

MINK DISEASES

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MINK DISEASES

ANIMAL PATHOLOGY DIVISION
HEALTH OF ANIMALS BRANCH
CANADA DEPARTMENT OF AGRICULTURE

This publication has been prepared as a reference for mink ranchers, veterinarians and others concerned with the mink industry. Detailed information is given on the serious and most common diseases, with shorter descriptions of other conditions.

The booklet should not be considered a replacement for the advice of a veterinarian. Because some diseases resemble others differing in cause and severity, correct diagnosis is the key to successful treatment. Therefore, working cooperatively with your veterinarian yields the greatest chance of success in identifying and solving disease problems.

PREVENTION OF DISEASE

The greatest possible return, as far as disease is concerned, is from preventive measures. This is particularly true for mink, as few other classes of livestock are housed under such unnatural conditions or in such concentrated numbers. Lack of attention to sound husbandry and principles of sanitation can suddenly erupt in a serious situation that might easily have been averted. Economically, the cost of prevention is usually only a fraction of the cost of cure.

Every ranch should be surrounded by a suitable fence to prevent the entry of outsiders, both human and animal. Dogs, cats and wildlife can carry diseases transmissible to mink. Visitors may carry infectious organisms on their boots, clothing or persons, and should not be allowed to enter until their disease-carrying potential is known.

SANITATION

Sanitation is necessary to reduce environmental contamination in general, as well as to decrease the spread of specific diseases. Always burn waste feed and carcasses or bury them deeply. Remove manure often, especially in the summer, and spread lime over it periodically between removals.

Clean the feeding area regularly (daily during hot weather) to help avoid feed spoilage. Use a wire brush to remove uneaten feed and wash the area with a disinfectant. Use a torch or flame thrower to burn fur from wire, and a high-pressure water cleaner (minimum 500 psi) to remove dirt from other surfaces and cracks in pens and nest boxes. Steam also is good, but it may be hard to handle and must be used very close to the surface. Once areas are clean, apply a disinfectant.

There are a number of disinfectants available, with varying characteristics and active principles. In general, their effectiveness is limited by the sensitivity of the organism to the particular disinfectant, as well as the time and conditions under which the two are in contact. Under ranch conditions, the activity of many preparations is greatly reduced by the presence of fecal material or other organic matter. The ideal disinfectant is nontoxic, has a high resistance to organic matter (dirt), and has wide killing power for both bacteria and viruses.

FEED HANDLING

Mink are extremely susceptible to food poisonings and every effort must be made to avoid ration ingredients that may carry such organisms or their toxins. Do not feed carcasses of animals dead of septicemic (infectious) disease or of which the health status is unknown; avoid meat from animals sent for salvage; and never feed carcasses of mink, even from known accidental deaths. It is wise to follow the old axiom, "If in doubt, throw it out!"

Store all perishable feeds in clean, properly operating freezers or refrigerators; and do not refreeze any feeds that have been frozen and thawed. Keep refrigeration equipment in good repair to avoid breakdowns that may result in feed spoilage. Ranchers who rely on commercial storage facilities or ready-mix feed supply should make sure that these storages are cleaned regularly and that equipment is functioning properly.

Wash grinders and mixers daily, using boiling water, and do not let old feed accumulate in out-of-the-way parts of the equipment. In addition, remove uneaten portions of feed from

pens and nest boxes before adding fresh feed and flush out drinking cups (if used) with fresh water.

RODENTS

Rats and mice can carry a number of diseases transmissible to mink, and may foul feed and water with their urine and droppings. Control measures are essential on every ranch. Make sure that poisons are placed where the mink can't get at them.

FLIES

Flies can transmit disease organisms picked up from human and animal excrement, carcasses of diseased or dead animals, manure piles or other filth on which they land. Germs are carried both on the outside of the body and within the digestive tract of flies.

Under ideal conditions, flies complete their life cycle in about 3 weeks. Since their development is closely related to poor sanitation, the most effective control measure is regular removal and proper disposition of manure, garbage, sewage, dead animals and other litter. Intermittent dusting with insecticide powders or lime helps control larval development between removals.

To avoid open cesspools, which attract flies, provide the ranch with a good drainage system. Install screens on windows and doors of feed houses to keep flies out, and dispose of waste feed and animal carcasses in such a way that they do not provide a medium for development of flies. For current recommendations on chemical control of flies, contact your local agricultural or public health representative. If chemicals are used, make sure they do not reach the mink or contaminate feed or water.

VACCINATION

The diseases vaccinated against are distemper, virus enteritis and botulism. Always follow the instructions provided by the vaccine manufacturer, as suggested times and dosages vary. Use proper-sized needles and change them often. Sterilize vaccination equipment by boiling it for 20 minutes and use it only for vaccinating, not for killing at pelting.

Recently, a new method for distemper immunization has been introduced which involves spraying of kits in nest boxes. It seems to be effective in immunizing against distemper and certainly is convenient to use.

TREATING SICK MINK

Usually, for examination and treatment, mink need only to be caught by hand and held by the neck and hind legs or hips. For procedures where this form of restraint may not be adequate, or may interfere with the treatment to be performed, a catching cage is useful.

To hold a mink's mouth open, insert a block of wood or a pair of pliers; to hold it shut, use a special 'split key ring,' which is placed over the muzzle and held in place by a pin that passes through the mouth behind the canine teeth.

Local, general and topical anesthetics can all be used for mink. As tolerances with general anesthetics are somewhat narrow, ranchers should rely on professional assistance for procedures requiring these drugs.

Sick mink can be force-fed by passing a small, pliable rubber tube through the esophagus to the stomach. This tube must not be introduced by mistake into the trachea, as death will quickly follow the passage of any material through it into the lungs.

ADMINISTERING MEDICINES

In administering medicines to mink, the condition of the animal, the drug involved, and the purpose for which it is intended must all be considered. The following methods are used:

Orally

If the mink is eating, obviously medicine is most easily administered by mixing it with the food. Otherwise, use a spoon or eyedropper for liquid medicine, and place powdered medicine on the tongue or blow it into the mouth through a straw. Pulling the lips away from the teeth along the side of the mouth forms a pouch into which you can place fluids. Give medicines slowly, to allow the mink to swallow naturally between instillations.

Rectally

Rectal instillation is an excellent way to administer fluids to support a sick mink. Glucose solution and nutrient broth are often given in this way and are absorbed quickly from the rectum. Some medicines are given rectally, also, especially in capsules, but professional guidance is needed here.

By Injection

Subcutaneously (under skin) — Biological fluids, including

vaccines, vitamins and sera, are usually administered by injecting under loose skin beneath the front leg. A less desirable site is inside the hind leg.

Intramuscularly (into muscles) — Most antibiotics are intended for intramuscular injection. The site of choice is the thick muscles on the back surface of the hind legs.

Intraperitoneally (into abdomen) — Some fluids, including saline solution, are safe to inject directly into the abdominal cavity through the body wall, but only with professional guidance.

EUTHANASIA

This is the technical term applied to the process of bringing about easy, painless death. As applied to mink ranching, such methods should be followed at pelting time, to ensure that the humane aspects of animal care are considered at all times.

Some methods of killing mink, for example, neck breaking, cyanide gas or carbon monoxide, require special techniques that must be learned by apprenticeship. Injectable killing fluids are dangerous in the hands of a careless operator, but aside from that are the most reliable means of killing mink. Some ranchers use a mixture of one part nicotine sulphate (Black Leaf 40) to four parts of wood alcohol. One millilitre of this mixture injected hypodermically into the chest cavity kills quickly; if into the heart, it kills immediately. When using gases or killing fluids, make sure that skinning is done in a well-ventilated area. A powerful exhaust fan installed above the skinning bench, with air intakes on the opposite wall, will remove fumes and odors immediately.

A variety of contraptions have been assembled by ranchers to enable electrocution of mink. However, some of these are hazardous to the operator. Portable commercial equipment developed and manufactured in Norway is available to ranchers in Canada and is recommended as an alternative to other methods. This equipment utilizes battery current to stun mink safely and allows ample time for manual neck breaking. It is humane for the animal and safe for the operator. Also, it is inexpensive, highly efficient and causes no damage to the fur.

SUBMISSION OF SPECIMENS

Ranchers should work closely with local veterinarians, as well as with regional diagnostic laboratories. When submitting

specimens to one or the other for diagnosis and recommendations, keep the following in mind:

- Submit samples early in a problem and after discussion by phone.
- Do not freeze specimens, but keep them cool and ship as soon as possible.
- Deliver samples personally or use the fastest method of transporting them. In general, rail and bus lines are satisfactory for well-wrapped, leak-proof parcels containing ice packs.
- Time your shipment so that it does not arrive on a weekend, unless this has been prearranged.
- Include a complete history — owner's name, address, telephone number, number of mink, colors, number sick, number dead, clinical signs, distribution and length of illness, and recent diet changes.

Usually, dead mink are submitted, although sometimes other samples, such as live mink and feed, are requested. Where a certain disease is suspected, ask for professional assistance in selecting the best material to confirm the diagnosis.

DISEASES CAUSED BY BACTERIA

Many diseases caused by bacteria affect mink. The more common or serious ones are discussed here.

