Disease Concerns for the Deer Industry across North America

While conducting the research for import protocols it became very clear that there are several disease concerns that vary from jurisdiction to jurisdiction. Alberta is fortunate to be free from most; however freedom (in several cases) must be proven.

It is the AWMDA intention to work with governments to address the concerns on behalf of the industry and ensure that we have adequate programs available for producers wishing to export to outside markets.

It is up to producers to inform themselves about the diseases and ensure that they participate in the uptake of programs available. Without the proper surveillance and/or testing markets will continue to become increasingly difficult to access.

AWMDA remains committed to continue the lobby for access to markets within our own province, but the resistance from the anti groups and urban MLA's continues to hinder our progress.

Below are fact sheets, gathered from various sources to help our members understand the diseases, where the concerns are geographically situated, and what measures or mitigation may be possible.

The Diseases of concerns are:

Bluetongue/Epizootic Hemorrhagic Disease

Chronic Wasting Disease

Bovine Brucellosis

Bovine Tuberculosis

Parelaphostrongylus Tenuis (Meningeal Worm)

Paratuberculosis (Johne's disease)

Website Reference:
http://www.inspection.gc.ca/english/anima/heasan/disemala/tuber/tbwtbse.shtml

Bovine Tuberculosis In Wild Animals

What is bovine tuberculosis?

Tuberculosis is a contagious disease caused by an infection in the lymph nodes which spreads to other organs like the lungs. This disease affects practically all mammals, and before control measures were adopted, was one of the major diseases of man and domestic animals. Bovine Tb is one of the most infectious forms of Tb and is reportable under the Health of Animals Act.

Another form of Tb called Avian Tb has also been known to affect cattle, pigs, and a few other animals, but is usually restricted to birds.

What are the signs of disease?

Sick animals may show general symptoms such as weakness, loss of appetite, weight loss, and a fluctuating fever.

Suspicious lesions might be apparent when an animal is being field-dressed. Tb lesions may be found in any organ or body cavity of diseased animals. In the early stages of the disease, lesions are difficult to find. In later stages, nodules or lumps may be found in the lungs and associated lymph nodes as well as in the lymph nodes of the head and intestinal tract.

What species are susceptible to Bovine Tb?

Bovine Tb can be transmitted from cattle to other farm animals, bison, and all of the deer species under certain conditions. Cattle are the usual host for this bacteria, but transmission can occur between wild deer.

How is Bovine Tb transmitted?

The most common means is by airborne transmission. Infected animals exhale bacteria through breathing, coughing and sneezing. Animals are more likely to infect each other when they share a common watering and feeding place.

Is Tb common in wild animals?

Bovine tuberculosis is not a naturally occurring disease in wild animals. It is thought that it was introduced into wildlife populations through contact with domestic animals. Although it is known that wild herds can become infected with Tb, results from specific

hunter surveys in Manitoba and Alberta, and the capture and testing of wild animals for domestic and zoological purposes indicate that the disease is still not common in wild animals in Canada.

What does CFIA do to control Tb in wild animal population?

Responsibility for controlling disease rests with the agency that has legal jurisdiction over wildlife species. Animals within national parks, are a Parks Canada responsibility. Animals that are free roaming or on provincial crown land, are a provincial responsibility. Although the CFIA does not have a program specifically designed to control disease in wildlife populations it can provide information, advice, scientific and laboratory support to agencies which have jurisdiction over wild populations.

What are the alternatives\methods for controlling Tb in a wild animal population?

Realistically, there are limitations to what can be done. Fences have been used in some national parks to reduce the possibility of wildlife leaving the park. Farmers or ranchers can fence off hay bales\stacks to prevent wildlife from having access to hay, and to prevent contact with domestic livestock. Specific, targeted depopulation may also be considered.

Buffer zones have been tried to control disease outbreaks but have not found to be effective.

Can humans contract Bovine Tb from eating the meat of an infected animal?

It is unlikely that a person field-dressing or eating meat of an animal infected with bovine Tb could become infected. Hunters should practice sanitary precautions, including washing after field-dressing an animal. Hunters that suspect their kill of being infected should contact their provincial or federal agencies.

What does CFIA do when domestic cattle have been exposed to wild animals with the disease?

As a precautionary measure, cattle, bison, elk and deer are tested for Tb. If livestock has a positive reaction to the test, they are ordered destroyed and tissues are collected for laboratory examination.

Why are domestic animals infected with, or exposed to, Bovine Tb destroyed?

