Fur Animal Research

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Dr. Jim Oldfield

It is a real pleasure to relay to you that Fur Commission U.S.A. has made a substantial gift to the John R. Gorham Fellowship in Veterinary Medicine at Washington State University. Disease is the greatest enemy that the fur industry has, and John Gorham is the greatest enemy of fur diseases. In the course of his distinguished career, he has virtually eliminated some fur animal diseases and has developed better diagnostic methods and treatments that ease many others The citation for this gift follows:

This is to notify you that this October, Fur Commission USA made a donation of $5,00 to the Dr. John R. Gorham Fellowship in Veterinary Medicine at Washington State University.

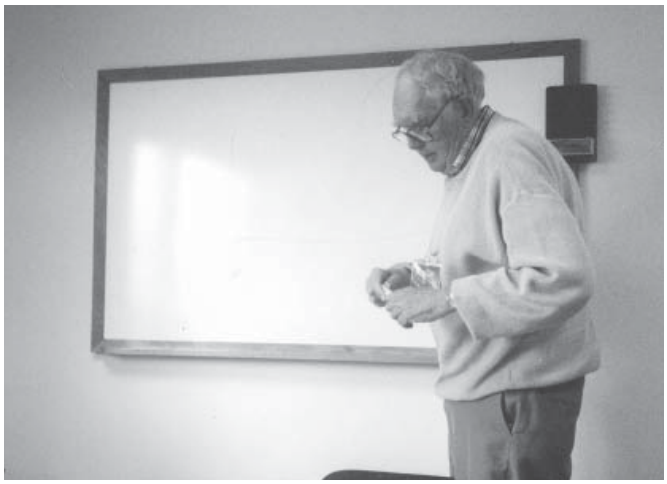
Following is a summary of Dr. Gorham's illustrious career, and of the fellowship itself, provided by Washington State University:

Dr. John R. Gorham, professor of veterinary microbiology and pathology at Washington State University's College of Veterinary Medicine holds the longest tenure of any faculty member ever employed in the college. Since receiving his DVM degree from WSU in 1946, he has continuously taught, conducted research, and provided service for the profession, the USDA, and the university.

A double alumnus of Washington State University with a Ph.D. from the University of Wisconsin, Dr. Gorham has published nearly 500 publications with more than 250 co-authors in the areas of viral, bacterial, genetic, and nutritional diseases and across clinical, applied, and basic research.

In 1970, Dr. Gorham received the Gaines Medal from the American Veterinary Medical Association for his landmark research into the acute viral diseases of dogs, and in 1963, he was awarded the highest honor granted to WSU alumni, the Regents' Distinguished Alumnus Award.

In 1985, Dr. Gorham received the XII International Veterinary Congress award given by the AVMA and in



the same year, he received the USDA's Distinguished Service Award for his remarkable legacy of research on the acute and slow viral diseases of domestic animals. Most notable among these were equine infectious anemia, Aleutian disease of mink, and distemper in dogs and mink. He was also the co-discoverer of the rickettsial cause of salmon poisoning.

In 1985, Dr. Gorham was the first veterinarian inducted into the USDA Hall of Fame and in 2001 he was given the American Epidemiology Society's Gold

Headed Cane.

In 2007, he received the Lifetime Excellence in Research Award from the AVMA. He is only the second recipient of the award that recognizes lifetime achievement in veterinary medical research.

The Dr. John R. Gorham Fellowship in Veterinary Medicine was established to foster the education and scientific curiosity of graduate students studying animal diseases. Virtually every living WSU veterinary alumnus in the world has had at least one lecture during their professional training delivered by Dr. Gorham.

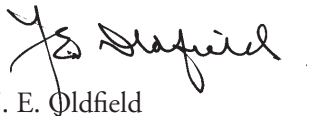
Today we ask you to join us in furthering this important fund that will live on long after Dr. Gorham is gone in the same way that his legacy of teaching, research, and service will.

If you would like to make a gift to the Dr. John R. Gorham Fellowship in Veterinary Medicine, contact Lynne Haley, Washington State University, College of Veterinary Medicine, P.O. Box 647010, Pullman, WA 99164-7010; Tel: (509) 335-5021; e-mail: lhaley@vetmed.wsu.edu.

