DISTEMPER IN FUR BEARING ANIMALS AND NEW DEVELOPMENTS IN MINK ENTERITIS

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I — DISTEMPER

Since 1932, when Shaw[1] first described the disease in mink on this continent, the word "Distemper" has been a by-word for fear among mink ranchers. A disease which may present a varied and confusing picture clinically and one which has challenged our skill in control methods for the past twenty years, even now according to a report from the Department of Lands and Forests[2], it causes the loss of over 1700 mink in one year, on Ontario ranches.

CONTROL —

Careful scrutiny of the difficulties involved in the control of distemper on mink ranches reveals this to be a two fold problem involving the mink rancher himself with the headaches involved in the operation of a modern mink ranch, and the gaps in our knowledge concerning distemper as a disease entity.

Although mink ranchers have a comprehension of practical mink genetics which puts most of us to shame, their appreciation of the importance of sanitation, and its implications in disease control, is frequently inadequate. On the modern mink ranch the practice of keeping mink in individual pens is of definite importance in inhibiting the spread of disease, but this is overbalanced by the tendency to crowd 200 or more pens under one roof, and to congregate as many as 2,000 animals on an acre of land.

Preventive vaccination would be a possible solution to the problem, but most ranchers operate on a small margin of profit at best and are therefore unwilling to undertake a programme of annual vaccination which will slice that margin even thinner, especially when there is apparently no distemper outbreak and to their knowledge therefore, no source of infection in the neighborhood.

The complications which present themselves when one considers transmission of the distemper virus, are manifold. The incubation period for distemper in experimentally inoculated mink is approximately 10 to 14 days — however observation of distemper in the field indicates the possibility of a much longer incubation period. Raccoon, skunk, foxes, ferrets, and dogs, may be susceptible to infection by the virus of distemper, and are therefore potential carriers. Airborne transmission, and arthropod or rodent vectors have also been suggested as sources of infection. From field experience it has been recognized that outbreaks of distemper tend to recur on mink ranches 1 to 3

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or even 5 years following the initial outbreak. Often no source of reinfection appears to be present. Where has the virus hidden in the intervening years?

Gorham[3] described an experiment in which the virus of distemper was demonstrable in the nasal discharge of mink for from 46 to 51 days after initiation of infection or about 14 days after signs of lesions on the eye, nose, and skin had disappeared. He also demonstrated the survival of the virus on gloves for as long as 20 minutes following their use in handling infected mink.

Pens constructed partially of wood which were scrubbed with 1% lye solution after housing ferrets which died of distemper, were considered by the author to be the probable source of infection for an outbreak of distemper in ferrets housed in the same pens three months later.

Research workers at first chiefly concerned themselves with the clinical manifestations of distemper and the development of a useful vaccine. Since the successful cultivation of the virus of distemper in the embryonating egg as reported by Haig[4,5] and others[6,7] following the early lead of Plummer[8] more attention has been focused on basic research. There is a possibility that the serum neutralization test now being used in the laboratory will be a useful tool in determining whether there are strain differences in the virus of distemper as is indicated by the observation in the field of variation in clinical manifestations of the disease. It may also prove useful in determining the reservoirs of infection. Morse[9] has recently accomplished the serial passage of the distemper virus in the brain of suckling mice. This promises to be another very useful research tool.

Prevention

Consideration of prevention must include elimination of possible sources of infection. New stock should be quarantined for 60 days before being introduced to the ranch area — however this is not a simple matter when, as frequently happens, new stock is acquired a short time before the breeding season and close contact with other ranch animals is inevitable.

Dogs and other known susceptible animals are a possible source of infection, but most ranch areas are now fenced to exclude the larger prowlers, and modern methods of pest control will keep rodents at a minimum. Fur shows afford an excellent opportunity for spread of contagious disease. Four hundred or more animals from ranches over a wide area are congregated in one building, therefore it is a wise precaution to quarantine the animals on their return to the home ranch.

