Fur Animal Research

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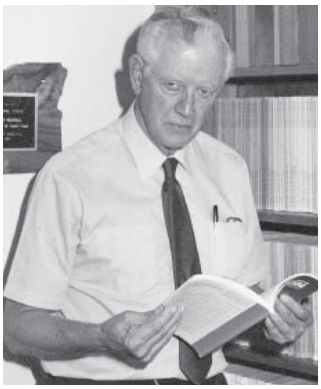
The Fur Industry is a global operation; we benefit from research done in many other countries and they benefit from our American fur research. My old home country of Canada has a long history of fur production and a strong program of research to back it up.



Dr. Jim Oldfield

There have been a number of important Canadian fur research scientists and I have commented on their work in earlier issues of this newsletter. Today, one of the outstanding Canadian fur scientists is Dr. Kirsti Rouvinen-Watt, who is Professor of Carnivore Nutrition and Physiology at Nova Scotia Agricultural College in Truro, Nova Scotia, Canada. She has a Ph.D. degree from the University of Kuopio in Finland – another country with a strong interest in the business of fur production. Dr. Rouvinen-Watt maintains her Finnish contacts as an External Examiner of graduate students at the University of Kuopio and the University of Helsinki. Although much of her research has been done with mink, she has also advised graduate students working with marten, foxes (both silver and blue), polar bears and sheep. Some of her graduate students have worked with the use of cage resting shelves (which we described in our last issue), various feed ingredients in mink diets, including protein produced from natural gas, kelp, ginseng and reishi mushrooms and disorders including nursing sickness and nematode parasitism. Certainly, we have reason to be grateful to Dr. Rouvinen-Watt for her extensive and thoroughly useful program of research.

Kirsti Rouvinen-Watt

We provide some of the financing for Dr. Rouvinen-Watt's research, and she has sent the following update on what she is doing through out Secretary, Dr. John Easley.

RECENT PROGRESS IN RESEARCH PROGRAM ACTIVITIES

Blood Sugar Regulations in Mink Females During the Reproductive Cycle

Nursing sickness, the largest cause of death and culling in adult mink females, is a metabolic disorder that occurs when the demands for lactation require extensive mobilization of body energy reserves. The high blood sugar and insulin levels observed in the sick dams are not clinical symptoms but likely causative factors with a strong parallel to acquired insulin resistance. Although mink nursing sickness manifests itself during lactation, our findings indicate that disruption in glucose homeostasis is observed prior to and throughout the reproductive cycle and is not solely a result of lactation demands. Breeder females that are in non-ideal body condition (too thin or heavy) show higher variability in their blood sugar regulation, while poor glycemic regulation at

the time of breeding and during gestation is linked to reduced litter size and increased mortality of the dams. Moreover, a short-term treatment of hyperglycemic mink dams with antidiabetic, antioxidant and/or anti-inflammatory agents effectively restores normoglycemia during late lactation.

Effects of Body Condition on Lipid and Glucose Metabolism in Mink

The farmed mink experience large seasonal changes in body condition with pronounced body fat deposition during the fall to achieve maximum size, followed by slimming down for breeding early in the spring. Both obesity and lack of adipose tissue are known risk factors for the development of insulin resistance. To better understand the role of body condition as a contributing factor, we have studied the impacts of autumnal fattening on the regulation of lipid and glucose metabolism. Mink kits fed at 120% of recommended dietary allowance (RDA) developed hyperglycemia with the highest insulin levels. The suppressed LPL gene expression in the subcutaneous adipose tissue in the mink fed at the higher feeding intensity levels is evidence that the adipose tissue LPL may have become insulin resistant due to the development of obesity. The lower LPL gene expression also reduced the postprandial clearance of triglycerides from the bloodstream and may have contributed to the development of hyperglycemia and (hepatic) insulin resistance.

