FUR ANIMAL RESEARCH

Volume 8, Number 3
September 2000

Published by the Mink Farmers' Research Foundation
A Committee of Fur Commission U.S.A.
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This newsletter is going to be a little different from the usual, since I am including material prepared for presentation at the VIIth International Scientific Congress in Fur Animal Production, held earlier this month in Kastoria, Greece. Specifically, I am presenting material prepared by Drs. Gorham and Westlake, and by myself. Drs. Gorham and Westlake have prepared a very interesting summary of results of use of 4-way type vaccine, that I feel sure will interest you. This is new material that has not been previously published. My own presentation is a brief, historical summary of some effects of metabolic antagonists in mink diet. These are substances that interfere with (are antagonists to) the normal effects of dietary nutrients. It is important to remember that there are two causes of nutrient deficiencies in diets. The first, obvious one is that there may not be enough of some essential nutrient in the feeds that make up your diet mix. The second is that, although the levels of dietary nutrients may be okay, there is something in the diet that interferes with their use. This "something" is a metabolic antagonist, and I describe several of them that have caused problems in mink production. Most of you will recognize the situations I describe – they are not uncommon in mink production – but you may not be aware of just how their involvement was first discovered. This is, in fact, a brief description of how nutrition research with mink is conducted, and I hope you find it interesting.

To these two more lengthy discussions I've added a few short topics on items that have been brought to my attention. These include some thoughts about feed testing for bacterial contamination: a topic that bothers many of us, and one about preservatives. The mink business is like no other type of livestock production in that it involves such a wide array of feed ingredients and additives. As producers, you want to know how well various feedstuffs and additives will work for you, and as your research arm, the Mink Farmers' Research Foundation will try to get the information you need to you. In later issues of this newsletter, I will report on other matters of interest that came up at the international meeting. Looking ahead, I wish you a successful pelting season. Kind regards,

J. E. Oldfield

SODIUM BISULFITE AS A PRESERVATIVE

I have been asked about the use of sodium bisulfite as a preservative in mink feeds. Some studies run some years ago, in the Scandinavian countries, cautioned against its use, noting that it destroyed vitamin B_1 (thiamin) in the diet, resulting in similar symptoms to those caused by thaminase in certain types of fish: Chastek's paralysis. In human foods, sulfiting agents are looked upon as effective food processing substances in a wide variety of applications, including dough conditioners, antioxidants and color stabilizers. There have been adverse reactions to sulfites, however, including asthmatic attacks, rashes and abdominal upsets. The U.S. Food and Drug Administration (FDA) acted in 1986 to reduce the likelihood that sensitive individuals would be exposed to sulfited foods. The level of sulfites added to foods must be declared, and the use of sulfites on fruits and vegetables that are to be eaten raw, and are presented to the public as "fresh" is prohibited. We suggest that, until more positive information becomes available, preservatives other than sulfites should be used in mink feed mixes. (from: Warner, C.R., D.W. Diachenko and C.J. Bailey, 2000. Sulfites: An Important Food Safety Issue. Food Testing and Analysis 6:8-14.)

METABOLIC ANTAGONISMS IN MINK

Nutrient deficiencies in diets for mink may be of two types: there may simply not be enough of a specific nutrient in the feed to meet the animals' requirements, or the nutrient may not be available, biologically, because of the presence in the diet of substances that interfere with its normal function. Such interfering substances are called *metabolic antagonists*, and they are the subject of this paper. For about 20 years, we operated a program at the Experimental Fur Farm of Oregon State University, to investigate metabolic antagonists that interfered with the normal health and well-being of mink, including effects on the color and quality of their fur. This program was directed by Dr. F.M. Stout.

Cotton Fur

The first problem we tackled was "cotton fur," a condition in which the dense underfur of the mink fails to pigment normally, but remains white and 'cottony' (Figure 1). The breakthrough in this research came when it was noticed at pelting time, that the carcasses of cotton mink were much paler in appearance than those from mink with normal fur coloration. Dr. Stout immediately suspected that anemia was involved and blood tests quickly confirmed this. Normal mink blood has a hemoglobin level of about 18 g% (grams/100 cc blood) but blood from cotton mink had values of about half that, and sometimes as low as 4 g% (Table 1).

