FUR ANIMAL RESEARCH

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As we end this first year of the 2,000's, it is comforting to see that a number of concerns about problems that a new century would bring never happened at all. The Y2K problems, for example, which were feared would destroy many computer programs, did surface, but at a much lower profile than many people anticipated. By this time, certainly, any such problems have been solved, and we are back to business as usual.

I have mentioned before that a century's end is a good time to look back, and this is especially attractive to old people like myself, who have a long way to look. My interest in mink began when I was doing graduate studies at the University of British Columbia in the late 1940's. My major professor, Dr. A.J. Wood, suggested that I do my Master's thesis on this "new" species of animal, and I have been fascinated with them ever since. It was at U.B.C., too, that I first met John Gorham, who had assumed leadership of the mink research program at Washington State University and I have admired John's work immensely over the years, and he has been a consistent and valued contributor to this Newsletter.

I moved from U.B.C. to Oregon State University in 1949 and found a mink research program in the Department of Fisheries and Wildlife (then "Fish and Game") which was ably directed by Phyllis Watt (now Wustenberg). If I needed any encouragement to continue my interest with mink, Phyllis certainly supplied it. Raised on a mink ranch at Bay City, Oregon, she combined her knowledge of the industry and its needs with her research abilities to form an effective program which she operated with great good humor. When Phyllis left, the program was run by Ken Davis, who went on to a distinguished career with the Charles Pfizer company, and later by John Adair and Ron Scott. The University administration decided that mink were really a domesticated species and moved the mink program from Fisheries and Wildlife to Animal Science, where I became associated with it. We hired an extremely able young scientist, Dr. Floyd Stout, who developed an outstanding research program relating nutrition to fur color and quality and accomplished the early work on effects of daylight exposure on priming of the winter pelt, which led eventually to the use of melatonin to accelerate furring.

Much of our work in Oregon was supported by the Mink Farmers' Research Foundation and I became acquainted with Dr. Hartsough and Ronald Stephenson, who organized the program of mink research supported by a check-off program from pelt sales. Dr. Hartsough was a remarkable individual, whose active mind devised solutions to many of the most pressing problems facing mink producers. His efforts are continuing, through studies under the direction of Dr. Dick Aulerich at Michigan State University, with support from the fund established in Dr. Hartsough's memory. Finally, I must pay tribute to the members of your MFRF Board. They have changed over the years, but all have been dedicated individuals who have worked hard to identify which industry problems deserve highest priority for research and then to find research personnel and facilities to deal with them. In this, first Thanksgiving season in a new century, I thank them, on your behalf, for the good things they have done and are doing. May you have a productive and profitable marketing season. Cheers!

J. E. Oldfield

SPONGIFORM ENCEPHALOPATHIES IN THE UNITED STATES

The Council for Agricultural Science and Technology (CAST) has issued a report on this problem, popularly called "mad cow disease." It identifies a number of types of the disease, including **bovine spongiform encephalopathy** (BSE), **scrapie** in sheep and goats, **felina spongiform encephalopathy** (FSE) in cats, **chronic wasting disease** in deer and elk, and **transmissible mink encephalopathy** (TME) in mink. A human form is called **Creutzfeldt-Jacob disease** (CJD). These are all fatal, neurodegenerative disorders. The mink form, TME, was first identified many years ago in Wisconsin, in 1947, and has since been found in Canada, Finland, Germany, and Russia. The cause seems to have been contaminated feed. It is important to remember that the ruminant form, BSE, has never been detected in the United States (from: **Transmissible Spongiform Encephalopathies in the United States**. Report #136. 36 pp. Copies of the report can be had from the Council for Agricultural Science and Technology, 4420 West Lincoln Way, Ames, Iowa 50014. The cost is $20 plus $3 shipping).

LECITHIN: A NEW DIET ADDITIVE?

From time to time, we pick up information outside of our own research program that might have relevance for mink. An example is a recent note about **lecithin** that is being used to enhance aquaculture (fish) diets. Lecithin is actually a mixture of natural phospholipids which are present in all living plant or animal cells. Its composition includes choline, inositol, phosphorus and fatty acids. Commercially, lecithin is produced as a by-product in soybean processing. Lecithin helps emulsify fats in feeds and since mink diets are commonly high in fats, it may have some application in them. In a 1997 study, lecithin was tested in diets for Atlantic salmon. With a mean starting weight of 1.5 grams, after an 84-day trial, the fish fed the lecithin supplement weighed 13 grams, versus 6.3 grams for fish fed the unsupplemented, control diet. The lecithin supplement made it unnecessary to add choline chloride to the diet, when it was fed at a level of 3% of the diet (from Zang and McCaskill. 2000. Lecithin to enhance aquaculture diets. **Feed Management** 51(4):26-27).