The blood glucose and insulin levels are affected by a number of factors, including oxidative stress. In nursing females, the level of oxidative stress, as measured by DNA strand breaks, was shown to increase from early to late lactation. In addition, during lactation, when mink dams were fed n-3 long chain polyunsaturated fatty acids (LCPUFA), a nearly significant increase in the uncoupling protein 2 (UCP2) gene expression was observed in the subcutaneous fat. In the n-3 enriched dams, blood insulin levels were positively correlated with blood leptin and mammary gland LPL and white adipose tissue LPL gene expression. The n-3 LCPUFA enriched dams also had significantly lower blood sugar levels indicating improved insulin signaling despite rapid body fat mobilization.

Mink Fatty Liver Disease – Causes and Metabolic Consequences

Most recently, we have carried out two feeding and fasting experiments in mink in order to study the time scale of the development of fatty liver and investigate the effects of feeding intensity and dietary fatty acid supply on the development of liver pathology. In the first experiment we studied the time scale of development of fatty liver in the mink in response to a period of fasting and re-feeding. Sixty farm bred standard black mink (30 male, 30 female) were fasted for a period of 0, 1, 3, 5, or 7 days and one group was re-fed for a period of 28 days after a 7-day fast. The mink were weighed before and after fasting. The livers were weighed, their fat content analyzed and they were histologically evaluated and graded for the type and severity of lipid accumulation using light microscopy. All except one mink in the 3-7 day fasted groups had moderate to severe macrovesicular hepatic lipidosis ($P<0.001$). Liver fat content (5.9% vs. 13.2%, SEM=1.5, $P<0.001$) and the hepatosomatic index (males: 1.1 vs. 1.7, SEM=0.2, $P=0.013$, females: 2.7 vs. 3.6, SEM=0.2, $P<0.001$) were also significantly elevated by day 3 in comparison to the non-fasted control. The livers showed recovery after the re-feeding period with the histomorphology, liver fat percent (5.9%±1.6), and hepatosomatic index

(males: 1.3±0.2, females: 2.9±0.2) not different from the control. Our results show that in mink, in response to short term food deprivation, moderate to severe hepatic lipidosis develops very rapidly, in as few as 3 days; however, lipid regeneration is possible even after the development of severe macrovesicular steatosis.

In the second experiment, the objective was to investigate the effects of dietary fatty acid supply and feeding intensity on the development of fasting-induced fatty liver in mink. Seventy-two (72) mink, 36 male and 36 females, were fed diets with canola oil (n-9), herring oil (n-3) or soybean oil (n-6), at two different feeding intensity levels (80%, 120%). Half of the mink were fasted at the end of the experiment. Overall, the highest liver fat content (15.7±1.3%) was observed in the n-6 120% group, whereas the lowest (6.6±1.3%) was found in the n-6 80% mink (P=0.035). When considering the interaction of feeding intensity and fasting (P=0.023), the highest liver fat content (21.7±1.0%) was found in the 120% fasted group with the lowest fat content (6.0±1.0%) in the 80% non-fasted mink. With regards to percent weight loss during fasting, the 80% fasted mink had a higher loss (15.0±0.4%) than the 120% fasted mink (11.9±0.4%) (P<0.001), and the fasted females lost more weight (14.8%±0.4%) than the fasted male mink (12.1±0.4%) (P=0.003). It is evident that the development of fatty liver in mink in response to short term food deprivation is influenced by the level of feeding intensity.

Future Direction of Research Program

The research will now continue with the analyses of the fatty acid composition of various adipose tissue depots and organs of these mink, the analyses of the hematological, and serum clinical-chemical and endocrinological profiles, further liver histology and immunohistochemistry, as well as the molecular analyses of the levels of messenger RNA encoding for key enzymes in the pathways regulating lipid and glucose metabolism in the liver, skeletal muscle an adipose tissue. The long term goals of my research program are to better understand the underlying biology of the metabolic disorders of mink nursing sickness and the associated fatty liver syndrome.

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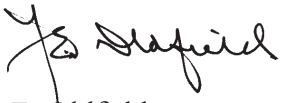
I wish you all a successful growing/furring season in this year's mink operations.