What had caused this? We examined the diets that seemed to be producing the most 'cottons' and found that they included fairly high levels of fish – primarily Pacific hake (*Merluccius productus*). Other fish have been shown elsewhere to produce similar symptoms. These included Whiting (*Merluccius bilinearis*), and, in the Scandinavian countries, 'coal fish' (*Gadus virens*). Our studies were reported in the Journal of Nutrition (Stout, Oldfield, Adair, 1960a,b).

The obvious way to correct the 'cotton' problem was to avoid feeding the causative fish species but we were interested to learn whether it could be overcome by diet changes. The occurrence of anemia suggested an iron deficiency, yet the diet contained adequate levels of iron. Examination of the flesh of the hake fish involved in the problem showed that it contained formaldehyde and this appeared to be interfering in some way with normal iron metabolism. Scandinavian workers (Helgebostad and Ender, 1968) later showed that the causative compound was a formaldehyde derivative, trimethylamine oxide (TMAO). Iron is necessary for the formation of melanin, the dark pigment in mink fur and when it was not available, the fur was not pigmented, hence the 'cotton' appearance. Iron is not well absorbed and we were not successful in preventing cotton pelts by supplementing diets with iron; however, it can be given by injection as iron dextran. The best

Table 1. Blood values for normal and "cotton" mink.

Mink Type	Hemoglobin, g %	Hematocrit, %	Number of Animals
Normal	18.7±0.6	45.0±3.1	32
"Cotton"	10.8±3.0	28.1±8.8	27

Figure 1: Pelts of "cotton" (left) and normal, dark mink, parted to show underfur.

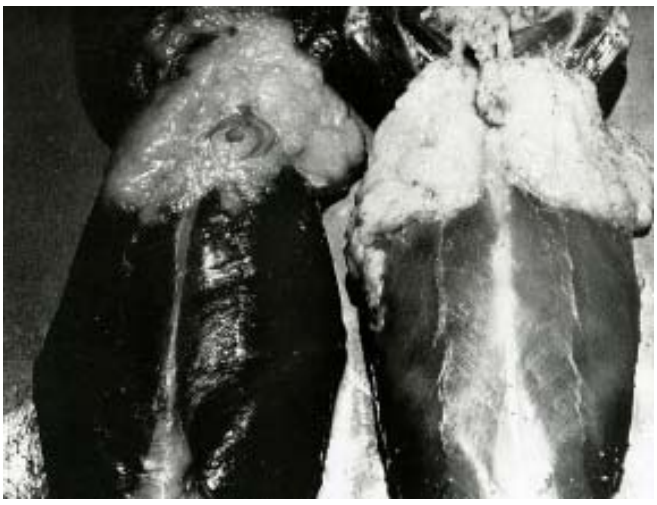


Figure 2: Pelted carcasses of normal (left) and "cotton" mink, illustrating the anemic condition of the latter.

protection continues to be restricting the level of causative fish (hake, in our experience) to about 10–20% of the diet.

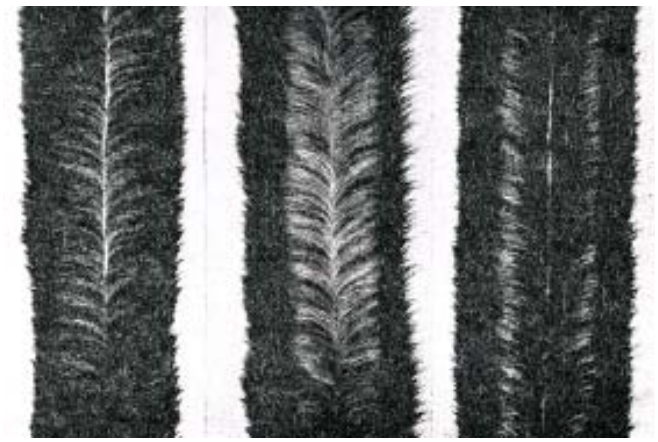
Thiaminase

We had been aware of the condition "Chastek Paralysis" which was first identified on fur farms in Minnesota, and we knew that it resulted from feeding high levels of certain fish which contained the enzyme, *Thiaminase*. This enzyme destroys the thiamine (vitamin B_1) and the consequent deficiency of it caused the paralysis (Figure 3).

