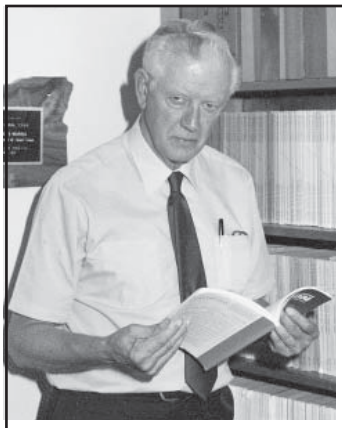
# Fur Animal Research

Published by Mink Farmers' Research Foundation, a Committee of Fur Commission U.S.A

Volume 15, Number 2 June, 2007



You have probably been reading in the newspapers, as I have, about problems with contamination of various brands of pet food. As a dog owner, I of course wanted to be sure my pet was not exposed to such poisons and I was relieved to find she had not been.

The U.S. Food and Drug Administration (FDA) has been investigating this situation since March 16 of this year, when **Menu Foods, Inc.**, recalled dog and cat foods that had been produced at two of their plants. These foods had been sold under a number of trade names and concern was raised when a number of dogs and cats fed the tainted products became ill and died.

The FDA identified the cause of the problem as contamination with melamine, and related compounds and they suggested that wheat gluten and a rice protein concentrate imported from China were the probable carriers of the melamine. It was also suggested that the reason melamine was being added to these feeds was that it contained nitrogen, which, in Kjeldahl analyses that were used in the nutrient evaluation of the feeds, showed up as "crude protein," which added to the price the product would bring.

I'm not suggesting that you would feed pet foods to your mink, but you might perhaps feed some of the pet food ingredients. This has happened with other species and the FDA reports they have evidence that some of these feeds have been used with swine, poultry and fish. The lesson we can take from this situation is to make sure the source of the various feeds that you include in your mink diets is reliable and the product is safe. Some manufacturers have effective safety programs already in place. The U.S. National Renderer's Association, for example, has very effective ways to ensure product quality and safety, so that products rendered in this country will probably be okay (from Caparella, Tina. 2007. Contaminated Pet Food Investigation Expands. Render, June, 2007, pp. 8-9).

On the brighter side, we are beginning to get the beautiful spring weather I was hoping for in our last issue. And if I may be permitted a personal observation: our Oregon State Beaver baseball team is headed towards another College World Series, beating a very good Arizona State team last evening, 12-5.

Kindest regards and best wishes to you all.

J. E. Oldfield

Dr. John Easley, Foundation Secretary, has forwarded me progress reports from the researchers that we provide with funding and I will include these in this newsletter.

# DISTILLERS' DRIED GRAINS AND SOLUBLES IN MINK FEED

We have written before about the likely availability of distillers' dried grains and solubles as the country increases its production of ethanol as a gasoline additive. Dr. Steve Bursian at Michigan State University has been looking at this product as a replacement for wheat middlings in a cereal formula.

The use of "new generation" distiller's dried grains with solubles (DDGS) as a feed ingredient is receiving considerable attention within the livestock industries. Distiller's dried grains with solubles is one of the three co-products produced in the dry mill ethanol plants along with fuel ethanol and carbon dioxide. The production of DDGS is increasing at a rapid rate due, in part, to many states banning methyl tertiary butyl ether (MTBE) as a gasoline oxygenation agent, which has led to an increase in ethanol demand. Currently, the fuel ethanol industry in the U.S. produces about 7.8 million metric tons of DDGS (http://www.usda.gov/oce/forum/speeches/markham.pdf).

Research has shown that DDGS can be a cost-effective partial replacement for corn, soybean meal and inorganic sources of phosphorus in diets of swine and poultry. Forty-five years ago, Schaible and Travis (1961) explored the use of DDGS in mink rations. They conducted a series of trials to determine if DDGS could replace: (1) portions of meat or cereal in mink rations during the growth and furring periods; (2) dried skim milk and liver products in dry pelleted feed during the periods of maintenance of adult mink; (3) fresh liver during reproduction and lactation. Their results indicated that DDGS gave good results during growth and furring when used as a replacement for 5% meat and as a replacement for up to 20% of a commercial cereal component of a typical mink ration. They also found that the product was a satisfactory replacement for dried skim milk and dried liver during the winter, summer and fall maintenance periods, but it could not be used to replace the fresh liver component of a mink ration during breeding, gestation, parturition and lactation.