BODY LENGTH AND BODY WEIGHT RELATIONSHIPS TO PELT LENGTH

In our last issue, I cited Finnish studies which suggested body length was a better indicator of pelt length than was body weight. This implied that fatness did not necessarily contribute to pelt length. Dr. Leoschke presented material at the International Scientific Congress on Fur Animal Production held at Kastoria, in Greece, that indicated a strong correlation between body weight and pelt length (see Figures 1 and 2). We will look for further information on this important topic and will report it to you, as available.

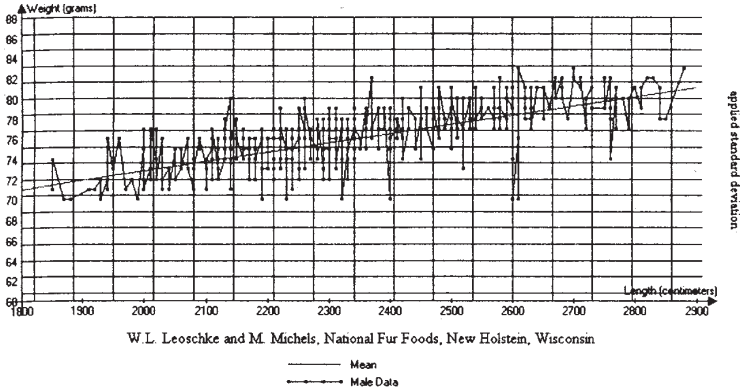

Figure 1. Correlation of pelt length with body weight (500 male, dark, kits).

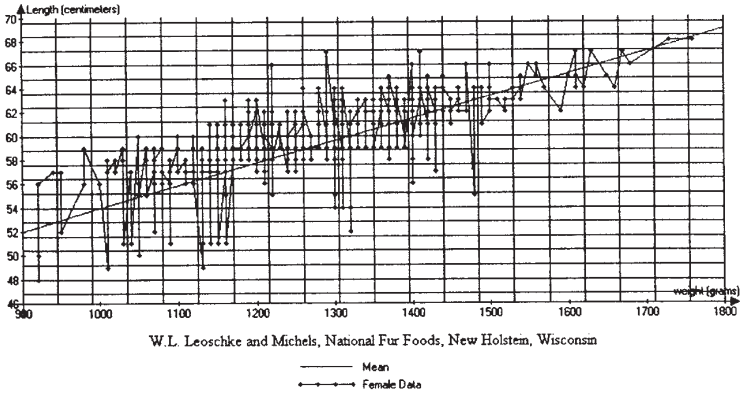

Figure 2. Correlation of pelt length with body weight (500 female, dark kits).

ALEUTIAN DISEASE

The International Scientific Congress in Fur Animal Production held 13-15 September of this year, in Greece, devoted a whole section to Aleutian Disease (AD) research. Dr. Gorham, who has probably had as much experience with AD as anyone in the world, presented the following, interesting review entitled, "Perspectives on Aleutian Disease," which we are pleased to reproduce here.

Of all the infectious diseases of mink, Aleutian disease (AD) has no doubt caused the greatest mortality and deleterious losses in reproduction and fur quality. Moreover, the immunosuppression caused by AD markedly influences the course of other viral, bacterial and nutritional diseases. It would be difficult to estimate the total economic harm for the mink industry.

Within six years after the Aleutian mutation aa appeared on an Oregon, USA, dark mink farm in 1941, we heard about the problems of keeping this beautiful mink alive. Because the deaths were seemingly confined to Aleutian mink, it is not surprising that the disease with its slow, downward course, weight loss, pale mucous membranes, bleeding at the mouth, marked thirst, and tarry diarrhea was called Aleutian disease. Everyone thought it was a hereditary disease and did not suspect that they were dealing with an infectious disease that had an incubation period measured in months and a carrier state of years.

The first suggestion that AD was an infectious disease was the widespread deaths of Aleutian mink following distemper vaccination (Gorham et al., 1965). The autogenous vaccine was made from the spleens of mink that were infected with distemper and formalized. No one knew that the vaccine was contaminated with the more formalin resistant Aleutian disease virus (ADV). While the "vaccine" caused the death of hundreds of Aleutian mink, there were no reports of deaths in dark mink. In all likelihood, the low virulent Pullman strain of ADV was present in the spleen used for the vaccine. After these vaccine incidents, there can be no argument that Aleutian mink is not the sentinel animal for AD *par excellence*.

