Fur Animal Research

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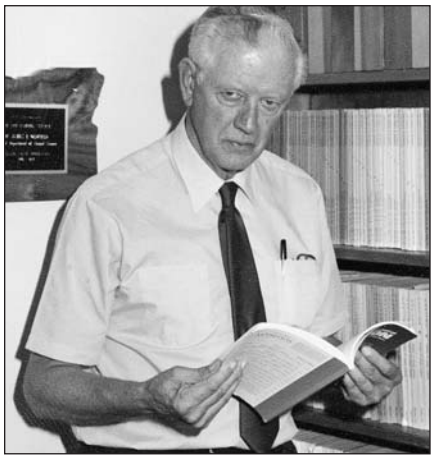

I was reminded of the "Good Old Days" of the fur industry this Fall when Ben Wustenberg enrolled as a graduate student in the Department of Animal Sciences at Oregon State University. Ben is the grandson of Phyllis (Watt) Wustenberg who was an influential figure in the American fur industry during the latter part of the 20th century. Phyllis grew up on a mink ranch in Bay City, Oregon, just north of Tillamook. Armed with a broad knowledge of the fur business, she was in great demand as a speaker on mink matters, both nationally and internationally. Phyllis and her husband, Don, had two sons, Bill and Mark, who both became professional veterinarians. Bill has a practice in Minnesota, and Mark in Tillamook county, Oregon. Ben is the son of Mark and Judy Wustenberg, and Judy has operated a nutrition laboratory, serving the fur farmers, for a number of years. Ben will not be working with mink at OSU, since we no longer operate a fur farm facility, but he will be majoring in animal physiology under the direction of Dr. Jim Males, the Animal Sciences Department Head. So you can understand that seeing Ben brought back many pleasant memories. But it also made me feel old - Phyllis's grandson in graduate school! Wow.

As I think back about what mink ranching was like then and what it is now, I marvel at the many technological advances that have taken place. Truly, the industry has come from an era of guesswork to one of application of scientifically-gathered information and the results are apparent in the beautiful animals that are raised today. My first association with the mink industry was at the University of British Columbia in the late 1940's. There were no computers then and no one would have believed that a few decades later both mink diets and breeding programs would be formulated on such machines. Indeed, the data that the computer needed were not then available.

We send copies of these newsletters to our friends overseas and this brings useful returns, for which we are grateful. Dr. Wilhelm Weiss, of the Danish Fur Breeders' Center at Holstebro, has been particularly helpful, sending me their annual reports and individual research papers from time to time. Some of these reports are in Danish in which I am not fluent, so I am learning a foreign language in the process.

We are fast approaching the Holiday Season, for which I send best wishes and also that most important time in the mink production year - the pelting season. May the fur markets be good to you in 2006.

J.E. Oldfield

DIAGNOSIS OF DISTEMPER IN MINK

When distemper is mentioned, ranchers have cause for alarm as it is the most serious disease found in mink in the United States.

The first report of distemper in mink appeared in a 1932 fall issue of the Black Fox magazine. Before that time many ranchers refused to accept the idea that mink had any contagious diseases. Undoubtedly distemper occurred on many ranches before this first report but these outbreaks were probably diagnosed as some other malady.

It has often been said that before any diagnosis can be made in mink, distemper must first be eliminated as a cause of the disease in question. At the present time, veterinarians have a variety of methods to diagnose this condition in mink. Diagnosis can be made on a clinical, pathological or a serological basis; animal inoculation may also be employed.

Clinical. A mink that is still living is necessary for a clinical diagnosis. If the signs of an illness are similar to those that occur in typical cases of a given disease - distemper let us say - a diagnosis is made. The history of an outbreak is a great help in solving the cause of the outbreak. Distemper does not spread rapidly in adult mink. When an outbreak is just getting underway, a rancher may observe only a dozen infected adult mink in the first two months. Such individuals frequently recover. In kits the story is quite different; the infection will wipe out entire litters within two or three weeks.

A diagnosis by clinical means is not difficult if the veterinarian has an opportunity to observe two or three mink at different stages of the disease. This applies more to adult mink and older kits, as a very young mink often dies so rapidly that signs of advanced distemper do not have time to develop. Nine to fifteen days after exposure in typical cases, the infected mink ha a watery discharge from both of the eyes and the nose. As the disease progresses, this discharge becomes thicker and accumulates as brown granular material that adheres to the skin and completely closes the nostrils and eyes (Figure 1).

