Progressive Retinal Degeneration in Ranch Mink

W. J. Hadlow

Department of Health and Human Services, Public Health Service, National Institutes of Health, National Institute of Allergy and Infectious Diseases, Epidemiology Branch, Rocky Mountain Laboratories, Hamilton, Mont.

Abstract. Retinal degeneration was prevalent in a large group of sapphire and pastel mink (Mustela vison) kept for studies on slow viral diseases. Nearly 78% of those two to eight years old were affected. The retinopathy was equally common in both sexes but more frequent in sapphires (85%) than in pastels (63%), and it was severe more often in sapphires than in pastels. By light microscopy, the primary change appeared to be progressive degeneration of fully developed photoreceptors, beginning in their outer segments. In many mink, including some younger ones, the rods and cones and outer nuclear layer had disappeared from all but the far periphery of the fundus. The inner retinal layers were spared until late in the disease, and the pigment epithelium remained essentially unchanged. The cause of the retinopathy was not established. It may represent an abiotrophy in which the structural integrity of the photoreceptors began to wane in many mink after they reached two years of age. Apart from reducing visual acuity, the retinopathy has implications for the photoperiodic control of fur growth and reproduction in this highly light-sensitive carnivore.

Primary degeneration of the retina in animals may occur in diverse ways: it may result from nutritional deficiency or from exposure to toxic chemical substances; it may be determined genetically; or it may accompany aging. Sometimes its cause is unknown, or at least uncertain. Accordingly, its pathogenesis and natural history are varied. Regardless of the mechanism by which it arises, endstage light microscopic appearances of the retina are similar. This similarity often poses a dilemma for veterinary pathologists attempting to determine the cause and histogenesis of naturally occurring retinopathies.

Such is the case with the retinal degeneration I found prevalent in a large group of ranch mink (Mustela vison) kept for studies on slow viral diseases. Although the cause of this retinopathy was not established, its main features are described here to document its occurrence in an animal whose ophthalmic lesions seldom have been reported.

Materials and Methods

Eyes were collected from male and female sapphire and pastel (royal) mink obtained from a local closed herd when they were 6 to 24 months old. (Eyes of a few younger mink were from kits of this stock born at the laboratory.) They were housed individually in galvanized wire mesh cages hung in fly-proof, open-sided sheds. Oriented in a north-south direction, the sheds provided adequate natural lighting while protecting the mink from direct rays of the sun. Each cage had a wooden (redwood or spruce) nest box that contained pine wood shavings as nesting material. The mink were fed a standard ration (a ground wet mixture of marine fish offal, chicken offal, beef by-products, and a commercial cereal, sometimes with added tetracycline) and were given untreated well water in galvanized metal cups. None had been vaccinated for canine distemper, viral enteritis, or botulism. Most had been used in experiments on Aleutian disease or the spongiform encephalopathies.

All eyes examined histologically were from mink killed by exsanguination while under ether anesthesia. The globes were removed promptly, freed of orbital soft tissues, fixed in Zenker's fluid, and processed by a procedure described. Vertical meridional sections of the globes were stained with hematoxylin and eosin-phloxin (HEP), usually with periodic acid-Schiff reagent (PAS) as well, and sometimes by other standard staining procedures. Eyes from 375 mink were examined. Except for 122 affected with experimental Aleutian disease, all were chosen at random from live mink coming to necropsy for varied reasons.

Results

Normal retina

The retina of the mink resembles that of other vertebrates (fig. 1). From the optic nerve head, its peripheral extent is about the same in the dorsal, ven-
nal, and lateral (temporal) directions but is slightly greater in the medial (nasal) direction. In all quadrants, the thickness of each layer is fairly uniform from the optic disc to a point nearly two-thirds of the distance to the ora ciliaris retinae. More peripherally, the retina becomes thinner. The ganglion cell layer is a single row of small and medium-sized cells with a few larger (18 μm) ones interspersed among them. More ganglion cells are found centrally than peripherally, and occasionally one is situated deep in the inner plexiform layer. In the dorsolateral region of the fundus, about 2.5 mm from the optic disc, ganglion cells have been reported to be relatively more numerous than elsewhere in the retina—an observation I did not attempt to confirm.

