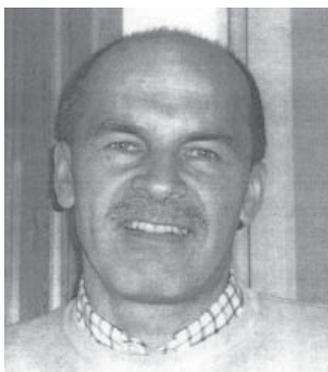




Many people who are not directly involved in it tend to think of farming as a static business that goes on in the same way, without change, year after year. This is not so, and I submit that farming has continuously changed as new technologies have become available. The fur farming industry is a good example of this. One pertinent area is that of diet formulation by means of computers. I have recently received from Ilpo Polonen of the Finnish Fur Breeders, a description of their computer program called Ration.

The first version of this program was developed about 1993 and it was designed for planning experimental diets. It has since been expanded as computers have become faster and more widely used and has been translated into English so it can be used by fur ranchers themselves. A most important part of the modern Ration program is its least-cost feature. Raw feed ingredients are ranked, based on their prices of digestible protein, metabolic energy or dry matter, and a rancher can build from this data the most economical diet to suit his particular circumstances. The use of Ration requires an IBM-compatible computer, a color screen, mouse and a printer.



Ilpo Polonen

Another thing that is happening is that the direction of mink research is changing to reflect concerns about the comfort of the caged animals and ways that it may, perhaps, be improved. I hasten to add that most mink producers have always shown a

concern for their animals' health and comfort, but adding a research approach to this is something new. As an example of this, I cite a study of "climbing cages" that has been undertaken by Finnish mink scientists.

The experiments were carried out on a private Finnish mink farm. The juvenile animals were housed from weaning to pelting either in male-female pairs in standard mink cages (cage 84x31x45 cm, nest box 30x31x40 cm, LxWxH) or in groups of two males and two females in climbing cages that consisted of a standard mink cage with a second floor (56x31x45 cm). There were three color types in the experiment: black (growing season 2003), wild (2003 and 2004) and mahogany(2003). The results are based on the data from altogether 1083 animals. The housing system had no effects on the skin length, fur quality and skin price,

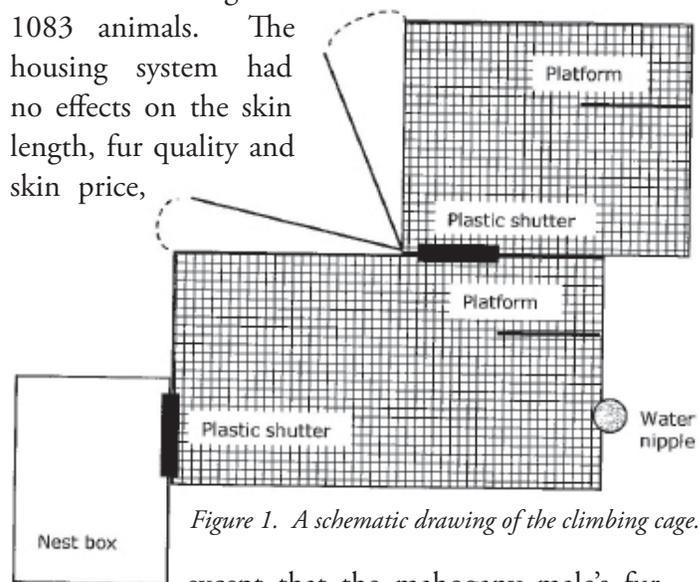


Figure 1. A schematic drawing of the climbing cage.

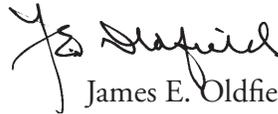
except that the mahogany male's fur quality was slightly worse in the climbing cages than in the standard cages. The mink farmer's experience of climbing cages were positive in terms of both animal care and their productivity. From farmers'

point of view the climbing cages seem to be a feasible way to produce mink. However, since in particular aggression may be a problem in group-housed mink, further studies with focus on the welfare of mink are still needed.

The fur industry is, indeed, changing with the times, as are other types of animal production. This

will lead to better, more comfortable quarters for the animals and likely to improved productivity.

I wish you a happy and pleasant summer.