There was no research on the disease and fifteen years after AD first appeared, we still did not know the cause, how the disease was transmitted on farms or how it could be prevented (Hartsough and Gorham, 1956). Our early transmission experiments failed because we only had non-Aleutian mink as test animals and we used the low virulent Pullman strain. We had no laboratory means to detect an infected mink.

Microscopic descriptions of the liver, spleen and lymph nodes revealed the massive numbers of plasma cells and Obel (1959) named the disease *plasmacytosis* - a more descriptive term. As one might expect, the marked proliferation of plasma cells resulted in hypergammaglobulinemia. The excessive immune response is ineffective and does not neutralize the ADV. Antibody combines with the virus to form infectious immune complexes that are deposited in the glomeruli and arteries. The disease slowly progresses and the mink die of uremia and renal disease (Hensen et al., 1976; Porter et al., 1980). It is the overwhelming immune response that is primarily responsible for the development of lesions, which is somewhat surprising. Other than interstitial pneumonitis of the newborn kits, the virus does not directly cause lesions and death.

Since the ADV has been demonstrated in the saliva, urine and blood, handling mitts and pens are a likely source of infection (Gorham, 1965). While we can assume that shedding of the virus continues throughout the course of the disease, at least with highly virulent strains, transmission between pens seems relatively low when compared to distemper and mink virus enteritis. The question of whether ADV can be transmitted by the airborne route has not been answered because such research is difficult to accomplish. Field observations have suggested that wind and high-pressure cleaners may spread ADV between infected and non-infected sections of a farm (Hansen, 1985). Chriel (1990) described possible wind-borne transmission between neighboring farms by using mapping methods. One wonders if the highly virulent Utah-I strain is more transmissible and may account for the explosive outbreaks of AD on some Utah farms.

Most mink farmers and veterinarians involved in control programs have long considered that kits from AD infected females will be infected with AD (Larsen et al., 1984). Alexandersen and Bloom (1987) clarified the role of ADV maternal antibody. If the kit's mother is not infected, she will not have maternal antibodies to confer to her litter. If her kits are infected from an outside source and do not have protective maternal antibody, they will frequently develop a fulminating interstitial pneumonia within a few days after birth. If the infected females pass maternal antibody to their kits (and ADV as well) the course of the disease is modified and the kits will

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show signs of classical disease after a few months. And if newborn non-Aleutians are infected with low virulent strains, the kits probably become carriers of ADV. The complex relationship between the mink's age and genotype with high and low virulent strains of ADV remains to be determined by molecular virologists.

There will not be a serious effort to develop a vaccine for AD in the near future. First, it was shown by Porter et al. (1980) that mink immunized with formalin inactivated crude tissue vaccine developed the disease more rapidly when challenged with live ADV than unvaccinated control mink. The increased accumulation of viral antibody complexes in the kidney and other vascular locations led to the earlier deaths of the vaccinated mink. Furthermore, if specific antibody produced by the vaccine combines with the challenge virus, there is a suggestion this complex might promote an ADV attack on mink macrophages (Porter et al., 1980; Bloom et al., 1994). Finally, one would be concerned about the validity of the CIEP test if antibody against a vaccine produced a positive test.

Research using live mink is expensive, cumbersome and requires a great deal of time. The definition of susceptible cell types and the propagation of ADV cell culture by Porter et al. (1977) in which a parvovirus was isolated ushered in new areas of research, particularly molecular biology. While cell cultures will not replace live mink in many experiments, they are a very valuable tool. Bloom and his former and present coworkers (Bloom et al., 1994) are using molecular procedures to (1) sort out strains of AD with high and low pathogenicity and virulence; and, (2) relate genomic differences of isolates to clinical disease such as neurotropic AD in adults and fulminant interstitial pneumonia in newborn kits. Perhaps DNA amplification such as the PCR will lead to sensitive tests on the farm to detect infection, although the studies done to date suggest that this approach has limited usefulness. Recent studies defining the AD virus particle by using cyroelectron microscopy may yield insights as to why the interactions of ADV with the immune system of mink are so unusual (Bloom, 2000). While the molecular approach to the study of ADV may lead to novel vaccines, there is nothing on the horizon at this time.

Because of the complex interplay between the genetic factors of the mink and the different viral strains, AD is a fascinating disease to study. The pathogenetic mechanism where all Aleutian mink infected with any strain of ADV succumb is an enigma. However, since all Aleutian mink have the Chediak-Higashi syndrome, it is tempting to speculate that phagocytic cells with their big bags of lysosomes do not destroy the AD virus but allow the virus to replicate (Gorham et al., 1965). On the other hand, the ADV strain itself may be the most significant factor in the pathogenesis of disease in non-Aleutians. Why does the Utah-I strain cause overwhelming disease and death in non-Aleutian mink and the Pullman strain causes infection but does not lead to disease (Hadlow et al., 1983, 1984, 1985)?

