Johne’s Disease
Management for New Zealand dairy herds
The Johne’s Disease Research Consortium is an Unincorporated Joint Venture established in 2008 with a mandate to reduce the impact of Johne’s Disease on farm in New Zealand.

It has as its participants Beef + Lamb NZ Ltd, DairyNZ Inc, DEEResearch Ltd, Massey University, University of Otago, AgResearch Ltd and Livestock Improvement Corporation.

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Introduction

Johne’s Disease (JD or paratuberculosis) is caused by a bacterial infection of the gut in cattle and other ruminants with *Mycobacterium avium paratuberculosis* (MAP). Gradual thickening and inflammation of the intestinal wall eventually prevents uptake of nutrients. Clinical JD is characterised by ill-thrift, progressive weight loss and profuse diarrhoea. No cure is available and the condition is eventually fatal.

Calves and young stock are particularly susceptible to infection. However, the disease has a very long ‘incubation period’ so that clinical signs of JD typically appear several years later in the adult cow. Shedding of MAP (primarily in faeces) also increases with age and advanced stage of infection.

**Johne’s Disease was first discovered in New Zealand over 100 years ago in a Taranaki Jersey cow.** Since the diagnosis in 1912, JD has been found in farmed ruminants throughout New Zealand. It is widespread in dairy and most herds probably harbour some MAP infection. While disease incidence is very low in many herds, JD causes significant animal health problems and production losses in some herds.

**Top control priority: Reduce calf and heifer exposure to adult faecal matter.** Although the epidemiology of JD is not fully understood, it is clear that specific risk factors favour transmission of MAP resulting in higher incidence and severity of clinical disease. Some farming practices, such as type of effluent spreading, may inadvertently lead to a gradual build-up of MAP in the environment and increased JD. Once a threshold level of herd infection and MAP shedding is reached, a vicious transmission cycle may ensue. At this point, specific interventions are necessary to bring MAP infection in the herd under control.

**The impact of Johne’s Disease can be minimised** An intervention such as test-and-cull may produce some quick benefits by limiting production losses due to the clinical disease. However, the full advantage of changed management practices that reduce transmission will only be seen after several years because of the protracted duration of subclinical stages of MAP infection.
The MAP lifecycle in the herd

**NOT INFECTED**
- Adults are less susceptible
- Some early stage cattle recover
- False positives extremely rare
- But a negative test result does not confirm absence of infection.

**LATENT MAP INFECTION**
- Not shedding any MAP
- Rarely test positive
- Some animals may recover.

**AT RISK CALVES AND HEIFERS**
- Highly susceptible to infection
- Rarely test positive if infected.

**CLINICAL JOHNE’S DISEASE**
- High MAP shedding - including super-shedders
- Mostly test positive
- Ill-thrift, wasting, death.

**EARLY SUBCLINICAL MAP SHEDDER**
- Low level MAP shedding - may shed intermittently
- Mostly test negative
- Appears clinically healthy.

**ADVANCED SUBCLINICAL MAP**
- High MAP shedding - including super-shedders
- Mostly test positive
- Milk production drop.

**TRIGGER**
Stresses e.g. calving may trigger onset of clinical JD.
The purpose of this guide is to provide the New Zealand dairy industry with risk management tools to combat Johne’s Disease and protect against the spread of MAP infection in the herd.

JD eradication from commercial dairy herds in New Zealand is not feasible with today’s technology, thus control measures are aimed at risk reduction. A series of management strategies have been designed to minimise transmission of MAP to new and young stock and to limit the impact of JD in the herd.

This toolbox provides specific and targeted interventions for dairy farmers struggling to cope with high annual losses arising from clinical JD, while also being relevant to all dairy farmers and rearer swishing to guard against spread of the disease. Therefore, this guide provides management tools without being prescriptive. Not all tools, especially the higher input measures will suit all herds. But herds with severe JD and incurring considerable financial losses should benefit from applying one or more of the management interventions. With the help of their veterinary advisors, farmers should select the most appropriate practical interventions from the toolbox.

To help decision making, the management practices for each of the five strategies are presented in three categories:

**BEST PRACTICE**
Control actions target MAP transmission to the calf. They are intended to be practical, but individual affected herds may consider some too difficult or costly to implement.

