Tuberculosis- Case Reports.

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INTRODUCTION:

In 1989 I was presented with several large deer herd breakdowns in herds previously accredited as being "tb free".

Four years on we are confident that the problems are now behind us with two of the herds re-accredited and the third off movement control and close to being re-accredited.

This paper covers some of the details surrounding these outbreaks and the the methods adopted to control and finally eradicate infection in these herds. Time will not allow a detailed examination of all the testing proceedures and results and therefore I will only be able to present this in summarised form.

HERD ONE:

Large "run" property located in the head waters of one of the Canterbury river valleys. Running approximately 1500 red deer, 18,000 sheep and 500 cattle.

Commenced deer farming in 1973, all deer were captured on the property and NO deer had been introduced. That was until 1986, when a single imported German stag was purchased and held in isolation on a seperate property for a further 18 months prior to being bought into the main herd for sire duties.

Deer are extensively managed at approximately 1 deer to the acre in large native oversown tussocck blocks of up to 500 acres in a single paddock.

This property had been long regarded as a very "safe" property to purchase deer from because of the "closed herd" policy of not buying in deer plus the geograpic isolation with no neighbours.

The herd commenced whole herd tuberculin testing in 1985 and was subsequently accredited in October 1987.

The herd tb breakdown was identified as a consequence of another herd breakdown in a North Canterbury property which had purchased a sire stag from herd one in March 1988.

In October 1988 this stag was positive to the standard skin test on the North Canterbury property, and was retested in March 1989 using the comparitive cervical test to which it was postive.

This same North Canterbury property purchased a further three sire stags in March 1989 from herd one and two out of three of these were positive to a standard skin test applied as a post-sale replacement test. In April 1989, all three were BTB tested and two were positive. In May 1989 they were necropsied and all three had visible lesions of tuberculosis.

Following receipt of this information, on the 20th of May 1989, I carried out the first part of a whole herd test on herd one. 513 deer tested with 21 reactors. One of the reactors, a six month old fawn was necropsied in the field and lesions consistent with a diagnosis of tuberculosis were seen in a popliteal lymph node.

The balance of the herd (770) were tested with a further 108 reactors being identified.

At this stage we realised we had a major tuberculosis breakdown and we set about identifying the cause of the infection and to eradicate the disease from the herd.

HERD TWO:

A Canterbury foothill property of 8500 acres running approximately 4,000 red deer, 7000 sheep and 600 cattle.

Commenced deer farming in 1978 with all but a small number of deer helicopter captured in the surrounding river valleys and mountains, care was taken to capture in areas with no history of tuberculosis in feral populations.

The small number of purchased deer were imported European stags and hinds, which were managed as a seperate unit.

The property had started whole herd tuberculin testing in 1983 and was accredited in August 1987.

In May 1989 the herd breakdown was identified as a result of a lesion being noted in a single bullock slaughtered after being finished on the property. This bullock was a hand reared dairy calf and had been grazing over areas where the deer were farmed.

During June 1989, we completed a whole herd check test with 3744 deer being tested, 52 reactors and on slaughter 28 of these had visible lesions consistent with tuberculosis.

Clearly, another major breakdown in a large deer herd.

HERD THREE:

This property located in the foothills of the Fairlie basin, had only recently commenced deer farming running approximately

200 red deer, 5000 sheep and 50 cattle.

They had carried out their first whole herd test in March 1985 and had been accredited in July 1987.

The bulk of this herd had been purchased from herd one, the latest being sire stags in 1988 and 1989, hence the reason for a check test.

In July 1989 we carried out a whole herd check test with 203 deer being tested, 46 reactors and 35 of these showed visible lesions of tuberculosis at slaughter.

Given the size of the herd this was again a very significant herd breakdown, at least in this case we knew where the source of infection came from.

ACTION PLAN:

Given the extent of the herd breakdowns and the widespread location from each other (we were also dealing with other herd breakdowns associated with purchase of deer from herd one), there were a number of questions being asked and not too many answers forthcoming!

The basis for some action was centered around a series of meetings which involved many of the interested parties.

1. MAF, local Office, Laboratory Lincoln and Wallaceville, Head Office and other regional Offices.

2. Pest Boards, to assist with local possum surveys and control operations.

3. Otago University, namely Dr. Frank Griffin and his team were consulted and their various testing proceedures were used extensively during the early stages of the control programme.

4. Fellow Veterinary Surgeons from deer practice were involved and provided valuable information and support during the difficult early stages of the testing programme.

5. Deer Farmers were involved from the start and their input and contribution was invaluable.

The outcome of all of these discussions was that the three property owners wished to test their deer clear of tuberculosis. The advice at the time was that it was not going to be easy but we would try and implement a control programme with the ultimate aim of eradicating the disease from these herds.

This task required 100% cooperation from the farmers and their staff, 110% effort from the testing Officer and 100%

cooperation from the local DSP (Fortex) and Animal Health Laboratories both Lincoln and Wallaceville.

The action plan involved a number of important steps:

1. Determine the source of infection:

Without the knowlege of where the infection had originated there is little chance of controlling a disease situation, particularly if you are going to encounter a re-infection problem.

Herd one, with it's geographical isolation posed a few problems and the only real source following detailed investigation was the imported German stag.

Herd two, was located adjacent to an area previously known to have had Tb infected possums (last identified in 1975), but still possibly a source (this was subsequently proven to be wrong after further typing of the tb organism).