Agreeing with a recommendation from the Research Committee, Ryan Holt moved that FCUSA, from its admin budget (broken down two thirds to Research Committee and one third to AWC/GAC) make a donation of $5,000 to the Gorham Fellowship at Washington State University, Bob Zimbal seconded. Approved.

I am sure you all feel, as I do, that this is a wonderful and most appropriate action by Fur Commission U.S.A.

Have a great Holiday Season.



J. E. Oldfield

It is appropriate to follow this announcement with yet another example of Dr. Gorham's continuing contributions.

CONVULSIONS IN MINK

Although they are rare, convulsions sometimes occur in mink affected with Aleutian disease (AD) or transmissible mink encephalopathy (TME). They occur more frequently late in the course of distemper, sporadically in epilepsy and terminally when mink are near death. A screaming fit describes the convulsion that is seen in the late stages of distemper.

ALEUTIAN DISEASE

chronic disease, nervous symptoms are first noticed when areas of the affected mink's brain are damaged by the AD virus. Depending on what part of the brain is involved, the mink may become uncoordinated and incapable of synchronizing movements in running or climbing. In later stages, the hindquarters appear to "flounder." When it falls on its back, it has difficulty righting itself and it may walk in circles or weave from side to side. An affected mink's head may continually bob and shake and it struggles to get into the nest box. Shrill cries are heard.

The mink may live a month, gradually losing weight before dying.

The lesions of AD always involve the blood vessels and kidneys. Degenerative changes in the blood vessels of the brain may slowly reduce the flow of blood. This occlusion leads to the death of brain tissue because of the lack of oxygen.

There must be a high prevalence of AD on a farm before nervous symptoms are noticed. The following field observations suggest a difference in AD virus strains. In Pacific Northwest outbreaks of AD that I have observed, only 2-3% of affected non-Aleutian mink showed nervous symptoms. On the other hand, on a Wisconsin farm 160 of 1150 non-Aleutian mink succumbed to AD, and of those, 50% had nervous symptoms.

For sorting out nervous or high and low virulent strains, research is needed based on molecular virology and the mink genome. This would be difficult and time-consuming. The development of AD resistant mink and really effective vaccines won't be easy.

TRANSMISSIBLE MINK ENCEPHALOPATHY (TME)

Convulsions are rarely observed in TME. Affected mink become uncoordinated in the hind limbs. Some mink act sleepy. They may bite a handling mitt and seem to go to sleep or they may clamp their teeth to a wire mesh. Often an affected mink's tail curls over its back like a squirrel. After a course of 3-8 weeks, a TME-affected mink always dies.

DISTEMPER

A screaming fit describes the convulsion that is seen in the late stages of distemper

Convulsions or "screaming fits" is a form of distemper that occurs in the late stages of the disease and may be seen even though the mink has not had the early signs of puffy crusty eyelids or swelling of the foot pads. Affected mink may appear normal and apparently recover only to die in a later convulsion. The distemper virus invades and damages the brain, causing the mink to throw its head back in spasmodic jerks with saliva bubbling out of the sides of its mouth. It may roll about the pen and scream sharply. The mink may appear to recover but invariably will develop convulsions within 1-2 days and usually will die with its teeth clamped onto the wire netting.

Over the years there seems to be a gradual change in the clinical picture of mink distemper. Often the same number of unvaccinated mink die during an outbreak but the eye and nose discharges are less serious. On the other hand, the occurrence of convulsions in outbreaks seems to be about the same. These observations might suggest a change in the distemper virus or perhaps a shift in the genetic makeup of ranch mink populations that alters their response to the distemper virus.

EPILEPSY

Epilepsy is a disorder in which nerve cells of the brain release abnormal electrical impulses that may lead to a seizure or loss of consciousness. In mink, the convulsions may occur spontaneously for no apparent reason but often are triggered by a disturbance of some kind. Such disturbances might include handling during vaccination, anxiety created during separation, transport within the confines of a small catch cage or noise from the feed cart. Some mink may scream briefly and recover while others may lose consciousness and go into a spasm where they claw at their throat or paw at the air. Some farmers claim they can bring a mink out of a convulsion by either dowsing it with cold water or stroking it for a few minutes.