BANG'S DISEASE (BRUCELLOSIS)

This disease is caused by *Brucella abortus*. Symptoms include abortions, small litters and death of very young kits. Visible lesions are not present. Diagnosis is established by postmortem examination (necropsy), laboratory analysis of aborted fetuses and placentas, and serum tests on aborting females. Serum tests on other mink may identify positive animals for elimination. Submit aborted fetuses and placentas for laboratory examination.

No treatment is suggested, but most of the infection can be eliminated from the ranch by pelting all adults. To prevent brucellosis, avoid feeding contaminated beef by-products to mink and never feed them aborted bovine fetuses, as these are the most likely source of infection.

CLOSTRIDIAL INFECTIONS

Blackleg

Clostridium chauvoei infection in mink kits can cause severe losses. The disease usually stems from feeding carcasses of animals, especially cattle, that have died of blackleg. The result is heavy contamination of feed and equipment with these bacteria. Most kits are found dead without any warning. Postmortem examination reveals gas and fluid in swollen tissues of the hind legs. Laboratory diagnosis is made by isolation and identification of the bacteria from mink tissues and feed. Submit feed samples and dead mink to the laboratory for diagnosis.

No treatment is suggested. To prevent blackleg, do not feed carcasses from diseased animals; and always observe sanitary practices in the preparation and storage of feed.

Botulism

Botulism is always a ranch hazard because of possible contamination of meat and fish. Although total losses are not great, losses on individual ranches may be high. The cause is usually type C toxin produced by *Clostridium botulinum* when this species multiplies in unfrozen food. Mink are susceptible to extremely low toxin levels, and even more susceptible to a second dose within a few days or weeks. The first symptom is paralysis of the hind legs, then there is abdominal breathing, followed by a short period of coma and death. Onset is rapid, with death in from 3 to 24 hours of first symptoms. Diagnosis is aided by typical clinical findings. In laboratory examination, characteristic lesions are not found. Laboratory procedures include neutralization tests in which feed extracts are injected into mice. With low toxin levels, a mink feeding trial is necessary to confirm the diagnosis. Toxin may also be unevenly distributed in feed. Submit feed samples, including individual ingredients, and specimens of sick and recently dead mink.

Treatment is to inject 5 to 8 ml of polyvalent antitoxin into the abdominal cavity of every mink as soon as possible, certainly within 12 hours of onset of symptoms. Immunization against the disease, using polyvalent toxoid, is effective and is strongly recommended.

Enterotoxemia

Acute hemorrhagic enteritis has been described in mink in which *Clostridium perfringens* Type C has been isolated. Chicken waste being fed contained the same bacteria. Various

treatments were not successful. The disease could not be reproduced, so additional research is required.

DIARRHEA

Diarrhea is nonspecific, but is usually due to inflammation of the gastrointestinal tract and is often related to spoiled food. Feces are yellow in young kits. On postmortem examination, there is intestinal inflammation and general dehydration. Laboratory tests on intestinal contents and organs may aid in selecting antibiotics for treatment. For control, follow sanitary feed handling practices described earlier, including the cleaning of grinders and mixers.

Gray diarrhea, profuse and foul, is seen in older mink that gradually lose weight while eating well (malabsorption syndrome). In one study, persistent focal stomach inflammation was the only significant finding. Treatment is unlikely to be of value, although nitrofurazone has been found helpful in some cases.

MASTITIS

This infection of the mammary gland of nursing females is caused by several bacteria, including *Streptococcus* spp., *Staphylococcus* spp. and *E. coli*. Infection may occur following gland injury by nursing kits or sharp bedding material. Widespread infection may originate from the feeding of infected carcasses. Symptoms usually show up when the kits are 3 to 4 weeks old. There is marked swelling and inflammation of mammary glands, with the female refusing to nurse kits and showing loss of appetite. Most of the litter may die of septicemia (blood poisoning). For diagnosis, submit dead females and kits, and feed samples.

Treatment is to administer antibiotics and eliminate infection sources. Mark recovered females for pelting. To prevent mastitis, take care to avoid injury to mammary glands; and do not feed carcasses of infected animals to mink.

PLUM BLADDER DISEASE (CYSTITIS, PYELONEPHRITIS, URINARY CALCULI)

This disease affects the urinary system, mainly of pregnant females and male kits. Bacterial infection gains entrance through the external urinary passage, moves up the urethra to the bladder, and often on through the ureters to the kidneys. The action of the bacteria (usually *Staphylococcus aureus* or *Proteus* spp.) causes alkaline (basic) urine, which precipitates the minerals normally found in urine, forming calculi (stones).

Debris is at the stone center, with bacteria in all layers of the matrix. Most calculi are composed of magnesium ammonium phosphate. Symptoms are few, although sometimes bloody urine is passed. Male kits are usually found dead in early summer. Postmortem examination reveals a distended bladder containing bloody pus and often calculi (Figure 1); the urine passageway is usually blocked; and the kidneys contain pus and are often mottled. Diagnosis is by laboratory tests and examination of carcasses.

Outbreaks of plum bladder disease may be treated with soluble sodium sulfathiazole or novobiocin if the bacteria are sensitive. To help prevent the condition, provide good sanitation to reduce environmental contamination; increase the water supply; cull out families in which the condition is seen; and add feed grade phosphoric acid (0.6% before water) to the diet from March 1 to mid-June and from mid-July until pelting.

HEMORRHAGIC PNEUMONIA

Hemorrhagic pneumonia occasionally is seen, almost exclusively in kits, as an explosive outbreak in late summer or fall. It is usually caused by *Pseudomonas aeruginosa*, from contaminated feed or water, and often accompanies stress from cold, dampness, rapid temperature changes or furring. Symptoms include blood around the nose and mouth, difficult breathing and death within hours of the first sign. Spread is from kit to kit once ranch infection is established. Some mink strains seem to be more susceptible than others in any given outbreak. At necropsy, the lungs are solid, dark red and blood-filled (Figure 2). Laboratory tests are necessary to confirm the diagnosis, by isolation of the bacteria from tissues. The organism can be recovered from most of the organs of the animal.

For treatment, antibiotics are usually of little value, although oral sulfonamides (buffered with equal amounts of baking soda) have helped in some outbreaks. Autogenous bacterins have also been reported to stop outbreaks. To keep the disease from spreading, cull or dispose of all mink in the immediate area of the ranch as soon as possible. Clean and disinfect all sheds and wire; quaternary ammonium products are not thought to be effective against these bacteria. To prevent hemorrhagic pneumonia, apply good sanitation practices, especially relating to feed and water, and try to avoid stress in young mink.

SALMONELLOSIS

This infection, mainly of the intestine, is caused by *Salmonella* spp. It can be carried by healthy mink and precipitated by stress. Infection is introduced by contaminated feed, especially chicken offal. Diarrhea and abortion are occasionally noted in pregnant females. Diagnosis is by postmortem examination and laboratory tests on intestinal contents and mesenteric lymph nodes. Antibiotic treatment may be of value.

SEPTICEMIA (BLOOD POISONING)

Blood poisoning due to beta hemolytic streptococci strikes young kits at 5 to 6 weeks of age. Although there may be preliminary lethargy, refusal to eat and general weakness, usually the first warning is the sudden death of some of the kits. The disease may spread from kit to kit, so involved litters should be separated. Diagnosis is by laboratory tests and examination of dead kits. To control the infection, use an appropriate antibiotic and improve the sanitation.

SKIN INFECTIONS (DERMATITIS)

Usually due to *Staphylococcus aureus*, these infections are seen occasionally. They often start with small wounds. The lesions appear as raised, smooth lumps, often with a crusty exudate and with some containing thick, yellow pus. Diagnosis is made from these findings combined with laboratory tests. For treatment, clean the lesions and use a suitable antibiotic.

Boils are somewhat similar, but are most often seen in mink with the Aleutian factor because of their reduced infection-fighting ability. The infection usually follows an injury caused by a bone splinter or barbed grass. The boil appears as a swelling (usually on the head) that may develop to considerable size and may rupture if not attended to. For treatment, clip the area around the boil; lance, drain, and flush the boil with a mild disinfectant such as hydrogen peroxide; and apply a suitable antibiotic. In herd problems, look for and eliminate the source of foreign bodies.

TUBERCULOSIS

This is a rare infection, mainly of the lungs and liver, due to *Mycobacterium tuberculosis*. The more-common avian type is usually restricted to mink carrying the Aleutian factor; but the rarer, more-severe bovine type may also be seen in other mink. The usual source is infected poultry (layers) or beef in the feed.

During the incubation period of the disease (3 to 9 months), infected mink are in poor condition and gradually lose weight. Lesions include white foci in the lungs, liver and intestinal lymph nodes (Figure 3). Laboratory examination and tests are necessary to confirm the diagnosis. There is no treatment. To prevent the disease, do not feed carcasses of infected animals to mink.

DISEASES CAUSED BY VIRUSES

ALEUTIAN DISEASE (PLASMACYTOSIS)

Aleutian disease (AD) was first recognized in 1956 in homozygous Aleutian mink. It is caused by a heat-resistant virus, which is readily inactivated by formalin and ultraviolet light. All color types of mink are susceptible, but Aleutians, being generally less robust, may succumb more quickly. Kits from infected females are usually infected. Although the virus can cross the placenta and infect fetuses, the usual mode of spread is through excretion of virus in urine, feces and saliva of chronically infected mink.

