Acute Enteritis of Unknown Origin in Mink

by

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In January, 1967, we received word from a mink farmer in western Washington of an alarming number of deaths among his breeder mink. This report elaborates on our investigation of the outbreak and our failure to reproduce the condition in experimental mink.

We, as well as Dr. G. R. Hartsough of Pittsville, Wisconsin, have never observed a similar outbreak. Thus, our purpose is to describe the condition in the hope that other workers investigating similar conditions can provide more accurate knowledge. If we were faced with a similar outbreak at this writing, we would not know how to control it.

HISTORY OF LOSSES

At the beginning of the outbreak in January, 1967, the farmer had 186 male and 914 female mink of various genotypes. The animals were vaccinated against botulism, but not against distemper and mink virus enteritis. Vaccination against the latter was recommended and accomplished early in February. We feel that mink virus enteritis was not involved because (1) the losses continued until pelting, (2) transmission trials were negative in susceptible mink, and (3) affected mink did not go off feed suddenly—they continued to eat.

By the end of January, 1 or 2 mink were dying each day; both males and females were affected. The losses peaked in early March when 9 deaths were recorded in one day. From then on, the deaths did not cluster, and by July a total of 170 adults had succumbed. Almost 300 kits were lost from the disease itself and from lack of adequate nourishment and care by affected nursing females.

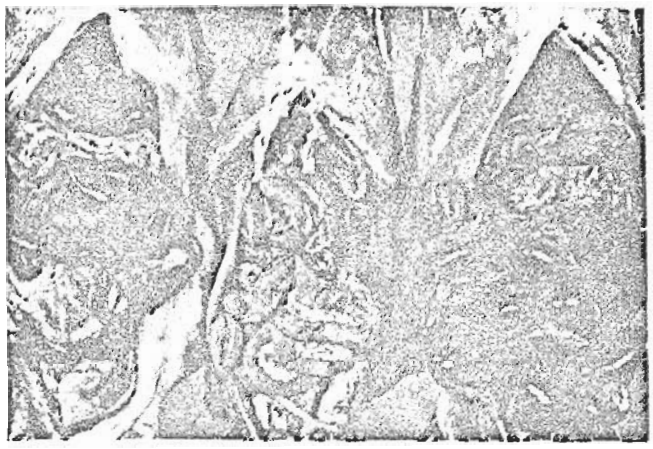
SYMPTOMS

One of the striking features noticed by the owner was bright, almost fluorescent, green droppings. They were not the greenish droppings seen when animals are off feed or affected with mink virus enteritis. Also, the feces of affected mink may be thin and bright red accompanied by watery mucus. Occasionally, the feces appeared like unaltered blood. As the disease progressed, dehydration and lassitude dominated the picture. After 3 to 6 days, the animals became weaker and death ensued or the mink slowly recovered. During this period the mink continued to eat a small amount of food. A few mink showed signs for 2 or 3 weeks.

THE NECROPSY

If one can do a necropsy on a dozen dead mink, he will see that the lesions follow a pattern that is distinct from other common mink diseases. The most frequent lesion is an extremely marked inflammation of the small intestine. Even without incision, the dark red involved portion can easily be demarcated (see accompanying photograph).

A pathologist would describe it as an acute hemorrhagic enteritis with necrosis and sloughing of the lining cells. Occasionally ulcers perforate through the walls of the intestine (manifested by circular greyish areas on the outside of the intestinal wall) leading to peritonitis. Inflammatory fluid is often observed in the peritoneal cavity. The lymph nodes in the abdominal cavity may be hemorrhagic and enlarged. The spleen may or may not be enlarged.



The involved portion of the intestine is a dark red color. A normal mink is included (center) for comparative purposes.

LABORATORY FINDINGS

We are indebted to Dr. Frank Crews of the Washington State Diagnostic Laboratory for his bacteriologic findings. He stained smears of the intestinal contents of dead mink and found large numbers of bacteria resembling **Clostridia**—a kind of bacteria that is probably found in normal mink ingesta, but in this instance almost all of the bacteria he observed were of this type.

From the livers of fresh dead mink, he isolated **Clostridium perfringes**, Type C in pure culture. This bacteria belongs to the same group as **Cl. botulinum**, but is distinct. Identification as to type was made by conducting toxin-antitoxin protection tests in mice.

Microscopic sections made by Dr. H. D. McCausland of the Veterinary Laboratory, Abbotsford, British Columbia and ourselves revealed massive numbers of large gram positive rods presumably **Clostridia** bacteria in the lumen and throughout the wall of the intestine. These were mink that had succumbed within an hour or two before necropsy. This organism usually migrates into the wall of the intestine and liver shortly after death. Fatty degeneration of the liver and kidney cells was also recorded.

Bacteriologic examination of one ingredient of that diet—ground chicken viscera—revealed the same organism. However, we discounted the importance of this finding inasmuch as the organism is probably always present in such chicken by-products, and millions of tons of this feed are fed each year with no apparent trouble.

CONTROL

Before obtaining the bacteriologic findings, we recommended a course of sulfaguanidine, a treatment we have used for 25 years with varying degrees of success. Since

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the losses continued, we advised that a neomycin product be administered in the feed. The owner repeated both treatments without reducing the deaths. When we received word of the **Cl. perfringes** isolation, the therapeutic attack on the outbreak shifted.

In the middle of February the owner obtained antitoxin directed against **Cl. perfringes**, Types B, C, and D. According to his observations, an intraperitoneal injection of 5 to 7 ml led to recovery of up to 90% of those affected. However, there was often a return of clinical signs and the mink had to be treated again within a few days.

Obviously, the continued treatment of a whole farm of mink was a time-consuming and an expensive procedure. In April, all the mink were vaccinated with Type C toxoid that later was shown to have no effect in protecting mice given challenge inoculation of Type C toxin.

In July, the kits and adults were vaccinated with a C-D toxoid of another commercial concern. This vaccination was repeated when the kits were 10-12 weeks of age.

The losses continued throughout the summer. To complicate the picture still further, antibody titers against leptospirosis were observed in September, 1967. Although this organism may have contributed to the preceding losses, it seemed that animals dying in September also had a yellow tinge to their mucous membranes. Needless to say, an investigation of the role of leptospirosis in mink is contemplated.

ATTEMPT TO REPRODUCE THE NATURAL DISEASE

In an attempt to reproduce the disease in mink and ferrets, surgical procedures were performed so that toxin, bacterial cultures or intestinal contents could be introduced directly into the small intestine and thereby by-pass the effect of the stomach. Atropine sulfate was administered to slow down the peristaltic action of the intestinal tract. No mink or ferret had signs of illness when inoculated directly in the lumen of the intestine. If, however, the inoculum leaked into the peritoneal cavity, the animal promptly died. The disease could not be reproduced in mink or ferrets by intubation of toxins or bacterial cultures or by scarification and direct inoculation into the lumen of the intestinal tract.

COMMENT

We are obviously confused as to the role of the **Clostridium** bacteria in this outbreak. Firstly, this organism was isolated from the mink feed in January and from the mink throughout the outbreak. Furthermore, the antitoxin was of temporary benefit but the toxoids probably were not —at least the losses continued. Even though leptospirosis was isolated, we still have the feeling that we overlooked crucial points leading to an accurate diagnosis.