FUR ANIMAL RESEARCH

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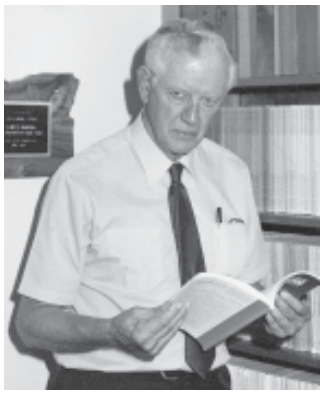


The upcoming millennium gives us an opportunity to look back over the 20th century and see just what we have accomplished. Actually, the American Fur Industry as we know it only came into existence during this past century. It has always been a dynamic and innovative group of people, and I submit that a prime example of this was the formation of a research program, under leadership of the Mink Farmers' Research Foundation. The Foundation came into being as the brainchild of two far-thinking individuals: Dr. G.R. Hartsough and Ronald Stephenson, who recognized that, like any other successful business, the Fur Industry needed continuing work in research and development. The two founders complemented one another: Hartsough was a veterinarian and research scientist; Stephenson was a progressive mink rancher. Stephenson then knew what needed to be done, and Hartsough knew how to do it. This kind of relationship continues today, with four ranchers on the MFRF Board of Directors and three support staff - two veterinarians and myself.

If you think back to those early days when so much of mink management was "by guess or by gosh" and compare it with the sophisticated ranch operations of today, you'll agree, I think, that the research program has been a successful one. All the major improvements - in breeding and selection, management, nutrition and disease control - have a basis in research findings. These have not all been made in this country but in the various facilities that house mink research worldwide. And in these days when so much attention is paid to the "bottom line" of profitability, good research brings about as high a dollar return on investment as anything you can do.

Looking ahead, we recognize that a large part of our function as your Mink Farmers' Research Foundation board of directors is not only in generating information, but in sharing it with those who can use it. This newsletter is one way that we do this and it is aimed particularly to you, as ranchers and primary fur producers. We have a responsibility, too, however, to share information with other agencies that operate in the research area. Recently, Dr. Westlake was approached and asked to speak before a joint meeting of personnel from the American Veterinary Medical Association and the U.S. Department of Agriculture. His topic, which he shared with Dr. Durrant, was the disastrous distemper outbreaks that have caused such heavy losses in the U.S. and Canada, particularly in 1998. A report of his involvement in this meeting appears later in this newsletter, and I think you'll find it encouraging.

As I put this letter together, it is the season of that essentially American holiday, Thanksgiving Day. Sometimes it seems that we are overwhelmed with problems and there's not much to be thankful about. But if you think things through carefully, I'm sure you'll find valid reasons for thanks buried among the many problems we face. In the words of an old song, I invite you to "accentuate the positive" and join me in gratitude for our blessings.

And have a Happy Holiday Season.

J. E. Oldfield

HARTSOUGH AWARD RECIPIENT

Although we grieve at the loss of our friend and longtime Secretary of the Mink Farmers' Research Foundation (MFRF), Dr. G.R. Hartsough, we rejoice that mink research is continuing in his name at Michigan State University. The fund at MSU, to which the MFRF contributed in your name, is now large enough to permit a $2,000 cash award annually. This year's winner is Chandra Sharma, who is pictured below with Dr. Dick Aulerich, leader of Michigan State's program of mink research.

Chandra Sharma is originally from Kathmandu, Nepal. She received her BS degree in Biology from Tri-Chandra College in Nepal in 1997 and a second BA degree in Environmental Science from Olivet College in Michigan in 1998. Chandra is currently working with Dr. Steve Bursian on a Master's program in the Department of Animal Science with a specialization in Environmental Toxicology. Her research is entitled, "Reproductive Toxicity of Ergot Alkaloids in Mink." She completed her research in summer and expects to graduate in Spring 2000.

