

# JOHNE'S DISEASE

An in-depth interview with

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# Johne's Disease

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## 1. What is Johne's disease?

Johne's disease is a condition that results in progressive weight loss and intermittent watery diarrhea in both wild and domestic animals. Progressive weight loss often occurs despite normal appetite and feed intake. Johne's is unresponsive to treatment and signs of the disease typically develop between 2 and 6 years of age.

Johne's disease is caused by a bacterial agent and infection is thought to occur primarily by ingestion of contaminated fecal material. Newborn calves are most susceptible to infection but clinical signs of Johne's disease or diarrhea are not observed at this time.

## 2. Why is the beef industry concerned?

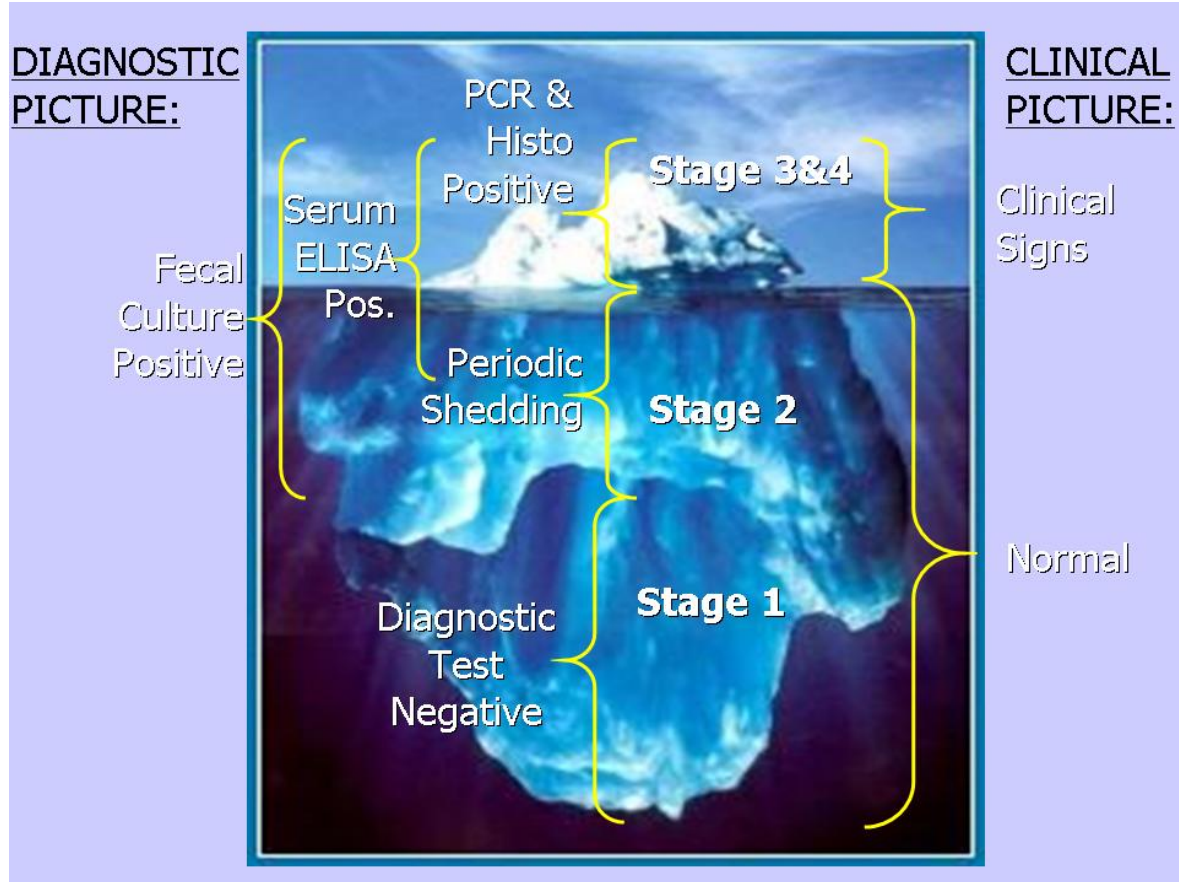
For individual animals, the economic costs of Johne's disease include production losses due to shortened productive lives for cows with clinical disease and reduced weaning weights for calves from infected cows. The magnitude of these losses has not been well documented for the beef industry. The biggest potential economic loss would be for purebred and seed stock producers who have spent their lifetime creating herd genetics. Once a producer confirms Johne's disease within the herd then an immediate question or concern for these producers is "Do I stop selling animals to prevent further spread of disease?"

At a herd level, difficulty in identifying infected animals prior to them showing clinical signs is one of the main issues associated with controlling this disease. For example, if all infected animals cannot be positively identified then it is difficult to eliminate the source of infection within the herd. Once one cow with Johne's disease is identified in a herd then it is difficult to know with certainty when all infected animals have been eliminated.

