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PSEUDOMONAS AERUGINOSA - Infection of Mink

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Pseudomonas aeruginosa is responsible for an acute infectious disease of mink often referred to as hemorrhagic pneumonia. The disease occurs sporadically in the United States, usually in the autumn months, and is associated with mortality of 1–50% of the mink in affected herds.

Clinical signs are usually not observed. Mink are often found dead with pale red frothy exudate around the nose and mouth. The lungs are diffusely hyperemic and hemorrhagic. One or more lobes may be consolidated. Microscopic lesions include several hemorrhage and necrosis associated with large numbers of bacteria, some of which may be within the walls of blood vessels. Consolidated lobes contain an infiltration of neutrophils and fibrin. Significant inflammation or necrotic lesions are found only rarely in tissue other than the lung and bronchial lymph nodes.

We have characterized the pathogenesis of the pulmonary lesions in mink infected intratracheally with P. aeruginosa (1600 organisms). Gross lesions were first visible at 16 hours post-infection (PI) as 1-3 mm red to tan foci scattered throughout the lung. Areas of discolouration increased in size with time so that at 36 hours large portions of lobes or whole lobes were affected.

Infected mink that died between 36 and 72 hours PI had consolidation in one or two lobes and hyperemia and hemmorhage in the remainder of the lung. Gross lesions in mink killed between 36 and 84 hours PI ranged from small foci to consolidation of entire lobes.

Microscopic lesions were first detected at 8 hours PI and consisted of small foci of neutrophils and macrophages within terminal bronchioles and adjacent alveoli. Ultrastructurally, there was an infiltration of neutrophils through the alveolar wall at 8 and 16 hours PI, but the alveolar lining cells were intact.

Characteristic foci of inflammation and necrosis were present at 24 hours PI. These foci involved 1-3 alveoli and consisted of neutrophils and fibrin associated with necrosis of alveolar septa. Ultrastructurally, there was necrosis and dissociation of alveolar lining cells in association with fibrin exudation.

The necrotizing inflammatory process spread with time as foci of necrosis became larger, often confluent, and were bridged by exudate resulting in gross consolidation of the affected lobes. Non-consolidated lobes contained foci of necrosis and hemmorhage of alveoli and terminal bronchioles. This event may have occurred due to spread of large numbers of bacteria via the airways.

Invasion of vessel walls by bacteria was a terminal event and was secondary to necrosis and bacillary invasion of surrounding tissues. In severely affected areas, large numbers of bacteria were present, often associated with leukocytes. However, many of the leukocytes were degenerate or necrotic.

In mink surviving beyond 48 hours, the inflammatory lesions tended to be less intense, focal and contained a prominent number of macrophages. Ultrastructurally, there was evidence of early regeneration with hyperplasia of type II alveolar epithelial cells.

Culture of various tissues from infected mink indicated that bacteria spread from the lung to the bronchial lymph nodes by 16 hours PI and to the liver and spleen by 24 hours PI. In spite of the presence of bacteria, there was no significant lesions present in the liver of spleen. P. aeruginosa was isolated from pharyngeal swabs from mink with well developed pulmonary lesions and from rectal swabs only from mink with advanced lesions.

Most cases of pseudomonas pneumonia in mink in the United States can be associated with two serotypes of P. aeruginosa Fisher immunotypes 1 and 7 (O antigen groups G and B, respectively). Other serotypes are occasionally found. In some outbreaks in which a number of isolates were typed, more than one serotype has been identified. This was especially true in herds in which a low number of mink died over an extended period.

P. aeruginosa appears to be a common inhabitant of mink even in herds without a history of pseudomonas pneumonia. We have often isolated the organism from the rectum of normal mink. These isolates are usually of the same serotypes that are associated with pneumonia. We have also examined mink from commercial herds for antibody to P. aeruginosa. Sera were obtained at pelting and examined for antibody to P. aeruginosa lipopolysaccharide antigen by an anzyme-linked immunosorbent assay (ELISA). With this assay, we detected antibody in mink sera that was non-reactive when tested by passive hemagglutination using the same antigen. Mink with antibody detectable by ELISA were found in all herds tested. Even in herds without a history of the disease, up to 20% of the mink had antibody to the serotypes of P. aeruginosa common to mink.

