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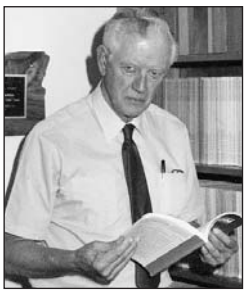
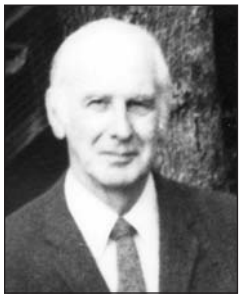


One of the things that seem to happen as you become more mature ("ancient," my wife says), is that you frequently find yourself thinking of times past. Just recently, I was thinking of the beginnings of the Mink Farmers' Research Foundation, which was the brain child of Dr. G. R. Hartsough and Ronald Stevenson. Dr. Hartsough, a veterinarian and Mr. Stevenson, an engineer/mink rancher, felt that the mink industry needed a continuing research program if it was to develop and progress, and they made a good team in getting the Foundation established.

Dr. G. R. Hartsough was a keen observer and he had the ability to translate his observations on mink ranches to solid suggestions for research to deal with them. Putting his contributions together would form a most useful textbook on mink diseases. He was the first person to suggest that yellow fat disease (steatitus) was the result of feeding high levels of unsaturated fat in the diet, and to a subsequent deficiency in dietary vitamin E. He was also the first to use sulfathiozole in the treatment of pseudomonas pneumonia outbreaks.



Dr. G. R. Hartsough

He was the first to observe that a thiamine deficiency (vitamin B1) in mink could be caused by fish containing the enzyme thiaminase. He worked with researchers at Oregon State University on the cause of cotton pelts. Mink farmers will always remember his suggestions for the control of nursing sickness. In recent years, he was concerned about water deprivation and the losses it caused in kits. Older farmers can recall that inactivated distemper vaccines failed to control many distemper outbreaks. He noted the advantages of live virus vaccines and was the first to use them in field outbreaks.

He saw some of the first cases of Aleutian disease in the early forties when Aleutian and sapphire breeding stock were shipped from Oregon to Wisconsin, and first described the disease along with Dr. John Gorham in 1956. He diagnosed the first cases of mink virus enteritis in the U.S. and he prepared the first inactivated virus vaccines for its control. Doctors Dieter Burger, Dick Marsh, and Hartsough were the first to describe and diagnose mink encephalopathy. It is a rare fatal disease that causes spongy changes in the brain, probably caused by the feeding of "downer cows." He recognized pseudorabies in U.S. mink for the first time. It is a rarely diagnosed viral disease caused by the feeding of infected pork lungs. (This material kindly provided by Dr. John Gorham).

Dr. Hartsough can conservatively be said to have saved the mink industry millions of dollars. He was a truly exceptional man and I have felt that there would never be another just like him. But there actually is one who comes close, and that is Dr. John Gorham, who collaborated with him in many of his studies.

Dr. Gorham has now reached the stage of his career where he is taking a look back at his very significant accomplishments in mink research. He has assembled a collection of his many publications, written from 1947-2004 on a compact disk (CD) which is available from the College of Veterinary Medicine at Washington State University, Pullman, WA 99164. The CD is easy to use, if you have access to a Windows-driven computer. And John is able to combine his really world-class mink research with a lively sense of humor. If you have heard one of his after-dinner speeches, I guarantee you will not soon forget it.

Dr. John Gorham

Have a great summer and fall, and keep cool!

J. E. Oldfield

MFRF MEETING

The Board of the Mink Farmers' Research Foundation held its annual meeting on May 12th in Seattle. Officers elected were Robert Zimbal, Sr., Chairman, Jim Wachter, Vice-chairman, and Dr. John Easley, Secretary. The following notes on the meeting were assembled by Dr. Easley.

Dr. Hugh Hildebrandt discussed the new lateral flow Aleutian Disease screening test that is being developed by Bob Stephon at Scintilla Development Company. The development is progressing rapidly and initial results are very encouraging. Studies comparing it to the CEP test have shown very high correlation in results.