A REVIEW OF EFFECTS OF PCB'S ON MINK

About fifteen years ago, serious problems occurred in Michigan, due to contamination of feeds and livestock facilities with PCB's (polychlorinated biphenyls), which came from a number of sources, including fire retardants, used electrical transformers and wood preservatives. These chemicals seeped into the top soil and from there into ground waters and ultimately the Great Lakes. Problems with mink arose when Great Lakes fish, contaminated with PCB's, were fed as part of the diet.

Dr. Aulerich, whose work we support, has been a leader in research on these toxicants, and he has summarized his research with them. He did this by feeding mink on diets containing 0 (control), 0.25, 0.5 or 1.0 ppm (parts per million) of PCB's provided in Saginaw Bay carp, which were substituted for ocean fish in the diets. He found that continuous feeding of 0.25 ppm PCB's, or more, delayed onset of estrus in breeding females and lowered the number of kits raised. Litters from females exposed to 0.50 ppm PCB's or more showed higher mortality and lower average body weight than controls. And, importantly, even short-term exposure to breeding mink to PCB's caused detrimental effects on survival of subsequent generations of mink, conceived even months after the parents had been switched to "clean" feed (from: Restum, J., S.J. Bursian, J.P. Giesy, J.A. Render, W.G. Helferich, E.B. Shipp, D.A. Verbrugge and R.J. Aulerich. 1998. Multigenerational study of the effects of consumption of PCB-contaminated carp from Saginaw Bay, Lake Huron, on mink. Effects on mink reproduction, kit growth and survival and selected biological parameters. J. Toxicol. & Environ. Health 54:343-375).

INTERAGENCY MEETING WITH AVMA AND USDA

Early in the fall, Dr. Westlake was contacted by Dr. Elizabeth Curry Calvin, of the American Veterinary Medical Association, and invited to describe the distemper problem that has affected so many mink ranches in the U.S. and Canada recently, and especially in 1998. Dr. Durrant accompanied Dr. Westlake to the meeting, which was held in October at the offices of the U.S. Department of Agriculture in Ames, Iowa. Dr. Westlake was originally allotted 15-20 minutes for his presentation, but interest in it extended the time to almost an hour and a half. He showed a videotape of an actual distemper outbreak on a Minnesota mink ranch and provided data from a survey he and Dr. Durrant had conducted on the distemper problem. It is good that major agencies like the American Veterinary Medical Association and the U.S. Department of Agriculture should learn, first-hand, about the seriousness of this problem, which lies outside their usual area of involvement.

An objective of the AVMA is to advance the science and art of veterinary medicine, including its relationship to public health, biological science, and agriculture. It provides a forum for discussion of issues of importance to the veterinary profession and for the development of official positions. The Association is the authorized voice for the profession in presenting views to government, academia, agriculture, pet owners, the media and other concerned publics. The Council on Biologic and Therapeutic Agents, to which Dr. Westlake reported, advises the Executive Board of the AVMA on the efficacy and proper use of biologic and therapeutic units in the practice of veterinary medicine.

Following the meeting, Dr. Westlake received the following letter from Dr. Steve Henry, Chairman of the Drug Advisory Committee of the AVMA:

"Dear Dr. Westlake: Thank you for the recent presentation to the Council on Biologic and Therapeutic Agents and Drug Advisory Committee in Ames, Iowa. You delivered well-documented information in a clear manner. I was pleased to receive such articulate input during our meeting. You addressed the very concerns that are under examination by the Council and Committee. Thank you for being an active AVMA member and for informing us of your concerns." (Signed) Steve Henry, DVM, Chair, Drug Advisory Committee.

Since this letter, Dr. Westlake has been informed that CVB's initial testing of the vaccine is in progress, with results from the studies due before the end of this year, which is encouraging news.

