Fur Animal Research

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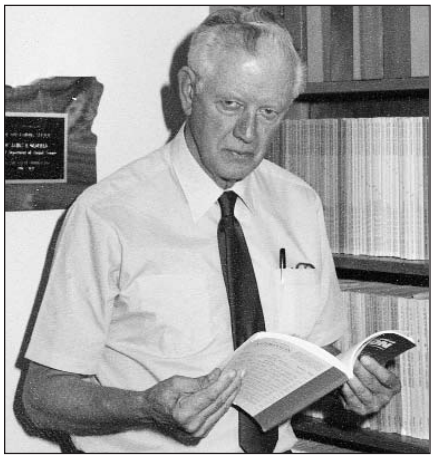

Since the last issue of this newsletter, your Mink Farmers' Research Foundation (MFRF) has held its annual meeting and we will report on that, among other things, in this issue. For a number of years now we have tried to move these meetings around the country to sites where mink research is being conducted. This year we met at Michigan State University, in East Lansing, where much of our best nutrition research is done. Dr. Steve Bursian, who leads the mink research program at MSU was a wonderful host and we were delighted while there to be able to visit with Dr. Dick Aulerich, who headed MSU's mink research for many years. We were also pleased to have the Head of the Animal Science Department. Dr. Karen Plaut, sat in on our Board meeting. She told us what mink research has meant to her Department and expressed pleasure that the MFRF was able to continue funding it. One of the highlights of our meetings has been consideration of research priorities for mink. The Board spent some time updating these and the result is included in this newsletter. As always, your comments and/or questions concerning them, are encouraged.

The word is that the May fur sale sponsored by American Legend and held at Whistler, British Columbia, drew a large number of buyers and was considered a great success. This is good news, indeed, and it is wonderful when business can be conducted in such a beautiful setting as mounts Whistler and Blackcomb. I know whereof I speak because we held a family reunion on the occasion of our youngest daughter's 40th birthday at Whistler. I must add that when you reflect that your youngest daughter is over 40, it makes you feel a bit ancient. I promised I would not say how old she is.

On Memorial Day, a flight of military planes on the way to the Corvallis National Guard Armory passed right over our home, and the noise was deafening. Our dog, George, hid under the Japanese maple tree and wouldn't come out. This got me thinking about effects of noise on other animals, especially mink. There used to be concern about planes flying over mink ranches, and Hugh Travis, who was then at Cornell University, did a study on it in Alaska. Predictably, on the first pass, the mink were excited and ran around the cages, but when the passes were repeated, they quickly adapted to the noise and some didn't even leave their nest boxes. So animals can be pretty resilient, and we hope we may soon have our dog back.

We have been pleased to learn that Dr. Anders Skrede, of the Department of Animal Science at the Agricultural University of Norway, will be visiting here during the week of June 13-18. Dr. Skrede is no stranger to the U.S.,, or to Oregon. He spent a sabbatic leave here some years ago, when our Experimental Fur Farm was still operating, to work with Dr. Floyd Stout. Anders is recognized as one of the world's top mink nutritionists, and it w/ill be good to have him back. I send best wishes to you for a most successful mink growing season.

J. E. Oldfield

WASTE DISPOSAL: A PROBLEM IN ALL ANIMAL OPERATIONS

From time to time I include information in these newsletters that has been generated with animal species other than mink. A recent article assembled at Ohio State University, relative to dairy cattle, has some items of interest. The point made is that it can be helpful to reduce the amounts of animal wastes and there are some ways that this can be done. Where mink are concerned, our MFRF President, Bob Zimbal has reported that including highly-digestible items, like cheese and eggs, in the mink diets reduces manure output.

Dr. W. P. Weiss, at Ohio State, has conducted 14 experiments, involving 232 cows fed 55 different diets. He reported that the average amount of manure produced daily by cows on his experiments was 64 kg (140 lb) and ranged from 27-102 kg/day. The greatest part of the cow manure was water (87.5%). Only 33% of the dietary nitrogen and 40% of the phosphorus eaten by the cows was retained in their bodies - the rest was voided in manure and urine where it causes environmental problems.

