TITLE: FIELD STUDIES: PSEUDOMONAS PNEUMONIA OF MINK

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PUBLICATION: AM J VET RES

DATE: 1981

SUMMARY

Epizooties of pneumonia in mink caused by Pseudomas aeruginosa were investigated to characterize the serotype of organisms and to identify possible predisposing factors. Most epizooties were associated with P aeruginosa Fisher serotype 1, and a few were associated with 3 other serotypes. There were no predisposing factors identified that could be used to differentiate farms affected and those not affected with pseudomonas pneumonia.

Cultural studies indicated that P aeruginosa was present in mink from affected and non affected herds. Organisms isolated included serotypes associated with naturally occurring disease. Serostudy results were similar among herds. A prospective field vaccination trial did not yield definitive results, since only slight losses occurred in both vaccinated nonvaccinated mink. Significant levels of antibody were detected in mink 15 to 17 weeks after they were given a e dose of P aeruginosa lipopolvsaccharide vaccine.

Pseudomonas pneumonia of mink (hemorrhagic pneumonia) was first reported from Denmark in 1953.' The disease has since been reported with a worldwide distribution. 2-7 Losses vary greatly, from 0.1% to 50% of the affected herds. (4.5) Many reports identified higher losses among kits than adults. (1.3.5.8.9) In some instances, mortality was greatest among male kits. 5,6 Pseudomonas pneumonia usually occurs during the autumn months, September through November. (1-6,8,9) During this period, the majority of animals on a commercial farm will be kits (4 to 6 months old).

The distribution of diseased mink on a farm may be sporadic or localized. In many epizootics, new cases appear in mink in the same cage or adjacent cages.(1,3) In one instance, (10) the direction of spread was with the prevailing wind. Losses may also have an apparently random distribution.

The clinical course in diseased mink is short and often unobserved. Mink are often anorectic the day before death. (2-1) Affected mink may be listless and show tachypnea. 4 Clinical

signs within the few hours preceding death included dyspnea, expulsion of red frothy fluid from the nares, gurgling sounds, and incoordinated locomotion. 2-4

Gross lesions have been similar in most cases. (1-4,7,9,11) Sanguineous exudate was present around the nose and mouth. The lungs were diffusely hyperemic, often with I or more consolidated lobes. Hemorrhagic pleural exudate has been reported. Bronchial lymph nodes were large and hyperemic. Microscopic lesions in the lung reflected the gross changes and ranged from inflammatory hyperemia and necrosis to acute consolidation, necrosis, and hemorrhage. (7,9,11) Bacteria morphogogically consistent with P aeuginosa were numerous in affected lungs.

Cross-agglutination tests of isolates from 13 separate epizooties of pseudomonas pneumonia in Scandinavia indicated that the isolates belonged to 2 SerotypeSI2 which were not further identified. Serotypes of P aeruginosa associated with pneumonia in mink have only recently been identified in a manner that allows cross-referencing. In Japan, several isolates have been identified as serotype 8 of Homa (O antigen group G).8

Little work has been done to identify factors that may predispose mink to infection with P aeruginosa. On 3 farms in Japan, the frequency of Aleutian disease (AD) viral infection was determined by the iodine agglutination test. 5 The farm with the largest number of agglutination positive-test mink also had the highest number of losses due to pseudomonas pneumonia. The agglutination test results of farms not experiencing pseudomonas pneumonia were not reported.

The purpose of the present study was as follows: (i) identify serotypes of P aeruginosa associated with pneumonia in mink in North America, (ii) determine whether P aeruginosa is present in normal mink, (iii) characterize disease epizooties to determine whether any predisposing factors could be identified, and (iv) evaluate a prospective vaccination trial under field conditions.

Materials and Methods

Survey of Stereotypes of Infective Strains: Isolates of P aeruginosa from 22 epizootics of mink pneumonia throughout the United States and Canada were obtained in the period 1975 through 1978. These isolates were identified by production of fluorescent pigment, characteristic reaction on triple sugar iron agar, growth at 41 C, and production of cytochrome oxidase and pyocyanin. Isolates were serotyped by the Fisher Immunotype schema, 13 with

antisera provided by Warner-Lambert/Parke Davis (in Detroit) and by the Habs schema, with commercially available antisera.

