Fur Animal Research

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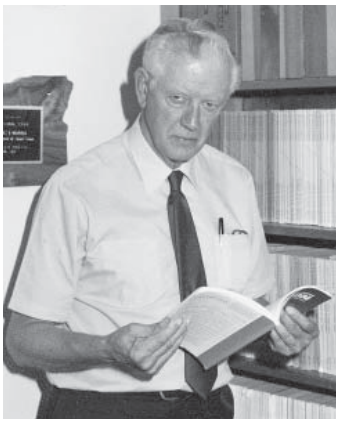


Every so often, my two office mates gently remind me that my desk is slowly disappearing under a mountain of reference material. As I responded to their latest admonition, I decided to focus on that part of the mountain that pertained to the fur industry.

We have recognized that the farmed-fur industry, which is fairly recent, doesn't enjoy the huge backlog of research information that exists for most other domestic animal species, so it doesn't have a great background of historic material.

To be sure, there were a few investigators who began the process of assembling fur animal research data, many of whom hailed from Canada. Since I come from Canada I have a few of those early compilations. The earliest I have is a book titled "*Fur Farming in Canada*" edited by J. Walter Jones that included information not only on mink and foxes, but also on martin, fisher, otter, muskrat and karakul sheep. My copy of this book is dated 1914 – nearly a century ago. Then there was "*The Principles of Mink Ranching*" by J. A. Allen and "*The Mink in Health and Disease*" by A. H. Kennedy, which was published b y the Fur Trade Journal of Canada in 1951 and cost me $6, not bad for a 300-page book! I also have "*History of the Early Mink People in Canada*" by a veterinarian, E. Rendle Bowness who I had the pleasure of meeting, years ago. He worked on Prince Edward Island, which was a center of the fur business in those days.

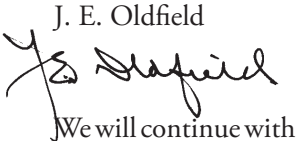
These old books make fascinating reading, and they have been joined since by more detailed reference volumes. One of my favorites, which I refer to frequently is "*Mink Production*" which was published by SCIENTIFUR under the editorship of Gunnar Joergensen. It contains a wealth of information about farm raising of mink much of which can be applied in this country, as well as in Denmark.

E. Rendle Bowness, D.V.M.

Canada Mink Breeders have come up with an attractive publication, "Mink … Biology, Health and Disease" which is edited by Bruce Hunter and Nathalie Lemieux, both of the University of Guelph. This is a collection of observations by a number of experts on mink, including some in this country, Dick Aulerich and Steve Bursian, for example. It also has a number of excellent color plates, illustrating various conditions in mink. Too, D. William Loeschke, who is no stranger to any of you, has been working on a publication incorporating observations of his long experience with mink and mink ranchers. I am sure that this will become a classic reference in the future.

Have a good summer and mink growing season.

J. E. Oldfield



We will continue with a presentation of progress reports from some of the scientists we support with funds. The first is from Dr. Kirsti Rouvinan-Watt, at the University of Nova Scotia, in Truro, Canada.

TREATMENT AND PREVENTION OF FATTY LIVER DISEASE IN MINK

Background

The liver has a central role in blood sugar and body fat metabolism. Fatty liver is a frequent pathological finding in mink and other species of livestock, where fat accumulation in the liver tissue most often occurs due to metabolic or nutritional causes. In fur bearing animals, hepatic lipid infiltration may result from a wide range of factors, including amino acid imbalance, fatty acid imbalance, and excess of carbohydrates, choline and vitamin B deficiency, poor feed quality and low feed palatability, obesity, as well as feed refusal and restricted feeding (Koskinen and Lassen 2006). In humans, fatty liver (hepatic lipidosis) is a common finding in diabetes and is considered to be a form of insulin resistance with strong evidence of an unfavorable n-3/n-6 fatty acid ratio (Adams et al., 2005).