J. E. Oldfield

PSEUDOMONAS PNEUMONIA IN MINK IN ROGALAND, NORWAY – MEDICAL AND PRACTICAL ASPECTS THROUGHOUT AN OUTBREAK IN 2006

by Gorm Sanson1, Michaela Falk2

Pseudomonas (Ps.) aeruginosa pneumonia in mink was not reported in Norway from late eighties till 2002, more than 15 years.

At Christmastime 2005, seven farms in Rogaland got Ps. aeruginosa infections nearly simultaneously. Infected mink were treated with sulfadiazine/ trimethoprim (Tribrissen® vet. Pulver til fisk (fishpowder)). Five farms got increased mortality rate again after treatment. In these five farms a new Tribrissen®-cure was initiated combined with vaccination with a commercial vaccine (Febrivac® 3 Plus.) In one farm (farm1) Tribrissen® did no longer reduce mortality and was substituted with Terramycin® (oxytetracyclin) to reduce the infection pressure during and shortly after vaccination. Terramycin had good effect, but Farm1 lost more than 25% of the mink in one house (600 out of 2000 minks) during January.

In early July 2006 farm1 got a new outbreak of Pseudomonas pneumonia. Due to the experience at Christmastime, treatment with Terramycin® was initiated. This time Terramycin® didn't work and was quickly replaced with Tribrissen® - with very good effect. Controlling mortality with Tribrissen®, the farmer started to vaccinate. Unfortunately, there seemed to be no effect of the commercial vaccine (Febrivac® 3 Plus). At the 20th of July another farm (farm2) got an outbreak of Pseudomonas pneumonia. This farm also had the infection at wintertime and therefore already vaccinated mink kits using Febrivac® 3 Plus. Again, a total failure of the commercial vaccine, and Tribrissen® was the only way to control mortality. A third farm (farm3) got an outbreak of Pseudomonas pneumonia at the beginning of August. Farm3 had no outbreak at wintertime and therefore yet not vaccinated against the bacteria. Due to the lack of effect of Febrivac® 3 Plus in farm1 and farm2, it was decided not to vaccinate farm3 with the commercial vaccine.

After the failure of the commercial vaccine, bacterial cultures isolated from mink that died from Pseudomonas pneumonia, were sent to the vaccine-producer IDT (Impfstoffwerk Tornau-Dessau), Germany, in early August. The idea was to have IDT producing an auto-vaccine in order to control the outbreak – a process taking at least 5 weeks.

Medication with Tribrissen® was effective till about September 10th. There was no possibility to intersperse periods without treatment to reduce the risk for the development of resistances. About September 10th the mortality rate raised dramatically, especially in farm3. The application of Terramycin instead of Tribrissen did not improve the effect. There died up to 500 mink per day in farm3 after September 15th.

In late September all three farms started vaccination with the auto-vaccine (Febrivac® Pseudomonas) produced by IDT, which had effect immediately. Within three to five days there was a considerable decrease in mortality. After five to seven days there was no longer mortality because of infection with Ps. aeruginosa on these farms. Total losses in the three farms were 18% of all mink farm1, 15% in farm2 and 50% in farm3.

From September 13th to December 30th fourteen more farms were diagnosed with Ps. aeruginosa infection. Vaccination with new vaccine was successful in all cases.

