Fur Animal Research

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The announcement in the recent newsletter of Fur Commission USA that Bob Zimbal is retiring from his job of Treasurer with them prompts me to write about this most dedicated man. I first met Bob years ago, when I served on the Board of the Mink Farmers Research Foundation and Bob was its Treasurer. I learned a great deal about the fur industry from him and the information was always freely given. Bob is the embodiment of volunteerism. No one could be busier than he is, running his several very large ranches, but he always seemed to be able to find time to help the industry. He often lands in Treasurer jobs and it is appropriate to say that he manages association finances as though they were his own. I'm sure you will join me in thanking Bob for all his efforts, and in wishing him and Audrey all the best, in future.

And while we're talking about people, it is time, too, to recognize another industry personality, John Gorham, who has been honored by the American Veterinary Medical Association for his lifetime achievements in veterinary research. His citation reads as follows: "John is a professor of veterinary microbiology and pathology at Washington State University. He retired from the Agricultural Research Service in 1995. He received his DVM degree and an MS degree from WSU and his Ph.D. from the University of Wisconsin. He is a charter member of both the American College of Laboratory Animal Medicine, and the American College of Veterinary Microbiology and an honorary member of the American College of Veterinary Pathology.

Dr. Gorham has authored more than 400 publications and he has traveled to veterinary laboratories over the world as a cooperating scientist and lecturer. In 1974 he led the first veterinary delegation to the Soviet Union. He has received countless awards, including the AVMA's 12th International Veterinary Congress Prize. John is one of the few veterinarians in the world who has a special knowledge of fur animal diseases and he has been inducted into the Fur Industry Hall of Fame. A cell-adapted Aleutian Disease virus type that is used in all mink-raising countries for diagnostic tests has been named after him."

Truly, John Gorham is a uniquely qualified individual and we are fortunate to have him working for the fur industry.

I wish you a profitable pelting season and happy holidays.

J. E. Oldfield

G. R. HARTSOUGH SCHOLARSHIP AWARD WINNER

Jennifer M. White: Originally from Connecticut, Jennifer attended Gettysburg College in Pennsylvania for studies in Environmental Studies and Biology. During her Junior year, she studied abroad in Ecuador. She traveled to diverse places such as the Galapagos islands, alpine volcanoes, coastal desert, and lowland jungle. While in the lowland rainforest, she worked on a research project studying spectacled and dwarf caiman ecology, which was later published with her co-author Dr. Jesus Rivas. After graduating from Gettysburg College she worked for five years as a Field Technician, traveling and working for many different ecological research projects. Projects included non-native plant survey in the Grand Canyon, herbivore interactions in Panama, swift fox ecology in Colorado, herpetological survey in South Carolina, and ocelot ecology in Belize. In the summer of 2005, Jennifer came to Michigan State University to work as a Laboratory Technician in Dr. Kim Scribner's lab with the goal of learning the skills of molecular ecology. She began her Masters program studying bobcat population ecology in the Fall of 2005 in the Department of Fisheries and Wildlife under the direction of Dr. Kim Scribner and Dr. Scott Winterstein. She will be continuing on for her PhD at the University of Washington, Seattle, beginning in 2008.

Jennifer White and bobcat.

DISTILLERS DRIED SOLUBLES FOR MINK

In his finial report on his assessment of distillers' dried solubles as a replacement for wheat middlings in the cereal portion of mink diets, Dr. Steve Bursian suggests that use of this material may result in significant savings in feed costs.

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 US produces about 7.8 million metric tons of DDGS a year. *(http://www.usda.gov/oce/forum/speeches/markam.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,

Table 1. Composition of Diets Containing Wheat Middlings (WM) or Dried Distiller's Grains with Solubles (DDGS) Fed During Whelping

	% Protein	% Fat	% Moisture	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	% Moisture	Diet	% Protein	% Fat	% Moisture
WM	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 2. Body Weights (g) of Mink Kits Fed Diets Containing Wheat Middlings (WM) or Dried Distiller's Grains with Solubles (DDGS) Through Lactation[a]

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.*

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 2). 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 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 3) 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 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 g ± 7g	213g ± 8g*
Birth to 7 months of age	1962g ± 48g	1997 ± 44g
	Females	
Birth to 6 weeks of age	241g ± 7g	189g ± 7g*
Birth to 7 months of age	1225g ± 28g	1269g ± 28g
	All kits	
Birth to 6 weeks of age	253g ± 5g	20g ± 5g*
Birth to 7 months of age	1556g ± 42g	1656g ± 41g

*[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 DGS group were fed that diet through six weeks of age and then switched to the WM diet through seven months of age.*

THE BIG FOUR MINK DISEASES

There are four diseases of mink capable of causing massive mortality over a very short time period on a farm. These include canine distemper, mink virus enteritis, botulism and Pseudomonas (hemorrhagic) pneumonia. No treatment is possible for the first three of these diseases, but vaccines are available to assist in their prevention. A vaccine is also available for hemorrhagic pneumonia but the bacteria that causes this disease is usually responsive to antibiotic therapy (sulfa drugs) and therefore treatment in the face of an outbreak is possible.