**ALTERNATIVE OPTIONS**
If implementing best practice is not practical, alternatives can help to mitigate risks.

**HIGH-RISK BEHAVIOURS**
All herds, also low risk JD herds, should aim to eliminate these high risk practices.
Calves and young stock
- Most susceptible to infection.
- Calves under 6 months are most at risk, but older calves remain vulnerable to infection.
- Repeated ingestion of high doses of MAP increases the progression and severity of the disease.
- Tests are not able to identify infected calves effectively.
- Shedding is rare in young stock.

Adults
- The major source of MAP.
- Cows begin to shed MAP before clinical signs appear and spread the disease.
- Clinical signs of JD often appear after many years with highest incidence usually amongst 5-7 year old cows.
- Tests are good at identifying cows with advanced infection especially high MAP shedders and early clinical JD.
- MAP are primarily excreted in faeces but cows in advanced stages can transfer MAP during pregnancy and via colostrum or milk.
- Super-shedders excrete huge bacterial numbers – up to a million per gram of faeces.

Mycobacterium avium paratuberculosis (MAP)
- Can survive for several months in effluent, water or on pasture.
- Dark, damp and cool conditions promote survival.
- MAP invades the intestinal wall and lymph nodes where it replicates and gradually causes increasing severe damage.

Other ruminant species
- All ruminants are susceptible to MAP infection and JD.
- Close contact or co-grazing may lead to cross-species transmission of MAP.
- Wildlife may be infected but their role is unclear but unlikely to be a significant JD risk to replacement heifers.
The management strategies and tools

JD risk management primarily revolves around protecting the calf from MAP infection. The interventions in this toolbox have been grouped into five strategies:

1. **Test-and-cull of clinical and high-risk cattle**
   *To eliminate a major source of MAP before calving and reduce losses from clinical JD*
   Cows with clinical JD disease are an obvious source of MAP and need to be removed from the herd ASAP. However, faecal shedding may start several years before JD signs appear. Cows in advanced subclinical stages are a major source of MAP. Some cows become super-shedders with the potential to infect many calves with a large dose of MAP. Fortunately, JD tests are good at identifying cows with advanced infection.

2. **Calving and colostrum management**
   *To minimise exposure to MAP before birth and at calving via dams’ faeces or colostrum*
   Calves that ingest high doses of MAP are more likely to develop JD earlier. While it is impossible to prevent all contact with faeces and other sources of MAP from the dams, it is important to try to limit exposure.

3. **Pre-weaning calf management**
   *To avoid contact with adults and prevent exposure to a MAP contaminated environment*
   Repeated ingestion of MAP can hasten the progression of the disease. For convenience the calf rearing shed is usually situated close to the milking shed. Do not allow contact with cows and protect calves from effluent.

4. **Replacement heifer management after weaning**
   *To remove susceptible heifers from any source of MAP until they join the dairy herd*
   Whereas adult cattle are less prone to a new infection than the young, calves remain highly susceptible at least for the first year and can be infected when older. Ideally the replacement calves should be removed from the dairy platform as soon as possible and managed at a rearing unit without adult stock (including other ruminant species).

5. **Biosecurity and purchasing low-risk stock**
   *To reduce the risk of importing MAP into the herd from high risk sources.*
Assessing the herd’s JD status

- Use whole-herd screening by the lab milk test.
- Monitor the herd for clinical signs of JD disease and use lab blood tests to confirm the diagnosis.
- Record all JD losses or diagnoses to determine the annual impact.
- Test milk of faeces of thin or older cull cows (4 years +) especially emergency culls or deaths.
- If 2-year old heifers develop clinical JD, it indicates the calves or heifers were exposed to massive MAP load.
- Lab tests of faeces or slurry tests may be useful to assess the amount of bacteria shed by cows and deposited into the farm environment.
- It is not possible to demonstrate freedom from MAP infection for a herd.
- However, whole-herd milk or serum testing with negative results suggests that the prevalence of MAP in the herd is low.

Factors that hasten the course of infection and severity of disease in individuals include:

- High infection exposure (number of bacteria).
- Repeated exposure to MAP.
- Age when infected – younger calves.
- MAP strains may differ in virulence.
- Breed – Jerseys are more prone to JD.
- Animal genetics – ongoing research to discover genes that confer resistance.
- Stress can trigger onset of clinical JD.

