

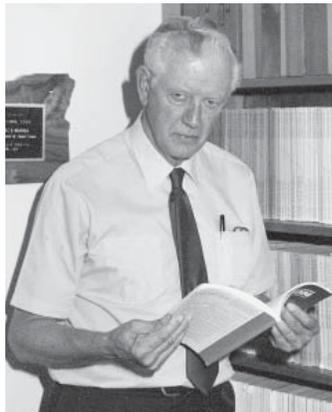
Fur Animal Research

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As a British poet once wrote, "Now that winter's o'er, can spring be far behind?" I would have responded this year, "I certainly hope not." I've had quite enough winter for one year, thank you. Not that our Oregon winters are that bad – rainy, but generally fairly mild – much easier than what many of you have been experiencing in the Midwest and New England states. But rain does get a bit monotonous. As I look out of my office window I see the coast range mountains, with their highest point, Mary's Peak, about 25 miles west. We have a saying here, "If you can see Mary's Peak, it's going to rain. If you can't see it, it's raining." Regardless of where we're situated, however, I think we all look forward to some nice sunny, spring days.

At its winter meeting, the Board of Directors of the Mink Farmers' Research Foundation (MFRF) approved a \$3,000 grant to Scintilla Development Company of Bath, PA to help their development of a new test for Aleutian Disease (AD). One of the Board members, Dr. Hugh Hildebrandt, has been running these tests and has done over 10,000 with apparently successful and repeatable results. The Board thanks the ranchers who made their animals available for these tests. Scintilla is applying to the U.S. Department of Agriculture for licensing of the

test. This is a continuation of the long program of research on AD that the MFRF has supported and it is good to see this support extended to a commercial laboratory.

Sometimes tests done with other species have important implications for mink. One such example is the test for BSE (bovine spongiform encephalopathy) which is run on cattle and is important because there is a similar disease that affects mink. The Neogen Corporation has developed a quick and effective test to determine presence of ruminant by-products in feed. It is thought that it is through feeding of these by-products that BSE is transmitted. In the United Kingdom, where most of the cases of BSE have been found, the government has banned the feeding of these products to ruminants, and since 1988 this has caused a dramatic reduction in the number of cases of BSE. (from RENDER, February 2003, pp. 10-12)

And just a brief personal note. The Department of Animal Sciences at Oregon State University, in which I have spent most of my career, is celebrating its 100th anniversary this year (1907-2007). Along with the Department of Crop Science (originally "Farm Crops") it was the first two departments formed in our College of Agriculture. I haven't been present for the whole hundred years, but can claim over half! Have a great spring and I wish you a great kit crop.

A handwritten signature in dark ink, appearing to read "J. E. Oldfield".

J. E. Oldfield

METHIONINE SUBSTITUTES IN MINK DIETS

As we know very well, it is important to feed high quality protein to mink, to achieve good growth of the animals and high quality pelts. Protein quality depends on the amino acids that the protein contains and this has raised the question whether supplementation of diets with some of the essential amino acids might allow use of lower quality, and cheaper sources of protein. Only a few of the amino acids are available commercially – one of which is methionine. There are also compounds that will take the place of methionine at a lower cost than methionine itself. Two of these are methionine hydroxy analog (MHA) and betaine. Danish investigators at their research center at Holstebro have looked at the use of these compounds with experimental groups of 120 each, male and female scanbrown mink. The basal

diet was a standard feed kitchen diet with 20% less methionine than normal. To this was added: group 1 – basal and methionine to make up the methionine deficiency; group 2 and group 3 – similar amounts of MHA and of betaine. They found that use of MHA or betaine had no negative effects on pelt length. Use of betaine produced similar pelt length and better fur quality than the methionine-fed group. They felt that MHA or betaine could be fed as replacements for dietary methionine if the level of methionine in the diet was 0.13 g methionine/100 K calories in the diet. (from Clausen, T., P. Sandbol and C. Hejlesen. 2003. Methionine or methyl donors for mink during the furring period. Annual Report, Danish Fur Breeders' Research Center, pp. 97-100)