Because research has indicated that DDGS can be used effectively in mink rations, it was of interest to reassess the applicability of the "new generation" DDGS in mink rations. Today's DDGS is produced in such a way that temperature is more carefully controlled, resulting in enhanced integrity of amino acids and other essential nutrients. We conducted a trial in which DDGS was used to replace the wheat middlings (WM) component of a basal mink ration during the lactation period of mink. Because the protein and fat content of DDGS is greater compared to wheat middlings, we were able to decrease the percentage of relatively expensive high protein/high fat components of the mink diet.

Thirty-five bred females received the diet containing 25% DDGS and 35 bred females received the traditional ranch diet containing 16% WM. The two diets were formulated to provide the percentage of protein and fat appropriate for the time of year. Table 1 provides the composition of the two diets used for the whelping period, which were fed beginning April 15. At whelping, kits from each litter were counted, sexed and weighed. Kits were weighed again at three and six weeks of age as were their dams. At six weeks of age, kits in the DDGS treatment group were switched to the WM diet through seven months of age. Body weights of kits in the DDGS treatment group were significantly less than body weights of the WM kits at birth and at three and six weeks of age (Table 1). Kits whose dams were fed the DDGS diet weighed approximately 5% less than kits from dams fed the WM diet at birth, 11% less at three weeks of age and 21% less at six weeks of age. Because the difference in body weights of kits in the WM and DDGS groups was progressively increasing, kits in the DDGS group were switched to

Table 1. Body weights(g) of mink kits fed diets containing wheat middlings (WM) or dried distiller's grains with solubles (DDGS) through lactationa

| Age | Dietary treatment | |
|---|---|---|
| | **WM** | **DDGS** |
| Birth | 9.8 ± 0.2 | 9.3 ± 0.2* |
| Three weeks | 113 ± 1.5 | 101 ± 1.5* |
| Six weeks | 263 ± 5.1 | 209 ± 5.3* |

[a]*Data are presented as mean ± standard error of the mean. * indicates significantly different from WM at p≤0.05.*

the WM diet through seven months of age to determine if the difference in body weight gain between the two groups would diminish. Table 3 indicates that body weight gain of DDGS males was 19% less compared to WM males from birth to six weeks of age, 22% less for DDGS females and 21% less for male and female DDGS kits combined. However, when the DDGS kits were switched to the WM diet at six weeks of age, body weight gain of both treatment groups was comparable over the seven month exposure period (Table 2) indicating that the reduced growth caused by feeding the DDGS diet was not permanent.

The results of this trial suggest that DDGS can be used as an inexpensive cereal component of the mink diet, reducing the cost of feed by as much as 50% (Table 1). However, further work needs to be done to determine the optimal dietary concentration of the ingredient at the different periods of the mink year so that reproduction and growth are not adversely affected.

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Table 2. Composition of diets containing wheat middlings (WM) or dreid distiller's grains with solubles (DDGS) fed during whelping

