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## Johne's Disease: Management of a Challenging Disease

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### ABSTRACT

In the last 20 years Johne's disease, caused by infection with *Mycobacterium paratuberculosis* (*Map*), has emerged as one of the most important bacterial diseases affecting productivity in New Zealand farmed deer. In addition to clinical losses, subclinical infection results in reduced growth rates of young deer, lower reproductive performance and lighter velvet weights. Traditionally, microbial culture of faecal material or gut tissue has been used to confirm the presence of infection. The Disease Research Laboratory has recently developed a refined ELISA test that monitors IgG1 antibody (Paralisa™) specific for antigens expressed uniquely by *M. paratuberculosis*. This test has acceptable sensitivity (75-91%) and specificity (>98%) for the detection of subclinically affected animals. The use of this test in severely infected deer herds can reduce the prevalence of reactors from high (>40%) to low levels (<5%) within 2-3 years and result in the elimination of clinical disease. Clinically detectable Johne's disease represents a minor proportion of the total number of animals infected in a herd so it is also important to have diagnostic tests that detect subclinically infected animals. Using the Paralisa™ test, it is now possible to implement management systems to electively remove infected deer to increase production and reproductive performances.

**Keywords:** Deer; Johne's Disease; immuno-diagnosis.

### INTRODUCTION

There have been reports of Johne's disease (Jd) in farmed deer in New Zealand since 1986 (de Lisle *et al.*, 1993). Jd in deer has also been diagnosed in a number of overseas countries; Belgium (Godfroid *et al.*, 2000), Czech Republic (Pavlik *et al.*, 2000), United Kingdom (Fawcett *et al.*, 1995) and USA (Manning *et al.*, 2004; Davidson *et al.*, 2004). The incidence of Johne's disease has been increasing in deer herds throughout New Zealand (de Lisle *et al.*, 2003). In deer, the process from infection to death can progress more rapidly, with animals dying from the disease from eight months of age (Mackintosh *et al.*, 2004). Older animals sporadically present with clinical Johne's disease that may be exacerbated by environmental stress or ageing (Mackintosh *et al.*, 2004). Johne's disease can be spread horizontally among adult animals (Whitlock & Buergelt, 1996) and infection may also spread vertically during pregnancy, since *Map* has been isolated from foetal tissues (van Kooten *et al.*, 2006). While most reports concentrate on clinical cases of Johne's disease, the prevalence and impact of subclinical infection has not been determined accurately. Previous results (Griffin *et al.*, 2003; Rodgers *et al.*, 2005) show that subclinically affected deer generally produce higher levels of antibody than previously reported in cattle (Collins *et al.*, 2005) or sheep (Sergeant *et al.*, 2003).

### MATERIALS AND METHODS

Blood samples were obtained from farmed deer properties throughout New Zealand, where a minimum of 10,000 animals were sampled annually. A small proportion (<10%) of the animals used in this dataset were obtained from herds considered to have significant Johne's disease problems. The majority of animals came from mobs within herds with no overt signs of disease where the farmer was attempting to establish whether the herd was infected and if so the likely level of exposure to *Map*. It should be recognised that the sample tested was not selected randomly so there may be bias in the sample relative to national prevalence rates for *Map* infection within the national deer herd.

A standard ELISA protocol originally described by Voller *et al.* (1979) and modified by Chinn *et al.* (2002) was used in this study. Native protoplasmic antigen (PpAg) from *Map* (Allied Monitor Inc, Fayette, MO, USA) was used in parallel with the standard tuberculins; PPDa obtained from *M. avium* and PPDj obtained from *Map* cultures (Lelystad, The Netherlands). An IgG<sub>1</sub> isotype antibody ELISA assay was used as earlier work (Chinn *et al.*, 2002) had established that this isotype is more sensitive than IgG-based ELISA assays for diagnosis of tuberculosis or *Map* infection (Rodgers *et al.*, 2005) in deer. 96-well micro-titre Maxisorp Immunoplates (NUNC) were

coated with 100  $\mu$ L antigen preparations (PPDa, PPDj, and PpAg) diluted in carbonate buffer (pH 9.6) to contain 50  $\mu$ g/mL. The test protocol was carried out as described in detail earlier (Griffin *et al.*, 2005). Animals were classified as test positive when reactivity in the ELISA was a minimum of 50EU with either PPDj or PpAg. The infection and disease status of animals included in the current datasets was confirmed by detailed pathological examination, histopathology of pooled gut, mucosal tissues and lymph nodes. Microbial culture was carried out using pooled gut mucosal tissues and gut associated lymph nodes.