I should mention an incidental problem with thiaminase. It also causes a complete loss of appetite, so one cannot take the usual route of supplementing the diet - the mink simply won't eat it. Giving thiamine by injection is quickly effective, however, and paralyzed mink so treated will be up on their feet, and eating, within an hour. Our National Research Council recognized the problems caused by thiaminase, and published an extensive table naming the fish species which contained it.

Our experience with thiaminase was an interesting one. We had not encountered thiamine deficiency on our fish diets, but in one experiment where we fed a high level of hake to produce cotton pelts, we noticed some of the mink became partially paralyzed. Dr. Stout cut open the hake and found that they had been feeding heavily on anchovies. The hake itself did not contain thiaminase, but the anchovies did. The results of this study were published in a short note titled, "A secondary induced thiamine deficiency in mink" (Stout, Oldfield, Adair, 1963) and the effects of the lack of thiamine on mink growth are charted in Figure 4.

Figure 3 (Above): Mink showing paralytic symptoms caused by a deficiency of Thiamine.



Turkey Waste Greying

The third, induced nutrient deficiency that I would like to describe to you involves one of the B-complex vitamins, *Biotin*. In the 1960's there was a thriving turkey-production industry in Oregon, and the visceral wastes from the turkey packing plants were available, at low cost, as mink feed. One year we were surprised to find that our dark mink were producing banded, partially grey pelts, which made them practically worthless (Figure 5).

Figure 5 (Below): Grey-banded pelts of standard, dark mink, resulting from a deficiency of biotin. Left - normal pelt; center - grey-banded pelt from mink fed 70% of turkey waste; right - grey-banded pelt from mink fed 50% turkey waste.

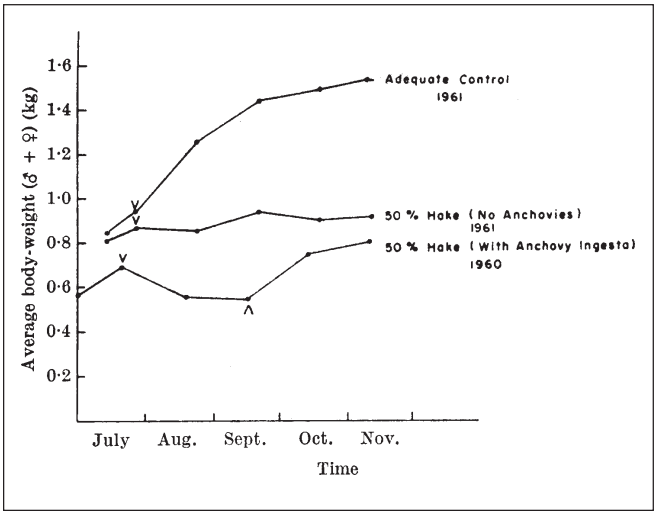


Figure 4 (Left): Growth curves of mink fed an adequate, basal diet and diets containing hake, with or without anchovy contamination.

These grey pelts were produced by mink on diets containing high levels (30-70%) of turkey waste and it seemed logical to assume that it was in some way involved in the pelt greying. A puzzling item was, however, that many Oregon mink farms were feeding similar levels of turkey waste without any problems.

We were aware that a deficiency of the B-vitamin, biotin, would affect pelt color and quality. A mild biotin deficiency causes loss of fur color (*achromotrichia*) and a severe deficiency causes loss of fur (*alopecia*). Dr. Stout correctly diagnosed a biotin deficiency and the next year showed that biotin supplementation of the diet would avoid the problem.