FEEDING DOGFISH

As demands for human food and for manufacture of higher-priced pet foods impact on our fish populations, the fur industry continues to seek fish species that might be more fully available for use with mink. One species that has been examined, off and on, for years, is the dogfish (Squalus acanthias) but there have been problems with its use because of high levels of urea in the flesh, which are not well tolerated by mink. Now, Kirsti Rouvinen, at the Nova Scotia Agricultural College, has provided useful information on both fresh dogfish and dogfish silage. She fed experimental groups of mink on diets containing 0 (control), 5%, 30% or 45% dogfish and 5% or 30% of dogfish silage. The dogfish replaced equal quantities of haddock-herring in the control diet. The rest of the test diets were composed of 10% beef tripe and lungs, 8% poultry offal, 10% cereal mix, 5% corn gluten meal, 0-2% vegetable oil, a vitamin-mineral premix, and water. The dogfish diets maintained growth of female mink, but caused male mink to grow significantly more slowly than the controls. Cotton fur, apparently caused by presence of trimethylamine oxide (TMAO) in the dogfish flesh, was a problem. Incidence of cotton pelts by diet groups was: Control 0/10; 15% DF, 6/20; 30% DF, 7/17; 45% DF, 0/19; 15% DFS, 2/18 and 30% DFS 3/17 pelts produced. The absence of cottons on the 45% dogfish diet was attributed to high ammonia formation interfering with the normal metabolism of TMAO. It is apparent that dogfish is not a satisfactory major feed ingredient in mink diets (from: Rouvinen, K.I., D.M. Anderson and S.R. Alward. 1998. Dogfish and dogfish silage as feedstuffs in growing, furring diets for minks. Can. J. Animal Sci. 78:189-197).

WELFARE OF MINK

Fur ranchers have always been concerned about the welfare of their animals, and now society generally is intensifying efforts to ensure that all species of domestic animals are well looked-after. Danish workers have recommended that practices in the selection of breeder mink should include attention to behavioral traits. They have been able to develop strains of mink that are quite different behaviorally. One strain is quite calm ("confident") and another is excitable ("fearful" strain). Thus, selection may be a means of producing mink that are better able to look after themselves but they suggest that any such selection must be imposed within a system that provides for adequate nutrition and disease control. (from: Malmquist, J. and P. Berg. 1998. Selection for increased welfare. **Scientifur** 23:31-36).

"PUSSY LUNG"

Accumulation of pus in the lungs of mink - called **purulent pleuritis** or more commonly "pussy lung" is a condition that has been around mink production areas for a number of years. We asked Dr. John Gorham if he would write a brief article on this condition, and he has kindly done so.

Purulent Pleuritis (Pussy Lung)

"Pussy lung" is the name mink farmers call purulent pleuritis (PP). Pathologists call it empyema or pyothorax. All of these terms mean the same thing - an accumulation of pus in the pleural space. It might be appropriate to briefly review the anatomy of a mink's chest cavity. The lungs are spongy, pink in color and are much lighter in weight than the liver. Each fills its side of the chest cavity and surrounds the heart. A thin glistening membrane - the pleura - covers the lungs and lines the inner side of the chest wall.

Only a potential space exists between the pleura covering the lungs and the pleura reflected over the inside of the chest cavity. When the pleura is inflamed, it is called pleurisy or pleuritis and fluid collects in the aforementioned space. In mink, this fluid is usually partly composed of pus and is termed a purulent exudate.

When the chest is opened, the pleural space contains a considerable quantity of thick, grayish, foul smelling yellow fluid that may be bloody. The pleural surfaces over the lungs and lining the chest cavity may be covered by stringy material called fibrin. The lungs are shrunken and solid appearing. Pneumonia (inflammation of the lung) itself is not an important part of what is called pussy lungs.

Disease Signs

On large farms, PP mink are usually found dead because disease signs of loss of appetite, rapid breathing and slight nasal discharge are frequently overlooked. Affected mink usually die within two to four days after signs first appear.