Figure 1. An advanced case of distemper. Note the appearance of the eye and the exudates that sticks the eyes shut. The foot pads are also enlarged.

About a week after the eye signs appear, the feet may become swollen two to three times their normal size. On the surface of the foot pads severe inflammation manifests itself as small scabs. This inflammation may extend up to the legs, and, if the animal lives long enough, may cover almost the entire body producing large folds in the skin. In some cases the hair may fall out. Small pustules sometimes are observed between the hind legs. The mink may succumb following convulsions. Many times, however, the animal appears to recover after all the signs mentioned above subside. Such mink may later die of so-called "screaming fits" or neurotropic distemper. In this condition the virus has attacked the brain causing the mink to bite the wire, froth at the mouth and throw itself about the pen. It usually emits a series of shrill screams. The duration of the convulsions usually varies from less than an hour to a couple of days. Frequently, the mink dies after one or two such attacks.

Pathological. By the signs mentioned above under the clinical description, i.e., eye and foot lesions, the diagnosis can be made by looking at the intact animal at autopsy. When the mink dies following neurotropic distemper, external signs of the disease are usually absent.

Animals dying in the fall or winter are often pelted before being sent to the laboratory. If this has been done, it is impossible to diagnose distemper without making microscopic slides or inoculating animals (see below). These slides are made to demonstrate

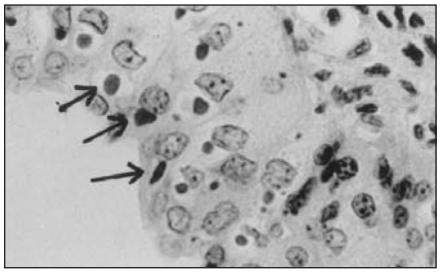

Figure 2. The arrows point to what are called distemper inclusion bodies, as seen under the microscope. These are indicators of distemper in mink, dogs and raccoons.

whether the cells of the bladder or trachea (wind pipe) have inclusion bodies in them. These inclusion bodies are small dots or specks and are considered to be "markers" of the virus (Figure 2). Unfortunately, inclusions can not be demonstrated at all stages of the disease so the absence of inclusion bodies does not necessarily mean that the animal did not have distemper.

Occasionally, distemper occurs at the same time as an outbreak of yellow fat disease, coccidiosis or food poisoning. If a diagnosis of these conditions is made without knowledge of the coexisting distemper virus, the losses will continue.

Serological. In this test, laboratory workers attempt to demonstrate antibodies in the blood of the mink. If they are detected, it is evidence that the mink has had or is experiencing an attack of distemper. This test presents many disadvantages for the diagnostician but is a powerful laboratory tool for the research man. Most of the work on the distemper virus using these tests has been done at Washington State University and the University of Wisconsin.

Animal Inoculation. If the interval of time following the death of the mink has not been too great or if the animal has been frozen solid, the inoculation of ferrets is probably the safest way of confirming a suspected clinical diagnosis. The method is relatively simple but it has the disadvantage of being time consuming. First, the spleen is removed from the suspected mink, ground up and made into a suspension and inoculated into a ferret or ferrets. If the spleen from the animal in question contains active virus, the ferrets will show signs of distemper in about 10-20 days. Ferrets are used because they are very susceptible to the distemper virus regardless of age. Many laboratories do not have ferrets available at all times of the year so this test is not usually employed.

Immunofluorescent tests: Most laboratories use this test to detect distemper virus in tissues microscopically. It will pick up the virus when regular tissue staining will miss distemper positive mink.

Summary. In most cases distemper can be diagnosed by observing affected living animals. If the veterinarian is not positive of his clinical interpretation, laboratory tests are available.