Clinical JD

Signs of Clinical Johne’s Disease
Clinical JD is the end stage of MAP infection. Thickening of the gut wall prevents uptake of nutrients and finally results in leakage of proteins. The animal essentially starves to death.

- Cows will often experience a drop in milk production before other signs appear.
- Visible signs include loss of body condition and ill-thrift in spite of normal appetite.
- Profuse diarrhoea causes muscle wasting.
- Bottle jaw (swelling under the jaw).
- No effective treatment.
- Death.

Clinical Johne’s Disease:

- Usually occurs in adults, particularly cows 4 years or older (3rd lactation onwards).
- Some infected cows do not develop clinical JD and may even clear the infection.
- Clinical JD in young cattle (2-year-olds) is an indication that the herd infection levels and transmission may be out of control.

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Johne’s Disease testing options

Tests may be used to 1) confirm JD, 2) help reduce MAP exposure and/or 3) select cows to cull before clinical JD develops.

1. Herd-test milk JD ELISA testing
   Herd-testing provides a convenient opportunity to screen the whole herd and cull high-risk JD cows before calving. Test costs are reduced by using an intermediate pooling step with a lowered cut-off.

   Results are ranked into (weaker) Positive versus High-positive categories. Overall, MAP shedding is heaviest amongst high-positive cows.

   See lab reports for more detailed interpretation.

   Note: Negative test results are reported as “Not detected” because in early stages most JD cows are negative and a few JD cows will remain test-negative throughout life

2. Blood serum JD ELISA testing
   Suspected JD cows and bulls can be diagnosed by a serum test. It can also check milk-test positive cows to rule out cross-contamination.

   Farms that experience severe JD losses and clinical JD signs amongst first-calf heifers, should consider bleeding rising 2-year-olds before they calve. Any test-positive individuals can be managed separately at calving and not bred but culled as soon as practical.

3. Faecal MAP PCR testing
   Real-time PCR tests can give a more accurate estimate of the amount MAP being shed by a cow in faeces. This requires quantitative interpretation by the laboratory and allows heavy and super-shedders to be identified.

   JD herds which are unable to cull all ELISA positive cows, may find this confirmation test useful to prioritise culling and management of high-risk cows.

<table>
<thead>
<tr>
<th>Milk JD ELISA result</th>
<th>Faecal Culture</th>
<th>Serum ELISA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Positive</td>
<td>Positive</td>
</tr>
<tr>
<td>All milk ELISA positive cows</td>
<td>77%</td>
<td>92%</td>
</tr>
<tr>
<td>‘High positive cows’ only</td>
<td>83%</td>
<td>98%</td>
</tr>
<tr>
<td>Remaining ‘positive cows’</td>
<td>47%</td>
<td>64%</td>
</tr>
</tbody>
</table>

Herd-Test Milk ELISA Positive Cows: % Positive on Confirmation Test
Real-time PCR results: Cows can be ranked by amount of bacteria in sample

Number of PCR cycles to reach threshold.

- **Super-shedding cows**: May shed up to a million MAP bacteria/gram
- **Heavy Shedders**: 100’s-1000’s MAP bacteria/gram
- **Moderate Shedders**: Less than 100 bacteria/g
- **Intermittent or low MAP shedders**: A few MAP bacteria/gram. Also may be false positive: pass-through bacteria.

Johne’s ELISA test performance: by stage of MAP infection

- Intermittent MAP shedders may be ELISA positive
- 70% moderate shedders
- >80% heavy shedders = ELISA +ve.

Data for IDEXX ELISA from Donat in 2014: based on 423 FC+ and 598 negative cows from 14 JD herds (between 2-50% FC positive cows).
Diagnostic Tests for JD

Diagnostic laboratory tests for JD/MAP can be broadly grouped into:

1. Tests which detect MAP
2. Tests that detect the immune response of the animal to MAP

Detecting MAP

- Common samples: Faeces and Slurry
- It is also possible to detect MAP in post-mortem, environmental or milk samples.
- Common test: PCR (DNA test)
- PCR stands for Polymerase Chain Reaction

Challenges

- Faecal PCR cannot detect non-shedders and low / intermittent shedders may be missed.
- PCR is a relatively expensive test.
- PCR may detect bacteria ingested during grazing ("pass-thru" bacteria), potentially giving some (weak) false positive results.

