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# PATHOLOGY OF OCULAR LESIONS IN FREE-LIVING MOOSE (*ALCES ALCES*) FROM SASKATCHEWAN

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ABSTRACT: Clinical signs of impaired vision or neurological disease occurred in seven of 74 free-living moose (*Alces alces*) from Saskatchewan, Canada, submitted for necropsy between 1969 and 1994. Several lesions were found in each eye, including retinal degeneration (seven cases), cataract (six cases), lymphocytic-plasmacytic anterior uveitis (six cases), corneal scars (six cases), keratitis (four cases), and microphthalmia (one case), but their cause was not determined. *Moraxella bovis* was isolated from the cornea of one moose. Lesions in the brain and spinal cord were mild or absent.

*Key words:* Pathology, ophthalmology, moose, *Alces alces*, keratitis, uveitis, cataract, retinal degeneration, microphthalmia, atrophia bulbi, *Moraxella bovis*.

## INTRODUCTION

Ocular lesions in free-living moose (*Alces alces*) may be important because, depending on their severity, they may cause blindness and lead to death. However, little is known about the ocular pathology of moose. Kurtz et al. (1966) found foci of ova and larvae, presumably of *Parelaphostrongylus tenuis*, in the optic disc of a free-living moose from Minnesota (USA) with granulomatous reaction in the surrounding tissue. The optic nerve had foci of swollen axons, demyelination, and gliosis. In the same eye there were numerous larvae in the area of the retinal pigment epithelium, with minimal reaction in the retina, but some congestion and plasma cell infiltration in the choroid. However, Saskatchewan, Canada, lies beyond the known range of *P. tenuis*, which has not been found in moose further west than southeast Manitoba, Canada (Lankester, 1974). Kronevi et al. (1977) found cataracts in Swedish moose, but could not determine the cause.

For the above reasons, in this retrospective study we describe the ocular and neurological lesions found in free-living moose submitted for necropsy to the Western College of Veterinary Medicine, Saskatoon, Saskatchewan, Canada, and speculate on the possible pathogenesis and etiology of these lesions.

## MATERIALS AND METHODS

Signs of neurological disease or impaired vision were described in the history of seven of 74 free-living moose from Saskatchewan submitted for necropsy to the Department of Veterinary Pathology, Western College of Veterinary Medicine, University of Saskatchewan, between September 1969 and August 1994 (Tables 1, 2). All seven were killed by shooting. Routine necropsies were carried out, including gross examination of all major tissues and histological examination of lesions. The brains of cases 1 and 2 were examined for rabies infection. In case 1, a fluorescent antibody technique and transmission electron microscopy (Leighton and Williams, 1983) were used; in case 2, only the fluorescent antibody technique was done. Bacteriological examination was carried out on a swab sample from the sheath of the right common digital extensor tendon and a brain sample of case 6, and on a swab sample from the left eye and a brain sample of case 7. All samples were cultured on blood agar with 5% sheep blood and MacConkey agar (Prepared Media Laboratory, Richmond, British Columbia, Canada). The inoculated media were incubated at 37 C in an aerobic atmosphere and examined daily for 2 days for bacterial growth. An isolated colony representative of each bacterial variant detected visually was selected and identified according to methods of Carter and Cole (1990). Age of the moose was estimated by body size; in cases 4 and 6 it was



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TABLE 1. Age and sex distribution of 74 free-living moose from Saskatchewan submitted for necropsy to the Department of Veterinary Pathology, Western College of Veterinary Medicine, University of Saskatchewan, between September 1969 and August 1994. For age categorization, 1 June was taken as the hypothetical date of birth.

| | Age (yr) | | | | | |
|---|---|---|---|---|---|---|
| Sex | Fetus | 0–1 | 1–2 | >2 | Unknown | Total |
| Male | 1[a] | 12 | 3 | 11 (3[b]) | 3 | 30 (3) |
| Female | 0 | 8 | 7 | 15 | 3 (2) | 33 (2) |
| Unknown | 0 | 7 (1) | 1 | 0 | 3 (2) | 11 (3) |
| Total | 1 | 27 (1) | 11 | 26 (3) | 9 (4) | 74 (8) |

[a] Number of moose.
[b] Number of moose of which only the head was submitted for necropsy.

determined by counting the number of cementum layers of an incisor tooth (Gasaway et al., 1978).