An epileptic mink may have repeated seizures and, on rare occasions, the seizure may result in the death of the mink. The condition also seems to have a hereditary component since litter mates may have convulsions. Almost all mink farmers have seen this condition. Affected mink are usually marked and pelted in the fall.

TERMINAL CONVULSIONS

When the processes that maintain life are shutting down, whether caused by a virus, bacteria, nutritional imbalance, toxic food poisons, etc., the affected mink will more than likely have a convulsion prior to death.

John R. Gorham, DVM
College of Veterinary Medicine
Washington State University
Pullman, WA 99164

INDIVIDUAL DIFFERENCES IN THE BEHAVIOR OF FARMED MINK:

HOW DOES VARIATION IN ACTIVITY (SPECIALLY STEREOTYPIC BEHAVIOR) REFLECT DEGREE OF ADAPTATION TO TYPICAL FARM CONDITIONS?

Georgia Mason, Maria Diez, Allison Bechard and Rebecca Meagher
Department of Animal and Poultry Science
University of Guelph
Guelph, Ontario, Canada

Steven Bursian, Angelo Napolitano and Jane Link
Department of Animal Science
Michigan State University
East Lansing, MI 48824

Last year we introduced this project and presented its rationale. This year we show the first results. To date, these are largely descriptive and not very practically useful, but they do provide the essential building blocks for the planned work to come. The bulk of our findings, especially concerning breeding, maternal care, and kit output, will emerge this coming summer and so will be presented in next year's report.

How enriched housing affects mink behavior

The enriched housing conditions are a great success. They have functioned quite well practically, and had the effects we wanted on behavior. Thirty-two litters were reared in these conditions, one mink/litter being kept to adulthood. Thirty-two litters were also reared in standard conditions. When assessed in November 2007 (43.3% for standard conditions compared to 17.3% for the enriched conditions). The enriched conditions affected males even more dramatically (male stereotypic behavior/ activity was 73.2% in standard cages, 8.4% in enriched). This broad pattern was still evident when animals were re-assessed in February 2008. During the summer of 2008, we hope to investigate why males benefit so much more from the enrichment than did the females. One possibility is that they out-compete their sisters for access to enrichments, when pair-housed, and this differential access has lasting effects on them as they mature.

These effects of housing on stereotypic behavior were not just by-products of the mink being observed in large enriched cage versus small standard cages. They really did represent fundamental, biological changes intrinsic to the differentially-reared animals (just as we had hoped). Thus, when these animals were all moved to small cages elsewhere on the farm, the enriched-reared animals remained less stereotypic than the standard-reared, an effect that was still evident after three to four weeks of this housing in females although it waned in males.

Two other effects were also seen in the enriched and standard cages: enriched-reared females tended to be less inactive than standard-reared females (an effect we discuss below), while enriched-reared males were significantly more inactive than standard-reared males. Despite their lower levels of inactivity and higher incidence of stereotypic behavior, standard-reared males were about 10% heavier in bodyweight than enriched males, a surprising finding that we need to explore further (e.g. is it caused by increases in body length, fat deposition, or both?) and see if it occurs again in 2008's cohort of males.

Individual variation in behavior

In both types of housing, individual mink showed a very wide range of responses: stereotypic behavior in individual females ranged from 0 to 90.5% in standard conditions, and 0 to 62.7% in enriched conditions. Stereotypic behavior in individual males ranged from 34.3 to 98.4% in standard conditions and from 0 to 25.0% in enriched conditions. Individual mink also varied in how inactive they were, and in how much time they spent in normal activity. Statistical "family effects" show that at least some of this variation is genetic.

In 2008, we will be exploring this individual variation to see which "behavioral types" of mink perform best (see also "Inactivity" section below). For example, in 2007, we gained some pilot data from nursing females suggesting that there is consistent individual variation in the degree to which dams lick and groom their neonates (a behavior that helps program infant stress responses in other species). If this is confirmed, in 2008 we will see which "behavioral types" direct more of this beneficial behavior to their offspring. We also collected some pilot data in 2008 suggesting that enriched females prefer to mate with enriched males. When given a choice of who to mate with, enriched females visited enriched males nearly twice as often as standard-reared males (an effect that was statistically significant), although they did not copulate more often or copulate for a longer period of time with them. Later in 2008, we will use paternity testing of the kits to see if females allocated more fertile matings (i.e. at ovulation) to some types of males in preference to others. Over 2008's reproductive season, we will also investigate whether housing and behavioral type affect

breeding females' litter sizes, kit growth rates, and the dams' vulnerability to nursing sickness.