Canada follows a strict testing and eradication program in domestic animals. Because tuberculosis may have a long incubation period and does not always show up in tests, regulations require that all infected animals as well as all exposed susceptible animals be destroyed.

Are the farmer or producer compensated for animals that are destroyed?

Compensation may be awarded for farmed animals ordered destroyed under the provisions of the Health of Animals Act. Compensation is also awarded for disposal, transportation costs and kill fees charged by the abattoir.

Website Reference:
http://www.inspection.gc.ca/english/anima/heasan/disemala/blufie/blufiefse.shtml

Bluetongue

What is bluetongue?

Bluetongue is a viral disease of domestic and wild ruminants that can be transmitted by insects, particularly biting midges of the *Culicoides* species. The range of animals that can be infected with bluetongue virus (BTV) includes most ruminants, but the severity of disease varies among different species. Sheep are one of the most severely affected species, with symptoms that may include fever, erosive lesions in the mouth and gastrointestinal tract, lameness, abortion, severe weight loss, and pneumonia. Cattle, although more frequently infected than sheep, generally have an inapparent infection or milder form of the disease. In non-domestic ruminants, the disease can vary from an acute, highly fatal hemorrhagic disease, as observed in white-tailed deer, to an inapparent disease as seen in the North American elk. Other animals that can be affected by BTV include goats, buffalo, antelope, and camels.

Do we have bluetongue in Canada?

Canada is currently free of bluetongue. Historically, the Okanagan Valley of British Columbia (B.C.) is the only area of Canada where occasional incursions of bluetongue have occurred. The last clinical cases reported in the Okanagan Valley were in 1999. The Okanagan Valley has been defined as a separate zone within Canada so that the rest of Canada can maintain bluetongue-free status according to international standards. BTV has a very wide global distribution and is found in regions where the insect vector (i.e. biting midges) is present. Bluetongue is commonly found in many countries, including the United States, Mexico, Africa, the Middle East, the Indian Subcontinent, and China.

Is there a human health risk associated with bluetongue?

There is no public health risk associated with bluetongue.

How is bluetongue transmitted?

Bluetongue is transmitted between animals by insect vectors, particularly biting midges. Cattle are considered to be the main amplifying host for BTV because the insect vectors

feed more abundantly on cattle and the disease may not be observed in cattle until one or two months after the virus is introduced into an area. Virus transmission can occur at any time during the year but is more active during rainy periods. The virus survives in locations where the climate allows biting midges to survive over winter. The bluetongue virus does not survive in the environment outside an insect vector or animal host. Therefore, contact with animal carcasses and products such as meat and wool is not a method of spread.

What are the clinical signs of this disease?

Signs of clinical disease vary among different species. Inapparent infection (lack of clinical signs) occurs in cattle and other species. The acute form of the disease that usually occurs in sheep and some species of deer is characterised by fever, widespread hemorrhages of the oral and nasal tissue, excessive salivation, and nasal discharge. The lips, tongue, and lower jaw become swollen and lameness may occur due to inflammation of the coronary band (above the hoof). The animals may also become emaciated due to reduced feed intake caused by sore mouths. The "blue" tongue that gives the disease its name occurs only in a small number of cases and is a result of cyanosis (discolouration due to the presence of oxygen-deficient blood).

How is bluetongue diagnosed?

Tentative diagnosis of bluetongue in sheep can be made based on the appearance of clinical signs and lesions. The prevalence of insect vectors is also taken into account. Laboratory tests are required to confirm the presence of BTV.

Are there any treatments for this disease?

There is no effective treatment for bluetongue.

What is being done to control and prevent the spread of bluetongue in Canada?

Bluetongue is a **reportable disease** under the *Health of Animals Act*. This means that all suspected cases must be reported to the Canadian Food Inspection Agency (CFIA). All reported suspect cases are immediately investigated by inspectors from the Agency.

Bluetongue has occasionally been found in the Okanagan Valley in B.C. As a result, the Okanagan Valley has been defined as separate zone within Canada so that the rest of Canada can maintain bluetongue-free status according to international standards. All animals that are susceptible to BTV infection (i.e. cattle, sheep and other wild and domestic ruminants) require permanent identification before they leave the Okanagan Valley zone. The BTV status of the Okanagan Valley zone is determined annually through a sentinel monitoring program, administered by the CFIA. If BTV is detected through the sentinel testing or from any other source, movement restrictions will be applied for the remainder of that insect season to contain the outbreak and minimise trade impact.