A detailed examination of the central nervous system and eyes of these seven moose was carried out. The brain, spinal cord (if available), and eyes were examined in situ and after removal from the body. The brain and spinal cord were fixed in 10% phosphate-buffered formalin for at least a week, and representative samples of the cerebrum, cerebellum, and spinal cord were taken. The eyes were immersed in Bouin's fluid for approximately 24 hr and then in 70% alcohol for another 24 hr. Each globe was cut in a sagittal plane to obtain a sample including the optic nerve and the lens. These samples were embedded in paraffin, sectioned at 5 μm, and stained with hematoxylin and eosin for examination by light microscopy (Luna, 1968). The globe of case 5 was also stained with Masson's trichrome stain (Sheehan and Hrapchak, 1980). The maximum vertical diameter of the eyeball and lens in these sections were measured. Exceptions in the histological examination of the eyes were cases 1 and 2. In case 1, a sample was only taken from half of each eye, and the sensory retinas and lenses were not included except for half of the lens capsule of one eye. In case 2, no sections of the lenses were available for examination.

The eyes of a 4-mo-old and an adult captive moose which had died of other causes and had no discernible gross or histological eye lesions were examined and used as controls.

## RESULTS

Corneal lesions were present in all cases examined. Case 5 only had diffuse stromal edema. Six moose had corneal scars characterized by irregular epithelial hyperplasia (cases 2, 3, 6, left eye (7L) of case 7) with keratinization (cases 6, 7L), mild superficial (cases 1, 2, 4, 6) to deep (case 7L) stromal fibrosis, diffuse edema (cases 3, 6), and mid-stromal vascularization, which varied from peripheral (cases 2, 7L) to complete (cases 3, 4, 6). Case 2 had mild, deep-stromal, suppurative keratitis, and cases 3 and 6 had moderate, diffuse keratitis with a mixed infiltrate of neutrophils, lymphocytes, and plasma cells. In case 6, eosinophils were also present. Case 7L had a moderate, focal keratitis with a mixed infiltrate of eosinophils, neutrophils, lymphocytes, and plasma cells (Fig. 1). *Moraxella bovis* was isolated from a swab taken from the corneal surface of this eye.

Six moose had a lymphocytic-plasmacytic anterior uveitis, which varied from mild (cases 5, 6, 7) to moderate (cases 3, 4) to severe (case 2), with anterior and posterior synechia (cases 3, 5, right eye (7R) of case 7), ectropion uveae (cases 4, 7L), and entropion uveae (case 6). Cases 3, 4, and 5 had plasmoid aqueous humor in the anterior chamber, cases 2 and 3 had a cyclitic membrane, and case 7L had a preiridal fibrovascular membrane (Fig. 1). In cases 3 and 5, the iris lay adjacent to the cornea, largely obliterating the anterior chamber and the filtration angle (Fig. 2).

Cataracts were present in all cases examined. Grossly, the lens was white and opaque (Fig. 3), and histologically, there was epithelial hyperplasia (cases 1, 3, 5, 6), formation of bladder cells (cases 5, 6), posterior migration of lens epithelium (cases 1, 5, 6), and liquefaction of lens stroma (Fig. 4, 5). The location of this liquefaction was subcapsular (cases 3, 4, 5, 6, 7R), anterior and posterior (cases 3, 4, 5, 6) or only posterior (case 7R). In case 6, posterior liquefaction was more extensive than anterior. Case 3 had multiple foci of calcification in the liquefied stroma. In cases 3 and 4, the lens capsule was wrinkled and there was extensive liquefaction of lens stroma, confirming hypermature cataract.

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TABLE 2. Date and location found, age, sex, clinical signs, and measurements of eyeballs and lenses in seven Saskatchewan moose with signs of neurological disease or impaired vision.

| Case number | Date found | Location found (latitude and longitude) | Age (yr) | Sex | Clinical signs | Diameter eyeball[a] (mm) | Diameter lens[b] (mm) |
|---|---|---|---|---|---|---|---|
| 1[c,d] | August 1979 | Prince Albert National Park (53°12′N, 104°46′W) | NA[e] | NA | bizarre behavior, apparent blindness | NA | NA |
| 2[c] | September 1980 | Cypress Hills (49°40′N, 109°30′W) | 0–2 | F[f] | circling, bumping into objects | 24, 25 | NA |
| 3[c,d] | September 1981 | Spiritwood (53°22′N, 107°31′W) | >2 | M | totally blind, circling to left | 24, 25 | 9, 12 |
| 4 | January 1988 | Little Bear Lake (54°20′N, 104°35′W) | 5 | F | blind | 35, 35 | 11, 12 |
| 5[c,d] | October 1991 | Nipawin (53°22′N, 104°00′W) | 0–1 | NA | circling to left | 22, 23 | 10, 11 |
| 6 | April 1994 | Garrick (53°29′N, 104°29′W) | 20 | F | bumping into trees, no fear of humans | 33, 36 | 15, 16 |
| 7 | June 1994 | Cypress Hills (49°40′N, 109°30′W) | >2 | F | circling to left, bumping into trees | 31, 33 | 14, 15 |

