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Hemorrhagic pneumonia in mink is an acute and fatal disease caused by Pseudomonas aeruginosa. The mink are typically found dead without prior clinical symptoms. The disease can be highly contagious and varying mortalities on the farm level has been reported. Hemorrhagic pneumonia in mink is seasonal with outbreaks almost exclusively occurring from September to November in Denmark. In human medicine, P. aeruginosa is regarded as a pathogen for immune compromised individuals but no underlying disease or immune defect has been identified in mink dying of hemorrhagic pneumonia. In fact, little research has been performed in this field and most published work is more than 25 years old.

The studies presented in this thesis aim at elucidating varying aspects of the disease:

**Article I** investigates the relationships of P. aeruginosa isolated from mink hemorrhagic pneumonia using pulsed field gel electrophoresis (PFGE) and a commercial typing system based on single nucleotide polymorphisms (SNP) on chosen strains.

The results presented in this article show that 70% of P. aeruginosa isolated from outbreaks of hemorrhagic pneumonia in mink consist of unique strains, while the remaining 30% belongs to either a cluster of closely related strains or unrelated but prevalent strains. This indicates that most outbreaks of hemorrhagic pneumonia are caused by environmental isolates and not by strains specially adapted to mink which spread among mink farms.

**Article II** compares the histopathological lesions in hemorrhagic pneumonia caused by P. aeruginosa and E. coli in diagnostic material. The distribution of the two pathogens is visualized using fluorescence in situ hybridization (FISH).

Two histological patterns were observed in the work presented in Article II; one was very hemorrhagic with few bacteria while the other was dominated by necrosis, neutrophils and massive amounts of bacteria. The hemorrhagic pattern was predominantly seen in P. aeruginosa infected lungs and this bacterium showed a preference for perivascular localization while alveolar edema was more frequently identified in hemorrhagic pneumonia associated with E. coli infection. The perivascular localization, tendency for a higher frequency of a very hemorrhagic response and alveolar edema were the only differences noted between hemorrhagic pneumonia caused by P. aeruginosa compared to E. coli.

**Article III** describes an infectious dose trial on mink. This experiment was performed in July and in November to elucidate whether the same infectious dose would be able to cause hemorrhagic pneumonia both in and out of the reported season for development of hemorrhagic pneumonia. Furthermore the ability to recover P. aeruginosa from the nasal mucosa of experimentally infected mink and from farm mink was investigated.

The results from this study suggest that some mink are predisposed to develop hemorrhagic pneumonia since wide ranges in infectious dose leading to hemorrhagic pneumonia were observed and a tendency towards greater mortality in November as compared to July could be demonstrated. It was possible to culture P. aeruginosa from the nasal mucosa up till eight days after experimental infection but no P. aeruginosa was ever recovered from the nasal mucosa of farmed mink.

**Article IV** focuses on the presence of respiratory syncytial virus (RSV) as a possible explanation for the high susceptibility of mink to development of hemorrhagic pneumonia due to P. aeruginosa. Polymerase chain reaction (PCR) was used to examine 50 lung tissue samples for the presence of both the bovine and the human type of RSV.

The results of this work showed that it was not possible to detect RSV in mink lung samples with hemorrhagic pneumonia caused by P. aeruginosa.

Results from investigations not included in articles are also presented. These include interviews with farmers experiencing outbreaks of hemorrhagic pneumonia in their mink, an attempt to culture P. aeruginosa from the farm environment and histology of tissue from the experimentally infected mink described in Article III.

It was only possible to culture P. aeruginosa from the farm environment in one case on a farm with an outbreak of hemorrhagic pneumonia. The most striking conclusion of the interviews with farmers experiencing outbreaks of hemorrhagic pneumonia among their mink was that the disease always started in the mink kits, never in the adults. Furthermore, 39% reported that most deaths occurred in the male mink.

The results presented in this thesis suggest that factors of the mink make them more prone to develop hemorrhagic pneumonia due to P. aeruginosa in the fall. This is based on the discovery that most outbreaks are not due to a special P. aeruginosa strain with an increased virulence for mink, but rather local strains. The observed tendency of mink to develop hemorrhagic pneumonia in the autumn irrespective of infectious dose and the difficulty in isolating P. aeruginosa in the farm environment also points to something in the mink as being crucial in the development of disease.

**General Information**

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