High levels of 'inactivity in the nest box' as a possible sign of poor welfare and poor performance in females

In our females in November 2007, there was no significant difference in inactivity levels between the two housing types. However, more females in standard conditions showed extremely high levels of inactivity. By February 2008, the gap between the two groups had widened, with standard females starting to look more inactive in general than enriched. This is important because we have collected some evidence that extreme inactivity, especially being inactive within the nest box, is a sign of poor welfare in female mink.

One piece of evidence is as follows. When differentially-reared mink were transferred to small standard cages to look for lasting effects of their rearing conditions (see above), the enriched-reared females reacted by becoming much more inactive: thus more inactive than they were in their enriched home cages (72% compared to 54% of the time), and more inactive than their standard-reared peers moved to the same conditions (72% compared to 63% of the time). For these females, this effect stayed fairly consistent over the three to four weeks spent in these small cages. Enriched-reared males, in contrast, reacted to the small cages by becoming increasingly stereotypic.

The second piece of evidence comes from data collected in parallel on a commercial farm. Here we examined the relationship between inactivity and reproductive performance in 350 females (color types: Black, Demi and Pastel). Behavioral data were collected through scanning observations conducted in the pre-feeding period, for four days prior to the commencement of mating. Although most females were stereotypic, some were inactive on over 90% of the scans (despite being watched in the most active period of their day, and one of the most active periods of the year). Our reproductive measures included nest quality scored around the time of parturition, litter size at birth and pre-weaning infant mortality. High levels of inactivity in the nest box prior to pregnancy predicted small litter sizes, and among experienced breeders, it also predicted higher kit mortality between birth and weaning. Some signs of a link between inactivity and poor nest quality were also found, although they were not consistent over time. We are currently replicating this study on another 200 females, and exploring the possible roles of fear, and/or of excess body fat, in these effects.

DEVELOPMENT OF AN ANIMAL MODEL FOR STAPHYLOCOCCAL ENTEROTOXICOSIS

Matti Kuipel[1], Dodd Sledge[1], Carol Volin[1] and Steven Bursian[2]
Departments of Pathobiology and Diagnostic Investigation[1]
and Animal Science[2]Michigan State University
East Lansing, MI 48824

Sticky kit disease is a devastating disease that affects mink kits often between 10 and 14 days of age. Large outbreaks at mink farms are not uncommon, causing death in 30-40% of affected kits. Disease is characterized by severe diarrhea and hypersecretion of cervical apocrine glands that result in severe dehydration, which can rapidly proceed to death. In necropsies of kits that died during an outbreak on a farm in Idaho, which were conducted at Michigan State University's Diagnostic Center for Population and Animal Health, it was shown that the intestinal mucosa of affected kits was colonized by gram positive coccal bacteria. Subsequent bacterial culture and typing showed that the bacteria were a strain of Staphylococcus intermedius that was capable of producing enterotoxins.

Staphylococcal enterotoxins have long been known to be a major cause of food poisoning in humans. However, the role of Staphylococcus enterotoxins in induction of emesis and diarrhea when ingested is poorly understood in regards to the underlying pathogenesis of disease. Also, of particular interest to this study, is the fact that it has been shown that Staphylococcus aureus can colonize the intestine of humans, similar to our observations of Staphylococcus intermedius colonizing the small intestine in mink with sticky kit syndrome.

The goal of this study was to inoculate groups of mink kits between 7 and 10 days of age with either the strain of Staphylococcus intermedius isolated during the outbreak in Idaho or enterotoxin derived from cultures of that strain of Staphylococcus intermedius or enterotoxin derived from Staphylococcus aureus that has been shown to cause food poisoning in humans. The results of this study indicated that sticky kit disease could not be produced by inoculation of the kits with the bacteria or enterotoxin. These results would suggest that other factors are required to produce this condition in mink.