MORE ABOUT DISTILLERS' DRIED GRAINS/SOLUBLES

We've written before about future availability of distillers' dried grains and solubles for use in mink cereal formulas, and here is more. The forecast for corn production for ethanol distillation is quite amazing. Corn production has doubled in the U.S. since 1963 and the prediction is that it may double again in the next five years to keep up with demand. This will, of course, bring significant increases in the amounts of distillers dried grains/solubles (DDGS) that will be available for livestock feeding. The biggest users of DDGS will probably be the swine industry, and we may learn something from them on how to best use this product. The usual advice to U.S. pork producers is to look at DDGS as a replacement for whole grain corn in their animal diets. We might look at DDGS as a replacement for other grains (we don't feed much corn) in mink diets. The quality

of DDGS tends to vary with production methods and we are advised that a dark-colored product is inferior and will reduce its feed value. A light-colored product is superior and has a higher content of the essential amino acid, lysine. We should note that the needs of pigs and mink are different: pigs are fed a largely plant-product diet which often benefits from supplementary lysine. Mink, on the other hand, get a diet with animal and fish products in it and usually don't need additional lysine. Nevertheless, a light-colored product is generally superior and should be used if possible. Any decision on use of DDGS, of course, should be based on their price in comparison with other cereal ingredients. (from Thornton, K., 2007. How DDGS looks from an American perspective. Pig International, March, 2007, pp. 11-15)

GIANT KIDNEY WORMS IN MINK

The giant kidney worm lives in the urinary system of the dog and especially mink. Both wild and ranch-raised mink have been found to be infected.

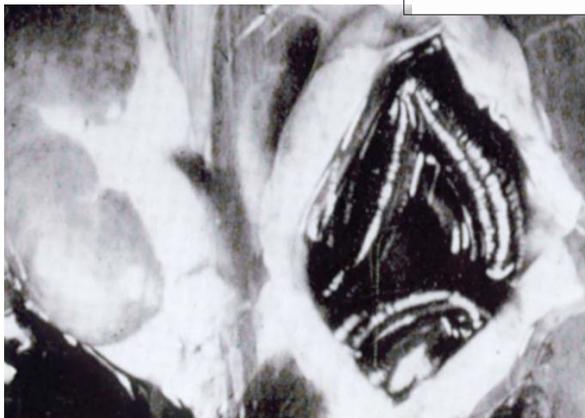
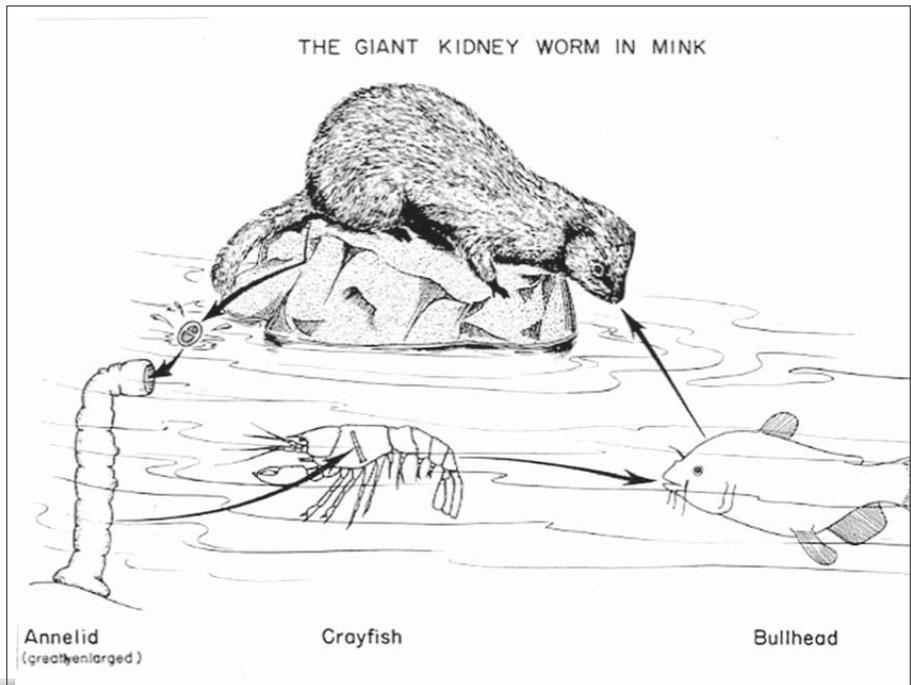
The life cycle of this worm is complicated. Eggs are laid in the mink kidneys and are expelled in the urine. Embryonation of the eggs normally occurs in water and is very slow. Small, leech-like worms (annelids) feeding on the bottom of ponds frequented by infected mink eat these embryonated eggs. The eggs hatch when swallowed by the annelid worms.