No one can answer the question which is crucial to the epidemiology of AD. Why does ADV persist in the lymphoid tissues in mink? That Hadlow demonstrated the virus in the mesenteric lymph nodes of an infected non-Aleutian mink two years after exposure confirms the persistence of ADV.

Obviously, persistently infected mink must be removed from the herd because they are a continuing source of disease. If we consider mink that have AD antibody by CIEP as virus carriers, AD can be eventually eradicated from a farm (Cho and Greenfield, 1978). The disease has been eradicated from Iceland by pelting all mink, disinfection and purchase of AD-free mink. Danish mink breeders using the program (pelting CIEP reactors, disinfection and purchase of new AD-free breeder mink) established by Hansen have been very successful. In 1995, 90 percent of 2,500 farms were free of the disease (Hansen, 1995).

The iodine agglutination test IAT (Mallen's) is a useful non-specific test for hypergammaglobulinemia that accompanies ADV infection (Henson et al., 1962). However, the level of gamma globulin must be at a level of more than 2 grams per 100 ml for the test to become positive. Thus, the IAT does not distinguish the population of subclinically infected mink on any farm. Furthermore, the IAT is a non-specific test and detects elevated gamma globulin levels irrespective of cause. As an example, avian tuberculosis and abscesses cause increases in gamma globulin that would be considered positive for AD.

It is fun to speculate about the history of AD strains as long as one is not taken seriously. In the states of Oregon and Washington, AD smoldered undiagnosed because dark mink were infected with the low-virulent Pullman ADV strain. Both vertical and horizontal transmission maintained the disease with some loss in production but little else. However, when the highly susceptible Aleutian

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genotype appeared in Oregon, the Pullman and all other strains of AD readily infected and caused the death of Aleutian mink.

Speculating on the history of the highly virulent Utah-I strain is more hazardous. In the 1930's and 1940's, the Utah mink farmers were more concerned with a proper diet and the devastating outbreaks of distemper and botulism than the undiagnosed cases of AD. But mink farming is highly concentrated in Utah and as the production of non-Aleutians increased, transmission between farms of the Utah-I strain expanded with a consequent increase in the number of outbreaks.

Thus, from the early days of mink farming, we have had to deal with the Utah-I strain that kills both Aleutian and non-Aleutian mink. There is no doubt that investigators in all mink farming areas have observations they could tell about the epidemiology of AD.

CIEP testing detects AD infected mink and currently provides the best means of control (Cho and Greenfield, 1978). The test is being applied aggressively on Danish farms and there is hope for the eventual eradication of AD in Denmark. The IAT (Mallen's test) will only distinguish infected mink with hypergammaglobulinemia. Since other AD infected mink that are carriers will not be detected, the use of the IAT offers no hope for eradication of AD on a farm. Moreover, a farm that has subclinically infected mink must be considered a potential source of the disease for nearby farms.

John R. Gorham

This investigation supported by the Mink Farmers Research Foundation

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EFFECT OF EXTRUSION TEMPERATURE ON FEED DIGESTIBILITY

Some of you have inquired of me whether putting mink diet ingredients through an extruder would increase their digestibility, and the following is a report on the topic from Norway - presented at the International Scientific Congress on Fur Animal Production. Kari Ljokjel and Anders Skrede from the Agricultural University of Norway, at Ås, prepared a feed mix on a Bühler EX-50/134 double-screw extruder at three different temperatures: 100°, 125° or 150°C. The composition of the feed mix was: fish meal 50.4%, wheat meal 30.2%, fish oil 19.2% and vitamin/mineral mix 0.2%. After extrusion, the feeds were coated with fish oil in a vacuum coater, while the unextruded feed had a similar amount of fish oil added. The prepared feeds were fed to groups of 10 each adult, standard dark male mink. One group received the diet unextruded as a control. Digestibility of dry matter, N (protein) and starch was measured (Table 1).