At a national level, there is concern that Johne's disease may be used as trade barrier to restrict the movement of cattle despite the presence of this disease on every continent.

Another potential concern to the beef industry is a possible public health risk due to a suggested link between Johne's and Crohn's disease.

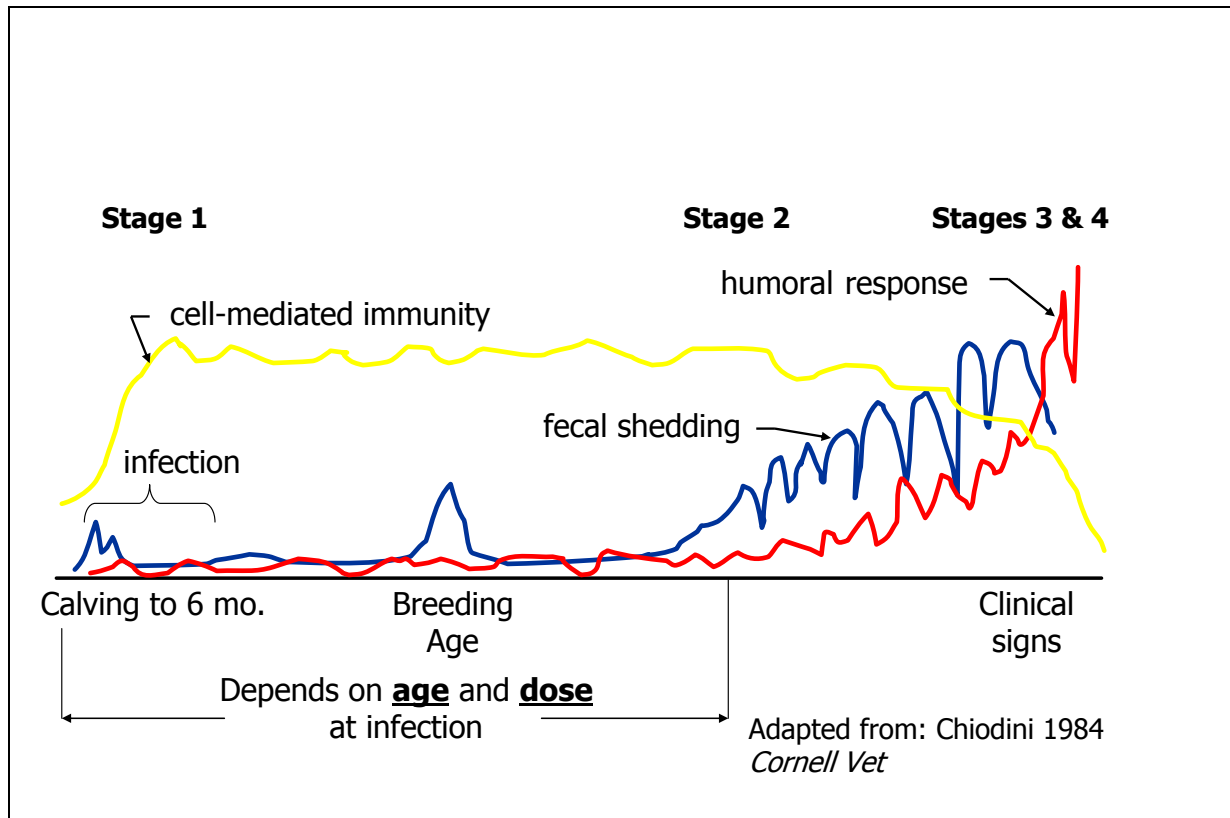
### 3. How does Johne's disease develop?



**Figure 1: The “Iceberg” Disease**

The development of Johne's disease can be described in terms of clinical signs. Animals appear healthy for 3 to 5 years (**Stage 1 and 2**) and during this time the available diagnostic tests fail to detect infection. Many animals never develop diarrhea and wasting but these animals may shed bacteria in their feces. Those animals that develop clinical signs of Johne's disease (**Stage 3 and 4**) are much easier to detect using a variety of diagnostic tests, including bacterial culture from feces, serum tests (**ELISA**), and post-mortem examination (**histology, bacterial culture, PCR**). Johne's disease has been described as an “iceberg” since detection of a single cow with Johne's disease in a herd implies many other animals may be infected.

#### 4. How do animals respond to infection?



**Figure 2: Johne's Timeline**

The time required for Johne's disease to develop (**Stage 1 and 2**) depends upon the animal's age and the dose of bacteria at the time of exposure. During this incubation period, animals may respond to infection with a protective immune response (**cell-mediated immunity**) and fecal shedding of bacteria is limited. As Johne's disease develops (**Stage 3 and 4**) the protective immune responses decline and serum antibody responses (humoral response) develop but fail to protect against disease.