Detection of bluetongue virus outside the Okanagan would lead to a re-evaluation of the zoning principles used to define infected and disease-free areas and the following actions may be taken:

- quarantine and movement controls of animals susceptible to BTV infection;
- surveillance and tracing of all potentially infected animals;
- re-evaluation of the BTV geographic zone; and
- institution of insect control measures during the biting fly season.

Website Reference:
http://www.inspection.gc.ca/english/anima/heasan/disemala/cwdmdc/cwdmdcfse.shtml

Chronic Wasting Disease (CWD) of Deer and Elk

What is it?

Chronic wasting disease (CWD) is a progressive, fatal disease of the nervous system of cervids such as mule deer, white-tailed deer and elk. Black tail deer and moose have also become infected naturally. It is known as a transmissible spongiform encephalopathy (TSE). Other TSEs include scrapie in sheep, bovine spongiform encephalopathy (BSE) in cattle, and Creutzfeldt-Jakob disease in humans. Although the exact cause of CWD is unknown, it is associated with the presence of an abnormal protein called a prion. There is no treatment or vaccine currently available for the disease.

How is it transmitted?

It is not certain how CWD is transmitted but based on experience with the disease in captive deer and elk in Colorado and Wyoming facilities, both lateral (animal to animal) and environmental (animal to premises to animal) occur. The most likely means of transmission is between animals that are in close contact with each other. In addition, the elk and mule deer placed in paddocks that had housed infected cervids for many years, became infected, even though there were no other cervids on the premises, leading to the assumption that the environment of a facility could transmit the disease on premises with multiple confirmed cases of CWD. There were a few cases of CWD in offspring of dams which developed the disease but it was not possible to rule out lateral transmission.

Is there a risk to human health?

There is currently no scientific evidence that CWD affects humans, but we must exercise caution since there is evidence to suggest that BSE can affect humans, which is another TSE also known as mad cow disease.

What are the signs?

Animals with CWD may show a number of different signs as the disease slowly damages their brain. They may include lack of coordination, separation from the other animals in the herd, excess salivation, depression, unusual behavior, paralysis, weight loss, difficulty swallowing, increased thirst and urination and pneumonia.

Signs usually last for weeks to months before the animal dies; however, some animals may not show clinical signs except for an acute pneumonia. Animals are usually three- to four-years-old before clinical signs appear, but clinical animals have been seen as young as 18 months or as old as 13 years.

How is it diagnosed?

The disease is tentatively diagnosed based on clinical signs, but can only be confirmed by laboratory examination of tissue from the affected animal after it is dead. There is currently no laboratory test available to test for the absence of the disease in live animals.

Where is CWD found?

Chronic wasting disease was first seen in Colorado deer belonging to several research facilities in 1967, and later was diagnosed in captive and wild deer and elk in northern Colorado and southern Wyoming. In the 1970s, CWD occurred at a zoo in Canada in mule deer that had been imported from a zoological park in Colorado. Fortunately, the disease did not spread. Since 1996, CWD has been diagnosed in ranched elk as well as farmed wild deer in both Saskatchewan and Alberta. CWD has also been found in deer and elk in the USA.

What does the CFIA do to prevent CWD from entering or becoming established in Canada?

The importation of elk and other cervids from the United States was prohibited from 1990 to 1999. Elk may now be imported under a CFIA import permit which contains specific measures designed to prevent the introduction of diseases including CWD.

Provincial surveillance programs have been implemented by the governments of Saskatchewan and Alberta. In the other provinces deer and elk sent to provincial laboratories for post mortem are screened for CWD as part of an ongoing surveillance program. In 1990 it became mandatory for a permit to be issued in order to move an animal, thus allowing for the monitoring of all movement of these species.

A CWD eradication policy was implemented in October 2000 and CWD was made a reportable disease under the *Health of Animals Act* and *Regulations* in April 2001. As part of the eradication program to control the spread of CWD, the CFIA has humanely destroyed about 9,000 ranched elk and white-tailed deer. There are approximately 810 elk farms and 60,000 farmed elk in Alberta and Saskatchewan.

The owners of any elk ordered destroyed receive compensation under the *Health of Animals Act*. Disease control measures have been taken by the CFIA working with the elk industry nationally and provincially. Actions taken by the CFIA are supported by provincial governments and the elk industry.

Are products like venison or powdered antler velvet from animals that have been exposed to CWD safe for human consumption?

According to Health Canada there are no studies available on the safety of tissues from deer and elk with CWD. In studies using mice experimentally infected with scrapie, another TSE, muscle and skin tissues were not found to be infectious at any detectable level. Similarly it is recognized that BSE is not transmitted by meat and skin.