| | % Protein | % Fat | % Moistuure | Diet | % Protein | % Fat | % Moisture |
|---|---|---|---|---|---|---|---|
| WM | 15% | 3% | 5% | 16.0% | 2.4% | 0.5% | 0.8% |
| Chkn | 19% | 9% | 66% | 26.0% | 4.9% | 2.3% | 17.2% |
| SD Liver | 58% | 12% | 10% | 3.0% | 1.7% | 0.4% | 0.3% |
| SD Eggs | 46% | 31% | 8% | 5.0% | 2.3% | 1.6% | 0.4% |
| Water | | | 100% | 36.0% | 0.0% | 0.0% | 36.0% |
| Fishmeal | 60% | 6% | 8% | 4.0% | 2.4% | 0.2% | 0.3% |
| SB Oil | | 100% | | 4.0% | 0.0% | 4.0% | 0.0% |
| Bld Prtn | 82% | 3% | 8% | 6.0% | 4.9% | 0.2% | 0.5% |
| | | | | 100% | 18.7% | 9.2% | 55.5% |
| | Cost = $0.23/lb | | | DW Basis | 42.0% | 20.7% | |
| | % Protein | % Fat | % Moistuure | Diet | % Protein | % Fat | % Moisture |
| DDGS | 26% | 10% | 13% | 25.0% | 6.5% | 2.5% | 3.1% |
| Chkn | 19% | 9% | 66% | 26.0% | 4.9% | 2.3% | 17.2% |
| SD Liver | 58% | 12% | 10% | 2.0% | 1.2% | 0.2% | 0.2% |
| SD Eggs | 46% | 31% | 8% | 2.0% | 0.9% | 0.6% | 0.2% |
| Water | | | 100% | 36.0% | 0.0% | 0.0% | 36.0% |
| Fishmeal | 60% | 6% | 8% | 2.0% | 1.2% | 0.1% | 0.2% |
| SB Oil | | 100% | | 3.0% | 0.0% | 3.0% | 0.0% |
| Bld Prtn | 82% | 3% | 8% | 4.0% | 3.3% | 0.1% | 0.3% |
| | | | | 100% | 18.0% | 8.9% | 57.1% |
| | Cost = $0.13/lb | | | DW Basis | 42.1% | 20.7% | |

Table 3. Body weight gain (g) of mink kits fed diets containing wheat middlings (WM) or dried distiller's grains with solubles (DDGS)a

| Period of Weight Gain | Males | |
|---|---|---|
| | WM | DDGS |
| Birth to 6 weeks of age | 264 ± 7 | 213 ± 8* |
| Birth to 7 months of age | 1962 ± 48 | 1997 ± 44 |
| | Females | |
| Birth to six weeks of age | 241 ± 7 | 189 ± 7* |
| Birth to 7 months of age | 1225 ± 28 | 1269 ± 28 |
| | All kits | |
| Birth to 6 weeks of age | 253 ± 5 | 201 ± 5* |
| Birth to 7 months of age | 1556 ± 42 | 1656 ± 41 |

[a]Data are presented as mean ± standard error of the mean. * indicates significantly different from WM at p≤0.05. Animals in the WM group were fed that diet from birth through seven months of age while animals in the DDGS group were fed that diet through six weeks of age and then switched to the WM diet through seven months of age.

# PREVALENCE OF COCCIDIA IN NORTHWEST MINK

**Introduction:** Coccidiosis is a disease of most animals, and is caused by microscopic protozoan parasites that can cause prolific 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 coccidian, 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 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%).

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

| Mink Ranch | # positive/ # examined | % positive | number 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% | 2 |
| 6 | 9/19 | 47.3% | 3 |
| 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 |

Eleven of the 12 farms had coccidia present in one or more samples. Numbers of parasites ranged from approximately 15 per gram of feces to several thousand per gram of feces. Three different species of coddidia 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.

**Future Plans:** Future work will include sampling kits prior to and after weaning to determine the effects of weaning stress on the numbers of parasites present. A second objective is to evaluate the efficacy of one or more drug treatments on the development of coccidia in mink and determine the effects if treatment on the growth and quality of mink.

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# TULAREMIA IN MINK

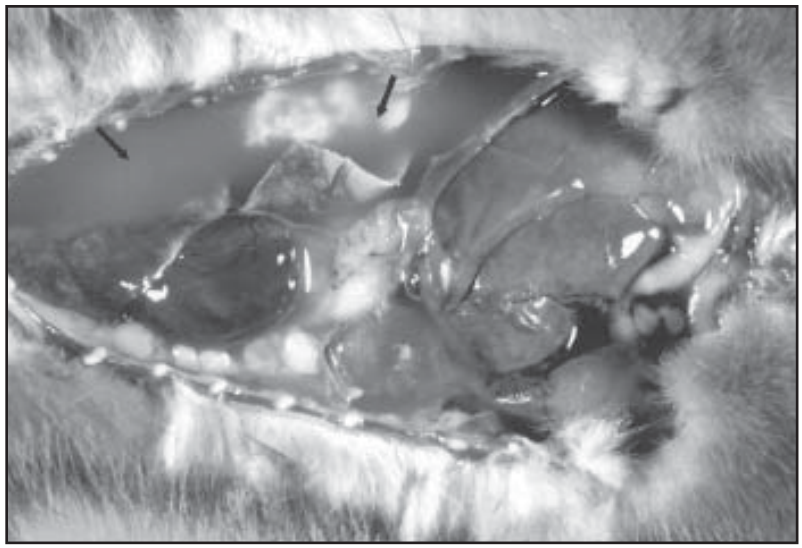
Dr. John Gorham shares some of his early research with mink investigating tularemia:

When I was just getting started in mink disease research, I received a telephone call from an old friend of mine in southern Idaho. Though he has since passed away, I think he would not mind my relating the following incident.