## RESULTS

The results given in Table 1 were obtained from 9 farms from Canterbury, Otago and Southland where detailed necropsy follow-up was carried out using gross pathological examination, histopathology and microbial culture of gut tissue samples.

**Table 1:** Incidence of gross pathology and culture positive findings from seropositive deer.

Farm	No. seropositive	Gross Pathology	Culture (+)
N = 9	126	10 (8%)	120 (95%)

In an attempt to establish the incidence of *Map* infected herds, samples obtained from herds throughout New Zealand were examined. The proportion of herds tested nationally, which had at least one animal that was seropositive for *Map* is given in Table 2. As a significant number of herd submissions involved animals that were reactive to the mid cervical skin test (MCT) for Tb, there is an obvious bias in the sample because these animals had been selected as having reactivity to mycobacterial antigens. Whereas the majority of samples were obtained from South Island submissions, there is a remarkable coincidence in the proportion of herds with test positive animals in both the North and the South Island.

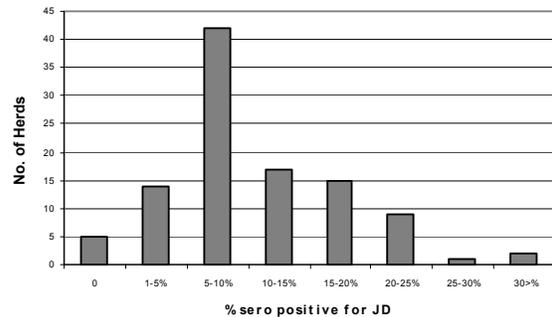
**Table 2:** Proportion of herds with one or more *Map* seropositive animal.

	No. Herds Tested	No. <i>Map</i> Seropositive	% Seroreactive
South Island	525	332	63.2
North Island	122	76	62.3

The seroprevalence rates shown in Figure 1 represent the proportion of Jd seroreactors identified in 104 herds throughout New Zealand, where a minimum of 30 animals per submission

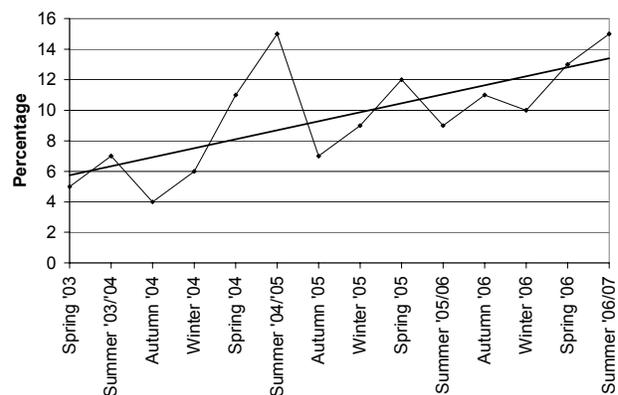
were sampled. Within the infected herds studied, a Jd seroreactivity rate > 5% was common with a small proportion of herds showing reactivity rates > 20%. Although lower numbers were tested from North Island herds there was a similar trend in the prevalence of seroreactivity (data not shown).

**Figure 1:** Jd seroprevalence rates in 104 herds with confirmed *Map* infection throughout the New Zealand.



As can be seen in Figure 2, observed Jd seroreactivity varies with season. Lower levels are evident during the autumn period but overall Jd seroreactivity rates have increased from 5 – 15% over the past four years. While seasonal trends in reactivity may be influenced by the category of animal tested, the consistent increase in the incidence of seropositivity seen over the past three years suggests that the trend for increasing *Map* infection is real.

**Figure 2:** Seasonal Prevalence of Jd seropositive animals sampled over 4 years.



While the data shown in Figure 2 was obtained from herds where samples were submitted for routine *Map* diagnosis, an equivalent trend was seen with skin test positive (MCST+) animals that were part of the Tb eradication scheme.

