Fur Animal Research

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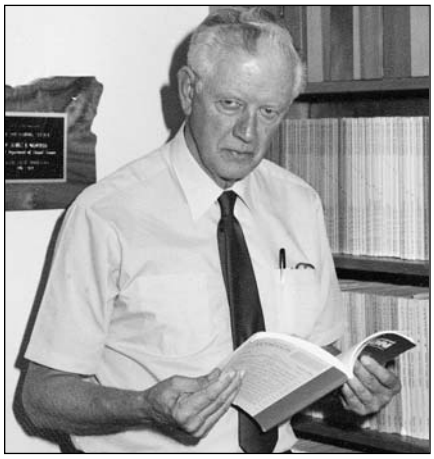
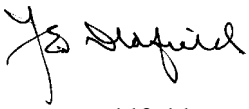


With this first issue of the Fur Animal Research newsletter of the new (2005) year, we are including an Index for articles published last year. The purpose of these indexes is to make it easier for you to find all the articles on a given subject and we hope you find it useful. The various articles are arranged under headings and we began with 5: Disease, Nutrition, Physiology, Genetics and a catch-all we called Awards and Reports. I recall mentioning in the first issue in the year 2000 that we were including a new area of research in our fundings, relating to environmental protection and problems related to it, and I predicted that our involvement in this area would grow. It has, and with reports on this type of work now becoming available we've added a new heading to our indexes: Management and Environmental Concerns. We hope you find these listings useful.

As most of you know, I am a displaced Canuck, so the decision by American Legend to move its February sale to Vancouver, B.C. was more than slightly interesting to me. They did a good job of outlining ways to get there and things to do, when not directly involved in the auctions. I would add a tip of my own - too late for the sale - but maybe useful in future visits you may make to the far Canadian west. The main border crossing on its I-5 freeway, at Blaine, WA, becomes very busy at times causing long periods of waiting in line. I avoid this by turning off I-5 at Bellingham, WA (at a large shopping mall called Bellisfair) and drive due north to another border crossing point near Aldergrove, B.C. This is usually far less busy and seldom involves much waiting time. If you have ever visited Lu Berneman's ranch, it is near the Aldergrove border crossing. Then, you follow straight north for a few miles until the highway meets Canada I, which takes you right into Vancouver.

Some of you in the Midwest and eastern states have told me that your weather this winter is not indicative of global warming, which is thought to be caused by damage of so-called "greenhouse gases" to the earth's protective ozone layer. I haven't reached a conclusion about this yet - whether the warming that has occurred is due to such damage, or whether it is part of a long-term cycle of warming/cooling weather. On the west coast, we have had a very mild fall, which is causing headaches to operators of our many ski resorts since the snow level is much lower than normal, but some of the ski season may be rejuvenated if we have a wet spring. I've been told that when you start writing about the weather, you're running out of more interesting topics, and its time to stop. We have what I hope are interesting topics in this newsletter but I'll stop anyway, wishing you success in your soon-to-begin breeding season.



J. E. Oldfield

HEATING-OIL PRODUCTION FROM MANURE

In a recent letter, I mentioned that an agricultural engineer at the University of Illinois, Dr. Yuanhui Zhang, had developed a process to generate heating oils from swine manure. Here is a little more information on the process. It is called thermochemical conversion (TCC) since it involves application of heat (275 C to 350 C) in a reactor. This is not new; TCC processes for energy production were studied, using wood sludge, paper waste, and municipal sewage sludge as base materials in the oil crisis of the 1970's. These carbonaceous materials are first converted to liquefied products by a series of chemical reactions and are then used to produce oil. The heating process takes place under pressures of 5.5 - 18 MPa and carbon monoxide is used as a reducing agent. The highest oil yield in the tests, which were made with swine manure was 76.2% of the total volatile solids in the base material. The only gaseous by-product of the operation is carbon dioxide and the solid by-product weighed only 3.3% of the total solids input.