He told me that he was losing mink and asked if he could bring some of the dead ones to Pullman for me to examine. He flew in his own plane and taxied up to my car at the airport which in itself is a bit surprising.

Then he asked if I could autopsy his mink at the airport inasmuch as the weather was "socking in" over the Rocky Mountains and he wanted to fly home that night. Also, the Pullman airport is not Kennedy International!

I hesitated but I have done stupid things before and probably will do them again, so I agreed. He had about a dozen dead mink. We lined them upon the edge of the wing on his plane.



*Small, white spots on the lung of a mink that was fed wild jackrabbits that were carriers of tularemia bacteria.*

I had a jackknife with me and proceeded to open them up, thinking that if his mink had something simple to diagnose such as yellow-fat disease, I could give him a quick diagnosis.

I went from one mink to the next and noticed small white spots, technically called multiple white foci, on the liver, spleen and lungs of each one. After I got to the last mink and my hands were smeared with blood, I asked, "What have you been feeding your mink?"

He named a whole list of things that swim, crawl, walk or fly. As an afterthought he added, "Oh, by the way, I am feeding about 10 percent wild jackrabbits."

My answer was not one of the classics of medicine, but I do recall it. "My friend, your mink have tularemia. You have tularemia. I have tularemia."

He jumped into his plane and flew back to Idaho with this tentative diagnosis, then contacted his veterinarian and started immediate treatment of his mink. I told him to watch out for himself and his caretakers and advised them to see a physician if they felt ill.

I loaded the mink in my car as carefully as I could and took them back to our laboratory that night. In a few days the tularemia diagnosis was confirmed. For the next week or so, I was a bit concerned about the health of all the human contacts but none of us contracted the disease. The outbreak subsided on the mink ranch; either the treatment was successful or the disease terminated without intervention.

This is an illustration that I use frequently when lecturing to a class of veterinary students on how not to conduct an autopsy. A windy airport at sundown is no way!

*Dr. John R. Gorham*
*Pullman, Washington*

# RINGWORM IN RANCH MINK

**Overview:** Ringworm is a fungal disease of the skin of animals caused by a group of fungi known as the Dermatophytes. These fungi affect only the keratinized layers of the skin and hair. The fungus does not invade living tissues. The typical lesions include focal areas of hair loss, stubbled hairs, scaling or crusting of the skin and inflammation (dermatitis). The main sites affected are the head, neck, the feet, hips and base of the tail. Extensive skin lesions may severely damage the pelt of fur animals resulting in significant economic loss from reduced pelt quality. This scenario was experienced on several Ontario mink farms in 2006. The following is a review of ringworm in ranched mink and suggestions on prevention and control.

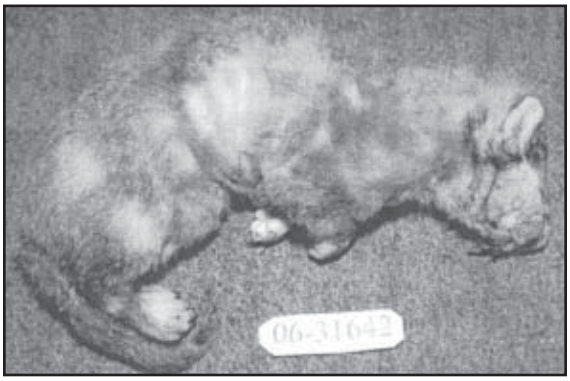



*Figure 1. Mink kit with ringworm. Note the areas of hair loss over the body and the thickened skin around the face and feet.*

**Cause:** Ringworm is the common term used to describe a skin infection caused by a group of dermatophilic (skin-loving) fungi. It is not caused by a worm or a parasite. Several different fungi have been reported to cause ringworm in mink:

1. Microsporum canis is the most frequently reported. Despite the scientific name of M. canis, cats are the natural host for this fungus. In cats, asymptomatic carriers are common, particularly in longhaired varieties. Mink infected with M. canis develop circular areas of hair loss and they usually recover within a few weeks without treatment.