Field Survey: In 1977, 4 commercial mink ranches in Washington and Idaho were specially studied. Ranches A and B had had epizootics of pseudomonas pneumonia, and ranches C and D had been free of the disease. Ranches B and C were approximately 1 km apart, and movement of personnel between the rances was common.

Management practices on the 4 ranches are common to commercial mink ranches. Feed was commercially available cereal, mixed on the premises with fish, poultry byproducts, and other protein sources as available. Approximate September mink populations were as follows. Ranch A, 12,000-, ranch B, 10,000-, ranch C, 10,000; and ranch D, 5,000. Negligible numbers of mink with the Chediak-Higashi syndrome (CHS) 14 phenotype were present.

Laboratory samples were obtained at pelting (November 22 to December 5). Blood samples were collected from carcasses immediately after settings. Swabs from the pharynx and rectum were plated on pseudomonas isolation agar b; isolates were identified and serotyped. Sera were tested for antibody to AD virus by immune counterelectrophoresis (CEP).(15) Sera were also tested for antibody to P aeruginosa lipopolysaccharides (LPS), Fisher serotypes I and 7, by passive hemagglutination (PHA).(16) Sera were tested untreated and treated with 2-mereaptoethanol (2-ME) to inactivate inununoglobulin (1g)M. Sera were tested for antibody to P aeruginosa exotoxin A by PHA.(17)

Vaccine Trial: In 1978, mink on ranches A and D were vaccinated with heptavalent P aeruginosa LPS vaccine. On ranch A, approximately half of the herd, including representative numbers of all ages and both sexes, was vaccinated. On ranch D, 50 female kits were vaccinated and 50 female kits in the same shed were designated as controls. The vaccine (0.3 ml) was given subcutaneously in the thigh; this was done between July 29 and August 7. All mink dying between vaccination and pelting were necropsied. Samples were obtained at pelting as previously described. Sera from vaccinated and nonvaccinated mink were tested for antibodies to P aeruginosa LPS by PHA and by enzyme-linked immunosorbent assay (ELISA) (11); the ELISA results were expressed as the mean optical density (OD) of test wells minus plate controls.

RESULTS

FIELD SURVEY

Serotype of Infective Strains: The occurrence of various serotypes of P aeruginosa from 22 epizooties of mink pneumonia is shown in Table 1. Relationships among various typing schemes are also shown. Isolates from ranches A and B in 1977 are included. More than I serotype was found in 2 of the ranches. The majority of isolates were Fisher serotype I (O antigen group G). Group B organisms were the next most common.

Description of Epizooties - Ranch A – Deaths due to pseudomonas pneumonia occurred on ranch A from early September to early November 1977. Losses were sporadic but seemed somewhat more numerous in 1 shed during the last 2 weeks of September. Both adults and kits were affected, with a predominance of males. Total loss due to pseudomonas was approximately 130 (about 1%). Mink were rarely seen to be ill, but were found dead with a pink blood tinged exudate around the nose and mouth. Some of the mink were anorectic the day before death.

Seven mink (5 male and 2 female) were necropsied. The gross appearance of the lungs varied from diffuse hyperemia (1 mink), red consolidation (3 mink), or tan to gray consolidation (3 mink). The consolidation involved 1 to 3 lobes of the lung, while the other lobes were hyperemic and edematous. Microscopically, the lungs reflected the gross morphologic changes and were similar to those reported previously. (7,9,11)

Four of the mink (2 males and the 2 females) had microscopic lesions consistent with those produced by Ad viral infection. (18) The female mink with pulmonary hyperemia also had areas of necrosis in the liver and subacute meningitis. One of the male mink with lesions Of AD had hemorrhagic pericarditis and lesions of chronic passive congestion in the liver. Pseudomonas aeruginosa was recovered in pure culture from the lungs of all 7 mink and from the pericardial exudate of the mink with pericarditis. Four of these lung isolates were serotyped as Fisher serotype 1. Microbiological cultural examination of drinking water from the main water line before entry into the mink shed did not reveal any P aeruginosa.

Fatal pseudomonas pneumonia occurred in 1 mink in March 1978: a yearling male, dying within hours after he had been noticed to be depressed. The lung changes were predominately of acute (red) consolidation, and there were no lesions Of AD. Pseudomonas aeruginosa (Fisher type 3/ 7) was recovered from the lung.