## **5. What is the cause of infection?**

Johne's disease is caused by a bacterial infection (*Mycobacterium avium* subspecies *paratuberculosis* [MAP]) which occurs following ingestion of bacteria from contaminated milk, feed, or water. This infection is thought to occur most frequently in young calves but diarrhea or scours do not occur at the time of infection. The mycobacteria invade cells inside the animal, making it difficult for the immune system to control or eliminate these infections.

## **6. What is the prevalence of Johne's infection in Canadian beef herds?**

Serological surveys indicate the prevalence of Johne's disease is relatively low in beef cattle, with 0.8 to 1.7% of seropositive animals identified in 3 to 11% of beef herds in Western Canada. In contrast, surveys in the U.S. indicate Johne's infected animals are present in approximately 60% of dairy herds.

In contrast, a serological survey of Alberta dairy herds in 2002-2003 determined that two or more infected animals were present in 58.8% of herds. This may be an important consideration when purchasing dairy calves to foster on a beef cow.

## **7. Does every exposed animal become infected for life?**

There is insufficient research to know whether every animal exposed to MAP will become infected. We do not know if there is a minimal infective dose of bacteria or if the risk of infection changes with the age of the animal. Although newborn calves are considered to be at greatest risk for infection it is also possible that older animals become infected if exposed to a sufficient dose of MAP.

## **8. Can an infected animal clear the infection?**

There are no published studies proving an infected animal can eliminate MAP infection but a very small number of animals exposed to MAP develop clinical disease. Therefore, we presume healthy animals can develop protective immune responses and clear infection if there is a single or repeated exposure to a low dose of the bacteria.

## **9. Does every infected animal shed MAP?**

Shedding of MAP is thought to occur primarily through feces but there is evidence MAP can infect the udder and be shed in milk. Detection of MAP in feces is difficult since methods used to culture MAP are relatively insensitive and require weeks to months to produce a diagnosis when low levels of MAP are present in samples. Shedding of MAP in feces varies with the stage of the disease. Young calves and cows with preclinical disease appear to shed low or non-

detectable levels of MAP. All infected animals are thought to eventually shed MAP when clinical disease develops but this shedding may be intermittent (Figure 1).

### **10. How is MAP infection transmitted from animal to animal?**

MAP is a ‘manure bug’, which means infection occurs primarily by the oral route (fecal/oral transmission). Suckling calves may also be exposed to MAP through colostrum and milk but the major risk of infection is thought to be through exposure to feces. The highest levels of MAP are found in the feces of animals with Johne’s disease.

### **11. When do animals become infected?**

Infection may begin during pregnancy but *in utero* infections are thought to be relatively rare. Calves from cows with clinical Johne’s disease have a 25% chance of being infected. It is suspected that newborn calves are at greatest risk of infection and the majority of calf infections occur by ingestion of contaminated feces. There continues to be a debate whether older animals are at the same risk for infection.

### **12. What risk factors are associated with Johne’s disease?**

Fecal contamination of the environment and water is the major risk factor for Johne’s disease. The presence of animals with clinical Johne’s disease and their progeny is a major source of environmental contamination and the level of bacterial shedding by these animals may increase during calving and periods of stress.

Grazing practices that increase contact with soil and feces may also change the level of exposure but the infective dose of MAP is not known. Very high numbers of MAP can be shed per gram of feces during clinical Johne’s disease. Therefore, there are potentially high doses of MAP in a small amount of feces.

There are, however, regional differences in the prevalence of Johne’s disease throughout Canada. Factors which may contribute to regional differences include weather, soil type, and wildlife contact with cattle.

Johne’s disease is also found in sheep, goats, bison, elk and deer, and MAP has been cultured from rabbits, dogs, cats and coyotes. It remains to be determined whether these animals are a potential source of infection for cattle.

Epidemiological studies have also implicated commercial colostrum and milk replacer products as potential risk factors for Johne’s disease, but the actual role these products play in disease transmission remains uncertain.

### **13. Can the risk of infection be controlled?**

MAP is a very stable bacterium in the environment. Studies done under Western Canadian conditions suggest the bacteria can survive in the environment for at least one year. Therefore, proper hygiene and prevention of feed contamination are of utmost importance, especially for the calving environment.

Since manure is a major source of potential MAP infection it shouldn't be spread on pasture or hay fields. Water sources should be clean and standing surface water should be eliminated.

Animals identified with clinical Johne's disease and their progeny should be removed from the herd as quickly as possible.

If a producer feeds calves fresh milk from a neighbor's herd they need to be aware that this may be a potential source of MAP infection.

Carcasses from cattle with Johne's disease should be disposed of in a manner which minimizes environmental contamination or ingestion by wildlife as this may also help reduce disease transmission.