So, here is a process that may have possible application in the mink business. It produces a useful energy source (oil) from the farm wastes. It helps overcome the odor problem, if in a continuing mode, since it removes the manure, as such, from the premises, and it reduces the solids that need to be disposed of by over 90%. The Illinois studies have been made with swine manure which is generated in considerable quantities on large swine farms and the much smaller quantities of manure produced by mink may make the process non-economic on fur farms. We will continue to monitor applications of this process and report further. (from: He, B.J., Y. Zhang, T.L. Funk, G.L. Riskowski and Y. Yin. 2000. Transactions, Am. Soc. Agr. Engineers 43:1823-1833).

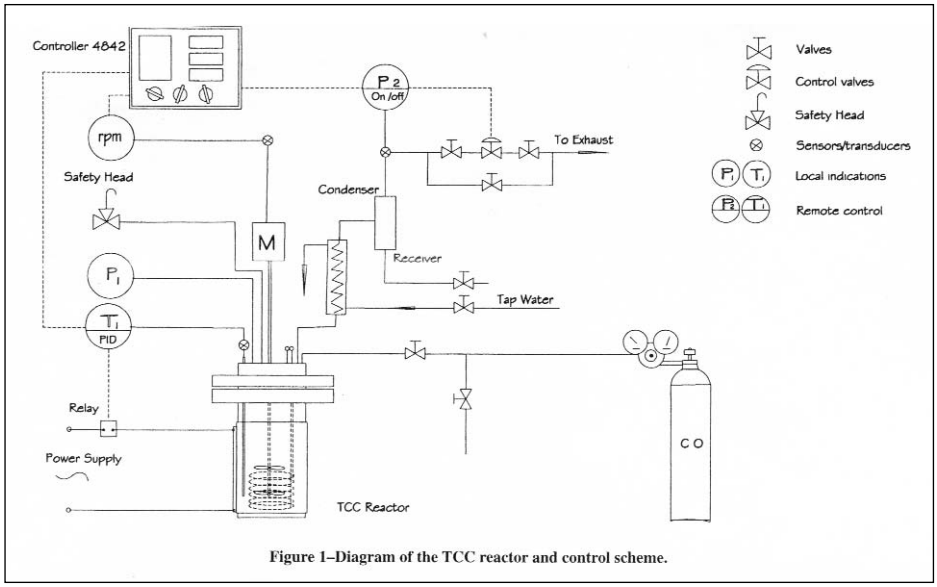


Figure 1–Diagram of the TCC reactor and control scheme.

TULAREMIA IN MINK

A few years ago, jackrabbits were plentiful and were sold for mink feed. If processed properly, these rabbits were an inexpensive source of protein. On the other hand, fur farmers should be aware of a common bacterial disease in rabbits known as tularemia or "rabbit fever."

Tularemia has a worldwide distribution and has been recognized in many areas of the United States. In addition to jackrabbits, snowshoe rabbits, cotton tails, muskrats and beaver may be infected. Tularemia is also a serious disease in mink, fox and humans.

The jackrabbit population of the West is never constant. The number of rabbits rises and falls in cycles. Veterinary epidemiologists believe

that these cycles may be due to biological regulators such as tularemia. When jackrabbits become numerous, an outbreak of tularemia follows and the jackrabbits are reduced in number. The cycle is then repeated every six to 10 years. It is Mother Nature's system of checks and balances.

Rabbits are easily infected with these bacteria. The most striking lesions at autopsy are multiple whitish necrotic areas in the lungs, liver and spleen. Healthy appearing rabbits can carry the bacteria and show no changes in the internal organs.

An outbreak among ranch-raised mink occurred in Illinois in 1956 after the farmer added two infected wild rabbits to his mink ration. In 1962 tularemia occurred in a mink herd in Wisconsin. The owner and one of the laborers also were infected.