Our recent research indicates that the mink readily accumulates body fat when fed above the Recommended Dietary Allowance (RDA) and is subject to visceral obesity, developing hyperglycemia and hyperinsulinemia (Rouvinen-Watt et al. 2004). Female mink fed 20% above RDA were in heavy or obese body condition in October and had elevated blood glucose readings (120% RDA 6.04 mmol/l, 100% RDA 4.48 mmol/l, 80% RDA 4.84 mmol/l), whereas both males and females in the 120% RDA group showed hyperglycemia (6.59 mmol/l) in December (100% RDA 4.95 mmol/l, 80% RDA 5.21 mmol/l) (Rouvinen-Watt et al. 2004). The males in the overfeeding group also had higher insulin levels (2.1 ng/ml) than animals in any other group (range 1.2-1.5 ng/ml) (Rouvinen-Watt et al. 2004). It is evident that the increased body fat deposition and obesity interfere with glucose disposal in the mink. The mink, and other closely related members of the weasel family, are also susceptible to the development of fatty liver syndrome after a short food deprivation period (Bjornvad et al. 2004; Mustonen et al. 2005, 2006, Nieminen et al. 2006a,b, under review). Despite the high body fat percentage, their ability to recruit body fat reserves during fasting may be limited (Mustonen et al. 2005). The most important biochemical manifestations in the mustelid species during food deprivation are the consistent decreases in 18:3n-3, 20:5n-3, 22:6n-3 and total n-3 polyunsaturated fatty acids (PUFA) together with an increase in the n-6/n-3 PUFA ratio in the liver and the white adipose tissue

We have also demonstrated that poor glycemic regulation is linked to poor reproductive performance and impaired health in the female mink. Blood glucose values > 8.0 mmol/l during breeding were associated with increased dam mortality, whereas values >7.0 mmol/l during gestation resulted in reduced litter size. Glucose concentrations > 7 mmol/l can thus be considered indicative of disruption in glucose homeostasis and critical in predicting potential problems with dam health and reproduction (Hynes and Rouvinen-Watt 2007a). Also, a short term treatment of hyperglycemic mink dams with anti-diabetic, antioxidative and/or anti-inflammatory treatments has been shown to effectively restore normoglycemia during late lactation (Hynes and Rouvinen-Watt 2007b). The pathophysiology of nursing sickness (Rouvinen-Watt 2003) and the associated fatty liver syndrome in mink show evident similarity to diabetes and the non-alcoholic fatty liver syndrome in humans, both manifestations of acquired insulin resistance (e.g. Adams et al. 2005), the cause of which is unknown. Especially, the mechanisms leading into excessive triglyceride accumulation in the liver tissue are poorly understood.

Objectives

The overall goal of this research is to develop a fundamental understanding of the metabolic and molecular level mechanisms leading to the development of fatty liver disease using the mink as a model animal,, and to examine plant bioactive compounds for their potential to act as regulators of gene expression for the prevention and reversal of hepatic lipid accumulation. The project will take place at the Nova Scotia Agricultural College, Canada (screening of plant bioactive compounds, fasting and feeding exposures, sampling and mRNA expression), at the Faculty of Biosciences, University of Joensuu, Finland (tissue fatty acid profiles, blood clinical-chemistry, endocrinology) and at AKVAFORSK, Norway (development of mink liver cell culture). The analytical techniques described in the following have been validated during previous studies of the research group.