The committee discussed how the testing development should be funded. Approximately $3000 has been spent so far, with a like amount to be needed in the near future. Scintilla Development will own rights to the test and its distribution. Drs. Hildebrandt and Easley indicated that they would be having discussions with Scintilla about further financial responsibilities for development and the development of test protocols and sampling techniques.

The research priorities were discussed, and no new research proposals had arrived at that time. The priorities were re-established; as follows.

The budget was discussed and it was agreed that 90% of the funds available for research should be allocated. There is approximately $60,000 available for grants for research. It was agreed to fund the existing proposals at last year's levels. Then at the annual meeting in July 2006, the remaining funds will be allocated.

It was discussed and agreed upon that next year's spring meeting should be held at Washington State University.

INVESTIGATION OF A COMMERCIAL FEED SUPPLEMENT

Recovery® is a commercial product that is a complex of iron, copper, zinc and cobalt. A liquid preparation that is added to drinking water at a rate of 1 ml/liter, Recovery® has been used in the swine and poultry industry to enhance immuno-competence. It was suggested to us by Bob Westlake that we evaluate the effect of Recovery® on kit survivability and growth as well as on pelt quality. Anecdotal information suggested that mink fed a diet containing Recovery® produced a shinier pelt.

Eleven pregnant females were given a diet containing Recovery® (1 ml/liter dietary water) and 11 pregnant females serving as controls were produced on the same diet without Recovery®. Animals were given these diets approximately one week prior to whelping through weaning when the kits were six weeks old. At weaning, kits were maintained on their respective diets until mid-December.

Control females had an average litter size of 6.2 kits while the Recovery® group had an average litter size of 5.9 kits. Kit survivability through six weeks of age was 96% for the control group and 88% for Recovery® group. Kit body weights at birth were similar between the two groups (10.1 g for the control group compared to 10.2 g for the Recovery® group). However, at 3 and 6 weeks of age, kits in the Recovery® group were significantly heavier than the control kits (135 g compared to 121 g at 3 weeks of age and 336 g compared to 295 g at 6 weeks of age). From birth to 6 weeks of age, kits in the Recovery® group gained significantly more weight (327 g) than did control kits (284 g). After weaning, the apparent growth advantage offered by Recovery® was no longer evident. On August 16, both the males and females in the Recovery® group weighed slightly less than their control counterparts (1,670 g compared to 1,728 g for the males, and 1,106 g compared to 1,142 g for the females). By mid-December, the Recovery® males weighed significantly less than the control males (2.327 g compared to 2,495 g) and the Recovery® females weighed slightly less than the control females (1,286 g compared to 1,324 g). Casual observation suggested that the difference in growth of the juveniles was due to a decrease in feed consumption by the animals on the Recovery® diet.

Twenty-five males from each group were pelted in December. The pelts were uniquely tagged to allow

subsequent identification of treatment. After processing, pelts were graded by an experienced individual who had no knowledge of the treatments. The grader could not detect a difference in pelt appearance between the two groups.

The results of this study suggest that the inclusion of Recovery® has a beneficial effect on kit growth through weaning but that advantage disappears as the animal continues to grow. It is possible that the ion complex reaches a saturation point in the animal and the animal then decreases feed intake in a compensatory manner. It would be of interest to determine if the weight advantage offered by Recovery® through weaning could be maintained if those animals were then placed on non-supplemented feed until pelting. Alternatively, the concentration of Recovery® in the feed could be reduced.

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THE MINK FARMERS' RESEARCH FOUNDATION: RESEARCH PRIORITIES (Revised June 15, 2002)

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 ??. 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 Additives:** Test usefulness of feed additives against specific problems, e.g., electrolytes in times of heat stress, enzyme 'cocktails,' probiotics and DL.	**Early Kit Loss:** Continue studies to identify causes and prevention of losses of neonatal 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. Protocols for virus eradication and disinfection of AD-infected farms. Biosecurity protocols for AD prevention.
II	**Blue Mink Problems:** Investigate boils, pussy lungs and various problems occurring; predominantly in blue mink.	**Feed Processing:** Investigate methods of preserving fresh feeds including acidification, irradiation, ensiling, and use of preservatives (formaldehyde).	**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. **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.