James E. Oldfield

MINK VIRUS ENTERITIS: ITS FIRST OCCURRENCE

Anyone who studies infectious diseases is always interested in the first case in an outbreak. When, where and how was the first mink infected with mink virus enteritis?

Since mink virus enteritis has been observed in the field and studied in the laboratory for over 50 years, we thought it might be useful to comment on certain aspects of the malady even though we lack adequate experimentation. A virtue of a good speculation is that it can be proved wrong. At least it should create intellectual conflict among one's contemporaries.

Early History

Although there may be previous reports, our earliest source of published information are the minutes of the meeting of the Dominion Council of Canadian Fur Breeders that was held in Fort William, Ontario, July 1950. The following is part of a discussion by a rancher from that area.

In the summer of 1947, a nephew of Mr. Schoales lost 16 of 150 mink kittens. The following summer,

Schoales lost 700 of 1700 animals on his ranch. His lucid description of affected feces signaled the disease as we recognize it today. "The first thing that happens is a softness – not exactly a diarrhea; the droppings are usually grey and then turn yellow. The



E. Rendle Bowness, D.V.M.

animals will lay down a deposit more than that of a hen. It is sometimes pink and sometimes nearly white and sometimes like cheese which is the size of the intestine. It may be followed by a splash or two of mucus." While he did not mention abrupt loss of appetite, which is typical of the disease, the late Dr. Rendle Bowness observed sudden anorexia and dehydration in affected animals when he visited the area in late August of 1947. Dr. Bowness necropsied about 1,000 mink from 20 different ranches and observed lesions that are consistent with present

descriptions. Recovered animals were apparently immune to further attack.

The malady was first diagnosed in the Kitchener Waterdown area of Ontario in 1949, in Wisconsin in 1950 and in Manitoba in 1954. For an unknown reason, mink virus enteritis was confined, for a few years, to two or three ranches in North Central Wisconsin before spreading elsewhere. At the present time, the disease has been diagnosed in major mink producing areas of the world.

Origin of Mink Virus Enteritis

The malady is so easily recognized in its typical form that we postulate that the Fort William area was the site of the first outbreak. Furthermore, this outbreak can probably be pinpointed to the summer of 1947.

We are indebted to Dr. Bowness for providing a resume of his early observations. "I had been in contact with the Fort William ranchers on an annual or semiannual basis from 1938, which was nine years previous to the recognized outbreak. The cause of the losses that occurred during this time was easily identified. It is my opinion that virus enteritis did not exist in epidemic proportions prior to 1947 and its spread after that time was primarily due to carriers. Furthermore, I think that the original Fort William outbreak was much more violent than later outbreaks in other areas."

If this was the first area of disease, the central perplexing question arises: what was the source of the virus on the ranch belonging to the nephew of Mr. Schoales?

The First Case?

It is highly likely that the feline panleukopenia virus – a real killer of cats and raccoons – mutated into a new virus that we now call mink virus enteritis.

That the virus mutation occurred on Mr. Schoales' farm might not be true but it makes a good story.

There is always a good chance for exposure of the cat virus to farm raised mink. Hardly a ranch in the world does not have cats on its premises and the feline panleukopenia virus is frequently present. It is a stable virus and, unlike distemper, it can contaminate barns and feed equipment for at least a year.

For an example of the exposure of panleukopenia virus to mink, the feces of panleukopenia-infected cats could contaminate the mink nest box hay stored in a barn. Of course, there is always the possibility that infected cats could contaminate the mink food directly with their feces.

The Canadian investigators at the Ontario Veterinary College deserve a great deal of credit for first showing that Fort William Disease was caused by a new virus that they named mink virus enteritis. Furthermore, Frank Schofield and Gordon Wills related this mink enteritis to feline panleukopenia and provided the first vaccines for control of mink virus enteritis.

Summary

Most researchers feel that mink virus enteritis is so characteristic in its present form that the malady as reported in 1947 in Fort William, Ontario was actually the first outbreak of a new mink disease. The actual source of this virus that has now spread throughout the mink raising world will probably never be known.

