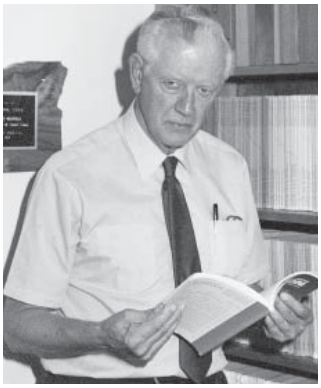
Fur Animal Research
Published by Mink Farmers' Research Foundation, a Committee of Fur Commission U.S.A
Volume 16, Number 1 March 2008



John Gorham has sent me word of the death of Dr. Richard Max Shackelford, at his home in Madison, Wisconsin, at age 92. Dr. Shackelford was truly one of the giants in fur animal research and he was considered a world authority on fur animal genetics. He was instrumental in describing the genetics of various color phases of mink that made it possible for producers to develop breeding plans for their propagation. The following material on Dick Shackelford was provided by the University of Wisconsin/Madison.

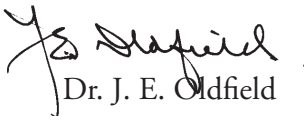
For nearly 40 years Richard Shackelford conducted extensive research on the color phases of fur-bearing animals at the university's Fur Animal Research Laboratory, sponsored in part by the U.S. Department of Agriculture, Division of Animal Husbandry. He was a valuable consultant to mink ranchers in the U.S., Canada and Scandinavia and was elected to the Fur Farm Industry's Hall of Fame. He had many friends in the Great Lakes Mink Association (GLMA), which developed the Blackglama trademark and the Mutation Mink Breeders Association (EMBA), for which he was instrumental in developing new color phases. In 1949, he established a two-day Mink Farmers' Summer School in Madison, which attracted 200 mink breeders from home and abroad. He also served for more than 15 years as chairman of the Classification Committee and chairman of the Educational Committee at the international mink shows in Milwaukee and in 1969 was named "Mr. International" at this annual event. On his promotion to the rank of full professor in 1968, he was considered the world authority on the genetics of fur animals. In 1974, he received a distinguished alumnus award from Murray State University.

In the latter part of his tenure at the University of Wisconsin he enjoyed teaching and coordinating an undergraduate course in the biology of companion animals. He retired in 1984, but maintained a close association with colleagues and took part in cultural activities at the university and in the community for many years. He loved reading, gardening, tending orchids, raising (and eating) guinea fowl, breeding canaries and watching wild birds in rural Mount Vernon, where he lived for 45 years and was known locally as "the professor on the hill." He was an active member of the Madison Orchid Guild, attending meetings and shows with his close friends, Jan and Judith Rapacz. After he reluctantly quit driving at age 91, he was grateful for the support of the Dane County Outreach, who sent a van to pick him up for lunch in Mount Horeb on weekdays. Thanks to a daily check-in with neighbors, Andy and Florence Connors, who picked up his mail, plowed his driveway, drove him on errands and did countless odd jobs around his house, Dick was able to continue his independent lifestyle right up to his sudden death from a fall while cooking dinner. He is survived by his son and daughter-in-law, Jole and Frankie Shackelford of St. Paul; grandsons, Gorm of London, England and Leif of Oakland, California;

sister-in-law, Marilyn Shackelford; nephews, Steven and Tim Shackelford and niece, Susan Davis and their families.

It is always sad to report the passing of one of our fur industry's active researchers but the nature of Dick Shackelford's genetic studies will ensure that his contributions will continue well into the future. Much of his work has been published in a book, "Genetics of the Ranch Mink." I am reminded that the University had the first Department of Genetics in the U.S. – Dick Shackelford focused on fur animals, mink and fox, and a friend of mine, Dr. A. B. Chapman headed work with larger farm animals, especially cattle and sheep.

I wish you all the best in 2008.



