Fur Animal Research

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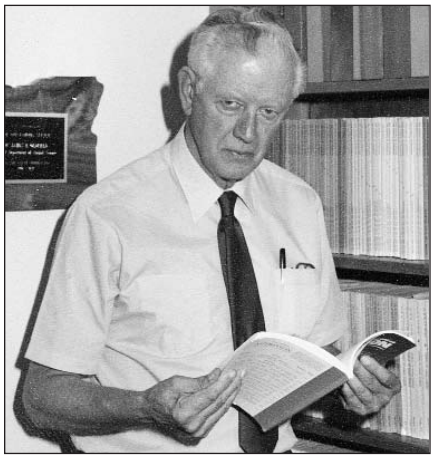

The American Fur Industry lost one of its leaders recently with the death on July 9th of Archie Gardner who raised mink and involved himself in industry affairs from his ranch near Sublimity, Oregon. During his long career, Archie served as President of both the Oregon Mink Ranchers' Association and the National Mink Breeders' Association (EMBA).

And seeing Archie's name reminded me of an interesting and, at the time puzzling, situation that we encountered in our diet studies at the Oregon Experimental Fur Farm. At the time, Dr. Floyd Stout was investigating the feeding of turkey waste (viscera, heads and feet) and we were buying the turkey product from Archie Gardner. Dr. Stout found when our mink were fed the waste product that they developed a grey banding pattern in the pelts, which greatly diminished their value. Through careful research he was able to pinpoint the cause of the fur banding: it was caused by a deficiency of one of the B-vitamins, Biotin, which in turn was caused by presence of a substance called avidin in the feed. The puzzling part of the situation was that Archie Gardner, who fed more of the waste product than we did had no grey-fur problem. Archie couldn't understand why we had the problem and we couldn't understand why he didn't, and relations between us were a bit strained for awhile. The answer, when Dr. Stout discovered it, was quite simple. Avidin is highest in turkey eggs and occurs at lower levels in the viscera. At our small Experimental Fur Farm, we used to buy all the fresh feed ingredients at one time and store them in the freezer. It so happened that we bought the waste at a time when breeders were culling their hen turkeys and waste from them contained the eggs, which were high in avidin. Archie fed largely waste from tom turkeys, which contained no eggs, so he had no problem. After we got this worked out our relations with Archie improved and we were good friends again. This was probably the first time that biotin deficiency has been diagnosed in mink and it exemplified the outstanding nutrition research that Dr. Stout developed. We summarized his work in an earlier issue of this newsletter (volume 11, number 1; April, 2003).

United Vaccines, Inc., is cutting back on its staff, as you know, and recently issued the following statement of their laboratory testing procedures:

CHANGES TO CEP TESTING

(Effective immediately)

Due to recent downsizing of the United Vaccines, Inc. CEP testing laboratory, we can no longer accept the volume of mink blood samples for testing that we have accepted in the past. Because of this smaller capacity you must schedule your samples with customer service prior to sending them to our laboratory.

SCHEDULING

You must call customer service prior to sending your samples and arrange a schedule for testing. Currently our lab can test a total of 2,500 samples per day for four days of the week, or a total of 10,000 tests per week. Customer service will explain this new policy to each customer at the time you order your testing supplies. All samples will be tested in the order that they are scheduled.

Samples received without prior scheduling through Customer Service may not be tested and could be returned to the sender.

UNITED VACCINES, INC.

August 17, 2005

We hope you are not having trouble with hot weather. In the Northwest, we have had as long as a week over 90°F which is hard on the mink. When we were operating the Experimental Fur Farm at Oregon State University, we installed sprinklers on top of the houses, which kept the cage temperatures about mid-80's F, and the mink seemed to tolerate this. Have a great summer!

J. E. Oldfield

NERVOUS DISTEMPER IN MINK

While there are other causes of "screaming fits" in mink, the prospect of distemper should always be considered first. A farmer may go out into his yard and hear a mink violently screaming. By the time he reaches the animal, it may appear normal but rather unsteady and have a string of saliva hanging from the corners of its mouth. This animal should be completely isolated from the herd and not killed. It should be watched very carefully to determine if the convulsions are repeated. If the mink actually has distemper, it will usually have more convulsions and succumb in a terminal episode. It is well to point out that normal mink occasionally have convulsions when they are excited but a good axiom to follow is that it is distemper until proven otherwise.

