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accelerating drug development. exactly.

Mycoplasma pulmonis

Classification

Small, pleomorphic bacteria that lack a cell wall

Family

Mycoplasmataceae

Affected species

Mice and rats are primary hosts; guinea pigs and hamsters are susceptible to experimental infections.

Frequency

Rare in modern laboratory animal populations; common in pet and wild populations of rats and mice; guinea pig and hamster prevalence data is not available.

Transmission

M. pulmonis is transmitted through direct contact, aerosol, and transplacentally.

Clinical Signs and Lesions

Typical sites of colonization for *M. pulmonis* are the middle ear and nasopharynx. Although subclinical infections are possible, animals usually present with clinical signs. Clinical signs in mice include weight loss, ruffled hair coat, dyspnoea ("chattering"), hunched posture, and reluctance to move. In rats, clinical signs are similar, although dyspnoea in rats is described as "snuffling", and rats may also exhibit porphyrin staining of the nose and eyes. Occasionally, arthritis is seen in association with *M. pulmonis*. Reproductive effects may also be noted, such as infertility, pup infection, low birth weight, abortion, or neonatal or fetal death. Age, strain, gender, concurrent viral infections, and immune status all affect the severity of and susceptibility to mycoplasmal disease.

Gross lesions noted on necropsy vary with the duration of infection and the tissue infected. Mycoplasmosis causes suppurative infection of the respiratory and reproductive tracts, and occasionally of joints. Early infections may have only a slight exudate, most often evident as suppurative rhinitis or otitis media. In advanced infections, the middle ear, bronchi and bronchioles (which can become greatly dilated), and occasionally uterus fill with suppurative material. Microscopically, there is chronic suppurative bronchopneumonia with prominent hyperplasia of bronchus-associated lympoid tissue. Suppurative inflammation, with a hyperplasia of associated lymphoid follicles, is also seen in any other tissues affected, including uterus, middle ear, and joints.

Diagnosis

Finding characteristic clinical signs and histologic lesions may aid in diagnosis of advanced mycoplasmosis. Colony surveillance for diagnosis of mycoplasmosis is often through serology (MFIA™, ELISA, or IFA), as the organisms persist despite the presence of antibodies. However, animals may be infected for some time, perhaps months, before antibodies develop against these surface-dwelling organisms. Thus, culture and PCR remain useful in detecting early infections. Culture of *M. pulmonis* is feasible, although the organism is fastidious in its growth requirements, and a slow grower. PCR, typically on lavage fluid, is also available and is a valuable screening adjunct for colonies. Cilia-associated respiratory bacillus is a frequent co-pathogen with *M. pulmonis* and diagnostic investigations should include screening for this organism, usually by serology, PCR, or histopathology.

Interference with Research

M. pulmonis infection has the potential to interfere with a wide variety of research. Animals that carry *M. pulmonis* are not suitable for use in research. Animals with this infection may be clinically ill, rendering them unfit for use. *M. pulmonis* may disseminate widely in the host and affect a number of organ systems. The lung is the primary target, however, and long-lasting changes occur. Some changes described by researchers include: decreased ciliary function, derangement of airway innervation, and altered endothelial cell morphology and function. Mycoplasmal infection affects the immune response and may predispose to other infections. Animals with mycoplasmal infections have decreased delayed hypersensitivity responses, T cell

subset changes, and increased total lymphocyte and neutrophil counts. Finally, *M. pulmonis* infection may interfere with the use of animals for the study of other mycoplasmas.

Prevention and Treatment

Prevention of entry of *M. pulmonis* into a facility should focus on the entry of animals and biologic materials. Animals should be sourced from reputable vendors or quarantined and screened before entry into the animal house. *M. pulmonis* is common among wild rats and mice, so a vigorous pest control program should be in place. Pet rats and mice also commonly harbor *M. pulmonis*, and workers should not keep pet rodents or have secondary employment that places them in contact with pet or wild rodents. *Mycoplasma* spp. are common contaminants of animal and human tumor and cell lines, but *M. pulmonis* is rarely confirmed in these materials, which are much more often contaminated with non-rodent mycoplasmas. Nonetheless, these materials should be screened via PCR or antibody production tests for infectious agents before being injected into animals.

There is no effective treatment for mycoplasmosis in rodent colonies. Depopulation and repopulation is one option for infected colonies, as is rederivation. Rederivation may be accomplished via hysterectomy and fostering or via embryo transfer. *M. pulmonis* can be found in both male and female reproductive tissues, so the pre-treatment of donor animals with antibiotics may be helpful in decreasing the chance of vertical transmission embryo transfer, rather than hysterectomy rederivation, may be the best choice for a *M. pulmonis*-infected colony.

Generally, mycoplasmas, due to their cell membrane construction, are not considered to be viable for long periods of time outside of a host. Some mycoplasmas are able to form biofilms, which may afford them better resistance to heat and desiccation than previously thought. Decontamination appropriate for more robust non-spore-forming bacteria should be sufficient to decontaminate after an *M. pulmonis* contamination. As with any other contamination, non-essential materials should be discarded, and items cleaned with an appropriate disinfectant and/or autoclaved as necessary, before new animals are introduced.

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