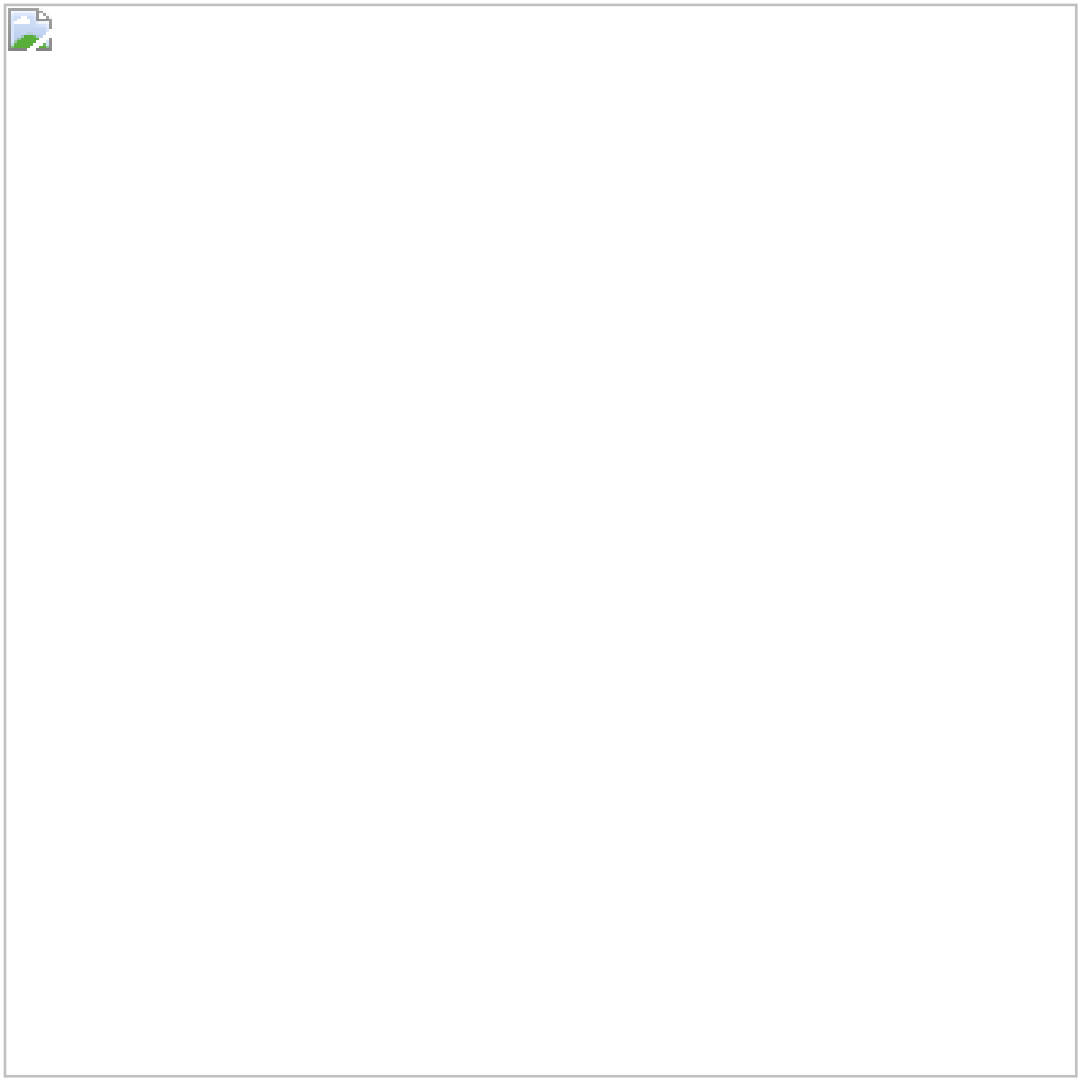

# Blindness Associated with Retinal Dysplasia in a Prairie Falcon, Falco mexicanus

Authors: Dukes, T. W., and Fox, G. A.

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cutaneous, mesenteric, etc.) were low or exhausted. Bone marrow fat content was 23%. This figure reflects an animal in a low nutritional plane (Franzmann and Arneson, 1976, J. Wildl. Manage. 40: 336–339). The animal otherwise appeared to be in good health.

This is believed to be the first report of a hernia in moose. It was not possible to determine its cause. It seems most likely that it was either a result of trauma or of umbilical origin. The hernia was apparently a long-standing condition. Based upon (a) reports that the moose had survived for more than 1 yr in its condition, (b) the fact that it had produced and raised a calf, (c) its ability to run and negotiate obstructions, and (d) the normal functioning of the affected organs, it would appear that the hernia posed no direct threat to the animal's health. This was most likely an isolated case which has no implications to the moose population of the area as a whole.