Dr. J. E. Oldfield

ENZYME ADDITIONS TO MINK DIETS

Mink have a very short digestive tract which means that the time of passage of their feed through the tract is short, too. This means that much of the mink feed may pass through the system undigested. Digestion is brought about by enzymes that break down feed ingredients into particles small enough to be digested. Norwegian workers have tried to increase digestibility of the mink diet by adding enzymes to it.

Introduction

Dietary enzyme addition is universally established as an efficient approach to improve nutrient digestibility, feed conversion ratio and performance in monogastric animals. The use of feed enzymes also contributes to reduced output and impact of environmental pollutants such as phosphate and nitrogen. Especially in poultry, use of feed enzymes has been shown to allow greater ingredient flexibility in least cost formulation.

The extensive use of feed enzymes in diets for poultry and pigs is targeted towards the use of cereal grains as major energy contributors in the diets. The level of metabolisable energy in a cereal will depend on the content of cell wall non-starch polysaccharides (NSP). The main NSP fractions in barley and oats are the mixed-linked (1→3) 1→4)-ß-glucans, whereas wheat and rye contain mainly arabinoxylans. Moreover, antinutrients like phytates (myo-inositol hexaphosphate) or salts of phytic acid, are major problems associated with most vegetable feed ingredients.

Although most studies with poultry and pigs have shown benefits from dietary enzyme supplementation, only very few studies have been carried out with other monogastric animals such as mink and foxes. However, promising results have been obtained in mink fed wheat-containing diets. As a carnivore species with short digestive tract, rapid feed passage, relatively low amylase activity, and limited microbial fermentation in the large intestine, the mink has lower capacity to digest carbohydrates than most terrestrial monogastrics. Dietary NSP, such as the) ß-glucans and arabinoxylans, are considered to be indigestible and may protect nutrients such as starch from the action of digestive enzymes.

Previous studies have shown that lactic acid fermentation of barley increases carbohydrate digestibility in mink and Atlantic salmon and improves growth and feed conversion ratio in broiler

chicken. Lactic acid fermentation may also eliminate indigestible NSP and other antinutritional factors in soybean meal. The present study was carried out to investigate effects of lactic acid fermentation of barley, using two different strains of lactic acid bacteria, on contents of antinutrients and nutrient digestibility in mink with or without enzyme supplementation. In this paper main attention is directed towards enzyme supplementation, whereas a more comprehensive presentation of results regarding effects of fermentation on antinutrients in barley and digestibility in mink has been published previously.

Material and Methods

Digestibility experiment

A digestibility study was carried out with 9 month old male mink (Mustela vison) of the standard genotype, kept in individual cages equipped for quantitative feeding and collection of feces. The animals were allotted to eight groups of four animals each. Wet diets with approximately 20% of metabolisable energy (ME) from carbohydrates were used. The diets were made 1 day prior to start of the experiment, and stored at –20ºC until start of thawing in a refrigerator about 20 hrs before feeding.

The animals were fed daily individual rations of approximately 200 kJ ME during a 3-day preliminary period and a 4-day fecal collection period. Feeding and collection of feces were carried out once daily. Drinking water was available ad libitum. Pooled feces from each animal were freeze-dried, and ground and sieved for removal of hair pending analysis.

The diets contained barley as the sole source of carbohydrate (Table 1). The barley was ground on a hammermill to pass a 3 mm screen at Center for Feed Technology, Ås, Norway. The experiment included four treatments of the barley: untreated (Diet 1), fermented with a Lactobacillus plantarum/penosus strain isolated from a spontaneously fermented sourdough from Norwegian rye (Diet 2), fermented with a starch-degrading Lactobacillus plantarum (Diet 3), and irradiated using ^{60}Co γ rays at 25 kGy (Diet 4). The fermentation procedure involved mixing of the cereal with water (1:1.2 w/w), inoculation with a 1 x 106 bacteria g-1, and incubation at 30ºC for 18 h. The irradiated barley was soaked in water (1:1.2 w/w) and incubated at the same condition as used for fermentation.

