

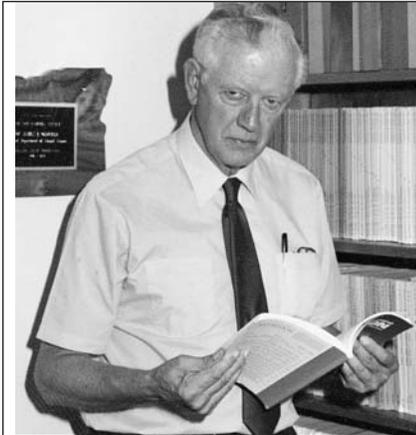
Fur Animal Research

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Well, after a long and unusually wet spring (even for Oregon) we are now enjoying bright, sunny and unseasonably warm days. This reminds me, once again, of the diverse weather conditions that mink are raised in, in various parts of our country. Last evening (May 17) as I watched the weather reports on TV, I saw that the New England states were being flooded by exceptionally heavy rains.

As I report to you on research done to help the mink industry, I am reminded that mink research may also help other animal species, including humans. A case in point is research with melatonin, which many of you use to speed up the fur-ring process in the fall. There are now reports on TV and in other news media, that melatonin may be

useful to humans to help them overcome "jet lag" experienced after long trips by air through several time zones. And, carrying this a step further, some suggest that it may be helpful as a sort of "sleeping pill," to enable people to get a good night's sleep. So, to those who question the value of the fur industry to society, we can point out that mink, over and above the beautiful pelts they produce, may be useful laboratory animals for testing products, like melatonin, that may benefit the public at large.

Sometimes research brings unexpected results. A survey was conducted two years ago in Denmark to assess the losses of kits in the first few days after whelping. When the figures were assembled, it was noticed that a disproportionate number of the kit losses occurred on ranches served by a single feed manufacturing plant, so this research effort, designed merely to identify and quantitate the losses, ended up giving a possible cause of the kit deaths.

Dr. John Gorham, at Washington State University, who we all recognize as a world leader in mink disease research, tells me that he has reached an age where he feels he should sum-

marize his tremendous output of disease research and possibly publish it in book form. As he does this, he has kindly offered to send me some of the articles he will include and I hope to include these in future issues of this newsletter. I first met John in the late 1940's, when I was doing mink nutrition studies at the University of British Columbia, and John came up from Washington to see what was going on. I have since admired his work and we have been good friends for many years.

Bob Zimbal, who chairs our Mink Farmers' Research Foundation Board, tells me that he has invited two Wisconsin veterinarians to join the group. They are Dr. John Easley, of Glenbuelah and Dr. Hugh Hildebrandt of Medford, and we welcome them to this group. If you know these men, please congratulate and thank them for undertaking this important work for the mink industry.

I wish you all a successful mink growing season.

A handwritten signature in dark ink, appearing to read "J. E. Oldfield". The signature is fluid and cursive.

J. E. Oldfield

THE GOOD LIFE - MINK RESEARCH

I have had a great time and an interesting research career investigating mink diseases. Since I am 83 years old, I thought it would be fun to write about some of the experiences and observations that I had on mink farms and in our laboratory. I will not hesitate to point out some mistakes I have made. I am not on an ego trip but if mink farmers find my rambling enjoyable, I will be pleased.

I never saw a mink until I got a part-time job with the Fish and Wildlife Service at Washington State University in the fall of 1942. Dead mink were sent to the laboratory from all over the Northwest for diagnosis. Abscesses, yellow fat disease, non-specific enteritis, calculi and problems associated with the diet were frequently observed but of the most concern was a diagnosis of distemper because there were no commercial distemper vaccines. I should mention that in the 1940s there were no vaccines for any mink disease.