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CORTISOL, AS AN INDICATOR OF STRESS

In my experience, most mink producers are concerned about the welfare of their animals and this fits within the concerns expressed by the public about animal welfare, generally. Scientists have been trying to identify chemicals in the animals' bodies, the levels of which may indicate whether or not the animal is suffering stress. Danish investigators think they may have found such a compound in cortisol, and its derivatives. Cortisol is the main glucocorticoid hormone secreted by the adrenal cortex. The exact mechanisms by which corticosteroids work is incompletely understood, but they do seem to suppress acute and chronic inflammatory processes. The Danes believe increased levels of cortisol may indicate stress in animals and they measured levels of cortisol derivatives in the feces of mink. They injected mink with adrenon-cortico hormone (ACTH) at the start of the experiment to induce stress. The level of the cortisol metabolite, 11-17 dioxoandrostan was significantly higher in the feces of the ACTH-injected group than in non-injected mink and they suggested that levels of this compound may be a useful indicator of stress in mink (from: Malmkvist, J., R. Palme, S.W. Hansen and B.M. Damgaard. Cortisol og Corticoide Nedbrydningsproducter I Minkfaeces. 2003. Ann. Report Danish Fur Breeders' Research Center. pp. 7-15).

VITAMIN E FOR MINK

Vitamin E is recognized as one of the most effective natural antioxidants available and is widely used to protect animals from the damaging effects of oxidizing fats in their diets. There is still a lack of accurate information on the amounts of vitamin E needed and since the supplementary vitamin is expensive, this is a matter of some concern. The Mink Farmers' Research Foundation has funded research at Michigan State University to add information on this matter and the following report outlines their findings:

Estimated dietary vitamin E requirements for mink are in the range of 5-25 mg/kg diet but one report suggests that Scandinavian fur ranchers now use average concentrations of 60-80 mg vitamin E/kg feed in mink diets to ensure appropriate vitamin E supplementation. Several studies have demonstrated the beneficial effects of adding vitamin E above the recommended levels to swine diets as a means of enhancing the immune system of the pig. Heifer dairy calves had overall greater weight gains when fed typical calf diets supplemented with 125 or 250 mg vitamin E/calf day over a 24-week period than un-supplemented calves. Similar observations of improved weight gain and feed efficiency in stressed beef calves were noted following vitamin E supplementation. The present report summarizes the effects of feeding mink a diet supplemented with vitamin E above the recommended concentration.

Eight female, natural dark mink and their 47 kits were fed a basal ranch diet (containing 54 mg vitamin E/kg feed) that was fortified with an additional 1,080 mg vitamin E/kg feed from whelping to weaning of kits at 6 weeks of age. Twelve female, natural dark mink and their 66 kits were fed the basal diet containing 54 mg vitamin E/kg feed and served as the control group. At weaning, the kits were continued on their respective diets throughout the growth period until October 31, 2002. The estimated daily intake of vitamin E (assuming a daily feed intake of 150 g/animal) was 8 mg/animal in the control group and 170 mg/animal in the vitamin E-fortified group.

Survivability of the kits in the vitamin E-fortified group (66%, 64% and 51% at three weeks, six weeks, and six months of age, respectively) was enhanced compared to survivability of the control kits (41%, 41% and 38% at three weeks, six weeks and six months, respectively), but the differences were not statistically significant. Body weights of the adult females in the control and vitamin E-fortified groups declined by 17% and 16%, respectively, from whelping to weaning. Body weights of the control male kits were 9% and 6% greater at birth and at six weeks of age compared to body weights of vitamin E-fortified males, but by the end of the trial, when the kits were approximately six months old, the vitamin E-fortified males were 2% heavier than the control males. The same trend was apparent for the females in that the controls were 16% and 12% heavier at birth and at six weeks of age, respectively, than the vitamin E-fortified females, but by 12 weeks of age and continuing until the end of the trial, the vitamin E-fortified females were 10% heavier than their control counterparts. There was no difference between the two groups in the time required for blood samples to clot, indicating that the vitamin E supplementation was not excessive, which could result in disruption of vitamin K clotting activity.

In conclusion, supplementation of a basal ranch mink diet with 1080 mg vitamin E/kg feed caused a numerical, but not significant, enhancement of kit survivability from birth through six months of age. Additionally, both male and female kits fed the vitamin E-fortified diets were slightly heavier than their control counterparts by 12 weeks of age. A reproductive trial utilizing the vitamin E-supplemented diet is in progress.