Each diet was fed either without added feed enzymes or with addition of Porzyme 811 (0.11 g kg-1 as fed) during diet preparation, containing ß-glucanase (250 U/g), xylanase (400 U/g) and amylase (1000 U/g).

Table 1. Ingredient composition of diets fed to mink (g kg-1 as fed)

Ingredients	
Barley*, unfermented or fermented	189.9
Fish meal†	91.9
Raw fish filleting scrap	339.8
Soybean oil‡	50.0
Vitamin and mineral premix¶	50.0
Water§	

* Sunnita variety, Bjørke experimental station, Ilseng, Norway.

† Norseamink, Norsildmel, Bergen, Norway.

‡ Denofa AS, Fredrikstad, Norway.

¶ Norsk Mineralnaering, Hønefoss, Norway. Ingredients per kg: Vitamin A; 2,000,000 IU, vitamin D3; 200,000 IU, vitamin E; 50,000 mg, vitamin B1, 15,000 mg, vitamin B2; 3,000 mg, vitamin B6; 3000 mg, vitamin B12; 20 mg; pantothenic acid; 3000 mg, niacin; 5000 mg, biotin 30 mg, folic acid; 300 mg, Fe (amino acid-chelated); 20,000 mg, Zn oxide; 7,500 mg, Mn oxide; 15,000 mg and Cu sulphate; 1,250 mg.

§ Added to achieve the same total water content in all diets.

Results and Discussion

All animals remained healthy throughout the course of experiment, and there were only minor feed refusals.

Addition of feed enzymes to the diets as well as type of diet (treatment of barley) had significant effects on apparent digestibility of CHO, starch, CP and fat (Table 2). Digestibility of CHO showed significant interaction between diet and enzyme addition ($P<0.05$), whereas no such interaction was seen regarding digestibility of starch, CP and fat.

In the present study, dietary enzyme supplementation increased significantly the overall digestibility of starch from 75.2 to 79.8%. CHO digestibility was increased by enzyme addition to all diets used in the experiment with an average increase from 51.4 to 60.2%. Fermentation of barley with bacteria strain AD2 caused higher starch and CHO digestibility than fermentation with the lactic acid bacteria strain AM4.

Carnivore animals are usually fed lower levels of cereals and vegetable protein sources than poultry and pigs, and there are few experimental data justifying use of supplemental feed enzymes. In Atlantic salmon, another carnivorous species, on-line enzyme supplementation during feed processing has been shown to improve digestibility of minerals, i.e. phosphorus, zinc and magnesium (Denstadli et al., 2006a).

In the chicken, ß-glucanase supplementation has been shown to improve starch digestibility of normal and waxy barley (Ankrah et al., 1999). Børsting et al. (1995), working with mill fractions of wheat for mink, also showed improved starch and CHO digestibility following enzyme supplementation. It should be noted that our study was carried out with wet diets, and neither the barley nor the compound diets were subjected to the heat and moisture required for starch gelatinization.

Except for the feeding of diets with and without feed enzymes, the different treatment of the barley was the only difference among diets. The barley contributed only minor proportions of dietary CP and fat; fish meal and fish filleting scrap were the main protein sources, and soybean oil was the major fat source. Nevertheless, there was a significant overall increase in CP digestibility from 85.2 to 86.4% due to enzyme addition, and a corresponding overall increase in fat digestibility from 92.8 to 93.7%. These rather minor but significant effects of enzyme supplementation on digestibility of protein or fat, may partly reflect disruption of cell walls and release of entrapped nutrients in the barley, but may also be related to associative effects on digestion of protein and fat from other sources in the diet.