Ranch B - Losses due to pseudomonas pneumonia on ranch B during 1977 were similar in time of occurrence and total loss (approx 1%) to those on ranch A. Most of the losses were concentrated in two 1-week periods in September. Early losses occurred in mink of both sexes and all ages, but were most severe among male kits in 1 shed. Many of the mink were anorectic and depressed for 1 to 2 days before death, and some of them had exaggerated respiratory movements.

Necropsies were conducted on 8 males and 2 female mink that had died over a 7-day period. In all, 1 or more lobes of the lung were consolidated and red-tan to gray. The remainder of the lung was congested and edematous find sometimes contained irregular hemorrhages. Lymph nodes and spleens were often large; other gross lesions were not seen. Lungs from 4 of these mink yielded pure cultures of P aeruginosa Fisher type 1.

Field Serosurvey –The occurrence of antibody to AD virus, P aeuginosa LPS 1 and 7, and exotoxin A are shown in Table 2. Fourteen of 75 mink with antibody to LPS 1 and/or 7 were present on ranch A, and 15 on ranch B had similar antibodies. There were fewer mink with anti-LPS antibody on ranches C and D, but the difference is not statistically significant. Ranches B and C had almost the same number of mink with antibody to AD virus, whereas ranches A and D differed greatly. Antibody to exotoxin A was detected in only 3 mink.

Field Survey-Bacteriologic Cultural Examination –The pseudomonas organism was isolated from 14 of 75 mink on ranch A. The isolates were from the rectum of these mink, and from the pharynx of I mink. Eleven isolates were 0 antigen type B, I isolate was 0 antigen type G, and 2 could not be typed. One mink on each of ranches B and D had P aeruginosa 0 type G isolated, I from the pharynx and I from the rectum. The organism was not isolated on ranch C.

VACCINATION TRIAL

Serotest Results –The differences in ELISA OD values for P aeruginosa LPS I and 7 between vaccinated and non-vaccinated mink of ranches A and D are shown in Table 3. Average ELISA results for each treatment and antigen tested for were similar on both ranches. Values for vaccinated mink were significantly greater than those for non vaccinated mink ($P < 0.05$, Student's t test).

The number of mink with antibody to LPS I detectable by ELISA, PHA, and IPHA + 2-ME are shown in Table 4. An ELISA-positive reaction for LPS I was determined to be OD> 0.5 by previous studies.(11) There was a significant difference between vaccinated and non-

vaccinated groups in the lumbers of mink positive by ELISA ($P < 0.01$ binomial confidence limits). Fewer mink with antibody were detectible by PHA, and a significant difference between vaccinated and non-vaccinated mink was present only on ranch D tested by PHA without 2-ME.

The response of mink to the other LPS types as tested by PHA was similar to the response of LPS type 1. Differences were noted between antigen types. The antigen types with largest numbers of reactors in the vaccinated groups tended to be associated with the largest number of positive mink in the non-vaccinated groups.

The ELISA results for LPS types 2 through 6 were similar to those for LPS type 1 (Table 3). The saturation limit of the ELISA test system was approached by many mink in the vaccinated groups. A few mink in the non-vaccinated groups had ELISA-positive reactions to 1 or more LPS types.

Bacteriologic Cultural Examination-Isolates of P aeruginosa from mink at petting are shown in Table 5. Fewer isolates were made from mink in the vaccinated group, but the differences were not significant. On ranch A. there appeared to be a trend from isolates falling within the Fisher typing scheme in the non-vaccinated mink to non-Fisher serotypes in the vaccinated mink. All isolates on ranch D were of the 2 Fisher serotypes commonly associated with pneumonia in mink (type 1 and type 3/7). Most isolates were from rectal swabs and a few isolates were from the pharynx; occasionally isolates were obtained from both sites. There was no serotype from consistently in either site.

Pneumonia –On ranch A, from the time of vaccination to pelting, 29 mink died. Of these, 9 could be verified as having died of pseudomonas pneumonia-6 from the vaccinated group and 3 from the non-vaccinated group. The distribution of serotypes was as follows: 0 antigen B (Fisher 3/7)-2 vaccinated; 0 group D (no Fisher type) -I each group; 0 group G (Fisher type I) -2 each group; and untyped -I vaccinated.

