NATIONAL FUR NEWS
MINK DISEASES
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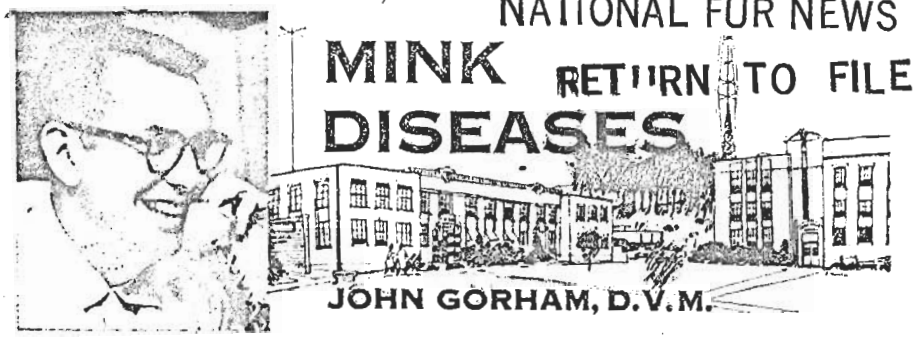

The Early History of Aleutian Disease*

By James B. Henson, George A. Padgett and John R. Gorham

A historical survey of aleutian disease sheds considerable light on the rapidity of its spread throughout the United States and foreign countries. Prior to 1940, in the early years of the mink industry, the ranches were small and relatively inefficient. Mink were trapped in the area surrounding the ranch or occasionally were brought from as far away as Alaska or Labrador. They were considered to be what is termed a "Standard Dark" or "wild type" mink. The problems associated with a proper diet and the devastating outbreaks of distemper and botulism were the ranchers' main concern. Thus, one cannot say for certain whether an insidious disease such as aleutian disease was present in these first ranch-raised Dark mink. However, if it was present, it smoldered along unnoticed or perhaps was confused with some other malady.

The first ranchers, not realizing the possibility of commercially important mutations, promptly disposed of "off-colored" mink. In 1941, an astute rancher near Astoria, Oregon, saved for breeding gunmetal-colored mink that were noticed in a litter of standard Darks.

The color phase was called "Aleutian" after the Aleutian fox which has a similar pelt color. The gene, which was inherited as an autosomal recessive trait, was designated by the symbol "a". By crossing Aleutian mink with other mutant mink, several substrains were developed which were collectively called "Blue mink." Some the pelts were desirable colors, the mink were extremely valuable and commanded high prices. There was a brisk sale of breeding stock and most of the industry geared itself for production of these mutants. In the early forties, Aleutian mink were sold to ranches in various parts of the country. Soon ranchers began reporting losses due to a condition seemingly confined to "Blue mink."

Mink ranchers first noticed the condition when their "Blue mink" lost weight, although there was usually no noticeable drop in food consumption. Faulty digestion was manifested by cereals and/or other undigested food stuffs in the feces. Dark tarry droppings were occasionally observed. Affected mink exhibited marked thirst. About 10 to 20 percent of those visibly sick bled at the mouth. Ragged ulcers at the gingival border, usually involving the teeth, bled when the mink were handled. Ranchers called them "bleeders." The mink were anemic as evidenced by pale mucous membranes and foot pads. They became progressively thinner until death intervened.

Hartsough (1) first observed the gross lesions of aleutian disease in 1946. These lesions were recorded in "Blue mink" shipped to the Midwest. At first the disease was thought to be limited to the aa genotype inasmuch as AA and Aa mink raised on the same ranch appeared resistant. Some ranchers tried to cope with the malady by crossing back their Aleutian mink to AA stock and/or breeding Aa and Aa. While the aa progeny were somewhat stronger than their original Aleutian breeding stock, they were still more responsive to the disease than Aa or AA mink.

As knowledge of the malady increased, it became known as "kidney disease." Depending on the stage of the disease, the kidneys were enlarged and reddened, or swollen, pale and pitted. Other necropsy findings included initial enlargement of the spleen and lymph nodes, and occasional yellowish brown mottling of the liver.