My thought is two-fold: we should try to assemble for mink the kind of data Dr. Weiss did, for dairy cattle and we should experiment on ways to alter the mink diet so as to lessen the amount of waste and particularly the amounts of nitrogen and phosphorus in those wastes - without lowering the health and productivity of the animals, of course. (from Lundeen, T. 2005. Ingredient choice may influence volume of feces, urine produced. Feedstuffs, Jan. 10, 2005. p. 8).

IMPROVING MINK WELFARE

There has been some confusion about what practice will improve mink welfare and which will not. The Council of Europe recommended allowing swimming and providing group housing as means to accomplish this, but in test trials neither has been shown to be effective. Research in Denmark looked at four different practices that they thought might improve mink welfare: (1) Selection for confident behavior, (2) putting an empty cage between pairs of mated females, (3) Separating litters at 1-2 weeks after weaning, at 8 weeks of age and (4) furnishing cages with shelves and occupational objects.

The test animals included 100 males and 600 females, which were moved from their home farm to the research center at the end of February. They were housed in a shed with six rows of conventional wire-mesh cages. The "activity" items were either a shelf, or a short tunnel made of wire.

Provision of an empty cage between mated females seemed to be effective. Dams isolated in this way were calmer and ran about less in their cages. Later weaning was questionable, since it apparently allowed more damage to the kits and also more aimless running about by the females. Where "play" items were concerned, provision of a shelf at first calmed the females who apparently crawled on the shelf to get away from the kits. When the kits became large enough to crawl up on the shelf themselves, this advantage was lost. Selection of mink for confidence appeared to help as it reduced fearfulness among the animals. (from Jeppesen, L.L. 2004. Mink welfare improved by combined implementation of several small initiatives. Proceedings VIII International Congress in Fur Animal Production. S. Hertogenbosch, The Netherlands, 15-18 September, 2004).

ROLE OF PROLACTIN IN MINK REPRODUCTION

Prolactin (PRL) is considered to be the major hormone responsible for terminating embryonic diapause in mink. Because of the loss of viable embryos during diapause, a better understanding of the role of PRL-signaling in the mink uterus, should result in methods to shorten or eliminate diapause and increase litter sizes. Therefore, beginning in early March, mink were treated with melatonin (MEL) to inhibit PRL secretion, haloperidol (HAL) to increase PRL secretion, and as controls. The animals were sacrificed in early August, the uteri collected and RNA extracted and subjected to RT-PCR. PRL mRNA abundance did not differ between the uteri of control or HAL-treated mink, but surprisingly was almost non-detectable in MEL-treat-

ed mink. This suggests that MEL acts either directly on the uterus to inhibit PRL expression, or that the MEL-induced reduction in circulating PRL, was necessary to maintain uterine PRL production; that is, PRL directly or indirectly stimulates PRL gene expression in the mink uterus. Uterine PRL-L-R mRNA abundance was greater (although not statistically different) in MEL-treated mink, and much less in HAL-treated animals. This trend in receptor production strongly supports the hypothesis that PRL down regulates its receptor in the uterus, at least in response to very high PRL levels. These data clearly demonstrate the production of PRL and its receptor by the mink uterus, and that the expression of both genes are influenced by PRL. It is our hypothesis that PRL (a): stimulates uterine glandular secretions, and (b): down regulates the expression of insulin-like growth factor binding protein-5 (IGFBP-5), which results in greater free concentrations, and activity of IGF-I within the uterine lumen. Both actions, we feel, are important to promote blastocyst activation and subsequent implantation. In an effort to test our hypotheses, future studies, utilizing in situ hybridization and RT-PCR for uterine IGFBP-5, PRL and PRL-R mRNA expression will be conducted to determine, among other things, if PRL decreases IGFBP-5 as a prerequisite to blastocyst implantation.