## DISCUSSION

While earlier estimates (de Lisle *et al.*, 2003) suggested that the herd prevalence of Jd infected herds throughout New Zealand was at least 5%, the current data suggests that more than 50% of NZ deer herds have serological evidence compatible with exposure to *Map* infection. There also appears to be an incremental increase in the prevalence of infection within infected herds, based on serological evidence obtained from sequential sampling (Griffin *et al.*, 2005). Sensitivity and specificity estimates obtained for the IgG1 ELISA for diagnosis of *Map* infection (Griffin *et al.*, 2005) suggest that the data obtained in the present study fairly represents exposure rates of NZ herds to *Map* infection. In the current study, a dataset of 126 animals that were seropositive in the ELISA, 95% were confirmed as infected by microbial culture, indicating a high correlation between seropositivity and confirmed *Map* infection.

It has been shown that there is a significant correlation between the amount of antibody and the severity of Johne's disease, and thus the culling of seropositive animals is likely to remove the most seriously affected animals that are also most likely to pass on infection to other deer. Nevertheless, a key observation from the current study is that infected animals with gross pathological evidence of disease represent less than 10% of all animals that were confirmed as *Map* infected, suggesting that subclinical infection, rather than clinical disease is the predominant presentation. This finding mirrors the belief held by many farmers who see no evidence of animal wastage or lesions at slaughter yet serological findings show evidence of infection by *Map* in apparently healthy animals. Farmers are often incredulous when informed that apparently healthy animals are seropositive and considered to be infected by *Map*. As significant clinical losses appear to occur infrequently, the major concern currently is whether subclinical infection by *Map* has any significant impact on herd performance values. Earlier studies (Rodgers *et al.*, 2005) show that significant mortality and production losses may occur in apparently healthy fawns that are seropositive at 6 months of age and in adults with no overt symptoms of Johne's disease. There is evidence that the presence of subclinical infection in adult stock impacts negatively on overall venison production (Rodgers *et al.*, 2005). Considering that subclinical *Map* infection may be present in more than 10% of animals in infected deer herds, there are major implications in the long term for the impact of subclinical infection on production within the national deer herd. Another

major consideration is that progression from subclinical infection to active disease in individual animals, not only affects their production but also results in significant environmental contamination as the animals progress from subclinical infection to scouring and wasting, resulting in persistent exposure of uninfected animals to *Map*. While environmental contamination via faecal spread is important in facilitating spread of infection between adults or from adult stock to fawns, van Kooten *et al.* (2006) have shown that pseudovertical transmission of infection from infected hinds to their fawns *in utero* may also be an additional risk factor for spread of infection within deer herds. Apart from the direct effects on production, the presence of *Map* infection with a deer herd places significant constraints on the sale and movement of live animals. Increasingly the principle of *caveat emptor* is being applied by purchasers of live animals, especially expensive stud stock, where assurances of evidence for freedom-from-infection is being sought from the vendor.

## ACKNOWLEDGEMENTS

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## APPENDIX

### A farmer's experience managing Johne's disease in deer

Dr. A.BELL

Over the last 20 years the incidence of Johne's Disease (JD) in deer herds in New Zealand has increased to a point where it is impacting significantly on the industry across all sectors. The last 2 years have seen a significant amount of progress in knowledge to control and manage JD in deer in NZ. Research has provided solid science for management tools and there are a number of communication projects focused on getting the science and information disseminated to farmers and the wider industry. For farmers, confirming their JD status and then managing the status is still critical. Every property should have a planned programme to manage Johne's Disease on-farm according to their status. Until this reaches a critical mass we will continue to battle the disease.

There has also been a significant increase in the incidence in both new infections on properties and increases in the within-herd prevalence. This is supported by data from DRL, AgResearch Wallaceville and communication with the AHB, who are dealing with diagnosis of the Tb-like

lesions post slaughter. To alter this trend we need to manage the disease more effectively.

We require continued research to investigate the causes and the different expressions of disease, to develop effective vaccines that won't interfere with Tb testing, and to refine tests to diagnose infection effectively in the slaughter plant and on-farm.

