# Uncontrollable Spread of Tb within a Deer Herd

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## Introduction

During the initial stages of development of deer farming in the 1980's, the spectre of tuberculosis and its spread within deer herds was the central concern of New Zealand's Deer Farmers. Deer herds were being set up using stock from multiple sources involving combinations of farm raised and helicopter captured deer. In the late 1970's uncontrolled spread of Tb was first observed in a herd of farmed deer (Beatson, 1985). The conventional bovine tuberculosis skin test was adapted for deer and used to diagnose Tb in this herd. Continuing spread of Tb within the herd over a two year period resulted in total depopulation.

While there has been a preception worldwide that farmed deer are uniquely susceptible to tuberculosis, this has not been borne out in the National statistics for tuberculosis in new Zealand's deer. Over the past two decades there has less than one New Zealand deer farm *per annum* depopulated, because of uncontrollable Tb. Most infected herds have less than 1% stock involved and disease is usually eliminated after a series of whole herd tests, carried out at three monthly intervals. Tb infected deer farms in New Zealand have been reduced from more than 400 in 1993 to around 200 in 1998. The herd history reported here was dramatic not only because of the rate of spread of disease but due to the fact that the disease could not be effectively contained despite using an extensive and rigorous testing protocol with radical culling of any suspect stock.

## Description of outbreak

The herd was first set up in 1990 (Table 1) and had increased in size to a 590 head breeding herd by 1995. Stock was purchased from herds considered to be free from tuberculosis.

**Table 1: Herd Buildup 1990- 1995.**

| | Acquisition Females | Males | Accredited |
|---|---|---|---|
| 1990 | 60 | 0 | 3 |
| 1991 | 76 | (1 Hired Stag) | 3 |
| 1992 | 15 | 1 Stag (1 Hired Stag) | 3 |
| 1993 | - | 1 Stag (1 Hired Stag) | 3 |
| 1994 | - | 1 Stag | 3 |
| 1995 | - | 21 Stags | 3 |

The tuberculosis testing history is as given in Table 2. Whole herd mid cervical skin testing (MCST) was carried out annually in 1992, 1993 and 1994. Apart from two MCST reactors in 1993, no reactions were found. The MCST(+) animals identified in 1993 were rechecked with a comparative cervical test (CCT) and cleared as non-specific reactors. By contrast, the whole herd skin test, carried out in August 1995 identified a significant numbers of MCST(+) reactors (>2%) for the first time in the herd's history.

**Table 2:Tb Testing History 1992-1996**

| Date | No. Tested | Age and Sex | Type of Test | No. of Reactors | Slaughter Outcome |
|---|---|---|---|---|---|
| 1992 | WHT | All | MCST | 0 | |
| 1993 | WHT | All | MCST | 2 | |
| | 2 MCST(+) | | CCT | 0 | |
| 1994 | WHT | All | MCST | 0 | |
| 1995 Aug | WHT | All | MCST | 22* | |
| 1995 Nov | 22 MCST(+) | MAH, MAS*, 2yoS, 2yoH | CCT | 19 | |
| 1995 Dec | 19 CCT(+) | | | | 6Tb Lesions |
| 1996 Feb | PHT | Yearlings, MAS | MCST | 28 | |
| | 28 MCST(+) | | BTB | 25 | |
| | 116 MCST(-) | | ELISA | 3 | |
| | 28 BTB(+) or ELISA(+) | | | | 22 Tb Lesions |
| 1996 March | PHT | MAH | MCST | 108 | |
| | 108 MCST(+) | | ELISA | 67 | |
| | 108 | | | 108 | 64 Tb Lesions |
| 1996 April | PHT (160) | Weaners | MCST | 23 | 5 Tb Lesions |
| | 137 MCST(-) | | ELISA | 8 | 5 Tb Lesions |
| 1996 June | PHT (75) | MAS | ST | 6 | 1Tb Lesion |
| | 118 MCST(-ve) | MAH | ELISA | 36 | |
| | 118 | All slaughtered | | | 24 Tb Lesions |

*2yoH - 2 year old Hind; 2yoS – 2 year old Stag; MAH – Mixed Age Hind;
MAS – Mixed Age Stag; AS – Adult Stag; WHT – Whole Herd Test; PHT – Partial Herd Test.

Based on the previous test history the risk from tuberculosis was considered to be low so it was decided to retest the 22 MCST(+) animals by CCT after a 90 day period. The results of the CCT showed that 19 were CCT(+) and risk from Tb was considered to be high so the animals were slaughtered. On *post mortem* 6 animals had lesions typical of tuberculosis on histology and 13 had no visible lesions (NVLs). From this time the herd was considered to be infected and there was special concerns that reactors were found both in different age groups and sexes.

A partial herd MCST was carried out in February 1996 on 143 yearling hinds, yearling and mixed aged stags. MCST(+) reactions were identified in 4/50 yearling hinds , 2/50 yearling

stags and 22/43 mixed aged stags. The 28 MCST(+) animals were tested by BTB (Griffin *et al* 1994) and 25/28 were positive. A parallel ELISA test was carried out on the 117 MCST(-) animals and 3 were ELISA(+). The 25 MCST(+)/BTB(+) animals and the 3 MCST(-)/ELISA(+) were slaughtered and 22/28 had lesions typical of tuberculosis.

By March 1996 it was established that the affected herd was at high risk from actively spreading tuberculosis, especially in the adult stock. Based on *post mortem* findings and ancillary BTB blood testing, it was clear that there was no major issue of non-specific false(+) MCST reactivity in this herd. The high prevalence of active Tb lesions within the MCST(+) mixed aged stags suggested that there was rapidly spreading disease among the males, at a rate similar to that found in the females earlier. The low prevalence of ELISA(+) reactions in the MCST (-) animals inferred that there was no major concern from residual chronically infected MCST(-) *'anergic'* animals.