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(from research funded by MFRF)

G.R. HARTSOUGH AWARD WINNERS: 2005

We are pleased this year to have been able to offer two G.R. Hartsough Memorial awards at Michigan State University. The winners are Tim Hiller and Sarah Hamer and brief biographies of the two of them follow. The Hartsough Memorials offer a means of remembering Dr. Hartsough and all he did for the fur industry, as well as accomplishing research that may provide industry benefits.

Tim Hiller

Having been raised on a family farm near McClelland, Iowa, Tim Hiller naturally took to trapping, hunting, and fishing. Tim started trapping at about age seven with his grandfather. Soon after, he bought his first trap with the muskrat being his first species. Since then, Tim has predator-called and trapped in four states, capturing many different species of furbearers for scientific research and damage control purposes. Tim graduated from Iowa State University in 1995 with a B.S. in fisheries and wildlife biology, and soon after was employed by Story County Conservation as a technician. Tim graduated from Oklahoma State University in 2004 with an M.S. in wildlife ecology, where he studied the nocturnal ecology of northern bobwhites under Dr. Fred Guthery. Currently, Tim is pursuing a Ph.D. in wildlife under Dr. Henry (Rique) Campa III, studying some aspects of landscape ecology of white-tailed deer. Tim is a member of the National Trappers Association, Fur Takers of America, Iowa Trappers Association, and Michigan Trappers Association. Public education is very important to Tim, so he gives presentations and has introduced many people to the trapping of furbearers.

Sarah Hamer

Sarah Yaremych Hamer is in her second year of a doctoral degree program in the Department of Fisheries and Wildlife, and the Program in Ecology, Evolutionary Biology, and Behavior at Michigan State University. Sarah grew up in suburban Chicago, and earned both her bachelors and masters degrees in Natural Resources and Environmental Science from the University of Illinois at Champaign-Urbana. At MSU, Sarah is part of a new specialization in Fish and Wildlife Disease Ecology and Conservation Medicine, and has initiated an ecological study investigating the emergence of Lyme disease in Michigan. Sarah is interested in the mechanisms of invasion and establishment of tick vectors and the Lyme disease pathogen, and the risk of disease that these species pose to canines and humans throughout Michigan. The differential roles of vertebrate hosts within the system, including mice, chipmunks, raccoons, opossums, skunks, rabbits, squirrels and many avian species, are being defined through trapping, collections of parasitizing ticks and blood samples, and diagnosis of infection status.

AMINO ACIDS IN MINK DIETS

We have learned, over the years, that the level of protein in a diet for mink is not as important an index for performance as the levels of the various amino acids. Danish scientists have compared various mixtures of amino acids in diets for mink during the fall furring period. Three groups of 50 male mink each were used. They were each caged together with a female in a 1-row, open shed with six cages per section. The experiment took place during the furring season, from September 4th until pelting. The diets were formulated, amino-acid wise, to equal the accepted (in Denmark) mink normal diet (N) the amino acid content of the mink body + fur (M) and the normal cat diet (C). Amino acid contents of these three diets are listed in Table 1.

Raw material	Mink norm (N)	Mink body + pelt (M)	Cat norm (C)
Fish offal	-	-	48.40
Poultry offal	29.94	-	-
Barley, heat treated	7.99	8.80	7.94
Wheat, heat treated	7.99	8.80	7.94
Swine pulp	6.00	3.224	8.00
Fish meal	3.48	-	-
Haemoglobin meal	0.69	-	0.05
Wheat gluten	-	-	0.36
Soya bean meal, (Protao)	2.40	3.50	1.10
Feather meal	0.73	-	0.22
Meat & bone meal	3.00	-	2.00
Potato protein	0.90	0.56	0.53
Corn gluten	0.87	-	2.00
Slaughter offal	-	47.08	1.15
Soya bean oil	4.27	6.90	6.78
Swine fat	2.13	3.45	3.39
Wheat bran	1.00	1.00	1.00
Vitamin/mineral premix	0.25	0.25	0.25
Toasted soya beans	0.30	2.63	-
dl-Methionin	0.17	0.08	0.08
l-Threonin	-	0.11	0.16
l-Cystine	-	0.05	0.17
l-Tryptophan	-	-	0.03
Water	27.9	13.56	8.45

