EXPERIENCES WITH LEPTOSPIROSIS IN NEW ZEALAND FARMED RED DEER (*Cervus elaphus*)

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Introduction

This paper gives a brief overview of two outbreaks of leptospirosis in red deer. Leptospirosis occurred on a Rotorua farm during 1981 and on a Southland farm in 1982 and 1983. For detailed information on the Rotorua outbreak the reader should consult the clinical communication by Fairley et al(2). Details of the Southland outbreak are to be published in full elsewhere. These two outbreaks were associated with deaths in young red deer calves. The occurrence of a case of suspected *Leptospira interrogans* serovar *pomona* infection in an adult deer has been recorded by Ruakura Animal Health Laboratory(1). Table 1 gives details from the two outbreaks in summary form.

A review of leptospirosis in deer is provided elsewhere in these proceedings.

Clinical history and laboratory findings

A: Rotorua outbreak

Four calves were found dead from a mob of 70 hinds and 70 calves. No illness was noticed prior to death. Calves were 3-4 months of age and died over a three week period in April and May 1981.

At necropsy the four calves showed enlarged, pale kidneys (2-3 x normal). Microscopically there was a severe chronic-active nephritis and death was probably due to renal failure.

Serological investigations revealed variable, mainly high MAT titres to *pomona* (range 1:100 to 1:12,800) in incontact calves. One calf had a 1:100 titre to *hardjo*. Despite low mortality serological evidence demonstrated that infection was widespread (Table 1).

Five of these calves were chosen for urine sampling in order to confirm the serological identity of the serovars by cultural examination. Calves were given 100-125 mg of frusemide as a diuretic to stimulate fresh urine production. A few drops of urine were transferred to a leptospiral culture medium. Four of these calves yielded *pomona*, and *hardjo* was recovered from the calf which was seropositive to this serovar(2).

B. Southland outbreak

Three calves were found dead on this farm. A neonate died in December 1982 and two 7 month old calves from a different mob died in June 1983 about one month after having been brought to the farm. These calves had severe jaundice with an underlying pallor of the carcase and watery blood. Urine when present in the bladder was tinged red. Their livers were enlarged and yellow with rounded margins. Leptospires were demonstrable histologically in silver stained sections of liver or kidney.

The majority of incontact calves had rising MAT titres to *pomona* (range 1:24-1:768) and *pomona* was recovered from the urine of a selection of these individuals. One calf had a MAT titre of 1:24 to *hardjo* and *hardjo* was recovered from this calf's urine, however it was seronegative at the time of urine sampling. The use of diuresis and transport media was used as described for the Rotorua calves.

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Control

In both herds whole-herd vaccination was carried out. The manufacturers recommendations for cattle were followed ie. two innoculations at least four weeks apart and yearly boosters.

Discussion

The existence of widespread infection with low mortality is perhaps not unexpected and is a common feature of *pomona* infections.

The high percentage of seropositive calves prior to vaccination suggests vaccination may have been of little immediate value for the calves. It did not stop leptospiruria in those with renal infection(2).

On both farms vaccination was carried out after the first bleeding and before the second bleeding. Titres to *pomona* rose in most calves in the Southland outbreak and so interpretation of rising titres is difficult. Despite vaccination on the Rotorua farm titres did not change significantly(2).

Leptospires were not recovered from any of the dead calves, although they were demonstrated histologically by silver staining in the Southland calves. The diagnosis of leptospirosis in the Rotorua calves was by association of a nephritis with serological and cultural evidence of infection in incontacts.

The source of infection was not clearly established in either outbreak but young 15-18 month old dairy bulls were suspected to be the source in the Rotorua outbreak. They were rotationally grazed 2-6 days ahead of the hinds and their calves. Approximately 50% of the bulls bled had MAT titres of 1:200 or greater to *pomona* or *hardjo*.

In a closed herd situation (including no cattle introductions or associated pig farming) it is likely that *pomona* infection in the herd would eventually be self limiting. By analogy to the epidemiology of leptospirosis in other animals(3), in order to maintain the infection and infect succeeding generations deer would need to be leptospiruric for at least 12 months. Studies on the Rotorua deer suggest that leptospiruria in excess of 12 months would be unlikely(2), however experimental studies would be necessary to confirm or refute these conclusions.

* Leptavoid (Wellcome N.Z. Ltd.)

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References

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(3) Hathaway, S.C. (1981); Leptospirosis in New Zealand: an ecological view. N.Z. vet. J. 29: 109-12.
(4) Marshall, R.B.; Wilton, B.E.; Robinson, A.J. (1981); Identification of leptospiral serovars by restriction - endonuclease analysis. J. med. Micro. 14: 163-6.

TABLE 1: Summary data from two outbreaks of leptospirosis in red deer calves

Outbreak	No. of calves	No. died	Ages	History	Time of year	Number seropositive *pomona* / Number incontacts bled	% seropositive *pomona*	Number urine culture +ve *pomona* / number incontacts cultured	Number urine culture +ve *hardjo* / number incontacts cultured
Rotorua 1981	70	4	3-4 months	Found dead	April/May	9/12	75	4/5	1/5
Southland 1982-83	>100 accurate figures not available	3	Neonate x1 7 mths x2	Found dead	Dec. '82 (neonate) June '83 (7 mth)	10/16	62	5/6	1/6

Leptospiral isolates identified by Dr R.B. Marshall, Massey University(4).