2. Microsporum gypseum is uncommon in commercial mink. It has been reported as a cause of skin infection in dogs. M. gypseum is a geophilic fungus indicating that it survives naturally in soils.

3. Trichophyton mentagrophytes is found in a range of species including household pets, cattle and horses. This is the most pathogenic of this group of fungi and is the variety of fungus identified as the cause of problems in Ontario mink farms in 2006.

Infection is initiated when the surface of the skin has been altered by scratches or abrasions, or if the skin has been in constant contact with damp or moist bedding. The branching hyphae of the fungi colonize in the surface of the skin, the hair follicles and the hair shafts. Hair shafts have an outer keratinized portion called the cuticle. The fungus penetrates the hair cuticle and tunnels extensively through the central portion of the hair shaft, eventually forming round arthrospores within the hair (endothrix) or on its external surface (ectothrix) (see Figure 1). The fungus causes irritation of the skin by excreting toxic substances like trichophytin. The affected skin becomes itchy (pruritic) and often painful. The affected hair shafts fall out leaving hairless areas of skin (alopecia) that may be thickened, corrugated and crusty. Often secondary bacterial infections with common bacteria like Staphylococcus sp. or Streptococcus sp. occur.

Diagnosis of ringworm can be made in several ways. Affected hair shafts plucked from the periphery of the skin lesion can be treated with potassium hydroxide (KOH) to dissolve the cuticle and then examined under a microscope for fungus. Some varieties of fungus will fluoresce using a Wood's Lamp and this technique is used as a screening tool for ringworm in many veterinary clinics. Fungus can be identified by microscopic examination of the skin. However, laboratory culture and identification of the actual type of fungus remains the "gold standard" for diagnosis. Representative affected animals should b submitted to a veterinary diagnostic laboratory or to your veterinarian for diagnosis. The disease can result in too serious an economic

loss for you to guess at the diagnosis.

Animals that are affected at pelting time may have significant damage to the pelt. Affected areas of skin fail to prime properly and are prone to damage and tearing during fleshing and pelt preparation. These animals may be difficult to identify if only the fur side of the pelt is examined. Running your hand over the entire body of the mink prior to pelting may reveal areas of roughening of the skin or scabby areas under the hair. After the pelt has been removed, areas of damage can be easily seen as dark, non-prime areas from the skin surface.

If ringworm is diagnosed on your farm there are several options:

1. Remove affected animals from the farm (this includes the entire litter as all animals will have been exposed).
2. Isolate and treat affected animals. If only a few litters are involved, individuals may be treated. If numerous litters are involved, treatment of the entire herd should be considered.
3. Treat herd with griseofulvin (15 mg/kg body weight given in the feed for a period of 10 days to 3 months depending on disease severity and response). Note that this drug cannot be used in pregnant females or in breeder males within 6 months of breeding as the drug causes birth defects in developing kits and sperm defects in breeding males. Contact your veterinarian for proper advice.
4. Isolate litters and treat individual mink with one of the following miconazole (an antifungal agent), dilute bleach solution (1:10 dilution 2x per week), or lime-sulfur (2-4% solution 2x per week). Each of these treatments involves dipping affected animals or sponging on the solution. Lime-sulfur has an unpleasant odor and stains the animal yellow. Bleach solutions can be irritating and potentially cause damage to eyes. All of these options require that the person administering the treatment wear protective gloves and eye wear. Treatments should continue for several weeks depending on response. Use separate gloves to handle affected animals.
5. Nest boxes and the local environment will be contaminated with arthrospores and vigorous cleaning and sanitation is recommended. Old wooden nest boxes should be burned and replaced with new ones. All nest box bedding (including those in pelting sheds) should be removed and burned. Nest boxes should be vacuumed, physically washed with hot water and soap and then disinfected with an appropriate disinfectant that has antifungal properties such as bleach (1:10 solution). Hair on wire should be removed (burning with a torch).
6. In cats and in cattle, vaccines have been used with variable success. There are no vaccines licensed for use in mink.