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MAXIMIZING MINK KIT SURVIVAL

Maximizing kit survival is an important target for successful mink farming. Some farmers keep accurate records of the number of kits whelped but many rely on 10 day counts or weaning counts. Weaning numbers correlate better with the number of marketable mink but fail to consider the lost potential associated with early kit losses. Records maintained on individual mink cage cards are a good first step, but mink farmers are encouraged to keep a daily and weekly summary of mortality data by shed and by farm so they can recognize as early as possible if kit mortality rates are too high. This information is important so that treatment and prevention measures can be implemented as quickly as possible.

Normal mortality rates between birth and weaning approach 20-25% of all kits whelped if all losses are considered including aborted, stillborn and weak-born kits. Approximately half (50%) of these dead kits are stillborn, i.e. dead at birth and have not breathed air (if they have not breathed their lungs will sink when placed in water). Another 35% of this early mortality is due to a failure to nurse for a variety of reasons (these kits can be identified because there is no milk curds in the stomach at necropsy). Most losses occur during the first 72 hours post whelping, so if you are only doing 10 day counts, you will have missed the early mortality in our statistics.

A normal healthy newborn kit should weigh 11-12 grams or sometimes even more. Kits with a birth weight of under 8 grams do not survive. These kits are either late-term deaths in utero or they are born too small or too weak to nurse or compete with larger siblings. Undersized kits examined at

necropsy often do not have pathological lesions and a specific cause of death may not be established. Most of these are simply physically and physiologically underdeveloped kits. Whether this relates to an asynchronous implantation or inadequate placental nutrition is unknown. In cats, dogs and humans there is a direct correlation between birth weight and postnatal survival. For example, small and premature human infants have great difficulty breathing due to underdeveloped lungs. In other multiparous species like pigs, mice and rats it has been shown that there are preferential sites for optimal growth in the uterus ... presumably areas with the best nutrient blood supply.

Some of the most common reasons for early kit losses include dystocia (difficulty in birthing due to mal-positioning in the uterus or the kit being too large, see Figure 1), systemic infections (usually *E. coli*, *Beta Streptococcus* spp., *Staphylococcus aureus*, *Pasteurella multocida* or mixtures of the above), external trauma associated with dystocia and the mother physically pulling the kit (see Figure 2) or mothers lying on kits and suffocating them,

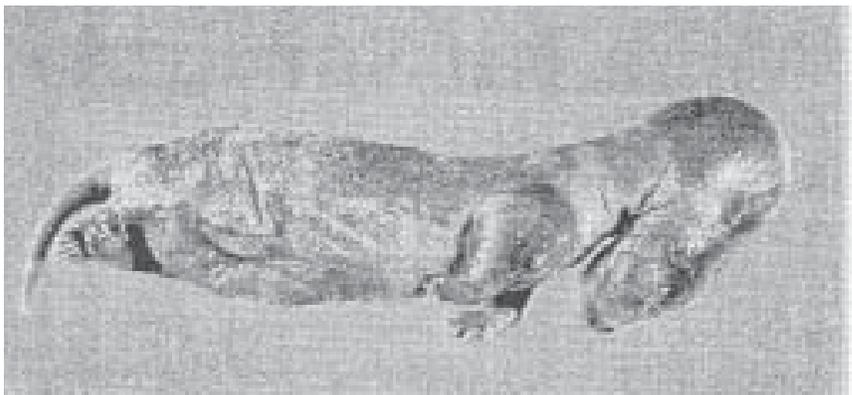


Figure 1. Kit that has died of dystocia due to being malpositioned in the uterus. Note the bent head and the swelling of the neck. This kit was jammed or stuck in the birth canal. If it was the first kit in the birthing sequence the siblings may have died in utero as they could not get out.

anasarca (swollen or edematous kits) and congenital anomalies (quite a wide variety have been reported). Occasionally, bacterial infections acquired at birth may cause infections of the neonatal skin gland over the neck resulting in pimply kits.

Remember that mink kits are highly susceptible to temperature fluctuations because their thermoregulatory mechanisms are not fully functional until they are 25-35 days old. Adequate bedding and most importantly, protection from drafts, is critical during this early period and so your nest box management (i.e. proper bedding, deciding if nest box covers should be left on or off, etc.) has to be tied to external weather conditions and mink shed design.