We were still perplexed that we would have such a severe problem, while other mink farmers feeding apparently similar diets did not. The answer, when it became evident, was simple. The actual compound causing the deficiency was *avidin*, which is a metabolic antagonist to biotin. The avidin is produced in eggs of chickens and turkeys, and is particularly high in turkey eggs. At our small Experimental Fur Farm, we followed the practice of buying all our feed for the year at one time, and keeping it frozen until needed. In the year we experienced the pelt-greying problem, we happened to buy our turkey waste at a time when turkey breeders were culling their hen flocks, so we got viscera from hen turkeys, which contained eggs. Other Oregon fur farmers, with much larger operations, bought turkey waste through the year, on a continuing basis. In this way, they received mostly viscera from tom turkeys and any small amounts of hen turkey viscera were diluted to the point where they caused no problem (Table 2). The antagonistic factor was apparently heat-sensitive, since cooking the turkey waste avoided the problem.

Unlike the cotton fur condition we had worked on previously, the greying of the pelts did not constitute complete lack of pigment - nor was it accompanied by the severe anemia, so we understood that we were working with a different entity. We developed recommendations on biotin supplementation, and these are generally being followed in the industry, wherever eggs, or viscera containing them, are being fed.

Summary

I have described three instances of metabolic antagonism, each of which has caused significant problems in the fur industry. The nature of these antagonisms differs. In the cotton pelt problem, trimethylamine oxide reacts with, and binds iron, so that it is not available to the animals. In the thiaminase problem, the enzyme attacks, and destroys vitamin B_1 (thiamine). And in the turkey-waste greying situation, the antagonist is avidin, which combines with the diet's biotin, making it unavailable for its normal metabolic functions. Continued research is needed so that the fur industry can identify such problems as they appear and propose ways to avoid, or overcome them.

— J. E. Oldfield

References

Stout, F.M., J.E. Oldfield and John Adair. 1960a. Nature and cause of the "cotton-fur" abnormality in mink. J. Nutr. 70:421-426.

Stout, F.M., J.E. Oldfield and John Adair. 1960b. Aberrant iron metabolism and the "cotton-fur" abnormality in mink. J. Nutr. 72:46-52.

Stout, F.M., J.E. Oldfield and J. Adair. 1963. A secondary induced thiamine deficiency in mink. Nature 197:810-811.

Stout, F.M., J. Adair and J.E. Oldfield. 1966. Turkey waste greying – an answer. Natl. Fur News 38:13, 19.

Ender, F. and A. Helgebostad. 1968. Studies on the anemiogenic properties of trimethylaine oxide in fish-induced anemia in mink. Acta Vet. Scand. 9:174-176.

Table 2. Occurrence of fur-greying on high-turkey waste diets.

Diet	Turkey type	Resulting fur color
88% turkey waste	Adult, breeder hens	All grey
88% turkey waste	Young birds	All normal
70% fish + raw turkey eggs		All grey
70% fish + cooked turkey eggs		All normal

THE AVIDIN/BIOTIN RELATIONSHIP: GOOD OR BAD?

As mink ranchers, we are familiar with the problems caused by avidin, a substance found in eggs - particularly turkey eggs - which interferes with the activity of the B vitamin, biotin, and causes greying of dark mink pelts. Presence of avidin in eggs or in poultry waste containing eggs has created biotin deficiencies in mink diets and necessitated supplementation with biotin - a costly process. I found it interesting to read recently evidence that avidin isn't all bad. Scientists at a U.S. Department of Agriculture laboratory at Kansas State University have found it possible to introduce avidin, which is a protein, into corn grain. The reason for doing this is to control some of the insect pests that infest corn crops. Biotin is necessary for the normal growth and development of these insect pests. When avidin is introduced into the corn grain, the insects develop a biotin deficiency and die. The researchers say that avidin is a better protectant than another bacterial substance, *bacillus thurengiensis* (Bt), which is sometimes used to protect against insect infestations. Apparently avidin corn is not a problem to consuming animals, and it stores much better, over longer periods of time. Avidin is used in medical and biochemical kits as a kind of diagnostic protein, and when it is produced in corn, the cost is lower than when it is isolated from chicken eggs, the usual source. Chicken-egg avidin sells for around $3,000 a gram. So it's interesting that a substance that has been bad news for the mink industry turns out to have an important, beneficial use in the cereal grain business. (from: McGraw, L. 2000. Avidin. In *Agricultural Research* 44:8-9.)