After a period of free living in water, these annelids commonly attack the gills of crayfish. When crayfish bearing annelids on their gills are eaten by such fish as bullheads, mink become infected if they eat the bullheads. The whole life cycle may take as long as two years.

When mink eat the infected fish, the freed larvae penetrate the intestinal wall of the mink and

then penetrate the kidney. The presence of the worm in the kidney results in the destruction of the tissues. The presence of the worm in one kidney does not usually produce any signs of illness. If both kidneys become infected, the mink dies.

Because there is no known treatment, control is based on prevention of the infection. Mink ranchers



Mink kidney cut open to show the giant kidney worm.

who make use of local fish as part of the food for their animals should be aware of this life cycle. Such fish as bullheads or catfish suspected of being infected should be thoroughly cooked or frozen before being used as mink feed.

*John R. Gorham, DVM
College of Veterinary Medicine
Washington State University*

MINK WELFARE IN PRACTICE

Mink producers have always been concerned for the welfare of their animals, but public concern has increased in the last few years, justifying some research on the subject. In 1997 the Danish Fur Breeders' Association initiated discussions with the Society for the Protection of Animals in Denmark (similar to our SPCA) as a basis for such research. Items studied were: (1) Selection of mink for confidence, (2) 10% weight loss in females from December until the beginning of flushing, (3) placing mated dams in every second cage before birth of kits, (4) removal of dams from kits at 8 weeks of age and separation of litters into male/female pairs 2 weeks later, and (5) placing shelves and tunnels in the cages just below the cage-top.

Female kits preferred the shelf as a resting place over the tunnel. Male kits used the shelf and tunnel equally, but the fur on their bellies was worn when they used the tunnels. In the confidence-selection study the selected females were calmer during lactation than the controls. It appeared that females could safely be separated from their kits when they were 8 weeks old, and some females could be separated even earlier.

These studies demonstrated that research on animal welfare is a viable topic and can lead to improved handling procedures. (from: Jeppeson, L., T. Simonsen, and V. Pederson. 2003. *Project Welfare in Practice*. Annual Report, Danish Fur Breeders' Research Center, pp. 35-44)

FUR CHEWING

Fur chewing is a condition where a mink decides to chew all the guard hairs it can reach on itself (see accompanying figure). Only the underfur remains in the chewed area. It often starts with the mink licking its tail and then continues when the mink chews the hair off the tail. Occasionally the mink does not stop with just chewing hair. It will chew the flesh and bones of its tail until finally reaching its anus. Then the mink will die of infection.

Since fur chewing occurs in individual mink and it is not farm-wide, the condition does not cause a great deal of concern. Every big farm will have a few fur chewers. There are no answers to the question. What is the cause of this abnormal behavior? It will probably take a mink psychologist to figure this one out. Small pens, genetic factors or nutritional

deficiency have been suggested as the cause. The only advice that I could give would be to pelt the dam and sire.

