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Retrospective Study of Diseases in a Captive Lemming Colony

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ABSTRACT: Fifty-four ill or nonproductive lemmings (*Dicrostonyx* spp.) were evaluated for signs, lesions and causes of disease for 5 yr in a domestic colony. Parasitic granulomas caused by *Encephalitozoon cuniculi* were the most common finding and were seen in 22 lemmings. The disease was characterized by circling and torticollis with granulomas in many tissues, especially the central nervous system. Suppurative otitis occurred in 12 lemmings and was associated with *Klebsiella pneumonia* infection; circling was the common sign. Hepatic microabscesses were present in seven lemmings but a cause was not identified. Five lemmings had neoplasms and 14 had either suppurative processes, aspermia, or ovarian cysts.

Key words: Lemming, *Dicrostonyx*, encephalitozoonosis, hepatic microabscesses, neoplasms, disease.

Lemmings are small arctic rodents of the genera *Lemmus*, *Myopus*, *Synaptomys*, and *Dicrostonyx* (Nowak and Paradiso, 1983). Members of the genus *Dicrostonyx*, also called collared or varying lemmings, have been used in research as a model animal for studies in brucellosis (Dieterich, 1975). Our interest in the lemming resulted from frequent mortality, often associated with a nervous disorder, in the breeding colony at the National Animal Disease Center, Ames, Iowa (USA). The newly established colony initially consisted only of the species *Dicrostonyx stevensoni* and was gradually replaced over 5 yr by the species *Dicrostonyx rubricatus*. The lemmings originated from Umnak Island (53°15'N, 168°20'W), and were supplied by the Arctic Health Research Center, Department of Health, Education and Welfare, Fairbanks, Alaska (USA) and the Institute of Arctic Biology, University of Alaska, Fairbanks. We previously reported encephalitozoonosis in the colony (Cutlip and Beall, 1989). Our objective in this retrospective study was to document other

diseases seen in the captive lemming colony over a 5-yr period.

Husbandry was similar to that reported by Dieterich (1975). Breeding pairs were housed together in laboratory-constructed plastic cages (18 × 28 × 46 cm) equipped with wire mesh tops. Wood shavings and cotton were used for bedding and nesting materials. Weekly, the cages were washed and disinfected (Wexford Labs Inc., Kirkwood, Missouri, USA). Secondary housing was a concrete-block building with the temperature at 12.5 to 18.5 C, relative humidity at 35% of maximum, and a lighting cycle of 20 hr per day. Cage light was at 70 to 110 candelas. The diet, fed *ad libitum*, was lettuce, carrots, wheat germ, sunflower seed, oats and complete rabbit chow (Code# 5315, Ralston Purina Company, Minneapolis, Minnesota, USA). The lemmings were checked at least daily. The mean monthly population of mature lemmings over the last four yr of the study period was 30 males and 31 females (SE = 1.28 for females and 0.64 for males). Replacement breeders were chosen either from the most prolific and healthy of the colony or purchased from the source. Replacement schedule was dictated by the loss of breeding adults through either usage or disease.

Fifty-four lemmings were examined for type and cause of disease; 38 were moribund or had other clinical signs of disease, eight were found dead, six were barren breeders and two had no clinical signs. Live lemmings were anesthetized with ethyl ether and killed by exsanguination. Animals ranged from one to 16 mo old and included both sexes. Post-mortem examination was done as soon as possible after death. Brain, heart, lung, liver, kidney, spleen, and any tissues with macroscopic

lesions were fixed in neutral buffered formalin, embedded in paraffin, sectioned at 6 μ m, and stained with Harris' hematoxylin and eosin. Suppurative exudates were collected on cotton swabs and cultured aerobically at 37 C on 5% defibrinated bovine blood in trypticase soy agar (Baltimore Biological Laboratory, Inc., Cockeysville, Maryland, USA). Bacterial isolates were identified by the methods of Cowan (1974) and Edwards and Ewing (1972).

