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Diseases of Mink

Thomas M. Ford, D.V.M.*

The mink ranching industry is a natural outgrowth of trapping mink and selling the pelts. To supply the increasing demand for garments, mink ranching has steadily expanded until at present it has grown into an industry with an annual gross product of approximately 100 million dollars. By breeding and raising mink under controlled conditions, ranchers are able to produce a higher quality pelt in a variety of mutant colors. However, by nature the mink is not adapted to living in a small cage in close proximity to hundreds or thousands of other mink and this has made it susceptible to many disease conditions. Also, the intensive inbreeding programs carried on by ranchers to develop the mutant color strains has brought out some undesirable characteristics from a disease standpoint.

The potential role a veterinarian can play in providing medical service to the mink rancher becomes apparent when one recognizes the rapid expansion of the industry and the resultant increase in disease problems. In many instances the mink rancher has been in need of veterinary assistance and advice but has been unable to obtain it. The veterinarian can be of real service to the mink rancher merely by familiarizing himself with some of the special problems and diseases that are peculiar to mink and by applying his basic veterinary medical education.

The four major types of diseases that mink are likely to contract are viral, bacterial, nutritional and parasitic.

**Viral Diseases**
- Distemper
- Virus Enteritis

**Bacterial Diseases**
- Abscesses
- Anthrax
- Botulism

**Enteritis, non-specific**
- Klebsiella Infections
- Pneumonia
- Salmonellosis
- Sinusitis
- Streptococcus Infections
- Tuberculosis
- Tularemia

**Nutritional Diseases**
- Chastek’s Paralysis
- Fatty Degeneration of the Liver
- Gastroenteritis
- Nursing Sickness
- Nutritional Anemia
- Rickets
- Steatitis (yellow fat)
- Urinary Calculi
- Urinary Incontinence (acidosis, wet belly)

**Parasitic Diseases**
- Coccidiosis
- Flesh fly, flea, lice and mange mite infestation
- Fluke infestation
- Roundworm infestation
- Tapeworm infestation
- Trichinosis

**Miscellaneous**
- Hereditary
  - Hydrocephalus
  - Screw neck
- Poisoning
  - Diethylstilbestrol
  - Lead
  - Salt
  - Streptomycin
  - Sulfonamides (sulfaquinoxaline)
  - Warfarin
  - Wood Preservatives
- Vices
  - Tail Chewing
  - Fur Clipping
  - Aleutian Mink Disease
  - Dehydration
  - Grey Diarrhea
  - Heat Exhaustion

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The following is a discussion of some of the more common disease conditions a veterinarian would encounter in a mink herd.

DISTEMPER

Distemper is caused by the same virus responsible for distemper in the dog. Infections can be seen at any time of the year, but occur most often in the summer and early fall months. Kits are more susceptible than adults; the average death loss in nonvaccinated adults is 30 to 40 percent, but may go as high as 90 percent in nursing kittens. The infected mink transmit the disease to their kits or to other adults by direct contact or by coughing and sneezing. Although the virus can be simply destroyed in a few hours by heat, it can resist low temperatures (below freezing) for several days. Feed and feed dishes may become contaminated, and it is also thought that humans can carry the virus on their clothing for a considerable length of time.

The symptoms of distemper are quite distinctive. Nine to fourteen days after exposure the skin around the eyes, ears, nose and chin becomes reddened. The feet may become enlarged and tender, giving rise to the term “boxing glove feet”. Ocular and nasal discharges are also seen. Death usually occurs two to three weeks after the onset of symptoms. However, some animals may appear to recover, only to die 10 to 12 weeks later with a nervous syndrome the rancher calls the “screaming fits”.

Post mortem findings are not conclusive. Enlargement of the spleen may be seen, but it is not constant. Laboratory diagnosis can be made by finding distemper inclusion bodies in the trachea or urinary bladder.

When an outbreak has occurred in a nonvaccinated herd, all animals should be vaccinated as soon as possible with a modified live virus vaccine. Mink distemper vaccines are available from several biological houses. Distemperoid vaccine of ferret origin should never be used with mink, as the virus attenuated by ferret passage retains its pathogenicity for mink. Every effort should be made to keep down contact between infected and noninfected animals.

In a preventative program, breeding stock is vaccinated after pelting and before the breeding season. Kits should be vaccinated between 10 to 12 weeks of age. Some ranchers vaccinate only their breeding stock and not the kits, depending on maternal immunity to carry the kits until pelting in December.

Due to widespread immunization by mink ranchers, the incidence of distemper has declined steadily over the last 10 years. Outbreaks still occur, however, especially in nonvaccinated herds.

VIRUS ENTERITIS

Mink virus enteritis (MVE) is caused by a virus which is considered to be similar to, or the same as, the virus causing feline panleucopenia. The disease was first reported in the Fort Williams, Ontario area in 1947, and is sometimes known as Fort Williams Disease. MVE has been spreading steadily, and since carrier states probably exist, the shipment of breeding stock will eventually carry the virus to all mink producing areas of the world.

