Johne’s disease in New Zealand livestock

by Cord Heuer, Peter Wilson and Kaylene Larking

All veterinarians and most farmers are aware of paratuberculosis, or Johne's disease (JD), a chronic progressive, incurable conditional associated with diarrhoea and wasting in ruminants.

Clinical JD occurs at a low case incidence of about 0.7% in yearling deer (Glossop et al, 2008) and <1% in dairy cows (Norton et al, 2009). JD is widespread globally (Nielsen and Toft, 2009) and has been known in New Zealand since 1912, with case reports steadily increasing since 1980 (De Lisle, 2002).

Despite intensive research worldwide, there is a lack of control methods that have long-term feasibility for either prevention or therapy. Although clinical incidence of JD is low, the causing pathogen, *Mycobacterium avium* subsp. *paratuberculosis* (MAP), is extremely widespread in normal deer (Stringer et al, 2009), cattle (Wells et al, 2009) and in the environment of MAP-infected dairy herds (Raizman et al, 2004).

In addition, there are frequent scientific and media reports of an association between MAP and Crohn’s disease (CD), a chronic, incurable bowel disease of humans with striking pathological similarity to JD of animals. There is, however, no conclusive evidence of a causal association between MAP and CD. In meat and milk exporting countries such as New Zealand there is concern about MAP as a potential public health hazard, irrespective of its causation being proved or believed by the consumer.

The distribution of MAP and the incidence of JD in cattle, sheep and deer has been subject to Massey University studies since 2008 under the umbrella of the Johne’s Disease Research Consortium (JDRC), a joint initiative of Beef + Lamb New Zealand Ltd, DairyNZ Ltd, DEEResearch Ltd, AgResearch, Massey University, Livestock Improvement Corporation, University of Otago and the Ministry of Science and Innovation. This article reports highlights of their recent findings.

Distribution of JD and MAP in New Zealand

Through the voluntary support of 28 veterinary practices in seven selected regions, 8,000 questionnaires were mailed to practice clients inquiring about observed clinical JD and production performance in all farmed livestock (dairy, beef, sheep, deer). The same practices were subsequently contracted to sample 20 randomly selected animals per species mob in 238 farms selected from the 1,940 farms (24%) that responded to the survey (Figure 1). The sample was designed to include farms with and without observed JD and, in similar proportions, farms holding either single or multiple livestock species. This was done with the intention of evaluating whether MAP was likely to be transmitted between species and whether the clinical occurrence of disease was associated with different genetic strains of MAP.

Faecal samples were sent to the AgResearch laboratory at Wallaceville for bacterial culture. In the same laboratory, MAP isolates were subjected to genotyping methods developed by JDRC/AgResearch since 2008. At New Zealand Veterinary Pathology (NZVP) and University of Otago, animals from culture negative species mobs were examined by serum ELISA for evidence of antibodies against MAP.

Conclusion

This short article cannot hope to do justice to the complexity and breadth of this topic, or to the options that are developing for the management of cancer in the veterinary patient. Hopefully the few examples presented here will whet your appetite for considering options rather than euthanasia for the oncology patient.

To discuss any of the issues raised in this article or to discuss cases, contact Jonathan Bray on oncology@massey.ac.nz or by phone on 06 350 5329.

References


Massey applied new statistical techniques, “latent class analysis”, to adjust for the sensitivity of culture and sensitivity and specificity of ELISA tests, none of which are 100 percent. Thus, more accurate estimates of flock or herd prevalence of MAP infection were obtained and subsequently weighted by sampling fraction to reproduce the distribution in the original population of 1,940 farms. The results describe, for the first time, the extent of JD and MAP infection in New Zealand livestock farms. The analysis showed that about two-thirds of sheep flocks (68%) and deer herds (60%) were infected, and there was a clear differential between islands: more sheep flocks were infected in the North Island whereas more deer herds were infected in the South Island. The prevalence in beef herds was lower (31%) with only a small difference between islands (Table 1).

Table 1: Herd/flock prevalence of MAP infection in sheep, deer and beef cattle in seven regions of North and South Islands, New Zealand (238 farms sampled, JDRC epidemiology survey 2008–10)

<table>
<thead>
<tr>
<th>Species</th>
<th>Island</th>
<th>Herds/flocks sampled</th>
<th>Prevalence</th>
<th>95% Confidence interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sheep</td>
<td>North</td>
<td>96</td>
<td>74%</td>
<td>64% – 76%</td>
</tr>
<tr>
<td></td>
<td>South</td>
<td>66</td>
<td>60%</td>
<td>48% – 82%</td>
</tr>
<tr>
<td>Beef cattle</td>
<td>North</td>
<td>86</td>
<td>33%</td>
<td>23% – 43%</td>
</tr>
<tr>
<td></td>
<td>South</td>
<td>30</td>
<td>27%</td>
<td>14% – 42%</td>
</tr>
<tr>
<td>Deer</td>
<td>North</td>
<td>41</td>
<td>42%</td>
<td>21% – 82%</td>
</tr>
<tr>
<td></td>
<td>South</td>
<td>58</td>
<td>73%</td>
<td>49% – 94%</td>
</tr>
</tbody>
</table>

The high prevalence of infected herds and flocks detected by sampling was in contrast to results collated from the postal survey where a lower proportion of farmers observed JD in their herds or flocks during the past three years. Deer (35%) and dairy farmers (22%) reported the highest occurrence of clinical JD – sheep (14%) and beef farmers (4%) to a much lesser extent (Table 2).