With the cooperation of the rancher such contacts can be controlled. When the source of infection is unknown and previous theories of carriers, etc. seem to be inapplicable, the obvious course is protection by immunization.

Protection of the herd can best be accomplished by vaccination of the breeding males and females in January — after pelting has been accomplished

and before the breeding season commences. This procedure followed by vaccination of the kit crop shortly after they are weaned, gives maximum protection at all times.

Two types of vaccine are in common use — the inactivated virus vaccine, and the modified live virus vaccine.

The inactivated virus vaccine was used by Pinkerton[10] in 1940 and was the only widely used method for approximately ten years. Although it possesses the advantage of being a safe method because no virulent organism can be introduced to a ranch by its use, it has the inherent limitations of any inactivated virus vaccine. The immunity produced is transient. Hartsough[11] found that animals vaccinated during the winter were often susceptible to field strains of distemper by the following summer.

Haig[5] in 1949, reported that after serial passage in the embryonating hen egg the distemper virus showed marked attenuation in virulence for ferrets. He advocated the use of this attenuated strain of virus for immunizing purposes. Hartsough[11] used a vaccine of this type on approximately 60,000 mink on 120 ranches which were free of distemper. Neither systemic reactions nor outbreaks of infection were observed following its use.

Crawley and Walker[12] used a similar type vaccine on more than 8,500 mink on 12 non-infected Ontario ranches in 1952. Distemper had not occurred in any of these vaccinated animals during a 12 month period following vaccination. On one of these ranches distemper occurred in the non-vaccinated adults causing some mortality but not one of the vaccinated kits showed evidence of disease.

Insufficient data is available concerning the duration of immunity in mink following vaccination with attenuated virus vaccine, but it is probable that it results in a more lasting immunity.

Cabasso et al[15] reported that ferrets given a single injection of a chick embryo adapted live virus vaccine were found to be immune to challenge with a lethal dose of virulent distemper virus as long as two years following the inoculation.

Crawley and Walker[13] state that six months following inoculation of mink with a live virus vaccine 56.6% of the inoculated controls succumbed to challenge with a field strain of virus, but only 2.4% of the vaccinated mink died of distemper.

Treatment of an Outbreak

The importance of early diagnosis in the control of distemper during an outbreak cannot be overemphasized. Distemper outbreaks may occur at any time of the year but are more common during the summer when the mink population has been suddenly trebled by the advent of the highly susceptible kit crop.

In its early stages distemper in mink may be hard to differentiate from any other disease, but observation of 2 or 3 animals in various stages of infection will facilitate diagnosis. First signs of infection are usually a squinted expression and slight watery discharge from the eyes, followed by swelling of the eyelids, and purulent lacrimal exudate which dries and causes the eyelids to stick together. There may be a purulent nasal exudate. A slight swelling of the bridge of the nose sometimes gives a "Roman nosed" appearance. The lips may develop pustules which become massed into a dried crusty exudate around the mouth. The foot pads which at first are very hyperemic and swollen become encrusted with light brown granular material. The skin ventrally may be hyperemic. Anorexia is inconstant in the early stages. Occasionally clinical manifestations of involvement of the central nervous system are evidenced — often in the later stages of the disease, and the animal dies in a "screaming fit". A diagnosis of distemper cannot be made by gross examination of the pelted carcass. Clinical diagnosis may be substantiated by laboratory examination. Characteristic inclusion bodies can usually be found in stained sections of the bladder and trachea.

Immediate destruction of all mink showing signs of infection and vaccination of all other mink on the ranch is the most effective method of dealing with an outbreak. Previously, vaccination with inactivated vaccine was the only available method for attempting to control the spread of distemper on a ranch, and results showed that it was difficult to produce immunity rapidly enough to be effective.

Cabasso and co-workers[15] in 1951, first reported the use of an egg adapted modified virus vaccine during an outbreak of distemper on two mink ranches. The results were apparently satisfactory.

