor cells with increased chromosome numbers and reduced tumourigenicity (Matsuya et al., 1978)
- reduced transplantability of hamster tumor cells in combination with augmented cell-mediated immunity (Ogura et al., 1980; Yamada and Hatano, 1972)
- altered host responce to transplantable tumors (Wheelock, 1966, 1967; Collins and Parker, 1972; Matsuya et al., 1978)
- strong influence on chemically induced carcinogenesis (Peck et al., 1983)

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