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# Blindness Associated with Retinal Dysplasia in a Prairie Falcon, *Falco mexicanus*

**T. W. Dukes,** Agriculture Canada, Animal Diseases Research Institute, NEPEAN, P.O. Box 11300, Station H, Nepean, Ontario K2H 8P9, Canada; **and G. A. Fox,** Toxic Chemicals Section, Canadian Wildlife Service, 100 Gamelin Boulevard, Hull, Quebec H8Y 1V9, Canada

Retinal dysplasia results from the response of the developing retina to unusual stimuli. The morphologic changes include retention of an embryonic appearance, retinal folds or retinal rosettes which have been classified. These may occur alone or in association with other ocular lesions. Several pathogenetic mechanisms associated with specific factors or agents affecting the developing retina have been proposed (Silverstein et al., 1971a, Am. J. Ophthalmol. 72: 13–21; Lahav et al., 1973, Am. J. Ophthalmol. 75: 648–667).

Many reports have been written on retinal dysplasia in man but there are fewer concerning its occurrence in animals. Cases have been reported in dogs, horses, pigs, deer, rats and mice (Lahav et al., 1973, op. cit.). We are reporting the occurrence of apparent congenital blindness associated with retinal dysplasia in a prairie falcon. We were unable to find reports of retinal dysplasia in birds or reports on the effect of viruses, toxic chemicals, or abnormal incubation temperatures on the developing avian retina.

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During a study of the effects of toxic chemical residues on the reproduction of birds of prey, a clutch of four prairie falcon eggs with thin shells were collected in southern Alberta and brought to the Endangered Species Unit in Wainwright for incubation. Only one egg hatched and the chick was blind at hatching. It was sent to us for evaluation and study when 6 wk old.

The bird, a female, was alert. Ophthalmoscopically, there was variable pigmentation of the fundus with the choroidal vasculature being obvious in most of the fundus. There were focal areas of bluish opacity and pigmentation in the retina (Fig. 1). The pupils were widely dilated and responded poorly to light. The eye preservation reflex was absent but corneal sensitivity was present.

At necropsy, no changes were macroscopically apparent in the eyes or other tissues. Tissues were fixed in Bouin's solution overnight and were washed in running water and stored

in 50% ethanol. Routine histological techniques were used in preparing tissue sections.

Histologically, focal malformed areas were dispersed in the retina. These were cystic spaces containing elongated cell processes or fibrils, surrounded by one or two layers of cell nuclei (Fig. 2) and resembled the primitive unilayer rosettes described in the classification by Lahav et al. (1973, op. cit.). One eye was more severely affected than the other. Some retinal folds and minimal retinal gliosis were also evident. The rest of the globe and lids were morphologically unaltered. No lesions were seen in the other body tissues including brain.

No viruses were isolated from this bird using standard egg inoculation and tissue culture techniques for avian viruses.

Both brain and liver tissue were submitted to the Ontario Research Foundation for organochlorine and mercury residue analysis. Trace levels (less than 0.01 ppm wet weight) of dieldrin, dichlorophenyldichloroethane (DDD), dichlorophenyltrichloroethane (DDT), heptachlor epoxide, hexachlorobenzene (HCB) and mercury were detected in the brain accompanied by 0.15 ppm dichlorophenyldichloroethylene (DDE) and 0.57 ppm polychlorinated biphenyl.

Retinal dysplasia is a nonspecific altered response of the retina at certain stages of its development, having variable histologic changes, different etiologies, and several pathogeneses. The retina may have an embryonic appearance, is often formed into folds, and may contain rosettes. Gliosis and calcification may be seen but these are considered to be nonspecific secondary changes (Lahav et al., 1973, op. cit.). Associated ocular abnormalities that may be seen include coloboma, persistent hyperplastic primary vitreous, and changes in the pigment epithelium. Retinal dysplasia is present at birth in those animals born with a mature retina such as man, cattle, and horses, but it can develop neonatally in kittens, puppies and rodents which are born with immature eyes.

Lahav et al. (1973, op. cit.) reviewed a series of human cases and classified them clinically and histopathologically. Dysplasias were divided into those occurring as isolated lesions and those associated with other ocular changes. Four morphologic types of rosettes were described in dysplastic retinas: (1) three-layer rosettes; (2) two-layer rosettes; (3) single-layer rosettes; and (4) primitive unilayer rosettes. Correlation of the clinical and pathological classifications was limited. The primitive unilayer rosette was seen only in the group with isolated retinal lesions.