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CAGING MINK FEMALES WITH A MALE KIT

There is a trend developing to keep the breeding female together with one male kit after weaning and until pelting time. It is not documented how widespread this routine is and until now it has not been examined how this routine affects the breeding female with regard to her welfare. A pilot study was set up with the aim to examine some few parameters directly or indirectly linked to welfare of the breeding female when kept with one or two male kits until pelting. In the end of June, 192 breeding females were distributed into 4 groups. The females had between 6-10 kits and had given birth within the same week in April/May. The litters were then between 7-8 weeks old at weaning time. Group 1 females were housed singly, group 2 litters were then between 7-8 weeks old at weaning time. Group 1 females were housed singly, group 2 females were housed with 2 male kits from another litter, but of the same age as her own kits, group 3 females were housed with one male kit from another litter and of the same age as her own kits, and group 4 females were housed with one of her own sons. All animals were moved from their home cage and re-housed in another shed in similar sized cages measuring 90x30x45 cm with one nest box. Scan sampling observations of behavior and position in the cage were performed 30 times, divided over 6 days (2 at start, 2 in the middle and 2 at the end of the study) with 5 samplings per day with 30 minute intervals. After being killed the mink was marked, and individually measured for skin size from the snout to tail root. Bites on the leather side were counted for different regions: belly, back of the neck, back, hips and tail. After being processed the skins were checked and registered for fur damages on the same regions.

The results showed that being together with a partner increased the amount of positive behaviors such as grooming and play, but it also increased the amount of fights and being pursued. The number of animals stereotyping were reduced in groups with one male kit, and highest in the group with singly housed females. In the group with 2 male kits, the amount of stereotypes was high as well as fights and being pursued (from Pedersen, V. Adfaerd og Productionsparametre som Indikation pa Velfaerd hos Aylstaever underForshellige Sociale Indhusningstyper i perioden fra Fravaenning til Pelsning. 2003. Annual Report, Danish Fur Breeders Research Center, pp. 17-26).

BOTULISM IN MINK

One of the "old" problems that still crops up occasionally is botulism, and since its effects may be catastrophic, it is useful to summarize what we know about the disease. The following has kindly been provided by Dr. Gorham, at Washington State University:

Botulism is a poisoning caused by a potent toxin produced by Clostridium botulinum bacteria found in soils and in the intestinal tracts of mammals and fish (U.S. FDA 2001). At the present time, there are eight known botulism toxins (types A, B, C1, C2, D, E, F and G) but by far type C1 causes the most severe problems in mink. Mink of all ages are susceptible to botulism type C (Loftsgard et al., 1970). Mink are also susceptible to types A and B but to a much lesser extent (Quortrup and Gorham, 1949). The spores of botulism type C are very heat resistant; in fact, they can be boiled for 60 minutes and still be viable. The spores themselves are harmless but under the right conditions, spores can hatch and produce deadly toxin.

Transmission

A small amount of toxin will kill thousands of mink. Ingestion of spoiled feed is the main way mink are infected with botulism. Raw feed at temperatures above 50 degrees F (15 degrees C) and anaerobic conditions (no oxygen) provide the perfect environment for the bacterial toxin to be produced (Joergensen 1985). In this way, cooling and washing slaughterhouse offal can reduce the risk of botulism by decreasing the likelihood the toxin will be formed (Joergensen 1985). Once the toxin is formed, however, freezing the meat will only preserve the toxin. Botulism toxin is

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BOTULISM IN MINK CONTINUED

susceptible to heat and will be destroyed by heating food products to 176 degrees F (80 degrees C) for 10 minutes (U.S. FDA 2001). Boiling raw food products also helps reduce the risk of botulism toxicity (Joergensen 1985).

Clinical Symptoms

The clinical signs of botulism usually appear within 18-36 hours of ingestion of spoiled feed although symptoms have been seen as early as 4 hours and as late as 8 days. In the early stages of disease, mink will exhibit incoordination, stiffness and muscle tremors. The breathing of affected mink will be labored due to paralysis of the muscles between the ribs. Mink may be seen heaving their flanks in an attempt to inflate their lungs. Affected animals will later become paralyzed with paralysis beginning in the hind legs and progressing forward to the front legs and neck (Figure 3). When held, mink will be flaccid, their heads lolling to one side (Figure 4). Mink may also exhibit excessive salivation, convulsions and even coma. Sick mink will be unable to right themselves if turned onto their backs. Eventually death will ensue as a result of respiratory paralysis. The time of death depends on the amount of toxin ingested. In general, many deaths will occur on the first day of the outbreak with fewer and fewer deaths occurring on subsequent days.

