Study confirms genetic link to Johne's disease resistance

Preliminary results from a longitudinal study led by Colin Mackintosh, AgResearch Invermay, and funded by the Johne's Disease Research Consortium (JDRC), have indicated that resistance or susceptibility to Johne's disease appears to be highly heritable. The results were presented to at the New Zealand Veterinary Association Deer Branch's* Cervetec 2010 *conference in Queenstown in early June.

As well as noting the strong genetic link to heritability, the study showed that at least one of the animals, sired by a Johne's-resistant stag, appeared to have completely cured itself of mild Johne's disease-related lesions within nine months of becoming infected with the disease.

The study was designed to isolate the impact of the sires' genotype, by controlling husbandry-related variables. The deer studied were of the same age, born into the same mob, run together and given the same animal health treatments. At four months of age, they were challenged with the bacteria that cause Johne's disease – *Mycobacterium avium* subspecies *paratuberculosis* (MAP).

The JDRC study has followed a collaboration between the Otago University Disease Research Laboratory and the Peel Forest Estate deer stud, where the stud's extensive records for its bloodlines has allowed researchers to identify animals thought to have a high level of heritability for resistance (R) or susceptibility (S) to natural MAP challenge. The stags are identified as R or S on the basis of their offspring's response to natural challenge from the disease.

Semen from two Peel Forest-bred red stags, one R and one S stag, were used to artificially inseminate 24 randomly selected red hinds. The hinds were from a property with no history of clinical Johne's disease which had all tested negative for the disease. Eighteen calves were sired, nine from each stag. There was also a wapiti-cross calf, sired by a chaser stag. The offspring were run together and, at four months of age, challenged with an oral dose of the virulent bovine strain of MAP.

Throughout the 49-week trial, the animals were grazed together and lymph node biopsies taken at four and 13 weeks following the disease challenge. Histopathology of these samples, and a range of other diagnostic techniques, were used to assess their response to the disease challenge.

Five animals died during the trial. One died following biopsy surgery and the other four animals were euthanised after developing clinical cases of Johne's disease between 18–25 weeks after disease challenge. Two of these were S animals, one R and the last the wapiti-cross. The animals showed typical signs of the disease, such as rapid weight loss and scouring.

At week four of the study, two S animals had mild lesions detectable in their lymph nodes. By week 13, all 18 animals on the trial had lesions, ranging from mild to severe.

By week 49, when all of the remaining animals were slaughtered, the disease pattern was distinct, with animals falling into two groups: eight animals (seven R and one S) with no, or very mild disease and six animals (one R and five S) with severe disease. The results are preliminary but strongly suggest that the genetic impact on disease resistance or susceptibility was showing, with the stag R or S status dominating the disease outcome for the offspring. It was noted that each sire had offspring with disease outcomes at the opposite end of the spectrum with the R sire having two severely affected offspring, and the S sire one offspring that was only mildly affected by the disease. These "outliers" were not unexpected as the stags were bred across randomly selected hinds with unknown Johne's resistance status.

The authors said the only common variable not controlled was gender of the offspring, but that had no significant effect on the outcome.

It was noteworthy that as well as one R animal appearing to have completely self "cured", almost all the R offspring showed a lower lesion score at slaughter than at week 13 of the trial. This indicated that "cure" was occurring at varying degrees across all the mildly affected R offspring.

There seemed to be a strong relationship between low antibody response and resistance to the disease, with four of the R animals remaining Paralisa™ negative throughout.

The authors noted that this strong genetic impact on Johne's resistance would be harder to see on a commercial farm and outside the setting of a controlled trial, because other factors such as timing and degree of challenge would come into play. But there appears to be little doubt that resistance to this devastating disease is likely to be highly heritable, which should be of great excitement to the industry.

Lymph nodes collected during the study are being used in further investigations of gene expression to determine the underlying genetic basis for Johne's disease resistance and susceptibility.

Reference

C Mackintosh, B Tolentino, G de Lisle, G Clark, S Liggett, F Griffin (2010) Longitudinal study of resistant or susceptible red deer to challenge with *Mycobacterium avium* subspecies *paratuberculosis* – preliminary results. Proceedings of NZVA Deer Branch Conference, 2010 (pub. pending).

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