The most characteristic symptom is the appearance of casts or slugs in the drop-
pings. These casts may be greyish white to pinkish in color and are composed of fibrin, mucus, and epithelial debris. The feces may be very mucoid and blood streaks may be seen. The mink refuse to eat; they have a tendency to remain in the nest box; they appear dull and their eyes become squinted. Kits are the most susceptible, although adults are occasionally affected. Death usually occurs two to three days after symptoms are first seen. Mortality may run from 10 to 80 percent.

At necropsy, the most striking finding is a marked enteritis. The intestines may be ballooned, and a marked necrosis and desquamation of columnar epithelium of the intestinal mucosa exists. Many times a diagnosis can be made on the basis of symptoms and history alone. This can be confirmed by the demonstration of inclusion bodies in the columnar epithelium of the intestine. These inclusion bodies are acidophilic with H and E stain.

The virus is quite resistant to environmental conditions, and can exist on contaminated pens or nest boxes for at least a year. Thus, infection may reappear on a ranch a year or more after a previous outbreak.

When an outbreak occurs, vaccination of all animals on the ranch should be carried out immediately. Two types of vaccine are available, an autogenous product prepared from the viscera of infected mink, and feline panleucopenia vaccine. Along with vaccination, strict sanitation and quarantine measures should be instituted. All animals showing symptoms should be separated from the rest of the herd. Traffic on and off the ranch should be kept at a minimum; visitors and non-essential personnel should be excluded from the ranch. Every effort should be made to control the fly population. Utensils, pens, and equipment should be disinfected with a strong sanitizing agent.

In a preventative control program, all kits should be vaccinated during the early part of July. Since older animals are more resistant, opinions vary on the need of vaccinating the breeding stock. However, the chances of a severe outbreak in the kits will be lessened if the breeders are also vaccinated.

BOTULISM

Botulism is fairly uncommon among mink and from the standpoint of total losses, this disease is responsible for a relatively small percent. However, when botulism strikes on a ranch the effect can be devastating, often resulting in 100 percent mortality. The source of the botulism toxin (usually type C) is most often traced to some meat component of the ration. Two of the most common reasons for botulism outbreaks on mink ranches are including spoiled meat in the ration and meat from a sick animal. Since a mink ration contains a large percent of meat and poultry by-products, including intestines, the ration should never be allowed to warm up or stand for long periods of time before being fed.

Onset of symptoms is rapid (18 to 96 hours) and is characterized by muscular incoordination and stiffness. Paralysis usually develops first in the hind quarters and mink will be seen dragging themselves around by their front legs. It becomes increasingly difficult for the mink to breathe, as is evidenced by its heaving flanks. Shortly the mink lapses into a coma and dies.

In the case of an outbreak, polyvalent antitoxin should be given to the entire herd immediately. The sooner this is done the greater will be the number of mink that are saved. The dose is five to eight cc., I.P., depending on the size of the animal. It is very important that all of the food suspected of containing the botulism toxin be removed from the pens and feedboards and discarded.

Most mink ranchers now vaccinate against botulism. The toxoid can be given to unvaccinated adults in January or February and to the kits in June or July. One injection will impart a lifetime immunity, so that no booster is needed.

In summary, there are three diseases for which a mink rancher will commonly vaccinate: distemper, mink virus enteritis, and botulism. In each case the adults or breeding stock are immunized between pelting and the breeding season (i.e., January or February) and the kits in late June or early July. It has been found that distemper vaccine and botulism toxoid in-
jections may be given simultaneously. Separate syringes should be used and the injections given at different body sites.

CHASTEK'S PARALYSIS

In the early 1930's, a new disease of foxes was discovered on the fur farm of J. S. Chastek at Glencoe, Minnesota. It was found that the paralysis and death occurring in his foxes was caused by the raw fish in the ration. The fish contained the enzyme thiaminase, which destroys thiamine (B1) in the body. Therefore, when fox or mink are fed thiaminase daily, their reserves of thiamine are quickly exhausted and they develop a paralysis and die. The course and symptoms are characteristic. There is a loss of appetite and weight, the gait becomes unsteady, and paralysis and convulsions are seen shortly before death.

The condition may be prevented by cooking the fish used in the diet, which will destroy thiaminase; or if raw fish is fed, it should be added to the ration only on alternate days. When an outbreak occurs, intraperitoneal injections of five to ten milligrams of thiamine should be used and ample amounts of Brewer's yeast should be included in the ration. If uncooked fish is being fed, it should be removed from the ration immediately.

NURSING SICKNESS

Nursing sickness, sometimes referred to as nursing anemia, is a common condition in lactating females. It is characterized by a marked loss of flesh, progressive weakness and a high mortality.