Hartsough[11] in 1953, reported the use of a similar vaccine on 35,000 mink from 23 ranches where there were infected animals. Vaccination was accomplished on each ranch not more than 2 or 3 days following positive diagnosis of distemper on the premises. Animals showing signs of distemper were killed and removed from the herd. The mortality due to distemper on these ranches did not exceed 3% in any case.

Baker, et al[16] observed that ferrets which received a strain of attenuated virus two or more days before challenge with a lethal strain of virus were apparently immune to distemper. Cabasso and co-workers[12] noted ferrets to be resistant to infection as early as 24 hours following vaccination with an egg adapted strain of distemper virus. Both groups however, suggest the possibility of this early resistance being a manifestation of the interference phenomenon. Whatever the cause, this exhibition of early resistance in ferrets following vaccination with egg adapted strains of virus may explain the apparently successful use of live virus vaccines in the control of distemper on some mink ranches.

However Baker, et al[16] also report that ferrets inoculated simultaneously with egg-adapted virus and the challenge dose of virulent virus succumbed to distemper, as did those inoculated with the egg-adapted virus 2 days following challenge. Remembering that the incubation period for distemper in mink may be 10-14 days, or possibly much longer, one cannot expect to obtain 100% protection by vaccinating the mink on a ranch during an outbreak, when there can be no estimate of the number of animals already infected. It is inevitable that on some ranches the disease may prove difficult to control.

II — INFECTIOUS ENTERITIS OF MINK

HISTORY —

During the late 1940's an apparently new and highly contagious disease suddenly appeared on mink ranches in the Fort William area of Ontario. This disease known as Fort William disease, virus enteritis, or infectious enteritis, continued to cause severe losses, (as high as 90 percent of kits), on ranches in that area for several years. In 1950 this disease appeared also on two ranches in Southern Ontario. Since that time it has been diagnosed each year on Southern Ontario ranches, the losses at first being alarmingly high. During 1953, approximately 10 ranches were affected. The nature of the causative agent has been considered by Schofield[17] to be a virus. Why this disease suddenly appeared in such widely scattered localities, however, is still unknown.

DIAGNOSIS OF THE DISEASE —

Clinical

Infectious enteritis in mink is a disease primarily affecting kits. Adults may contract it, but less frequently and with a much lower death rate. Outbreaks occur commonly between the time the kits are weaned and the early part of October. The first sign of illness is complete loss of appetite and a tendency to remain in the nest box. Following this the feces may present a slimy appearance or a cast may be noticed. A "Cast" may be described as slightly pink to greyish-white soft material of about the size, shape, and length, of one's little finger. Very loose, curd-like, or blood-stained feces may sometimes be seen. The eyes may have a slightly dull, squinted appearance. Death most frequently occurs within five days following the first signs of illness. The disease spreads rapidly, and the mortality on a ranch becomes increasingly alarming.

Pathological

Post mortem examination usually reveals that the intestine is very hyperemic, indicating severe inflammation. The spleen and liver may be darker than normal. Similar post-mortem findings, however, may be encountered in other diseases; therefore a diagnosis cannot be made on this alone. Laboratory examination of stained sections from the intestine reveals pronounced swelling

of some of the epithelial cells lining the crypts, described by Schofield[17] as "ballooning".

Wills[18] was successful in producing from a formalized emulsion of infected mink tissue a vaccine which appeared to limit the morbidity and shorten the duration of an outbreak. In use since 1951, this vaccine was given in two doses, seven days apart. On ranches where an early diagnosis was made and immediate vaccination undertaken, the number of deaths rapidly decreased after the second vaccination. On ranches where a controlled experiment was conducted, a significant drop in the number of infected animals was noted in the vaccinated group, as compared with an unvaccinated control group, deaths being reduced by about two thirds. In continuing his work, Wills[18] has revealed some very interesting facts concerning infectious enteritis in mink.

1) Sulfonamides and antibiotics were found to be ineffectual in limiting the course of the disease.

2) Finding that infected mink in the later stages of the disease showed leucopenia, he correlated this with feline enteritis or panleucopenia, in which affected kittens show a similar leucopenia.