Discussion

- All farms obtain feed from the same feed kitchen (Rogaland Pelsdyrfôrlag AL).
- Adversarial in use of Tribrissen® fishpowder is its hydrophobia, which is caused by its adjuvant, the flour of limestone. Consequently, an inconstant dispensation is a possible reason for the ineffectiveness of Tribrissen® in at least one case (farm1).
- A combination therapy with Terramycin® and Tribrissen® had to be portioned on two feeding times because a watery mixture of Terramycin® and Tribrissen® resulted in a heavy foam production.
- The commercial vaccine seemed to have sparse or no effect, but on the unvaccinated farm (farm3) the loss of mink was highest (50%)
- Time is a crucial factor in the treatment of Pseudomonas pneumonia. In one later case, there was no Tribrissen® available, and Terramycin® was prescribed. After two days with no effect of treatment, we got Tribrissen® airborne from Denmark. At this point animals were dying all over the farm. Tribrissen® stopped the mortality within 3 days, but the farm got a total loss of 14%.
- There seem to be something weird with sensitivity reactions of Pseudomonas aeruginosa in vitro and its actual sensitivity to antibacterials in vivo. For instance, the bacteria always seems to be sensitive to oxytetracyclin in vitro, but Terramycin® very seldom had any good effect in vivo. Over the past few years we have seen several cases of poor sensitivity to sulfadiazine/trimethoprim in vitro, but still Tribrissen® works well in vivo.

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MINK AND "MAD COW DISEASE"

Following the outbreak of mad cow disease in Alberta, Canada and after the first case of mad cow disease appeared in Washington in December 2003 and in another case in Texas in June 2005 and in Alabama in March 2006, mink ranchers became concerned as to the susceptibility of mink to the disease.

Mink can be infected and will succumb if they eat meat from cattle that have mad cow disease or eat rendered cattle tissues containing the mad cow agent.

Mad cow disease, which is called by pathologists bovine spongiform encephalopathy or simply BSE, probably has occurred in England and Scotland before it was first diagnosed in 1986. Cattle become infected by eating rendered bone and meat meals that harbor the BSE agent. BSE is caused by a unique agent called a prion that resembles a virus in many ways. This agent is very resistant to the heat and solvents of the rendering process.

After an incubation period from two to eight years, infected cattle have a slow progressive downward course characterized by nervousness, aggression,

incoordination and loss of appetite. Often the affected cows lose their balance and strength and fall. They are termed "downer cows." All affected cows die or are killed prior to death.

Currently, there is no test to detect BSE in a live cow. It is diagnosed by blood and tissue tests in a laboratory after a cow dies. The BSE brain has a characteristic perforated spongy appearance. This is why the disease is named spongiform encephalopathy.

During the period from November 1986 (when BSE was first diagnosed in England) until December 1995, 155,600 head of cattle were diagnosed and died or were killed because they had symptoms of BSE.

After the British officials prohibited the inclusion of rendered ruminant-derived proteins that potentially could carry the BSE agent, the cattle losses dropped to less than 300 per week.

While the number of English and Scottish mink farms decreased markedly in the 1970s and 1980s, there still was a slim chance that a mink farmer might have fed some products containing BSE or BSE contaminated meat meal or bone meal in the cereal part of the ration. British veterinary pathologists who have first knowledge of all aspects of the disease did not know of any mink outbreaks that could have been caused by BSE.

EXPERIMENTS AT WASHINGTON STATE UNIVERSITY

Dr. Mark Robinson of the U.S. Department of Agriculture at Washington State University and his coworkers fed mink BSE tissues and injected BSE tissue suspensions directly into the brains of mink. Nervous signs appeared about 12 months after the BSE agent was injected into the minks' brains and more importantly, 15 months after feeding the BSE agent. Of course, the natural route of transmission would be by mouth. Prior to death the mink lost their appetites and became lethargic and uncoordinated. Microscopic examination of their brains showed the characteristic spongy changes. Thus, it is highly likely that mink are susceptible to BSE when fed by-products containing the BSE "mad cow disease agent."

As all veterinarians familiar with cattle diseases know, a cow can go down and not get up for a variety of causes. These include terminal states of bacterial and nutritional conditions and trauma to pelvic nerves during delivery of a calf.

Mink farmers who have fed downer cows to their mink must be aware that a cow "goes down" for a reason.

CHRONIC WASTING DISEASE (CWD) IN DEER AND ELK

Because CWD is closely related to mad cow disease (but not the same), it is suggested that deer and elk carcasses not be fed to mink.

Deer affected with Chronic Wasting Disease, which occurs in some mink farming areas.