Figure 3: Botulism-affected mink are first depressed and show difficulty in running, or climbing the cage wire. Such signs are followed by distressed breathing, flaccid paralysis and death.

Figure 4: When a botulism-affected mink is picked up, the hind legs hang limp. The toxin paralyzes the muscles used in breathing.

Diagnosis

The diagnosis of botulism can be tricky. Pathologic changes in animals dying of botulism are not specific and this is not helpful in diagnosis. The only consistent finding is lung congestion. Therefore, both carcasses and feed samples are necessary to reliably diagnose botulism. Clostridium may be cultured from submitted samples but this takes 5-7 days. The fastest way to diagnose botulism is via the mouse neutralization test that detects toxin in the serum of sick mink or in food the mink ate (U.S. FDA 2001).

Control of Botulism

The key to dealing with botulism is prevention. No effective treatment for botulism exists (Aulerich and Bursian 1996). Vaccination is the best defense against infection. Botulism vaccines consist of a toxoid (a toxin that has been detoxified by formalin). The injected toxoid will elicit an immune response resulting in the formation of antibodies directed against botulism. Three weeks after vaccination the mink's immune system will be ready to ward off botulism toxicity. The duration of immunity varies but to be safe, all mink should be vaccinated each year. Vaccination should occur no later than June since the risk of botulism is highest during summer months (Joergensen 1985). Kits should be vaccinated at 6-7 weeks of age. Some maternal antibody protection is conferred prior to this age as was demonstrated by an outbreak that occurred in 1957. Interestingly enough, botulism vaccines can be administered at 6-7 weeks, whereas mink virus enteritis and distemper vaccines cannot be administered prior to 12 weeks to avoid interference by maternal antibody. The maternal antibody for botulism must decline more rapidly than

that for mink virus enteritis and distemper.

Botulism vaccines are highly efficacious. During an outbreak in 1957 the percentage of vaccinated adults dying of botulism was 0.3% while the unvaccinated herds suffered losses around 45% (Gorham 1957). Even unvaccinated kits from vaccinated females were protected (only 0.01% died versus 46.75% of kits from unvaccinated females).

Botulism Outbreaks

On the 25th of December 1988, 150 tons of feed were distributed in southwestern Norway from a large feed kitchen. The first mink deaths occurred on the 26th and continued on the 27th and 28th. It turned out the meat products in the feed were not refrigerated properly prior to feeding. In the end, nearly 200,000 unvaccinated mink died of botulism. This loss represented 60% of Norway's mink. One farmer alone lost 12,000 mink (his entire herd) in 24 hours.

Botulism is a devastating disease with mortality rates of 90% or higher (Figure 5) that can be prevented with vaccinations and proper food preparation. In Denmark where there are strict regulations regarding food preparation, botulism is rarely seen in mink.

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Figure 5: Losses can be very heavy if botulism vaccine is not used to protect the mink.

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RAPID-FIRE TESTS DETECT BOTULISM TOXIN

Scientists in the UW-Madison Medical School and the College's Food Research Institute have developed a pair of rapid-fire tests for botulinum toxin, a feat that could underpin new technologies to thwart bioterrorism and spur the development of agents to blunt the toxic action of the world's most poisonous substance.

The two assays vastly improve on current technologies to detect the deadly poison. One is a real-time assay, which could be deployed in a kit and used in the field. The other is a cell-based assay that helps provide a glimpse of the toxin doing its dirty work in living cells. This technology promises a rapid screen for millions of chemicals to see which might inhibit the paralyzing effects of the toxin. The potential upshot of such a screening technology could be the development of drugs that act like a prophylactic to confer protection from botulinum poisoning.

The new tests can be conducted with ordinary lab equipment. They work by introducing into cells bioluminescent proteins whose glow extinguished in the presence of the toxin. The tests are capable of detecting all seven variants of the poison.

Currently, the most sensitive and common test for toxin activity is exposing mice to an a gent. The process takes time, and many animals are used and die in the process. (from: Devitt, Terry. University of Wisconsin College of Agricultural and Life Sciences Quarterly. Winter 2005).

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