Three or four poorly aligned rows of round nuclei with loosely arranged chromatin form the inner nuclear layer, whereas seven or eight well-ordered rows of much smaller, more densely staining, round nuclei form the outer nuclear layer. In the nontapetal fundus, this layer may be one or two rows thicker. The photoreceptor layer, 24 (tapetal) to 27 (nontapetal) μm thick, consists of rods and cones whose inner and outer segments are nearly of equal length. Because of their bulky, intensely staining (eosin-phloxin) inner segments, cones are distinguished easily from rods. Both visual cells occur throughout the retina, but relatively more cones are present centrally than peripherally. As a normal variation, an occasional cone nucleus is found in the photoreceptor layer. Blood vessels occur in all layers from the internal limiting membrane to the outer plexiform layer.

The layer of pigment epithelium is a single row of flattened cuboidal cells whose content of melanin varies within and between the two color phases studied. In the sapphire, a blue-gray color phase with brown or red eyes, melanin granules in the pigment epithelium of both color phases; except in some pastels, are sparse and spotty in the retinal pigment epithelium. In the royal pastel, a brown color phase with brown or red eyes, melanin granules in the retina (and uvea) characteristically are large, coarse, and widely separated. Some red-eyed sapphires are almost devoid of ocular pigment. In the royal pastel, a brown color phase with brown eyes, the melanin granules are much finer, more evenly dispersed, and generally more numerous. Even though melanin may be abundant in the uvea, deposits usually are sparse and spotty in the retinal pigment epithelium of both color phases; except in some pastels, most is in the peripheral part of the layer. The melanin granules tend to be concentrated at the cell surface nearest the photoreceptors; the large granules in sapphires may project into the outer segments. Neither color phase has melanin in the pigment epithelium of the tapetal fundus.

Several choroidal structures adjacent to the retina are noteworthy. As in several other domesticated mammals, Bruch’s membrane is not well developed; it appears to be nothing more than the thin PAS-positive basement membrane of the retinal pigment epithelium. It is prominent only peripherally in the fundus. Capillaries of the choriocapillaris often indent Bruch’s membrane, causing it to appear scalloped. At sites of such indentation, the basement membranes of the capillary and the pigment epithelium apparently merge.

A tapetum cellulosum is present in the dorsal half of the globe, but its full extent has not been determined (fig. 2). In mink three to five months old, it consists of four to six rows of thin, rectangular, tile-like cells. It may be only two or three rows thick in mink nine months old. In those more than one year old, it is irregularly identifiable as a thin, often discontinuous layer one or two cells thick. The cells may be compressed or greatly ballooned. Such a tapetal remnant was found in 6 of 18 yearling mink, 8 of 60 three-year-olds, 7 of 49 five-year-olds, 9 of 48 seven-year-olds, and 3 of 23 nine-year-olds. Sometimes it was present in one eye but not in the other. Even though the tapetum might not be apparent in these older mink, the tapetal fundus always can be identified by its melanin-free pigment epithelium.

Several choroidal structures adjacent to the retina are noteworthy. As in several other domesticated mammals, Bruch’s membrane is not well developed; it appears to be nothing more than the thin PAS-positive basement membrane of the retinal pigment epithelium. It is prominent only peripherally in the fundus. Capillaries of the choriocapillaris often indent Bruch’s membrane, causing it to appear scalloped. At sites of such indentation, the basement membranes of the capillary and the pigment epithelium apparently merge.

A tapetum cellulosum is present in the dorsal half of the globe, but its full extent has not been determined (fig. 2). In mink three to five months old, it consists of four to six rows of thin, rectangular, tile-like cells. It may be only two or three rows thick in mink nine months old. In those more than one year old, it is irregularly identifiable as a thin, often discontinuous layer one or two cells thick. The cells may be compressed or greatly ballooned. Such a tapetal remnant was found in 6 of 18 yearling mink, 8 of 60 three-year-olds, 7 of 49 five-year-olds, 9 of 48 seven-year-olds, and 3 of 23 nine-year-olds. Sometimes it was present in one eye but not in the other. Even though the tapetum might not be apparent in these older mink, the tapetal fundus always can be identified by its melanin-free pigment epithelium.

Retinal degeneration

Apart from those with overt retinal degeneration, few mink more than one year old had a retina resembling that of younger mink. In all others, by comparison, outer segments of the rods and cones were shortened slightly, sometimes reduced in number, and sometimes bent or partly fragmented distally (fig. 3). The outer nuclear layer, instead of having seven or eight rows of nuclei, usually had only six. Such structural changes were diffuse in the retina, which otherwise looked normal. Apparently non-progressive, the changes were the same in mink two or ten years old. They were interpreted as indicative of the slight degeneration that almost invariably supervened in the retina once the mink became about two years old. Overt retinal degeneration seemed superimposed on this fairly constant background of minor deviation from the normal. Even so, the two phenomena may not be separate or unrelated events.