[a] Maximum vertical diameter of the sagittal fixed section of each eyeball.
[b] Maximum vertical diameter of the sagittal fixed section of each lens.
[c] Only the head (and in case 2 also the neck) was submitted for necropsy.
[d] The head was stored frozen before necropsy.
[e] NA, not available.
[f] F, female; M, male.

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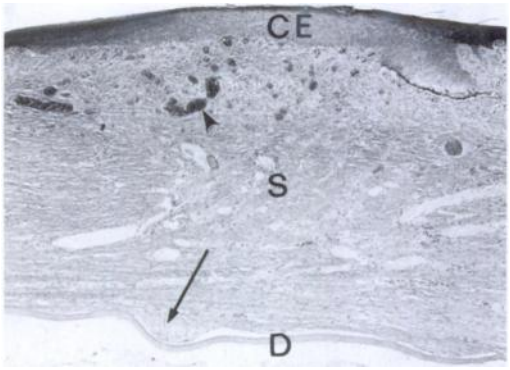



FIGURE 1. Moderate, focal keratitis, associated with *Moraxella bovis* infection in a moose (case 7L). The corneal epithelium (CE) is hyperplastic and keratinized, the stroma (S) has vascularization (arrowhead) and fibrosis (arrow), and there is a preiridal fibrovascular membrane (M). D = Descemet's membrane. H & E. Bar = 300 μm.

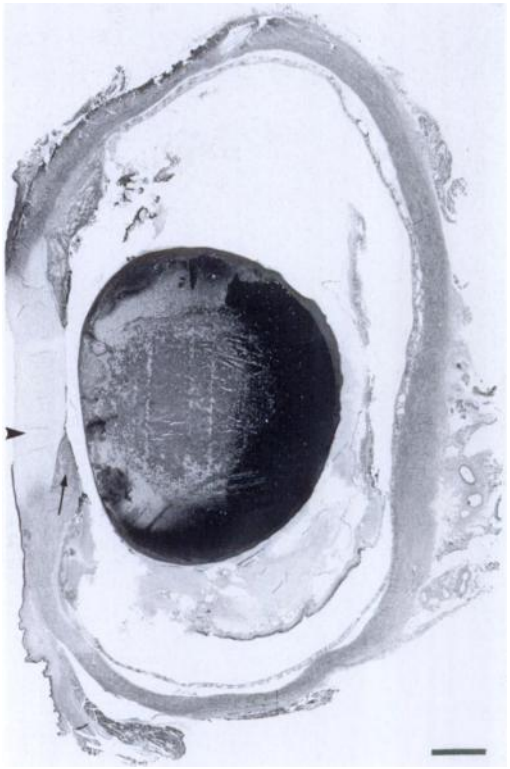


FIGURE 2. Microphthalmia in a moose (case 5). The iris (arrow) is apposed to the cornea (arrowhead), largely obliterating the filtration angle and anterior chamber. Masson's trichrome. Bar = 2 mm.

Retinal lesions were present in all cases examined. There was multifocal hypertrophy (cases 1, 2, 3, 5, 6) and hyperplasia (cases 1, 2) of the retinal pigment epithelium (RPE) (Fig. 6, 7). In cases 2, 4, 6, and 7R, there was retinal degeneration, which was multifocal (Fig. 7) to diffuse (Fig. 8), and mainly characterized by absence of the photoreceptor cell layer and outer nuclear layer and migration of RPE cells into the inner retinal layers. These degenerative lesions will be described individually.