Torticollis was the most frequent clinical sign ($n = 12$ animals) followed by rapid circling ($n = 9$), posterior paralysis ($n = 7$), lethargy ($n = 6$), infertility ($n = 6$), diffuse suppurations of cutaneous and subcutaneous structures of the head and neck including the eyes ($n = 5$), and palpable masses ($n = 3$). Eight lemmings were found dead and two had no discernible signs. Of the lesions observed, parasitic granulomas ($n = 22$), suppurative otitis ($n = 12$), hepatic microabscesses ($n = 7$), and neoplasms ($n = 5$) were the primary lesions. Miscellaneous lesions were seen in 14 lemmings and no lesions were seen in seven lemmings. Details of the granulomas have been reported (Cutlip and Beall, 1989). Suppurative otitis usually was associated with circling and the purulent exudate was usually confined to the middle ear but was present in all auditory structures, temporal bone and adjacent meninges in some lemmings. *Klebsiella pneumonia* was isolated from exudate taken from the ears of six of seven lemmings. Hepatic microabscesses were multifocal, irregularly distributed, and had necrotic centers. They were not associated with a recognized clinical entity or a cause. Neoplasms included two hepatic adenomas, two mammary adenocarcinomas, and one lacrimal adenocarcinoma. Miscellaneous lesions were aspermia, ovarian cysts, and lymphocytic encephalitis, as well as suppurative conjunctivitis, dacryoadenitis, rhinitis, dermatitis, pleuritis, and pneumonitis. A variety of bacteria, including *Escherichia coli*, *Alcaligenes fecalis*, *Proteus rettgeri*, *Acinetobacter calcoaceticus*, *Streptococcus*

bovis, *Staphylococcus epidermidis* and *Klebsiella pneumonia*, were isolated from the suppurative exudates.

We have observed four syndromes of clinical importance in the lemming colony: generalized protozoan disease, suppurative otitis, multifocal hepatic abscesses, and neoplasia. The protozoan was identified as *Encephalitozoon cuniculi* (Cutlip and Beall, 1989); in contrast to most rodents, the clinical response in the lemming was severe and closely resembled that in carnivores where death is common (McCully et al., 1978; Shadduck and Pakes, 1978). Suppurative otitis was reported by Dieterich (1975) as occurring frequently in lemmings and was a severe problem in our colony; we found *K. pneumonia* to be the etiology. Suppurative processes of the eyes and subcutaneous tissues of the head and neck and respiratory tissues were associated with several types of bacteria, but no type was consistently isolated. Hepatic microabscessation was a severe disease but infrequent in occurrence. Lesions in the liver were similar to those in other species with Tyzzer's disease (Cutlip et al., 1971) but typical elongated bacilli were not found in the lemmings. No bacteria were isolated and the etiology was not established. We confirmed the high prevalence of mammary adenocarcinoma reported by Van Pelt and Dieterich (1972) but did not find the chronic nephritis reported by Dieterich (1975) in captive-reared lemmings. Except for aspermia and ovarian cysts, we found no definite age, sex, or species predilection for any of the diseases; however, sex and age were not recorded for all lemmings and some data were scant. Information about diseases in free-living lemmings is slight. Several infectious diseases have been reported in true lemmings, *Lemmus lemmus*, but details are minimal (Wiger, 1971; Kornilova et al., 1975; Kataev et al., 1983; Shakhmatova and Litvinov, 1992). These diseases include tularemia, leptospirosis, listeriosis, Q-fever, arbovirus infection, Asian tick-borne rickettsiosis, trypanosomiasis, and infestations

of *Mastophorus muris* with hepatic migration. Very little has been reported about diseases, including those we report here, in free-living collared lemmings. Mites, lice and cestodes have been reported from collared lemmings of the Northwest Territories of Canada (Webster, 1974; Gill and Strandtmann, 1977).

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