Body Condition, n-3/n-6 Polyunsaturated Fatty Acid Balance and the Development of Fatty Liver Disease in Mink

Objectives of Proposed Research Program

The long-term goals of my research program are to better understand the underlying biology of the metabolic disorders of mink nursing sickness and the associated fatty liver syndrome. My objectives are to investigate in the liver the effects of dietary fatty acid supply and the role of different metabolic stressors, such as chronic over nutrition, rapid weight loss, and acute hyperglycemia, on liver fat synthesis and the activation of inflammatory pathways. I will also examine, under conditions of metabolic stress in the liver tissue as well as in cultured hepatocytes, the formation of Mallory bodies as an indication of the development of liver pathology.

Research Program Scope

It is to be noted that the proposed research program scope is somewhat different than what was previously proposed in the NSERC Research Partnerships Program as the application submitted in 2007 was not successful in obtaining funding. However, Dr. Rouvinen-Watt has since then renewed the NSERC funding support through the Individual Discovery Grants Program to support her research program titled "Body Condition, n-3/n-6 PUFA Balance and the Regulation of Lipid and Glucose Metabolism in the Mink" during 2008-2013. Investigating the causes and metabolic consequences of the fatty disease in mink is an integral part of t his research program. The research approach uses

both animal experiments as well as liver cell culture techniques to examine the physiological and molecular level mechanisms that lead to the development of liver pathology in response to different metabolic stressors. As the overall research program scope had to be redesigned for the Discovery Grants Program, the development of the liver cell culture component has been postponed and is now planned to begin during 2008-09 followed by the hepatocyte cell culture experiments as outlined in the following detailed research plan. The FCUSA funding during 2007-08 has been used to support part of the salary of two summer students and the fat, glycogen and liver morphological analyses in the recently completed mink fasting experiments.

Methods and Scientific Approach

The proposed research hypotheses will be investigated using the experimental approach outlined followed by the description of the scientific approach.

Hypothesis 1: The mink will develop fatty liver as a result of chronic over nutrition, rapid weight loss, acute hyperglycemia and induced oxidative stress, but the overall metabolic response will be altered by the tissue n-3/n-6 PUFA profiles.

Exp. 1.1 Effects of short-term food deprivation (rapid weight loss) and re-feeding on the metabolic response in mink: This study focuses specifically on the development of fatty liver and the associated metabolic changes. The animal experiment has already been completed with analyses underway. In brief, 30 male and 30 female mink were subject to 0, 1, 3, 5 and 7 d of fasting and a re-feeding period of 4 wks, 5 males and 5 females per treatment, in order to establish the time course for development of fasting-induced hepatic lipidosis and the subsequent regeneration of the liver tissue.

Exp. 1.2 Effects of body condition and dietary fatty acids on the metabolic response, inflammation and oxidative stress in the mink during rapid weight loss: This animal experiment has also been completed and included 72 juvenile mink, each treatment group consisting of 6 males and 6 females. The mink were fed at two feeding intensity levels: below (80% RDA) and above requirement (120% RDA) to induce lean and obese body types. Varying tissue fatty acid profiles were introduced by diets containing either soybean oil (predominantly n-6 PUFA; C18:2n-6), fish oil (rich in n-3 LCPUFA; EPA and DHA), and canola oil (rich in oleic acid C18:1n-9, ALA C18:3n-3). The diets were isocaloric and isolipidic. At the end of the feeding experiment half of the mink were fasted for 5 days, to examine the effects of rapid weight loss.

Exp. 1.3 Effects of fasting, acute hyperglycemia and oxidative stress on the metabolic response in female mink with varying body conditions and fatty acid profiles: This research will be carried out with 72 juvenile female mink from fall until winter. Within each dietary fatty acid source (soybean, fish, canola oil) and feeding intensity (80%, 120%) group there will be four treatments: control (CTRL; normal feeding), fasting (FAST; 5 day total fast), hyperglycemia (STZ; acute hyperglycemia induced by Streptozotocin), and oxidative stress (ROS; increased superoxide production induced by a mitochondrial complex III inhibitor).