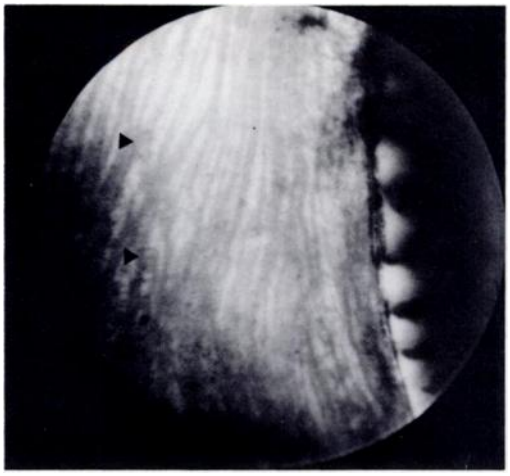

FIGURE 1. Fundus photograph of the prairie falcon with retinal dysplasia. Focal areas of altered transparency were evident (arrows).

The first pathogenetic mechanism proposed is that the lesion is primary, may be hereditary and is associated with detachment of the retina with consequent dissociation of the developing retina from the pigment epithelium. This concept is based on the idea that the pigment epithelium is the organizing factor of the developing retina so that separation of the retina from the pigment epithelium during a critical stage of development would lead to disorganization and dysplasia of the retina. This type tends to be associated with other anomalies and to have a preponderance of Lahav et al.'s two- or three-layer rosettes.

This type of retinal dysplasia in man has classically been associated with chromosomal abnormalities and hereditary conditions (Lahav et al., 1973, op. cit.). Hereditary retinal dysplasia associated with other ocular abnormalities has been reported in dogs, specifically Bedlington terriers (Rubin, 1968, J. Am. Vet. Med. Assoc. 152: 260–262), Labrador retrievers (Barnett et al., 1970, J. Small Anim. Pract. 10: 755–759) and Sealyham terriers (Ashton et al., 1968, J. Pathol. and Bacteriol. 96: 269–272). It has also been associated with the sclera ectasia syndrome of collies and inherited multiple anomalies of beagles (Rubin, 1974, Atlas of Veteri-

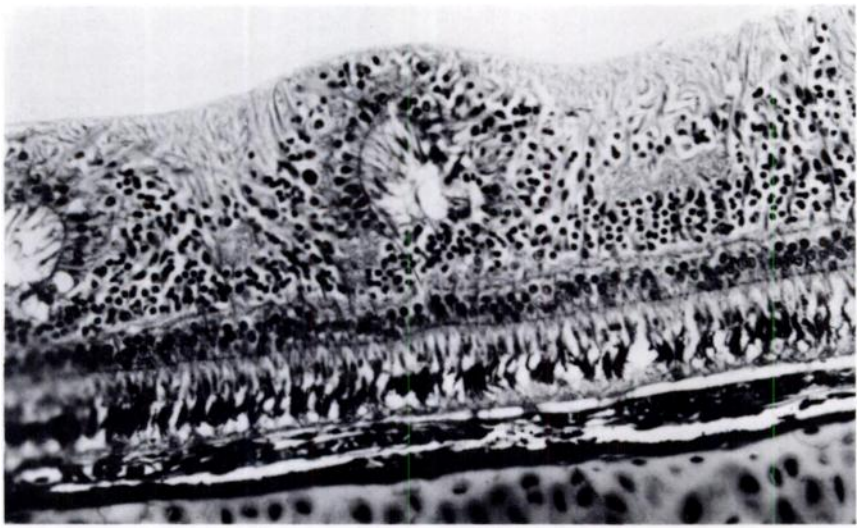

FIGURE 2. Histologic appearance of the retinal rosettes. Cystic spaces were evident surrounded by rows of nuclei and frequently contained fibrillar material suggestive of photoreceptor processes.

nary Ophthalmoscopy, Lea & Febiger, Philadelphia, Pennsylvania, 470 pp).

In the second type, the lesion is secondary to a toxic or viral insult to the pigment epithelium or dividing retinal epithelial cells. Silverstein et al. (1971b, Am. J. Ophthalmol. 72: 22–34) reported experimental evidence that viral infection during a critical period in gestation can induce maldevelopment of the retina in lambs without separation of the pigment epithelium. The viral or toxic insult might produce necrosis, inflammation, disorganization and abortive attempts at repair resulting in dysplasia. This pathogenesis tends to be associated with the isolated dysplasia described by Lahav et al. (1973, op. cit.). Several viral infections have been incriminated in spontaneous retinal dysplasias. These include herpesvirus canis (Percy et al., 1971, Vet. Pathol. 8: 37–53), feline panleukopenia virus (Percy et al., 1975, J. Am. Vet. Med. Assoc. 167: 935–937), bovine virus diarrhea virus (Bistner et al., 1970, Pathol. Vet. 7: 275–286), bluetongue virus in sheep (Silverstein et al., 1971b, op. cit.), lymphocytic choriomeningitis virus in rats (Mojan et al., 1972, Invest. Ophthalmol. 11: 850–856) and simian virus 40 in rats (Friedman et al., 1973, Invest. Ophthalmol. 12: 591–595). Ionizing irradiation of the human embryo and newborn dog has also induced retinal dysplasia (Shirley et al., 1970, Invest. Ophthalmol. 9: 888–900).