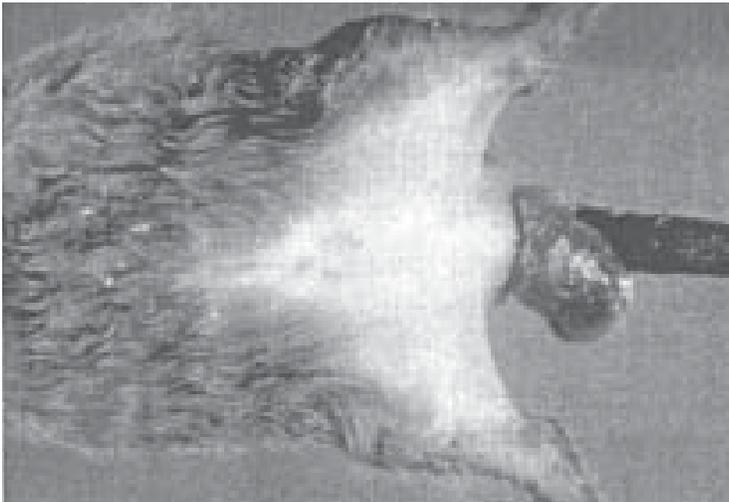
It is important that you recognize as soon as possible when a kit mortality problem is occurring on your farm. This can only be accomplished if litters are checked regularly. Most mink farmers check each litter on the day of whelp (either clued in by black tarry droppings under cages or kit crying sounds in the nest box). Some farmers check again on day 2 postwhelp and then often do not check again until day 10. The assumption is that it is best to disturb the litter as few times as possible. This is probably true, however every year there are disasters where kits were checked at day one and then not again until day 10, and at the day 10 check many nests are empty. The kits have died within the first few days and the females have removed the evidence. These early losses may be due to diseases like pimply kits or weak kits that were born alive but infected in utero with *Campylobacter* or some other bacteria or with females that for whatever reason (e.g. coming into whelp too thin or having inadequate nutrition post whelp) are not milking well. Checking nest boxes more regularly would allow the farmer to spot some

of these problems and initiate treatment.

After the kits have reached 4 days of age their survival rate is much improved. Most of the kits with early problems have died and been removed. Bacterial diseases often associated with nest box sanitation become more important. Common environmental contaminants like *E. coli*, *Streptococcus* and *Staphylococcus* bacteria may cause septicemia or pneumonia. Adequate milk supply from the female is obviously critical for kit survival. Energy and fluid demand from the kits exacts a huge toll on the mother and this is obviously correlated with litter size. Adequate amounts of good quality water should always be available for the female and her water consumption will increase greatly in warm/hot weather. Water consumption will decrease if the water is stagnant or hot. Inadequate intake of milk will result in short term dehydration and long-term starvation of kits. If litters are not thriving and growing well, the female should always be carefully examined to determine if she has the correct number of nipples, is producing milk (the mammary tissue is well developed), that the nipples are patent (you can assess this by gently squeezing the nipple to obtain a drop of milk) and that she does not have mastitis or sore nipples. Simple things like rough nest box openings can traumatize the mammary tissue as the female jumps in and out of the nest box. Sore mammary glands will make the female reluctant to allow babies to nurse. Low grade or sub-clinical mastitis (i.e. there is a mild infection in the gland but not enough to make the female go off feed or look ill) is under-diagnosed and a common cause of poor-doing kits. Females with low grade mastitis will often have small, black tips on each nipple due to dirt collecting on the exudates and babies not feeding

from that nipple. Mastitis in a few individual mink occurs on every farm, but if numerous females are affected, the milk should be cultured and antibiotic sensitivity patterns determined. Herd treatment may be necessary.

Kits should be regularly monitored throughout the pre-weaning period. Conditions like flea infestations and ringworm will occur in pre-weaned mink kits. Sticky kits can occur in both the pre- and post-weaning periods. There are many causes for sticky kits. Affected mink kits generally have a fever and often have diarrhea. Nest boxes become contaminated and bedding must be changed. *Campylobacter* is a common bacterial cause of kit diarrhea during the early weaning period as kits get exposed for the first



*Sticky kit. Note the wet, sticky hair coat and the prolapsed intestine due to straining associated with severe diarrhea. This mink kit has an enteric *Campylobacter* infection.*

time to bacteria present in the solid feed. Bacterial enteritis is more common if feed is fed on top of nest boxes as it is more difficult to maintain good nest box sanitation with this management system. There are

a number of viral causes of kit diarrhea including enteric calicivirus (the same virus causing 3 day enteritis in adult mink), corona virus, astroviruses and likely others that have not yet been determined.