At the present time only one company (Schering-Plough Animal Health) has mink vaccines authorized for use in Canada through issuance of a Veterinary Biologics Product License by the Canadian Food Inspection Agency. Distribution of these vaccines in Canada is coordinated through Springbrook Fur Farms, St. Agatha, Ontario. The product line available in Canada is limited compared to what the industry had access to 10 years ago and comparable to the products available in Europe. There are only three products available in Canada: a vaccine specifically against distemper virus, a 3-way vaccine combination containing distemper (CD), mink virus enteritis (MVE) and botulism, and a 4-way vaccine that contains distemper, mink virus enteritis, botulism and Pseudomonas. Although virtually every mink farmer uses these combination products it is important to remember their limitations and understand that your young mink are potentially unprotected against mink virus enteritis and botulism during certain important times of the year.

The purpose of this fact sheet is to remind mink farmers of the basic principles of vaccination, critical periods where vaccine protection is at risk and emphasize the importance of proper transportation, storage, handling and use of these biological products.

Basic facts around vaccines and vaccination:

1. Vaccination is only one of the tools (although an important one) used to reduce the risk of a specific disease occurring on your farm. Vaccination alone can never compensate for inadequate biosecurity and poor management practices.

2. Vaccines are never 100% effective in preventing disease. The general rule is that a properly administered vaccine will provide about 80% efficacy in preventing a disease. In fact, the USDA requirements for quality control testing of commercial batches of vaccine use 80% efficacy as the standard target.

3. Vaccines must be stored at appropriate temperatures, kept out of direct sunlight and handled properly even during the time the product is being administered to the mink, or the efficacy of the vaccine will drop dramatically.

4. Vaccinating multiple animals with dirty equipment can result in disease spread through your farm. However, improper cleaning of equipment and leaving residues of bleach, alcohol or disinfectant on the vaccinating equipment may inactivate the vaccine and destroy its effectiveness. No matter how many years you have been working in the mink industry, it is important to re-read the manufacturer's instructions on proper vaccine handling that accompany the vaccines.

The distemper vaccine is a "modified live" product. This means that the vaccine contains live distemper virus that has been modified so that it is no longer pathogenic for mink but still able to replicate in the animal and stimulate a strong immune response. Proper handling of this type of product is particularly important because if the vaccine virus is accidentally killed or the titre of the vaccine virus reduced because of improper handling it will be incapable of replicating in the mink and proper protection will not develop.

The mink virus enteritis vaccine is a "killed" product. The vaccine virus has been killed and inactivated by heat or chemical means but still retains the important antigenic properties to stimulate an immune response in the mink.

The botulism vaccine is a "toxoid". A toxoid is a modified bacterial toxin that has been rendered nontoxic (commonly with formaldehyde) but retains the ability to stimulate the formation of antitoxins (antibodies), thus producing an active immunity.

The hemorrhagic pneumonia vaccine is a "bacterin". This bacterin is made of killed Pseudomonas bacteria supplemented with an adjuvant (a substance that enhances the immune response to the vaccine).

How does the body react to the vaccine?

An antigen is a molecule or molecules whose shape and structure triggers an immune response in an animal. Infectious agents such as viruses or bacteria, contain numerous components that may be antigenic. Many of these are proteins associated with the capsule or surface of the agent. A vaccine contains this important antigenic material that is specific to the particular disease-causing agent.

After vaccination, the mink's immune system first recognizes these antigens as being foreign to the body and then it reacts by producing antigen-specific antibodies. Antibodies are special proteins formed by blood lymphocytes that bind to antigens and assist in protecting the animal against infection.

Initial protection against the disease begins to develop within a few days after administering the vaccine and certain classes of antibodies are produced by about day 7 post-vaccination. Ideally, a second (booster vaccine) would be given approximately 10-14 days after the first to stimulate a robust secondary immune response that would provide protection lasting a year or longer. Unfortunately in the mink industry, booster vaccinations are rarely given, mainly because of the limited product lines available and the difficulty in handling and vaccinating uncooperative mink.