In case 2, the sensory retina was detached at the level of the photoreceptor cell layer and lay tortuously folded in the vitreous humor, which contained several migratory RPE cells. There was diffuse retinal edema and multifocal retinal degeneration, which was characterized by decreased density of nuclei in the nuclear layers, fusion of the nuclear layers, and lack of ganglion cells. Numerous migratory RPE cells were scattered throughout the retina. In case 4, the photoreceptor cell layer and corresponding outer nuclear layer were degenerate and the outer nuclear layer was fused with the inner nuclear layer from approximately the equator to the optic disc. In case 6, the retina was progressively more degenerate from the level

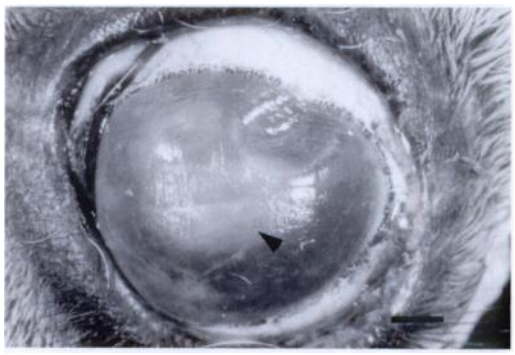


FIGURE 3. Cataract in a moose (case 6). The lens (arrowhead), partly visible through the pupil, is white and opaque. Bar = 4 mm.

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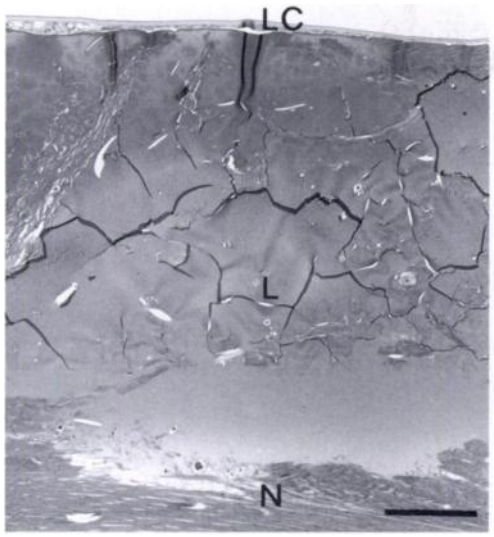



FIGURE 4. Lens with subcapsular cataract in a moose (case 6). The lens stroma adjacent to the lens capsule (LC) is homogeneous due to liquefaction (L). The cracks in this material are processing artifacts. N = normal lens stroma. H & E. Bar = 300 μm.

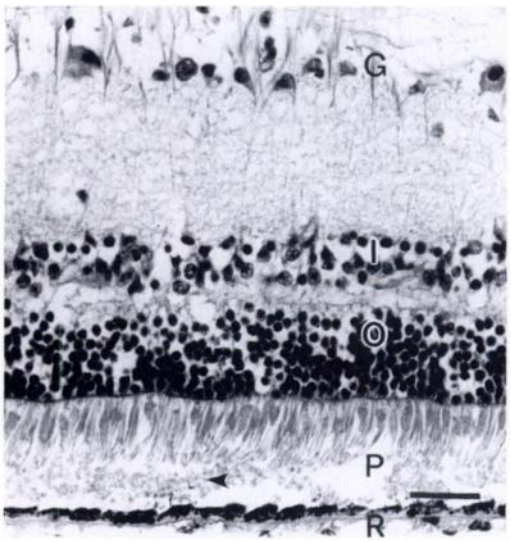



FIGURE 6. Normal retina in a moose (juvenile control). Although the eye was fixed within a few hours after death, there is some autolysis (arrowhead) of the photoreceptor cell layer. G = ganglion cell layer, I = inner nuclear layer, O = outer nuclear layer, P = photoreceptor cell layer, R = retinal pigment epithelium. H & E. Bar = 30 μm.

of the equator to the optic disc. The mildest changes were small foci of swollen RPE cells, some of which were also present in the inner nuclear layer. In some of these foci, the photoreceptor cell layer and

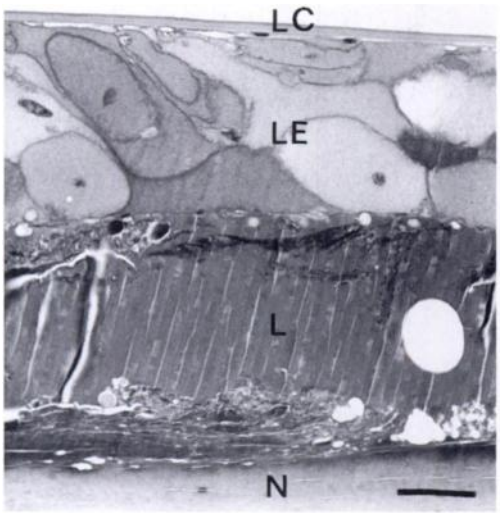



FIGURE 5. Lens with subcapsular cataract in a moose (case 6). The lens epithelium (LE) is hyperplastic and contains bladder cells. LC = lens capsule. L = liquefied lens stroma. N = normal lens stroma. H & E. Bar = 50 μm.