Table 2. Effects of enzyme supplementation to diets

	Diet 1	Diet 1 + enzyme	Diet 2	Diet 2 + enzyme	Diet 3	Diet 3 + enzyme	Diet 4	Diet 4 + enzyme	Pooled SEM	P-values Diet	Enzyme	Diet x enzyme
Digestibility												
CP	84.2^{d}	85.5^{abcd}	85.2^{cd}	86.9^{a}	86.0^{abc}	86.5^{abc}	85.3^{bcd}	86.6^{ab}	0.30	0.007	0.001	0.24
Fat	92.2^{b}	93.0^{ab}	93.2^{ab}	93.9^{a}	92.6^{ab}	93.8^{ab}	93.2^{ab}	94.2^{a}	0.31	0.02	0.001	0.89
Starch	68.^{8d}	77.5^{b}	83.0^{a}	84.7^{a}	75.6^{bc}	80.9^{ab}	73.4^{cd}	76.1^{b}	1.13	0.001	0.001	0.43
CHO	44.5^{d}	55.2^{bc}	58.8^{b}	66.1^{a}	51.1^{c}	59.9^{b}	51.4^{c}	59.3^{b}	1.31	0.001	0.001	0.03

^{abcd} *Numbers with different superscript within a row are significantly different ($P<0.05$).*

with differently treated barley on apparent nutrient digestibility in mink (%).

In the present study the contents of soluble ß-glucans, as well as ð-amylase inhibitors and phytate were reduced by all treatments of the barley, including γ-irradiation, soaking and incubation. This may partly explain the observed effects of diet on starch and CHO digestibility. Thus, it is interesting to note that enzyme addition to Diet 1, which contained untreated barley, resulted in greater increase of starch digestibility than enzyme addition to the other diets. However, fermentation with the AD2 bacteria strain improved digestibility of starch and CHO compared with γ-irradiation, subsequent soaking and incubation, although there were rather minor differences in contents of mixed-linked (1→3) (1→4)-ß-glucans and ð-amylase inhibitors. Other workers observed no effect of a wide range of dietary phytate levels on digestibility of starch or organic matter in the Atlantic salmon. Thus, the differences in starch digestibility in our study were probably not caused by different contents of inositol phosphates. Other factors affected by fermentation in addition to contents of mixed-linked (1→3) (1→4)-ß-glucans and ð-amylase inhibitors and phytate, such as reduced content of oligosaccharides, may therefore have been of importance for the digestibility of carbohydrates from the barley used in our study.

Enzyme addition after fermentation with the lactic acid bacteria strains AD2 and AM4 promoted a further increase in digestibility of CHO, but no significant effect on starch digestibility. This indicates that fermentation may convert indigestible fibre to digestible components, and degrade antinutrients such as ð-amylase inhibitors, capable of interrupting the effects of digestive enzymes. This could also explain why the effects of fermentation of barley and enzyme addition on CHO digestibility in mink to some extent appeared to be additive.

Cereals used in diets for fur animals are usually heat treated which may improve carbohydrate digestibility due to starch gelatinization, in spite of loss of endogenous enzymes, like ß-glucanase, in the cereal. We conclude that it m ay be possible to improve digestibility of nutrients in diets for fur animals by enzyme supplementation. The target enzyme substrates in diets for fur animals would be partly different from those of major importance for pigs and poultry. Thus, further studies should be carried out to explore digestibility as well as long-term effects with a variety of common feed ingredients. The current study was carried out with an enzyme product designed for use in pig diets. In the industry, advanced fermentation technology is used to produce a wide range of enzyme products intended for different markets. The suitability of specific feed enzymes may depend on the species of animal, and further improvement in effects on digestibility may be obtained by adapting the enzyme product to the characteristic digestive system of carnivorous mink and foxes.

Anders Skrede and Øystein Ahlstrøm
Norwegian University of Life Sciences
Department of Animal and Aquacultural Sciences
P.O. Box 5003, NO-1432 Aas, Norway

A PSEUDORABIES OUTBREAK IN MINK

John R. Gorham, DVM • Washington State University, Pullman, WA99169

I received a call asking me if it was safe to feed raw pork by-products to mink. I answered by telling the caller about a virus disease of pigs called pseudorabies that has caused mink outbreaks in Europe, Russia and Japan and in the United States.

