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Has Rabbit Haemorrhagic Disease worked in New Zealand?

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ABSTRACT

This paper reports on some results of a field study of Rabbit Haemorrhagic Disease (RHD) since its illegal release in New Zealand in August 1997. The presence of fresh carcasses and seropositive rabbits of different ages were used to indicate the presence of RHD in rabbit populations, and changes in rabbit densities were used to infer its efficacy.

Serological evidence from two Otago study sites where rabbits were sampled before and five times after the initial epidemic shows that (a) biociding left more seropositive, and so immunised, adult survivors than were left after a natural epidemic; thus natural epidemics are best if they persist, (b) the proportion of seropositive rabbits in cross-sectional samples declined over the 6 months after the initial epidemic, and (c) a second epidemic of RHD broke out naturally at both sites in September 1998.

At 36 study sites around New Zealand, the initial 1997 epidemics caused declines in rabbit densities averaging 32±23 % (95% S.E.) in the North Island and 56±23% in the South Island. However, success was not universal and rabbits increased at a few sites. Causes of failure at some sites have been attributed to poor-quality biociding where rabbits were vaccinated rather than infected. Variable kills were also seen in natural epidemics at sites where the initial rabbit densities were less than c. 25 rabbits seen /spotlight km, while consistently high kills were evident at intial densities above this threshold. Overall, rabbit numbers increased at most sites measured between winter 1998 and early 1999, despite second epidemics being confirmed at some of these sites.

The optimal strategy to manage rabbits as pests of pastoral production in New Zealand cannot be determined because we cannot yet predict the behaviour of RHD, and we have insufficient information on the relationships between rabbit densities and their impacts on production, although a single unreplicated trial we did in Otago showed that the reduction in rabbit numbers due to RHD reduced the grazing impacts of rabbits by 77%. Therefore, in the absence of any robust measure of benefits we cannot judge how much it is worth spending on conventional control. These information gaps are hindered by the old paradigm of rabbit control – "knock them down and hold them down". If this strategy was ever the best for a boom-bust pest, it is certainly not so now that a biological control agent is at least partially successful and persistent.

Keywords: Rabbits; *Oryctolagus cuniculus*; biological control; Rabbit Haemorrhagic Disease; RHD.

INTRODUCTION

Rabbit Haemorrhagic Disease (RHD) virus was illegally released in New Zealand in August 1997 and subsequently spread by a variety of baiting methods and by natural means over most of New Zealand (Parkes *et al.,* 1999). The motivation for introducing a biological control agent to control rabbits (*Oryctolagus cuniculus*) in New Zealand was simple (Williams, 1998). Changes in government funding policies had shifted the costs of rabbit control from taxpayers and ratepayers largely onto landowners. Many landowners could not earn enough from the land to pay for rabbit control, yet control was often enforceable (and enforced) under Regional Pest Management Strategies.

The introduction of RHD also needs to be placed within the wider context of the changing management of rabbits in New Zealand. Past management often confused means (killing the pest) and ends (protecting a resource). This meant the focus of managers and researchers, was usually tactical, i.e., focussed on the rabbit and how to kill it. Control strategies have been based on sustaining a low density of rabbits, a species particularly adapted to a boom-bust lifestyle and rapid population recovery when conditions are right. Apart from broadscale monitoring of vegetation trends (e.g., Gibson and Bosch, 1996), in which the role of rabbits was difficult to separate from other causal factors, and an uncalibrated grazing model (Barlow, 1987), the only work in New Zealand on the impact of rabbits has been one small study comparing pasture production with and without rabbits (Norbury and Norbury, 1996) and a study on their diet (Reddiex, 1998). We still have no good information to relate rabbit densities to their impact on pasture, livestock production, or environmental values. This lack of information did not concern managers focussing only on the tactics of rabbit control, but we argue that a successful or partially successful biological control agent increases the need to focus on better understanding of the ends to be achieved if landowners are to optimise the benefits from RHD by being able to reduce their control costs and/or increase productivity.

Two systems and their interactions will need to be understood to optimise decisions on rabbit control. First, we will need to understand how RHD has behaved since its introduction and ideally be able to predict how RHD will behave. Second, we will need to understand the nature of rabbit impacts as their density changes. Only then can we do a proper cost-benefit analysis to judge when and how much conventional control is worth doing (e.g., Choquenot and Barlow, 1998) - and, if we have mixed production, environmental and conservation goals, who should pay for it.

This paper reports on our attempts (to April 1999) to answer the first of these questions by summarising how RHD has behaved during its first two years in New Zea-

land, and goes some way towards answering the second by giving some preliminary results on the benefits of RHD to pastoral production.

