charles river
accelerating drug development. exactly.

Streptococcus pneumoniae

Classification

Alpha-hemolytic, Gram-positive, encapsulated, aerobic diplococcus

Family

Streptococcaceae

Affected species

Primarily described as a pathogen of rats and guinea pigs. Mice are susceptible to infection. Agent of human disease and human carriers are a likely source of animal infections. Zoonotic infection is possible.

Frequency

Rare in modern laboratory animal colonies. Prevalence in pet and wild populations unknown.

Transmission

Transmission is primarily via aerosol or contact with nasal or lacrimal secretions of an infected animal. *S. pneumoniae* may be cultured from the nasopharynx and tympanic bullae.

Clinical Signs and Lesions

Inapparent infections and carrier states are common, and very few infected animals will ever show disease. Despite its periodic detection in large breeding colonies, no outbreaks of *S. pneumoniae* disease have been reported in rats in 35 years, nor in guinea pigs in 20 years, raising the possibility that previous "outbreaks" were the result of mixed infections of *S. pneumoniae* together with additional infectious agents. When it occurs, disease is usually seen in young animals, especially after perturbation of host defense mechanisms, such as concurrent infection, experimental manipulation, or a change in environment. Clinical signs can include general ill rodent signs, such as hunched posture, ruffled fur, inappetance, or death with no premonitory signs, or specific signs such as nasal discharge, conjunctivitis, and vestibular signs. In guinea pigs, stillbirths and abortions are part of the clinical presentation.

On necropsy, a serosanguineous to purulent exudate is often found in the nasal cavities and the tympanic bullae. The lungs can have areas of firm, dark red consolidation. Fibrinopurulent pleuritis, pericarditis, and peritonitis are other changes seen on necropsy of animals affected by *S. pneumoniae*. Histologic lesions are consistent with necropsy findings, and bronchopneumonia of varying severity and fibrinopurulent serositis are often seen.

Diagnosis

An *S. pneumoniae* infection should be suspected if encapsulated Gram-positive diplococci are seen on a smear from a lesion. Confirmation of the diagnosis is via culture of lesions or affected tissues. *S. pneumoniae* grows best on 5% blood agar and is alpha-hemolytic. The organism is then presumptively identified with an optochin test. PCR assays are also available for diagnosis. PCR-based screening for *S. pneumoniae* may be conducted on respiratory samples or feces. PCR may also be useful for confirmation of presumptive microbiologic identification or confirming the identity of bacteria observed in histologic lesions.

Interference with Research

Animals carrying *S. pneumoniae* are usually suitable for use in research. The organism should not be tolerated in immunocompromised animals. However, because in immunocompetent animals it is usually noninvasive, living on nasopharyngeal surfaces, its detection should not necessarily cause termination of ongoing studies. Zoonotic transmission of this agent from rats or mice to humans has never been reported.

Prevention and Treatment

To prevent transmission of *S. pneumoniae* to animals, the animals must be raised in strict bioexclusion housing, such as would be necessary to prevent immunodeficient mice from acquiring other human-borne bacteria. As rodent S. pneumoniae probably originates from humans, animal care workers should wear masks and use other standard PPE to reduce the

chance of contaminating the animals. Caretakers with pneumococcal pneumonia, otitis media, conjunctivitis, or other diagnosed or possible streptococcal infections should not work with animals until a course of antibiotics has been completed. Normal animal work precautions will prevent humans from acquiring *S. pneumoniae* from animals.

S. pneumoniae are susceptible to most common disinfectants used in animal facilities. Any chemical or mechanical sterilant will also serve to remove *S. pneumoniae* from the environment. Treatment of animals with antimicrobials may serve to treat illness, but rarely, if ever, resolves the carrier state, nor will antibiotic treatment eliminate bacteria from the bedding or cage surfaces. Thus, treatment is only recommended to ameliorate clinical signs. Human isolates of *S. pneumoniae* are often multi-drug resistant. To obtain a *S. pneumoniae*-free colony, animals should be rederived through embryo transfer or hysterectomy into/onto *S. pneumoniae*-free dams.

References

Adams LE, Yamauchi Y, Carleton J, Townsend L, Kim OJ. 1972. An epizootic of respiratory tract disease in Sprague-Dawley rats. J Am Vet Med Assoc 161:656-660.

Baker DG. Natural Pathogens of Laboratory Animals: Their effects on research. Washington, D.C.: ASM Press; 2003. 385 pp.

Cardozo DM, Nascimento-Carvalho CM, Souza FR, Silva NM. 2006. Nasopharyngeal colonization and penicillin resistance among pneumococcal strains: a worldwide 2004 update. Braz J Infect Dis 10:293-304.

Fallon MT, Reinhard MK, Gray BM, Davis TW, Lindsey JR. 1988. Inapparent Streptococcus pneumoniae type 35 infections in commercial rats and mice. Lab Anim Sci 38:129-132.

Fox JG, Anderson LC, Lowe FM, Quimby FW, editors. Laboratory Animal Medicine. 2nd ed. San Diego: Academic Press; 2002. 1325 pp.

Keyhani M, Naghshineh R. 1974. Spontaneous epizootic of pneumococcus infection in guinea-pigs. Lab Anim 8:47-49.

Percy DH, Barthold SW. Pathology of Laboratory Rodents and Rabbits. Ames: Iowa State University Press; 2007. 325 pp.

Saito M, Muto T, Haruzono S, Nakagawa M, Sato M. 1983. An epizootic of pneumococcal infection occurred in inbred guinea pig colonies. Jikken Dobutsu 32:29-37.

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