Table 1: Total Digestibility by Mink of Extruded & Unextruded Feeds

Temperature:	0	100°C	125°	150°
Item:	(control)			
Dry matter	86.3	82.6	81.7	83.2
N (protein)	93.1	91.4	90.9	90.8
Starch (CHO)	96.4	99.1	98.5	99.4

The extrusion process, at the temperatures shown, decreased the digestibility of the nitrogenous part of the diet but increased that of the carbohydrate protein (starch). Since mink are known to digest starch poorly, extruding mink diets may pay off in improved digestibility of the starchy ingredients. Economic studies are needed to show whether such improved digestibility is cost-effective. (from: Ljokjel and Skrede. 2000. Effect of feed extrusion temperature on digestibility of protein, amino acids and starch. Proc. VIIth Int. Sci. Congress in Fur An. Prod. **Scientifur** 24(4):3-6.)

NUTRIENT EXCRETION & MANURE MANAGEMENT

Increasing governmental regulatory activity directed at maintaining the quality of the environment directs that the fur industry look carefully at ways by which excretion of nutrients into the soil can be lessened. In a most interesting paper on the broad issue of nutrient management, Dr. Kirsti Rouvinen-Watt, of Nova Scotia Agricultural College, makes some pertinent suggestions about nutrient excretion and manure management. She notes that in mink production, about 80% of the feed nitrogen is excreted in the urine and about 90% of the phosphorus losses end up in the manure. Peat moss under the cages has improved the recovery of nitrogen from manure and the binding (to prevent losses) of ammonia. **Sphagnum** peat moss has been found to bind ammonia much more effectively than barley straw, oat straw, shavings and sawdust. Composting mink wastes, including manure, feed waste and carcasses, has been shown to be effective, using sawdust as an energy source and commercial applications of these procedures may come in the not-too-distant future (from Rouvinen-Watt, Nutrient Management in Carnivore Fur-Bearers. 2000. **Scientifur** 24(4):25-35).

EFFECTS OF STAGGERED CAGING ON REPRODUCTION

Mink producers have developed caging layouts for their animals over many years' experience, but research on them is continuing. A recent, interesting experiment from the Danish experiment station at Holstebro looked into possible benefits from caging reproducing females in alternate cages; that is, leaving an empty cage between each pair of females. Results showed that females placed in every second cage weaned more and larger kits and had lower kit losses from birth to weaning. Females housed this way were also less reactive and stayed in the nest boxes for longer periods. It appears that having fewer female mink in a shed had a positive effect on female welfare and this deserves further study. (from: Overgaard, L. 2000. Effect of an empty cage between female ranch mink in the reproduction period. **Scientifur** 24(9):25.)

NURSING SICKNESS

With the breeding season not far off, it is appropriate to think about one of the leading losses of both females and kits – so-called **nursing sickness**. This happens when the nutritional demands for the maintenance of the litter surpass the body reserves of the female. The symptoms of nursing sickness are weight loss, loss of appetite and severe dehydration, and the females usually die shortly after the symptoms appear. Older females seem more affected than young ones, and large litter size is an important contributor to the disease. There is a severe weight loss: whereas normal females lose about 14% of their body weight during lactation, those suffering from nursing sickness will lose over 30%. What to do about nursing sickness? It is most important to keep the females eating, by providing enough of a palatable and nutritious diet, and to ensure they are drinking plenty of water (from: Rouvinen-Watt, K. 2000. Nutrition management in carnivore fur bearers. **Scientifur** 24(4):29-30).

FERMENTATION AND HEATING IMPROVE CEREAL DIGESTIBILITY

Wheat and barley fed as the only cereal ingredients to adult mink were inoculated with lactic acid bacteria (fermented), autoclaved (heat-treated) or subjected to both fermentation and heating. Fermentation and heating together improved the carbohydrate digestibility of the cereals more than heating alone (11% higher digestibility). Fermentation also decreased both fecal moisture and total feces which is important to manure disposal. In this study, conducted in Norway, the investigators concluded that lactic acid fermentation of wheat and barley products had beneficial effects on digestibility and on intestinal performance in mink (from Skrede, A., G. Skrede and S. Sahlstrom. 2000. Effects of lactic acid fermentation and heat treatment of wheat and barley on digestibility in mink. **Scientifur** 24(4):40).

RESTRICTED FEEDING AND "STICKY KITS"

The practice of restricting the feed to female mink before breeding ("flushing") has been found to increase litter size, and is widely followed on commercial mink ranches. A recent Danish study suggests, however, that there may be some side effects. In four mink ranches, in 1994, the number of litters including "study kits" was larger among the restricted group than in control females fed the same diet composition; however, the difference was not statistically significant. The authors concluded that restricted feeding might contribute to the "sticky kit" problem, and urged that ranchers be on the alert for this (from: Moller, S.H. and M. Chriel. 2000. Health effects of feeding strategies in the pre-mating and gestation periods of mink. **Scientifur** 24(4):57-58).

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