However, we have to be very cautious in using these results to predict the safety of products from infected or exposed elk, since test results from one species do not necessarily apply to another. Velvet and other products or by-products from elk or deer known to be infected with CWD are not allowed to enter the human or animal food chain. Velvet is used as a medicinal alternative.

Website Reference:
http://www.spc.int/rahs/Manual/BOVINE/BRUCELLOSISE.HTM

B103 - BRUCELLOSIS (BOVINE)

Nature of the disease

Bovine brucellosis is a highly contagious bacterial disease, almost exclusively caused by *Brucella abortus* causing late term-abortion and infertility in cattle. The disease is also a serious zoonosis, causing undulant fever in humans.

Classification

OIE List B disease

Susceptible species

Main natural hosts are cattle, horses and humans. Infection has been reported in a range of other species but these are not considered important in the maintenance of the disease. Infection with abortion occurs rarely in pigs, sheep and goats.

Distribution

Until recently, bovine brucellosis was widely distributed throughout the world. A number of countries, including several in Europe and Scandinavia, Australia, New Zealand, Canada, Israel and Japan have succeeded in eradicating the disease.

Clinical signs

The dominant feature of the disease in cattle is abortion.

Cattle

- Usually occurs at about 5-7 months. Full-term calves may die soon after birth
- Abortion rates in herds vary. In fully susceptible herds rates may vary from 30% to 80%, although in some cases, abortions may be more insidious
- Retained placenta and secondary metritis is common and may lead to permanent sterility
- In bulls acute or chronic infections of the reproductive tract may occur (orchitis, epididymitis, seminal vesiculitis · hygromas, particularly of the carpal joints, occur in some animals in chronically affected herds

Horses

- Chronic bursal enlargements of the neck and withers with chronic draining sinuses
- 'Poll evil' localisation of infection between the nuchal ligament, atlas and axis
- 'Fistulous withers' inflammation of bursa between the nuchal ligament and dorsal spines of thoracic vertebrae

Humans

- 'Undulant fever' (fluctuating, irregular fever)
- Chills, depression, weakness
- Headache, joint pains, generalised aches
- May be complications affecting cardiovascular or central nervous systems

Post-mortem findings

In cattle there is considerable variability in uterine lesions after abortion

- Mild to severe endometritis

- Placenta is thickened, oedematous, yellow-grey and may have exudate on surface
- Mammary gland lesions
- Inflamed regional lymph nodes

While some aborted foetuses will appear normal, others may show:

- Varying degrees of sub-cutaneous oedema
- Blood stained fluid in body cavities
- Enlarged orange-brown discoloured liver
- Fibrous pleuritis and focal pneumonia

Differential diagnosis

Other cause of abortion and reproductive failure in cattle include:

- Trichomoniasis
- Vibriosis
- Leptospirosis
- Listeriosis
- Infectious bovine rhinotracheitis
- Mycoses

Brucellosis should always be suspected when there are multiple late-term abortions in a herd.

Specimens required for diagnosis

Live animals

- Milk samples from each quarter and vaginal swabs from affected cows
- Blood samples (for serum) from a number of cows in the herd, together with a pooled milk sample

At post-mortem

- Samples of lymph nodes, spleen, mammary gland and uterine tissues from cows
- Spleen, lung and stomach contents from aborted foetuses, and cotyledons from foetal membranes

Transmission

Bovine brucellosis is almost always transmitted from herd to herd through the movement of infected cattle. Cows shed large numbers of organisms when they abort. Bacteria are

also excreted intermittently in milk throughout the lactation.

Animals become infected through ingestion of contaminated feed or water, or after licking an infected placenta, foetus or genitalia of another cow, after it has aborted.

Most cows remain chronically infected. Urine, faeces and hygroma fluids are also sources of bacteria. Infected bulls may excrete the organism in their semen. Congenital transmission may occur through in utero infection.

Humans are infected through handling infected cows or their tissues, or through drinking infected milk. Pasteurisation will prevent the latter.

Risk of introduction

Importation of infected animals is the highest risk for introducing bovine brucellosis. The disease could also be introduced through semen from infected bulls.

Imports should be restricted to brucellosis-free countries, or if genetic stock from infected countries is desired, from certified free herds. In the later case, strict quarantine procedures including testing should be applied (see OIE International Animal Health Code).

Control / vaccines

Infected cattle can be treated with tetracyclines. Treatment is not used as part of control schemes.

Live attenuated vaccines are available which provide good protection. However, vaccination can produce persistent antibodies, which interfere with subsequent diagnostic tests.