The organochlorine residue levels found in this case would not appear to be high enough to induce lesions since, in our experience, most raptorial bird tissues have levels 10 or more times higher than those found in this bird. The low levels of the measured pesticides and mercury suggested that these toxins were not involved since many birds have higher tissue levels with no dysplasia of retina. Although no viruses were cultured and there were no inclusions or viral-specific lesions in this bird the presence of viruses in the egg prior to hatching could not be ruled out.

Several hundred prairie falcon nestlings from the same population as this bird have been handled in the wild over the past 10 yr and this is the first observed case of congenital blindness. In other species hereditary retinal dysplasia is usually associated with other ocular lesions (Ashton et al., 1968, op. cit.; Rubin, 1968, op. cit.; Barnett et al., 1970, op. cit.). None were seen in this case. Dysplasia was never noted in any of the numerous other prairie falcon chicks hatched under identical artificial incubation conditions. Such lesions must, therefore, be a rare occurrence and not likely inheritable or the result of artificial incubation conditions.

There were no lesions in the brain and optic nerves that might result in central blindness. We found that the rosettes in the falcon retina were not associated with other ocular changes and were of the primitive unilayer type which Lahav et al. (1973, op. cit.) found incidentally with no relationship to other ocular lesions. Although we did not demonstrate any viruses and the levels of common environmental pollutants measured were low, the morphologic lesions in the falcon were like those seen in other species secondary to injury by toxins or viruses.

We thank Mr. Richard Fyfe, Endangered Species Specialist, Canadian Wildlife Service, Edmonton, Alberta, for submitting the specimen. Mr. Lincoln Reynolds, Ontario Research Foundation, performed the residue analyses.

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# Immobilization of Wolves Using Ketamine in Combination with Xylazine or Promazine

**Todd K. Fuller and David W. Kuehn,** Forest Wildlife Populations & Research Group, Minnesota Department of Natural Resources, Grand Rapids, Minnesota 55744, USA

Wolves (*Canis lupus*) have been most commonly immobilized with phencyclidine and promazine (Mech, 1974, Proc. Intl. Congr. Game Biol. 11: 315–322) or with etorphine (Fuller and Keith, 1980, J. Wildl. Manage. 45: 271–273). Phencyclidine is not currently available and etorphine is a highly potent narcotic that must be used with extreme care. Ketamine, with subsequent injections of promazine, has been used to immobilize both captive and wild-caught wolves (U. S. Seal, L. D. Mech, and S. Fritts, pers. comm.), and xylazine has been used to restrain captive wolves (Philo, 1978, J. Am. Vet. Med. Assoc. 173: 1163–1166).

Ketamine acts primarily on the central nervous system and produces a dissociative anesthesia (Harthoorn, 1976, The Chemical Capture of Animals, Bailliere Tindall, London, England, 416 pp.). Muscle relaxation is generally poor when ketamine is used alone so we used it in combination with xylazine, a sedative and muscle relaxant, or promazine, a tranquillizer, to immobilize wild-caught wolves during telemetry studies in northcentral Minnesota.

Wolves were captured in No. 4 or 14 Newhouse steel traps during spring (April–May) or fall (August–October). Trapped wolves were immobilized initially with combinations of ketamine hydrochloride (ketamine [ketaset] Bristol-Myers Co., Syracuse, New York 13201, USA) and xylazine hydrochloride (xylazine [rompun] Haver-Lockhart Laboratories, Shawnee, Kansas 66201, USA), ketamine and xylazine with atropine sulphate (atropine [atropine sulphate] Med-tech Inc., Elwood, Kansas 66024, USA), or ketamine and promazine hydrochloride (promazine [sparine] Wyeth Laboratories, Inc., Philadelphia, Pennsylvania 19101, USA), injected intramuscularly in the hindquarters with a 2-m jab stick. Additional injections were administered similarly, if necessary. The wolves' legs were bound and their mouths were taped shut throughout handling. Animals were ear-tagged, weighed, measured, fitted with mortality-sensing radiocollars, inspected for injuries and general condition, and given an antibiotic (benzathine penicillin G and procaine penicillin G [bicillin fortified] Wyeth Laboratories, Inc., Philadelphia, Pennsylvania 19101, USA) in the case of trap damage to the foot. Blood samples were drawn from the femoral artery and were handled and analyzed as outlined by Karns and Crichton (1978, J. Wildl. Manage. 42: 904–908). Heart rates, respiration rates, and rectal temperatures were recorded for some wolves. Radiocollared wolves were subsequently located and observed from the air.

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During 5 September–17 October 1980, 22