In March 1996, 179 mixed aged hinds were tested by MCST and 108 were identified as reactors. These were tested by a confirmatory ELISA and 67/108 were positive, indicating high risk of active disease among the MCST(+) adult females. Slaughter of the 108 MCST(+) animals showed 64 with lesions compatible with tuberculosis. Sixteen of the lesioned animals were condemned, mostly because of involvement of carcase lymph nodes.

A MCST test, carried out in April 1996 on 160 mixed sex weaners, produced 23 MCST(+) reactors. The residual 137 MCST(-) animals were tested by ELISA and 8/137 were ELISA(+). On slaughter, 5/23 MCST(+) animals had Tb lesions and 5/8 ELISA(+) deer had lesions, two of the ELISA(+) animals were condemned.

The adult stags (75) were submitted for MCST in June 1996 and 6/75 were MCST(+). The extent of Tb and evidence for continuing spread suggested that it would be difficult to salvage an acceptable number of adult animals in any management programme. Slaughter of the 6 MCST(+) showed only one with lesions. It was decided to cull the adult hinds (118) at this time and an ELISA test was carried out to evaluate the overall level of risk in these animals prior to slaughter. The ELISA identified 36/118 animals as positive and when the hinds were slaughered 24/118 were diagnosed as lesion positive.

As infection rates were lower in the weaners it was decided to carry out repeated BTB tests on the remaining 122 weaners to determine if salvage was feasible. The animals were placed in three categories based on the lymphocyte transformation (LT) and ELISA test results. Data given in Table 3 shows that 48/122 weaners were considered to be at high risk because they were LT(+)/ELISA(+). These animals were slaughtered and 39/48 had Tb lesions, with a significant number showing lesions in body nodes (prescapular/popliteal).

**Table 3: Repeat BTB test carried out on 122 weaners**

| | Group I LT(-)/ELISA(-) | Group II LT(+)/ELISA(-) | Group III LT(+)/ELISA(+) |
|---|---|---|---|
| August 1996 | 38 | 36 | 48* |
| September 1996 | 27 | 35 | 12 |

* Slaughtered in August 1996: 39/48 with Tb Lesions.

Group II animals classified as moderate risk [LT(+)/ELISA(-)] and Group I; classified as low risk [LT(-)/ELISA(-)), were retained and retested by ELISA one month later. At the repeat test 9/36 animals from Group II had to be reclassified as Group III (LT(+)/ELISA(+)] while 8/38 Group I animals had converted to Group II [LT(+)/ELISA(-)] status and 3/38 had become LT(+)/ELISA(+) requiring that they be reclassified as Group III. The increasing spread and severity of disease seen in the weaners over over the one month interval suggested that salvage of Tb free young stock would not be cost-effective. All the remaining weaners (71) were slaughtered in November 1996 and 11 had Tb lesions.

The residual mixed aged stags (68) were tested with BTB in September 1996 and 15 were BTB(+). All stags were slaughtered in November 1996 and 10/68 had Tb lesions. Though this group shared the lowest prevalence of Tb in any of the cohorts tested within the herd, the prevalence of infection was still too high to justify salvage.

## Discussion

A retrospective analysis of this herd history highlights how Tb infection in a deer herd may spread over a short time period. In spite of the extensive surveillance and testing in this herd it was not possible to accurately identify the source of infection or the time that Tb infection first entered the herd. Apart from the adult stags all other groups of deer within the herd showed massive spread of Tb over a one year period. The skeptic might suggest that this is a classical demonstration of the unique susceptibility of farmed deer to bovine Tb.

Our experience with hundreds of Tb infected deer herds, studied over a decade, plus the New Zealand Deer Industry statistics in general, suggest that the pattern of Tb spread in the above herd was quite exceptional. Healthy well managed farmed deer do not appear to be more susceptible to Tb than dairy cattle. The more likely explanation is that phenotypic stressors associated with the management of the deer, rather than any intrinsic genotypic susceptibility, may have have precipitated the rampant spread of tuberculosis within this herd.The typical stressors that influence disease resistance include nutritional deprivation, adverse climate and pregnancy.

Close scrutiny of the records and ongoing herd visits confirmed that the herd showed no evidence of nutritional deprivation or sub-optimal management. The factors which precipitated the spread of Tb within the herd probably relate to adverse climatic conditions over the 1995 winter period and the fact that Tb was present, but as yet undiagnosed, in the pregnant hinds during the winter of 1995. One could only speculate as to whether hinds became infected through contact with an infected stag during the 1995 rutting season, or that the reverse may have occurred. It is possible that an infected hind may have been excreting Tb and caused transmission of infection to a stag during the 1995 mating season, and subsequently caused Tb infection to spread within the breeding hinds. It is recognised that fawns born to infected hinds are uniquely at risk from infection, so it is probable that Tb from the infected hinds spread to the fawns born in 1995. Overall the most likely explanation for the dynamics of Tb spread is that infection became established in the hinds early in the breeding cycle and subsequently spread uncontrollably within adults that were phenotypically

susceptible due to breeding (rutting or pregnancy) and climatic stressors. Spread from infected hinds to fawns could be the result of immunological naivety in immature neonates exposed to infectious *M. bovis*

## Conclusion

The above case history provides compelling evidence that undiagnosed tuberculosis within a deer herd can have catastrophic effects should nature's confounding variables come together in concert. While this occurs only very rarely, its consequences are devastating for the farmer, debilitating for the veterinarian and humbling for the scientist. At worst it provides compelling evidence that this disease refuses to disclose the secrets that would allow us mortals to contain its ravages.

### References

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