A number of countries have successfully undertaken control programs that have led to eradication. The programs use combination of vaccination, test-and-slaughter, surveillance and abattoir traceback. Experience has shown that vaccination should be maintained well into the advanced stages of the program to protect clean herds from serious breakdowns.

Website Reference:
http://www.michigan.gov/dnr/0,1607,7-153-10370_12150_12220-26647--,00.html

Epizootic Haemorrhagic Disease (EHD)

Description

Epizootic hemorrhagic disease (EHD) is an acute, infectious, often fatal viral disease of some wild ruminants. This malady, characterized by extensive hemorrhages, has been responsible for significant epizootics in deer in the northern United States and southern Canada.

A similar hemorrhagic disease called bluetongue also occurs throughout the U.S. and Canada. The two diseases are antigenically different.

Distribution

Since 1890, deer die-offs from diseases which might have been EHD have occurred in various parts of North America. These early die-offs were variously diagnosed as blackleg, blacktongue, bluetongue, mycotic stomatitis or hemorrhagic septicemia or they were undetermined. The causative agents were never confirmed. A review of the case histories, signs and lesions, seasonal occurrence, and lack of a bacterial agent suggests that they might have been EHD.

The first occurrence and subsequent identification of EHD occurred in 1955 when several hundred white-tailed deer (*Odocoileus virginianus*) succumbed in both New Jersey and in Michigan. It was considered a new disease of deer and the name `epizootic hemorrhagic disease' was suggested to describe its main clinical and pathological features.

Since these initial confirmed outbreaks of EHD, documented epizootics have occurred in white-tailed deer in South Dakota, North Dakota, Wyoming and Alberta, Canada. Suspected EHD outbreaks have occurred in Missouri, Washington, Nebraska, Iowa and British Columbia. South Dakota, Missouri and Nebraska have experienced periodic outbreaks of EHD and the disease might be considered enzootic in these areas.

Since the initial 1955 outbreak, this malady has occurred primarily among white-tailed deer, although occasionally mule deer (*O. hemionus*) and pronghorn antelope (*Antilocapra americana*) have succumbed.

Additional die-offs attributed to EHD occurred in Michigan in white-tailed deer in 1974 and 2006. The 1974 die-off occurred in several counties and resulted in approximately 100 deer dying. The 2006 die-off occurred in the southwestern portion of the state in Allegan county and involved 50-75 animals.

Transmission and Development

The mode of transmission of EHD in nature is via a *Culicoides* biting fly or gnat. *Culicoides variipennis* is the most commonly incriminated vector in North America. A common observation in outbreaks involving large numbers of deer - as in Michigan, New Jersey and Alberta - is that they are single epizootics which do not recur. Die-offs involving small numbers of deer - as experienced in South Dakota and Nebraska - occur almost annually, and the disease appears to be enzootic in these areas. All documented outbreaks of EHD have occurred during late summer and early fall (August-October) and have ceased abruptly with the onset of frost.

Experimentally, the disease can be transmitted to susceptible deer by the inoculation of virus-laden material from infected deer by subcutaneous, intramuscular, intravenous or oral routes.

Clinical Signs

Clinical signs of EHD and bluetongue are very similar.

White-tailed deer develop signs of illness about 7 days after exposure. A constant characteristic of the disease is its sudden onset. Deer initially lose their appetite and fear of man, grow progressively weaker, often salivate excessively, develop a rapid pulse and respiration rate, and finally become unconscious. Hemorrhage and lack of oxygen in the blood results in a blue appearance of the oral mucosa, hence the name 'bluetongue'. Eight to 36 hours following the onset of observable signs, deer pass into a shock-like state, become prostrate and die.

Pathology

The gross and histological lesions of EHD have been characterized by, as its name implies, extensive hemorrhage. The hemorrhages range from pinpoint to massive in size, and involve different tissues and organs in individual animals. No organs appear to be exempt from hemorrhage, with the most regularly involved being the heart, liver, spleen, kidney, lung and intestinal tract. Extensive hemorrhaging is the result of interference with the blood-clotting mechanism together with degeneration of blood vessel walls.

Generalized edema and increased pericardial fluid are consistently found in EHD. These changes also reflect the widespread interference with normal blood circulation.

The virus can be recovered from a variety of tissues of animals which have succumbed to EHD. These include blood, liver, spleen, kidney, lung, heart and muscle.