There are elements of bias that must be considered when interpreting farmer-observed reports of disease. Recognised factors for bias include low disease incidence, the different intensity by which farmers observe disease events in their livestock and the ability of the farmer to recognise and accurately assign the cause of disease, particularly in the absence of testing. Therefore, data derived from interviews or postal surveys must be regarded with care.

It can be concluded from the data, however, that MAP infection and herds experiencing clinical JD are common in New Zealand pastoral livestock species.

Table 2: Percentage of farmers reporting the occurrence of confirmed or suspected clinical JD during the previous three years (1,940 survey farms, JDRC epidemiology survey 2008-10)

<table>
<thead>
<tr>
<th>Species</th>
<th>No. farms</th>
<th>Confirmed JD</th>
<th>Suspected JD</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deer</td>
<td>237</td>
<td>18%</td>
<td>17%</td>
<td>35%</td>
</tr>
<tr>
<td>Dairy cattle</td>
<td>614</td>
<td>19%</td>
<td>3%</td>
<td>22%</td>
</tr>
<tr>
<td>Sheep</td>
<td>1,257</td>
<td>5%</td>
<td>9%</td>
<td>14%</td>
</tr>
<tr>
<td>Beef cattle</td>
<td>1,265</td>
<td>2%</td>
<td>2%</td>
<td>4%</td>
</tr>
</tbody>
</table>

Transmission between species

In addition to describing the distribution of MAP infection and Johnes’s disease, the survey data were stratified by the species composition of farms to evaluate whether animals of different species grazed on the same pasture (parallel or in succession) were at a higher or lower risk of MAP infection than animals grazed in isolation. This analysis provided evidence for interactions between species, either posing a risk for increased infection (inter-species transmission) or reducing infection dynamics or clinical disease incidence. It is hypothesised that mixed-species grazing could reduce JD by adding genetic diversity with respect to innate host resistance, by reducing exposure through different grazing preferences or by introducing less pathogenic strains and causing some level of acquired immunity and lower clinical disease incidence. An increased risk could arise from higher stocking density (more frequent contacts), introduction of more pathogenic strains with different host preference (eg, type I cattle strain) or greater exposure to MAP near the soil surface through grazing competition.

Results were as follows.

Sheep
Co-grazing sheep with deer did not alter the risk of MAP infection but was associated with a lower incidence of clinical JD in sheep. Contact between sheep and beef cattle increased the risk for MAP infection, but did not affect the incidence of clinical JD in sheep. However, co-grazing sheep with both cattle and deer increased clinical JD in sheep, whereas MAP infection was not affected. This suggested that farmers co-grazing sheep, deer and beef with contact among all species may be particularly prone to report clinical disease in sheep, possibly due to either farming system-specific management factors or a biased awareness.

Beef cattle
Co-grazing beef cattle with sheep increased both the MAP infection risk and the reported incidence of clinical JD in beef cattle. In contrast, co-grazing beef cattle with deer tended to reduce the infection risk and clinical disease in beef cattle.
Contact with beef cattle increased the risk of infection and occurrence of clinical JD in deer. Co-grazing deer with sheep did not alter the infection risk, but reduced the rate of clinical disease in deer. Similar results were reported from an earlier case-control study of deer herds (Glossop et al, 2008).

As a general trend, deer benefitted from co-grazing with sheep but experienced higher infection and clinical disease rates when in contact with beef cattle. Beef cattle also increased the risk for sheep, and were themselves at higher risk when in contact with sheep when grazed in isolation. But both beef cattle and sheep benefitted from the presence of deer. The results suggest that MAP is often transmitted between livestock species through the contamination of the environment, especially pasture. Strain typing results will be available during 2011 and are expected to add further understanding to the observed transmission patterns.

New questions
This set of data coupled with our existing understanding now raises important questions for the research community.

- If infection is widespread, why do only a small proportion of animals appear to develop clinical symptoms? Hence, what are the determinants of disease in infected animals (other than age at infection and infectious dose)?
- How important are “super-shedders” for the propagation of infection within and between herds or flocks?
- Are there any detectable virulence differences between the major MAP strains in our animal populations?
- Can resistance or susceptibility to MAP be measured?
- Are infected but clinically normal animals likely to be less productive?
- Can we identify infected animals and predict that they are likely to develop clinical disease and/or to start shedding a large amount of MAP and, thus, propagating MAP through herds and flocks?

These are core questions for JDRC science providers in the next period of JDRC research.

New opportunities for JD control
Gene markers may become available to identify resistant dams and sires for selective breeding. To overcome the long incubation period required for bacterial culture of faecal samples, modified available diagnostic tests are being evaluated for their ability to predict the onset of shedding and clinical disease well into the future. The removal of super-shedders has been advocated overseas as an effective way to achieve a major reduction in new infections while keeping the amount of culling, and thus the cost of control, at a minimum. Research is required to show whether such interventions are effective in New Zealand’s ruminant livestock species. Vaccines are known to reduce the incidence of clinical JD but have little effect on shedding.

References