Distemper Vaccine Catastrophe

To control distemper on the farms, the head of our laboratory made what was known as a killed virus tissue vaccine. This type of vaccine was not difficult to make. After distemper was diagnosed, we would rush off to the farm having the outbreak and collect the spleens of mink that were dead or dying of

distemper. Back at the laboratory, we ground up the spleens in a kitchen blender and added saline that contained a low level of formalin. After the mixture was incubated for a few days at room temperature, this vaccine was sent to the farmer to be injected as soon as possible into all the mink that were not showing distemper signs.

Aleutian disease (AD) losses occurred within a few months on several farms where the vaccine was used. In 1949, one rancher lost 500 mink after vaccination. Floyd Marsh, a truly colorful Oregon mink rancher told me "they didn't make baskets big enough to pack out my dead Aleutians and Sapphires." I might add that besides being contaminated with AD, the vaccine failed to stop the spread of distemper on the farms.

The Source of Aleutian Disease for Ranch Raised Mink

There is no doubt that AD had been present in some wild mink populations for many years. Low virulent strains were occasionally transmitted mink to mink but transmission from an AD-infected female to her kits would seem to be the most effective way of maintaining the virus in nature. I would like to speculate that live-trapped wild mink in the Pacific Northwest carried low virulent AD to the mink farms. If the farmer was raising black mink, the

AD virus might have lowered the kit average a bit but this minor loss was overlooked by the farmer.

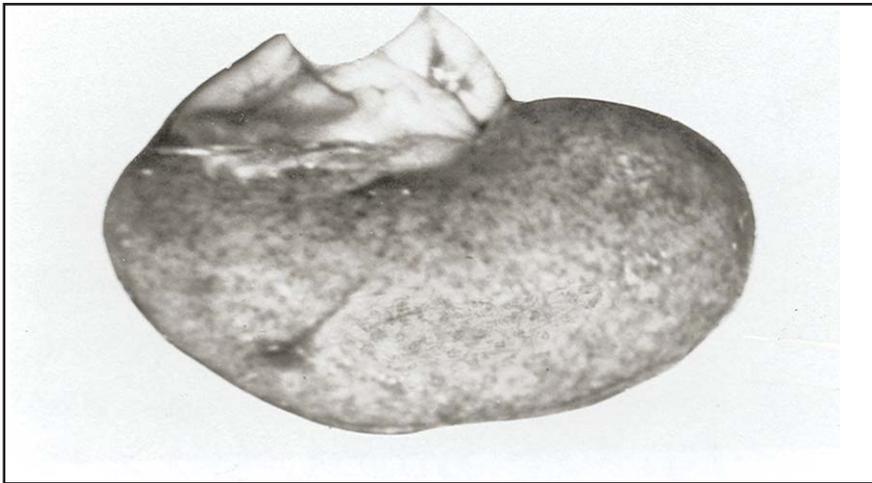
In 1941, Andy Waris, who had a mink farm near Astoria, Oregon, saved for breeding a gunmetal colored mink that was noticed in a litter of standard dark mink. Andy called this new mink Aleutian after the Aleutian fox that had a similar coat color. The Aleutian mink gene was inherited as a recessive trait designated by the symbol (aa). These mink carried the gene for the Chediak Higashi syndrome that will be discussed later. In a few years there was a brisk sale of breeding stock because the mink had beautiful coat color. Aleutian mink were and still are the sentinel for AD on the farm.

The First Appearance of Aleutian Disease

Ranchers first noticed AD when their Aleutian mink lost weight. Dark tarry feces were often observed and affected mink were very thirsty. Because of mouth ulcers, about 10-20% bled at the mouth. If the blood vessels of the brain were involved, the mink had nervous signs. The mink became anemic with pale gums and foot pads and they became progressively thinner. They died within weeks or months after the onset of symptoms.

Lesions of Aleutian Disease

If a mink is handled, ulcers in the mouth tend to bleed. When the abdominal cavity is opened, depending on how far the disease has progressed, the kidneys are at first enlarged and reddened. Later in the course of the disease, they are swollen and pale with many small red foci. The spleen and lymph nodes are enlarged. In the final stages before death, the pale kidneys are shrunken and pitted. Mink farmers not only called the condition Aleutian disease but also mouth bleeders, grave diggers and "one year mink."