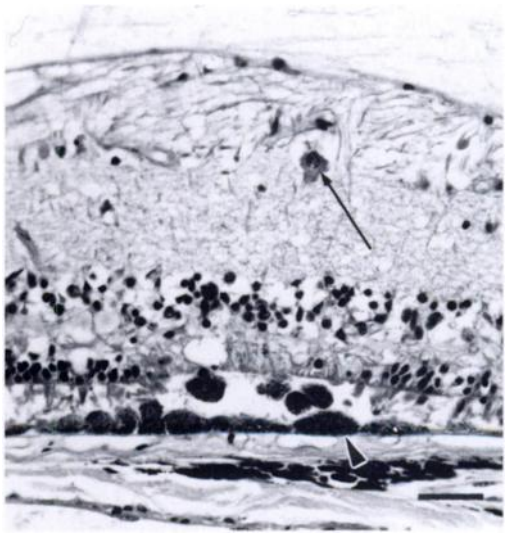


FIGURE 7. Retina with multifocal degeneration in a moose (case 6). In the focus shown here the retinal pigment epithelium is hypertrophic (arrowhead), and the photoreceptor cell layer and outer nuclear layer are absent. The inner nuclear layer is less dense than normal and only one ganglion cell (arrow) is visible. H & E. Bar = 30 μm.

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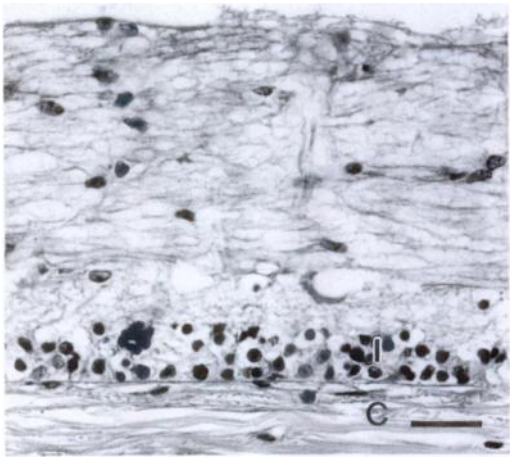



FIGURE 8. Retina with diffuse degeneration in a moose (case 6). The outer retinal layers are absent, and the inner nuclear layer (I) is directly apposed to the choroid (C). There are no ganglion cells visible. H & E. Bar = 30 μm.

outer nuclear layer were absent, and there was eosinophilic homogeneous material in the resulting cavity (Fig. 7). Further towards the optic disc the outer layers of the retina were not present, and the inner nuclear layer was directly apposed to the choroid (Fig. 8). In this area the density of ganglion cells was less than normal. In case 7R, large areas of the retina consisted only of the RPE, the inner and outer limiting membrane, a few glial cells and part of the inner nuclear layer. These degenerate areas were interspersed with small areas of normal retina.

Two moose had abnormally small eyes (Table 2). The eyes of case 3 were smaller (24 to 25 mm in diameter) than those of the control adult moose (38 mm in diameter). The lenses were also smaller (9 to 12 mm in diameter) than the lenses of the control adult moose (15 to 17 mm in diameter). Case 5 had smaller eyes (22 to 23 mm in diameter) than those of the control juvenile moose (25 to 26 mm in diameter), whereas the lenses (10 mm in diameter) were the same size (Fig. 2). The eyes of the other moose appeared to be normal in size.

Mild brain or spinal cord lesions were found in five moose (cases 1, 2, 5, 6, 7). In case 1, many neurons in or adjacent to the pyramidal layer of the hippocampus contained eosinophilic intracytoplasmic inclusion bodies, but no viral particles were detected in these inclusion bodies by transmission electron microscopical examination, and the brain did not have rabies virus antigen by use of a fluorescent antibody test. There was some glial satellitosis around neurons and very mild perivascular hypercellularity in the motor cortex of the cerebrum, but no evidence of migrating nematodes or other infection anywhere in the brain. Case 2 had an amber nodule of 3 mm diameter on the dorsal midline of the spinal cord at the level of the third cervical vertebra. It was not examined histologically. The brain did not have rabies virus antigen by use of a fluorescent antibody test. In case 5, there was slight cerebellar coning with deviation of the herniated area to the right, in association with fracture of the petrous part of the left temporal bone and adjacent subdural hemorrhage. In case 6, there was mild perivascular cuffing with mononuclear cells and hemosiderin-laden macrophages in the cerebrum, cerebellum, brain stem, and cervical, thoracic, and lumbar spinal cord. The brain of case 7 was edematous. Histologically, there was a mild focal infiltration of plasma cells and lymphocytes in the leptomeninges, and atrophy and non-suppurative neuritis of the right optic nerve.