**Zoonotic potential:** Ringworm is a zoonitic disease meaning that it can be transmitted from affected mink to people. Farm workers and those working with untanned mink pelts (pelt processing facilities and auction houses) should always practice proper hygiene. Any worker with skin lesions after handling mink should be advised to see their physician.

**Summary:** I have diagnosed ringworm in farmed mink a number of times over the past 30 years. These infections had always involved only a few litters on a farm and were generally mild and self-limiting. The outbreak reported in Oregon in 2003 and those that occurred in Ontario in 2006, all caused by Trichophyton mentagrophytes, caused serious economic loss. These cases reinforce the need for upgraded biosecurity, careful purchasing practices, prompt disease diagnosis (including identification of the type of fungus) and sound health management practices.

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# FEED USE OF A MANNANOLIGOSACCHARIDE TO ENHANCE KIT SURVIVAL AND GROWTH

Enteric pathogens must attach to the mucosal surface of the gut wall to establish themselves in the gastrointestinal tract. Because attachment is often mediated through binding of bacterial lectins to gut receptors containing the sugar D-mannose, inhibition of bacterial attachment can be accomplished by addition of mannose, or similar sugars, to the diet. Because relatively high concentrations of mannose are required to control colonization of pathogenic bacteria, the cost of using pure mannose in commercial animal production is prohibitive, even for short periods of time. However, mannose-based sugars occur naturally, such as in yeast cell walls. Recently, mannanoligosaccharides (MOS), derived from yeast cell walls, have been evaluated as to their ability to bind enteric pathogens.

In vitro tests have shown that MOS binds several strains of E. coli and a number of species of Salmonella and Clostridia. In vivo tests with turkey poults and chicks demonstrated that inclusion of MOS into the diet decreased colonization of several enteric pathogens in the gastrointestinal tract, presumably by adsorbing the bacteria and thus preventing them from adhering to the gut wall.

Because mink kits are particularly sensitive to enteric pathogens, it was of interest to determine if inclusion of a mannanoligosaccharide in the diet of growing mink would be useful in maintaining a healthier balance in the microflora of the gut and thus reduce kit mortality and enhance growth. Therefore, the objective of the present project was to assess the efficacy of a commercial mannanoligosaccharide preparation (Bio-Mos; Alltech, Inc.) in promoting enhanced kit survivability and growth from whelping until pelting.

Sixteen female, natural dark mink and their 102 kits were fed a basal ranch diet with Bio-Mos (Alltech, Inc., Nicholasvile, KY) incorporated at a rate of 6 lb/ton from whelping to weaning of kits at six weeks of age. In addition, 12 female, natural dark mink and their 66 kits were fed the basal ranch diet without Bio-Mos and thus served as the control group. At weaning, the kits were continued on their respective diets throughout the growth period until October 31, 2002.

Survivability of kits at three and six weeks of age was slightly greater in the Bio-Mos group compared to the control group (54% vs. 41%, respectively, at three weeks and 44% vs. 41%, respectively, at six weeks). At the end of the trial, percent survivability in the Bio-Mos group was 41% compared to 38% in the control group. Body weights of the adult females in the control and Bio-Mos groups declined by 17% and 22%, respectively, from whelping to weaning. Birth weights of control kits were 4.1% greater than birth weights of the kits assigned to the Bio-Mos treatment (11.2 g vs. 10.7 g, respectively), but by six weeks of age, the Bio-Mos kits were 5.8% heavier than the control kits (365 g vs. 345 g, respectively). At approximately six months of age, the Bio-Mos females were 5.5% heavier than their control counterparts (1652 g vs. 1566 g, respectively), while the Bio-Mos males were 1.9% heavier than the control males (2122 g vs. 2082 g, respectively).

In conclusion, the incorporation of Bio-Mos, a commercial mannanoligosaccharide preparation, into a basal ranch mink diet at a rate of 6 lb/ton caused a slight enhancement of kit survivability from birth through 6 months of age. Additionally, body weight gain of kits fed the diet containing Bio-Mos was slightly greater compared to kits fed the basal diet without Bio-Mos over the same time period.