Herd three, the source was obvious (herd one) and later proven using typing techniques developed by Wallaceville.

2. Maintain groups:

It was demonstrated early in the investigation that the reactors were concentrated in discrete groups within each of the herds. Special efforts were made to ensure these "groups" were kept isolated and remained together where possible.

There was considerable evidence that some "groups" on each of the involved properties had no infection established and every was made to maintain this status.

3. Infectious versus Infected status:

Clearly, the "infectious" deer is the most likely candidate to spread infection to other deer, probably via direct contact.

Every effort was made to prevent deer from becoming "infectious". This involved a number of measures:

3.1 Shorten the testing interval down to 60 days in some instances.

3.2 Target again the groups with known infected status.

3.3 Test the herd regularly throughout the whole year. There are times of the year when for management reasons deer cannot be yarded, by careful planning these can be minimised. We also carried out tests on stags in the rutting period (quite exciting!) and hinds with fawns at foot during the summer months.

3.4 Use of "other" tests, this included the use of the Elisa blood test as applied by the Otago University Deer Laboratory.

4. Management:

Carefull attention to management and the grazing of different "groups" was undertaken. In particular, the detailed recording of tag numbers and management of "groups" of deer.

Attention to fences to prevent "trading" between paddocks.

Grazing of the "high" risk groups in the most isolated area on the property and the handling of these deer last or separate from the less "at-risk" groups.

Improve shed hygiene to include regular cleaning and disinfecting, attend to drainage and effluent & carcase disposal.

RESULTS:

While it is still early days with all of these herds, there have been some very encouraging results as evidenced by both regular skin testing results and following animals which have been slaughtered at the DSP's.

The following is the latest situation with regard to the testing status of the herds in question.

HERD ONE:

In March 1991, the last lesion reactor was identified following the completion of the sixth herd retest.

Two whole herd tests "clear" of tb lesion reactors followed.

March 1992, in one "high" risk group we identified 36 reactors in a group of 77 rising 2 year hinds. 20 of these had lesions at slaughter and the balance of the group was slaughtered. This group had been separately managed in complete isolation for their entire life on the property and we are confident that infection had little or no direct opportunity to spread to the rest of the herd (subsequent testing and slaughter has confirmed this).

Three further whole herd retests carried out in June 92, October 92 and July 1993 have all been clear of lesion reactors. In addition, in excess if 300 stags and hinds have been slaughtered at the local DSP and no visible lesions of tuberculosis have been identified.

During the testing programme we have carried out 13 whole herd retests on this herd involving 15858 deer tested, 456 reactors

have been identified and 295 of these had visible lesions at slaughter.

The source of the infection we feel almost certain to be the one imported German stag, this is supported by Wallaceville's typing of the strain to be unique in New Zealand. This is particularly disappointing following the number of "clear" skin tests and blood tests carried out on this stag during his export/import process, plus the on farm quarantine applied. This stag, finally reacted to a skin test carried out in December 1989, on post mortem he was GTB.

This herd which was undoubtedly the most seriously infected of the three when we started, has shown remarkable progress towards eventual eradication and we are confident that re-accreditation is not far away.

HERD TWO:

Following two whole herd tests, no further lesion reactors have been identified. A further four whole herd clear tests resulted in this herd being re-accredited in August 1992.

A further whole herd retest carried out July 1993 has also been clear.

The testing programme has extended over eight whole herd tests involving 29,891 deer, resulting in 181 reactors being identified with 30 of these showing visible lesions of tuberculosis.

The infection source in this herd was shown to be associated with infection having been bought onto the property through their "Safari Park" where a number of Fallow bucks were purchased from South Kaiapara Head area and not tested. This has been confirmed by Wallaceville typing the strain of Mycobacteria.

This herd is annually slaughtering 1000 plus deer clear of any tb lesions and this coupled with the continuing clear testing status has shown encouraging progress.

HERD THREE:

Due to it's size this herd has been somewhat easier to manage and perhaps clear of infection. Given the fact that almost 25% reactors were identified at the initial test this is still a major achievement.

Lesion reactors were cleared from the herd after the second whole herd retest. A further five whole herd clear tests followed with the herd being re-accredited in March 1992.

A further retest in March 1993 was also clear.

During the testing programme a total of 1161 deer were tested over eight whole herd tests, 84 reators were identified and 44 of these had visible lesions of tuberculosis.

We are confident that infection has been cleared from this herd.

SUMMARY:

1. Tuberculosis can be eradiacted from severely infected deer herds.

2. The programme required to achieve this result needs to be specifically tailored for the property involved and needs 100% commitment from all parties.

3. Carefull attention to details and record keeping is essential if success is to be found.

4. Regard the discovery of tb in a deer herd as a challenge and not a disaster, all vets are encouraged to try their best to solve the problem and not to give-up.

Too often the "soft" option of herd slaughter is taken without giving it a go.

5. Look to your Veterinary colleagues for their support and guidance if you are presented with a problem, this should include not only the MAF but also other practitioners.

ACKNOWLEGEMENTS:

I must thank my clients involved in this programme for their continued faith and support despite the enormity of the task in front of us. The MAF Officers, located at Timaru, Lincoln, Wallaceville, Head Office, Invermay and various other locations throughout New Zealand. My fellow colleagues whose advice and support was comforting at times when I felt a little vunerable, special mention to Drs John Hunter and Ian Walker. The Otago University Deer Laboratory Staff who provided much time and special effort to investigate the various problems we were presented with, sometimes at very short notice.