The earliest evidence of overt retinal degeneration was further shortening and greater loss of outer segments of the rods and cones (fig. 4). Many had disintegrated into small PAS-positive globules or beaded

Retinal Degeneration in Mink

19

Downloaded from vet.sagepub.com by guest on November 7, 2016
fragments. Coincident with this, the outer nuclear layer was reduced to four or five rows of nuclei. The inner retinal layers and the pigment epithelium still looked normal. Usually such degeneration was most apparent centrally in either the tapetal or nontapetal fundus, or both. With further degeneration, considered here as moderate, only stubby remnants of the rods and cones remained in much of the retina, and the outer nuclear layer was reduced to three rows of normal-appearing nuclei (fig. 5). The bulker inner segments of the cones persisted longer than the slender ones of the rods (fig. 6). In some mink, many photoreceptor nuclei, apparently mostly from cone cells, had migrated into the photoreceptor layer comprised of remaining inner segments and a small amount of debris. In a few sapphires, these accumulations of nuclei were dense and were associated with a jumbled plethora of nuclei in the outer nuclear layer (fig. 7). Their combined bulk sometimes puckered the inner retinal surface centrally. Small groups of swollen pigment epithelial cells contained increased amounts of PAS-positive material. Some had become detached into the debris from disintegrated rods and cones, but these or other phagocytic cells were never numerous. Usually only focally apparent, the
debris did not accumulate as the degeneration of photoreceptors became more severe.

In the severely damaged retina, the rods and cones were mere vestiges or mostly absent, and no more than one or two rows of nuclei remained in the outer nuclear layer (figs. 8, 9). Where the photoreceptors, their nuclei, and the outer plexiform layer had disappeared, only the external limiting membrane separated the inner nuclear layer from the thin layer of pigment epithelium (fig. 10). Where it too had sometimes disappeared through atrophy or degeneration, the external limiting membrane directly apposed Bruch's membrane. Whether adhesions formed between them at these sites was never certain. Thickened Müller's fibers spanned the retina between the two limiting membranes (fig. 11). Melanin in the pigment epithelium remained sparse, and melanin-bearing cells had not migrated to the inner retinal layers. Rarely, a few pigment epithelial cells laden with PAS-positive material (lipofuscin) were seen there. Only late in the course of the retinopathy did the inner nuclear layer become noticeably narrower, ganglion cells less numerous, and the optic nerve fiber layer greatly attenuated (fig. 11). Overt atrophy of the optic nerve supervened only in a few mink when the retina was mostly a glial scarred remnant (fig. 12). In general, the degeneration was more complete centrally than peripherally. Yet in many mink, all but the far periphery of the retina was affected severely. At all stages, the extent and severity of the degeneration usually were similar in both eyes.

Other microscopic changes were seen mainly in the older mink, notably pastels. Retinal blood vessels often were thickened slightly, though otherwise unchanged. In a few mink, small knobby thickenings of PAS-positive material occurred along Bruch's membrane, usually centrally. The choriocapillaris seemed normal, but walls of the large choroidal arteries often were hyalinized. Except for this and the cellular infiltration in mink affected with Aleutian disease, the choroid was free of changes.

The degeneration was seen in a few yearling sapphire females, but it first became common when mink were two years old (fig. 13). Thereafter through eight years of age, it was prevalent in both color phases. Although equally common in both sexes, it was more frequent in sapphires than in pastels. Thus, among mink one to eight years of age, 85% (162/190) of sapphires and 63% (95/151) of pastels were affected to some degree. In mink older than four years, moderate to severe degeneration occurred far more often in sapphires (80%) than in pastels (53%) (fig. 14). With advancing age, this difference became even more striking. The severity of the degeneration was not clearly related to age, for total loss of photoreceptors was found in both color phases from two to eight years of age. Only moderate degeneration was present in some mink seven or eight years old. Most older pastels were affected only mildly, but severe retinal damage predominated in the oldest group of sapphires.

Most age groups included mink born from 1970 to 1975. Some from each of those years were affected when examined at two to eight years of age. A larger percentage of those born in 1972, 1973, and 1974, however, had the retinopathy than did those born earlier or later. Of 213 mink (56.8% of the total) born during that three-year period, 72.7% were affected. Even though many nine- to eleven-year-old pastels born in the period 1966 to 1972 had the disease, it was nearly always mild.

