Studies on the Wet-belly Disease of the Mink WILLIAM L. LEOSCHKE, Valparaiso University

The wet-belly disease of the mink is characterized by an accumulation of urine in the fur adjacent to the urinary orifice (Fig. 1). De-

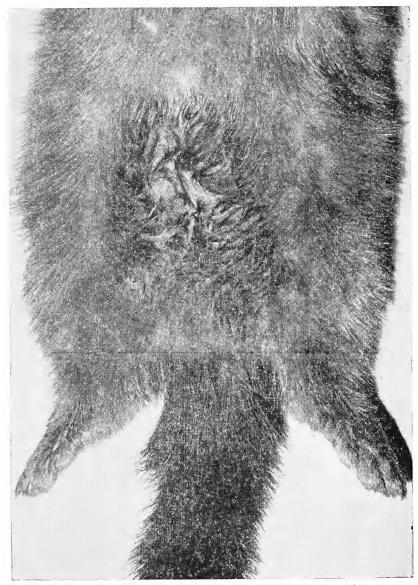


Figure 1. Typical symptoms of wet-belly disease of the mink.

CHEMISTRY

composition of the urine results in permanent damage to the pelt. Nutritional studies have indicated a correlation between the incidence of the disease and ranch diets containing high levels of fat (2,3) or an imbalance of calcium and phosphorus (3,4). Bacterial infection may also be a contributing factor in this complex malady (1).

The wet-belly disease is primarily limited to male kits in the late fall months or during the breeding season in March. These ranch observations led to a study on the role of male hormones (methyl testosterone) in the wet-belly disease of the mink. This work was supported in part by a research grant from the Mink Farmers Research Foundation, Milwaukee, Wisconsin.

Experimental

The 60 day experimental feeding program was initiated in late September 1960. A group of 80 pastel kits were ramdomly assigned to 3 groups consisting of 10 males and 10 females for each experimental group (A and B) and 20 males and 20 females for the control group (C). All animals received a practical ranch diet consisting of cereal mix, 15%; lactalbumin cheese, 6%; cooked eggs, 9%; chicken heads, 37%; chicken heads and entrails, 13%; beef rumen, 9%; and ocean fish, 11%. The control mink received this ranch diet supplemented with 1% corn oil. The experimental animals received this same ranch diet and a 1% corn oil supplement providing either 0.4 mg (Group A) or 4.0 mg (Group B) of methyl testosterone per pound of wet feed. With the supplemental corn oil the control and experimental animals received a diet containing about 21.7% fat on a dehydrated basis.

Results and Discussion

Observations of the mink for the characteristic symptoms of wetbelly diseases were made once a week during October and November. By mid-November a significant increase in the incidence and severity of the wet-belly disease was noted in the male kits of the hormone supplemented groups (Table 1). No wet-belly disease symptoms were noted

	Wet-belly Disease	
Grouping ¹	Incidence ²	Severity ³
C Control	5%	1
A 0.4 mg	20%	3
B 4.0 mg	40%	6

TABLE 1

Dietary Methyltestosterone Supplementation and Incidence and Severity of Wet-belly Disease.

¹ mg methyltestosterone per lb. wet feed.

² Incidence on Nov. 19, 1960, point of highest wet-belly disease incidence.

³Arbitrary classification with the number representing total points per 10 animals: 1—slight case; 2—good case; 3—severe case and 4—very severe case. Figure 1 is representative of a 2 rating. in the female kits in either the control or experimental groups. Cessation of the hormone treatment resulted in an immediate drop in the incidence and severity of the external manifestations of the disease.

Examination of the kits at pelting in late November revealed the presence of yellow stained fur and an area of blackened leather about the urinary orifice of the wet-belly diseased animals. The fur quality of the experimental and control animals was similar. Significant clitoris development was noted in the female kits receiving methyl testosterone. It is interesting to note that the rancher observed a direct correlation between the level of male hormone supplementation and the difficulty of removing the pelts from the experimental animals. Pelts from the female kits receiving the male hormone were about as difficult to remove as normal male pelts. Pelts from the male kits treated with methyl testosterone were almost impossible to remove without tearing.

It is apparent from the data presented that the presence of a male hormone (methyl testosterone) in the diet of ranch mink resulted in a significant increase in the incidence and severity of wet-belly disease. It is difficult to pin-point the exact role of male hormones in the production of this disease. It is possible that the male hormone may increase the kidney excretion of those chemicals responsible for the characteristic symptoms of the wet-belly disease of mink. This may be done by a direct action on kidney function or by influencing the levels of certain chemicals in the blood. It has been well documented that male hormones raise blood levels of the beta-lipoproteins (containing high levels of cholesterol and triglycerides) while female hormones raise the level of alpha-lipoproteins (containing high levels of phospholipids and protein). Reference has already been made to the correlation between dietary fat levels and the incidence of wet-belly disease of the mink.

Literature Cited

- GUNN, C. K. 1962. Actiology of wet-belly in ranch-bred mink. Nature 194: 849-851.
- LEOSCHKE, W. L. 1959. Wet-belly disease research. Amer. Fur Breeder 32(6): 16-17.
- 3. OLDFIELD, J. E., F. M. STOUT and J. ADAIR. 1962. Investigation of factors causing wet-belly disease in mink. Progress Reports of the Mink Farmers Research Foundation, Milwaukee, Wisconsin.
- SCHAIBLE, PHILIP J., H. F. TRAVIS and G. SHELTS. 1962. Urinary incontinence and wet-belly in mink. Quart. Bull. Michigan Agr. Exp. Station 44(3):466-483.