Fur Animal Research

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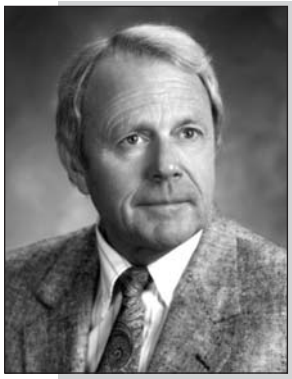


Dr. Bob Westlake

It is always sad to record the passing of one who has been an important part of the Fur Industry, for many years, as was Dr. Robert Westlake, who died in December of last year. Bob was a member of the Ranch Services group which for years provided sound veterinary advice for the solution of problems in ranch mink. He got out to the ranches where the problems were - never relying on "armchair diagnoses" from his office. Truly international in his work area, he was as well known in Canada as he was in the U.S. There was never anything stuffy about Bob Westlake; he was fun to be around, and I will miss him. On behalf of all of us, I send condolences to his wife and family in Detroit Lakes, Minnesota.

It is interesting at this time of the year to note the weather conditions in various parts of the country, much of which means extra work for fur farmers. In the midwest, prolonged freezing makes provision of water to the animals difficult - requiring specialized heating equipment. But in the Northwest, where I'm located, we have had no problems supplying water. There's been all we needed, and more. In January, we had 31 straight days of measurable rain which caused a good deal of flooding and isolated some shed areas. To make matters worse, the long-term forecast is for development of an "El Nino" which will bring even more rain.

The Commission of European Communities has developed an Action Plan for the Protection and Welfare of Animals that has some implications for American mink ranchers. They have defined five major areas of action:

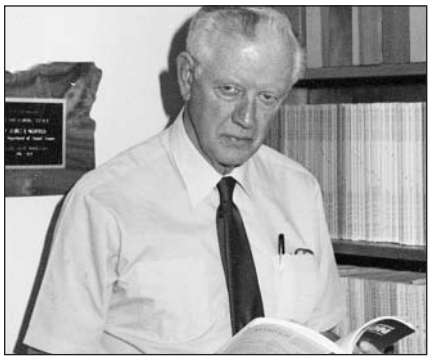
1. Upgrading existing minimum standards for animal protection and welfare in line with new scientific evidence and socio-economic assessments as well as possibly elaborating specific minimum standards for species or issues that are not currently addressed in EU legislation. A particular priority will be designing EU rules in order to secure efficient enforcement and to take account of rules governing international trade.

2. Giving a high priority to promoting policy-orientated future research on animal protection and welfare and application of the 3Rs principle: in order to respect the obligations under the EC Treaty Protocol to pay full regard to the welfare of animals in formulating and implementing these policies in parallel with enhancing the development, validation, implementation and monitoring of alternative approaches to animal testing.

3. Introducing standardized animal welfare indicators: to classify the hierarchy of welfare standards applied (from minimum to higher standards) in order to assist the development of improved animal welfare production and husbandry methods and to facilitate their application at EU and international levels. On this basis, options for EU labeling will be explored in a systematic manner.

4. Ensuring that animal keepers/handlers as well as the general public are more involved and informed on current standards of animal protection and welfare and fully appreciate their role in promoting animal protection and welfare. In respect of farm animals, for example, this could include working with retailers and producers to facilitate improved consumer trust and awareness of current farming practices and thus more informed purchasing decisions, as well as developing common initiatives in the field of animal welfare to facilitate the exchange of information and the application of best practices.

5. Continue to support and initiate further international initiatives to raise awareness and create a



greater consensus on animal welfare, including engaging with Developing Countries to explore trade opportunities based on welfare friendly production systems. The Community should also actively identify trans-boundary problems in the area of animal welfare, relating to companion or farm animals, wildlife etc., and develop a mechanism to tackle them in a more timely, efficient and consistent manner.

As I write this, you will be readying your pelts for the February sales. I wish you all success and hope last year's strong prices will continue.

J. E. Oldfield

SALMONELLOSIS OF MINK

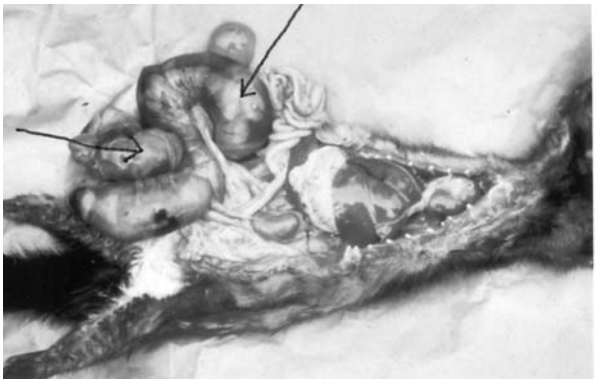
This paper deals with the role of Salmonella, a microscopic rod-shaped bacterium, as a disease producing agent in mink. Salmonella is infrequently found in mink submitted for post-mortem examination at this laboratory at Washington State University. Salmonella isolations are also rare in German ranch-raised mink. H-Chr.Loliger recorded Salmonella organisms in 25 of 1,602 mink (1.6%) received for autopsy at the Federal Research Station located at Celle, West Germany.