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# HORMONAL EFFECTS ON UTERINE IMPLANTATION

The hormones 17ß-estradiol (E2), progesterone (P4) and prolactin (PRL) have been shown to have direct actions on the uterus of many species that are essential for preparing the organ for embryo implantation. Our objectives were to determine the effects of E2, P4 and PRL on uterine luminal and glandular epithelial cell growth in anestrous mink, an animal for which such knowledge is lacking. Mink were treated with E2, P4 or haloperidol (HAL; a dopaminergic antagonist that increases PRL cell heights measured using Image J software (NIH). E2 or P4 increased luminal and glandular cell height relative to controls ($P<0.001$). Glandular cell height was approximately doubled in response to P4 when compared to control or E2 treated mink ($P<0.001$), and E2+P4 increased glandular cell heights, compared to #2 alone ($P<0.001$). Interestingly, HAL caused luminal and glandular cells to be taller than controls ($P<0.05$). When combined, HAL+E2 increased glandular cell height to a greater extent than in response to E2 alone. Glandular cell height was taller in response to HAL+P4 than HAL+E2 ($P<0.001$). Luminal cell height in response to HAL+E2+P4 was greater than HAL, HAL+E2 or HAL+P4. Because blood PRL levels increase throughout the breeding season of mink, we hypothesize that PRL, in combination with E2 and P4, plays an essential role in creating an optimal uterine environment for blastocyst implantation.

**Summary**

These findings suggest to us that E2 (perhaps during the ovarian follicular phase) has greater effects on the luminal epithelium (compared to the glandular epithelium) that serve to prepare the uterine lining for blastocyst implantation. It is most likely that because these mink retained their ovaries, we did not observe an estrogen-priming effect in E2 + P4 treated mink. That is, E2+P4 was not greater than E2 alone on luminal epithelial growth because even low E2 production by the ovaries may have maintained production of the P4 receptor. The slight reduction in glandular cell height in response to E2 + P4 (when compared to P4 alone), may have been the result of P4 induced down regulation of E2 receptors. The greater effect of P4 on glandular epithelia than luminal epithelia is in keeping with the known effects of P4 on uterine secretion. Finally, the increased glandular and luminal epithelial cell heights in response to elevated PRL (HAL-treatment), are of particular interest. In addition to the well-established luteotropic actions of PRL in mink, these findings suggest PRL may have direct actions on the uterus that support implantation. The synergistic actions of E2 + HAL, to increase uterine glandular epithelial cell growth, may represent a requisite action of these hormones that prepares the uterus for implantation. If this is true, then perhaps the short-term increase in E2 secretion, coupled with the direct actions of PRL on the uterus, are in part responsible for the termination of embryonic diapause in this species.

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# PUSSY LUNGS

Whenever Aleutian mink raisers get together, the subject of pussy lungs will eventually come up. A mink farmer in Mt. Angel, Oregon asked me, "What are you doing about pussy lungs?" I said, "Not very much." He answered, "That is what you told me 30 years ago!"

The purpose of this article is to tell you what is known about pussy lungs, technically known as purulent pleuritis (PP).

**Symptoms of Pussy Lungs:** Affected mink show a loss of appetite, rapid breathing and a slight nasal discharge that is often overlooked. In most cases, the mink are found dead. Pussy lungs are diagnosed in 10-20% or more of the dead mink on Aleutian mink farms.

**Bacteriology:** Aerobic (in the presence of air) cultures of the lung and purulent fluid do not result in growth of significant bacteria. Anaerobic cultures (grown in the absence of oxygen) were positive and included large numbers of a variety of different bacteria. Among these bacteria, Fusobacterium spp (including F. necrophorum), Bacteroides spp., and Prevotella spp. were most commonly identified.