HORMONAL EFFECTS ON MINK PRODUCTION AND REPRODUCTION

Reproductive physiology: As part of a larger study, we measured serum prolactin (PRL) levels in male mink during development and adulthood (Kabbaj et al., 2004). During both puberty and the active spermatogenic phase of the reproductive cycle of adult male mink, PRL levels were low. Subsequently, PRL levels increased sharply during the first half of testicular regression, then rapidly declined, remaining low until the start of the next recrudescence period. Increasing serum PRL levels in the spring may cause macrophages to reduce hormone sensitive lipase (HSL) expression and activity in the testis, and this may in part account for the reduction in testosterone secretion by Leydig cells at this time. In support of this hypothesis, Huang et al., (2001: J. Cell Biochem., 83:313) showed that PRL reduced testosterone secretion in the rat, via mechanisms that involved cells other than Leydig cells. It would seem reasonable to further investigate the possibility of inhibiting PRL secretion in male mink during the spring, as a potential means of increasing testosterone secretion, sperm production and/or viability.

Studies are in progress to analyze PRL receptor (PRL-R) production in the testis, epididymis, uterus and ovaries of mink. Thus far, we have shown that the concentration of PRL-R's in the epididymis (45.8 7.0 fmoles/mg protein) is much greater than in the testis (18.8 2.9 fmoles/mg protein; $P<0.01$) of adult male mink. Thus, PRL may play roles in both testicular steroidogenesis and sperm maturation within the epididymis. We attempted to increase PRL secretion in male mink prior to breeding using the drug haloperidol. Unfortunately, the sedative effect of the drug rendered the animals unresponsive to females during the breeding season. Other drugs will be investigated for this purpose.

Recently, (12/2/2002) we collected testicles from both pubertal (7 months of age, N=85) and adult (17 months of age, N=90) mink. In addition, uteri and ovaries were collected from pubertal (N=120 complete uteri and ovaries) and adult (N=120) females. The cell membranes from all of these tissues have been isolated and PRL receptor assays will be conducted this summer. The goal will be to determine if the PRL-R concentrations in these tissues differ between pubertal and adult animals, which may reflect part of the maturational process of the reproductive systems.

Fur Growth Studies: This past year we have shown that PRL simulates the development of mink hair follicles (Guard and Under hair) during the summer fur growth cycle, and inhibits them during the winter fur growth cycles. Interestingly, artificially increasing serum PRL levels, and then plucking the fur to induce growth in the spring, significantly reduces under hair density, but increases both guard and under hair fiber diameters. In contrast, reducing PRL secretion in the spring, results in a much greater number of under hair follicles being activated, but the diameters of both guard and under hair fibers are reduced. PRL had no effect on guard hair density. We also found that treatment of mink with melatonin (MEL) to advance winter priming significantly increases the diameter of guard hair fibers, but has no effect on guard hair density or ellipticity of fibers. Because ellipticity of fibers (shape in cross-section) is negatively correlated with fiber strength, this should be of interest to ranchers that use MEL implants. Moreover, under hair density and diameter following MEL-treatment were not different from control mink that developed the winter pelage at the normal (later) time. Thus, we can find no negative effects of MEL on hair density or upon hair fiber characteristics.

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ALEUTIAN DISEASE: THE IODINE AGGLUTINATION TEST (IAT)

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In his memoirs, Dr. John Gorham describes early investigations by himself and others into ways of testing for Aleutian disease (AD).

Up until 1962 when the IAT was first reported, there was no way to diagnose Aleutian disease other than by typical clinical signs and autopsy findings. Both were well described in the fur journals. We were dealing with dead and sick mink, and the mink farmer needed a simple test to pick out mink that had AD.

We knew that most but not all AD infected mink had a marked increase of gamma globulin in their blood serum. When the gamma globulin increases from about a normal 13% (which would be IAT negative) to about 22% or above, it would indicate an IAT positive AD infected mink. In Scandinavia it was called the Mallen test.

Even though it was a non-specific test, millions of mink were tested in the United States, Canada, and worldwide. It is safe to say prior to the CIEP test the IAT was a big help in the control of AD.

With any test for gamma globulin levels in the IAT, the CIEP, or the newly developed lateral flow test, the virulence of the AD virus strains (discussed later) and the genetic differences of the mink must be considered. As an example, any Aleutian aa genotype mink will die of AD when infected with any strain of AD virus. Therefore, by sequential IAT testing, Dr. G. R. Hartsough was able to completely eradicate AD on a 100% Aleutian mink farm.