### **14. How is Johne's disease diagnosed?**

Currently there is no one test available for rapid, sensitive, and accurate detection of Johne's disease. Diagnosis of MAP infection in live animals is currently done by genetic testing of feces, culturing the organism from feces, or serum analysis for antibodies.

Culture of MAP from feces takes two to four months and the present culture tests also lack sensitivity due to intermittent shedding of MAP in feces. There may be many false negatives and a positive diagnosis may require repeated fecal samples. This test is costly and creates a considerable waiting time for producers after a fecal sample is collected from a cow. For example, a producer might collect manure at the time of a pregnancy check and four months later the cow may have already calved. Management changes or culling the cow and her progeny before that time could prove beneficial for herd health.

Polymerase chain reaction (PCR) is a genetic based test which amplifies specific DNA sequences from the MAP organism. This test can be applied directly to manure and provides a more rapid diagnosis of a week or less. The major limitation with PCR tests is a lack of specificity because of other related bacteria and the presence of inhibitors in the manure.

Serum testing by ELISA detects MAP-specific serum antibodies which are produced when the cow develops an immune response to the bacteria. MAP-specific antibodies are only produced late in the disease process and are usually detected 6 to 18 months after fecal shedding begins (Figure 2). Serum antibodies may be detected prior clinical Johne's disease signs but the sensitivity of ELISA is lower than bacterial culture, and similar to PCR testing. The advantage of



ELISA testing is the relatively low cost and rapid diagnosis and may be most valuable for confirming whether there has been herd exposure to MAP.

Johne's disease can also be diagnosed at the time of post-mortem examination by collecting tissues (ileum, mesenteric lymph nodes) for bacterial culture or special stains which visualize the bacteria within tissue sections.

### **15. What should a producer do with a suspected Johne's case?**

It is not essential to cull these animals but the producer should be aware of the potential risks associated with these animals. It is recommended that these animals be tested to determine if they are shedding MAP and present a risk of environmental contamination or disease transmission.

### **16. What should a producer do following a positive Johne's diagnosis?**

In the beef industry, where cows and calves are commingled, the first response is to cull the infected animal and progeny.

It is also important to institute management practices which minimize the risk of further environmental contamination and reduce the risk of exposure for remaining animals (refer to #13).

Cows with clinical Johne's disease can be very thin and weak and these animals are at a much higher risk of injury during transport. These animals should be humanely euthanized on farm and commercially rendered rather than being left for scavenging by other species which may create a disease reservoir. There is no evidence whether rendering completely inactivates MAP and it is uncertain whether composting inactivates the organism. These areas are presently being investigated.

The sale and disposal of healthy cohorts of infected animals is an ethical issue. The recommendation would be not to sell these animals as breeding stock.

### **17. Is there a vaccine to control Johne's disease?**

A licensed vaccine is not available in Canada but there is a vaccine in the USA which is reported to reduce fecal shedding but not prevent infection. Thus, the vaccine may reduce bacterial contamination of the environment. Calves need to be vaccinated in the first weeks of life and the vaccine is given in the brisket causing a large grapefruit-sized swelling which lasts throughout the life of the animal. This may not be acceptable to purebred producers.

## **18. Are there Control Programs for Johne's disease?**

Some countries have instituted voluntary control programs, such as Australia ([www.animalhealthaustralia.com.au/programs/jd/njdcp.cfm](http://www.animalhealthaustralia.com.au/programs/jd/njdcp.cfm)) and the USA, ([www.johnes.org/handouts/files/JD\\_Program\\_Standards.pdf](http://www.johnes.org/handouts/files/JD_Program_Standards.pdf)) which restrict movement of animals with Johne's disease. Other countries, such as the Netherlands, use routine testing of milk to monitor the prevalence of MAP infection in dairy cows.

Control programs vary by region in Canada. Alberta has a voluntary control program in place, but participation in the program is minimal. Voluntary programs have been developed in a number of provinces. There is a framework for a national program to be implemented. The overall goal of these programs is the use of good management practices rather than "test and cull" programs to control Johne's disease.

## **19. Is there evidence for a causal link between MAP and Crohn's disease?**

Similarities in intestinal tissue changes during Johne's disease and Crohn's disease are suggestive of a common causal agent. A Canadian survey of intestinal tissue samples confirmed MAP organisms were present in 59% of Crohn's patients (n = 17) and 14% of control patients (n = 35) (Jeyanaythan et al. 2007). Furthermore, antibiotic treatment of Crohn's disease provided equivocal results which may support the idea that Crohn's is caused by a bacterial agent (Behr and Hanley, 2008). It is very difficult, however, to dismiss or prove a causal relationship between MAP and Crohn's disease. While MAP infection may occur many years prior to Crohn's disease, Crohn's may also predispose to MAP infection.

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