Cataract, usually bilateral, was the only other ocular lesion often found in the mink. Unlike that of the retinopathy, its prevalence was about the same in both color phases and steadily increased with age (fig. 15). Both lesions were equally common in nine- and ten-year-old mink. These contrasting patterns of prevalence suggest that the two lesions occurred as independent phenomena.

**Discussion**

The light microscopic findings are consistent with the interpretation that the primary pathologic change in this retinopathy is progressive degeneration of the fully developed photoreceptors. The process begins in their outer segments, especially centrally, while their inner segments and nuclei are intact. Eventually, the rods and cones and the outer nuclear layer disappear from all but the far periphery of the fundus. This selective disappearance of the photoreceptors contrasts with the preservation of the inner retinal layers until late in the course of the disease. Debris from disintegrating photoreceptors neither accumulates in excessive amounts nor persists in the subretinal space, as it does in retinopathies that arise when the pigment epithelium is defective.²⁴ Debris-laden phagocytic cells originating from the pigment epithelium or from other sources are uncommon. No doubt, products of the degeneration never become conspicuous because the tempo of the process is slow and because the normal pigment epithelial cells have a remarkable capacity to ingest and metabolize such debris.²⁷ I do not know whether this ability of the pigment epithelium to digest photoreceptor debris is impaired in the sapphire mink as it seems...
Fig. 6: Moderate degeneration of mid tapetal retina in 2-year-old sapphire female. HEP.
Fig. 7: Massive accumulation of photoreceptor nuclei in layer of rods and cones in 5-year-old sapphire male. Central nontapetal retina. HEP.
Fig. 8: Severe degeneration of mid tapetal retina in 2½-year-old pastel female. HEP.
Fig. 9: Severe degeneration of mid tapetal retina in 3½-year-old pastel female. HEP.
Fig. 10: Extreme degeneration of mid tapetal retina in 7-year-old pastel female. HEP.
Fig. 11: Thickened Müller's fibers in remnant of mid tapetal retina in 7-year-old sapphire female. HEP.
Fig. 12: Glial scarred remnant of central tapetal retina in 7-year-old sapphire female. HEP.

to be in the beige mouse,\textsuperscript{51} which, like the sapphire, is affected with the Chediak-Higashi syndrome.

Furthermore, unlike what happens secondarily in some retinopathies of animals,\textsuperscript{2} the pigment epithelial cells do not acquire more melanin or regularly migrate to the inner retinal layers. Nor do they form aggregates of hypertrophic cells containing abundant lipofuscin, as in an apparently inherited dystrophy of the pigment epithelium in the dog.\textsuperscript{4} Instead, they remain largely unchanged, except at the endstage of the retinopathy, when they may undergo atrophy or degeneration and disappear. The focal hyaline thickening of Bruch's
membrane found in a few old mink no doubt is an age-associated change unrelated pathogenetically to the retinal degeneration. The choriocapillaris remains unchanged, so diminished capillary blood supply does not account for the degeneration of the photoreceptors.

Passage of some photoreceptor nuclei through the external limiting membrane commonly occurs, but the massive influx of such nuclei into the layer of rods and cones in some mink is not understood. The jumbled appearance of the outer nuclear layer often associated with it ostensibly resulted from an excessive number of nuclei that distorted the retina into ripples or near folds suggestive of retinal dysplasia. Indeed, this apparently anomalous proliferation of photoreceptor nuclei probably is not a part of the essential pathologic process, for it did not necessarily accompany appreciable degeneration of rods and cones, nearly always occurred centrally in the retina, and appeared only in eight sapphires five to seven years old.

The apparent disappearance of the tapetum cellulosum in most mink more than one year old is an unusual and unexplained finding. It resembles that seen sporadically in some beagle dogs, but differs from the hereditary tapetal abnormality seen in others. Whether it is peculiar to the mink I examined or occurs generally in ranch mink was not determined. In any event, it does not seem directly related to the retinal changes. Degeneration of tapetal cells, however, may accompany degeneration of photoreceptors, as in cats fed a taurine-deficient diet or treated for a long period with chloroquine.