Methods: At the end of each of the animal experiments, blood, liver, skeletal muscle and various adipose tissue samples will be collected, snap frozen and stored in 80^{o}C. The diets and adipose tissue samples will be analyzed for fatty acid profiles in order to determine the n-3/n-6 PUFA balance. I will evaluate the animals' metabolic response by measuring serum clinical-chemical parameters and

endocrinological indicators, such as growth hormone, cortisol, glucose, insulin, leptin, ghrelin and adiponectin. The gene expression of selected regulatory proteins will be assessed by quantitative real time polymerase chain reaction (qRT-PCR) assay of messenger ribonucleic acid (mRNA) levels. 18S rRNA will be used as a normalizing control. All samples will be run in triplicate with an acceptable coefficient of variation <10%. Adipose tissue, liver and the skeletal muscle will be analyzed for the gene expression of enzymes and proteins involved in lipid synthesis, storage and mobilization, gluconeogenesis, glucocorticoid recycling, insulin-dependent glucose transport, and the mitochondrial production of reactive oxygen species. My lab has mink-specific primers and optimized qRT-PCR assays for several of these target mRNA. All adipose tissue depots will be quantitatively dissected and weighed. Liver fat content will be analyzed and the type and severity of hepatic lipidosis assessed histologically. We will analyze the following indicators of oxidative stress and inflammation: glutathione (GSH/GSSG), malondialdehyde, DNA damage (comet assay), C-reactive protein, TNFα, and IL6. Further analysis of the liver tissue is described below in Exp. 2.1-2.3.

Hypothesis 2: Chronic over nutrition, rapid weight loss, acute hyperglycemia and oxidative stress will activate the inflammatory pathways and will result in the development of liver pathology. I plan to study the activation of the inflammatory pathways and the development of liver pathology using liver tissue collected from the mink as well as liver cell culture.

Liver tissue:
Exp. 2.1 Effects of short-term food deprivation and re-feeding on the activation of the inflammatory pathways and development of Mallory Bodies: Liver tissue samples for this research will be obtained from the fasted and re-fed mink in Exp. 1.1 as above.
Exp. 2.2 Effects of body condition and dietary fatty acid supply on the activation of the inflammatory pathways and development of Mallory Bodies: This experiment will use the liver samples collected in Exp. 1.2.

Exp. 2.3 Effects of fasting, acute hyperglycemia and oxidative stress on the activation of the inflammatory pathways and the development of Mallory Bodies: Liver samples for this study will be obtained from Exp. 1.3 described above.

Hepatocyte culture: Exp. 2.4 Effects of the inflammatory pathway on the development of Mallory Bodies: These studies will be carried out using hepatocyte cell cultures, with four independent experiments for each series. In the first series, I will study the activation of the inflammatory pathways by selective inducing and blocking of the signaling systems associated with oxidative stress and the sympathetic response.

Exp. 2.5 Effects of fatty acid nutrition and lipid synthesis pathway on the development of Mallory Bodies: In the second series, I will selectively induce and block the oxidative stress, hyperglycemia and sympathetic response as above in order to induce the lipid synthesis pathway. I will block this pathway with an antagonist. Methods: Oxidative stress will be induced by a respiratory chain inhibitor and blocked by a reactive oxygen species scavenger and hyper- and normolycemia will be implemented with varying concentrations of glucose, while epinephrine and propranolol will be used to stimulate and block the sympathetic β-adrenergic receptors.

Activation of the inflammatory pathway: As indicators of the inflammatory pathway activation, I will measure

the levels of the key enzymes and the end product prostaglandin E2 (PGE2) in the liver cells.

Activation of the lipid synthesis pathway: The response parameters will include the upstream regulatory element-binding protein (SREBP)-1c, and the lipogenic enzymes acetyl-CoA carboxylase (ACC), and fatty acyl synthase (FAS). The gene expression of these will be assessed using qRT-PCR.

Development of Liver Pathology and Formation of Mallory Bodies: I will examine the formation of Mallory bodies using immunofluorescence analysis. In brief, the liver cells will be fixed and incubated with primary antibodies for the target proteins present in the Mallory Bodies and then incubated with secondary antibodies. The morphometric analysis of the antibody-stained cytoplasmic inclusions will be done using fluorescent microscopy. I have access to all necessary facilities and equipment at NSAC to carry out this work.