Diagnosis

A combination of case history, characteristic signs and lesions, and the isolation of the virus is necessary for a diagnosis of EHD. Useful aids in obtaining a diagnosis are the epizootic nature of the disease, its seasonal occurrence, and its spectacular hemorrhagic lesions. Because of the similarity of its symptoms to other diseases, such as bluetongue and malignant catarrhal fever, the isolation and identification of the virus is essential.

Methods to be used for virus isolation are: (1) inoculation of cell cultures; (2) inoculation of susceptible sheep or deer combined with serologic monitoring; and (3) intravenous inoculation of embryonating chicken eggs.

Treatment and Control

There is no known effective treatment or control of EHD. Theoretically, an oral vaccine could be developed for administration through a supplementary winter feeding program, but this is presently impossible, impractical and unwarranted.

Significance

Because of its very high mortality rate, EHD can have a significant effect upon the deer population in a given area, reducing numbers drastically. Hemorrhagic disease can be transmitted to other wild ruminants. The EHD virus can infect domestic animals but rarely causes disease.

In all probability the virus does not infect humans

Website Reference:
http://www1.agric.gov.ab.ca/$department/deptdocs.nsf/all/agdex742

Johne's Disease

Cause | Signs of infection | Diagnosis | Vaccination | Control | Prevention and control programs in Alberta

Johne's disease (paratuberculosis) is a chronic, debilitating disease that affects the intestines of all ruminant animals, including cattle, sheep and goats. Although many animals in a herd may be infected, usually less than five per cent of infected animals develop clinical signs of disease. This is called the "Iceberg effect." Under circumstances of stress, inadequate nutrition or parasitism, more of the infected animals may develop clinical disease. Once clinical disease develops, affected animals eventually die due to dehydration and starvation as a result of malabsorption of nutrients. There is no treatment for Johne's disease.

The true prevalence and economic losses associated with Johne's disease have not been determined. This is because there is no practical diagnostic test that reliably detects infections of Mycobacteriurn paratuberculosis (MAP) in living animals that have not developed clinical disease. The fact that MAP grows very slowly delays the immunological response and detection of the bacterium in serum and in feces.

The epidemiology of the disease is different in dairy and beef herds, mainly due to management factors. The differences in cow and herd prevalence, along with the long incubation period of MAP, causes a broader range in sensitivity of the diagnostic tests (10 to 50 per cent) and high variation in the serology versus fecal culture tests, mainly during early stages of the disease when it remains sub clinical.

Several studies have been performed in Alberta in the last few years to determine the prevalence of Johne's disease. In these studies more than half of dairy herds and approximately 14 per cent of beef herds appear to be infected with MAP. However, these numbers may be higher due to the factors explained before.

The most significant economic losses associated with Johne's disease are decreased milk production and reduced salvage value of clinically affected animals. Milk production from dairy cows affected with Johne's disease has been estimated to drop by 590 kg in the third lactation and up to 1,270 kg in the fourth lactation. Affected animals have an increased risk of being culled early and the slaughter weight of these animals was 59 kg less than non infected animals.

Cause

This disease is caused by *Mycobacterium paratuberculosis*, which is closely related to the organisms that cause tuberculosis and leprosy. This organism does not cause disease in non-ruminant animals, however, these animals may perform as reservoirs, creating a risk to transmit the disease. Infection with M. paratuberculosis is usually acquired by consuming feed and water contaminated with manure from infected animals. Fecal shedding from clinically affected animals has been estimated to reach more than 500 billion organisms per day. Obviously, the environment can become

very heavily contaminated and most animals on these farms will be exposed to the infection.

Up to 35 per cent of cows with advanced clinical disease will shed *M. paratuberculosis* in their milk. As well, there are reports of calves being born already infected, but this appears to occur only among dams that are shedding very high numbers of the organism.

Mycobacterium paratuberculosis is resistant to environmental degradation, as well as many disinfectants. This organism survives in stagnant water, manure or deep soil for up to a year. It also withstands freezing at minus 14°C for up to a year. The ability of this organism to survive in the environment is reduced by the presence of urine or by the ensiling process.

Calves under six months of age are the most susceptible to infection. Depending on the number of organisms the calf is exposed to, only about a third of exposed calves become chronically infected with *M. paratuberculosis*. Clinical signs of disease are seldom observed in animals under two or three years of age. Consequently, this is a disease of adults in which the infection was acquired in the first few months of life.

M. paratuberculosis infects cells that line the intestines, but does not damage them. It is also not detected by the immune system, or does not trigger an immune response enough to eliminate it at early stages in younger animals. However, as the disease progresses clinical signs may appear as a result of the body's immune response to the presence of M. paratuberculosis, not by direct damage of the intestinal cells by the organism itself.