The somewhat earlier appearance, greater prevalence, and greater severity of the retinopathy in sapphire than in pastel mink parallels other differences in responses between these dissimilar color phases: greater susceptibility of sapphires to pyogenic bacterial infections, Aleutian disease, periodontal disease, and a lymphoreticular proliferative disease. Presumably in all, this greater susceptibility is largely an expression of profound constitutional differences, some affecting longevity, brought about by the pleiotropic effects of the Aleutian gene in sapphires. No doubt, some of these effects are manifestations of the cellular abnormalities that characterize the Chediak-Higashi syndrome in man and animals. But what bearing these differences between the two color phases really have on the occurrence and severity of the retinopathy is not clear. The decreased prevalence of severe retinopathy in the nine- and ten-year-old pastel mink is unexplained. Apart from color phase, perhaps the year of birth rather than age has some as yet unrecognized epidemiologic significance.

Despite the apparent variation in the age at onset and progression of the retinopathy, attested to by its variable severity in most age groups, I have assumed it represents a single etiologic entity. But none of the ways primary retinal degeneration may come about in animals was established as the cause.

Of the nutritional deficiencies, that of vitamin A is foremost because it causes retinal changes much like those in the mink. Signs of this deficiency in growing mink include night blindness, but the morphologic basis for it has not been described. Published information does not indicate whether a similar visual deficit occurs in naturally deficient adult mink. Although the vitamin A content of the ration fed the mink
examined no doubt varied from one season to another, overt deficiency was not likely. Prolonged vitamin E deficiency also results in retinal damage in several mammalian species.30,52 Mink naturally deficient in this vitamin suffer from yellow fat disease.22 In its usual acute form, it affects mainly rapidly growing male kits three to four months old when their diet contains rancid fats or is high in polyunsaturated fatty acids. Vitamin E deficiency was not a probable cause of the retinopathy in these mink, for their ration was fortified regularly with alpha tocopherol, their retinal changes differ from those associated with this deficiency in other species, and none had lesions of subclinical yellow fat disease at necropsy. Deficiency of taurine, a sulfonic amino acid, causes retinal degeneration in cats fed commercial dog food.1 I do not know whether mink require dietary taurine or whether they can meet their need for it by biosynthesis. Nevertheless, their usual diet of fish, chicken, and beef by-products should have a high concentration of this amino acid. To have accounted for the epidemiologic pattern of the retinopathy in the mink, these or other nutritional deficiencies perforce would have had to occur repeatedly over several years. This was highly unlikely with the standard ration fed.

Many chemical substances, including drugs, can cause selective damage to the outer layers of the mammalian retina.17,46 Some act primarily on the visual cells, others on the pigment epithelium. In either case, degeneration of the outer segments of the photoreceptors ensues. Species vary in their susceptibility to such damage,17 but nothing is known about the possible responses of the mink to retinotoxic chemical substances. Although no exhaustive search of the mink’s environment was made for them, none was apparent that might have accounted for the retinal degeneration—and in view of its widespread occurrence over several years, none seemed likely.

In several species of animals, ambient light, even at low intensities, can cause degeneration of the photoreceptors like that seen in the mink.30 Outer segments are affected first and then the inner segments and nuclei. Eventually, all disappear, leaving an otherwise apparently intact retina devoid of visual cells. Usually, prolonged exposure is necessary to bring about such light-induced degeneration. By itself, ordinary daylight, to which the mink were exposed (46°10′N latitude), probably would not be detrimental to the photoreceptors, but nothing is known about the susceptibility of the mink’s retina to the damaging effects of light. Perhaps the mink, like the albino rat,48 is unusually sensitive to light. And as in the rat,49 this might be especially so in those color phases, such as sapphire, blue iris, violet, hope, and triple pearl, which have little ocular pigment19,53 and are photophobic.10 In any event, ambient light, even though not a primary cause of the retinopathy in mink, could accentuate the degeneration arising from other causes.

In view of its high prevalence at a relatively early age, the retinal degeneration may be largely an expression of an inherent constitutional state in which the structural integrity of the photoreceptors began to wane in many mink sometime after they reached two years of age. Even if this alone did not account for the eventual disappearance of the photoreceptors in some affected mink, it might have made the visual cells more vulnerable to further damage by other means. The term abiotrophy, as first proposed in neuropathology16 and as later used more specifically in ophthalmic pathology,11,12 would seem to apply to this phenomenon, though it does not explain how the degeneration comes about. Of all the possible explanations for the retinopathy in these mink, this seems the most plausible now in both color phases, despite their obvious genetic dissimilarity.