Ohio Outbreak

Pseudorabies was diagnosed on a farm that had 6,600 adults and 24,000 kits. A total of 525 (approximately 2%) died over a 6-7 day period. It is a fatal disease. Any adult or kit that showed signs of the disease died.

Symptoms. After eating the raw pork scrap, the mink refuse to eat, salivate, become depressed, uncoordinated and their muscles tremble, followed by posterior paralysis. Death occurs within 12-24 hours after the first signs were observed. Sometimes a mink will chew its foot, leg or back (see accompanying photograph).

Autopsy. Small hemorrhages were seen in the lungs, stomach and intestines. There was blood-tinged fluid in the lung cavity. Actually, there were no findings that could nail down a diagnosis. Laboratory tests, cell culture and rabbit inoculations confirmed the diagnosis.

The ration. Because of a favorable price, an affected mink farmer incorporated raw pork lungs (approximately 12%) as part of the ration for his mink. The ingredients of the ration included ocean fish, poultry byproducts and cooked eggs. The rancher was advised that the lungs should be **cooked at 190 degrees F. for 30 minutes** before being added to the ration to prevent possible pseudorabies virus contamination. The cooking of the pork lungs for the prescribed time was discontinued after approximately one year because of the hassle of cooking the lungs. After again feeding uncooked pork lungs for 120 days, the pseudorabies deaths re-occurred.

It is a good suggestion to cook raw pork products because of the possibility of pseudorabies-contaminated pork viscera. Furthermore, mink farmers should not feed raw pork byproducts to pregnant female mink because of the potential danger of abortions caused by Salmonella. In Russia, all pork byproducts are cooked if they are to be included in mink rations.

Ferret exposed to pseudorabies virus has chewed itself. Infected mink go off feed, show signs of salivation, padding movements and posterior paralysis prior to death.

MASTITIS IN MINK

Introduction

The classical clinical signs of mastitis (rubor, tumor, dolor, calor) are rarely seen in lactating Danish mink females. Mastitis in mink has not been regarded as a significant problem in farm mink and has been sparsely investigated and is rarely mentioned in surveys of diseases in mink. Mastitis has been reported in Alaskan ranch mink. An epizootic of mastitis on a Connecticut mink ranch due to Staphylococcus aureus and Escherichia coli infections has been associated with feeding of condemned beef. Mixed staphylococcal and coliform mastitis with septicaemia in farm mink has been associated with severe necrotizing mastitis in mink with Aleutian disease. Mastitis has been hypothesized as a causal factor for "Greasy kits" syndrome in mink, but this theory has been rejected based on studies of mammary glands of female mink with litters suffering attacks of greasy kits. Recent investigations indicate that mastitis may be an underdiagnosed and important cause of neonatal death in mink. Results of pathological and microbiological investigations of neonatal death in Danish mink kits during 2005-2006 indicated that a relatively large proportion (13.2-38.1%) of neonatal losses is associated with bacterial infections in the mammary glands of the female mink. This report concerns the preliminary results of investigations of mastitis in Danish farm mink during the period 2005-2007.

Materials and Methods

The materials included 71 female mink females collected with lost or significantly reduced litters 0-2 days post partum. These mink were collected during the breeding seasons 2005-2007. Furthermore, 38 mink females were collected from 7 farms experiencing problems with mastitis in the breeding season of 2007.

Pathology. The animals were necropsied. Mammary gland tissue sections (approximately 1 cm2) and sections from liver, intestine and uterus were collected in phosphate-buffered 10% formalin, embedded in paraffin and sections were stained for histological examination. Mammary gland tissues were evaluated in hematoxylin and eosin (HE) stained slides for degree of inflammation using a method previously described. The cells in the lumen of each alveolus were counted in each of 10 lobules in a section. A mean number of cells per alveolus were obtained for each mammary gland section. Infiltrating cells not in the alveolar lumen was not considered.