Kidney from a mink in advanced stages of Aleutian Disease. Kidney failure leads to uremia and death,

Aleutian Disease is Contagious

By 1950 it was apparent to many mink farmers that AD was contagious. When mink were moved from a farm that had AD cases to a "clean farm," AD appeared in the mink on the clean farm within a few months. Infected Aleutian mink were sold to

Wisconsin farmers and AD losses appeared on these farms. Overseas shipments to Scandinavia by U.S. farmers may have spread the disease. But AD may have been present in Denmark, Sweden, Finland and Holland prior to the U.S. exports and just was not recognized.

In 1956, G. Hartsough and I wrote a short article about Aleutian disease that was published in the National Fur News. There were a lot of questions we couldn't answer. All attempts to find the cause failed. Tissues from AD mink did not produce the disease in black mink. No

doubt we were dealing with such a low virulent strain that the injected black mink showed no signs of the disease. This was one of the many blind alleys in research on this disease. Now we know that if we had injected Aleutian mink with the low virulent strain, we would have

showed transmission of AD. At the same time, no significant bacteria were isolated which meant that broad spectrum antibiotics that were injected or put in the diet were of no help. All mink farmers realized that the industry was dealing with a very serious disease and we did not know the cause, prevention or treatment.

Discovery of the Iodine

Agglutination Test (IAT)

In the spring of 1960, we knew that AD-infected mink had a tremendous level of gamma globulin in their blood. In some instances this serum protein was four times the normal value. Running the test in the laboratory to determine the level of gamma globulin was not difficult, but it required expensive equipment and was time consuming. Something more practical had to be devised for use by the mink farmers.

Early in October in 1960 my co-worker, Dr. Jim Henson, walked into the lab and said, "I can diagnose Aleutian disease in a live, healthy-appearing mink by testing for this increased amount of gamma globulin. Furthermore," he said, "it doesn't take six years of college to run the test." You can imagine my answer when I found out he mixed together a drop of an iodine solution and a drop of mink serum with a toothpick! He had modified Mallen's test which was employed for human disease. We quickly started

THE GOOD LIFE - CONTINUED

work on the iodine agglutination (IAT) test which, by the way, was supported by funds supplied by the Mink Farmers' Research Foundation. In November of that year we were ready for field testing and called on Wallace and Gladys Leonard. Like virtually all Aleutian mink raisers, their farm was loaded with the disease. There was nothing that they or anyone could do except

pelt the obviously sick mink.

We asked a couple of mink farmers to go into the Leonards' sheds and select some mink for testing. They came back with five big, beautiful triple pearl males. We tested them and found three IAT-positive. We autopsied all five. The three that had tested positive had AD and the negatives showed no gross changes of AD in their organs.

Everything was not "beer and skittles" with the IAT. Sensitivity problems showed up that I will point out in a forthcoming article.

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DELAYED IMPLANTATION, OR DIAPAUSE IN MINK REPRODUCTION

Reproduction in female mink is characterized by an arresting of development of the fetus at the blastocyst stage. Fertilization occurs in the oviduct from 53-70 hours after mating and the embryo develops into a blastocyst which migrates to the uterus 6 days after mating. Then, delayed implantation or diapause occurs and the further metabolism of the blastocyst is restricted. Canadian researchers investigated the possibility of avoiding this dia-

pause by treating the females with prolactin. Within 72 hours of continuation of development both protein and DNA synthesis markedly increased and embryo diameter increased. They concluded that during the reinitiation of embryo growth following diapause embryo growth is characterized by only gradual increases in protein synthesis, accompanied by mitosis of the trophoblast and inner cell mass. There appeared to be a pattern of

differential proliferation with the trophoblast phase occurring mainly during the early reactivation stage, while inner cell mass proliferates more rapidly nearer time of implantation. (from: Desmarais, J.A., V. Bordignon, F.L. Lopes, L.C. Smith and B.D. Murphy. 2004. The escape of the mink embryo from obligate diapause. *Biology of Reproduction* 70:662-670.)