A farm with sticky kits should work with their veterinarian and the diagnostic lab to determine the cause. Feed should be carefully monitored for bacterial quality (bacterial counts) and particular care should be taken if the weather is hot and feed spoilage is more rapid. Feed supplies should be checked for ingredients. Chicken offal is commonly incriminated with *Campylobacter* infections. Nest box bedding should be kept dry and as clean as possible and changed frequently in nest boxes containing sticky kits.

Dr. Hugh Hildebrandt spoke at the 2007 CMBA annual meeting about the importance of regular monitoring to determine the causes of kit losses in order to establish patterns of disease on your farm. Once you know what normally happens on your farm it becomes much easier for you to recognize abnormal patterns of disease and react more quickly to the problem. Farmers are encouraged to establish regular farm visits with their veterinarians to help set up this type of program.

So, in summary, there are many disease conditions that affect kits in the period from whelping to weaning. Good management practices including early disease detection is critical to maximize kit survival during this important time on the farm.

*Dr. Bruce Hunter, Department of Pathobiology
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THE EARLY HISTORY OF EPIZOOTIC CATARRHAL GASTROENTERITIS (ECG)

When a new enteritis appeared in the 1970s, no one knew the cause and it was diagnosed as food poisoning or the “waste basket” diagnosis of non-specific enteritis. Dr. Austin Larsen, my roommate when we were in Veterinary College, was the first to describe the disease on a Utah farm. It soon became known worldwide as “Utah enteritis” or “three day disease.”

We saw the same disease in the Pacific Northwest within a few months after Dr. Larsen called me about the first outbreak. We reported the new enteritis in 1975 and named it epizootic catarrhal gastroenteritis or simply ECG. This technical name replaced the unpopular designation “Utah enteritis.”



Dr. Austin Larsen

Several million mink have been affected since ECG was first reported in 1975. In addition to outbreaks in the U.S., similar outbreaks have occurred in Canada, Scandinavia, Russia, France and China.

Symptoms

Five to eight days after exposure, both male and female mink partially or completely go off feed for one to five days. Some mink show signs for as long as two to three weeks. At least 50 percent of the mink with depressed appetites have a diarrhea characterized by green, yellow or occasionally pink tinged mucus on the surface of the feces.

Blood or intestinal casts in the feces that are seen in mink virus enteritis are rarely observed. Affected

mink show no other signs. Although many mink are affected, few die. However, fatalities may occur when mink are affected concurrently with ECG and Aleutian disease.

Those mink affected with Aleutian disease lack an effective immune system. According to farmers, female mink with nursing sickness and ECG have a higher death rate. Abortions and reduction in litter size have not been associated with the disease but if the outbreak occurs when the females are nursing, the kit losses may be severe due to starvation.

Autopsy Findings

The stomachs of affected animals seldom contain food. The linings of the stomach and intestines are reddened and covered by sticky, thick, clear-to-white mucus.

The abdominal lymph nodes are moderately enlarged. In mink virus enteritis, the intestines usually are markedly reddened, often ballooned and hemorrhagic.

Cause and Diagnosis

Further studies are necessary to determine definitely the cause but we feel that coronaviruses play a role in the U.S. outbreaks of ECG. Workers in China and at the North Dakota State Diagnostic Laboratory also have demonstrated coronaviruses in cases of ECG. However, other viruses such as parvoviruses, calciviruses or hemolytic E. coli or other pathogenic bacteria could add to the severity of the disease. Danish investigators have detected a rotavirus in the feces of ECG mink.

Dr. Bruce Hunter of the Ontario Veterinary College has isolated calciviruses in Canadian ECG outbreaks.

Green slime disease of ferrets is an enteritis caused by coronaviruses. The disease is characterized by green mucus in the feces. While most ferrets recover, this condition is a concern to ferret owners.