Bacteriology. Primary cultures were made on blood agar (blood agar base, OXOID, supplemented with 5% calf blood), Drigalski, MacConkey agar and Enteric medium (Statens Serum Institut, Copenhagen, Denmark) and subcultured on blood agar. All media were incubated aerobically at 37°C for 18-24 h. Bacteria were identified from their appearance on agar media, haemolysis, odour, cell morphology, catalase and oxidase reaction and Gram properties. If necessary, identification kits were used (API ID 32E for E. coli and, API 20NE for P. aeruginosa and P. multocida, API ID 32 STAPH for S. intermedius, and API rapid ID 32 STREP for streptococci, bioMérieux, Marcy l'Étoile, France). Identification of S. intermedius was supplemented with a positive test for coagulase and negative test for hyaluronidase and identification of haemolytic streptococci with a test for positive reaction with Lancefield's group G or C antiserum (OXOID Diagnostic Reagents). All E. coli isolates were frozen at -80°C and freeze dried for later investigations. Salmonella species were detected according to the ISO 6579 guidelines.

Antimicrobial susceptibility testing. A semi-automated antimicrobial sensitivity testing system (Sensititre, Trek Diagnostic Systems, East Grinstead, UK), based on the broth dilution method, was used together with customized ready-to-use microtitre plates containing two-fold dilution amounts of

antimicrobial compounds. Different panels were used for different bacterial species. MIC breakpoints were as defined by Pedersen et al. Significance tests for differences between proportions of resistant isolates were calculated using StatCal in Epi-Info™ version 6. A significance level of 5% was applied ($p<0.05$). Fisher's exact test (2-tailed) was used when appropriate.

Other microbiological methods. Lung tissue was analysed for presence of CDV antigen by indirect immunofluorescence assay applied to freeze sections of lung tissue. Two monoclonal anti-CDV antibodies produced in mice (Laboratory of Clinical Virology, Huddinge) in 1:100 dilution were used as primary antibodies. Fluorescein isothiocyanate conjugated (FITC) were used as secondary antibodies essentially as described previously. The slides were assessed essentially as described previously. Blood samples from all mink were tested for antibodies against Aleutian disease virus (ADV) by counter immunoelectrophoresis. This test was performed at the Danish Fur Breeders Laboratory, Glostrup, Denmark.

Number of females	71
Dystocia	25/71 (35,2%)
Mastitis	22/71 (31,0%
Underdeveloped mammary glands	20/71 (28,2%)
Endometritis	12/71 (16,9%)

Table 1. Autopsy findings in female mink submitted to the Danish National Institute with an anamnesis of neonatal death (lost or significantly reduced litters) during the period 2005-2007.

Number of females	21
Farm size	2000-3000 females
Mortality due to mastitis	Low (< 5%)
Nesting material	Wood chip + straw (71, 3%)/straw (14.3%))/wood chip (14.3%)
Colourtype	Dark (89,0%)/light (11,0%)
Submission period	1-10 May (42.9%) and 21-31 May (57.1%)

Table 2. Findings in 27 mink from 7 farms with mastitis submitted from Danish farms with an anamnesis of problems with mastitis during breeding period 2007.

Results

All animals were negative for distemper, Aleutian disease, viral enteritis and salmonella and Chlamydia. Bacteriological analysis showed that a variety of bacteria, mainly Escherichia coli and Staphylococcus intermedius was involved in mastitis in mink.

Number of females	92
Light colour type mink	12 (13,0%)
Haemolytic Escherichia coli	38 (41,3%)
Haemolytic Staphylococcus spp.	23 (25,0%)
Streptococcus spp.	4 (4,3%)
Others (non-hemolytic Escherichia coli, micrococcus)	2 (2,1%)

Table 3. Bacteriological findings in mink 92 mink diagnosed with mastitis. The mink were submitted from 29 farms during the period 2005-2007.