Bacteriology

Aerobic (in the presence of air) cultures of the lung and purulent fluid do not result in growth of significant bacteria. Cultures for *Mycoplasma* spp. were negative from all mink tested. Anaerobic cultures (grown in the absence of oxygen) were positive and include large numbers of a variety of different bacteria. Among these bacteria, *Fusobacterium* spp. (including *F. necrophorum*), *Bacteroides* spp., and *Prevotella* spp. were most commonly identified. These bacteria have been shown to be primary disease agents, but are often opportunistic invaders. In healthy Aleutian mink used as controls, the pleural space was found to be essentially sterile.

Route of Infection

Growth of large numbers of anaerobic and only a few aerobic bacteria is not surprising as the pleural space has no direct source of oxygen and is considered an anaerobic space. The perplexing aspect of this disease is how the bacteria gain access to the pleural space. As mentioned previously, the pleural space is considered sterile. The bacteria cultured from the PP exudate are organisms commonly found in the environment. Several hypotheses have been presented as to how the bacteria enter: (1) Eating small, sharp bones or bone fragments may cause damage and possibly perforate the esophagus as it runs through the thorax; (2) Penetrating trauma into the thoracic cavity (such as a bite wound or needle puncture inoculating bacteria from the skin); and/or (3) Abscesses from the lung burst releasing bacteria into the surrounding pleural space.

No route of entry of the bacteria was found during postmortem exami-

nations. No wounds in the chest wall or esophagus were seen and not all affected mink have lung abscesses. This does not rule out these routes of infection, however. The inoculation of bacteria may have occurred several days or weeks prior to death. The route of entry still remains a mystery, but the isolation of large number of different anaerobic bacteria is consistent with inoculation with bacteria frequently isolated from the environment of mink.

The Occurrence

Because of its sporadic widespread occurrence over a period of time and because the deaths are rarely clustered, PP mink are not usually autopsied. About 5 percent of the mink submitted for autopsy at the Ontario Veterinary College are diagnosed with PP. This percentage is about the same number of PP diagnoses made at Washington State University. Whitie Johnson of the Heger Company conducted a PP survey on farms in the midwest, Pennsylvania, Oregon, Massachusetts, and Montana. While for the most part the mink were opened by the farmer and not autopsied at a laboratory, the farmers felt that between 4 and 63 percent of their losses were caused by PP during January, February, and March 1999.

Purulent Pleuritis and the Chediak-Higashi Syndrome

All Aleutians have the Chediak-Higashi syndrome (C-HS). In the C-HS, the white blood cells can "gobble" up bacteria (A) normally, but the small bags of enzymes (B) in the white blood cells fail to function normally. The accompanying figure shows a white blood cell with the bag of enzymes in a cell (B), characteristic of the Chediak-Higashi syndrome. The enzymes kill the bacteria. But these bags containing the enzymes do not break open and empty their enzymes onto the bacteria (C). Normal granules in non-Aleutians release their enzymes. If the enzymes are not released, the white blood cells cannot destroy the bacteria. The bacteria are not destroyed, and in many instances, they multiply and produce pussy lungs and/or abscesses.

No one can explain the role of these bags of enzymes in the case of Aleutian Disease virus. Presently, all we can say is that if an Aleutian mink is infected it will invariably die.

The prospects look rather poor for finding Aleutian mink that do not have an inherited susceptibility factor. Every mink we have examined (several thousand) with the Aleutian genotype (aa) has also had the abnormalities characteristic of C-HS. Therefore, the gene for coat color and the gene for C-HS are probably either closely linked or the same gene. If they involve the same gene, we will never be able to separate the coat color and the susceptibility to disease factors. If they are separate genes, the solution to the problem will depend on locating an animal in which the chromosomes have split and crossed leaving one or the other of the characteristics behind. While this is a possibility, it will likely be a long and tedious search for an Aleutian mink without the Chediak-Higashi syndrome.

Vaccination

As in the case of treatment of PP, the development of vaccine is really not possible because no one can pinpoint the cause. We have autopsied more than 100 mink suspected of having PP and examined them for bacteria that might be the cause. If we could consistently find the same bacteria, a vaccine might be possible. Different bacteria, *Fusobacterium* spp., *bacteroides* spp., *Prevotella* spp., have been isolated from the pleural fluid on a few occasions. Veterinarians have vaccinated mink with *Fusobacterium* spp. to prevent PP but it is difficult to evaluate the results. The vaccinated mink in these field trials must be autopsied along with the unvaccinated control mink. Again, because of the low prevalence and sporadic nature, these field tests are difficult to interpret.