MILK AND CHEESE BY-PRODUCTS AS MINK FEEDS

I know that many mink ranchers include cheese in their feed mixes - it is a very good feed. There is some evidence, from work with pigs, that other milk/cheese by-products may be useful, too. Whey has been shown to be a good feed for baby pigs but the researchers cautioned that there are several different types of whey which may have different nutritional values. Whey that has been overheated in preparation (brown whey) is not considered a good feed. Sweet whey is a better product than acid whey, probably partly due to its higher palatability. Some nutrient values of sweet whey are given in the accompanying table. High quality whey should have a creamy, off-white color. Some batches of whey are a yellowish color due to higher levels of carotene in the milk. This is okay, but a brown color indicates overheating which is not good. Spray-dried whey is usually a good product because the heating is less than in other forms of dried whey. (from: Mavromichalis, I. 2006. Which whey for piglets? *Pig International* 36(3):6-9.)

Typical chemical specifications for sweet whey

	%	SD
Drymatter	96.4	2.1
Crude protein	12.6	2.3
Lipids	2.1	0.9
Ash	8.7	2.1
Lactose	72.9	
Calcium	0.82	0.15
Phosphorus	0.69	0.07
Sodium	0.64	
Chlorine	1.76	
Lysine	0.94	
Methionine	0.20	

(INRA [2002])

NURSING SICKNESS IN MINK

Nursing sickness is the greatest cause of deaths in adult female mink. It is a metabolic disorder that develops when the high demands of lactation cause extensive mobilization of the female's body energy reserves. It is characterized by a progressive weight loss, emaciation and dehydration, and is accompanied by high levels of glucose and insulin in the blood. The condition varies in incidence from year to year, for a number of reasons and old females and mothers with large litters are most often affected. Some form of stress may trigger the onset of the

disease. Dr. Kirsti Rouvinen-Watt of Nova Scotia suggests that the underlying cause of nursing sickness is an acquired insulin-resistance in the mink, coupled with over-fatness and a high rate of oxidative protein breakdown. She recommends the practice of keeping breeder females in a moderate flesh condition during the fall and winter months and ensuring that the females have ready access to drinking water. (from: Rouvinen-Watt, K. 2002. New hypothesis for pathogenesis of nursing sickness in mink. Proc. NJF Seminar no. 347. 11 pp.)

ASSESSING THE TEMPERAMENT OF MINK

Some mink producers in the Scandinavian countries are including assessment of temperament among the criteria that they use in selecting their breeding stock. Mink are described as fearful, exploratory, or aggressive and then breeders can be selected according to the producer's wishes. They use what they call the "stick test" to assess the animal's temperament. This simply involves pushing a stick into the cages and observing the mink's behavior towards it. They report that, after a 30-minute training session, mink farmers can perform the test with 70-100% agreement in results with those of an experienced tester. On average, they found that 60% of the adult female mink were classified as "exploratory"; however, this percentage varied among farms. They feel that the welfare of mink can be improved by adapting the production system to the needs of the animals, and/or selecting animals that are best adapted to their production system. (from: Hansen, S.W. and S.H. Moller. 2001. The application of a temperament test to on-farm selection of mink. *Acta Agr. Scand., Section A. Animal Science Supplement* 30, pp. 93-98).

WATER SUPPLY FOR LACTATING MINK

The lactation period is the most critical time in the life of female mink and we recognize this by special attention to her nutritional needs. It is just as important to provide carefully for water requirements and a recent study of types of watering devices carried out at the University of Copenhagen has produced some interesting and useful results. One of the aims of the work was to compare effectiveness of open water cups with drinking valves (nipples).