*John R. Gorham, DVM
Washington State University*



In fur chewing, the mink pulls out all the fur it can reach which leaves it with a lion-like look.

EFFECTS OF PESTICIDES ON MINK REPRODUCTION

Sometimes ingredients in the mink diet are contaminated with pesticide residues and we need to know what effects these may have on mink production and reproduction. Canadian workers have examined this situation by treating mink feed with three pesticides: Lindane @ 1 mg/kg/day, Carbofuran @ 0.05 mg/kg/day or Pentachlorophenol 1 mg/kg/day over their entire life spans after they were weaned. This was done over 3 generations of animals. The second generation mink had actually been exposed to the pesticides in utero and in their mothers' milk. The third generation were offspring of mink that had actually been exposed from conception onwards. No signs of toxicity were observed, nor did the pesticide

contamination affect the percentages of mink that were mated. The Lindane treatment, however, reduced the numbers of mated mink that subsequently whelped and the litter sizes of the mink that did whelp. Testis size was decreased in the 3rd generation of Lindane-treated mink. Serum concentrations of cortisol, testosterone and estradiol were not changed by any of the pesticide treatments. The effect of the Lindane treatment on reproductive efficiency was calculated to reduce the number of kits born by 60%. (from Beard, A.P. and N.C. Rawlings. 1998. Reproductive effects in mink exposed to the pesticides Lindane, Carbofuran and Pentachlorophenol in a multigeneration study. *J. Reproduction and Fertility* 113:95-104).

YELLOW FAT DISEASE

Dr. Gorham reminisces about some of his early research with yellow fat disease in mink:

When I first started to work in 1946 at the College of Veterinary Medicine, Washington State University, bags of dead mink kits were sent to us that had fat the color of canary yellow typing paper. The mink farmers called the condition "yellow fat disease" which was a good description. About the same time I went to Ketchikan and Petersburg, Alaska to investigate a condition they called "watery hide disease." The Alaskan mink farmers said that there was a lot of fluid under the skins of mink kits when they were autopsied.

It is important to point out that the farmers in the Pacific Northwest and the Alaskan farmers were feeding high levels of salmon scrap because it was a cheap food.

Shortly after my Alaska trip I visited a pelting/

fleshing service in Puyallup, Washington and found that the fat adhering to the pelts from some farms was discolored yellow while the fat on pelts from other farms was a normal white in color. When I checked the mink from farms that were fed high levels of salmon scrap that had been in storage, they had yellow fat. It was a question that needed an answer. Mike Dederer, President of the Seattle Fur Exchange, said that yellow fat and watery hide disease cost the mink farmers in the 1940s millions of dollars in dead mink and damaged pelts.

Dr. G. Hartsough and I were able to control outbreaks on farms by changing the ration so that it contained a large amount of fresh, unfrozen horse meat and additional fresh liver. This diet prevented yellow fat disease but was too costly to feed.

About this time, a farmer in Northeastern Oregon who was a real character told me that he

could prevent yellow fat disease by feeding all the wheat germ meal a mink would eat. Unfortunately, I did not follow his suggestion or we would have been able to control the disease a lot sooner.

I got my first lead from the Danish workers who reported that rats raised on vitamin E deficient diets containing highly unsaturated fatty acids developed a characteristic pigment in their fat tissue. Voila! We found the same pigment in mink that were fed salmon scrap that contained high levels of unsaturated fatty acids. At that time, mink farmers were not feeding additional Vitamin E in their rations. This was one heck of a finding. Any research worker worth his salt would not trade it for a lot of money.

In the Northwest, the disease appeared where the farmers fed high percentages of salmon scrap in the ration. Such a diet provided a rich source of highly unsaturated fatty acids and was deficient in Vitamin E. Consequently, we conducted an experiment to determine if Vitamin E could prevent yellow fat disease.