METHODS

Rabbit densities were indexed by standard spotlight count methods (Norbury *et al.*, 1998) at 36 study sites from Hawke's Bay to Otago. These sites were chosen because we had rabbit density indices taken within 3 months prior to the arrival of RHD at each site, and often for many years before that. Counts were made on all sites several times after the initial epidemic; we use the counts taken in early 1998 to estimate changes in rabbit numbers over the period when RHD was first active. Counts were again taken on most sites in the winter of 1998 and early in 1999 and were used to estimate the effect of the second epidemic. To make the data more succinct we have pooled the data from sites by region and by the method of viral release (i.e., biociding, spot baiting, or natural spread).

Samples of rabbits were shot at the two Otago sites (Earnscleugh and Bendigo) once just before the arrival of RHD and on 7 or 8 occasions between November 1997 and February 1999, after RHD arrived at the sites. Age classes were determined from eye lens weights (Dudzinsky and Mykytowycz, 1961) and a blood sample taken from each rabbit. The presence of antibodies to RHD was tested at the National Centre for Disease Investigation, Wallaceville, using the competition ELISA method and reagents supplied by the Instituto Zooprofilattico Sperimentale della Lombardia e dell'Emilia in Italy. Four dilutions were tested and rabbits were considered to have antibodies to RHD if the percent inhibition was above 50% at the 1:40 dilution (O'Keefe *et al.*, 1998).

We infer the temporal pattern of RHD epidemics from the proportion of each cohort of rabbits that were seropositive. Rabbits in semi-arid Otago have a discrete breeding season from about September through to February (Fraser, 1988). We grouped the rabbits sampled into (a) those born in the 1996/97 breeding season and earlier, (b) those born in the 1997/98 breeding season, and (c) those born in the 1998/99 breeding season.

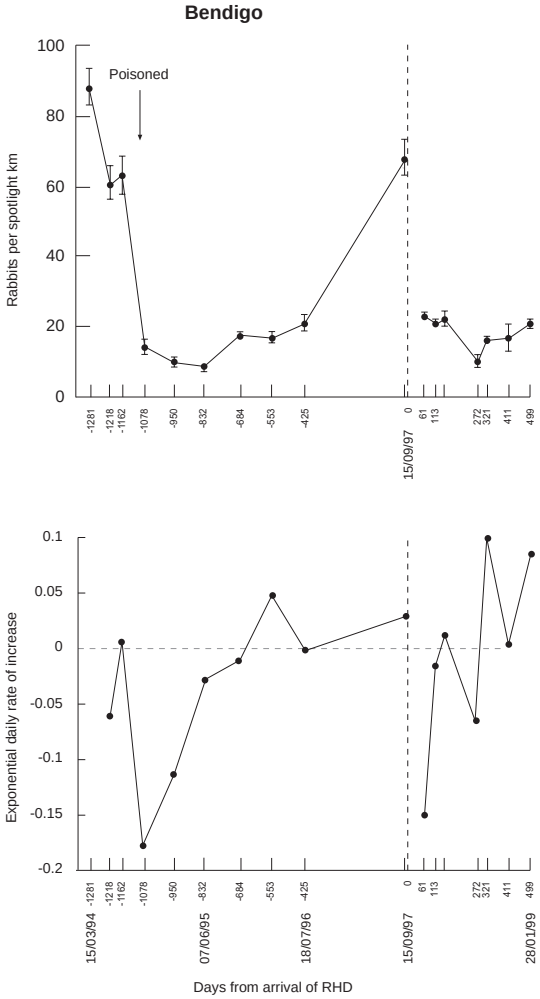
The benefits of RHD to pastoral production were assessed by comparing the accumulation of pasture biomass in open plots and exclosures, corrected for year effects (i.e., rainfall) during spring in 1994 and in 1998 at Earnscleugh, one of our Otago study sites. The methods for this trial are described in Norbury and Norbury (1996).

RESULTS

Efficacy of the initial epidemics of RHD

The detailed changes in rabbit density indices and rates of change are illustrated for the two Otago sites (Figs. 1 and 2). Rabbit Haemorrhagic Disease was introduced to the site at Bendigo by spot- baiting on 18 August 1997 and then by biociding (= mass aerial spread of baits treated with various unknown concoctions of virus) on 15 September 1997. The disease arrived naturally at Earnscleugh on 26 September 1997. Rabbits declined from high densities by an average of 67% at both sites (Figs.1 and 2).