Signs of Infection

Less than five per cent of infected animals develop clinical signs of illness. The reason for this is unknown. Infected animals without clinical signs act as carrier animals and are a source of infection to the environment on the farm. Clinical signs seldom develop in animals under two to three years of age. Affected animals may develop intermittent bouts of diarrhea that gradually become more frequent. Other animals suddenly develop diarrhea, which persists until death. Progressive weight loss is typical of this disease and may begin before diarrhea develops. Although affected animals appear unthrifty, with a rough hair coat and declining milk production, their appetite remains normal until the terminal stages of the disease. As a result of the chronic protein loss through diarrhea, affected animals may develop ventral edema (bottle jaw).

Weight loss without diarrhea is the main sign of disease in sheep and goats.

Diagnosis

Animals with chronic, non-responsive diarrhea and progressive emaciation, coupled

with a normal appetite should be viewed with suspicion. Johne's disease is confirmed by a postmortem examination where increase thickness and transverse folds in the intestinal lining are observed.

It may be difficult to detect infected animals that do not exhibit clinical signs. Culture of feces for M. paratuberculosis is expensive because of the long time required to grow the organism. Cultures must be incubated for up to four months before they can be called negative. Intermittent shedding also makes a negative fecal culture difficult to interpret. Several blood tests are available, but the number of false positives and negatives makes these tests unreliable.

Vaccination

Vaccines have been developed in the United States, Europe and New Zealand. They are effective in reducing the number of clinically affected animals. Unfortunately, vaccination does not reduce the total number of infected animals in the herd.

Reactions at the injection site in animals or in humans injected accidentally are major concerns regarding the use of paratuberculosis vaccines. These vaccines are not available in Canada because they interfere with subsequent tuberculosis tests.

Control

Producers should attempt to maintain a disease free herd by rearing their own heifer replacements. Purchasing replacements is risky because of the lack of a reliable test to detect infected animals that are not showing clinical disease.

Once the disease has been diagnosed in a herd, avoid selling the animals for dairy or breeding purposes. Infected herds have up to 20 times more carrier animals than those showing signs of disease.

Because of the difficulty in detecting carrier animals, eradication of Johne's disease from a herd is unlikely to be successful. Even complete depopulation may be unsuccessful due to the risk of purchasing carrier animals to repopulate the herd. Rigid culling procedures and improved herd management will reduce Johne's disease to acceptable levels.

Recommended management practices for infected herds include the following:

- Cull animals exhibiting signs of clinical disease that suggest of Johne's disease. Have the carcasses examined to confirm the diagnosis.
- Cull all offspring, dams and siblings of confirmed cases.
- Separate unthrifty animals from the herd.
- Clean and disinfect areas where affected animals have been kept. M. paratuberculosis is susceptible to 10 minutes exposure to five per cent formalin, 1:32 cresylic disinfectant, 1:40 phenol, 1:1000 mercury bichloride

and 1:50 calcium hypochloride.
- Remove manure from the barn yard regularly and spread on cultivated land. Avoid spreading manure on pastures.
- Pasture calves on clean pasture and maintain in winter quarters separate from adults until the heifer enters the milking herd.
- Drain, fill or fence off sloughs.
- Protect young animals from adult manure drainage.
- Ensure feed and water are not contaminated with manure. Drinking water should be piped from clean sources.
- Remove calves from their dams immediately after birth and put them in clean quarters separate from adult animals.
- Clean the udder before drawing colostrum for calf feeding.
- Rear calves in individual pens and switch to a high quality, powdered milk replacer after 72 hours of age.

Prevention and Control Programs in Alberta
Currently, there are two programs and one project in Alberta:

The Alberta Voluntary Johne's Cattle Herd Status Program (VJHSP)
This Alberta initiative was started in September 2001 to reduce the likelihood of Johne's disease in Alberta's cattle herds. The goal of the herd status program is to: identify and categorize herds on the strength of apparent freedom from Johne's disease, provide a simple system to communicate to cattle buyers the risk of Johne's disease infected animals and stop the spread of this infection to non-infected cattle herds. This four-level voluntary program offers to interested cattle producers the opportunity to work with accredited veterinarians and to evaluate the Johne's disease status of their herds using specific sampling and testing protocols. Each level represents an increase in confidence that the herd is free from Johne's disease.