The source in the Wisconsin outbreak apparently was skinned jackrabbits obtained commercially as the mink had no access to rabbits or rodents. There also have been some unconfirmed cases in mink farmers who skinned rabbits for mink feed and infected themselves as well as their mink.

An outbreak occurred in ranch mink in southern Idaho in a herd of about 5,000 mink. The ration included cereals, scrap ocean fish, beef tripe and rabbit and sheep carcasses. The owner stated that a loss of 26 animals occurred over a 10-day period. Some of the mink that died had gone off-feed slightly but others dropped dead with no other signs.

When the mink were autopsied, they were in fair body condition and had normal pelts. However, they appeared somewhat dehydrated. The most significant findings were small, ivory-colored nodules scattered throughout the lungs, liver and spleen. With the history of feeding wild jackrabbits and the typical autopsy picture described above, a tentative diagnosis of tularemia was made.

We say, "It is tularemia until proven otherwise!"

A final confirmation of the outbreak was made by a bacteriologic examination. The two possible sources if infection in the Idaho outbreak were rabbit and sheep carcasses. Tularemia has been found in both of these species in that part of Idaho.

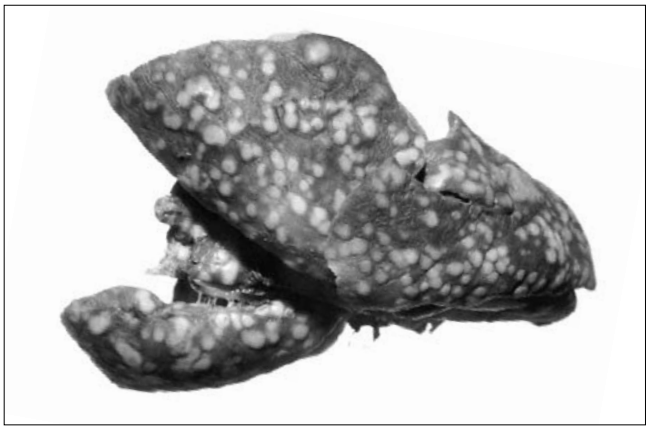


Figure 2: Lungs from a mink infected with tularemia bacteria. Note the white foci of dead tissue on the surface of the lungs.

At the time of the outbreak, we recommended adding therapeutic levels of oxytetracycline to the diet and injecting the sick mink with streptomycin. The losses ceased after about 60 mink had died. However, we did not know whether the treatment controlled outbreak or whether the losses stopped as a natural termination of the disease.

Some Other Facts:

We believe that the bacteria entered the mink by the intestinal tract.

However, tularemia bacteria are capable of entering the body through a wound cut, abrasion or directly through the unbroken skin. It is important, therefore, that skinners and other persons handling rabbit carcasses wear rubber gloves whether the disease is suspected to be present or not. We also warned the owner that he had been exposed.

These bacteria have been in carcasses 133 days and in hides as long as 40 days after death. Thorough cooking of infected tissue will kill the bacteria. Some jackrabbits are poisoned with strychnine, 1080, zinc phosphide or other toxicants. It is important that the mink farmer know the source of the rabbits or he or she will be buying trouble.

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GENETIC (INHERITED) DISEASES OF MINK

1. Tyrosinemia

With thousands of mink raised on an acre or two of land, coupled with excellent breeding records of knowledgeable mink farmers, the discovery of many genetic diseases of mink was not difficult.

There are diseases in which the coat color serves as a useful marker, i.e. Aleutian mink and the Chediak-Higashi Syndrome and deafness in Hedlund white mink. Then there are mink that are observed to be more susceptible to virus diseases. The susceptibility of the pastel color phase mutation to distemper has been recognized worldwide and the reported susceptibility of black mink to coronavirus infections has also been reported. No doubt the susceptibility and/or resistance to disease agents involve several genes.

Of course, in an overwhelming intoxication such as botulism or infectious-like anthrax, the genotype of the mink probably plays a minor role in whether the affected mink lives or succumbs.