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EFFECTS OF CLIMBING CAGES AND GROUP SIZE ON BEHAVIOR AND PRODUCTION IN JUVENILE MINK

Abstract. The effects of climbing cages and group size on behaviors and bite marks were studied in 330 juvenile mink. Mink of the color types Demi buff and half Sapphire were housed from weaning in pairs in standard cages, in pairs in climbing cages, 2 females and 1 male in climbing cages or 2 females and 2 males in climbing cages. Behaviors were observed during sun rise and sunset from July until November. In November an optical inspection for bite marks on the leather side were made. The occurrence of stereotypies were very low. Pair housed mink in standard cages showed no differences in behaviors or frequency of bite marks compared to pair housed mink in climbing cages. There was an increase in aggression and more bite marks as a consequence in group housing were not observed. The group size, though, affected behaviors. Mink in groups were more active out in the cage, used the enrichments less and spent less time in the nest box than pair housed mink. In conclusion our study showed that an increase in cage size and complexity of cage environment did not affect behaviors or frequency of bite marks in juvenile mink housed in pairs and the frequency of bite marks did not increase in group size of 3 or 4 individuals.

Introduction. Housing conditions for farmed mink has been thoroughly discussed in Sweden during the past years, and several reports have been written (SOU 2003:86; DS 2005:32). According to Swedish animal welfare legislation, animals kept for production shall be housed in a way that allows them to behave naturally (4 § SFS 2003:1076). Providing the opportunity for those natural behaviors that satisfy the needs of the animals should be considered when new housing systems are being developed (Lidfors et al. 2005). Farmed mink has been found to show abnormal behaviors such as stereotypies (Blidsøe et al. 1990a; Mason, 1994). Several studies have shown that environmental factors such as enrichments can decrease stereotypies (Hansen, 2007). Climbing cages could offer a better welfare for farmed mink because it provides a more complex environment for the mink that can stimulate both physiological and physical needs. Climbing cages increase the total available space for the animals when housing juvenile mink in groups. However, mink is a solitary animal (Dunstone, 1993). Thus, aggression may be a problem in group housing and is of importance when concerning the welfare of farmed mink. In this study we investigated the effects of group size and climbing cages on behaviors and bite marks, on the leather side, after pelting in juvenile mink.

Materials and methods. The study was carried out at a private farm in the south of Sweden. In total 330 juvenile mink were used in the study. Mink of two color types were selected and were represented in each treatment, 165 Demi buff and 165 half sapphire, to investigate possible differences in aggression between color types. Mink were housed from July until pelting in November, in two kinds of cages and assembled in different groups:

(1) 1 female and 1 male in standard cages (S)

(2) 1female and 1 male in climbing cages (C2)

(3) 2 females and 1 male in climbing cages (C3)

(4) 2 females and 2 males in climbing cages (C4)

The size of the standard cage was (length 90, height 45, breadth 30) with a total floor size of 2550 cm2. Standard cages were provided with a wire net shelf and plastic cylinder placed on the floor of the cage. The size of the climbing cages were: bottom cage (L 90, H 45, B 30) and top cage (L 55, H 45, B 30) with a total floor size of 4250 cm2 (Fig. 1). Climbing cages were provided with the following enrichments, one wire

net shelf in the bottom cage and one in the box where mink in groups of 3 or 4 were less in nest box than pair house ($p<0.001$). No effects of group size were found on stereotypies (n.s.).

Females were more active out in cage ($p<0.001$) and showed a tendency for a higher frequency of stereotypies ($p = 0.10$) than males. Females also showed a tendency of more time spent in nest box ($p = 0.10$). Time spent in nest box increased over time ($p < 0.001$) and when fed ad libitum ($p < 0.001$) in all treatments. Males used the plastic cylinders more than females ($p < 0.001$). Stereotypies increased over time ($p < 0.001$) and were less frequently performed when fed ad libitum ($p < 0.01$). No differences were found between color types concerning behaviors (n.s.).