Treatment

It is difficult to treat any disease if the cause is not known. The respiratory signs in a single mink are frequently treated with penicillin injections or other antibiotics, but the mink invariably die. Long-term or "flushing" herd treatments with newer antibiotics and sulfa drugs are expensive and do not reduce the sporadic losses.

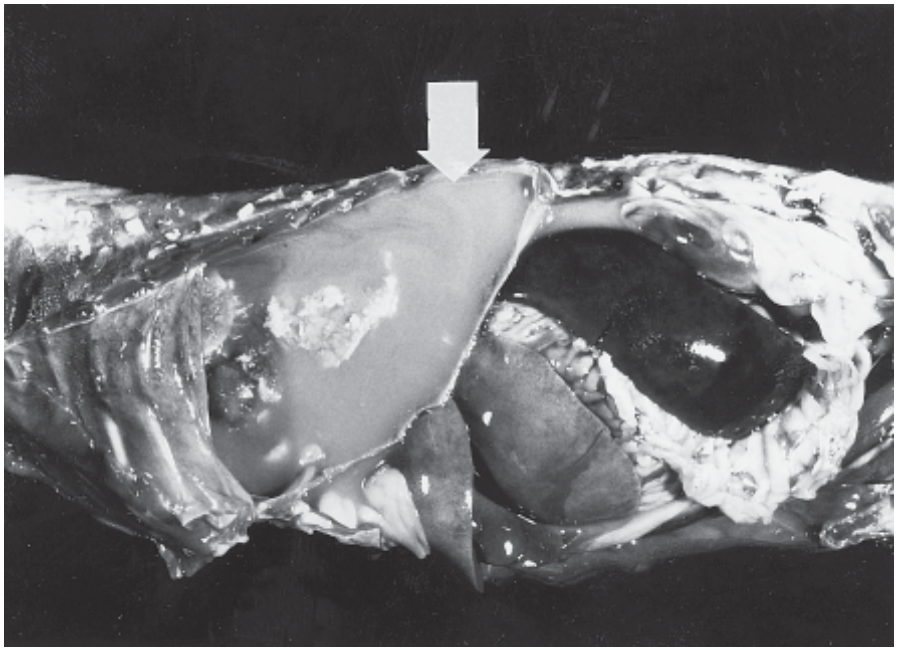
Summary

All Aleutian mink have the CH-S. Unfortunately, the beautiful hair coat of this genotype is tied to the gene or genes that make them highly susceptible to pussy lungs, abscesses, urinary tract disease, other bacterial diseases and Aleutian Disease. While Aleutian Disease can be eradicated on a farm, Aleutian mink will be subject to bacterial infections.