Symptoms

Affected mink lose weight, are listless and seem to be thirsty all the time. A common symptom is increased water consumption. The appetite is moderately good at first, but decreases as the condition advances. Now and then food goes right through and appears in the feces, which also may be black and tarry from intestinal bleeding. Often, shallow ulcers develop on the gums, sometimes with bleeding and loose teeth. In the late stages of the disease, mink are emaciated and some may show nervous symptoms, such as rolling or convulsions. Aleutian disease is usually fatal, after symptoms lasting from 2 weeks to several months, but it may be nonprogressive and last several years. Stress, such as cold weather, can speed up the normal course of the disease. Occasionally, the first indication of trouble in an affected mink is sudden death from a massive internal hemorrhage due to its spleen rupturing when the mink is caught.

Diagnosis

A common finding in mink infected with the virus of Aleutian disease is a tremendous increase in the gamma globulin fraction of the serum. This increase, with resulting disturbance of the normal serum albumin/globulin ratio, has made it possible to use the iodine agglutination test (IAT) as an

aid in diagnosing AD. A small amount of blood is obtained by clipping a toenail sufficiently close to cause bleeding. The sample is drawn into a fine glass tube, one end of the tube is sealed and the sample is spun in a centrifuge to separate serum from cells. The tube is broken to obtain the serum fraction, which is expelled onto a glass plate. An equal volume of a special iodine reagent is added and the mixture is stirred. Agglutination (or clumping), which occurs in a short time, indicates a positive test.

Although the IAT is useful as a ranch test, it is nonspecific and should not be relied on solely for diagnosing AD. In general, the test indicates impaired liver or kidney function and any condition producing this can give a positive reaction. Another limitation is that infected mink may give negative reactions in both early and terminal stages of AD. Positive reactions should always be confirmed by laboratory examination before a diagnosis of Aleutian disease is made. In mink dead from AD, the spleen is tremendously enlarged, dark, engorged with blood and easily ruptured (Figure 4). Early in the clinical stage, the kidneys are enlarged, orange-yellow in color and speckled with tiny hemorrhages, but later they are shrunken and studded with whitish blotches. Microscopically, the most characteristic changes include large numbers of plasma cells in kidneys, liver, spleen, and lymph nodes; hyaline (translucent tissue) changes in certain areas in the kidneys; proliferation of bile ducts in the liver; and collections of cells and hyaline material in arterial walls.

Recently, a counterimmunoelectrophoresis (CEP) test has been developed, which can identify infected mink very early in the course of AD. For this laboratory test, a blood sample is obtained as for the IAT, to provide a portion of serum. The serum is subjected to an agar-gel-diffusion procedure, using an antigen extracted from the tissues of AD-infected mink. In the test, antibodies in the serum of infected mink react with the antigen to form visible lines in the agar; no such lines are produced with serum from negative mink. With the CEP test, animals infected as recently as 7 days have been recognized; and the test is regarded as being very specific, whereas the IAT is known to be nonspecific. On the other hand, the new procedure cannot be conducted by ranchers on their own premises, and there is still limited experience with it. In the meantime, the IAT can still be useful in attempting to reduce infection on ranches where the presence of AD has been confirmed.

The following series of tests is suggested:

- Test potential breeders in the late fall after grading, but before pelting, and eliminate positive animals when prime. Stop testing several days before pelting to avoid soiling the pelts.
- Retest just before breeding, to detect and remove animals that have become positive since the first test.
- ‘Misses’ may be tested after whelping, to determine if AD was a factor in the failure of some females to reproduce.
- Test potential additions before allowing them to leave the ranch of origin.

Do not buy any mink reacting positively to this test, even if the reaction is due to something other than AD.

Treatment and Prevention

No treatment is available for Aleutian disease and there is no vaccine to prevent it. All you can do to eliminate the disease is to isolate infected mink, take sanitary precautions to minimize spread and pelt all infected stock.

DISTEMPER

This highly contagious disease affects mink of all ages, although it is most common in nonimmunized kits. In the absence of any immunity, the virus can spread quickly and cause high mortality. Since the same virus also produces canine distemper, infection can originate with any nonimmunized dog or with certain species of wildlife, including raccoons, foxes, weasels, ferrets and skunks.

The virus is spread by coughing and sneezing, or indirectly by contaminated equipment, clothing and catching mitts. Although it is inactivated by heat (about 40°C) and sunlight, freezing temperatures allow the virus to remain infective for months, especially in the form of discharges dried on equipment. Useful disinfectants include 1% formaldehyde, 1% lysol and tamed iodines.

Symptoms

The first symptom — most often watery eyes — usually appears about 2 weeks after exposure to the virus, but the interval can be longer. The condition becomes progressively worse, until the eyelids are encrusted and stuck together and



Fig. 5a. Distemper — swelling and encrustation of footpads.

there is a crusty nasal discharge adhering to nostril openings. Sometimes the nose and footpads are swollen and covered with thick, dried crusts (Figures 5 and 5a). Other areas, such as the belly, often show thickening of the skin along with crusting and loss of hair; and the coat is unkempt, dirty and stained by urine and feces.

Some mink may have a nervous form of the disease and not show any of above symptoms. In these cases, the virus invades the brain and causes convulsive seizures known to ranchers as "screaming fits." Although some mink recover, most die within 2 weeks of their first seizure.

Diagnosis

The presence of a highly contagious disease causing heavy mortality and producing nasal and ocular discharge, with swollen and crusted feet and convulsions in some, suggests distemper. This diagnosis can be confirmed by microscopic examination and the finding of characteristic inclusion bodies in the bladder and trachea. Where further confirmation is required, the causative virus can be isolated and identified in the laboratory. It should be borne in mind that outbreaks can

occur on premises where partial immunity can alter the clinical picture from that described.

Treatment and Prevention

Treatment of individual infected mink is not usually effective. The use of antisera, antibiotics or other reagents is not recommended, since they usually do little except prolong the period during which affected mink serve as a source of virus on a ranch. To avoid further spread of the disease, dispose of affected mink; soak and thoroughly clean all contaminated cages, nest boxes and other equipment in a 2% solution of lye, a 1% solution of formaldehyde or tamed iodines; and store disinfected equipment for a period in a sunny location.

Vaccination — When a diagnosis of distemper has been confirmed, it is important to move quickly to protect as many mink as possible by vaccination. Isolate the part of the ranch on which the disease appears as well as you can and, beginning away from this infected area, vaccinate all mink. Try to keep persons caring for unaffected mink away from the contaminated area, and do not move mitts or any equipment from one area to the other. When the same people must work in both areas, make sure that they change outer clothing and footwear.

Effective vaccines to prevent distemper include injectable and spray preparations. To provide adequate protection, vaccinate all kits at about 10 weeks of age; and revaccinate breeders when selecting them at pelting time or in the winter before breeding. During their first few weeks of life, kits born to an immunized mother may be protected by antibodies in the mother's milk, but the presence of these antibodies may interfere with the ability of a vaccine to confer immunity in the kits. This is why vaccination is delayed until kits are about 10 weeks old.

ENCEPHALOPATHY (SCRAPIE-LIKE DISEASE)

Encephalopathy is a rare fatal disease caused by a virus similar to the one that causes scrapie in sheep. The virus attacks the central nervous system and is extremely resistant. It withstands boiling for 15 minutes, is not inactivated by ultraviolet light and is resistant to 10% formalin. Another similarity to scrapie is the very long incubation period between exposure to the virus and development of symptoms. In natural infection, the virus is ingested in feed and clinical symptoms appear some 8 months later. The disease, therefore, is never seen in kits.

Symptoms

The disease starts gradually, with slight changes in the normal habits that are hardly noticed. However, these changes become more apparent and soon a normally clean mink is leaving feces in the nest box and scattering them over the wire, rather than depositing them in a single area. As the disease progresses, the mink neglects her kits, becomes noticeably excited and shows some incoordination. Often, a jerky motion of the hind legs is seen and the tail is curled up like that of a squirrel. Central nervous system involvement is shown by circling, biting their own tails and uncoordinated leg movements. In the advanced stages of the disease, this hyperactive state is replaced by one of somnolence; the mink becomes inactive and lies for long periods without moving, often pressing its nose into a corner. If disturbed, it may rouse briefly, but returns to its former state when left alone. If handled, it may bite the catching mitt and retain its grip indefinitely if the mitt is taken off and left with it. Death invariably results, usually within a month of first symptoms. When the mink is found dead, its teeth are often fixed to the wire.

Diagnosis

Diagnosis is based on the symptoms described. It must be confirmed on necropsy by microscopic examination of the brain, which shows a typical degeneration of brain cells.

Treatment and Prevention

There is no treatment or vaccine available for encephalopathy. Since the natural history of the disease is not yet fully understood, little can be said concerning its prevention. It seems certain that it is a food-borne infection, so affected mink should never be allowed to contaminate feed. Some authorities, because of this disease's resemblance to scrapie, caution against including sheep meat in mink rations.