Tyrosinemia is a hereditary disease that affects standard black mink (mustela vison). A deficiency in the activity of hepatic tyrosine aminotransferase (TAT) causes an accumulation of tyrosine in the blood of affected mink. Only mink that are homozygous for the recessive tyrosinemia gene will show signs of the disease. Which means the kit received a defective gene from each parent, making the parents carriers of the disease. Since tyrosinemia is a hereditary disease, not a communicable disease, it cannot spread from animal to animal within a herd.

Tyrosinemia was originally named pseudo distemper because its clinical signs closely resembled distemper. There are three forms of the disease, which differ in their time of onset, the duration of disease, and the severity of the symptoms. The disease that occurs in kits at about six or seven weeks of age is called the *early form* or the *spring form.* The *intermediate form* is seen in kits around three months of age and the *late form* or *fall form* appears around six months of age.

The Three Forms of Tyrosinemia: The *spring form* was the first form of tyrosinemia to be recognized. The clinical signs that characterize it are clouding of the cornea of the eyes, which is followed by development of crusts and raw areas about the eyes, nose and footpads a few days later. The farmer may notice the mink have bright red, raw noses and crusts around the eyes. There may be blood on the wire pen bottom due to raw areas on the feet. These animals begin to drink copious amounts of water and stop eating. Kits with the *spring form* of tyrosinemia rarely live longer than a week and usually die two to four days from the onset of the clinical signs.

The *intermediate form* of tyrosinemia is often seen in August through September. The symptoms are very similar to the *spring form* yet clinical signs appear at three months of age. Kits often have red or raw noses and toes which bleed readily even if handled with the utmost care. Their hair-coat is dull and the abdomen of the males may be wet due to frequent urination. Many of the males may have secondary bacterial urinary infections due to the frequent urination associated with this disease. The corneas look cloudy and the area around the eyes becomes matted after the cloudiness of the corneas is noticed. The kits may not have a hearty appetite or may quit eating. The affected animals may be in poor general condition and are usually smaller than the rest of the litter. At autopsy, most of the affected animals develop kidney lesions and many of those form yellow-brown kidney stones. Tyrosinemia has also been misdiagnosed as "plum bladder." Tyrosinemia compromises the lower urinary tract defenses which allow bacteria to gain entry. This bacterial invasion of the lower urinary tract is the probable cause of the urinary

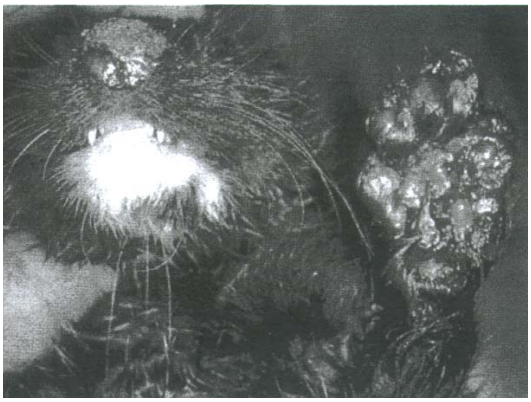

Figure 3. Tyrosinemia. Raw areas of denuded, bleeding skin on the foot pads. Their squinty eyes and nose become crusty and resemble a distemper-infected mink.

bladder stones. Death usually occurs 1-3 months following the onset of clinical signs.

The *late form* or the *fall form* has identical clinical signs as the *intermediate form* but the symptoms first appear during the time of pelting, when the kits are about six months old. Like the *intermediate form*, the kits may live for one to three months after the onset of clinical signs. These kits may live until pelting with minimal clinical signs, but are often not kept as breeders because they are in such poor general condition when compared to the rest of the herd. The only abnormalities these mink may show at pelting are their poor physical condition and kidney lesions. A pathologist may be necessary in order to make a definitive diagnosis in these individuals.