What is maternal immunity & why is it important?

If the female mink has been properly vaccinated the previous summer and has high levels of circulating antibodies, these will be passed on to the kits through the placenta during pregnancy. The antibodies are transferred from the mother's blood to the kit's blood, a process called "passive transfer" because those antibodies were never actively formed by the kit's immune system (the kits have not yet been vaccinated or exposed to the disease agent). These antibodies will provide protection against that specific disease for several weeks after the kit is born, but this protection will decrease with time.

Maternal protection against distemper lasts approximately 10-12 weeks. Protection against mink virus enteritis lasts approximately 6-8 weeks. There is not good information on maternal protection against botulism but it likely is very short and kits are likely susceptible as soon as they are on solid food. If a vaccine is given to a kit before the maternal protection wanes, the passive, maternal antibody in the kit's blood may render the vaccine ineffective. So in order to get a vigorous and effective immune response in the kit, timing of the vaccine is important so that it is given as soon as possible after the maternal immunity wanes.

The use of a single dose of the 3- or 4-way vaccines then holds an inherent risk for the mink farm. Most farmers vaccinate one time when the kits are 10-12 weeks old and use the 3-way or 4-way product. Waiting until kits reach 10-12 weeks of age allows the maternal protection against distemper virus to decrease sufficiently to ensure the kit's immune system will be properly stimulated. The problem is that MVE maternal protection was gone much earlier at around 6-8 weeks of age and the kits are susceptible to botulism at around the same time.

Vaccinating once with the 3 or 4-way products then leaves young mink at risk to MVE and botulism from about 8 weeks of age until they are vaccinated at 10-12 weeks. The best method to reduce this risk would be to use the 3- or 4-way product at 8 weeks of age followed by a booster at 12 weeks of age with the same product or with the distemper-only product. This is rarely done because of logistics, costs of vaccine and labour and the difficulty in handling fractious mink. Biosecurity and the best food quality are critical during this period to reduce the risk of kits coming in contact with MVE virus or encountering botulism toxin in the feed.

Quick review of the big four diseases:
Canine Distemper

Cause: virus in family paramyxoviridae
Properties of the virus: CDV is not a hardy virus and is killed by most disinfectants and direct sunlight. It survives for only a short period of time outside of the mink.

Host range: Very wide including all canids (dogs, foxes, coyotes, wolves), raccoons, other mustelids (skunk, wild mink, weasels, etc.). The virus can even infect bears and certain species of big cats. Distemper is a very common disease in raccoons and skunk and not uncommon in foxes so there are many wildlife species that could introduce the virus to a mink farm. Biosecurity and pest control is critical in keeping the virus out.

Transmission: The virus must be introduced to the farm, usually by purchase of mink incubating the disease or wild animals or dogs gaining access to the farm. The incubation period (time from infection to clinical disease) is about 9-14 days. Transmission is by direct contact with infected body secretions (usually respiratory) or by the farmer transferring infected material from animal to animal on gloves or equipment.

Disease: A highly contagious viral disease. Clinical signs include nasal or ocular discharge, loss of appetite, laboured breathing, thickening and crustiness of the skin on the nose and feet, neurological signs (screaming fits) or a combination of these. Treatment/control: There is no treatment for this disease other than providing supportive care. Obtaining a prompt and accurate diagnosis is essential. Antibiotics may help prevent secondary bacterial infections but will not treat CD. Vaccination in the face of an outbreak may help control spread on a farm. Prevention is good biosecurity (wildlife and dog exclusion) and a consistent vaccination program.

Mink Virus Enteritis:

Cause: Virus in family parvoviridae, closely related to feline panleukopenia virus (cat distemper) Properties of the virus: Parvoviruses are extremely resistant viruses that can survive for days to months in manure and organic material. The virus remains viable for long periods in frozen material. Clean up requires careful physical cleaning and strong disinfectants with antiviral properties like formalin or strong bleach.

Host range: Host range includes mink, raccoons, cats, blue fox. Biosecurity and pest control is critical in keeping the virus out.

Transmission: The virus must be introduced to the farm, usually by purchase of mink incubating the disease or recovered mink carrying the virus, or by infected cats or raccoons gaining access to the farm. The virus can also be carried onto the farm by flies or on the farmer's boots or clothing or other objects contaminated with virus. The incubation period is about 4-7 days. Transmission is by ingestion of material contaminated with virus. Virus is shed via feces of infected mink.