AVOIDING BACTERIAL CONTAMINATION OF FEED

I get a number of questions about possible problems of bacterial contamination of mink feeds, and how to guard against it. Many ranchers regularly have plate counts run on their feed ingredients, but are unsure of what the results mean, and, in fact, whether the tests are worthwhile. I have talked with microbiologists at the University and throw out a few suggestions for your consideration. First, there is no universally applicable bacterial test. Choice of the test to use depends on the nature of the diet. Eggs, which are commonly being fed in quantity, are likely to be contaminated with different strains of bacteria than, say, fish or meat-based diets. Bacillus tests, for example, are less useful on eggs than tests for Salmonella. Your testing laboratory should be helpful to you in deciding on what tests will be most useful.

Many of you acidify your feeds to lower the chance of bacterial problems, and this is often done with phosphoric acid. Acidification is measured as pH, and a pH of 4.0 or lower generally holds bacterial growth in check. It is important to remember that a low pH stops bacterial growth but does not actually kill the bacteria. This means that you have to be careful about what happens after the acidified feeds are mixed with the rest of the diet. When eggs, at a pH of 4.0 are mixed with other diet ingredients, the pH may rise to ineffective levels. Another thing that keeps bacteria in check is low temperatures, so if you are able to keep your feed mixes near freezing until the feed is put on the cage wire, it usually helps. We have bacterial problems with mink diets that are uncommon in diets for other animal species because of the nature of the mink diet, which contains fresh feeds – fish, meat, eggs and their by-products – that are excellent media for bacteria to grow on. Dry feeds are usually not a problem bacteria-wise.

CUTTING BACK ON PHOSPHORUS

Environmental regulations are becoming increasingly strict about substances that are released, via manure from animal operations, into soil and groundwater. One such substances is phosphorus, and some domestic animal producers are looking at phosphorus levels in their animal diets to see whether they can be lowered without affecting productivity of their livestock. There is some evidence that they can. A recent release from the Agricultural Research Service (ARS) of the U.S. Department of Agriculture suggests that dairy producers are feeding diets containing more phosphorus than necessary. By adjusting dietary phosphorus levels downward by 20%, the phosphorus needs of the cows can still be met while dairy producers in the U.S. would save $100,000,000 a year in ration costs and ground water quality would improve, says Dr. Larry Satter, who is with the U.S. Dairy Forage Research Center in Madison, WI. One way of dropping dietary phosphorus levels is to use lower-phosphorus feeds. The same thing might be done with mink diets, although many of the meat/fish/poultry products fed to mink tend to be naturally high in phosphorus. The NRC book "Nutrient Requirements of Mink and Foxes" lists phosphorus requirements of mink at from 0.3 – 0.6% of the diet dry matter. The lower figure is for maintenance of adult mink; the higher one is for feed during gestation/lactation. When you are having nutrient analysis run on your mink feed mixes, it might be worthwhile to get phosphorus run, just to see where you stand, relative to the NRC recommendations (partly from Caparella, T., 1999. Cutting Phosphorus on the Minds of Researchers. **Render** 28(5), p. 7).

DISTEMPER AND ITS CONTROL

At the International Scientific Congress in Greece, Drs. Westlake, Gorham and Durrant presented a review of the current status of distemper and vaccines used to control it and have kindly permitted me to reproduce it for you here.

While the cardinal signs of distemper (eye and nose exudates, swollen footpads and nervous signs) are recognized by most mink farmers, the first cases that occur on large farms are frequently not diagnosed. This situation may occur when there has been a failure of the vaccine to effectively immunize. On these farms, the farmer feels secure that the mink are protected and is not concerned about distemper.

Distemper Outbreaks

There are two unfavorable outcomes for vaccination failure. Obviously, if the exposure occurs in the kits that are considered to be immune in the summer or fall, and if the first cases are missed, the outbreaks will be underway before the kits are revaccinated with an effective vaccine.

It is even more devastating when the exposure occurs in the unprotected females and her kits during the following May, June or July. Here there is no maternal antibody conferred to the kits because the females were not effectively immunized the previous summer. Not only are the first cases of distemper in the kits often missed, but outbreaks during May, June and July are very difficult to control.