Rouvinen-Watt, K., Prithiviraj, B. and Rupasinghe, V.
Nova Scotia Agricultural College
Truro, Nova Scotia, Canada B2N 5E3

MINK NURSING SICKNESS

Mink nursing sickness, a major health and welfare problem affecting the best producing females on the ranch, is characterized by hyperglycemia (elevated blood sugar), hyperinsulinemia, and potentially insulin resistance. This project investigated the impact of body condition and obesity on the regulation of lipid and glucose metabolism in the mink. Thirty mink kits from five litters, one male and one female from each were fed, from August-December, according to three feeding regimes: 80, 100 or 120% of the recommended dietary allowance (RDA). At pelting, samples were obtained from the blood, the liver, the adipose tissue, and the skeletal muscle of the animals. The blood serum was analysed for glucose content and for lipid peroxidation products (TBARS). The collected tissues were analysed for messenger ribonucleic acid (mRNA) levels of regulatory proteins governing lipid and glucose metabolism. This included the isolation, purification and characterization of mRNA using quantitative Real-Time Polymerase Chain Reaction (qRT-PCR). 18S ribosomal RNA (rRNA) was used as a control and the results were reported as a ratio of mRNA/18S rRNA. The mink in the 120% RDA group showed hyperglycemia (6.59 mmol/l) in December (100% RDA 4.95 mmol/l, 80% RDA 5.21 mmol/l). The males in the 120% RDA group also had higher insulin levels (2.06 ng/ml) than animals in any other group (range 1.17-1.51 ng/ml). A significant association was observed between the liver glycogen concentration, the liver fat content and blood insulin levels ($P = 0.034$), where Liver glycogen (%) = $19.29 - 2.08 \times$ Liver fat (%) + $8.75 \times$ Insulin (ng/ml). The significant negative relationship observed between the liver glycogen and fat content indicates reduced hepatic glycogenesis in the presence of fat accumulation in the liver and is clear evidence of the development of insulin resistance in the mink. The mRNA analyses showed no significant differences in the 11ß-HSD1 gene expression in the mink livers due to sex, treatment or six and treatment interaction, whereas the levels of the lipoprotein lipase enzyme mRNA in the subcutaneous fat were significantly influenced by the feeding intensity treatment in both male and female mink ($P=0.038$). The 80% RDA group (10.96±1.069) different from the 100% (6.76±1.013; $P=0.014$) and 120% RDA groups (8.14; $P=0.066$). It appears that the higher feeding intensity of the mink causing heavier body condition and increased adiposity had resulted in the suppression of the LPL gene expression. This in turn reduced the postprandial clearance of triglycerides from the blood stream, and may thus explain the development of hyperglycemia documented in the heavy-obese mink. Parallel with the quantification of the mRNA in the gene expression studies, we isolated the complementary DNA created in order to provide partial nucleotide sequencing of the target genes. Our findings strongly indicate the central regulatory role of the liver in the whole body glucose homeostasis in the mink and the intimate association between the regulatory pathways of blood sugar and body fat storage and utilization, where excessive fat accumulation interferes with insulin signaling. Further research will be particularly important during the lactation period in order to understand how the rapid weight loss in the nursing female influences blood sugar regulation. This will be significant regarding the development of mink nursing sickness, which results from a disruption in glucose homeostasis during lactation and is closely associated with the fatty liver syndrome.

Dr. Kirsti Rouvinen-Watt
Nova Scotia Agricultural College
Department of Plant and Animal Sciences
Truro, Nova Scotia, Canada, B2N 5E3

TESTING ALPHAMUNE – A FEED SUPPLEMENT FOR MINK

Alphamune™ G is a feed supplement that is a combination of ß-glucans (a type of polysaccharide or complex sugar) and mannans (polysaccharide-protein complexes that are derived from the cell walls of food-grade brewers yeast (Saccharomyces cervisae]). Alphamune™ G has been used as an antimicrobial growth promoter in the swine and poultry industries. It has been reported that the inclusion of Alphamune™ G in poultry and swine diets enhances the immune system, reduces colonization of harmful bacteria in the intestinal tract and enhances growth of the beneficial bacteria Lactobacillus spp. Mink ranchers have expressed interest in utilizing this feed supplement to enhance survivability of kits prior to weaning and to promote growth as the animal matures. The purpose of this proposal is to evaluate the efficacy of this product in terms of kit survivability and growth.