The CanWest DHI Johne's Disease Prevention Project
This project was initiated in Ontario in 2005 and in 2006 it was extended to the western provinces. Several industry organizations, government agencies and academia are the project partners. This project began in Alberta on March 2006. It provides assistance to veterinarians and dairy producers to develop sound calf rearing programs that help prevent the spread of Johne's disease. This project is executed through trained veterinarians who conduct risk assessments and evaluate the potential financial impact of Johne's disease in a particular herd.

The National Johne's Disease Prevention and Control Program
Alberta producers, including dairy, beef, sheep and goat producers, have agreed to join the National Johne's Disease Prevention and Control Program, an initiative proposed by the Canadian Animal Health Coalition. This organization was formed by a broad group of stakeholders in 2006. The program provides two pathways. The first is the testing pathway, which is designed for herds with low prevalence of infection. The second is the prevention pathway, which takes many elements from the CanWest DHI project. The key element of the prevention pathway is keeping the healthy and

susceptible animals in the herd (i.e. young calves) disease free by reducing the risk of transmission from those animal that are presumably infected with Johne's disease.

This strategy is designed to determine the risk factors present at each farm that influence the occurrence of Johne's, and then to gradually reduce the presence of the disease by eliminating those risk factors. A trained veterinarian works with the producer to design a plan of best management practices for implementation. Every year this plan is reviewed and updated, with the ultimate goal of reducing and eventually eradicating the problem. Some degree of testing may be required during this process to provide information on the progress and the impact of the new management practices.

Website Reference:

http://www.unbc.ca/nlui/wildlife_diseases/parelaphostrongylus_tenuis.htm

PARELAPHOSTRONGYLUS TENUIS -
MENINGEAL WORM - BRAIN WORM

Meningeal worm or brain worm has been transferred from the genus *Pneumostrongylus to Parelaphostrongylus,* hence is now known as *Parelaphostrongylus tenuis.* This species is a slender, delicate roundworm found in the cranial cavity of cervids. The white-tailed deer is the normal host of *P. tenuis.*

The adult worms are normally located between the membranes (meninges) covering both the brain and spinal cord, but are found more frequently in the meninges of the brain. The adult worms deposit eggs either on these membranes or directly into blood vessels. Those deposited on the membranes, hatch and the larvae enter small blood vessels to be carried to the lungs where they enter the alveoli. Eggs deposited into blood vessels are carried to the lungs and eventually hatch with larval penetration of the alveoli. This activity in the lung tissue produces an interstitial pneumonia. The larvae pass up the respiratory tract from the alveoli, are swallowed and then eliminated in the faeces. Larvae appear in the faeces about three months after the host becomes infected.

The larvae then penetrate into gastropods (snails and slugs) which act as intermediate hosts. Development of the larvae in the gastropod to a stage when they are infective to the vertebrate host takes about three weeks. Cervids become parasitized by ingesting infected gastropods with their food.

In the final host, development of the larva to the adult worm takes place in tissues of the central nervous system, particularly the spinal cord. Parasites leave the tissues of the spinal cord after about 20-40 days and locate between the spinal membranes where they mature. Subsequently they tend to accumulate in the cranial region. The adult worms are about 50 mm in length and may be seen fairly readily when free in the cranial cavity. From one to 20 worms have been found in the crania of infected deer.

P. tenuis seldom causes damage in white-tailed deer.

In other cervids there is often extensive damage to tissues of the brain and spinal cord. The resulting neurologic disease is characterized by weakness, fearlessness, lack of coordination of movement, circling, deafness, impaired vision, paralysis and subsequent death. When in moose this disease is often called “moose sickness” or, “moose disease”.

The life cycle of *P. tennis* and the relationship between this parasite and "moose sickness" were first elaborated experimentally by Anderson (1963, 1964). The early experimental work stimulated extensive study of meningeal worm in the natural environment.

In Canada, studies by Anderson (1965, 1971, 1972), Bindemagel and Anderson (1968), Kelsall and Prescott (1971), Lankester and Anderson (1968) and Saunders (1973) have substantiated the importance of this problem in management of big game and have given us some indication of the dynamics of the hostparasite relationships among wild populations.

P. tenuis has a wide distribution in eastern and central North America. Within Ontario in the last 100 years, changes in the environment have resulted in the northern extension of the deer range. Both deer and meningeal worm have shared range with moose. "Moose sickness" has been restricted to areas where deer and moose ranges overlap. Declines in the moose populations of New Brunswick, Nova Scotia, Maine and Minnesota appear to be associated with this disease.

Failure to consider the importance of the effect of this parasite on cervids other than white-tailed deer resulted in the loss of a small herd of reindeer introduced from Norway to Ontario in 1969.