There have been outbreaks of Aleutian Disease in which nervous signs were common (Dyer and coworkers). On at least one occasion, an AD outbreak was confused with distemper because of screaming fits. In such outbreaks, it is well to secure the services of a diagnostic laboratory to differentiate between the two virus infections. The possibility of both diseases occurring in the same herd at the same time must always be considered.

Signs of Nervous Distemper

Mink affected with nervous distemper may go into a convulsion at minor disturbances such as feeding and watering. The animal first becomes rigid with one or more limbs extended. As in the accompanying picture, the head is thrown back. Violent spasms occur with the animal twisting and rolling in the pen. The jaw muscles are frequently affected, resulting in chattering teeth and foaming at the mouth. Short, piercing screams that can be heard over the entire yard are common.

Many times the animal will die during these convulsions. In other cases, however, there is a gradual or abrupt return to consciousness and in one or two minutes the animal appears normal. The convulsions become more frequent, terminating in a long, continued coma until death intervenes.

Distemper Virus Strains

If a farmer has recorded a large number of the usual catarrhal cases of mink exhibiting either swollen, crusty eyelids or swollen foot pads or both, there will be proportionately a greater number of cases of nervous distemper. Almost invariably the farmer feels that he is dealing with a different virus strain because of the many cases of screaming fits.

If there are many distemper cases at the time of vaccination, the vaccine will not control the outbreak. Then, until many weeks following vaccination, the farmer feels that he is dealing with a new strain of virus, a different disease or that the vaccine failed to protect. It is not the fault of the vaccine in these instances. It simply has not had

Neurotropic signs in a distemper-infected adult mink: the head is thrown back in a violent spasm and stringy saliva bubbles from the corners of the mouth.

enough time to develop an effective immunity in the herd.

In rare cases, however, there does seem to be a definite distemper virus strain difference. Many cases of nervous distemper are recorded without the usual number of mink showing eye, nose and feet signs. In these instances, normal appearing mink, with good appetites and no other signs of disease, succumb during convulsions.

Prevention

As in the case of distemper in general, the nervous form can be prevented by routine vaccination. Even though nervous cases may predominate in rare instances, the regular vaccine protects against this form of the disease. Treatment is to no avail. Once the distemper attacks the mink's brain it causes irreparable damage. It is well to kill these animals showing distemper convulsions as they offer a continuing source of the virus to other mink.

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FIBER IN THE MINK DIET

In formulating mink diets, we tend to emphasize fat (as a source of energy) and protein (as a source of body-building materials, including fur). Sometimes we overfeed on these nutrients which causes problems. Overfeeding fat has been implicated as a cause of wet-belly disease, which significantly lowers pelt values, while overfeeding protein increases the cost of the diet since protein is expensive. This raises the question of what to use to take the place of these major nutrients. One answer is the cereal mixes, which are an accepted part of most mink diets. Cereals are largely carbohydrates, and the carbohydrates can be of two types: soluble carbohydrates, like starches and sugars, and insoluble carbohydrates, like fiber. Advantages of adding fiber are said to include its effect on animal fullness, which may restrict the diet intake, and investigators at the Danish Fur Breeders Research Center, at Holstebro have looked into this. The fiber source they chose was barley hulls, which they fed at about 12% of the diet dry matter. They found that barley hulls at this level increased the time spent by the mink in feeding, while tending to restrict the total amount of diet eaten and they suggested that barley hulls could be used as a satiety factor in mink feed (from: Hejlesen, C. and P. Sandbol, 2003. Fiber as a Satiety Factor in Mink Feed. NJF Seminar no. 354, Lillehammer, Norway, 4 pp.).

DIETARY VITAMIN A LEVEL AND GROWTH & FUR QUALITY IN MINK

Vitamin A (Retinol) is a fat-soluble vitamin that is essential for animals to promote normal vision, protein synthesis, bone and joint development and reproduction. Retinol is stored by animals in their livers and, consequently, dietary liver is a good source of the vitamin. Vitamin A can also be formed by the animal from plant carotenes, especially beta-carotene. Finnish research has set the dietary requirement for vitamin A at 3500 I.U. per kg of dry matter.