In 1998, widespread outbreaks of distemper occurred in the United States and Canada when there was a failure of the distemper vaccine to immunize. Two scenarios will be described. Table 1 shows dates of diagnoses and vaccination, the estimated kit deaths and percentage of deaths on farms where distemper was diagnosed prior to primary vaccination. It is obvious that the protection afforded by maternal antibody did not occur. Also, when the kits were vaccinated, the distemper losses were higher than one would anticipate if an effective vaccine was employed. The table illustrates 30,183 of a population of 93,700 (32%) died.

Similarly, the same vaccine failed to adequately protect kits that had been vaccinated at 10-12 weeks of age and later exposed to distemper. The dates of diagnoses, revaccination and estimated kit losses due to distemper are provided in table 2. One nine farms, the estimated kit deaths to distemper were 37,833 of 177,280 (21%).

Four Component Vaccines

A vaccine containing: (1) live attenuated distemper virus, (2) formalin-killed mink enteritis virus, (3) formalin-killed pseudomonas bacteria, and (4) botulism Type C formalin prepared toxoid is termed a 4-way vaccine designed for a single injection.

The product is widely used in the United States and Canada and was employed in the outbreaks described above.

Separate evaluation for efficacy and safety for each of the four aforementioned components should be done before the product is released for use. The live virus distemper component should always be tested after the addition of the three formalized components. While there are procedures to neutralize the formalin, there may be a question on whether residual formalin or some

other factor might reduce the efficacy of the live distemper component when the four components were mixed together prior to injection.

The time interval required for the development of distemper immunity in mink is inversely related to the number of units of vaccine virus units in the inoculum. It requires about two weeks for immunization with a minimal dose of 30 units of vaccine virus (Svehag and Gorham, 1962). In order to provide a margin of error and possible detrimental environmental effects on the vaccine, commercial vaccine should contain more than 3,000 units per mink dose.

Duration of Immunity

There has been almost no research done in the United States and Canada regarding the duration of immunity to distemper following primary immunization (after the kits reach 10 weeks of age or older). On field evidence, we feel that a single effective vaccination protects mink for their "economic life"; i.e., 2 to 3 years of age. Even if a few mink lose their immunity at 2 to 3 years, it should be difficult for an outbreak to start on a farm and maintain a "train of infection." In this situation, the outbreak would subside. Moreover, most mink, particularly Aleutian genotypes, are pelted after 2 years.

REFERENCES

Svehag, S.E., Gorham, J.R. An attempt to demonstrate the minimal number (estimated by probit analysis) of biological units of chicken embryo-propagated distemper virus required to immunize ferrets and mink. *Archiv ges Virusforsch* 12:250-258, 1962.

Table 1. Estimated kit deaths on farms where distemper was diagnosed prior to vaccination.

Farm Number	Distemper Diagnosed	Vaccinated	DV Deaths/Total Kits	% Kit Losses
10	16 May 98 in Kits	6, 8, 10, 14 weeks of age*	15,083/54,000	28
11	8 June 98	15 June - 3 Aug 12-18 June**	1,100/2,700	41
12	10 June 99	6-15 July*	14,000/37,000	38

A 4-component vaccine was used*
- Distemper-attenuated live virus
- Mink virus enteritis – formalin killed virus
- Pseudomonas bacterin – formalin killed
- Botulism toxoid – formalin killed

Attenuated distemper vaccine only as an injectable or as a spray vaccine**

Total kit deaths	30,183
Total number of kits	93,700
Percent kits dead	32.0

Table 2. Estimated kit deaths on farms where distemper was diagnosed after kits had been vaccinated at 10-12 weeks of age.*

Farm number	Distemper diagnosed	Revaccination	Deaths/Total Kits	% Kit Deaths
1	27 July 98	28-29 July 98	3,500/7,000	50.0
2	28 Aug 98	12-20 Sept 98	23,000/27,600	83.0
3	23 Sept 98	1-3 Oct 98	971/2,880	34.0
4	25 Sept 98	25 Sept/Oct 5 98	1,500/6,800	23.0

From: Current Infections Disease Problems in the United States: Mink Distemper

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