If this supposed proneness to early retinal degeneration is an inherent characteristic, it is not determined genetically in a simple way, as are the inherited retinal degenerations and dystrophies of animals.2,24,35 Other than those autosomal genes (mostly recessive ones) that have recognizable effects mainly on coat color, the genetic constitution of ranch mink generally is understood poorly.28,29 Under breeding practices that prevail in commercial herds, it probably is extremely variable in most mink. This genetic heterogeneity may have been less so in the mink I examined. From 1960 to 1978, when the last ones were obtained, each color phase had been selectively random-bred (for fecundity and fur quality) in a small (250 females) herd without the addition of other breeding stock. Whether this
uncommon practice had any bearing on the occurrence of the retinopathy in them, however, is problematic. Moreover, I do not know whether the retinopathy is peculiar to mink from this herd or whether it occurs also in mink with a different genetic background.

Of the various natural and experimental diseases affecting the mink examined, only scrapie has been associated with progressive degeneration and loss of photoreceptors like that in these mink.\textsuperscript{6,7} Thirteen mink, mostly two and three years old, were affected with either scrapie or transmissible mink encephalopathy, which are caused by the same virus. Five had moderate or severe retinal degeneration no different from that in the other mink. Whether it resulted from scrapie virus infection is not known, but because of the widespread occurrence of the retinopathy in non-infected mink, it probably did not.

The clinical effects of the retinal degeneration were not evaluated, but reduced visual acuity surely attended it. Residual photoreceptors in some affected mink no doubt allowed partial visual function, particularly light-dark discriminations. The apparent absence of photoreceptors in others must have made them blind. Even so, when ranch mink are confined to familiar surroundings, visual deficits, even blindness (as from bilateral cataract), do not restrict unduly their normal daily activities, such as eating and drinking or moving about the cage.

Perhaps of more practical concern are the effects of photoreceptor damage on biologic rhythms controlled by periods of light and darkness.\textsuperscript{26} In the mink, a photoperiodic animal, the annual furting and reproductive cycles are influenced profoundly by light.\textsuperscript{26} Thus, in northern latitudes its winter pelage is initiated about mid August by the decreasing length of the day. The coat becomes prime in late November, when gonadal recrudescence occurs in the male; the winter molt soon follows. Completed in early July, it remains intact until late summer, when the winter molt begins again. By exposing mink to artificial light schedules, these closely related cycles of fur growth and reproduction can be accelerated or retarded, sometimes so they are six months out of phase with the normal.\textsuperscript{57}

Damage to the photoreceptors need not cause blindness, only a reduced light response, to disturb such rhythmic functioning.\textsuperscript{36} In mink, a reduced light response might affect not only fur quality but also spermatogenesis, estrus, length of the gestation period, and litter size—phenomena not evaluated in the mink examined. (In suggesting this, I have assumed that in the mink, as supposedly in mammals generally,\textsuperscript{26} photoperiodic control depends exclusively on the retina.) Until the responses of the mink’s eye to light, its intensity and wavelength, are better understood and until more is known about the endogenous control of the annual rhythms in mink, further comment seems unnecessary. Besides, nothing is known about the prevalence of retinal degeneration in ranch mink generally, and because they usually are culled from a breeding herd by 3½ years of age,\textsuperscript{46,27} the occurrence of retinopathy may not be considered of practical importance anyway. In view of my findings, however, a significant number of mink could become affected before they reach that age. (Incidentally, reduced reproductive performance is the main reason for culling mink when still young.\textsuperscript{57}) Thus, both commercial breeder and biologist working with mink should be aware of the implications retinal degeneration has for the control of annual rhythms in this highly photosensitive carnivore.

Acknowledgements

I am grateful to William Anderson, animal caretaker, Merry Schrumpf and Monte Thompson, histotechnologists, Liza Hamby, librarian, and Charles Taylor, photographer, for their help with this study. I thank Dr. Leon Z. Saunders for comments that improved the manuscript.

References

28 Johansson, I.: Studies on the genetics of ranch-bred mink.

I. The results of inbreeding experiments. Z Tierzücht Züchtungsbiol 75:293–297, 1961
29 Johansson, I.: Studies on the genetics of ranch-bred mink. II. Fecundity, viability and body size of various color mutants. Z Tierzücht Züchtungsbiol 81:55–72, 1965
44 Padgett, G.A.; Gorham, J.R.; Henson, J.B.: Mink as a biomedical model. Lab Anim Care 18:258–266, 1968
47 Prieur, D.J.; Collier, L.L.: Chediak-Higashi syndrome.

Request reprints from W.J. Hadlow, Rocky Mountain Laboratories, Hamilton, MT 59840 (USA).