VIRUS ENTERITIS

This highly contagious disease causes high mortality in kits, which are its main victims. The virus that produces mink enteritis is closely related to the one causing panleucopenia (infectious enteritis or distemper) of cats. It can infect all the cat family, including lions and tigers, as well as raccoons and ferrets. The disease is characterized by severe inflammation of the intestines.

Although the virus is readily inactivated by 0.2% formalin,

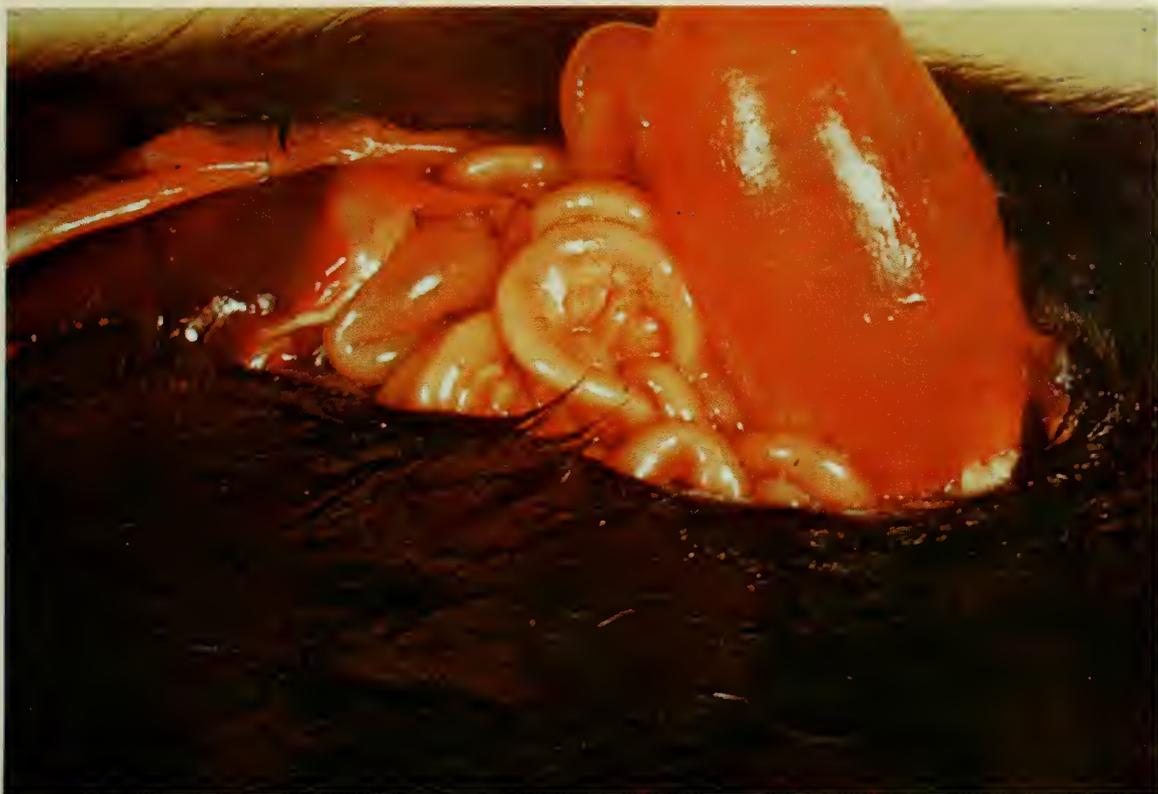


Fig. 1. Plum bladder disease — distended bladder due to calculi blocking urethra.



Fig. 2. Hemorrhagic pneumonia — lungs showing congestion and hemorrhage.



Fig. 3. Tuberculosis — lesions in abdominal organs.



Fig. 4. Aleutian disease — *L to R*: spleen, liver, kidneys; *top*, infected; *bottom*, normal.



Fig. 5. Distemper — swelling and encrustation of nose.



Fig. 6. Virus enteritis — stool sample with typical mucus "slug" on top.



Fig. 7. Rickets in kits — spinal column: *L*, normal; *R*, twisted due to rickets.



Fig. 8. Strangulation of tongue due to tracheal ring.

it can persist on a ranch from one season to the next. It is present in all secretions and excretions of affected mink, and recovered mink can excrete the virus for long periods. Infection is spread by contamination of feed or water (often by flies), or on contaminated mitts, clothes or equipment. Redistribution of unconsumed feed has been known to spread the infection and should be discouraged.

Symptoms

The first symptom noted is sudden loss of appetite, with most of the feed being left on the wire. Examination of feces reveals a grayish-white or clear to pinkish layer of mucus on top of, or occasionally mixed into, a stool that appears otherwise normal (Figure 6). There also may be blood in the feces. In most cases, kits die in 4 or 5 days. Any that recover do not develop normally and also serve as carriers of the infection.

Diagnosis

Appearance of mucus on the surface of stools and the high mortality in kits showing severe inflammation of the intestines suggest virus enteritis. The diagnosis is confirmed by microscopic examination of the intestine, which shows typical cell degeneration. On necropsy, most mink have dilated, flabby, inflamed intestines, with liquid, blood-tinged, foul-smelling contents. The spleen is usually enlarged and congested with blood.

Treatment and Prevention

Destroy all mink showing signs of enteritis; and vaccinate all remaining mink as soon as the diagnosis is confirmed. To prevent the occurrence of this disease, vaccinate all kits at about 10 weeks of age.

INHERITED CONDITIONS

CHEDIAK-HIGASHI SYNDROME (CH)

This is an autosomal recessive trait found in homozygous Aleutian mink (aa), which makes them more susceptible than other mink to many diseases. In infection, the white blood cells known as neutrophils are a very important part of the defence mechanism. Normally, these cells engulf invading organisms (bacteria and viruses) and release small enzyme bags (lysosomes) that destroy them. In Aleutian mink, the neutrophils move to infection sites more slowly. Once there, they pick up invaders, but lysosomes are not released to destroy them. With bacteria, multiplication often continues, eventually forming abscesses.

DEAFNESS

Certain white mutant mink are born deaf. It is due to the absence of certain parts of cells within the inner ear, which leads to severe degeneration.

DWARFISM (SHORTIES)

Occasionally, a kit is born tiny but without hair loss. The body is about half as big as usual, head and feet are of normal size, and some kits have undershot jaws and bulldog faces. Most die early in life. Littermates and parents should be pelted.

EHLERS-DANLOS SYNDROME

This disease is detected only at pelting time when the skin tears easily, ripping to shreds when fleshing is attempted. Microscopic examination of the dermis (inner layer) of the skin shows that collagen fibers, which normally give strength to the skin, are unequal in size, unorganized, and have a 10-fold decrease in tensile strength. Control of this autosomal dominant trait is difficult, as it is seldom possible to trace the parentage of pelter mink.

HYDROCEPHALUS (WATER ON THE BRAIN, BIG HEAD)

Affected kits have greatly enlarged heads with soft or thin skull bones. Rarely, the condition is internal, with a normal skull. Kits are dull, sleep a lot, lack size and muscle coordination, and most die early. The cause is an accumulation of fluid in the brain ventricles (cavities), which forces the brain against the soft developing skull bones. Both parents must carry the recessive gene and should be pelted, along with normal littermates of affected kits as these may also be carriers.

HYPOTRICHOSIS (HAIRLESSNESS, NAKED)

Kits with this anomaly are normal at birth but by a week old have only a few hairs, or none at all, and wrinkled skin. At 2 weeks they are small, very wrinkled and have a declining growth rate. Few live longer than 10 weeks, but by this time fur is present on the extremities. The adult coat develops on survivors, but all die by the end of November with the largest being dwarfs about half the expected size. This condition results from homozygosity for an autosomal recessive gene (nn). Ranchers must pelt normal littermates and parents.

MUSCULAR DYSTROPHY

As early as 2 months old, kits with this anomaly show

progressive signs of weakness, skeletal muscular atrophy (wasting away), and reduced appetite. As the condition advances, large muscles, including those of the head, undergo atrophy and lack tone. Kits have an unsteady, undulating gait, with impaired running ability. Blood serum enzymes such as CPK and SGOT are elevated. Diagnosis is by microscopic examination of muscle tissue. Littermates and parents should be pelted.

POSTERIOR PARALYSIS

This fairly common disease, seen in a few kits on a ranch, appears suddenly and recovery is rare. There is complete paralysis of the hind legs, with loss of excretory function control, but appetite is normal. Mink are born with an abnormality known as hemivertebra in the thoracic (rib) area; either one half of the bone fails to develop or the two halves fail to fuse. This allows the vertebrae later to push upward and put pressure on the spinal cord, causing the paralysis. Littermates and parents should be pelted, as the disease is inherited as a simple recessive gene.

WOBLER MINK

Abnormal lipid deposits in brain cells are responsible for this inherited defect, seen in all colors. There is progressive ataxia (incoordination). Littermates and parents should be pelted.