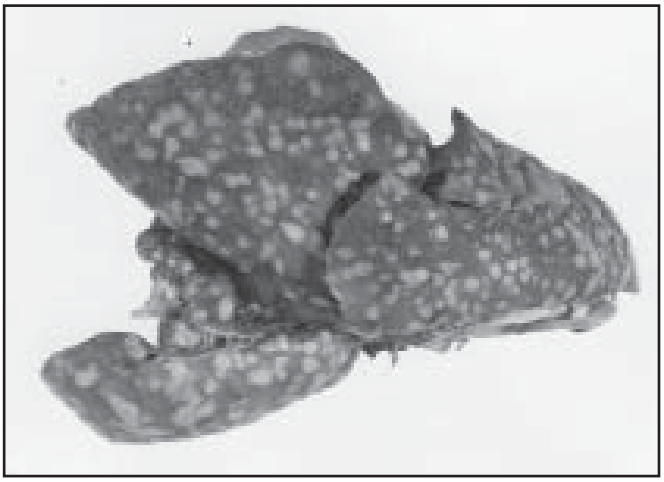
**Route of Infection:** The perplexing aspect of this disease is how the bacteria gain access to the chest cavity. Several ideas have been presented as to how the bacteria enter the chest cavity. (1) Eating small, sharp bones or bone fragments may cause damage and possibly perforate the esophagus as it runs through the thorax; (2) Penetrating trauma into the thoracic cavity (such as a bite wound or needle puncture inoculating bacteria from the skin) and/or (3) Abscesses from the lung burst releasing bacteria into the chest cavity.

We found no route of entry of the bacteria during postmortem examinations of mink sent to Washington State University's College of Veterinary Medicine. No wounds in the chest wall or esophagus and no lung abscesses were observed in PP mink.

**The Occurrence:** Because of its sporadic wide-spread occurrence on farms over a period of time and because the deaths are rarely clustered, PP mink are not usually autopsied. About 5% of the mink submitted for autopsy at Washington State University are diagnosed with PP. Pussy lungs are rarely observed at autopsy of non-Aleutian mink.

**Purulent Pleuritis and the Chediak-Higashi Syndrome**

All Aleutians have the Chediak-Higashi syndrome (C-HS). With the C-HS, the white blood cells can "gobble up" bacteria normally but the small bags of enzymes in the white blood cells fail to function normally.



*Aleutian mink carcass showing pussy lung which contains about 5 ounces of thick, white pussy fluid. Sometimes the fluid shows fibrin (stringy, white strands) and it may appear reddish if blood is present.*

The accompanying figure shows a white blood cell with the bag of enzymes in a cell (arrow). The enzymes kill the bacteria. But these bags containing the enzymes do not break open in the C-HS mink and empty their enzymes onto the bacteria. Normal granules in non-Aleutians release their enzymes. If the enzymes are not released, the white blood cells cannot kill the bacteria. The bacteria are not destroyed; instead, they multiply and produce pussy lungs and/or abscesses.

The prospects look rather poor for finding Aleutian mink that do not have an inherited susceptibility factor. Every mink we have examined (several thousand) with the Aleutian genotype (aa) has also had the abnormalities characteristic of C-HS. Therefore, the gene for coat color and the gene for C-HS are probably either closely linked or the same gene.

If they involve the same gene, we will never be able to separate the coat color and the susceptibility to disease factors. If they are separate genes, the solution to the problem will depend on locating an animal in which the chromosomes have split and crossed, leaving some characteristics behind. While this is a possibility, it will likely be a long and tedious search for an Aleutian mink without the Chediak-Higashi syndrome.

**Vaccination:** As in the case of treatment of PP, the development of vaccine is really not possible because no one can pinpoint the cause. We have autopsied more than 500 mink suspected of having PP and examined them for bacteria that might be the cause. If we could consistently find the same bacteria, a vaccine might be possible. Veterinarians have vaccinated mink with Fusobacterium ssp. but it is difficult to evaluate the results. The vaccinated mink in these field trials must be autopsied along with the unvaccinated control mink. Again, because of the low prevalence and sporadic nature, these field trials are difficult to interpret.

**Treatment:** It is difficult to treat any disease if the cause is not known. The respiratory signs in a single mink are frequently treated with penicillin injections or other antibiotics but the mink invariably die. Long-term or "flushing" herd treatments with antibiotics do not reduce the sporadic losses.

**Summary:** All Aleutian mink have the C-HS. Unfortunately, the beautiful hair coat of this genotype is tied to the gene or genes that make them highly susceptible to pussy lungs, abscesses, urinary tract disease, other bacterial diseases and Aleutian Disease. While Aleutian disease can be eradicated on a farm, Aleutian mink will always be subject to bacterial infections.

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