Resistance among E. coli isolates was highest for ampicillin, streptomycin, sulphonamides, and tetracycline. Very low levels of resistance (<5%) were recorded to fluoroquinolones, gentamicin, florfenicol, amoxicillin with clavulanic acid, ceftiofur, chloromphenicol, colistin, nalidixic acid, and apramycin. Resistance to ampicillin (p=0.017) was significantly higher in isolates from the urogenital tract or mammary glands than in isolates from feces or intestine.

No S. intermedius isolate showed resistance to amoxicillin with clavulanic acid or fluoroquinolones, and resistance was also low for fucidic acid, cephalothin, kanamycin, potentiated sulphonamides, and chloramphenicol. The far highest resistance was recorded for tetracycline, while resistance to penicillin, macrolides, lincosamides and spectinomycin was all around 20%.

There were no significant difference between haemolytic E. coli isolates and haemolytic staphylococcal isolates from mammary glands and isolates from other organs.

Histopathological investigations showed that E. coli most often caused a peracute, necrotizing mastitis, while staphylococcal mastitis typically resulted in milder infections and abscessation of affected glands. The majority of the E. coli isolates (4%) were haemolytic coli bacteria.

Discussion and Conclusion

Bacteriological and pathological findings were consistent with previously published descriptions of mastitis in mink. The microbiological findings in the mink females supported previously published results, indicating that bacteria in the close environment of the animals (e.g. E. coli) may be associated with neonatal death (Table 3). These results indicate that optimization of the feeding management of breeding mink and hygiene management in the neonatal period are important preventive tools in relation to mastitis in mink farms. Results obtained from other species (cow, sow) indicate that certain management practices and environmental factors influenced the development of mastitis, which may contribute to the development of methods useful for controlling the disease. It is likely that predisposing factors for mastitis in mink may include poor nest box and cage sanitation, rough or sharp edges to the entrance of nestboxes, and high bacterial contamination of feed. Treatment and prevention involve improving management and treating individual animals or the herd with appropriate antibiotics based on sensitivity testing.

Mastitis was diagnosed in 31% of females submitted with an anamnesis of neonatal death (lost or significantly reduced litters 0-2 days post partum) included in this study (Table 1). The kit mortality is presumed to be due to bacterial infection or starvation because of reduced milk production.

Haemolysin is considered a virulence factor for E. coli in mink. In the present investigation, we distinguished between haemolytic and non-haemolytic isolates. The majority of the E. coli isolates (4%) were haemolytic. In a previous investigation of E. coli

from mink, haemolytic isolates were found to be more resistant than non-haemolytic isolates to tetracycline, amoxicillin, sulphonamides, trimethoprim, and spectinomycin. Many studies, executed during the last decade, indicate that the severity of E. coli mastitis in cattle is mainly determined by the immune status of the cow and the surrounding environment rather than by E. coli pathogenicity. During E. coli mastitis, the host defense status is a cardinal factor determining the outcome of the disease.

S. intermedius is one of the most commonly isolated pathogenic bacteria in mink as well as many other carnivores. This bacterial species is involved in many types of infectious conditions in mink, such a pneumonia and pleuritis, dermatitis, urinary tract infections, metritis, and mastitis. Thus, S. intermedius is closely connected with carnivores, and the infections are therefore likely to be caused by strains from the mink themselves, whereas the infections with other types of haemolytic staphylococci may have been acquired from other sources.

(from Mastitis in Mink; Anne Sofie Hammer, Charlotte M. Sørensen og Trine H. Jensen. Danish National Veterinary Institute, Department of Poultry, Fish and Fur Animals, the National Veterinary Institute, Technical University of Denmark, Hangøvej 2, DK-8200 Århus, phone: +45 72 34 61 17, email: ash@vet.dtu.dk.

INDEX

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

SECRETARY:

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

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

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

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

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

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

Author Dr. J.E. Oldfield • Desiged by Sandra Wallace