Disease: MVE is a highly contagious viral disease. Clinical signs include loss of appetite and severe diarrhea (often gray or pink with casts of intestinal lining cells and some blood streaking). Affected mink may vomit and dehydration is often severe.

Treatment/control: There is no treatment for this disease other than providing supportive care including keeping fresh feed and water available. Obtaining a prompt and accurate diagnosis is essential. Antibiotics like neomycin may help prevent secondary bacterial infections but will not treat MVE. Vaccination in the face of an outbreak may help control spread on a farm. Prevention is by good biosecurity (including careful purchase policy for new mink and wildlife and cat exclusion) and a consistent vaccination program.

Botulism

Cause: Preformed botulinum toxin (usually Type C, rarely others) in feed.

Properties of the toxin: Botulism toxin is produced by the spore-forming bacterium Clostridium botulinum. The spores of this bacterium may be present in many locations including the soil and intestinal contents of domestic animals including chickens. Under certain conditions that include optimal warm temperatures, an anaerobic environment, proper organic substrate, etc., toxin production occurs. Toxins may be present in blocks of frozen feed prior to the farmer purchasing it or it may develop in feed that is improperly stored or handled while on the farm. There is no method to detect the toxin prior to feeding the product. Botulinum toxin is destroyed by heat but survives freezing.

Host range: Botulinum toxin is the single most poisonous substance known. Mink are very susceptible, foxes and dogs more resistant. Botulism is a common disease of wild waterfowl and is an increasingly more important disease in broiler chicken and turkey production. I have seen a serious outbreak on a mink farm associated with feeding cull chickens from broiler farms even though the poultry operation did not recognize they had a disease problem in the chickens.

Transmission: Ingestion of feed containing preformed toxin. There is no mink to mink transmission.

Disease: The toxin blocks the transmission of nerve impulses to muscles and the mink develop rapid, progressive paralysis usually starting in the hind end and moving forward to the head and neck. Mink eventually die due to suffocation as the muscles controlling the diaphragm become paralyzed. Mink are affected within 12-24 hr after ingesting toxin. Mortality may be extensive.

Treatment/control: There is no treatment for this disease other than providing supportive care and keeping fresh feed and water available. Suspect feed should be removed immediately (including scraping left over feed from the wire) and a fresh source of feed obtained. This decision may be difficult as it is often not possible to identify which component of the diet was the source of toxin. It may have been a single contaminated block of frozen product.

Obtaining a prompt and accurate diagnosis is important, but the above control measures need to be done immediately as the diagnosis will take several days as it involves the diagnostic lab performing mouse inoculation and protection tests. Getting sick mink back on feed as soon as possible is important but often difficult. Tempting mink with fresh uncooked liver, vitamin B-complex injections or steroid treatment using dexamethasone are sometimes helpful.

Preventative vaccination is imperative. Further, farmers should make every effort to obtain quality feed and maintain good feed storage and handling techniques on the farm. Remember that young mink are susceptible to botulism as soon as they start on solid feed and will be unprotected until they are vaccinated at 10-12 weeks of age.

Pseudomonas (Hemorrhagic) pneumonia:

Cause: The bacterium Pseudomonas aeruginosa is a

gram-negative, water-loving bacteria.
Properties of the bacteria: Pseudomonas is a common bacterium often found in the nasal cavity of mink and other places like water lines. Although certain serotypes of this bacteria cause disease in mink, there usually is some predisposing stress that triggers disease. Outbreaks can occur at any time of the year but most commonly occur in the early fall when the stress of furring-up is combined with cold nights and warm days. The bacterium is susceptible to good hygiene and most disinfectants.

Transmission: Transmission is from pen to pen by infective nasal/respiratory secretions, or through drinking water that has been contaminated with these same secretions. There is usually a distinct pattern to the disease as it moves down a shed row from animal to animal. Spread of the disease via waterlines is less a problem now that pressurized nipple drinkers are in standard use. Pseudomonas can also be spread widely in a shed by aerosol if high-pressure washers are used in the cleanup.
Disease: Affected mink are usually found dead with a frothy bloody discharge from the nose or plugging the wind pipe. On necropsy the lungs are dark red, very firm and have a liver-like consistency. One or several lung lobes may be involved. The disease must be differentiated from E. coli pneumonia as treatment and control measures are different for each disease, so obtaining a prompt and accurate diagnosis is important.

