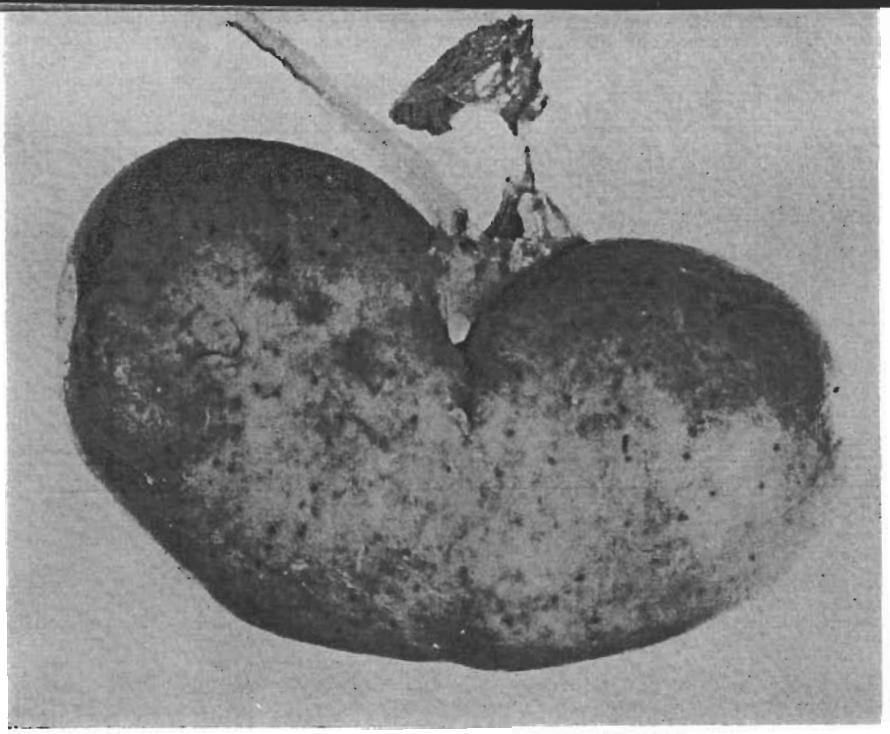

Aleutian Disease In Mink

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WITHIN a year or two after the observation of the Aleutian mutant (1946), ranchers reported losses that seemed to be confined to this mutation. Deaths in Aleutian stock were observed from all of the mink raising areas of North America and Scandinavia. The purpose of this paper is to provide a description of the most common disease in Aleutian stocks and to present what limited knowledge there is available.

[1]Associated Fur Farms, and Great Lakes Ranch Service.
[2]Fur Animal Disease Research Laboratory, A.R.S., U.S.D.A., in cooperation with the Department of Veterinary Science, Washington Agriculture Experiment Stations, The State College of Washington.

Cause—All attempts to determine the cause of this malady have met with failure. Tissues from Aleutian mink dying of the disease have not proved infectious for other mink and laboratory animals such as young chicks, mice, rats, hamsters, and guinea pigs. No significant bacteria have been isolated through the use of agar plates grown in the presence and absence of oxygen. Special tissue staining and culture for leptospira have not been conclusive. Limited blood studies have not revealed data which are pertinent. However, the marked increase in the number of affected mink following the administration of improperly prepared formalized tissue vaccines from affected Aleutians suggests that the cause may be transmissible to other color phases under artificial conditions.

Since the disease is found in ranch mink homozygous for the Aleutian gene, field observations indicate that there may be a genetic relationship to the cause. Out breeding has provided some measure of control. Ranchers experiencing high Aleutian losses nearly always report that Darks and hybrids were not affected. However, to further confuse the situation, the disease is occasionally reported in other color phases. The possibility of two or more unrelated conditions occurring as "Aleutian disease" must be given consideration.