DISEASES RELATED TO NUTRITION

CHASTEK'S PARALYSIS

This condition is caused by a dietary deficiency of vitamin B₁ (thiamine). It occurs in mink that have been fed certain freshwater fish containing the enzyme thiaminase, which inactivates thiamine. Fish high in this enzyme include freshwater smelt, carp, alewife, sucker, whitefish, saltwater herring, channel catfish, bullhead, buckeye shiner, burbot, white bass, creek chub, fathead minnow, goldfish, mud minnow, menominee whitefish and sauger pike. With the daily feeding of thiaminase-containing fish, reserves of vitamin B₁ are used up and symptoms of Chastek's paralysis commence. The mink stop eating and become weak and thin, and develop a rather typical paralysis in which the head is pulled back almost to the tail. This is soon followed by muscular incoordination and further paralysis, then death.

Chastek's paralysis is easily prevented by not feeding fish known to contain thiaminase, or by cooking the fish

to inactivate the enzyme. The recommended procedure is to cook the whole batch of fish at 100°C for 15 minutes; and to cool the cooked fish quickly to minimize spoilage.

In an outbreak, immediately remove all thiaminase-containing fish from the diet unless it is properly cooked. Give injections of thiamine to affected mink and supplement the diet with a good source of thiamine, such as liver, meat meal, brewer's yeast or wheat germ. Most commercial cereals contain sufficient levels of thiamine to be of assistance in treating this deficiency, once the cause has been eliminated.

COTTONY UNDERFUR (COTTON PELTS)

Mink with this condition have a coat of weak, whitish underfur instead of the normal dense growth. Cottony underfur is associated with anemia. It is caused by feeding certain uncooked ocean fish (whiting, coalfish, hake and trawlfish) that interfere with iron absorption. It can be prevented by cooking the fish. Recent work indicates considerable heritability of underlying resistance to this condition in dark mink.

GRAY UNDERFUR

Gray underfur is caused by a deficiency of the vitamin biotin, which is necessary for normal pigmentation of hair. Affected mink have dirty-looking, nondescript underfur, their eyes discharge and crusts form on their skin. The condition occurs when uncooked turkey hens or their raw eggs are included in the mink ration. Turkey eggs contain a substance that interferes with the normal use of biotin. Cooking the turkey meat or eggs for 30 minutes at 100°C inactivates this substance. Where cooking facilities are not available, biotin can be added to the ration as a supplement. Affected mink respond to injections of 1 mg of biotin twice weekly.

NURSING SICKNESS (NURSING ANEMIA)

Nursing sickness can cause high mortality in females with large litters (five or more). Although it is the litter's excessive demand for milk that precipitates this condition, the actual cause is dehydration due to salt depletion. A lactating female loses a large amount of salt in her milk and, if this increased requirement is not filled, nursing sickness can result. Early encouragement of kits to eat, by making feed and water available to them, reduces the demand for milk and thereby the likelihood of this condition occurring.

Marked weight loss is one of the symptoms of nursing

sickness. After about a month's nursing, the female becomes very thin and may wander aimlessly about the cage, sometimes carrying uneaten feed in her mouth. These early signs are followed by unsteadiness of gait, then coma and death. Postmortem examination reveals dehydration, fatty liver and a complete absence of subcutaneous fat.

The addition of salt (NaCl) at 0.3 to 0.5% of the ration throughout the nursing period is sufficient to prevent nursing sickness. Since mink are susceptible to salt poisoning, an adequate supply of water must be available. Do not use more salt than recommended and mix the salt thoroughly into the ration before feeding. Where the condition of individual mink has advanced beyond a certain point, treatment is seldom successful. Control of nursing sickness depends on vigilance during the nursing period, to prevent females developing marked symptoms. At the first sign of weight loss, separate the kits from the female. Encourage her to eat by providing special food, such as fresh liver, milk, or a freshly killed bird. Intraperitoneal injections of vitamin B and normal saline (8 to 10 ml per day) may aid the recovery of dehydrated females. To make normal saline, add 9 g of salt to 1 litre (1 teaspoon to 1 pint) of **sterile** (boiled) water.

RICKETS

Rickets, once a serious problem, is a deficiency disease caused by insufficient vitamin D, with consequent disturbance of calcium-phosphorus metabolism. The same condition can result from a primary deficiency of either of these elements (ash) or an improper ratio between them. Affected kits, generally smaller than normal, crawl about with their hind legs bent outward somewhat like a frog's. The leg bones are rubbery and can be bent. On occasion, the spine also becomes twisted (Figure 7).

The first step in preventing rickets is to provide pregnant females with a diet containing adequate calcium and phosphorus (ground bones are a good source); and to supplement the diet with fish oil for vitamin D. If rickets appears in kits, add fresh fish oil supplement and a calcium-phosphorus source to their diet immediately. Moving affected kits into the sunlight is also beneficial.

SCREW NECK

This condition is associated with certain pastel mink, which show marked twisting of the neck, nervousness and imbalance. Affected mink tilt their head to one side or the other and may

throw it over the back when turning around. Sometimes the animal rotates its head through 180°, the underside of the jaws facing upward. Mildly affected individuals may show signs only when under stress. If severely disturbed, the mink may race about wildly, showing some degree of incoordination and sometimes somersaulting. Affected mink eat normally and are capable of breeding.

Diagnosis is by clinical symptoms. A test has been described that detects the condition through the inability of affected mink to swim normally; they assume a vertical position in water, rather than horizontal, and in advanced cases have difficulty in keeping their heads above water.

Screw neck is due to a reduction or complete absence of otoliths, structures in the inner ear that maintain equilibrium in normal animals. The condition can be prevented by the addition of manganese sulfate ($MnSO_4 \cdot H_2O$) to the ration at 3.1 g per kg of feed. Affected mink should be pelted.

STEATITIS (YELLOW FAT DISEASE, WATERY HIDE DISEASE)

Steatitis occurs in young, rapidly growing male kits during late summer and early fall, because they lack vitamin E. The condition is caused by feeding fish or meat that has been stored for so long that the fats have turned rancid. This change in the unsaturated fatty acids makes vitamin E in the fish or meat unavailable to the mink. Usually, the first sign of steatitis on a ranch is the finding of a number of dead kits. Other kits less severely affected may leave their feed; and some show an abnormal gait varying from mild unsteadiness to hopping, possibly due to inflamed fat deposits in the groin area. These symptoms usually progress to coma, followed by death. Mortality among affected kits may reach 75%, with survivors never furring outbn addition, hemolytic anemia and poor kit survival have been described for this condition in dark mink.

Postmortem examination reveals the presence, especially in the groin area, of yellowish fat deposits, which also appear swollen and 'wet.' Often, the spleen is markedly enlarged and hemorrhages are visible on the surface of the body fat. Microscopic examination shows inflammation of subcutaneous and visceral fat, liver and muscle degeneration, and calcification of the left atrium (part of heart).

In another form of steatitis affecting growing kits, the first abnormality noted is a swollen head. The swelling is confined to the top and does not extend behind the ears. Occasionally,

such mink also show extensive hemorrhage behind an eye, which may protrude from its socket. When mink with this form of the disease are skinned, a clear, jellylike substance is found adhering to the undersurface of the skin over the head. The urinary bladder is often found distended with reddish-colored urine containing brown adherent material, which is hemoglobin from the blood (hemoglobinuria). This atypical form of steatitis mimics certain bacterial infections (clostridial, streptococcal), but diagnosis is confirmed by microscopic examination of the fat, which reveals changes characteristic of vitamin E deficiency.

Kits affected with steatitis can be given 10 to 20 mg of vitamin E by injection daily for several days.

Control consists in correct storage of meat and fish to prevent rancidity of fat and subsequent inactivation of vitamin E. Make sure that freezers are functioning properly. If necessary, add stabilized vitamin E as a concentrate in the feed, to provide 3 to 5 mg of vitamin E per mink per day. This is sufficient both for lactating females, to prevent steatitis in nursing kits, and for growing kits after weaning. Usually, the level of stabilized vitamin E in commercial cereal is adequate to meet the animals' needs. However, if mink are fed a large proportion of rancid horsemeat or fish, extra vitamin E is necessary. It is recommended that such rations be supplemented with 0.5 to 1 kg of stabilized vitamin E (about 10,000 IU/kg) per ton of mixed feed, in addition to that in the cereal.

WET BELLY (URINARY INCONTINENCE)

Wet belly affects males more often than females and is most common in the autumn. Because this is during the early furring season, it has serious economic impact. In affected mink, urine dribbling from the urethral opening keeps the belly wet and causes severe staining and loss of fur. The pelts have patches of dark, unprime leather due to some regrowth of fur and have to be discarded or drastically trimmed.

The cause of wet belly is complex. It may be partly due to feeding a high proportion of raw fish or chicken; or to high-fat diets, calcium/phosphorus imbalance, bacterial infection, excessive male hormones or decreased water intake. Recommended treatments include reducing the fat level to 20-22% (dry basis) in rations during furring; adding phosphoric acid to the diet (calcium/phosphorus ratio should be 1:1); always feeding rations with low bacterial counts (possibly

cooked); and providing more water in the fall. Some people believe that the condition is inherited and affected families should be pelted.