There are laboratory tests to diagnose MVE. At the present time, mink coronaviruses can be detected only by demonstration of virus particles by electron microscopy.

Confusion with Mink Virus Enteritis

Because the signs of ECG, loss of appetite and mucus in the feces, are similar to that of mink virus enteritis (MVE), it can easily be confused with MVE, particularly in mink that have not been vaccinated against MVE.

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REVIVAL OF APPARENTLY LIFELESS MINK KITS AT BIRTH

It is common practice to collect cold and apparently lifeless mink kits for revival in an incubator. The literature does not report any results concerning survival to day 42 of mink kits revived in an incubator and whether or not this practice is economically feasible. The present investigation was carried out in order to shed some light on the matter.

The results show that about 10% of the females giving birth also have kits which end up in the incubator. A total of 73% of the kits placed in the incubator were revived. Of these almost half (42.7%) were still alive on day 42 after birth. The survival of revived kits depended on whether they came from a female which had delivered one or more kits to the incubator. There was no difference in revival between kits coming from a female that delivered one or more kits (72.7% and 72.5%, respectively). However, the chance of survival to day 42 was much higher for the kits coming from a female that only delivered one kit to the incubator (72.5%) in comparison to kits coming from a female that had delivered more kits (35.1%).

In spite of the relatively high mortality amongst kits revived in an incubator, it is possible to conclude that the work effort is economically feasible. (Hvam, K. 2007. Survival of mink kits stimulated in incubator. Annual Report 2006, 155-158, Danish Fur Breeders Research Center, Holstebro, Denmark.)

ENTERITIS IN MINK KITS PRIOR TO WEANING

Some mink farmers have had a yearly problem with “sticky kits.” These are kits that have diarrhea and have not been cleaned by the female. Along with inflammation of the intestinal mucosa, the intestine is filled with fluid.

E. coli is considered a normal bacterial isolate after the kits begin eating solid food at 3-4 weeks of age. However, *E. coli* has been considered a pathogen in cases of diarrhea, especially when isolated from the lungs, spleen and liver, indicating a septicemia.

Several isolates of *E. coli* from sticky kits were tested and none of the strains revealed virulence factors in healthy or diseased kits. Both ourselves and Danish investigators feel that there may be virulence factors for which there are no currently available tests.

We feel that another approach is needed in the study of “sticky kits.” The electron microscopic (EM) examination of feces collected from kits with diarrhea may provide leads to the cause and control. Since some enteric viruses cannot be propagated in cell culture, electron microscopy must be used.

We as well as Dr. Hunter at the University of Guelph in Canada have isolated a calicivirus from three-week-old kits with diarrhea. Our identification of coronaviruses from outbreaks of epizootic catarrhal gastroenteritis was made by EM examination of the feces of post weaning kits with diarrhea. Dr. Lena Englund at the National Veterinary Institute in Stockholm, Sweden has made an extensive study of “sticky” kits or “greasy” kits to determine whether enteric virus infections may be the cause of pre-weaning diarrhea.

Tissue samples from the intestines of 180 sacrificed mink were examined and checked histologically.

Fecal contents were examined by EM. Astrovirus was detected in abundance and thought to be of significance on farms that had sticky kits. Other factors, i.e. low body weight and the presence of calcivirus, were shown to increase the risk of pre-weaning diarrhea. However, the astrovirus was considered the primary pathogen.

The 2007 trials to determine the microbiologic flora of normal Chediak Higashi (CH-S) (Blue Iris) and Standard Dark Mink Genotypes were conducted. We do not know the significance of the *Mycoplasma mustelae* isolation. We will attempt to isolate this agent from future autopsies and to determine its role in mink diseases. No pathogenic aerobes or anaerobes were found.

The most interesting finding was the identification of coronaviruses from both standard dark and CH-S mink. These isolations were from a farm that did not have a history of enteritis. Since we and others have found coronaviruses in high numbers from the intestines of mink in large outbreaks of Epizootic Catarrhal Gastroenteritis (ECG), we must assume a carrier state occurs. It may be possible that mink must be immunosuppressed or that there are other factors that can precipitate coronavirus ECG outbreaks.

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