The Experiments

All the mink were fed a basal ration that served as the control in the experiment. It consisted of 85% ocean fish scrap, 13% cereal and 2% brewer's yeast. We added increasing amounts of Vitamin E to groups of mink diets. The control group was fed the basal ration and received no Vitamin E.

The experiments using kits were started on July 6 and concluded on September 22, 1950 when the level of pigment in the fat in all kits was recorded.

- Basal (control) group: 22 of 22 had pigment in fat tissue.
- Basal plus 3 mg of Vitamin E: 22 of 22 had pigment in fat tissue.
- Basal plus 10 mg of Vitamin E: 1 of 22 had pigment in fat tissue.
- Basal plus 20 mg of Vitamin E: no mink had pigment in fat tissue.
- Basal plus 5% wheat germ: 10 of 10 had pigment in fat tissue.

- Basal plus 10% wheat germ: No mink had pigment in fat tissue.

The pigment in the fat was the criterion of yellow fat disease in this trial. As the amount of vitamin E was increased, there was a corresponding decrease in pigment formation. There was a total absence of pigment when the mink were supplemented with adequate amounts of Vitamin E.

To sum it up, the diet that contained high levels of unsaturated fatty acids and a deficiency of Vitamin E (which acts as an anti-oxidant) led to production of a pigment that turns the fat to yellow and grayish color. Dr. William Leoshke reported that horse meat that was stored for a long period could cause yellow fat in mink. The pigment played a role in the inflammation of the fat so I named the disease "steatitis". The disease is no longer a mystery. It can be prevented by feeding a nutritionally sound diet with added Vitamin E.

Yellow Fat Disease in Pigs

I visited a mink farmer near Seattle who owned a few pigs. He told me that they were condemned at slaughter because of the peculiar yellow color of the fat. I asked him what he was feeding and he answered that he fed uneaten mink food that he removed from the mink pens. Incidentally, he had an outbreak of yellow fat disease in his mink that same summer. That was probably the easiest diagnosis I ever made.

Yellow Fat Disease in Cats

My co-worker, Dr. Don Cordy, autopsied a cat at Washington State's College of Veterinary Medicine and found the same lesions that mink showed. Canned cat food containing high levels of fish, particularly tuna fish, and a deficiency of Vitamin E led to the disease in cats. Now almost all commercial cat food contains added Vitamin E.

*John R. Gorham, DVM
Washington State University*

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Mink Farmers' Research Foundation Board

Members of your Research Foundation Board of Directors invite your input into the ongoing program of research. Please contact any of the Board with suggestions or comments. You may reach them at:

OFFICERS:

Chairman: Robert Zimbal, Sr.
2111 Washington Ave.
Sheboygan, WI 53081
PH.: (920) 452-7380
FAX: (920) 803-0662
rjz@execpc.com

DIRECTORS:

Dr. J. E. Oldfield
Dept. of Animal Sciences
Oregon State University
Corvallis, OR 97331-6702
PH.: (541) 737-1894
FAX: (541) 737-4174
james.e.oldfield@oregonstate.edu

Paul Westwood
8137 South, 1800 West
Spanis Fork, UT 84660
PH.: (801) 798-1786
FAX: (801) 298-1482
pwstwd@msn.com

SECRETARY:

Dr. John S. Easley
W 8866 Spring Valley Drive
Glenbeulah, WI 53023

Dr. Gary Durrant
Utah Fur Breeders Co-Op
8700 South 700 West
PH.: (801) 255-4228
FAX: (801) 255-4678
garydurrant@comcast.net

Jim Wachter
N 5350 Country Aire Road
Plymouth, WI 53073
PH.: (920) 892-4287
FAX: (920) 892-4287
jwachter@wi.rr.com

Dr. Hugh Hildebrandt
Medford Veterinary Clinic
898 S. Gibson Street
Medford, WI 54451

Ryan Holt
9762 S. Tayside Drive
South Jordan, UT 84095
PH.: (801) 280-1428
FAX: (801) 255-4678
rhino@networld.com