We also require support for management programs, including a JD expert network to work on programs with farmers and industry, solid communication for information flows in both directions between science and industry, and attention to various issues such as the management of lesions in slaughter plants and the use of a modified ETB test.

#### Case Study

The following discussion notes and tables follow the progress and management of JD infection on a deer property from 2002 to 2007. Properties with JD infected deer seem to follow a

similar pattern, with low infection rates in Year 1 that steadily increase to an incidence in Year 3/4 with significant losses. There are numerous variables making the disease very challenging to manage. Control programs are best carried out on an individual basis coupled with a strong cost-benefit analyses and annual reviews.

**Property Details**

This herd contained elite and commercial entities, based primarily on venison production. Its genetic base was Hungarian red deer (120 kg hinds), and had a high percentage of R2yr hinds in the herd. The Tb status was C7 (Elite) and C4 (commercial herd). Hinds and stags were drenched annually, and weaners were drenched at weaning and 3 times thereafter within the first year. Hinds received Yersiniavax and selenium pre-calving and copper at weaning.

**2002 - JD was first diagnosed**

JD was identified at Tb testing (+ve skin test, BTB +ve). Post mortem and culture confirmed *Mycobacterium paratuberculosis* in 2 yearlings in October 2002.

**2003 – Sample testing of hinds (refer Table 2**

In January 2003 are IGG1 tested 201 yearling hinds. Of these 4 were positive and 18 suspect we

culled all reactors (Table 2).

**2004 – No further testing was conducted this year**

Notification of Tb-like lesions from works increased as positive for JD. The R2 hind replacements were not tested at this point as there was a scarcity of solid information on causes, diagnosis and management options coupled with a cost analysis of testing and low venison schedules.

**2005 – A substantial number of hinds were tested following increases in ‘detains’ at the slaughter plant and a number of yearling losses on farm.**

We culled all clinical cases. This led to a further decrease in clinical cases on farm and of lesions detected at the slaughter plant (latter not necessarily a result of lower number of lesioned animals). There was a higher percentage of lesioned animals in second half of season. We tested 500 replacements hinds and 300 R3 hinds and placed 30 hinds noted as JD suspects in a hospital paddock. They were drenched and preferentially feed. Their condition improved initially. They were culled with very high JD reactivity (retested pre-slaughter).

**Table 1:** A summary of financial losses on the case farm attributable to Johne’s disease, spring 2002 to autumn 2003.

	Total animals	% affected	Number	\$/animal	Total	Comment
R1/2 on-farm losses	2200	1.5%	33	\$220	\$7260	
MA Hinds on farm losses	2500	0.5%	1	\$350	\$5250	
Detain at slaughter	2200	2%	44	\$100	\$4000	Some DSP’s now pay 90%
Post Tb testing costs	3100	2%	62	\$30	\$1860	ETB cost Reactor price
Total losses					\$18370	
Management program – basic R2 hind replacement test	200		200	\$18	\$3600	Cost to bleed R2 and test with Paralisa

**Table 2:** Results of testing of R2 hinds with the IGG1 blood test.

	Result	Deer #	Action	Lesion
Tb reactivity	EQV	4	Cull	NAD
	Positive	1	Cull	NAD
	Negative	196	Retain	
JD reactivity	Suspect	18	JD farm tagged	
	Positive	4	Cull	1
	Negative	179	Retain	

**Table 3:** A summary of financial losses to a deer farmer with medium JD prevalence, 2005.

	Total animals	% affected	Number	\$/animal	Total	Comment
R1/2 on-farm losses	850	8%	68	\$250	\$17000	
Opportunity cost			68	\$100	\$6800	
R1/2 Ill thrift	850	15%	127	\$50	\$6350	10% lighter at slaughter (10kg) \$5.00/kg
MA Hinds on farm losses	1000	1%	10	\$300	\$3000	
Opportunity cost			10	\$100	\$1000	
R2 detain at slaughter	850	2%	44	\$20	\$880	Some DSP's now pay 90%
Post Tb testing costs	1850	2%	37	\$40	\$1480	ETB cost
				\$125	\$4625	Reactor price
Total losses					\$41135	
Management program – basic	200		200	\$18	\$3600	Cost to bleed R2 and test with Paralisa
R2 hind replacement test						

Note: 1: Losses such as decreased reproductive performance are not taken into account.