Supplemental vitamin A is expensive and it should be remembered that some mink feeds (e.g. liver) contain significant amounts of vitamin A - possibly even more than needed. Finnish research studied the effects of dietary vitamin A on health, growth and fur quality in mink. Eighty dark, male mink were involved and were fed the test diets from July until pelting. Four treatments were involved: (1) Control (no supplementation); (2) Control + 1000 I.U. vitamin A/kg feed; (3) Control + 5000 I.U. vitamin A/kg feed; (4) Control + 10,000 I.U. vitamin A/kg feed. The control diet consisted of herring, beef and pork by-products, cooked barley, a protein concentrate and soy oil as a source of energy. A vitamin mix which contained no vitamin A was added.

All animals were healthy throughout the experiment, and grew similarly on the different test diets (Figure 1). The Finnish Fur Breeders' Association recommends 3,500 I.U. vitamin A/kg feed, so diets 1 and 2 were below the Finnish recommendation while diets 3 and 4 were above it. Observations on the pelts produced showed no significant differences across treatments for skin length and weight, fur density and quality. It appears that commonly-fed mink diets, such as the one used in these investigations, can be safely and profitably fed without supplementary vitamin A.

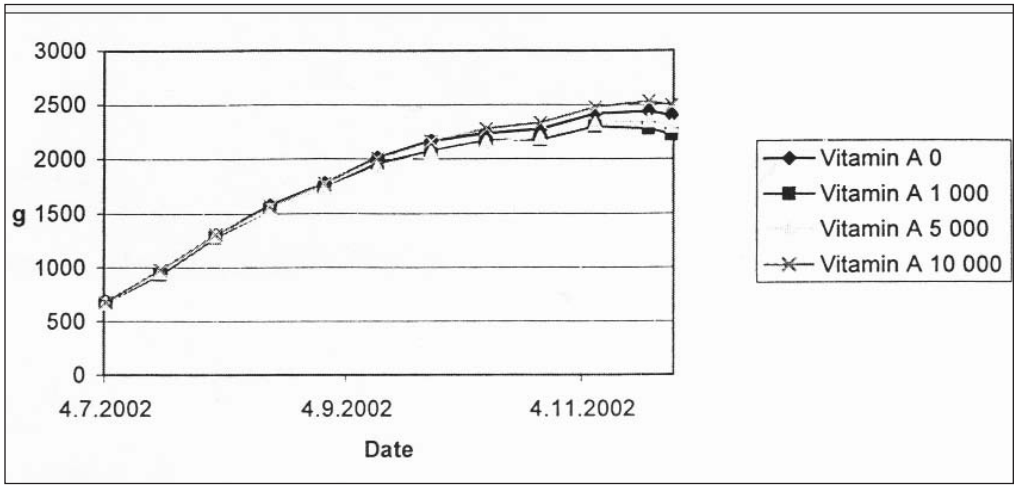

Figure 1. Weight gain of the mink (g) (from Nenonen, N., I. Polonen, T. Rekila and J. Valaja. 2005. Influence of dietary vitamin A level on growth performance and fur quality of mink. Finnish Fur Breeders Association. Vantaa 78)

USE OF IRRADIATION TO PRESERVE MINK FEEDS

Because irradiation is an effective methodology to preserve human foods, it is of interest to assess the process as a means of sterilizing and preserving fresh mink feed ingredients/complete feed to determine if disease-causing bacteria can be eliminated or reduced and to determine how long ingredients/feed can be stored in a non-frozen state without rapid spoilage. The objectives of the present proposal were: (1) to assess the length of time that irradiated duck offal and complete feed can be kept in a cooler before high bacterial counts and oxidative damage render it unusable and (2) to assess palatability of the treated product.