Thirty-five bred females received the traditional ranch diet made with wheat middlings to which Alphamune™ G was added at 0.1% of the diet (AM) and 35 bred females serving as controls received the traditional ranch diet made with wheat middlings and containing no Alphamune™ G (WM). The diets were formulated to provide the percentage of protein and fat appropriate for the time of year. Table 1 provides the composition of the WM and AM diets that were used during the whelping period. Females started on their respective diets on 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. Kits were weaned between seven and eight weeks of age and continued on their respective diets through the furring period (December 1). At weaning, animals were weighed on a monthly basis to assess growth.

As presented in Table 2, body weights of kits fed the Alphamune™ G diet were numerically greater than body weights of kits fed the basic wheat middlings diet at birth and at three weeks of age and significantly greater at six weeks of age. From birth through six weeks of age, kits fed the AM diet were on average 5% heavier than kits fed the WM diet. Body weight gain from birth through lactation and from birth through the furring period was not significantly different between the WM and AM groups, although, in general, the weight gain of mink fed the AM diet was numerically greater compared to the WM group (Table 3).

In conclusion, the inclusion of Alphamune™ G in the diet of mink from whelping through the furring period did appear to have a beneficial effect on growth of mink. It would be of interest to assess the effects of Alphamune™ G through the entire life cycle of an animal to determine if a beneficial effect on reproduction could be demonstrated.

Table 1. Composition of the Wheat Middling Diet (with and without 0.1% Alphamune™ G) Fed During the Whelping Period.

	% 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%
			DW Basis	42.0%	20.7%		

Table 2. Body weights (g) of Mink Kits Fed Diets Containing Wheat Middlings with (AM) and without (WM) 0.1% Alphamune™ G through Lactation[a]

Age	Dietary treatment	
	WM	AM
Birth	9.8 ± 0.2	10.4 ± 0.2
Three Weeks	113 ± 1.5	117 ± 1.6
Six Weeks	263 ± 5.1	282 ± 5.6*

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

Table 3. Body Weight Gain (g) of Mink Kits Fed Diets Containing Wheat Middlings with (AM) and without (WM) 0.1% Alphamune™G Prior to Weaning and at Pelting

Period of Weight Gain	Males	
	WM	AM
Birth to 6 weeks of age	264 ± 7	288 ± 8
Birth to 7 months of age	1962 ± 48	1892 ± 45
	Females	
Birth to 6 weeks of age	241 ± 7	255 ± 8
Birth to 7 months of age	1225 ± 28	1305 ± 30
	All kits	
Birth to 6 weeks of age	253 ± 5	201 ± 5
Birth to 7 months of age	1556 ± 42	1674 ± 43

[a]Data are presented as mean ± standard error of the mean.

S.J. Bursian, J. Link and A. Napolitano
Department of Animal Science
Michigan State University
East Lansing, MI 48824

MINK AND CHRONIC WASTING DISEASE OF DEER

We know that mink are susceptible to the related bovine spongiform encephalopathy agent (mad cow disease) but the susceptibility of mink to chronic wasting disease (CWD) in deer and elk is unknown. Thousands of deer and elk are currently being killed to control CWD. However, we feel that the feeding of deer and elk carcasses to mink would be a risk that is not worth taking.

Transmissible mink encephalopathy (TME) has been recognized for almost 50 years yet this noncontagious neurological disease is still considered rare. Originally described in Wisconsin, TME was responsible for 5 outbreaks involving 11 farms in the U.S. since 1947 and has also been reported in Canada, Finland, Germany, and Russia. The most recent case of TME in the U.S. was in 1985. The exact source of the disease is unknown but appears to stem from an infectious agent in mink feed.

The possibility of CWD as a cause of spongiform encephalopathy in mink is being investigated at the College of Veterinary Medicine, Washington State University. Mink have been inoculated with brain material from an elk with CWD. The intracerebral and oral routes were used. The experiments are currently underway. The mink are fed National pellets to preclude any opportunity for an outside source of transmissible mink encephalopathy agent or other sources of contamination. All mink in these trials are dark mink and were tested negative on the CEP test.