The Infectious and Contagious Nature—The losses from aleutian disease increased each year. Then a series of "vaccine incidents" occurred, which focused attention on the malady, suggesting that the disease might be of an infectious nature (2). Prior to the advent of chicken-embryo-origin attenuated distemper vaccines, it was common practice to prepare inactivated autogenous tissue vaccines. To make this vaccine, spleens were collected from distemper-infected mink and ground with saline containing 0.3 to 0.5 percent formalin. After the mixture was incubated for a variable length of time—often dependent on how badly the vaccine was needed—it was injected. Following the use of these vaccines, widespread aleutian disease losses occurred. In 1949 one rancher lost 500 mink to aleutian disease after vaccination for distemper with such a vaccine. Floyd Marsh, a

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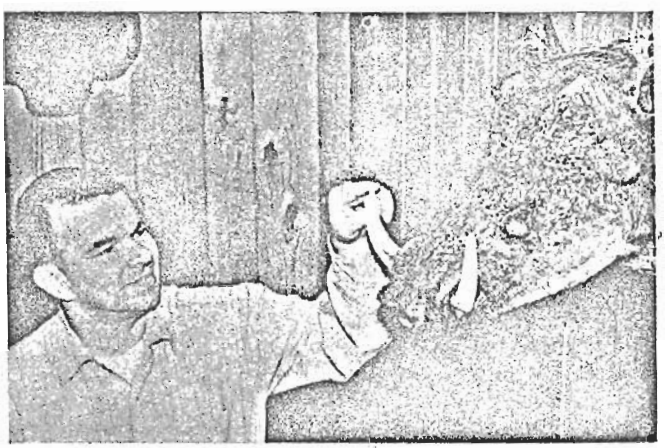

Paul Autio—well-known mink farmer and equipment manufacturer. In 1941 two Gunmetal kits appeared in one of his litters. This was the first time the Aleutian gene was recognized.

Table I.—Losses From Naturally Occurring AD on One Ranch Comparing aa and Aa or AA Mink From December 1960 to December 1961

	Aleutian mink aa			Non-Aleutian mink Aa or AA		
	Total number of animals	Total death loss	Mortality rate (percent)	Total number of animals	Total death loss	Mortality rate (percent)
Adult females (animals carried over for breeding)	655	311	47.5	740	44	5.9
Adult male (animals carried over for breeding)	164	128	78	185	18	9.7
Kittens (born during current year)	1,800	162	9	2,130	38	1.8

* This paper was taken from NINDB Monograph No. 2. Slow, Latent, and Temperate Virus Infections, pp. 279-285.

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Virginia Casey Visits Eau Claire Mink Ranch; Holds Style Show for Women's Club Members

Modeling mink garments for Women's Club style show are Mrs. James Hobart of Eau Claire, Mrs. Earl Morrison of Chippewa Falls and Mrs. Arnold Wold of Eau Claire.

Emba's Virginia Casey brought her traveling style show to the women of the Chippewa Falls and Eau Claire, Wis., Women's Clubs on October 4. She spoke to over 200 women on the "Magic of Mink" telling about American mink from the raising of the animals on the ranch to the finished fur garment.

Fur garments of Emba and Great Lakes mink were modeled by members of the clubs. Miss Casey scored a terrific hit by modeling some of the outfits herself. The garments were from New York and Minneapolis furriers.

On October 2nd, prior to the style show, Mr. and Mrs. H. T. Helfrich held a buffet supper for Virginia Casey. Mrs. Helfrich, whose husband is a mink rancher, is a member of the Women's Club and helped with the arrangements for the show.

Some ranchers and their wives from the Chippewa Valley were present for the supper. Guests included Mr. and Mrs. Glen Harmston of Menomenee, Mr. and Mrs. John Wenstadt of Elk Mound, Mr. and Mrs. John Klinger of Eagle Point, Mr. and Mrs. James Thorson of Augusta, Mr. and Mrs. Everett Mueller of Chippewa Falls and Mr. and Mrs. Walter Becker of Eau Claire.

Following the meal, Miss Casey told the ranchers of her work, her visits to all parts of the United States holding fashion shows and telling of Emba's program, and her contacts with furriers to guarantee that the ranchers' products would not be misrepresented.

The guests modeled some of the mink garments that were stored in the Helfrich home prior to the Women's Club style show.

Ranchers' wives dress up in mink after buffet at Helfrich home. Kneeling are Ann, 7-year-old daughter of the Helfrichs, and Mrs. Wenstadt. From left are Mesdames Thorson, Harmston, Helfrich, and Virginia Casey.

Barry Coward (left), president of Hudson's Bay Co., presents plaques to William Fitzgerald (center) and Earle Cantor, newly elected president and board chairman, respectively, of New York Auction on behalf of the New York Fur Dressers. The occasion is a cocktail reception at the Plankinton House in Milwaukee during the mink rancher association meetings in October. The dressers cited Cantor and Fitzgerald for industry leadership and business achievements.

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truly colorful mink raiser, said "they didn't make baskets big enough to pack out my dead mink."