*Staphylococcus aureus* and alpha-type *Streptococcus* sp. were isolated from the sheath of the right common digital extensor tendon of case 6, which had a chronic suppurative tenosynovitis.

## DISCUSSION

All seven moose with signs of neurological disease or impaired vision had marked eye lesions, whereas lesions in the brain and spinal cord were absent or mild. Leighton and Williams (1983) describe the brain lesions of case 1 in more detail. *Moraxella bovis* (case 7L) was the only etiolog-

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ical agent detected that may have caused eye lesions. This pathogen causes infectious bovine keratoconjunctivitis (Wilcock, 1993); hence it may have caused the focal keratitis in this case.

Because of the presence of multiple lesions in each eye, and their chronic and non-specific character, it was not possible to determine with certainty whether they were related to each other. However, in some cases the retinal degeneration may have occurred first and have contributed to other ocular lesions observed. The nature of the retinal lesions, characterized by degeneration of the outer retinal layers, is evidence that there was injury to the photoreceptor cell layer. The resulting degeneration of the photoreceptor cells would have resulted in hypertrophy, hyperplasia, and migration of the adjacent RPE cells (Wilcock, 1993). Subcapsular cataract may have occurred secondarily to the retinal degeneration, as a result of the release of toxic lipid peroxidation products of the retina into the vitreous humor (Zigler and Hess, 1985). This hypothesis is supported by the presence of more severe cataract in the posterior part of the lens in some cases. The lymphocytic-plasmacytic anterior uveitis may have been caused by the leakage of liquefied lens stroma (Wilcock, 1993). The impaired vision and neurological disease would have predisposed these moose to physical trauma to the eyes. The diffuse corneal lesions, characterized by epithelial hyperplasia, stromal fibrosis, vascularization, and keratitis, are consistent with this cause (Wilcock, 1993).

Retinal degeneration may be caused by senile change, nutritional deficiency, metabolic disorder, or injury caused by infectious, parasitic, chemical or physical agents. Especially in severe cases, retinal degenerations with different pathogenesis have a similar histological appearance in their end stages (Wilcock, 1993). Possible causes in the above cases include the following parasites, which have been found in moose and are known to cause eye lesions in moose or other species: *Parelaphostrongylus tenuis* (Kurtz et al., 1966; Anderson and Prestwood, 1981), *Elaphostrongylus* sp. (Anderson, 1992), *Elaeophora schneideri* (Worley et al., 1972; Anderson, 1992), *Setaria* sp. (Wobeser, 1985; Wilcock, 1993), and *Toxoplasma gondii* (Kocan et al., 1986; Siepierski et al., 1990; Barker et al., 1993).

Cases 3 and 5 differed from the others in the reduced size of the eyes. The differential diagnoses for this lesion are microphthalmia, which is a developmental anomaly, and atrophia bulbi, which is the shrunken end-stage of a severe ophthalmitis (Wilcock, 1993). In addition to decreased size of the eyes, case 3 had moderate keratitis, anterior and posterior synechia, moderate anterior uveitis, and a cyclitic membrane. These inflammatory lesions may have been primary and resulted in shrinkage of normal sized eyes. Alternatively, they may have occurred secondarily in eyes that were already too small. We could not distinguish between the two possibilities. In case 5, there was no evidence of severe inflammation at present or in the past, so that this probably was microphthalmia. Microphthalmia has been related to genetic defects and maternal vitamin A deficiency (Saunders, 1968). A genetic defect causing microphthalmia has been reported in red deer (*Cervus elaphus*) (Dahme and Helwig, 1960), and such a defect may also have been the cause in these moose. It is unlikely that free-living moose suffer from vitamin A deficiency, as precursors of this vitamin are available in adequate amounts in green plants.

Although we found marked ocular lesions in 9% of free-living Saskatchewan moose submitted for necropsy between 1969 and 1994, we were unable to determine their cause despite detailed examination. Ocular disease may have an important effect on the survival of individual moose, and we hope that the description of these lesions will stimulate further studies of their pathogenesis and etiology.

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