John R. Gorham
College of Veterinary Medicine
Washington State University
Department of Comparative Medicine
University of Washington

EFFECTS OF ESTRADIOL, PROGESTERONE AND PROLACTIN

The hormones 17beta-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 #2, 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 secretion), alone or in combination. Histological images of the uterus in cross section were digitized and cell heights measured using Image J software (HHIN). E2 or P4 increased luminal and glandular cell height relative to controls ($P<0.001$). 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.

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.

Most mammalian prenatal losses occur after fertilization, but before implantation, emphasizing that optimal uterine conditions must be established to ensure reproductive success. The uteri of rats, mice and humans accumulate glycogen as an energy source, in a reproductive cycle-dependant pattern. And, many reproductive failures (i.e. spontaneous abortions), are correlated with low uterine glycogen concentrations. Our objectives were to determine: 1. if the mink uterus contains glycogen as an energy source, and 2. the effects of estrogen, progesterone and prolactin (PRL) on uterine glycogen accumulation. Female mink (4/group) were assigned at random to one of 8 groups. Mink in Group 1 received no treatment (Controls). Mink in Groups 2, 4, 6 and 8 each received a slow release Silastic implant containing 100 mg estradiol-17beta (E), while mink in groups 5, 6, 7, and 8 each received an implant containing 200 ug Haloperidol (HAL) to increase endogenous PRL secretion. One week later, mink in groups 3, 4, 7 and 8 each received an implant containing 100 mg progesterone (P). Mink were sacrificed one week later and uterine samples assayed for glycogen concentrations by glucose hexokinase chromophotography assay. Both E and P increased glycogen concentrations ($P<0.05$), but exhibited no synergism when given together. Interestingly, HAL-treated mink exhibited significantly

elevated uterine glycogen concentrations ($P<0.05$), and mink treated with HAL + E had the greatest concentrations of glycogen, suggesting a synergism between E and PRL. It would not appear that in addition to their many other direct actions on the uterus that are essential for implantation, uterine glycogen synthesis is also stimulated by these hormones in mink. Because blood PRL levels increase in mink prior to and during breeding season, we hypothesize that PRL and E may promote the development of a more receptive uterine endometrium for implantation, in part, by increasing uterine glycogen reserves.

Conclusions

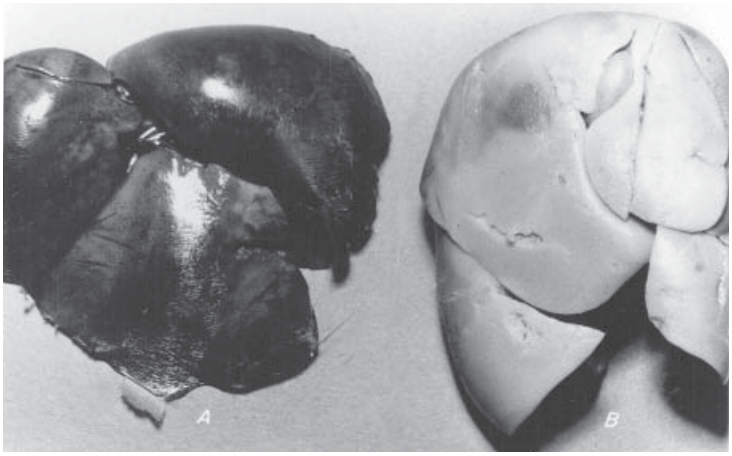
(1) E2 is a major stimulus for glycogen synthesis in the mink uterus, having a greater effect on the glandular than luminal epithelium.
(2) P4 stimulates glycogen synthesis to the greatest extent in the myometrium (images not shown).
(3) PRL appears to act directly on the mink uterus to simulate glycogen accumulation in the endometrium and myometrium.
(4) The synergistic actions of E2, P4 and PRL on glycogen production by the uterine glands of the mink may contribute to the termination of embryonic diapause and initiate implantation.