The IAT and Non-Aleutian Mink

It didn't take long for some problems with the IAT to show up.

1.)The IAT did not detect AD infected mink with a gamma globulin level of less than about 22%. It was obvious that all IAT negative mink had to be retested.

2.)Some mink that tested positive later turned IAT negative. More importantly, some of these "negative" mink could be carriers and a continuing source of the AD virus on the farm.

3.) Mink infected with the low virulent Pullman strain may become AD carriers and may not be IAT positive.

4.) Mink experimentally infected with the AD virus required a long incubation period of about 3 weeks to become IAT positive.

5.) The IAT is a non-specific test and may become positive in mink with abscesses or avian tuberculosis.

6.) It was evident that IAT by itself would never really control AD on a farm.

The Counterimmunoelectrophoresis Test (It is easier to call it the CIEP)

In 1972, Cho and Ingram, researchers at the University of Guelph in Ontario, Canada, made a great contribution for the control of AD. Needless to say, the CIEP, which is a specific test for AD virus infected mink, replaced the IAT. The CIEP gave us the tool to establish first rate control measures and all of us that investigated AD were really pleased.

The CIEP tests for specific AD virus antibody. A positive test can be shown about a week after exposure, which means that a mink is infected. However, a few non-Aleutians may be positive and have cleared the AD virus from their body. In any attempt to control the disease, all positive mink including the few false positives should be pelted.

Dr. Mogens Hansen of the Danish Fur Breeders Laboratory designed a first rate program that showed that the CIEP was very effective. By multiple testing of virtually all of the mink on farms combined with large scale pelting of positive mink, only a limited number of farms still had AD infected mink. At the beginning of the program in 1976, the kit average was 3.9 kits per mated female. In 1994, there were 5.2 kits per mated female.

The Lateral Flow Test

The new test called the Lateral Flow Test was developed by Dr. Hugh Hildebrandt, Medford Veterinary Clinic in Medford, Wisconsin. Like the CIEP test, it is a test for AD antibody. A positive test indicates that a mink is infected with AD. The new "penside" test can be run by the farmer. Urine, blood or saliva is collected in a tube, and a special treated paper is placed in the tube. Lines appear on the paper which indicate a positive or negative mink.

Strains of Aleutian Disease Virus

We, as well as others, have recognized differences in the appearances of Aleutian Disease (AD) in field outbreaks and laboratory experiments.

Since any known AD virus strain will kill Aleutian mink

aa within a few months after infection, a clinical description of AD strains must be based on AD in non-Aleutian mink. Here, there are observable differences in the ability of AD strains to cause disease.

The Utah I strain was first described by Porter and Larsen and is considered to be the "classic" highly virulent AD virus. The Utah II virus strain, which was isolated by Dr. Gary Durrant, has a slightly different molecular makeup than the Utah I strain but is as virulent as Utah I. Raccoons were infected with AD virus in the areas where Utah II was isolated.

Dr. William Hadlow found in trials conducted at the Rocky Mountain Laboratory in Montana that the Utah I strain was highly virulent for darks, pastels, and all other non-Aleutian mink. Dr. Mogens Hansen, Danish Fur Breeders Cooperative, observed that the Danish AD virus killed a high percentage of non-Aleutian mink.

The Wisconsin strain is not only highly virulent for pastels and darks but it has another important identifiable feature - neurotropism. In this instance, the AD virus attacks the brain. It was estimated in a single Wisconsin outbreak that about 50 percent of the non-Aleutian mink died of AD showing convulsions and other nervous signs prior to death. With other strains of AD, only 1 to 2 percent of AD-affected mink show nervous signs.

Hadlow observed in experiments that the Ontario and Montana strains were not as virulent for pastels as Utah I but they were more virulent than the Pullman strain. Ontario and Bitterroot strains were arbitrarily designated as medium virulence strains.