Diagnosis and Control of Tyrosinemia: Once tyrosinemia is suspected, a definitive diagnosis is crucial in controlling this genetic problem within the herd. The affected mink should be necropsied and samples of their serum can be analyzed by a diagnostic lab for elevated levels of the amino acid, tyrosine. At the present time, distinguishing carriers of the faulty gene is not possible in humans or in mink. A carrier animal is discovered when it produces offspring that present with the disease.

In order for the kit to present with the disease, both of the parents must be carriers for tyrosinemia. When two carriers are mated, 25 percent of the litter will succumb to tyrosinemia (these are homozygous kits), 50 percent of the offspring will be carriers, and 25 percent of the kits will be normal, on average. The carriers look identical to normal animals. Due to this fact, control of tyrosinemia relies on recognizing kits with clinical signs of tyrosinemia and recordkeeping to identify the parents and the littermates of the affected kits. Once the carriers are identified, they must be pelted out so that the number of carriers is reduced within the herd, with the ultimate goal of eliminating all carriers from the herd. Even with accurate records of pedigree, it can take some time to track down and identify the families in a herd where carriers exist, but it is vital in controlling the incidence of the defective gene in the herd. This process will be much more time consuming if females are mated twice to two different males. About fifty percent of the litter comes from each male when this mating method is used and only the dam of the affected kits is reliably known, which makes finding the carriers much more challenging.

Presently, the only way to eliminate tyrosinemia from the herd is identifying affected (homozygous) kits and having a recordkeeping system that allows identification of the parents of those kits (heterozygous carriers). Once the carriers are recognized, they can be eliminated from the herd. In the future, a test to detect carriers will make identifying carriers much more efficient.

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HOUSING MINK KITS, SINGLY OR IN PAIRS

Danish investigators have studied the practice of housing a breeding female with one male kit, after weaning, until pelting - looking at effects on the mink's behavior and comfort. The test animals were selected at the end of June and allocated to four groups. The females had borne 6-10 kits each and had all whelped during the same week in April. Group 1 females were caged singly, group 2 females with 2 male kits from another litter, group 3 females were caged with one male kit from another litter, and group 4 females were caged with one of their own male kits. The results showed that having more than one mink per cage increased positive behavior, including grooming and play, but it increased biting and the number of fights. Stereotyping, which is aimless running back and forth, was reduced in the cages with one male kit, and was highest in the cages with one female alone. Stereotypes were also high in the cages with two male kits, as were fights and bitings, which caused pelt damage. It was felt that this grouping - female with two male kits - should be avoided. Stereotyping is sometimes felt to indicate a stress condition (from: Pederson, V. 2004. Behavior and production parameters as an indication of welfare in female breeding mink during different social housing conditions from weaning to pelting time. In: Annual Report 2003, pp. 17-26).

POULTRY BY-PRODUCTS PRESERVED WITH FORMIC ACID AS MINK FEED

Poultry by-products are generally well-established as a useful feed ingredient in mink diets. Such by-products normally contain the viscera, heads, feet and other discarded materials from the filleting of poultry for human consumption.

In Norway, poultry by-products preserved with formic acid (HOOCH) were fed at 3 different percentages of the diets to groups of 60 female mink each (30 brown and 30 dark genotypes). The experiment started on January 14 and ended at weaning when the kits were 42 days old. The test animals were kept in cages equipped with nest boxes and fed once daily. Feed was restricted prior to mating but the females were fed to appetite after whelping and during the pre-weaning growth period. Details of the diets fed are shown in Table 1.