Approximately 240 lbs of feed and 85 lbs of ground duck offal were thawed overnight and packaged in 2 gallon plastic bags (approximately 8 lbs/bag). Bags were then placed in styrofoam coolers and refrozen for 48 hours prior to ground transport to CFC Logistics in Quakertown, PA. Feed/duck offal was processed upon arrival at the CFC Logistics facility, which maintains a Genesis™ cobalt 60 irradiator. A dose of 3.00 kGy was requested, which is sufficient to inactivate spoilage and pathogenic bacteria. The actual minimum dose was 2.03 kGy and the actual maximum dose was 3.03 kGy. After irradiation, which required approximately 60 minutes, the product was placed in a freezer for 24 hours prior to the return trip to Michigan State University (MSU). Upon arrival at the MSU Experimental Fur Farm, the Styrofoam coolers containing the irradiated feed and duck offal were placed in a freezer until subsequent use.

Twenty 6-week-old kits were provided with irradiated feed (IF) while 20 kits were provided non-irradiated feed (NIF) for a 21-day period. At the beginning of the trial, male and female kits in the NIF group weighed 480 g and 418 g, respectively, compared to 453 g and 381 g for IF males and females, respectively. At 9 weeks of age, body weight gains of NIF males and females were 114% and 88%, respectively, compared to 130% and 100% for IF males and females, respectively.

The results of the feeding study indicated that the irradiated feed was palatable and that kits fed the irradiated feed had a greater relative weight gain compared to kits fed a conventional diet.

In the near future, irradiated and non-irradiated feed will be placed in the farm's walk-in cooler and samples will be taken on a daily basis for up to 21 days to assess bacterial growth. In addition, irradiated and non-irradiated feed will be kept at room temperature for up to 7 days with bacterial counts being determined on a daily basis. In addition, diets will be mixed using either irradiated duck offal or untreated ground chicken. At whelping, 10 females each will be provided one of the two diets through weaning of their kits. During the 6 week period, kit survivability and growth will be assessed.

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COCCIDIOSIS IN PACIFIC NORTHWEST MINK

Introduction

Coccidiosis is a disease of most animals, and is caused by microscopic protozoan parasites that can produce diarrhea and death. The parasite lives and reproduces in the cells of the intestine. When large numbers of intestinal cells are destroyed, animals lose the ability to absorb water and nutrients, resulting in diarrhea and weight loss. In mink, there are five different species of coccidia, including one species that infects the liver. Historically, coccidiosis was a common disease in mink when they were raised on the ground. However, since mink are now raised in elevated wire bottom pens with improved sanitation, the occurrence of coccidiosis in mink has been greatly reduced.

Coccidia are most common in young, growing animals because older animals often develop immunity. Studies in Wisconsin have shown that the 54% of the mink samples were positive for coccidia. Studies with drugs have been done to determine the effectiveness of coccidiostatic drugs against coccidiosis. Of eight different compounds tested, four of them were effective. These included amprolium, sulfadimethoxine, lasalocid and monensin. Lasalocid and monensin are commonly used in the cattle and sheep industries to prevent coccidiosis and increase feed efficiency.

Purpose of this Study

The purpose of this study was to determine the prevalence and intensity of coccidia in mink in the Pacific Northwest.

Methods

A total of 290 fecal samples were collected at random from 12 different mink ranches in Oregon (n=10), Washington (n=1), and Idaho (n=1). Fecal samples were collected in individual plastic bags, placed on ice, and transported to Washington State University where they were evaluated for parasites. All samples were examined with a sugar fecal flotation technique and results were recorded as number of parasites per gram of feces.

Results

A total of 72 of the 290 samples were positive for coccidia, including Oregon (53 of 230 samples - 23%), Washington (16 of 27 samples - 59%, and Idaho (3 of 33 - 9%).

Eleven of the 12 farms had coccidia present in one or more samples. Numbers of parasites ranged from 15 per gram of feces to several thousand per gram of feces. Three different species of coccidia were identified in the samples. These included: *Eimeria vison, Eimeria laidlawi, and Eimeria mustelae.*

Significance of Results

Coccidia were present in 24.8% of the samples collected indicating the potential for clinical coccidiosis to occur. It has been suggested that coccidiosis may be part of a syndrome in growing kits called the "June Blues" which manifests itself with diarrhea, weight loss, and some mortality. Diagnosis and potential treatment would be important in cases where coccidiosis is present.