Salmonella as a Cause of Abortion

With the exception of abortions, it is difficult to relate the occurrence of salmonellae in mink to other clinical signs or autopsy findings. Dr. G.R. Hartsough reported outbreaks in the springs of 1946, 1947 and 1960. There were few deaths in the pregnant females but many kits were aborted and lost. A number of aborting females were killed and examined bacteriologically and in every instance, Salmonella choleraesuis var. Kunzendorf was isolated from the uterus and un-expelled fetuses. In some instances, necrotic metritis (inflammation of the uterus with tissue death) and peritonitis was observed. The source of infection in all three outbreaks was Salmonella-contaminated inedible pork livers.

Inasmuch as normal appearing livers may harbor salmonellae, it is difficult to keep such livers out of the ration. Hartsough suggested that some consideration be given for the elimination of pork livers from the ration during the gestation period.

Loliger reported the first outbreak of Salmonella abortions in German mink. The cause was Salmonella infantis, a species closely related to the one isolated by Hartsough in Wisconsin. Shortly after aborting their young, a number of female mink became ill.



Salmonella: This uterus contains dead kits. Many kits are aborted. (Photo by Dr. A. Larson)

Autopsy revealed metritis with portions of the remaining placenta extending through the uterine wall with resultant gangrenous peritonitis. Pregnant females found infect-

ed with Salmonella early in pregnancy did not abort.

Dr. Austin Larson has described an outbreak of Salmonella abortions that occurred in the Utah area. That Salmonella bacteria can also infect humans was shown in this outbreak. The mink farmer put a kit in his mouth to "warm it up" and infected himself. He became ill and Salmonella Dublin was isolated from his blood.

Salmonella as a Cause of Enteritis

There has been a good deal of discussion concerning this group of bacteria as a cause of enteritis. Most of the mink that have eaten contaminated meat show no signs of disease but may be called carriers. When the salmonellae enter the intestine, instead of invading and causing inflammation of the lining membrane, they pass through the animal with the intestinal contents.

Loliger has pointed out that enteritis has been observed in mink in which Salmonella and coccidian (Eimeria or Isopora) were demonstrated. He was not able to conclude whether the Salmonella or the coccidia was the primary disease.

Investigation carried on at our station showed that normal and semi-starved mink were resistant to experimental rations containing Salmonella. Clinical disease was produced in only 2 of 64 mink. These animals were in a group that received reduced rations for an extended period.

With the exception of agents such as mink virus enteritis, coronavirus, calcivirus and coccidiosis, the causes of enteritis are unknown. It would be foolhardy to overlook Salmonella. It would be well to "leave the back door open" and state that if the conditions are appropriate, i.e., if the resistance of the mink was lowered by some factor, enteritis might occur.

Treatment

Salmonella organisms are susceptible to antibiotics. Since the treatment may vary among outbreaks due to the varying sensitivity of different strains of Salmonella, it would be well to consult a veterinarian regarding appropriate treatments.

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CARBOHYDRATE DIGESTIBILITY BY MINK

Mink are known to digest carbohydrates with difficulty and heat-treatment ("cooking") has been used to improve the situation. In an attempt to assess the effectiveness of the heat treatment, Danish workers at Holstebro have developed a laboratory test. A sample of the feed mix containing the heated carbohydrate is incubated in the laboratory with added enzymes pancreatin and amylglucosidase for 20 minutes and 120 minutes. The amounts of the sugar, glucose, that is released by this treatment allows the designation of the carbohydrates as rapidly (RDS) and slowly (SDS) digestible starch. Finally the sample is boiled, with potassium hydroxide, KOH and completely digested with amyloglucosidase, to give the total glucose content. The test has been modified, so that now the incubation period is simply 20 minutes for both pancreatin and amyloglucosidase, and this simplification was found not to alter the results. Across several cereals (barley, wheat and peas) the content of RDS correlated well with the actual digestibility in the living animals. In principle, it was proposed that this lab test could be used to determine whether or not the heat treatment of the feed had been carried out satisfactorily. (from: H.N. Loerke and C.F. Borsting. 1999. Kan kulhydrat og stivelsesfordojelighed hos mink bestemm es ved anvendelse af en laboratorilmetode? in annual report of research at the Holstebro research station, pp. 15-24).