DISEASES CAUSED BY PARASITES

COCCIDIOSIS

This disease used to be widespread and serious, but today, with wire-bottomed pens in general use, the incidence is much reduced. Coccidia are tiny protozoan parasites that multiply in cells lining the intestinal wall and are excreted as infective stages, called oocysts, in the feces. When mink were raised on earth or wood flooring, the oocysts developed in these locations and were ingested in contaminated feed and water. Warmth and dampness of litter greatly favor the survival and development of oocysts.

The most serious effects of coccidiosis are experienced by kits, although any age is susceptible. Severity depends somewhat on the numbers of infecting organisms and on individual resistance. Usually, the first symptom noted is that the feces contain more than a normal amount of mucus. At about the same time, mink may be slightly off-feed, lose weight and appear listless; and their fur may look dull. Droppings are usually loose, vary in color from reddish to black, and are often of a tarry consistency due to blood from the inflamed intestine. Older animals may develop resistance and show no symptoms after about 2 weeks, but they remain chronically infected and may disseminate the parasite throughout life. Also, the infection often makes them more susceptible to other diseases, so that they are less vigorous and less profitable.

Diagnosis depends on microscopic examination revealing oocysts in the feces or infected cells in the lining of the intestines of dead mink. Both sulfathiazole and sulfaguanidine provide effective treatment, but should not be given without professional advice because they may be toxic to mink. The best way to control coccidiosis is by prevention, through the use of wire-bottomed cages and good sanitation. Do not allow litter to accumulate, and remove feces regularly from water and feed pans.

FLUKES

Flukes are flat, often spade-shaped parasites that live in various organs of mink. All require alternate hosts for their development and infect mink through the inclusion of one of these other hosts in the ration.

Intestinal Flukes

Several species, ranging in size up to about 1 cm, can live within the intestines of both wild and ranch mink. Crayfish and frogs are the infective sources in the diet of wild mink; and raw freshwater fish can act as a source for ranch mink. Symptoms are unusual but may include diarrhea and dry, lusterless pelts, which show that the animals are not thriving normally. To control intestinal flukes, cook all freshwater fish before feeding.

Liver Flukes

The specific liver fluke of mink is *Metorchis conjunctus*, which has the common sucker as its intermediate host. This fluke can cause severe losses from its presence in the gall bladder and bile ducts. Symptoms are loss of appetite, general unthriftiness, weakness and jaundice. Jaundice is indicated by yellowish discoloration of the mucous membranes, skin, and especially eyes. In advanced cases, mucous membranes may blanch due to anemia. Diagnosis is by locating the adult fluke in liver or gall bladder or by examination of feces for the presence of characteristic eggs. Although there is no practical treatment, the disease is controllable by cooking suckers before feeding.

Lung Flukes

The lung fluke, *Paragonimus kellicotti*, is a common parasite of wild mink because they feed regularly on its intermediate host, the crayfish. This fluke also has a second intermediate host (certain snails), but crayfish are the usual carrier to mink. The adult fluke, approximately 0.75 cm long and 0.5 cm wide, occurs in cystlike cavities in the lungs. These cavities are quite large and appear on the lung surface as raised, bluish areas. When the cavities are cut into, flukes can be easily observed with the naked eye. Sometimes there is more than one fluke in a cyst, which also contains a thick, brownish fluid. The main symptom of infection is a harsh, dry cough. Diagnosis is by finding the parasite in the lungs. There is no available treatment. Infection is prevented by eliminating crayfish from the diet or by boiling them before feeding.

KIDNEY WORMS

The giant kidney worm, *Diocotophyma renale*, is reddish colored, about 0.5 cm in diameter and up to almost 30 cm long. Usually, only one worm is present and only one kidney is involved; but the worm has been known to affect both kidneys and also to occur free in the abdominal cavity. If both kidneys are involved the mink usually dies.

This parasite has a complicated life cycle, with its eggs passing out in urine to eventually reach crayfish. When the crayfish are eaten by certain fish (bullheads), the parasite develops further, forming cysts in the fish. These are infective when fed back to mink, completing the cycle.

Signs of infection in mink include irregular appetite and, occasionally, colic. The only definite diagnosis is by finding the worm within the kidney of a dead mink. There is no effective treatment, so control depends on cooking of bullheads before feeding.

MYIASIS

Myiasis means infestation of animal tissues by fly maggots (larvae). The most serious ones to mink, particularly kits, are those of flesh and blow flies. Although abundant throughout the summer, the flies are most active in northern latitudes during June. The adults are about twice as big as house flies, grayish to gray-black in color, and slow and deliberate in their movements. The female lays tiny, live maggots directly on the kits, preferring the face, neck or flank, or near the eyes or anus. Larvae are also often deposited on any skin wounds. These larvae immediately penetrate the kits' tender skin, which becomes inflamed, and serum exudes from the points of penetration, causing the hair to mat. Each maggot burrows under the skin, forming tunnels, and bores holes to the outside to obtain air. As it grows in size, boil-like swellings develop under the skin, marking the site of its development.

During initial penetration, infested kits whine and become very restless, and the female may drag them from the nest box and leave them on the wire to die of exposure. With continued development of the maggots, kits are unable to grow normally due to constant irritation. They show lack of appetite, increasing debility and emaciation, which may lead to death. After about 2 weeks, the maggots complete their development and fall to the ground, where they pupate before completing their life cycle. If kits survive this experience, they are left with severe damage to the skin and subcutaneous tissues, which may pave the way for secondary bacterial infection and also will devalue the pelts.

Treatment of infested kits can be undertaken but is laborious, as it involves removing the maggots with forceps and flushing the sites with a mild antiseptic. The best way to control the flies is to place Korlan or Ronnel dust in the nest-box material during late May or early June, taking care to follow

directions to avoid poisoning the kits. Although screening of sheds may provide some protection from the flies, screening of cages is impractical. Keep lids of nest boxes tightly closed.

TOXOPLASMOSIS

This disease affects all ages of mink and is caused by the protozoan *Toxoplasma gondii*. The parasite exists in two forms, one causing acute infections and the other chronic disease. It may be transmitted to other animals if they eat the tissues of infected animals. In the cat family, the parasite undergoes a sexual cycle in the intestinal tract, to produce a third form that is shed in the feces. Animals can then become infected by these oocysts in their feed or water. In addition to mink and cats, such animals as mice, rats, dogs, cows, pigs, sheep, pigeons and chickens can be chronically infected. Infective cysts in meat probably are the most common source of toxoplasmosis for mink. Contamination of mink feed with cat feces is another possibility, and infection of unborn kits of an infected female is known to occur.

The symptoms of toxoplasmosis vary with the number of organisms ingested, the tissues invaded and the immunity of the host. Nonspecific symptoms in adults may include loss of appetite for several days after infection. The mink then may develop a resistance that allows them to return to normal, with few losses. However, the chronic infection that results can infect their kits in the uterus. The effects of this type of infection are not evident until a severe problem of abortion, stunting or early kit mortality is experienced. If the parasite invades the brain, possible symptoms include extreme excitability, inability to chew and swallow normally, incoordination, convulsions and inability to find their feed (possibly due to blindness).

There is nothing distinctive about the postmortem appearance of mink dead from toxoplasmosis. Diagnosis depends on locating the parasite microscopically in the tissues. A nonsuppurative encephalitis (inflammation of the brain), with typical cysts, is present where nervous symptoms have been encountered. In other cases, areas of necrosis in tissues and organs associated with the presence of the parasite are diagnostic. A serological test (the Sabin-Feldman dye test) is available to assist diagnosis, and could be used as a ranch screening test.

At this time there is no known treatment or prevention of practical value. Although vaccines have been developed and

successfully applied experimentally, prevention should center on attempts to eliminate live parasites from the feed. Do not feed carcasses of wild animals to mink, as they are often infected. Try to control rats and mice, and keep cats away from the ranch, especially those areas where feed is stored, mixed and held. Feeding only cooked meat to domestic cats helps minimize the risk of such animals carrying the disease to the premises. Freezing at usual freezer temperatures does not necessarily inactivate the parasite.

There is a very real public health hazard with toxoplasmosis. Try to avoid handling uncooked meat with bare hands; and always practice strict hygienic principles. If toxoplasmosis is suspected, be sure to seek professional advice.

POISONS

AFLATOXIN

Aflatoxin, a mold toxin produced by some strains of *Aspergillus* sp., may be found in cereal grains. It causes liver degeneration in mink, induces cancer-type changes over a period of time and reduces production. Diagnosis is by laboratory examination of tissue and analysis of tissue and feed for the toxin.

CHLORINATED HYDROCARBONS

This group of chemicals includes many of our pesticides, for example, DDT, DDD and DDE. The importance of these compounds in reproductive and other problems in mink has caused a great deal of concern and speculation in the mink industry. However, at present, it appears that the danger from these chemicals is not as significant to mink ranchers as was believed.

In general, mink have a high tolerance for DDT, DDD and DDE, and the quantities required to produce acute poisoning are much greater than would normally be found in fish or other animal products fed to mink. Levels of DDT as high as 700 parts per million (ppm) have been found in the fat of mink without apparent clinical illness, illustrating the ability of this species to accumulate these residues over a period of time.