Advantages

- Demonstrates presence of MAP.
- Real-time PCR can give an indication of the amount of bacteria in a sample. This can be used to rank MAP shedders and identify the worst culprits.
- Traditionally faecal culture (FC) is the "gold standard", but costs and a slow turnaround time (6-12 weeks) make it impractical for farm use.

Detecting Immunity to MAP

- Common samples: Blood Serum or Milk
- Common test: ELISA (Antibody test)
- ELISA stands for Enzyme-Linked ImmunoSorbent Assay

Challenges

- Poor test performance in early stages of infection

Advantages

- Simple and relatively cheap.
- Good performance with advanced disease.
- Excellent for identifying high-dose shedders.
- False positive test results are rare.
- Ranking positive test results can improve predictive value: High-positive cows are likely to have advanced JD and high shedding.

Points to Note

- Testing alone will not control JD
- Use tests as a risk management tool.
- Ask your veterinarian for advice before you start testing.
- Your vet and laboratory can help with test interpretation.
Frequently asked questions

What is the difference between JD and MAP infection?

Johne’s Disease (JD) and paratuberculosis refer to the clinical disease caused by *Mycobacterium avium paratuberculosis* (MAP). The disease, often observed as diarrhoea and wasting), occurs several years after the initial infection with MAP bacteria. Up to this point ‘MAP infection’ is considered to be subclinical disease.

How do I know if I have a JD or MAP problem in my herd?

Look for clinical signs and confirm the diagnosis by a blood test, faecal PCR or post-mortem.

While clinical signs of JD such as diarrhoea and wasting can be severe and lead to death of the individual, a large part of the disease complex remains hidden during the subclinical phase and may be missed. In a herd with no more than 1% culling losses and deaths due JD annually, many more cows are likely to be infected.

On the other hand, a veterinary diagnosis is important to confirm JD and rule out other causes of diarrhoea or ill-thrift. To prevent MAP infection building up in the herd, keep a look out for JD and test cows with suspect clinical signs.

Clinical JD amongst two year old heifers in the herd indicates a very high MAP exposure of the replacements. Intervention options should be urgently investigated and implemented, based on a risk assessment with your veterinary advisor.

Can I vaccinate to protect my herd against JD?

Available vaccines interfere with TB diagnostics and, therefore, not used in New Zealand cattle herds.

Lamb vaccination (Gudair™) is a useful JD control tool for sheep farmers. Research suggests that one vaccine (Silirum™) can reduce the impact of JD in cattle herds and deer. In New Zealand, Silirum™ is occasionally used amongst finishing deer going direct to slaughter, but severe restrictions have been placed on the use of the vaccine for breeding stock as well as cattle. The problem:

- Vaccination causes false positive reactors with the TB skin test, requiring a blood test. More than 10% of vaccinated heifers may give false positives TB readings even two years after vaccination.
- In some TB infected cattle, vaccination results in false negative TB results.

How about test-and-cull to eradicate Johne’s Disease from the herd?

Laboratory tests are fairly good at identifying cows with advanced MAP infection but the typically long subclinical incubation period, with poor test performance in the early stages, renders test-and-cull programs expensive and ineffective. Instead, tests should be used as one tool in a wider management programme to reduce risks so that the impact of JD can be minimised. Some infected cows remain test-negative, but culling of any test-positive cattle will remove many high shedders to reduce calf exposure.

Does Johne’s Disease run in families?

A calf’s parentage may affect its chances of developing JD in two ways:

**Transmission**

A dam with advanced MAP infection or clinical JD is clearly one of the most important sources of transmission to her offspring. Not only is the calf exposed directly to MAP in the dam’s faeces (and via colostrum) after calving, but during the pregnancy MAP may be transmitted to the foetus in up to 10-40% of cows with advanced MAP infection or clinical JD. The risk of repeat exposure to high levels of MAP very early in life means these calves have a poor outlook and are more likely to develop clinical JD than their herd mates.

