



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**

**Sialodacryoadenitis Virus /
Rat Corona Virus**

Status September 2018

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Sialodacryoadenitis Virus / Rat Corona Virus

Background

- Sialodacryoadenitis virus (SDAV) and Parkers's rat coronavirus (RCV) are two naturally occurring prototype coronaviruses isolated from the rat. Although these viruses are considered to be different strains of one rat coronavirus, this dichotomy and terminology continues because of historical precedent.¹ Like mouse hepatitis virus (MHV), the rat coronavirus group is likely to contain numerous, constantly changing strains that differ in virulence and organ tropism.^{1,2}

Prevalence

- Serological surveys indicate a low prevalence of infections with SDAV/RCV in contemporary rat colonies, i.e. less than 1% of the samplings or sera tested were found to be positive.⁴⁻⁶ In a more recent survey, less than 5% of responding institutions in the U.S. reported SDAV in their rat colonies.⁷

Host species

- Rat

Properties

- RNA virus, antigenically related to MHV.
- Relatively stable at acid pH 8.
- Can be stored at -60 °C for at least 7 years, but loses infectivity rapidly at room temperature, freezing to -20 °C, heating to -56 °C and exposure to lipid solvents.^{8,9}
- Can be propagated in multiple cell lines.^{8,9}

Susceptibility

- LEW, WAG/Rij and SHR rats are more susceptible than other strains; less susceptible rat strains include WI, SD, LE and F344.^{3,10}
- In athymic rats, SDAV infection is more severe, is persistent and may be fatal.^{11,12}
- Mice have been shown to be susceptible experimentally.¹³

Organotropism

- Salivary glands, lacrimal (incl. Harderian) glands, cervical lymph nodes, thymus, and respiratory tract.³
- Virus is present in tissues for only about one week.

Clinical disease

- Enzootic infections: asymptomatic or mild conjunctivitis in suckling rats.
- Epizootic infections: cervical swelling, sneezing, nasal and ocular discharge (serous to seropurulent), porphyrin staining, squinting, photophobia, conjunctivitis, exophthalmus, corneal ulceration and keratoconus.^{3,9}
- Most clinical signs disappear in about one week.

Pathology

- Characteristic changes in salivary and lacrimal glands: coagulation necrosis of the ductal epithelium, interstitial edema, mononuclear and polymorphonuclear cell infiltration (during acute stages), squamous metaplasia of ductal and acinar structures (during reparative stages).
- Ocular lesions (secondary to impaired lacrimal function): interstitial keratitis, corneal ulceration, keratoconus, synechia, hypopyon, hyphema, conjunctivitis, megaloglobus with lenticular and retinal degeneration.
- Other changes: focal necrosis of the thymic cortex and medulla, focal necrosis and lymphoid hyperplasia in the cervical lymph nodes, necrotizing rhinitis, tracheitis, focal bronchitis and bronchiolitis, focal interstitial pneumonia.^{1,3,9}

Morbidity and mortality

- Morbidity: high
- Mortality: virtually none

Zoonotic potential

- No data

Interference with research

Oncology

- Reduction of epidermal growth factor in submaxillary salivary gland.¹⁴
- Higher prevalence of anterior pituitary tumors in infected male F344/NCr rats.¹⁵

Teratology

- No data

Infectiology

- No data

Immunology

- Inhibition of phagocytosis and IL-1 production in alveolar macrophages.¹⁶
- Increase of localized graft-vs.-host disease in salivary and lacrimal glands after bone marrow transplant.¹⁷
- Infected alveolar type I epithelial cells induce expression of CXC chemokines (CINC-2, CINC-3, LIX) in uninfected alveolar type I epithelial cells *in vitro*.¹⁸
- Influx of neutrophils and macrophages and expression of chemokines (LIX, MCP-1, CINC-1, IP-10) and surfactant protein SP-D in the lung.¹⁹
- Polymorphonuclear neutrophils express various proinflammatory cytokines (IL-18, IL-1 α , IL-1 β , TNF- α) and chemokines (CXCL-1, CXCL-2, IP-10, CXCL-11, CCL-2, CCL-4, CCL-7, CCL-9, CCL-12, CCL-22) when exposed to infected alveolar type I epithelial cells *in vitro*.²⁰

Interactions with other infectious agents

- Enhancement of nasal colonization with *Haemophilus influenzae* type b in infant rats.²¹
- Exacerbation of lesions caused by *Mycoplasma pulmonis*.^{22,23}
- Increased adherence of *Mycoplasma pulmonis* in tracheas of infected rats.²⁴

Toxicology

- No data

Physiology

- Interference with studies involving eyes, salivary and lacrimal glands or respiratory system.²⁵
- Reduced food consumption and body weight loss or slowing of growth rate.²⁶⁻²⁸
- Rise in serum amylase.²⁹
- Impaired olfaction and chemoreception for at least two weeks post-exposure.³⁰
- Increase in permeability of the alveolar epithelium.¹⁹

Cell biology

- Impaired axonal regeneration and diminished functional recovery in infected rats with unilateral tibial nerve transection.³¹
- Infected alveolar type I epithelial cells direct neutrophil chemotaxis and inhibit their apoptosis *in vitro*.³²

Assisted reproductive technology

- Disorders of estrous cycles and increased embryonic and postnatal mortality.^{26,27,33}

Special considerations

- No data

Updated by Michael Mähler, Hannover, September 2018

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