However, a related chlorinated hydrocarbon, dieldrin, is known to produce problems in mink at much lower levels. Mink die when fed levels of this compound as low as 2.5 ppm for extended periods. The long-term effects of lower levels of dieldrin and its possible effects on reproduction are not yet

fully known, but the growth rate of mink fed quite low levels is significantly lower than that of controls.

Apart from the possibility of such chemicals being biologically incorporated in feed, the main danger would seem to be in spraying mink or applying the material accidentally to their pens or feed. Obviously, precautions should be taken to prevent this, including the labeling of all supplies to ensure that mink do not become exposed inadvertently through misidentification.

DIMETHYLNITROSAMINE (DMN)

This chemical toxin causes extensive liver damage. At one time, sodium nitrate was widely used in fishing boats to preserve herring. During processing into fish meal, the fish was heated, causing the nitrate to combine with trimethylamines in the fish to form DMN.

When mink are fed DMN, they become very pot-bellied and die suddenly. On necropsy, the abdominal cavity is filled with fluid and blood, the liver shows degeneration and numerous tumors of blood vessels are evident. Diagnosis is based on these lesions and on analysis for DMN in feed.

LEAD

As mink are very susceptible to lead poisoning, the use of lead-containing paint or other material on a mink ranch is not recommended. The problem most often occurs following the painting of nest boxes with a lead-based paint or the use of red or white lead in oil applied to wire or other metal equipment as a preservative.

The disease may be acute or chronic. Acute lead poisoning occurs when mink are placed on newly painted wire before the paint is thoroughly dry. After 2 or 3 days, the mink are lethargic and stop eating. Soon muscular incoordination and trembling are noted, and pus collects in the corners of the eyes. Death follows in about a week, often with convulsions. The chronic form of the disease can occur in mink on treated wire even when the paint is allowed to dry completely. There are no dramatic signs, but there is gradual weight loss, with death occurring in 1 to 2 months.

There are no characteristic lesions (signs of disease) on postmortem examination, so diagnosis is based on relating the clinical signs to the use of paint or other lead-containing preservative. It should be kept in mind that distemper also produces convulsions and pus in the eyes. Once the diagnosis is

made, remove all animals from painted equipment as soon as possible. Give survivors vitamin D and dicalcium phosphate or calcium gluconate as supplements to the ration for about a month. These substances help remove lead from the blood and deposit it in bones. A veterinarian may be able to treat individual mink by using a chelating agent, such as calcium ethylenediamine tetraacetate.

POLYCHLORINATED BIPHENYLS (PCB'S)

These industrial chemicals gain entrance to the environment through transformer or hydraulic system leaks, or by burning plastics at low temperature. They become concentrated in the food chain and can occasionally be found in high levels in large fish. One example is their presence in Great Lakes coho salmon, which caused mink reproductive failures several years ago. Recent research indicates that very low levels cause reproductive failures and higher levels cause death. Diagnosis is by analysis, with highest levels being found in body fat.

SALT

Common salt, sodium chloride (NaCl), is an essential constituent of the body. Mink usually obtain the small amount they need in meat, fish, cereal or other ingredients in the ration. Extra salt can become incorporated in the mink ration by an error in mixing or perhaps by feeding large amounts of salt fish. If a great excess of salt is present, mink refuse to eat. The danger of salt poisoning occurs when a slight excess of salt becomes mixed with the ration, especially if the supply of drinking water is limited. In fact, the usual amount of salt in a mink ration can cause the problem if water availability is not normal.

The first symptoms of salt poisoning are weakness and depression, with the animal tending to lie curled in a corner. Later symptoms include nervousness and incoordination and, since the condition is partly due to dehydration, sunken eyes and weight loss. In fatal cases, death usually is preceded by convulsions.

The most characteristic sign on necropsy is marked reddening of the stomach lining. This may be general, affecting the entire lining or a large area of it, or focal, showing spots of hemorrhage. Once salt poisoning is suspected, remove all feed and check the water supply to make sure it is available on demand. Individual mink can be treated by a veterinarian to control dehydration and reduce nervous symptoms.

STILBESTROL

The term "stilbestrol" refers to a group of natural and artificial hormones that have been used in the past to stimulate growth in some animals, mainly poultry and cattle. The use of contaminated feed or of meat from such animals in mink rations can severely disrupt normal reproduction, even to the extent of complete failure to produce kits. As legislation now prohibits the use of these hormones for stimulating growth, they should no longer be a problem.

STREPTOMYCIN

Streptomycin is very toxic to mink. Symptoms, which commence within $\frac{1}{2}$ hour after a toxic dose, include extremely labored breathing, then muscular incoordination and collapse. Death usually occurs within a short time. Although it is possible to use this drug successfully by careful attention to the dose, as a general rule it should be avoided in treating mink.

SULFAQUINOXALINE

Although occasionally used to treat coccidiosis or other intestinal infection, this drug should never be given to mink, as it interferes with their blood-clotting mechanism. The compound produces spontaneous hemorrhage in the internal organs, causing death. On necropsy, the abdominal cavity is found to be filled with bright-red, unclotted blood.

THYROID GLAND TOXICITY

Frequently, gullets, windpipes, or pluck from cattle include portions of thyroid and parathyroid glands. These glands, near the upper part of the windpipe, are bean-shaped structures darker in color than the surrounding tissue. The glands should not be included in mink rations, as they can cause reproductive failure. Affected mink become thin and nervous; but the main effect is that the females either fail to produce a kit crop or they have a high proportion of stillbirths.

WOOD PRESERVATIVES

Many ranchers, in the interest of protecting their equipment, apply wood preservatives. Some of these contain compounds such as cresols and chlorinated phenols, which are toxic, especially to young mink. Affected kits usually die during their first 2 or 3 weeks, with no diagnostic signs or lesions. Losses in older mink may continue sporadically over a long period. To avoid such losses, do not use wood preservatives on mink pens, inside nest boxes or on any outside framework that

the mink might chew. The toxic property of these materials persists for more than a year, no matter how thoroughly the treated wood is dried and aged.

NONSPECIFIC CONDITIONS

CANNIBALISM

Cannibalism occurs occasionally among mink kits, but is usually not serious. The highest incidence is among mink kits 1 to 2 months old confined to the nest box by a long spell of bad weather. Such crowding and inactivity may provoke an outbreak of fighting among the naturally aggressive mink and on occasion can lead to the death or serious disfigurement of some kits.

Older kits can be separated and their wounds treated with antibiotic dressings, perhaps supported by injectable antibiotics. Since most cases occur before the kits can be safely weaned, however, individual treatment is difficult. Some success in controlling ranch problems has been obtained by increasing the fat level of the ration. The mechanism is unknown, but less frequent fighting within the litter is reported to follow this change.

CHOKER

Mink rations often contain ingredients that can cause choking or other eating difficulties. For example, bone splinters may become stuck between the teeth, either preventing the mink from closing its mouth or interfering with normal swallowing; or sometimes a section or ring of windpipe in turkey or chicken waste encircles and lodges around a mink's tongue (Figure 8). Though apparently hungry, the mink refuses to eat. It may paw at its open mouth and usually salivates profusely. The solution is to locate the foreign body (a flashlight is handy) and remove it. If the tongue gets stuck in a tracheal ring, it can become very swollen and full of blood, making location of the ring difficult. In severe cases, the tongue may slough; such mink can be fed very wet feed and they should be pelted in the fall.

DEHYDRATION

Dehydration occurs when a mink is deprived of some or all of its supply of fresh drinking water. Ordinarily, 3 days without water will kill a mink, but during hot, dry weather this can happen in 1 day. When a mink has been without water for a day, it stops eating, becomes restless and continually visits the

waterer. Depression follows, with skin and muscles contracting to give a thin, wasted appearance, and with eyes sunken and perhaps shut. Eventually, the mink has a convulsion and dies. Provision of adequate water at all times, along with frequent checking of automatic nipple waterers, prevents this condition.

HEAT EXHAUSTION

Mink exposed to abnormally high temperatures for a period of time may suffer from heat exhaustion. Both hot sunshine and a hot, humid atmosphere without proper circulation of air induce a state in which the animals' heat-regulating mechanism is no longer able to cope. Body temperature rises abnormally, respiration becomes difficult and the mink collapse. The whole chain of events can take a surprisingly short time. Usually many mink are affected at the same time and losses can be extensive, especially at whelping time or when kits are only a few days old.

At whelping, young kits with little tolerance succumb quickly without any particular signs. The uncomfortable female neglects her kits and may block the nest box entrance, reducing air circulation within. Older kits and adults become quiet, stretching out on their sides on the wire. They may make frequent trips to the water dish, but eventually seem unwilling to exert themselves even to this extent. If an attempt is made to pick one up, it may turn on its side, raise its head and make weak crying sounds; or it may crawl or show increased nervousness by biting at the wire. Respiration rate increases, breathing appears somewhat labored and sometimes saliva collects around the mouth. Death is preceded by a convulsion or coma. Postmortem examination reveals the lungs to be markedly congested. The large veins are often distended with blood which fails to clot normally when exposed to the air.