BLOOD AND BLOODMEAL IN MINK DIETS

Various products made from blood are available on the market as mink feeds. Some of these products have a low digestibility, however, due to heat treatment necessary for sterilization. In Denmark, two blood products were investigated as mink diet ingredients during the spring growing period. The products studied were Blosol, a blood product available in Denmark, and a commercial blood meal. Four groups of mink were used, each consisting of 120 each male and female scan brown mink. Three groups were fed Blosol, at levels of 2, 4 or 6% of the diet and the fourth group got blood meal fed at 6% of the diet. The best results came in the group fed 2% Blosol, which showed the best pelt lengths. Pelt lengths were significantly shorter in the groups fed 4% and 6% Blosol, and in the group fed 6% blood meal. The investigators recommended that these types of blood products be only fed at low levels (ca. 2%) in mink diets. (from T. Clausen and C. Hijlesen. 2003. Blood meal to kits in the growing-furring period. Annual Report, Danish Fur Breeders Research Center, Holstebro, pp. 81-84).

MINK ARE SUSCEPTIBLE TO 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, 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 had 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 (see figure.)

Cow affected with Mad Cow Disease, showing incoordination and early paralysis.

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.

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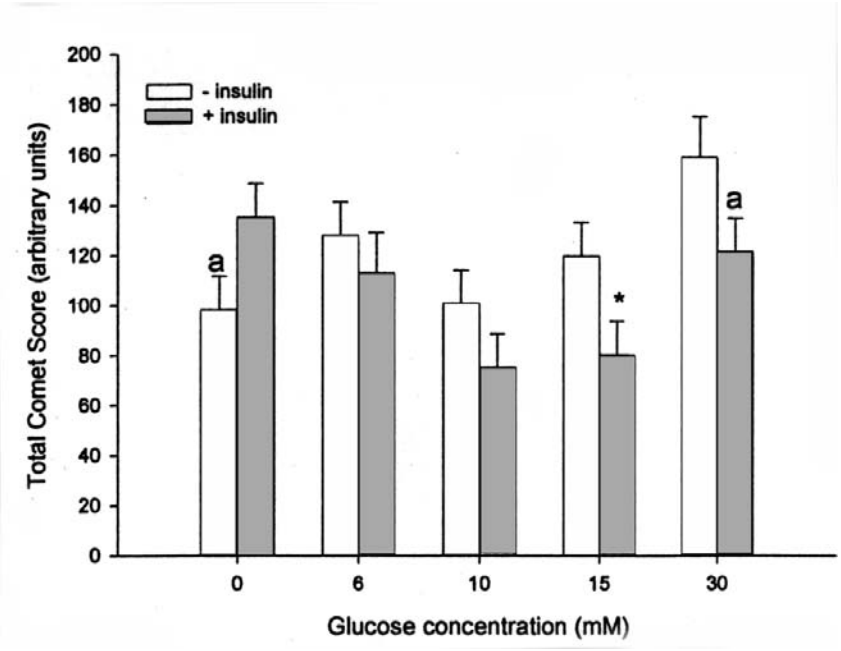
MINK LENGTH, WEIGHT AND PELT SIZE

Studies were conducted in Denmark for three years on organ sizes in light and heavy male mink kits. The animals were weighed and measured and various organs were removed and weighed. It was observed that when selecting mink in November for heavy body size, the kits become a little longer but they also get fatter. The body weight of the mink had a greater influence on the size of the pelt than did body length. Organ weights increased in fat mink more than in mink selected for body length. These observations may be useful in setting selection criteria for producing top quality pelts. (from T. Clausen. 2004. Investigation of organ size in big mink. Annual Report of the Danish Fur Breeders' Research Center at Holstebro. pp. 119-122).

CONTROLLING OXIDATIVE STRESS IN MINK

Animals, including humans, suffer oxidative stress when the balance between highly reactive forms of oxygen and the animal's antioxidative mechanisms is upset. Oxygen can be easily metabolized to form water, which is harmless, but the intermediary steps in this process may yield free radicals and hydrogen peroxide, which can cause stress and destroy some useful compounds in the body.

Nursing sickness is an important cause of mortality in adult mink, and it may be caused by formation of too much glucose (hyperglycemia) which in itself is caused by acquired resistance to insulin, and this may be a cause of oxidative stress. Such stress involves damage to certain intracellular compounds, including DNA which leads to altered cellular function. Investigators in Nova Scotia tried to use what is called the Comet Assay to detect oxidative damage due to increasing levels of hydrogen peroxide. It showed a clear cut increase in oxidative damage by increasing levels of hydrogen peroxide, from 0-1000 moles (M). This could be prevented by the administration of insulin at levels of 10 nM (nanomoles). More research is needed to confirm the suitability of this assay for measuring oxidative stress in nursing female mink, although these preliminary results are encouraging.



Mean total comet score (±SD) in mink leukocytes exposed to increasing concentrations of glucose with and without 10 nM insulin. Difference between least squares means with addition of insulin significant at 15 mM (P=0.041) and marginally significant at 30 mM (P=0.077). At 0 mM of glucose, total comet score was significantly increased with the addition of insulin (P=0.053).

(from R. Garbes and R. Rouvinen-Watt. 2004. Evaluation of oxidative stress in mink using the Comet Assay. Canadian Centre for Fur Animal Research, Nova Scotia Agricultural College, Truro, NS, Canada).

INDEX

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