**Genetics**

Besides apparent breed predispositions various studies point to genetic differences in susceptibility versus resistance to infection. Genes may influence the likelihood of exposure leading to MAP infection, or the progression and spread of the infection in the animal, or the development of clinical JD. Genes may affect the susceptibility to initial infection, development and spread of the infection or expression of clinical signs. Research (including JDRC funded work) aimed at gene discovery for JD resistance, has identified some candidates. In time it may be possible to select sires to breed less susceptible dairy cows and possibly use genome screening in the herd.
What is the difference between cattle and sheep strains of MAP?
MAP are often classed into two major strains (and many sub-strains). As a rule type I (also S strain) is found in sheep while type II MAP (also C strain) is found in cattle and deer. The significance of this is not clear – some researchers have postulated that type I may be less virulent in deer and possibly cattle. A recent, JDRC funded, study confirmed that type II is the predominant strain in New Zealand dairy cattle herds but that a number of herds also harbour type I MAP and suffer clinical JD.

Can I graze other livestock with dairy calves and heifers?
Avoid contact or common grazing, especially if the JD status is unknown.
Beef cattle, deer and goats may be infected and shed MAP bacteria in their faeces. Therefore, the grazing of dairy calves and replacement heifers with adult beef or dairy cattle or goats or deer of any age is a high risk practice. The same principle applies to other ruminants, such as llamas and alpacas. Sheep are affected by MAP and may be an important risk to heifers. Certainly the type I strain (normally associated with sheep) appears to be widespread amongst some dairy herds. However, sheep tend to graze pastures to a low residual which may decrease MAP survival in pasture sward. Non-ruminant species such as horses and pigs do not pose a risk to replacement heifers.

Are rabbits and other wildlife a JD risk to dairy calves and heifers?
MAP can infect a wide range of animals and has been isolated from several feral species in New Zealand including rabbits, hares, hedgehogs, possums and others. Overseas research has shown some association between high JD incidence in the herd and MAP in rabbit pellets. MAP may be ingested by rabbits on pasture and excreted in a different paddock. It is unclear if infected rabbits or other non-ruminant wildlife shed sufficient MAP bacteria to play a significant active role in MAP transmission in New Zealand.

Can MAP be transmitted by AI or embryo transfer?
This is highly unlikely. Small amounts of MAP have been found in semen from bulls with advanced infection/ clinical JD. However, the risk of MAP transmission by AI is purely theoretical and has not been demonstrated. Similarly, MAP has been recovered from uterine washes during embryo transfer but standard ET procedures ensure that most pathogens are eliminated before implantation.

Natural mating bulls are a potential source of MAP via faecal shedding. This risk can be mitigated with little effort by adding a JD test to the pre-purchase BVD and EBL screen for bulls.

Does MAP grow in the environment?
It may survive outside the animal for a long time but clear evidence of multiplication has not been found.
It is important to remember that MAP bacteria in the environment stem from infected animals:
MAP is very resilient and may survive for many months under ideal conditions, moist, shady, cool and neutral pH environments. Unless conditions are optimal MAP numbers will decline rapidly although residual contamination may remain for long periods in favourable spots.

Pasture
It is difficult to eliminate MAP completely from pasture so the focus should be on reducing risks. Do not introduce heifers to pastures recently grazed by adults. Shared grazing with the adult herd (e.g. set-stocking calves on the home farm) or immediately after the herd will result in the highest exposure and needs to be prevented at all cost. Accurate data regarding the exposure risk (likely bacterial dose) over time are lacking, but it is reasonable to expect risks to reduce significantly after each rotation, especially in the first few weeks.

Water
Under experimental conditions, MAP has been shown to survive in water for up to 6 months or more. Provide clean drinking water (e.g. bore water) and avoid access to stagnant water, particularly if it may be contaminated by adult cattle or other stock.

Effluent
MAP will survive for long periods in faeces and the effluent pond or storage tank. Any pastures irrigated with effluent should not be used for young stock at all. Wind drift during effluent spraying may spread MAP to neighbouring paddocks and might present a significant risk to calves.

Silage or Hay
Low pH, high temperatures and drying reduce MAP survival. In one study, researchers failed to recover MAP from good silage 2 weeks after it was made.

Milk
Souring or culture of pooled milk is not effective against MAP. Good pasteurisation will kill off most MAP in the milk. On-farm pasteurisers are available but may be impractical for seasonal dairy herds with compact calving season.
Pocket to put 5 x factsheets in