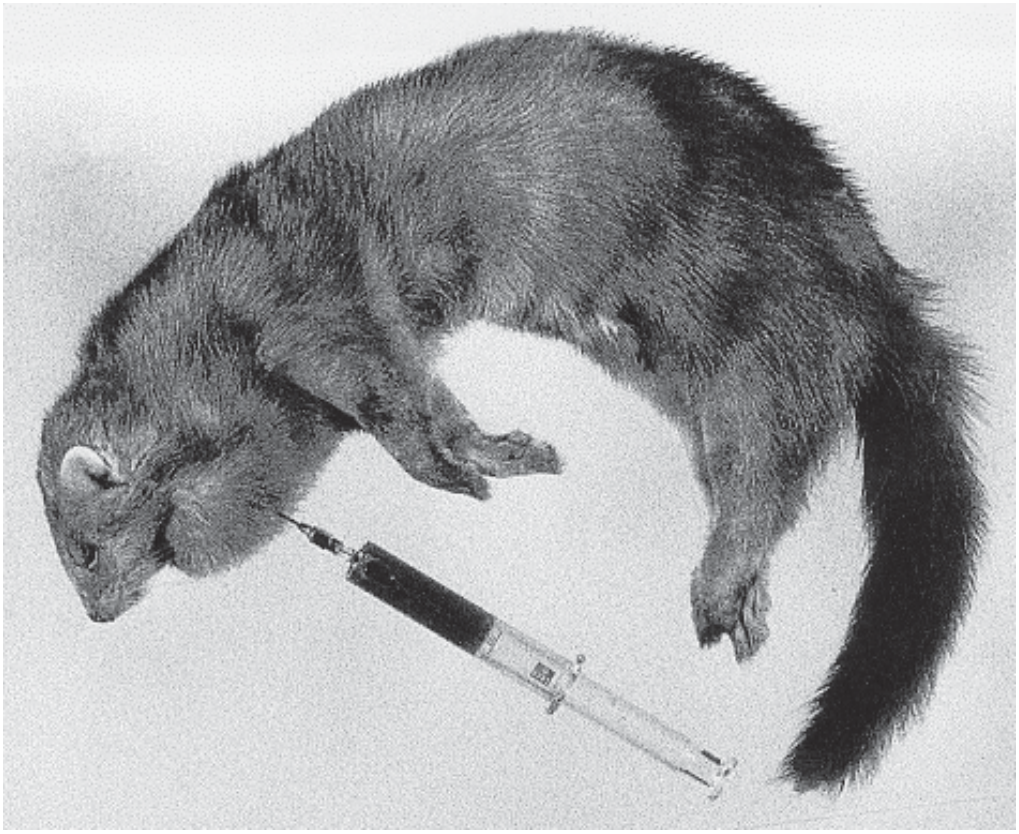
References

Momberg-Jorgensen HC. In Pelsdyreygdomme, 1952, p. 38.
Hunter DB, Lemieux. Mink, biology, health and disease. Graphic Print Services. University of Guelph, Guelph, Ontario, Canada, N1G2W1.

"PUSSY LUNG" Cont.