On ranch D, no mink deaths were recorded in the test shed, and deaths attributable to pseudomonas pneumonia were not recognized on the farm.

Discussion

Pseudomonas pneumonia of mink is caused by several serotypes of P aerugitiosa, but the majority of epizooties appear to be associated with organisms of Fisher serotype I (O antigen

group G). Vaccination with heptavalent P aeruginosa vaccine (16) would provide protection against the majority of isolates associated with infection. Addition of antigens of 0 type D organisms would enhance the range of protection. Combined vaccination with toxoids may help overcome problems of strain specificity. (19)

Factors predisposing mink to infection by P aeruginosa have not been characterized. Pseudomonas infections in persons are usually associated with predisposing conditions, such as hematologic malignancies and immune deficiency states, burn wounds, chronic heart and lung disease, cystic fibrosis, and prematurity.20-23 In contrast, pseudomonas pneumonia of mink often effects animals that otherwise appear to be in good health.

Aleutian disease is a chronic viral infection of mink that causes severe emaciation, anemia, hypergamrnaglobulinemia and humoral immunosuppression in terminally ill mink.18 Cellular immune responses may also be depressed. The present serostudy did not reveal any consistent differences in AD antibody status between herds with and without pseudomonas pneumonia. The CEP test performed at standard dilutions demonstrates past infections with the virus, but does not indicate the severity of the infection. It has been shown that 30% to 40% of mink without CHS may become CEP positive after infection, but do not develop hypergammaglobulinemia or lesions Of AD. 25,26 On ranch A during, the epizootic in 1977, it appeared as if mink with well-developed lesions of AD had more severe or disseminated disease due to P aeruginosa. Thus, advanced viral infection may increase the frequency or severity of infection by P aeruginosa. However, CEP-negative mink without demonstrable antibody to P aeruginosa are susceptible to challenge infection with relatively few organisms.11 Further work is necessary to determine whether AD renders mink more susceptible to infections by P aeruginosa, both on an individual and a herd basis.

Mink with CHS appear to be more susceptible to infection with certain bacteria (27) and are definitely more susceptible to AD.18 Although mink with eRs have been reported to become affected with pseudomonas pneumonia, (5,9,11) other phenotypes of mink also succumb to the disease. 2.3 In the present study, all of the losses examined were in non-CHS phenotype mink.

There may be other differences in susceptibility among strains, sexes, ages, or individual mink. On ranch B, the predominance of losses involved male kits, and this is also the case in several reports.(5,6,17) The losses on ranch A did not follow a definite trend.

Most epizootics of pseudomonas pneumonia occur in the autumn months. A number of factors are present on mink ranches at this time of year:

(1) Most of the population consists of kits and the total population is approximately 4 times what it was before whelping.
(2) Maternal antibodies, if present in kits, would be dissipated by this time.
(3) The weather during this time may be cool and/or damp.
(4) Mink are growing a winter pelt and kits are gaining in body size. This is generally considered to be a stressful period.

These factors occur simultaneously and cannot be separated under field conditions.

Pseudomonas aeruginosa is common in mink. The organism was obtained by cultural techniques from normal mink of herds affected and not affected by pseudomonas pneumonia. A large number of carriers was found on ranch A in both years and on rand D in 1978. The serotypes identified were generally similar to those associated with pneumonia, except in the vaccinated group on ranch A in 1978. It was not determined whether the apparent shift in serotypes between vaccinated and non-vaccinated mink on ranch A was due to the vaccine or an affect of different areas within the ranch.

Most of the infections detected at pelting were subclinical, since disease attributable to P aeruginosa was not recognized in these animals. In addition, only a few mink were detected with antibody to exotoxin A. Presence of antibody to exotoxin A in persons is associated with age, presumable due to repeated exposure, or with severe infections.(17, 29)

Determination was not done on whether there is a difference in virulence between isolates P aeurginosa from healthy mink and mink with pseudomonas pneumonia.This would coincide with studies to characterize virulence factors of P aeruginosa for mink. Various toxins have a role in the pathogenesis of infection by P aeruginosa.28 Indirect evidence of the importance of toxins in infection of mink by P aeruginosa has come from studies showing resistance of toxoid-vaccinated mink to experimental challenge infection.19