Devon Rasmussen, Daniel Mecham, Janie Thomas and Jack Rose
Idaho State University
Department of Biological Sciences Pocatello, 83209

FATTY CHANGE OF THE LIVER

Fatty change of the liver, often referred to as fatty degeneration, is frequently found on post-mortem examination of mink. It should not be considered as a separate disease but rather as an abnormal change accompanying many disease conditions.

This change is observed in intoxications resulting from bacterial toxins, toxic products of metabolism or chemical poisoning. It is frequently observed in females with nursing sickness. When females are autopsied in late pregnancy, fatty livers are often seen. The change can be produced experimentally by starvation and by including excessive fat in the ration. Significant fatty change of the liver is not found in experimental or actual field cases of steatites (yellow fat disease).



Fatty degeneration of the liver (right) is easy to diagnose but often difficult to determine the cause.

Because of the accumulation of fat in the cells of the liver, that organ appears yellowish or clay colored. It may be softer than usual and it has a tendency to separate into small pieces. When the liver is cut with a cold knife, a film of grease remains on the blade.

The diagnostic value of fatty degeneration is limited because it is seen in many disease conditions. Therefore, do not attempt to treat fatty change of the liver as such but rather look for the actual cause to provide a sounder basis of therapy.

John R. Gorham, DVM

ANNUAL MEETING, MINK FARMERS' RESEARCH FOUNDATION

The Board of Directors of he MFRF met in May, at Washington State University in Pullman.
Members present: Robert Zimbal, Sr., Jim Wachter, Ryan Holt, Dr. Hugh Hildebrandt and Dr. John S. Easley
Guests present: Dr. John Gorham, Dr. Austin Larson, Paul Mauer

1. Review of Research Priorities
   - Move Water Quality into the "I." priority ranking from the "II." priority ranking.
   - Move Feed Processing into "I." priority ranking from "II." priority ranking.
   - Move Feed Additives from "I." priority ranking into the "II." priority ranking.

2. A-D research into further typing, testing and control was discussed. It was agreed that A-D is still the mink industry's number one problem and that it should be addressed in as many ways as possible

3. Discussion on behavior studies: Canadian Mink Breeders are supporting a wide range of research. The minimum pen size per adult animal was discussed. A decision on a recommendation should come late this year.

4. Dr. John Gorham and his research team presented some preliminary findings on their BSE and CWD research. Mink are susceptible to BSE and are a good model for its study. Mink have not been shown to be susceptible to CWD when exposed in natural ways. A tour of the mink research facilities and the Washington State University Veterinary Hospital were provided to all in attendance.

5. Research proposals were reviewed and funding was allocated as follows:

Dr. Jack Rose, Idaho State University	$7,400
Dr. Steve Bursian, Michigan State University	$20,000
Dr. John Gorham, Washington State University	$10,000
Fur Breeders Agricultural Co-op	$9,000
Dr. Kristi Rouvinen, Watt	$10,000

Other funding allocations:

Travel	$10,000
Administration	$12,500
Ranch Services	$18,000
Newsletter	$6,000

Jim Wachter proposed a motion to accept the budget as stated. Ryan Holt seconded the motion. Motion carried. Jim Wachter proposed a motion to adjourn the meeting. Ryan Holt seconded the motion. Motion carried.