Vaccination protects mink from experimental challenge with P. aeruginosa and during field outbreaks of pseudomonas pneumonia. Japanese workers have demonstrated the efficacy of the common antigen (OEP) of P. aeruginosa and toxoids of protease and elastase in vaccinating

mink against infection with P. aeruginosa. We have used the lipopolysaccharide natigens of P. aeruginosa (Pseudogen Warner Lambert, Detroit, Michigan) for vaccination trials. In laboratory trials, 2 doses of lipopolysaccharide vaccine protected mink from intratracheally. The lethal dose for nonvaccinated mink was 10^3 but varied with the presence of naturally acquired antibody. A single dose of vaccine, given 2 weeks to 4 months before challenge was not as efficacious, but did offer some protection. Mink surviving challenge infection had no significant lung lesions when examined 2 weeks after challenge. The lipopolysaccharide vaccine is effective, but may be deficient in that an occasional mink isolate of P. aeruginosa from mink does not fit into the immunotype schema.

The variability in mortality and appearance of the disease on various ranches, and the number of apparently healthy carriers of P. aeruginosa led to the hypothesis that other factors may play a role in the pathogenesis of pseudomonas pneumonia. This was reinforced by the fact that in most other species, infections with P. aeruginosa are localized, or are secondary to other disease processes. Some individual cases of pseudomonas pneumonia, in mink occur in association with Aleutian Disease viral infection (AD). However, we could find no correlation in the field between the incidence of AD within a herd (tested by CEP) and the occurrence of pseudomonas pneumonia. The two diseases often occur independently.

Attempts at virus isolation from tissues of mink dying of pseudomonas pneumonia were negative. Pharyngeal and rectal swabs were obtained from mink housed adjacent to cages in which mink had died of naturally occuring pseudomonas pneumonia.

A picornavirus was isolated from pharyngeal swabs of mink from 2 ranches. This virus shares some of the physical characteristics of the calicivirus group but has not been definitely identified. It does not cross-react serologically by neutralization with antibody to the known caliciviruses.

An extensive serologic survey of mink from the United States and one herd from Japan was conducted. In all herds tested, most of the animals had serum antigody to the virus as measured by viral neutralization. There was no correlation between the occurrence of antibodies to the picornavirus and to AD.

Experimental infection of mink kits and ferrets with the picornavirus resulted in the development of viral neutralizing antibody in the ferrets and an increase in antibody titer in the mink. No clinical disease of lesions were detected. The negative results in the mink may not be valid as the test mink had low levels of neutralizing antibody before infection, and mink from the same group experienced seroconversion naturally one to two months later. Clarifrcation of the role of the mink calicivirus in producing disease, both alone and in association with P. aeruginosa will depend upon finding or producing mink without evidence of prior infection.

Second International Scientific Congress

The following is a list of some of the papers given at the 2nd International Scientific Congress in Fur Animal Production, Denmark 1980 —

1. A Pot Pourri of Disease Problems in Nova Scotia — Mink and Foxes. G.G. Finley, D.V.M. Canada
2. Some Significant Changes in Management on Northwood Fur Farms. A.A. Rietveld, General Manager. U.S.A.
3. Objectivisation of Methods of Exterior Evaluation of Standard Mink. Maciejowski Janusz, Slawon Jerzy.
4. Genetic Parameters of Some Traits in Mink and The Opportunity to Use Them in Fur Improvement. Dr. N. Pasternac, Romania.
5. Early Growth Performance of Dark and Pastel Kits At the Northwood Ranch. 1971–75. W.L. Leoschke, U.S.A.
6. Morphological and Histological Characteristics of Fur Defect "Metallic" — the number of cuticle layers, histology of hair follicles and mineral content of long guard hairs. Blomstedt Leena and Lohi Outi. Finland.
7. Towards a More Efficient Mink Production. Rafael Garcia Mata, Argentina.
8. Prenatal and Early Postnatal Mortality in Mink. Enai J. Einarsson, Norway.
9. Amino-Acid Profile of the Plasma, Pelt and Hair of Adult Mink. E.R. Chavez. Canada.
10. Amino-Acid Digestibility in Mink. Anders Skrede, Norway.
11. Lecithin Enriched Vegatable Oils in Mink Nutrition. J. Hertrampf, West Germany.
12. A Survey of Vitamin Status in Mink of Various Age, Sex and Pelt Qualities in France & Yugoslavia.
13. Technique of Feeding Pellets to Mink. A. Allain, J. Rougeot, G. Charlet-Lery and J.J. Sabaut. France.
14. Enzyme Linked Immunosorbent Assay of A.D. Viral Antibodies. P. Wright, F.J. De Pauli and B.N. Wilkie. Canada.
15. Experimental Food Poisoning in Mink. T. Juokslahti.