Three experimental groups of mink were involved. Group 1 had water supplied in cups placed about 4 cm above the cage floor. Group 2 were watered by valves placed 12 cm above the bottom of the cage and group 3 had valves set 4 cm above the

floor. The results showed that kits having open water cups (group 1) started drinking water 1-3 days sooner than those supplied by valves, and they licked saliva for a shorter period, which indicates that saliva licking is caused by thirst. Water intake from the cups was greater than that from the valve-supplied groups. Group 2 mink, which had the water supplied by valves 12 cm above the floor had the lowest water consumption of all groups. Group 3 (valves 4 cm above the floor) showed the largest kit weight gain and the lowest weight loss by the dams at weaning, which suggested that they experienced less stress than the other groups. Group 2 dams performed more stereotypies (aimless running

back and forth) just before their kits started drinking, indicating difficulty in coping with the higher valve placement. The kits spent most of their time in the nestbox at first, as might be expected, but towards weaning more time was spent out in the cages. Group 1 kits (open water) tended to be most active towards weaning. Putting it all together, open water cups appeared preferable to valves; however, this may be partly offset by potential danger of contamination of open water sources. (from: Steffinsen, L.K. 2003. Supply of drinking water for ranch mink in the lactation period. M.Sc. thesis. Dept. of Animal Behavior, Zoological Institute, University of Copenhagen. Abstracted in *Scientifur* 25(1):19, 20.)

FIBER IN THE MINK DIET AND STEREOTYPIES

There has been some indication that feeding mink a high fiber diet will reduce running about the cage, which is thought to be an indication of stress among the animals. In a Danish trial, 3 groups of female mink were fed a set diet with 3 levels of fiber - low, medium and high. The energy content of the diets was 199, 178 and 163 kcal/100 g for the low, medium and high fiber diets, respectively. Since the mink were fed at the same energy level, the high fiber group had to consume the most diet each day.

The experimental feeding period was from January 1 - February 24th. On January 13, 80% of the mink in the low-fiber group had no food left after 6 hours, while mink in the medium and high fiber groups took 10 and 12 hours, respectively, to eat their food. After 2 weeks on the experimental diets, the percentage of females performing stereotypies increased on the low fiber diet, while in the other two groups, it remained constant. There was no difference among groups in the number of live kits per female, or

in the number of kits at 42 days of age. It was concluded that high fiber feed reduced the time spent without eating and the occurrence of stereotypies. Feeding a high fiber diet during the winter had no effect on the number of kits per female. (from Hansen, S.W., T.N. Clausen, J. Malmquist, B. Damgaard and C. Hejlesen. 2003. Fiber in the Winter Feeding Period. Annual Report, Danish Fur Breeders' Research Center. Holstebro. p. 27).

FATS/OILS IN THE MINK DIET

The easiest way to add to the energy content of mink diets, when necessary, is to add fats or oils. Many of these are available commercially, but they vary a great deal in quality and usefulness. Danish studies have compared a number of these, including commercially-produced soybean oil, sunflower seed oil, rapeseed (Canola) oil, cold-pressed rapeseed oil and two different fish oils. These were added to mink diets fed through the mink gestation and lactation periods, with the oils being the only variable in the diets.

Analyses of the oils showed considerable difference in their content of essential fatty acids which varied from 0.1 to 24.7% of the total fatty acid content. These differences were reflected in the essential fatty acids in the fat of the dams' milk and the body fat of the kits. Results of feeding these different oils showed clear differences in the growth and development of the young mink. The best supplementary fat, overall, was the cold-pressed rapeseed (Canola) oil, which contained higher levels of carotenoids and other antioxidant materials than the other oils. The

two types of fish oil used showed some degradation, since fish oils are quite unstable to oxidative damage, unless protected by vitamin E, ethoxyquin, or other antioxidants. In this trial, the mink fed diets containing these two fish oils grew less rapidly than those on the other diets. (from: Bjerregaard, C., T.N. Clausen, T.M. Lassen, K. Mortinsen, H. Sorensen and J.C. Sorensen. 2003. Feeding Plant or Fish Oils to Gestating and Lactating Mink. Annual Report of the Danish Fur Breeders' Research Center, Holstebro, Denmark, pp. 53-66.)

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