No difference in bite marks between cage types (n.s.) or group size were found (n.s.), table 1. More females had bite marks than males, table 1. Female individuals had overall more bite marks and the bite marks were more severe than males in both cage types and in all group sizes, table 2.

	Cage type and number of animals/cage (n)	Male %	Female %
1/2 Sapphire	Standard cage (2)	27	67
	Climbing cage (2)	7	60
	Climbing cage (3)	0	73
Demi-buff	Standard cage (2)	13	60
	Climbing cage (2)	20	40
	Climbing cage (3)	20	50
	Climbing cage (4)	13	53

Table 1. Percentages of mink with bite marks on the leather side, in different cage types and in different group sizes shown for the different sex and color types. An optical inspection was done in November. Pair housed (2), two females one male (3) and two females and two males

Discussion and conclusions. In our study, pair housed mink in standard cages showed no difference in behaviors or frequency of bite marks compared to pair housed mink in climbing cages. This indicates that the size and complexity of the climbing cage did not affect juvenile minks, behaviors or frequency of bite marks, when housed in pairs. We also found that the occurrence of stereotypies were very low

Location of damages	Female % (S) ½ sapphire/ demi buff	Male % (S) ½ sapphire/ demi buff	Female % (S) ½ sapphire/ demi buff	Male % (S) ½ sapphire/ demi buff	Female % (S) ½ sapphire/ demi buff	Male % (S) ½ sapphire/ demi buff	Female % (S) ½ sapphire/ demi buff	Male % (S) ½ sapphire/ demi buff
Neck	40/47	0/0	53/40	0/0	53/47	0/7	23/47	7/3
Upper								
abdomen	7/13	20/7	0/7	7/7	0/7	0/0	0/3	0/3
Lower								
abdomen	7/0	0/0	0/0	0/0	3/0	0/0	0/0	0/0
Back	13/20	0/7	27/13	0/0	20/20	0/7	7/27/	10/7
Hips	20/7	7/0	0/7	0/0	3/0	0/0	3/10	0/0

Table 2. Percentages of mink with different kind of damages on the leather side, in different cage types and in different group sizes shown for the different six and color types. An optical inspection was done in November. One female and one male in standard cages (S), one female and one male in climbing cages (C2), two females and one male in climbing cages (C3), and two females and two males in climbing cages (C4).

in juvenile mink. Group housing of mink provides more individual and space and increase the physical complexity in their environment. Although mink are solitary animals, group housing may function as a social enrichment. However, increased aggression and bite marks have increased when mink were housed in groups (Pedersen et al. 2004). In our study we did not find an increased aggression or more bite marks as a consequence of housing juvenile mink in groups of 3 or 4 individuals. Females had more bite marks than males and had a higher frequency of neck bites and bite marks on their body. Bite marks on the body were mostly located around the tail area. This may indicate that the males performed neck biting as a mating behavior and were more aggressive than females. The group size affected the activity out in the cage, the use of enrichments and time spent in the nest box. Mink in groups of 3 or 4 individuals were more active out in cage, used the enrichments less and spent less time in nest box than pair housed mink. Group housing of mink may create more social play behavior and may also engage mink in more exploratory behavior, when housed in a more complex social and physical environment, which the climbing cage provides. Females were more active out in the cage and showed a tendency of performing more stereotypies. Activity has been shown to correlate with stereotypies (Blidsøe et al., 1990b; Hansen and Jeppesen 2001). Differences in activity between females and males may be explained by differences in physiology. Females in the wild also spend more time foraging per day from August until October (Dunestone, 1993). Stereotypies were more frequent before feeding. Females showed a tendency to spend more time in nest box and males spent more time in cylinders. Males mainly used the cylinders for resting.

In conclusion, our study showed that an increase in cage size and complexity of the cage environment did not affect behaviors or frequency of bite marks in juvenile mink housed in pairs. The frequency of bite marks did not increase in group sizes of 3 or 4 juvenile mink. Potential increase in aggression should, however, be considered when housing mink in groups.

Mink Farmers' Research Foundation Board

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