Symptoms—Mink ranchers first notice the condition when certain of their Aleutians or Sapphires seem to stop gaining weight or lose flesh. Usually there is no accompanying drop in food consumption. Faulty digestion is manifested by cereals and/ or other indigested food stuffs in the feces. Dark tarry droppings are occasionally observed. Affected mink are extremely thirsty. About 10-20 per cent of those that are visibly sick bleed at the mouth. The mink are often anemic and blood clotting may be delayed. Later in the course of the disease, they become progressively thinner until death intervenes. Imperial Sapphire are very prone to contract the malady.

Gorham Hartsough

Lesions — In those animals in which the mouth is affected, there is a marked inflammation of the gums around the teeth (gingivitis). At one or more points on the gums there are small or large ragged ulcers. When the mink is handled, these ulcers tend to bleed. In the later stages, the inflammation extends to and involves the front teeth, thereby causing decay and loosening of the teeth. Blackened ulcerated tongues have been reported. The lesions of the mouth may be the result of the retention in the blood of urinary waste products.

When the abdominal cavity is exposed, the most significant finding is the kidney lesions. Depending upon how long the disease has progressed, the kidneys appear enlarged and reddened, swollen and pale with small hemorrhages (Figure 1) or in the late stages of the condition, shrunken and pitted. Other findings include enlargement and dark splotches on the spleen, yellowish brown mottling of the liver—evidence of degeneration. Hemorrhage is occasionally found in the intestine. One gains the general impression that the animal is anemic because of pale membranes. The lungs are normal in color and consistency.

The microscopic findings consist of nephritis (inflammation of the kidneys), degeneration, inflammation and death of tissue in the liver. A change that is constant is that of a marked inflammation of the blood vessels.

Course—The condition tends to be chronic in nature. Only a small percentage of Aleutians or Sapphires less than six months of age are affected. In older individuals the incidence increases sharply. Ranchers have experienced mortalities during the second summer in excess of 50 per cent. Mink affected with this malady may die within two weeks or may linger for three months or more before dying. Nice infrequently after a cold snap in the fall many chronically affected individuals will die. These mink appear to be very susceptible to other infections. Handling often initiates a series of deaths.

Prevention and Treatment—There seem to be a few ranchers having large stocks of Aleutians and Sapphires that are remarkably free of the disease. Some ranchers have accomplished some measure of

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Floyd Marsh

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and you get a cheap pelt." He's a strong believer in horsemeat.

Going through the large, airy, clean sheds and admiring both the beauty and the number of newer types of mink, the question came to me over and over: Where did this ex-cop, who talked of color combinations with the ease of a skilled painter mixing colors, acquire such a wide knowledge of genetics? All progressive mink ranchers have a good working knowledge of genetics, but here was a fellow who seemed to have gone beyond what he could learn from books, lectures and actual experience. So I asked him.

"I owe most of what I know about genetics to Dr. W. E. Castle," Floyd said, "For forty years he was a teacher of genetics at Harvard. I believe he lectures now at the University of California. We've written back and forth for many years. I kept all his letters, still read them over. Dr. Castle started me in genetics."

"And you've been at it ever since?"

"My head's always full of it. I figure five years ahead on colors."

"And you still do some prospecting?"

Floyd grinned. "Uranium," he confessed. "Everytime I get back, I think more of my mink."

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trol by out crossing to vigorous strains of standard Dark and other mutant color phases. The avoidance of the Imperial Platinum gene, although a beautiful mink, should be given consideration.

Broad spectrum antibiotics such as aureomycin and terramycin in the diet, either at low or therapeutic levels, have had no influence on the course of the disease. Similarly, the use of sulfa drugs has been discouraging.

Injections of penicillin and crude liver extract seem to provide some temporary relief; however, almost all of these mink will eventually die of the disease.

Future Plans — Realizing the seriousness of a disease in a popular mutation in which the cause, prevention, or treatment is unknown, the **Mink Farmers Research Foundation** has provided a substantial grant to the University of Connecticut. This work is under the direction of Doctor Erwin Jungherr, one of the foremost veterinary pathologists in the United States. We feel confident that the work at Connecticut will contribute a great deal to the knowledge of the disease.

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