Table 1. Feed composition of the three test diets: Mink norm (N), Mink body + pelt (M), and Cat norm (C).

Groups C and M had live weight gains of 660 and 626 grams, which were significantly greater than those in group N (544 grams). The same groups, C and M, had significantly longer pelts than those in group N, however, there were no differences in pelt quality among the three groups. The investigators concluded that amino acid profiles that differed from the presently-accepted norm for mink might give better performance results. They estimated that 22% of the diet energy from protein was sufficient during the furring period. (from Sandbol, P., T.N. Clausen and C. Hejlesen. 2003. Amino acid profiles in the furring period of mink. N.J.F. Seminar no. 354. Lillehammer, Norway. Oct. 8-10, 2003.)

YELLOW FAT DISEASE IN PIGS

Sometimes you do not have to be too bright to make a reasonably good discovery. One time I was on a mink farm where the mink had an outbreak of yellow fat disease (steatitis) and the farmer told me that when he took his pigs for slaughter they were condemned because the fat was yellow and the pork smelled fishy. I asked him what he was feeding his pigs. He said, "Oh, I do not feed them very much. They just pick up what the mink drop under their pens and that's what they eat." Well, it was obvious that what they had was an outbreak of yellow fat disease so I went back to Pullman and my colleagues and I produced the disease by feeding high levels of fish and prevented it with vitamin E.

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THE ORIGIN OF 'MAD COW DISEASE'

Transmissible Spongiform Encephalopathies (TSE's) have been found in a wide variety of mammals, including mink. The most widely-publicized of these, because of its impacts on the world's food supply is bovine spongiform encephalopathy (BSE), commonly called "mad cow disease". There have been questions about the origin of these diseases, such as "Why did BSE break out in the mid-1980's?" and "Why did it occur in the United Kingdom?"

It is generally considered that cattle became infected by eating dietary additives made from the carcasses of infected animals and the timing of the outbreak in Great Britain seems to coincide with changes in the system of rendering carcass materials. The process of liquefying carcass materials into water-soluble and fat-soluble components was replaced by continuous movement of carcasses through the rendering equipment, which could result in incomplete exposure to heat, and a final extraction of the materials with hydrocarbon solvents under steam was eliminated. It is still uncertain whether the original infected feed came from the remains of sheep or cattle, but some believe it came from sheep infected with scrapie. (from American Scientist 92:335-338, 2004.)

THE EARLY HISTORY OF ALEUTIAN DISEASE

From the 1940's until the 1960's, Aleutian disease (AD) was an enigma to the mink farmer and researchers alike. We did not know what caused the disease, how AD was spread, or how to control it.

A historical survey of AD (Aleutian Disease) sheds considerable light on the rapidity of its spread throughout the United States and foreign countries. Prior to 1940, in the early years of the mink industry, the ranches were small and relatively inefficient. Mink were trapped in the area surrounding the ranch or occasionally were brought from as far away as Alaska or Labrador. They were considered to be what is termed a "standard dark" or "wild type" mink. The problems associated with a proper diet and the devastating outbreaks of distemper and botulism were the ranchers' main concern. Thus, one cannot say for certain whether an insidious disease such as AD was present in these first ranch-raised dark mink. However, if it was present, it smoldered along unnoticed or perhaps was confused with some other malady.

The first ranchers, not realizing the possibility of commercially important mutations, promptly disposed of "off-colored" mink. In 1941, an astute rancher near Astoria, Oregon, saved for breeding a gun metal-colored mink that was noticed in a litter of standard dark mink.