Apparently, nursing sickness is caused by a salt depletion in the nursing female. Since lactating female mink lose a great deal of salt in their milk, the condition occurs most often during the fourth and fifth week of lactation, which is the period of greatest milk flow. The only constant finding on post mortem is a complete absence of body fat. In many instances there is evidence of degenerative changes in the liver. The inclusion of salt in the diet at a level of 1/2 of one percent appears sufficient to prevent nursing sickness. After the condition has advanced to a certain point, treatment seems to be of no avail. Careful observation of nursing females will indicate those which are getting thin and special attention can be given both the mother and her litter. It is well to encourage the kits to eat and drink water as early as possible in order to reduce the heavy demands on the mother's system.

STEATITIS (Yellow-fat disease)

This is a seasonally occurring condition characterized by a gross discoloration (yellow fat) and steatitis of adipose tissue. It affects only kits and is seen in the summer and fall. Steatitis appears when rancid fat is fed in the ration. The rancid fat increases the demand for antioxidants, and if they are deficient or lacking, disease results. Some affected kits may appear perfectly normal until death occurs. Others may refuse to eat, develop an unsteady gait, posterior paralysis, become comatose and ultimately die. Mortality may reach 50 percent and survivors never again achieve full health.

At necropsy, the skin of the abdomen and inguinal region appears thickened and feels doughy. When the affected tissues are incised, a thin, watery fluid escapes. The subcutaneous and visceral fat is a characteristic brownish-yellow in color and the spleen is often enlarged and mottled.

Vitamin E is used as a preventative measure and also for treatment after the condition has occurred. A synthetic stabilized tocopherol powder added to the diet is satisfactory as a control measure. If the diet contains 15 to 20 percent cereal, four to five units of vitamin E per mink per day should be added. When an outbreak occurs, the tocopherol preparation should be added to the ration so that each kit on the ranch receives 20 units per day for two weeks. The rancher should remove storage fish and other stored meat products from the ration and substitute fresh unfrozen feeds. Affected kits can be given an injection of 15 to 20 units of vitamin E per day for three days.

URINARY CALCULI

In mink, urolithiasis occurs during two distinct periods of the year. Adult females die with urinary calculi during the spring months, often during parturition. The cal-
culi found in these cases are usually quite large in size. The second period occurs in late summer when male kits die due to a toxemia resulting from obstruction of the urethra by small calculi and/or fibrinous material associated with hemorrhaging, cystitis and pyelonephritis. The adult female may die without showing any symptoms. However, symptoms associated with calculi are dysuria, a straddling gait, wet fur between the mink’s legs and paralysis.

Etiology is unknown but the disease is probably a result of both nutritional factors and infection in the animals. Measures for the control of stones consist of the addition of either ammonium chloride or phosphoric acid to the diet. The basis for including these chemicals in the ration is to keep the pH of the urine on the acid side, since the crystalloid stones precipitate out when the urine is in the alkaline range.

Recommendations for the use of ammonium chloride are to feed it at the rate of one gram per mink per day (one pound for each 450 mink) from April 1 until June 15. It should first be dissolved in water and then added to the mixed feed. Recent research indicates that phosphoric acid may have advantages over ammonium chloride for the control of urinary calculi. Phosphoric acid is more palatable to the mink than ammonium chloride and also has a preservative action on the feed. Phosphoric acid should be fed at an 0.8 percent level of the ration. The correct amount of phosphoric acid should be weighed out and mixed with water before being added to the feed. The recommended periods of use are March 1 to June 15 and from July 15 until pelting. Care should be exercised in handling the acid as it is corrosive and may burn, causing injury to the rancher’s person or clothing.

When severe losses in young males are seen in the late summer, infections of the urinary tract should be suspected. Calculi frequently accompany these infections. The affected kits have difficulty micturating, become weak and succumb to infection and/or uremia. At necropsy, the most significant finding is a greatly enlarged urinary bladder filled with urine containing blood and purulent material. *Staphylococcus aureus* and/or *Proteus* are isolated when the exudate is cultured.

Recently, Lauerman reported that mink fed diethylstilbestrol (commonly found in rations containing chicken scrap) produced a squamous metaplasia, cornification and sloughing of urethral and prostatic epithelium. These changes permit bacteria found in the prepuce of normal mink to ascend the urethra, causing a cystitis and progressive destruction of the kidneys. If *Proteus* is involved, the enzyme urease, produced by the bacteria, splits urea in the urine forming ammonia. Calculi are readily formed in this alkaline medium. When *Staphylococcus aureus* is involved, fibrin plugs are formed which are just as effective as calculi in obstructing the flow of urine. Currently, the best treatment seems to be nitrofurantoin in the diet given at the rate of 15 milligrams per mink per day for four days. Care should be taken that mink have sufficient water available.