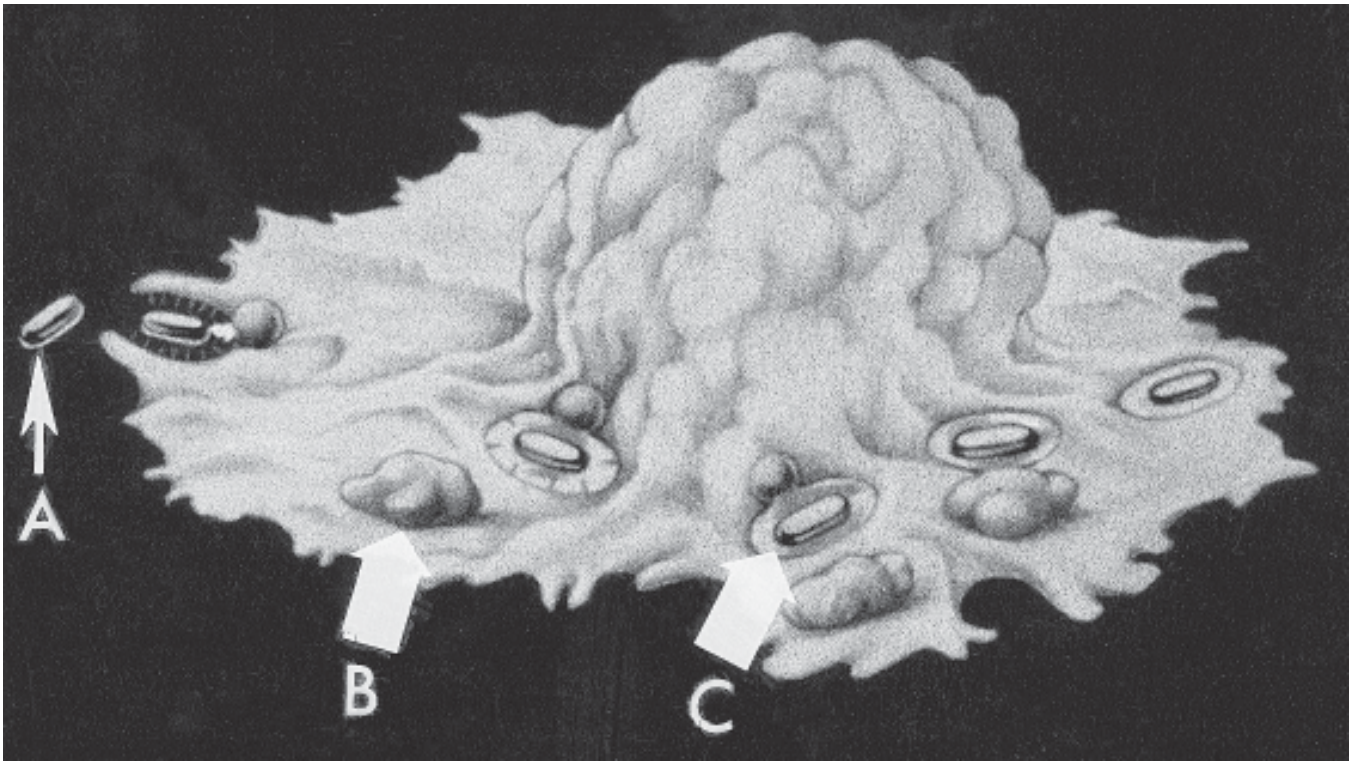


Aleutian mink carcass, showing pussy lung. The arrow points to the pleural cavity, which contains about 5 ounces of thick, white, opaque pussy fluid. Sometimes the fluid shows fibrin (stringy, white strands) and it may have a red color if blood is present.



Abscess in the neck of an Aleutian mink. Pussy fluid was withdrawn that contained staphylococci bacteria.

"PUSSY LUNG" Cont.



A white blood cell of an Aleutian mink with Chediak-Higashi syndrome. The bacteria (a) are engulfed by the mink's white blood cells in a normal manner. In order to kill the bacteria, small bags of enzymes (B) must be released into a vacuole containing the bacteria (C). In Aleutian mink, these enzymes are not released and the bacteria multiply, leading to pussy lungs and a variety of other disease. (from: Jeff Abbott, DVM; Tom Besser, DVM; John Gorham, DVM, Department of Veterinary Microbiology and Pathology, Washington State University, Pullman, Washington 99164).

MINK PARVOVIRUSES

Dr. Marshall Bloom of the Rocky Mountain Laboratory, whose work we support, has been collaborating with the Danish Veterinary Institute for Virus Research, in studies of parvoviruses. They note that the two parvoviruses of mink produce quite different disease symptoms. Mink enteritis virus (MEV) causes a rapid, high-level virus replication and acute disease. The other one - the Aleutian disease parvovirus (ADV) is associated with a persistent, low-level virus replication and a chronic, severe immune dysfunction. The ADV coding for structural proteins was present at a level at least 100 fold lower than the corresponding MEV proteins. These studies contribute to the ultimate goal of forming protective measures against these troublesome diseases. (from: Storgaard, T., M. Okelsiewicz, M. Bloom, B. Cheng and S. Alexandersen. 1997. Two parvoviruses that cause different diseases in mink have different transcription patterns: Transcription analysis of mink enteritis virus and Aleutian mink disease parvovirus in the same cell line. J. Virology :4990-4996.

INTERNATIONAL MINK CONFERENCE

The VIIth International Scientific Congress in Fur Animal Production will be held September 13-15, 2000 in Kastoria, Macedonia, Greece. Titles and abstracts for papers are due by January 30, 2000. Further information may be obtained from: SYMVOLI Conference Organizers, Ltd., Patmou 8, Kalamaria, 551 33, Thessaloniki, Greece.

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