The color phase was called "Aleutian" after the Aleutian fox which has a similar pelt color. The gene, which was inherited as an autosomal recessive trait, was designated by the symbol "a". By crossing Aleutian mink with other mutant mink, several substrains were developed which were collectively called "blue mink." Since the pelts were desirable colors, the mink were extremely valuable and commanded high prices. There was a brisk sale of breeding stock and most of the industry geared itself for production of these mutants. In the early forties, Aleutian mink were sold to ranches in various parts of the country. Soon ranchers began reporting losses due to a condition seemingly confined to "blue mink."

Mink ranchers first noticed the condition when their "blue mink" lost weight, although there was usually no noticeable drop in food consumption. Faulty digestion was manifested by cereals and/or other undigested foodstuffs in the feces. Dark tarry droppings were occasionally observed. Affected mink drank a lot of water. About 10 to 20 percent of those visibly sick bled at the mouth. Ragged ulcers at the gingival border, usually involving the teeth, bled when the mink were handled. Ranchers called them "bleeders." The mink were anemic as evidenced by pale mucous membranes and foot pads. They became progressively thinner until death intervened.

Hartsough first observed the gross lesions of AD in 1946. These lesions were recorded in "blue mink" shipped to the Midwest. At first the disease was thought to be limited to the aa genotype inasmuch as AA and Aa mink raised on the same ranch appeared resistant. Some ranchers tried to cope with the malady by crossing back their Aleutian mink to AA stock and/or breeding Aa and Aa. While the aa progeny was somewhat stronger than their original Aleutian breeding stock, they were still more responsive to the disease then Aa or AA mink.

As knowledge of the malady increased, it became known as "kidney disease." Depending on the stage of the disease, the kidneys were enlarged and reddened, or swollen, pale and pitted. Other necropsy findings included initial enlargement of the spleen and lymph nodes, and occasional yellowish brown mottling of the liver.

Kidney from a mink with an advanced case of Aleutian disease. Note the granular appearance.

The Infectious and Contagious Nature

The losses from AD increased each year. Then a series of "vaccine incidents" occurred, which focused attention on the malady, suggesting that the disease might be of an infectious nature. Prior to the advent of chicken-embryo-origin attenuated distemper vaccines, it was common practice to prepare inactivated autogenous tissue vaccines. To make this vaccine, spleens were collected from distemper-infected mink and ground with saline containing 0.3 to 0.5 percent formalin. After the mixture was incubated for a variable length of time - often dependent on how badly the vaccine was needed - it was injected. Following the use of these vaccines, widespread AD losses occurred. In 1949 one rancher lost 500 mink to AD after vaccination for distemper with such a vaccine. Floyd Marsh, a truly colorful mink raiser, said, "They didn't make baskets big enough to pack out my dead mink."

A few years later a Connecticut mink rancher who had AD on his ranch decided to try one of these homemade vaccines against the disease. It was reported that nearly all of the inoculated mink were dead of AD after 6 months!

By 1950, it was apparent to many mink ranchers that AD was contagious. When mink from an affected ranch were brought to a "clean ranch," the disease appeared in the purchased breeding stock as well as in other mink on the ranch. Such a prospective study was made in western Oregon in 1955 by our coworker, Dr. Keith Farrell. The rancher had many desirable mutations which, although he didn't know it, were infected with AD. They were sold to several other ranches and new foci of disease appeared on these ranches. Overseas shipments by many U.S. ranchers of infected mink may have spread the disease to Scandinavia, England, Japan, Canada, West Germany, and elsewhere. However, the possibility exists that Aleutian disease was present but not recorded in these countries prior to the U.S. mink exports. With the history of Aleutian disease in mink, we might speculate about the apparently sudden appearance of this "virus." Two alternatives appear possible: (1) the virus appeared about the same time and in the same area as the Aleutian gene mutation, or the virus might be associated with the Aleutian gene; (2) the virus was present in mink prior to selection of the Aleutian mutant but was not recognized until sufficient numbers of highly responsive aa mink became available in the ranch mink population. In considering the first possibility, it appears extremely unlikely that matching mutations would occur, not only at the same time, but in the same area.