**URINARY INCONTINENCE**

Urinary incontinence, also known as acidosis or “wet belly”, causes extensive damage to many male pelts and greatly decreases their value. The constant loss of urine by the mink results in staining of the fur and necrosis of the skin and subcutaneous tissues in the vicinity of the external urethral office. The cause of this condition is unknown and treatment is empirical. Among the procedures that are currently recommended are increasing the carbohydrates and lowering the fat level of the ration, adding more liver, and making certain the ration is not rancid.

**POISONS**

*Diethylstilbestrol.* One of the major problems of mink ranchers over the past decade has been sterility of mink caused by consumption of stilbestrol in the ration. The most common source of the drug is the necks of roosters which have been ca­ponized by placing a pellet in their neck. New regulations banning the use of stil­bestrol in chickens should ease this problem considerably. It has been found that the intake of 10 micrograms of stilbestrol
per day will cause sterility in mink. The mink will breed normally but no kits will be produced. In order to minimize the danger of sterility, most mink ranchers adopt a feeding schedule in which poultry by-products are fed only after whelping in May and are discontinued in December.

**Lead.** Mink are very susceptible to lead poisoning. Products containing lead should never be used as preservatives on nest boxes or any other equipment that mink might chew. No characteristic symptoms are seen with lead poisoning. The animals lose weight gradually and die in 25 to 40 days. Since there are also no characteristic post mortem lesions, a diagnosis must be made on the basis of a history of equipment being treated with lead. When a diagnosis is made, all mink should be removed from treated pens as soon as possible. Care should be taken that the survivors receive adequate amounts of calcium and vitamin D.

**Streptomycin.** The antibiotic, streptomycin, is toxic for mink and should be used with extreme caution. In combination with penicillin it is even more toxic. Adults should never receive a dose of more than 50 mg. Following an overdose, symptoms occur 5 to 30 minutes after injection. Labored breathing, followed by loss of muscular control, coma and death are typical symptoms.

**Sulfonamides.** Occasionally, sulfaquinocxaline has been given to mink for coccidiosis or other enteric infections. Very often the administration of this drug results in a loss of the normal blood clotting mechanism with resultant spontaneous hemorrhaging. The abdominal cavity of the mink will be found full of unclotted blood. Sulfaquinocxaline is always contra-indicated in the treatment of mink diseases.

**ALEUTIAN DISEASE**

With the development of the Aleutian color phase, a disease was noticed that seemed to be confined to mink homozygous for the Aleutian gene. The disease is characterized by a progressive weight loss, without anorexia, until death occurs. The affected mink are extremely thirsty and in some cases hemorrhaging occurs around the mouth. Since the mink are often anemic, mucous membranes appear pale and blood clotting may be delayed. Mortality of affected animals often approaches 100 per cent. At necropsy, the most significant findings are kidney lesions. Depending on how far the disease has progressed, the kidneys appear enlarged and reddened, with some hemorrhaging, or in the later stages, shrunken and pitted. At the present time, all attempts to isolate an etiological agent of the disease have failed. The medical profession is interested in the disease because it bears many similarities to aplastic anemia in humans. All treatment is symptomatic. Since there is apparently some genetic relationship to the Aleutian gene, outbreeding with standard dark mink has provided some measure of control.

**GREY DIARRHEA**

Over the past few years there has been an increase in a condition known as grey diarrhea or "putty droppings". Grey diarrhea is characterized by the animal having a ravenous appetite but also progressively losing weight. The affected animals produce a large volume of soft greyish droppings which often have a rancid fat odor. It appears that very little of the fat in the diet is assimilated but simply passes through the digestive tract and out in the stool. Often large fat droplets can be observed in the stool.

The cause of this condition is unknown. No bacterial or viral agent has been incriminated and no evidence of a parasitic infection has been uncovered. There is increasing evidence to indicate that the disease, or at least a predisposition to the disease, may be inherited.

Many forms of treatment have been tried, none of which have met with much success. The use of water soluble furacin in the ration seems to be of some value in keeping down the incidence. In view of the evidence that suggests heredity may play a role, care should be taken to select breeders that have no family history of this condition. This has been a rather brief description of some of the more important diseases that affect mink. There are several sources where more detailed information...
Stools of grey diarrhea may be obtained. One excellent reference is the Fur Farm Guide Book of the American Fur Breeder. This is issued annually and covers not only the diseases of mink, but all other aspects of fur farming as well. A good source for the latest information on diseases is the Progress Reports of the Mink Farmers Research Foundation. Through grants to various universities and research stations this foundation supports work on the important disease and nutritional problems confronting mink ranchers.

REFERENCES
The Mink Farmers Research Foundation, Progress Reports. September, 1959.

Annual Conference
The Annual Conference for Veterinarians will be held June 19 and 20 at the Iowa State University Memorial Union.