Although little can be done to treat young kits, older mink can sometimes be revived by dipping in lukewarm water and placing them in the shade. Efforts should be directed towards prevention. Make sure that animals always have an adequate supply of fresh water; inspect automatic waterers regularly, to ensure that all nipples are working. During warm weather, place wet sacks on the wire to help cool the air; and prop nest-box lids open on very hot days. Protect the animals from hot sun with permanent or temporary roofing, but take care not to cut off the circulation of air. Also, during the time when kits are confined, remove nest-box material to allow better air circulation within.

HYPERTROPHIC PULMONARY OSTEOARTHROPATHY

In this very rare disease, there is marked swelling of legs and feet, due to excessive bone production and soft-tissue swelling. The disease is associated with chronic lung lesions, such as tumors or abscesses. It may be confused with distemper, so postmortem examination is necessary for diagnosis although X-ray is of value. There is no practical treatment. (See Figure 9.)

KIT DEFORMITIES

On any ranch, there are always a few newborn kits with deformities. Most of these are born dead, but some are born alive and die within 24 to 48 hours. The most frequently encountered deformities discussed here are sporadic, noninherited defects resulting from chance alone. Where an unusual incidence is noted, professional help should be solicited.

Dropsy (Anasarca, Water Dogs)

Dropsy is the most common deformity of newborn kits. An affected kit has short, fat legs and a head swollen to resemble that of a bulldog. Essentially, the condition is due to massive accumulation of fluid beneath the skin, giving the kit a swollen, or distended, appearance. All colors can be affected, and usually only one kit in a litter is involved. In most cases, the kit is born dead, although there are records of live births followed almost immediately by death. Dropsy can induce a secondary problem by the size of the swollen kit causing a difficult birth; protracted whelping may lead to subsequent kits being born weak or even dead.

Umbilical Hernia

Sometimes a hernia of the intestines occurs through the navel when the umbilical cord separates after birth. The kit is usually alive at birth, but dies within a day or two.

Cleft Palate

Although kits appear normal, when the mouth is opened the palate is seen to be divided along its midline. This forms an opening between the mouth and nasal cavities. The kits are unable to nurse and die within 24 hours.

Hare Lip

This is a deformity of the upper lip, in which there is a partial or complete split at the center line. Kits are unable to

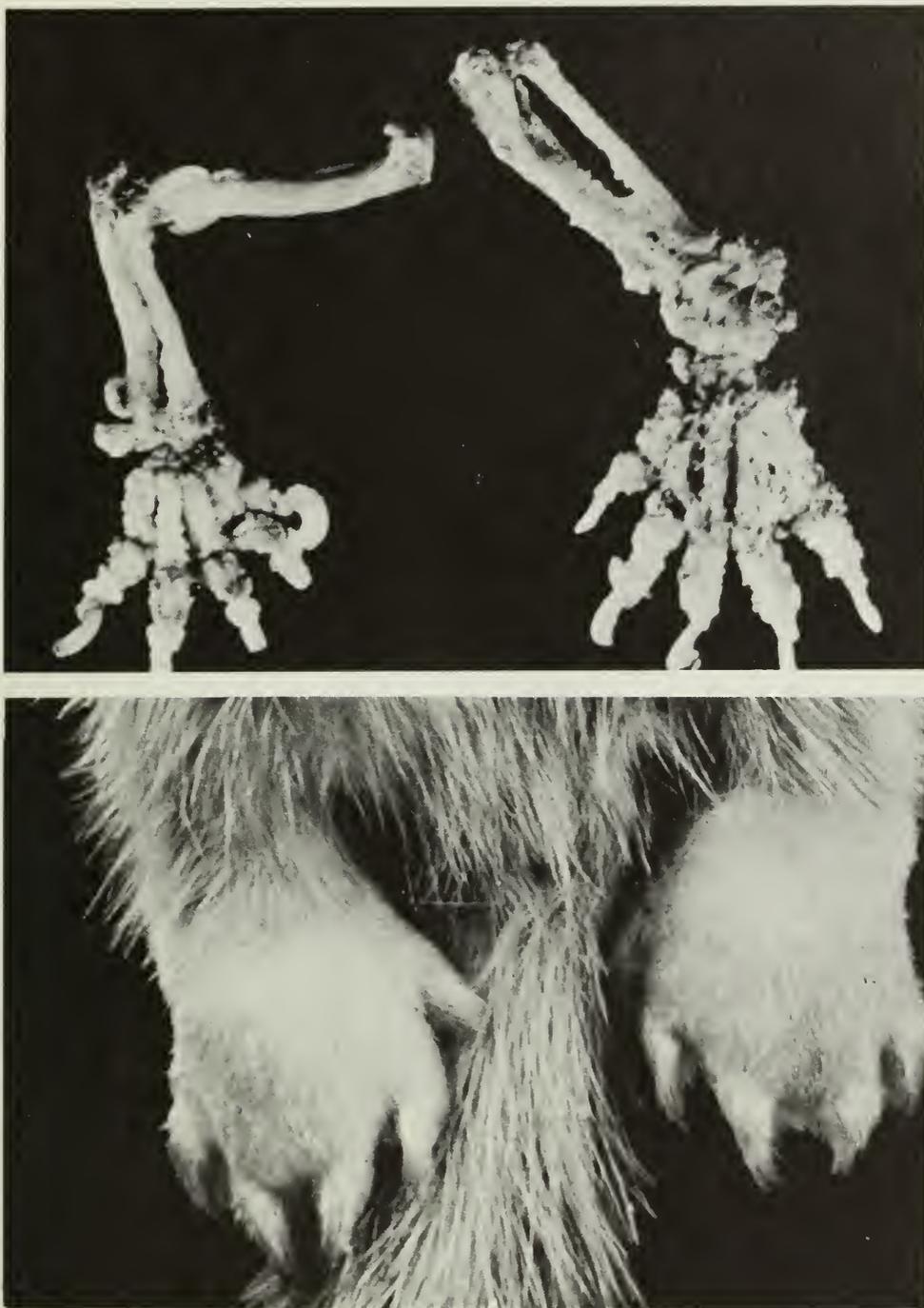


Fig. 9. Osteoarthropathy — *top*, bony deposits on feet; *bottom*, swollen hind feet.

nurse and die within 24 hours.

Brain Rupture

Affected kits have an opening in the midline of the skull, through which part of the brain and its surrounding membranes protrude. The kits are usually stillborn; if alive, they die soon after birth.

Cyclops

This is a rare deformity, in which both eyes lie within one larger-than-normal socket located in the center of the forehead.

PARAPLEGIA (BROKEN BACK)

The symptoms of paraplegia are identical to those of posterior paralysis, discussed under Inherited Diseases. The condition is usually caused by an injury, such as a fracture of the back or displacement of an intervertebral disc, which could happen during catching. Treatment is seldom curative, although some cases undergo spontaneous recovery.

RECTAL PROLAPSE

Rectal prolapse is usually associated with enteritis or bladder infection, although there are other causes. The lining of the rectum protrudes through the anus, exposing mucous membranes, which become dried and prone to injury and infection. Littermates may chew the exposed membranes, adding to the problem. Prolapse is rare in virus enteritis outbreaks, but commonly occurs with bacterial and nonspecific enteritis, when severe inflammation of the intestinal tract causes profuse diarrhea and straining. Immediate treatment is essential to control enteritis or bladder infection to avoid a serious situation.

Sometimes, individual kits suffer prolapse because of bladder infection or after a prolonged period of cold wet weather. Also, it appears that phosphoric acid in their diet can lead to prolapsed rectum and for this reason should not be incorporated in the ration between May 15 and July 15. In individual cases, early detection followed by surgical treatment may be successful. Mild ointments containing antibiotics and cortisone, as used for mastitis treatment, can be applied to the rectum.

SCLERODERMA (HARD SKIN)

This rare disease of unknown origin results in thickened skin, especially of the neck and legs. The skin is so tense and hardened that it cannot be elevated and results in a dog-paddle type of walk. The disease progresses to death.

TAIL CHEWING AND FUR CLIPPING

Tail chewing and fur clipping are vices associated with the artificial conditions under which ranch mink are raised. Their

close confinement and artificial diet are probably both partly to blame.

In fur clipping, a mink chews off all the fur it can reach on its own body, giving it a bizarre appearance somewhat reminiscent of a lion. Long hair (normal) covers the front part of the body and short, chewed-off hair covers the mink from behind the front legs backward. Fur clipping may be regular or sporadic. Affected animals should be disposed of.

Tail chewing usually starts by a mink sucking the end of its tail. It may then begin to chew, eventually mutilating itself to the point where it has removed its entire tail, bone and all. Usually secondary infection occurs and the mink dies from septicemia (blood poisoning). An additional cause of tail chewing has been attributed to impaction of the anal sacs. These small glands, one on each side of the anus, secrete a foul-smelling fluid that the mink discharges when disturbed and in the wild probably uses to attract a mate. Occasionally, the duct of a gland becomes blocked, so that the fluid secretion accumulates and the gland is perhaps infected. The resultant irritation provokes the mink to bite at the gland, inflicting wounds that may start the chain of events. Therefore, always check tail chewers for anal gland problems. Where these are found, squeeze out the contents of the glands by exerting pressure between thumb and forefinger; be sure to wear rubber gloves because of the foul smell. Repeat the treatment several times over a period of a week. If the contents appear infected, treat with injectable antibiotics.