3) He found that kittens infected orally with tissues from mink which had died of infectious enteritis showed a marked leucopenia and other features suggestive of feline enteritis.

4) He demonstrated that serum from mink which had recovered from infectious enteritis protected other mink from infection, if administered before exposure to the disease, but this serum did not prevent death when given to mink which already showed signs of the disease. This recovered mink serum also prevented leucopenia when administered to kittens which were simultaneously infected with tissue from mink which had died of infectious enteritis.

5) Commercial feline enteritis antiserum was used to prevent infection in mink. This serum was administered a day before and the day following the oral infection of mink with the virus of infectious enteritis of mink. The mink treated with antiserum showed no signs of disease, although control mink infected at the same time, showed typical symptoms of infectious enteritis. Feline enteritis vaccine has also been used experimentally to prevent infection in mink. This work however, needs further corroboration.

6) Laboratory examination of tissues from kittens naturally infected with feline enteritis as compared with tissues from kittens experimentally infected with infectious enteritis of mink, showed similar changes in the cells of the intestinal mucosa. Examination of mink tissue from naturally infected ranch animals, and from those infected experimentally, showed changes almost identical to those seen in the infected kittens. Smith[19] states that as in feline enteritis, intranuclear inclusion bodies can be demonstrated in the epithelial cells lining the intestines of infected mink. These specific changes in the cellular structure

of the mucous membrane lining the intestine are used routinely by Smith and Schroder[20] in the diagnosis both of panleucopenia in cats, and of infectious enteritis in mink.

7) Wills[18] concludes, therefore, that the virus causing infectious enteritis in mink is the same as, or very closely related to, that causing panleucopenia in cats.

The means of transmission of the virus are unknown as is the length of time of survival of the virus. It would seem reasonable to assume that cats may be a source of infection, but experience in the field seldom substantiates this assumption. However, it would appear to be a wise precaution to institute measures to prevent the entry of cats to the ranch, and to discourage all non-essential visitors, especially during the summer and early fall months. At the first sign of disease all animals showing anorexia should be isolated and strict sanitary measures taken.

The supply of formalized tissue vaccine is limited by the number of mink which succumb to the disease, therefore the greater the success achieved by control methods the smaller the quantity of available vaccine.

Feline enteritis antiserum or vaccine could probably be used as a prophylactic measure, but the cost would be prohibitive and the supply is somewhat limited. Therefore the veterinarian must stress the rancher's responsibility in mechanical methods of control, and be aware of the importance of an early diagnosis with immediate vaccination as a therapeutic measure.

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CANADIAN VETERINARY MEDICAL ASSOCIATION

The Canadian Veterinary Medical Association will meet in Ottawa on August 30th, 31st and September 1st, 1954. This is a pleasant time of the year to visit the Capital City and arrangements are being made to welcome Association members.

It is unnecessary to point out that the Canadian Veterinary Medical Association fills a long felt want in veterinary affairs in this country. It forms a united body of Canadian veterinarians. No matter what local interests members of the profession in this country may have, each one has an interest in the broad aspects of his profession. Certain factors of these are common to all parts of the country and in fact can only be dealt with by representatives of all parts of Canada.

Our profession in this country, comparatively speaking, is young and is scarcely yet getting into full stride. For many years there was a slow growth and a lack of proper organization. Much of this difficulty has been overcome and we are now set to use the tools which are at hand. Those who come to the Ottawa meeting will have an opportunity of hearing subjects of national importance discussed, of differences debated which affect the lives of all Canadian veterinarians and of hearing papers and veterinary demonstrations which will improve the capacity of each to serve the people of this country. It is expected that the Ottawa meeting will be the largest yet held.

One other point might be mentioned and that is, except for transportation, a visit to Ottawa need not be expensive. There are many hotels, motels, tourist cabins, etc. with varying rates which will accommodate the purses of all. From time to time this journal will endeavour to indicate the arrangements which the various committees are making for your accommodation and entertainment.