We showed that pastels, darks, and other non-Aleutian mink infected with the Pullman strain of ADV may occasionally succumb but most are infected without showing any signs of disease. Interestingly, Hadlow, who has done a great deal of solid AD research, has detected AD virus in the mesenteric lymph nodes of pastels two years after they were experimentally infected. This is good evidence that the AD virus can persist in non-Aleutian mink and serve as a continuing source of AD virus on a farm.

Aleutian disease virus - ADV-P (Porter) is a strain of tissue culture virus that was adapted in the laboratory from the Utah I strain. Similarly, ADV-G (Gorham) is another tissue culture strain that was adapted from the highly virulent Utah I strain. Both Dave Porter and ourselves tried for many years to grow AD virus in a tube full of live tissue culture cells. Porter won the race. It was a friendly rivalry. But our AD virus (ADV-G) grew a little better and is currently used in CIEP tests and in many worldwide research projects. Since the CIEP antigen apparently reacts with mink infected with any known strain of AD virus, it would appear that Utah I strain is closely related to all other recognizable strains.

Future strains of AD virus will yield significant information on virulence and on a variety of other factors that must be known for a better understanding of AD and its control.

The next article in this series will discuss the transmission of Aleutian disease.

The Virulence of Aleutian Disease Virus Strains in Non-Aleutian Mink Genotypes

Highly Virulent	Medium Virulence	Low Virulence
Utah I	Montana	Pullman
Utah II	Ontario	ADV-P (Porter) Tissue culture adapted
Danish		ADV-G (Gorham) Tissue culture adapted and used for the CIEP tests
Wisconsin (Neurotropic)		

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IRRADIATION AS A MEANS OF PRESERVING MINK FEED

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Irradiation has been used, successfully, as a means of preserving human foods, however its use has not become common. At our request, Dr. Bursian at Michigan State University has applied the practice to mink feed, where it has the potential to replace cold storage which is expensive.

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

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

In 2005, we reported that 20 6-week-old kits provided with irradiated feed for 21 days had average body weight gains of 130% (males) and 100% (females) during that time period, while controls had body weight gains of 114% (males) and 88% (females). The results of this feeding study indicated that the irradiated feed was palatable and that kits fed the irradiated feed had a greater relative weight gain compared to kits fed a conventional diet.

In the summer of 2005, a second trial feeding trial was conducted in which 9 pregnant females were provided feed made with irradiated duck offal and 11 pregnant females were fed the same diet made with duck offal that was not irradiated. Animals were provided these diets approximately one week prior to whelping through weaning when the kits were 6 weeks old. Control females had an average litter size of 6.2 kits while the irradiated feed group had an average litter size of 4.1 kits. Kit survivability through 6 weeks of age was 96% for the control group and 93% for the irradiated feed group. Body weights at birth, 3 weeks and 6 weeks of age were slightly greater for the irradiated feed kits compared to control kits, but these differences were not significant. At six weeks of age, the average weight of control kits was 295 grams and the kits fed the diet containing irradiated duck offal had an average body weight of 304 grams.

To assess the effects of irradiation on bacterial counts in mink feed, frozen samples of irradiated feed and frozen samples of non-irradiated feed were removed from the freezer and allowed to thaw for 24 hours in a cooler. Twenty-four hours after removal from the freezer, an aliquot of each type of feed was taken for bacteria counts and aliquots was placed on the feed grids of unoccupied cages for 24 hours. The next day, the feed aliquots placed on the feed grids were removed for assessment of bacteria counts. In addition, aliquots of both types of feed were again removed from the cooler for bacteria counting and additional aliquots were placed on feed grids for a twenty-four period prior to collecting for assessment of bacteria growth. This process was repeated for a third day. Thus, feed samples kept in the cooler from 1 to 3 days and samples that sat on feed grids for 24 hours after being kept in the cooler for 1 to 3 days were evaluated. The results of this trial indicated that there were no significant differences in the number of bacteria colony forming units between the irradiated and non-irradiated samples, regardless of sampling location and sampling time.

The results of the two feeding studies suggest that the use of irradiated feed/feed components does not offer a significant advantage over non-treated feed in terms of kit survivability and growth. At present, the logistical challenges and cost of irradiation make it an unfeasible method of reducing bacterial loads in mink feed. Furthermore, assessment of bacteria counts suggested that irradiation offered no significant advantage over freezing in terms of reducing bacterial loads.

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