Respectfully submitted,
John S. Easley, D.V.M.
Kettle Moraine Large Animal Clinic

(Revised May 11, 2007)

THE MINK FARMERS' RESEARCH FOUNDATION: RESEARCH PRIORITIES

AREA OF RESEARCH	DISEASE	FEEDS/NUTRITION	PHYSIOLOGY/MANAGEMENT ENVIRONMENT
PRIORITY RATING I	**AD:** Lateral flow test development and sampling protocols. **Nursing** Sickness & Sticky Kits: Identify physiological basis for nursing sickness and birth of Sticky Kits and study relationship to management practices. **Enteritis/Septicemia:** Identify and isolate various bacterial viral strains and develop control methods.	**Alternate Feeds:** Identify and analyze various potential levels for mink, including spent hens. Compile tables of nutrient values. Compare acceptability and nutrient values of fresh and frozen feeds. **Nutrient Requirements:** Assemble data on nutrient needs of mink at different stages of the life cycle. Combine these with data on feed nutrients in a form suitable for computer formulation of diets. Find specific requirements to protect against Nursing Sickness. **Feed Processing:** Investigate methods of preserving fresh feeds including acidification, irradiation, ensiling, and use of preservatives (formaldehyde).	**Early Kit Loss:** Continue studies to identify causes and prevention of losses of neo-natal kits. Investigate lactobacillus spray products as preventatives. **Environmental Problems:** Investigate and develop practical, cost-effective ways of lowering volume of excreta and disposing of mink farm wastes, including composting, and fly and odor control. Determine nutrient and fertilizer values for mink manure. Develop uses for it. **Water Studies:** Survey effects of mink production on ground water quality and develop means of improving it. Study effects of mink operations on different soil types, e.g., clay, sand.
II	**Blue Mink Problems:** Investigate boils, pussy lungs and various problems occurring; predominantly in blue mink.	**Feed Additives:** Test usefulness of feed additives against specific problems, e.g., electrolytes in times of heat stress, enzyme 'cocktails,' probiotics and DL.	Protocols for virus eradication and disinfection of AD-infected farms. Biosecurity protocols for AD prevention. **Hormone Studies:** Investigate effects of lighting on mink life processes. Continue investigation of ways in which hormones influence basic processes of growth, reproduction, lactation and fat production. Study possible involvement of melatonin in immunity with specific types of mink.
III	**Encephalopathy:** Study causes and devise prevention methods. **Viral Disease (AD and Distemper):** Continue studies to identify new virus strains and develop means of control.	**Food Poisons:** Continue investigation of toxins that may occur in, or contaminate, mink foods, and ways to control them.	**Housing:** Find optimum light exposure for mink. Investigate open vs. solid pen dividers and their effects on mink well-being.

Mink Farmers' Research Foundation Board

Members of your Research Foundation Board of Directors invite your input into the ongoing program of research. Please contact any of the Board with suggestions or comments. You may reach them at:

OFFICERS:

Chairman: Robert Zimbal, Sr.
2111 Washington Ave.
Sheboygan, WI 53081
PH.: (920) 452-7380
FAX: (920) 803-0662
rjz@execpc.com

SECRETARY:

Dr. John S. Easley
W 8866 Spring Valley Drive
Glenbeulah, WI 53023

DIRECTORS:

Dr. J. E. Oldfield
Dept. of Animal Sciences
Oregon State University
Corvallis, OR 97331-6702
PH.: (541) 737-1894
FAX: (541) 737-4174
james.e.oldfield@oregonstate.edu

Dr. Gary Durrant
Utah Fur Breeders Co-Op
8700 South 700 West
PH.: (801) 255-4228
FAX: (801) 255-4678
garydurrant@comcast.net

Dr. Hugh Hildebrandt
Medford Veterinary Clinic
898 S. Gibson Street
Medford, WI 54451
PH.: (715) 748-2341

Ryan Holt
9762 S. Tayside Drive
South Jordan, UT 84095
PH.: (801) 280-1428
FAX: (801) 255-4678
rhino@networld.com

Paul Westwood
8137 South, 1800 West
Spanis Fork, UT 84660
PH.: (801) 798-1786
FAX: (801) 298-1482
pwstwd@msn.com

Jim Wachter
N 5350 Country Aire Road
Plymouth, WI 53073
PH.: (920) 892-4287
FAX: (920) 892-4287
jwachter@wi.rr.com

Author Dr. J.E. Oldfield • Desiged by Sandra Wallace