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Table 1. Prevalence of Coccidia on Mink Farms in the Pacific Northwest - 2004

Mink Ranch	# positive/ # examined	% positive	# of coccidia species
1	2/23	8.6%	2
2	4/22	18.1%	1
3	1/21	4.7%	1
4	6/21	28.5%	3
5	4/20	20.0%	3
6	9/19	47.3%	4
7	0/19	0%	0
8	5/22	22.7%	3
9	15/16	93.7%	3
10	7/25	28.0%	2
11	3/33	9.1%	3
12	16/27	59.3%	3
TOTAL	72/290	24.8%	3

INTESTINAL COCCIDIOSIS IN MINK

Along with the results of his survey, Dr. Gorham has provided a useful description of the symptoms of coccidiosis.

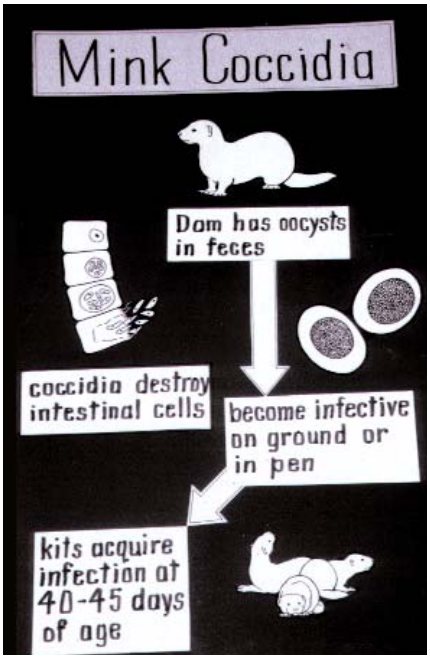
Coccidiosis is a disease caused by a minute protozoan parasite. The disease infects both wild and ranch-raised mink and can result in mortality among kits. It is most common in summer and early fall and appears to be equally prevalent in dry and wet climates. Although coccidiosis usually affects mink raised on the ground or in pens with board flooring, it may affect those kept on wire floors. Before wire bottom pens came into general use, losses among mink from coccidiosis were extensive.

Cause: The parasite (Eimeria mustelae, E. vison, Isospora bigemina, or I laidlawi) lives in the intestinal tract and single-cell oocysts are passed in the droppings. At this stage, the oocyst is not infective for other mink but, provided with warmth and moisture, the oocysts will develop into an infective stage. This development usually takes place in feed and water that has been contaminated with fecal material and the susceptible mink become infected when they swallow this material. The adult may become reinfected in the same manner. Once in the digestive tract, the parasite develops rapidly and liberates great numbers of tiny bodies that invade the cells of the gut wall. These young parasites in turn develop and produce mature oocysts that pass out in the droppings and begin a new life cycle. Infected adults are the carriers of the parasite and are a source of infection for the new kits in May.

Disease Signs: The severity of coccidiosis depends on the extent of infection and the mink's resistance. The signs, therefore, are not always clear-cut and specific. Usually, the first sign noted is the passing of mucous-coated droppings. The appetite may become irregular and impaired. The fur becomes rough, lusterless and frequently faded. Progressive weakness and loss of flesh usually follow. If the disease is associated with distemper or some other malady, losses may be great. Coccidiosis may also be chronic, especially in older mink. It runs its course in from 4 to 10 weeks after which the mink will have built up an immunity.

Control: As yet, there is no remedy that completely controls coccidiosis. If the disease has not gained too great a foothold, good sanitation will help greatly if the rancher can break the life cycle of the parasite. If susceptible mink cannot come in contact with contaminated feed or water, either through prompt removal of droppings or the use of wire bottom pens, the life cycle is interrupted and the possibility of re-infection is reduced. The mere use of wire bottom pens does not alone preclude infection. If the droppings are allowed to accumulate on the wire, little has been gained since the mink still have contact with the parasite-contaminated feces.

Some sulfa drugs such as sulfaquinoxaline are very toxic to mink and should never be used to treat mink for any disease. Prevention is still the best method and maintaining mink under good sanitary conditions is the primary ranch method to prevent losses from coccidiosis.



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