2: With the increase in schedule the cost of the losses has increased significantly for this season.

3: There is also recognized a variation in the ratio of clinical losses to subclinical losses due to different farm management practices. On those properties where animals have excellent feed and minimal stresses there will be a lower ratio.

**Table 4:** Testing of hinds with Paralisa May/June 2006.

	Class	Total tested	JD reactivity Positive/suspect
<b>Bred on property</b>	MA	399	12
	R2	531	88
<b>Purchased</b>	MA	136	1
	MA	110	2

**Table 5:** A summary of financial losses to a deer farmer with medium (2%) JD prevalence, 2007.

	Total animals	% affected	Number	\$/animal	Total	Comment
R1/2 on-farm losses	850	3%	25	\$250	\$6375	
Opportunity cost			25	\$100	\$2500	
R1/2 Ill thrift	850	6%	51	\$50	\$2550	10% lighter at slaughter (10kg) \$5.00/kg
MA Hinds on farm losses	1000	0.4%	4	\$300	\$1200	
Opportunity cost			4	\$100	\$400	
R2 detain at slaughter	850	2%	17	\$20	\$340	Some DSP's now pay 90%
Post Tb testing costs	1850	0.5%	10	\$40	\$360	ETB cost
				\$125	\$1250	Reactor price (See note)
Total losses					\$14975	
Management program – basic	200		200	\$18	\$3600	Cost to bleed R2 and test with Paralisa
R2 hind replacement test						

Note: 1: The R2 detain at slaughter has not changed significantly – still at 2%. The reason for this is that there is an increase in the diagnosis of the disease in late 2006 and 2007 due to work carried out by Johne's Management Ltd contractors with meat inspectors in the DSP's.

2: Post Tb testing costs are minimal as the management plan now has all capital stock tested and all replacement R2 hinds tested annually for JD with the Paralisa blood test prior to Tb testing.

3: There will be a two year time lag from initiating a control program where all capital stock are tested to seeing the benefits in the young stock with decreased losses and increased growth rates.

### 2006 Testing of remainder of capital stock (refer Table 4)

All stock were tested, and any positives or suspects were culled

There was a substantial increase in infected R2 hinds suggesting a combination of lateral and horizontal spread. Older hinds that came from the original JD source property were all culled.

### 2007

There were substantial production increases (refer Table 5) as well as decreased losses in all classes. Weaning weights and weaner growth rates increased and there was a smaller 'tail' weaner mob (100 in 2007 compared to 300 in 2006). There was also a decrease in 'detains' at venison slaughter.

Note that the progeny weaned in 2006 came

from hinds where less than 50% had been tested for JD. We undertook an intensive rabbit control programme in the latter half of 2006 and in 2007.

### 2008

Where to from here? Replacement hinds will be tested annually. All capital stock will be tested negative for JD before purchase. We will continue wildlife control programmes. We have an expectation of further decreases in yearling losses and decrease in 'detains' to a level of 2% or below due to all capital stock having been tested. All other production parameters should also show positive movement.

### Conclusion

On farms that have not yet determined the prevalence of JD on their properties:

- Do not assume your herd is free of infection despite a lack of clinical signs, slaughter programme diagnosis or non-specific reactors to Tb testing.
- Diagnosing and managing disease when

there are low numbers of subclinical cases is the most cost effective way to deal with JD.

- There is a time lag of a minimum of two years from initial control to production gains.

A JD programme requires routine long-term attention to detail and will fail if half-hearted. The control programmes need to focus on minimizing the clinical signs of JD rather than eradication of *M. paratuberculosis*.

Farmers that have implemented control programs over recent years are now reaping the benefits:

- Significantly lower clinical losses on farm, lower detains at the slaughter plants and increased growth rates.
- Gains in reproductive efficiencies.
- Confidence shown from buyers for live sales.
- Less farmer stress in dealing with the practicalities of the disease on farm.