Dr. Kirsti Rouvinen-Watt
Nova Scotia Agricultural College
Canadian Centre for Fur Animal Research
Carnivore Nutrition & Physiology Research Program

FEEDING UNCOOKED PORK BYPRODUCTS CAN LEAD TO SALMONELLA ABORTIONS

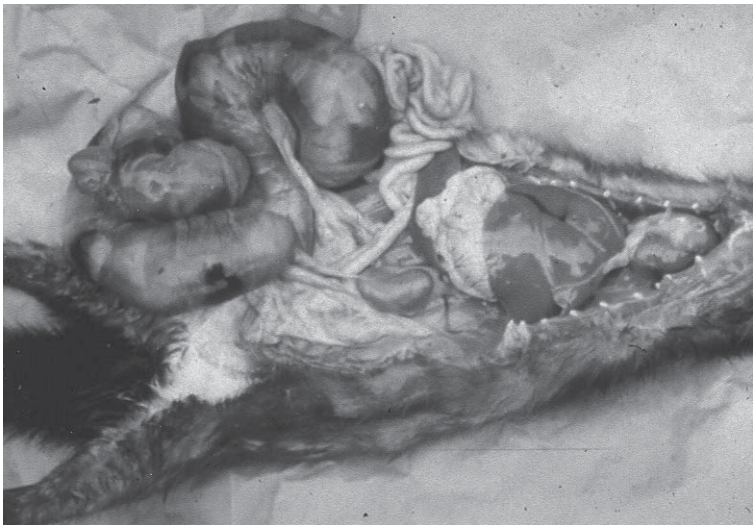
Dr. G. R. Hartsough reported abortion outbreaks in mink in the springs of 1946, 1947 and 1960. The source of infection in all three outbreaks was Salmonella-contaminated pork livers. With the exception of abortions, it is difficult to relate the occurrence of Salmonella to either clinical signs or autopsy findings. There were few deaths in the pregnant females but many kits were aborted and lost. A number of aborting females were killed and examined bacteriologically and, in every instance, Salmonella choleraesuis var. Kunzendorf was isolated from the uterus and unexpelled feti. In some instances, necrotic metritis (inflammation of the uterus with tissue death) and peritonitis was observed, extending through the uterine wall that resulted in gangrenous peritonitis. Pregnant females found infected with Salmonella early in pregnancy did not abort.

Austin Larsen of the Utah Fur Breeders Cooperative has also described an outbreak of Salmonella abortions that occurred in the Utah area. The mink rancher infected himself by placing a live kit in his mouth to "warm it up." The rancher was sent to the hospital where he recovered.

<u>Salmonella as a Cause of Enteritis</u>

There has been a good deal of discussion concerning Salmonella bacteria as a cause of enteritis. Most of

the mink that have eaten contaminated meat show no signs of disease but may be called carriers. When the Salmonella enter the intestine, instead of invading and causing inflammation of the lining membrane, they pass through the animal with the intestinal contents.



Dead unborn kits in the uterus of a female fed Salmonella-contaminated pork lungs.

Enteritis has been observed in mink in which Salmonella and coccidian were demonstrated. It was not concluded whether the Salmonella or the coccidian was the primary disease.

Investigation carried on at our laboratory several years ago showed that normal and semi-started mink were resistant to experimental rations containing Salmonella. Clinical disease was produced in only 2 of 64 mink. These 2 animals were in a group that received half rations for an extended period.

While normal appearing pork livers may harbor Salmonella, it is difficult to keep such livers out of the ration. Elimination of raw pork viscera during the gestation period is good advice.

H. Chr. Loliger reported the first outbreak of Salmonella abortions in German mink. The cause was Salmonella infantis, a species closely related to the Salmonella isolated in Wisconsin. Shortly after aborting their young, a number of female mink became ill.

With the exception of agents such as mink virus enteritis, epizootic catarrhal gastroenteritis and coccidiosis, the major causes of enteritis are unknown. It would be foolhardy to overlook Salmonella. It would be well to "leave the back door open" and state that if the conditions are appropriate, i.e., if the resistance of the mink was lowered by some factor such as a poor diet, enteritis might occur.

Treatment

Salmonella organisms are susceptible to certain sulfa drugs and antibiotics. Since the treatments may vary among outbreaks due to the varying sensitivity of different strains of Salmonella, it would be well to consult a veterinarian familiar with mink diseases regarding appropriate treatments.

John R. Gorham, DVM
College of Veterinary Medicine
Washington State University
Pullman, Washington 99164