The second proposal is not only more probable but is intellectually more acceptable. When the "blue mink" were shipped from the Oregon ranches, some were infected with AD while others were free of the disease. Infected animals established new foci of disease on clean ranches. On the other hand, when noninfected "blue mink" were sent to ranches where Aleutian disease was present in standard dark herds, the newly purchased "blue mink" served as sentinal animals by succumbing to AD.

There is no direct proof to support either of these possible explanations for the origin of the AD agent. However, if we could locate preserved tissues or tissue sections of standard dark mink with AD lesions in the late thirties, it would prove the latter possibility.

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THE MINK FARMERS' RESEARCH FOUNDATION: RESEARCH PRIORITIES

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AREA OF RESEARCH	DISEASE PROBLEMS INFECTIOUS, METABOLIC	FEEDS/NUTRITION	MANAGEMENT/ ENVIRONMENT PHYSIOLOGY
PRIORITY RATING			
I	**Antigen/Vaccine Studies:** Identify new sources of vaccines and antigens against AD. **Nursing Sickness & Sticky Kits:** Identify physiological basis for Nursing Sickness and Sticky Kits (Genetic, Nutritional, Management related). **Enteritis/Septicemia:** Check with Canadians on their work in this field. Note especially bacterial involvements.	**Alternate Feeds:** Test various alternative feeds for mink, e.g. Distillers Dried Grains & Solubles, which is a by-product in ethanol production. Compile tables of nutrient values and economic costs. **Nutrient Requirements:** Assemble data on nutrient needs of mink at various stages of their life cycles. Ensure that data can be used in computer diet formulation. **Feed Additives:** Look for alternatives to antibiotic use. Check usefulness of feed additives to combat heat stress and other metabolic problems.	**Early Kit Loss:** Studies of this should continue, since this is still a major problem in mink raising. Investigate lactobacillus spray products as preventatives. **Environmental Problems:** Compile data on amounts of manure produced by mink and its nutrient composition. Relate diet composition, e.g. feeding of cheese and eggs, to amount of manure produced. Continue studies of compos-ting mink wastes and assemble data on their fertilizer value. **Disinfectants:** Study use of disinfectants in controlling mink disease.
II	**Blue Mink:** Investigate problems to which blue mink are especially susceptible, e.g., boils, pussy lungs.	**Feed Processing:** Investigate methods of preserving fresh feeds, including irradiation, acidification, ensiling and use of various preservatives. Study means of removing water from fresh eggs, including possibly centrifuging to eliminate the need for heating, which is expensive.	**Water Studies:** Assemble data on the effects of mink production on ground water quality, under different conditions of soil (e.g. sand, clay). **Hormone Studies:** Investigate effects of lighting on various body processes of growth, reproduction and furring. Study possible involvement of melatonin with the immune processes in mink.
III	**Encephalopathy:** Study causes and devise prevention and control methods. **Footpad Disease:** Collaborate with Canadians on developing control strategies. **Viral Disease:** (Aleutian Disease and Distemper). Continue studies to identify new virus strains involved and develop means for testing and control.	**Feed Poisons:** Continue studies already done at Michigan State of toxins that may occur in mink feed ingredients, and develop strategies to control them.	**Housing:** Determine optimum light exposure for mink and effects on reproduction, furring. Compare pairing of mink in cages with single mink in each. Compare open vs. solid pen dividers, noting effects on mink behavior and well-being. **Animal Welfare:** Develop means of relating animal condition to their health and well-being, perhaps by use of certain chemical compounds as indicators.

Mink Farmers' Research Foundation Board

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