A few years later a Connecticut mink rancher who had aleutian disease on his ranch decided to try one of these homemade vaccines against the disease. The result was reminiscent of the scrapie outbreaks which followed the use of louping-ill vaccine (3). Helmboldt and Jungherr (4) reported that nearly all of the inoculated mink were dead of aleutian disease after six months!

By 1950, it was apparent to many mink ranchers that aleutian disease was contagious. When mink from an affected ranch were brought to a "clean ranch," the disease appeared in the purchased breeding stock as well as in other mink on the ranch. Such a prospective study was made in western Oregon in 1955 by our coworker, Dr. Keith Farrell. The rancher had many desirable mutations which, although he did not know it, were infected with aleutian disease. They were sold to several other ranches and new foci of disease appeared on these ranches. Overseas shipments by many U. S. ranchers of infected mink may have spread the disease to Scandinavia, England, Japan, Canada, West Germany and elsewhere. However, the possibility exists that aleutian disease was present but not recorded in these countries prior to the U. S. mink exports.

With the history of aleutian disease in mind, we might speculate about the apparently sudden appearance of this "virus." Two alternatives appear possible: (1) the virus appeared about the same time and in the same area as the Aleution gene mutation, or the virus might be associated with the Aleutian gene; (2) the virus was present in mink prior to selection of the Aleutian mutant but was not recognized until sufficient numbers of highly responsive aa mink became available in the ranch mink population. In considering the first possibility, it appears extremely unlikely that matching mutations would occur, not only at the same time, but in the same area.

The second proposal is not only more probable but is intellectually more acceptable. When the "Blue mink" were shipped from the Oregon ranches, some were infected with aleutian disease while others were free of the disease. Infected animals established new foci of disease on clean ranches. On the other hand, when noninfected "Blue mink" were sent to ranches where aleutian disease was present in standard Dark herds, the newly purchased "Blue mink" served as sentinal animals by succumbing to aleutian disease.

There is no direct proof to support either of these possible explanations for the origin of the aleutian disease agent. However, if we could locate

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preserved tissues or tissue sections of standard Dark mink with aleutian disease lesions in the late thirties, it would prove the latter possibility.

The Chediak-Higashi Syndrome—It was not until 1963 that a possible explanation for the remarkable genotype responsiveness of aa mink to aleutian disease virus was recorded. Leader, et al. (5) and Padgett, et al. (6) described a condition occurring in mink and cattle which resembled strikingly the Chediak-Higashi syndrome (C-HS) previously described in man (6).

In man, mink and cattle, the syndrome appears to be caused by a simple recessive non-sex-linked gene. Among mink, the only animals affected with this condition are those which are homozygous recessive for the aleutian gene "a". Thousands of mink have been examined, and we have never observed a mink with the aa genotype which does not have the C-HS.

In one study of C-HS in mink, Padgett, et at. (7) inoculated 40 aa and 40 Aa or AA mink with 10^4 ID_{50} of the aleutian disease virus. All animals developed aleutian disease. Of those with the aa genotype, all but four were dead within four months after inoculation. In the same period, only four of the non-aleutian (Aa or AA) animals were dead. After nine months, one-half of the latter group survived and six mink lived for more than a year. These findings and the results of similar work suggest that all mink are susceptible to aleutian disease, but early death occurs primarily in mink of the aleutian genotype. Indeed, it was for this reason the name aleutian disease was given to the condition. The difference in mortality rate between Aleutian and non-Aleutian mink in naturally occurring aleutian disease is illustrated in Table I.

The idea that the genetic constitution of the host has a profound effect on the severity of disease caused by a particular virus is certainly not new. One strain of potato X virus kills one variety of potato, causes a mosaic in another and is completely asymptomatic in a third. All three varieties of potato are capable of transmitting the virus.

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(3) Gordon, W. S. Scraple Panel, United States Livestock Sanitary Association, Proceedings Sixty-Third Annual Meeting, 286-294, 1960.
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(7) Padgett, G. A. Unpublished data, 1965.

W. A. Styer Promoted By Kellogg's Company

W. A. Styer has been appointed to assistant to the sales manager in the food department of Kellogg's according to L. C. Borsum, Feed Division Sales Manager.

W. A. Styer

Styer joined Kellogg's in 1956 as a member of the feed sales selling organization, assigned to Michigan, Ohio, Indiana and the East Pennsylvania area.

A native of Battle Creek, W. A. Styer graduated from St. Phillips High School and attended the University of Mississippi. In 1951, following four years of duty in the Michigan National Guard, he served 16 months in the U. S. Air Force.