Serotest results indicated that antibody to P aeruginosa was present in mink from affected and non-affected herds. Emphasis was placed on detection of antibody to Fisher types I and 7, because these are most commonly associated with infection in mink. However, antibody to other types was also present and responses to various serotypes were similar between herds. The relationship between ELISA and PHA results was similar to that reported previously.(11) The ELISA test detected more mink with antibody. Mink with naturally acquired antibody to P

aeruginosa Fisher tvpe, 1 are resistant to experimental challenge infection.(11). A few mink in each herd were found to have antibody levels that would indicate probable resistance to infection. Infection by a non-virulent strain, or enteric infection by a virulent strain may render a mink resistant to respiratory tract infecting by a virulent strain. The probability of previous exposure and, thus, resistance would decrease susceptibility in adult mink.

The ability of the heptavalent P aeruginosa LPS vaccine to confer immunity under field conditions was not assessed. Only a few mink on ranch A died of pseudomonas pneumonia. Most of the mink that died were in the vaccinated group. However, a few mink in this group may not have been vaccinated, or may not have responded. Two mink in the vaccinated group had a low ELISA OD to Fisher type 1 antigen. It is significant that the majority of vaccinated animals had antibody levels at pelting that presumably would be protective against Fisher type I organisms. This antibody level was maintained 14 to 17 weeks in minks given a single vaccination.

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TABLE 1—Serotype of Isolates of *Pseudomonas aeruginosa* from Mink with Pseudomonas Pneumonia, 1975–1978*

Serotyping scheme				
O antigen group	Fisher LPS type	Habs (Difco) type	No. of ranches†	No. of isolates
A	...	3	0	0
B	3, 7	2, 5, 16	5	9
C	6	7, 8	2	2
D	...	9	1	1
E	2	11	0	0
F	...	4	0	0
G	1	6	16	35

* Includes isolates from ranches A and B, 1977.
† Total No. of ranches = 22. Two ranches had 2 serotypes each.

TABLE 2—Serostudy of *P aeruginosa* Pneumonia in Mink, 1977

	No. of mink with antibody to AD virus and antigens of *P aeruginosa*			
Ranch*	AD†	LPS 1‡	LPS 7‡	Exotoxin A
A	73	4	11§	0
B	50	15	0	1
C	49	2	8§	0
D	10	5	5	2

* 75 mink were tested per ranch. † AD virus, tested by immune counterelectrophoresis. ‡ LPS of *P aeruginosa*, Fisher types 1 and 7. § 1 mink with antibody to both LPS types on each of ranches A and C.

TABLE 3—Comparison of ELISA Results Between Vaccinated and Non-vaccinated Mink, Field Vaccine Trial

Fisher LPS type	Ranch A		Ranch D	
	Nonvaccinated	Vaccinated	Nonvaccinated	Vaccinated
1	0.385 ± 0.742	2.225 ± 0.795	0.426 ± 0.941	2.347 ± 0.652
7	0.080 ± 0.132	0.401 ± 0.285	0.018 ± 0.027	0.497 ± 0.347

ELISA results are expressed as mean ± SD of average ELISA OD readings for 25 mink/group

TABLE 4—Mink with Antibody to *P aeruginosa* LPS 1 by Various Tests, Field Vaccine Trial

	Ranch A		Ranch D	
Serotest	Nonvaccinated (n = 25)	Vaccinated (n = 25)	Nonvaccinated (n = 25)	Vaccinated (n = 25)
ELISA positive	6	23	4	25
IHA positive	1	8	1	15
IHA + 2-ME positive	0	6	1	9

TABLE 5—Cultural Isolation of *P aeruginosa* from Mink at Pelting, Vaccine Trial

		Ranch A		Ranch D	
O Antigen type	Fisher LPS type	Nonvaccinated (n = 25)	Vaccinated (n = 25)	Nonvaccinated (n = 25)	Vaccinated (n = 25)
A	...	0	1	0	0
B	3, 7	3	0	5	4
D	...	1	0	0	0
E	2	2	0	0	0
F	...	0	6	0	0
G	1	7	3	4	1
Untyped	...	1	1	0	0
Total		14	11	9	5

Data are expressed as No. of mink yielding the various serotypes of *P aeruginosa*