Treatment/control: It is possible to vaccinate against hemorrhagic pneumonia in the face of an outbreak ,vaccinating a "buffer zone" around the affected portion of a shed or segregating an entire shed. Obtaining a proper antibiotic sensitivity for the bacteria is important. Most strains are susceptible to sulfa drugs but these are potentially toxic so it is important to determine if other drugs are useful.

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June, 2007

LEAD POISONING

A number of mink ranchers in the United States and Canada have had severe losses following the use of red lead paints on metal surfaces or lead paints on pens and nest boxes. Weathering of this equipment doe not alter the toxic properties of lead. There is no direct evidence as far as mink are concerned but the drinking of acid water that has been carried in lead pipes might conceivably induce lead poisoning. Inasmuch as lead sprays, especially those containing lead arsenate, have caused heavy mortality in cattle, such sprays should not contact mink food.

Symptoms. Experimental cases of acute lead poisoning were produced by placing mink on wire immediately after it was painted with red lead in oil. On the following day, the mink appeared normal but on the third day, they were sluggish and showed no interest in their food. These early symptoms were followed by muscular incoordination, stiffness (as evidenced by a stilted gait), trembling, complete loss of appetite, dehydration (removal of water from the body) and muco-purulent discharge around the eyes. Five to seven days after the test exposure, the mink showed terminal convulsions and died.

Chronic lead poisoning was experimentally produced by placing mink on red lead treated wire that was thoroughly dry. Some of the wire-bottom pens were painted as long as two months before the test animals were placed in them. No characteristic symptoms were recorded. The animals exhibited only a gradual loss of weight with death occurring in 25-40 days.

Necropsy Findings and Diagnosis. There are no characteristic changes that point to a definite diagnosis. Laboratory study of blood smears reveals changes in the red blood cells. The best evidence is the demonstration of increased amounts of lead in the liver and blood. In the absence of these procedures, one must rely on a history of the mink being exposed to lead for a diagnosis.

Treatment. As soon as a diagnosis of lead poisoning is made, the animals should be removed from the painted pens as quickly as possible. While the mink are in these pens, they are constantly taking in a small amount of lead each day by chewing on the wire or other painted equipment. Since it is a cumulative poison, a sufficient number of small doses will prove toxic.

Although we have had no opportunity to treat the condition, consideration should be given to the use of chelating agents such as the calcium complex of ethylenediamine tetra-acetic acid (CaEDTA) to form a non-ionizable lead complex which is excreted. Careful prevention of lead exposure is the best control measure.

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TESTING FOR ALEUTIAN DISEASE

Most mink farmers know that the iodine agglutination test (IAT) is a non-specific test. The test is positive (turns cloudy on the glass plate) when the antibody level in mink serum is markedly increased. Some diseases such as avian tuberculosis, abscesses and probably some other disease conditions may give a positive test. However, but it was the only test we had the AD until Cho and Ingram at the University of Guelph reported on their counter immoelectrophoresis (CEP) test in 1972.

The CEP Test. This is a procedure for specific Aleutian Disease (AD) antibody. It can indicate that a positive mink is infected with AD. The CEP test must be performed at a laboratory on blood samples sent by the mink farmer. It is currently the standard diagnostic test for AD.

Millions of mink have been tested in every mink raising country of the world. An AD control program designed by Dr. Mogens Hansen of the Danish Fur Breeders Association was very successful in controlling AD in Denmark.

Currently only one laboratory in the United States is testing CEP submitted mink blood samples.

Blue Cross Animal Hospital
Dr. Blau
401 North Miller Ave.
Burley, Idaho 83318
(208) 678-5553

The Lateral Flow Test. A new test that was first used to detect AD in ferrets was adapted as an AD test in mink by Drs. Easley and Hildebrandt. Like the CEP test, it is specific for AD antibody and does not cross react with other mink diseases, which means that a positive test indicates that the mink is infected with AD. The new test employs the ELISA technique and is called the Lateral Flow Test.

It has a big advantage over the CEP test because the Lateral Flow Test can be done on the farm "penside" by the rancher. It can be used on urine or blood obtained by toe nail clipping. The collected samples do not have to be sent to a laboratory for testing.

A northwestern rancher who is very pleased with the Lateral Flow Test said the urine collection is easy and it takes about one half the time to obtain urine as the time it takes to toe bleed. The results of the Lateral Flow Test can be obtained in between 10 and 30 minutes.

At the present time, there is ongoing research to compare the sensitivity of the CEP test and the Lateral Flow Test to detect AD antibodies.

The material given in this article is very limited. Interested mink farmers should contact:

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