The experimental diets were eaten readily by the mink females and their body weight gains averaged about 200

	Poultry by-products 5.7%	Poultry by-products 17.3%	Poultry by-products 29.9%
Fishmeal	3.4	2.3	1.2
Meat-and-bone meal	4.6	5.8	7.2
Acid preserved poultry by-product	**5.7**	**17.3**	**29.9**
Slaughterhouse by-product (cattle)	8.0	6.2	-
Preheated carbohydrates	14.9	15.0	15.5
Vitamin-mineral supplement	0.2	0.2	0.2
Water	27.1	23.7	22.1
Dry matter	35.4	36.8	38.4
Ash	5.1	5.2	5.4
Crude protein	13.6	14.1	14.5
Crude fat	5.9	6.3	6.1
Carbohydrates	11.0	11.2	11.0
Digestibility:			
Crude protein (apparent)	79.7 (0.9)	79.4(1.2)	80.1(0.9)
Crude fat	86.7 (1.9)	91.4 (1.0)	92.6 (0.9)
Carbohydrates (by difference)	62.2 (3.6)	62.8 (1.9)	60.8 (2.3)
Metabolisable energy (MJ/kg)	5.28	5.62	5.61
ME distribution (%) between protein, fat and carbohydrates	38-39-23	38-41-21	39-40-21
Hygenic quality and pH:			
pH	5.9	5.4	4.9
Total bacterial count, million/g	1.2	2.2	0.3
Coli bacteria, per g	15,000	12,000	290
Fungus spores, per g	6,000	4,600	500

Table 1. Feed composition (%), pH, analyzed average chemical content and digestibility (%), measured energy data and hygienic quality.

grams during gestation, which should give the females sufficient energy to face the critical lactation period. The two diets containing lower levels of poultry-waste by-products (5.7% and 17.3%) supported similar weight gains but those on the highest level (29.9%) were somewhat smaller. It was proposed that poultry waste by-products, preserved with formic acid, could be fed at levels up to and including 17.3% in diets for gestating mink (from: Ahlstrom, O., Anders Skrede and Helen K. Smith. 2004. Formic-acid preserved poultry by-products as a feed ingredient for mink in the reproduction and early-growth periods. NJF Seminar 354, October 8-10, Lillehammer, Norway).

BOTULISM: IMPROVED TESTS

The University of Wisconsin's Medical School and its Food Research Institute have jointly announced two new tests for botulism. They expect that these will improve the possibility of developing substances to lessen the impact of botulinus toxin, one of the world's most poisonous substances and also that they will help toward the dangers of botulism use by terrorists.

The two tests, they feel, are vast improvements on the current technologies to detect botulism. The first involves use of a test-kit and can be used in the field. The second is a laboratory test that can be done with common laboratory equipment. It works by introducing bioluminescent proteins into cells, whose glow is extinguished by the botulinus toxins. This lab test will allow for rapid screening of large numbers of chemical compounds, to see which might inhibit the paralyzing effect of the toxin. Current tests involve exposing mice to the toxin, which takes time. The new tests are fast and do not involve deaths of lab animals (from: University of Wisconsin/Madison College of Agriculture and Life Sciences Quarterly. Winter, 2005, p. 1.)

FIBER IN MINK DIETS

Fiber has been a controversial substance in mink feeds. It is essentially indigestible by mink. Since animals generally eat enough to meet their energy requirements, it was thought that inclusion of an indigestible substance would cause them to eat more, but this doesn't seem to be the case. Danish workers at the Fur Breeders' Research Center, at Holstebro, report an experiment using different levels of dietary fiber. Three groups of female mink were fed diets containing different levels of fiber - low, medium and high. The energy contents of the diets were 199, 178 and 163 Kcal/100 g diet, respectively, and the animals were fed the diets from January 1 to February 29. In mid-January, the group on the low-fiber diet had no feed left on their cages after 6 hours. Corresponding times to clean up the medium and high-fiber diets were 10 and 12 hours, respectively. There were no significant differences among the groups in number of kits per female, either at birth or at 42 days of age. It was concluded that high fiber feed reduced the time spent without eating, by mink (from Hansen, S.W., T.N. Clausen, J. Malmqvist, B.M. Damgaard and C. Hejlesen. 2004. Fibers in mink diets in the winter period. Influence on behavior and reproduction. Ann. Report 2003, pp. 27-34. Danish Fur Breeders Research Center, Holstebro).

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