FIGURE 1: Changes in rabbit density indices (±95% S.E.) along a standard 13.1 km spotlight route, and exponential rates of change at Bendigo Station, Central Otago between 1994 and 1999. Note: not all counts were repeated over two or more nights so some means do not have CLs.



Elsewhere, the disease has had variable results (Table 1). It killed an average of 88% of rabbits at nine Mackenzie Basin pastoral sites (all biocided) with low to moderate initial rabbit densities (Table 1). However, the disease had little effect on low density rabbit populations in the Mackenzie Basin river beds that are adjacent to the biocided areas but which were not themselves baited. At these sites, rabbits declined on average by 24% (Brown and Keedwell, 1998; G. Norbury, unpubl. data). In North Canterbury, rabbits halved at The Rock study site (Table 1) where the disease was released by spot baiting, but increased by 2% from very high initial densities at Tiromoana where mass biociding was used. However, the Tiromoana data are difficult to relate directly to RHD as the pre-RHD counts were measured in 1996, and the dry spring in 1997 had led to a burgeoning rabbit population. At five sites in Marlborough, rabbits declined by an average of 72% (Table 1). At seven sites in Manawatu/Wanganui, rabbits declined by an average of 34% . Rabbits at four sites in Hawke's Bay showed an average decline of 37% (Table 1),

FIGURE 2: Changes in rabbit density indices (± 95% S.E.) along a standard 14 km spotlight route, and exponential rates of change at Earnscleugh Station, Central Otago between 1994 and 1999. Note: not all counts were repeated over two or more nights so some means do not have CLs.

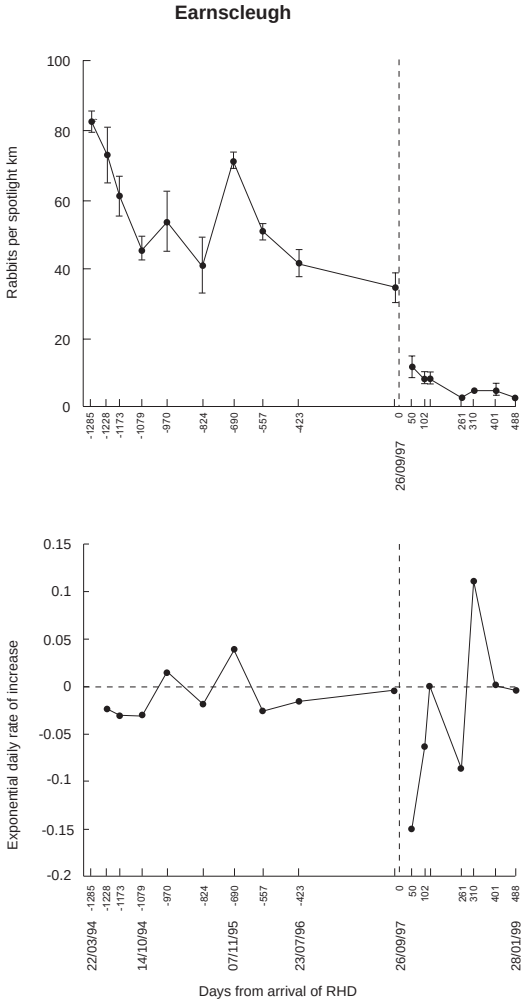


TABLE 1: Summary of changes in rabbit density indices before (usually winter) and after (usually summer) the initial epidemic of RHD in 1997 and a second epidemic in 1998.

Site	1997/98 Pre-RHD	1997/98 Post-RHD	1997/98 % change	1998/99 Pre-RHD	1998/99 Post-RHD	1998/99 % change	Notes
Earnscleugh	35	11.7	-67%	5.0	2.8	-44%	Natural epidemic
Bendigo	67.9	22.6	-67%	15.8	20.6	30%	Biocided 1997
9 Mackenzie Basin sites	14.3	1.7	-88%	2.1	3.6	71%	Biocided 1997
The Rock	41	21.2	-48%	29.4	29.4	0%	Spot baited 1997
5 Marlborough sites	17.4	4.8	-72%	4.3	2.9	-33%	Spot baited 1997
4 Hawke's Bay sites	27.4	17.2	-37%	NA	NA		Most spot baited
7 Manawatu / Wanganui sites	6.7	4.4	-34%	NA	NA		

but interpretation of the results are confounded by ongoing conventional control at some sites.

Efficacy of the second epidemics of RHD

As expected between breeding seasons, rabbit numbers at both Otago sites and most other sites continued to decline after the initial epidemic had ceased until the new breeding season in 1998 (c. day 330) (Figs. 1 and 2). In 1998, it appears that rabbits had begun to breed before the second epidemic as rates of increase were positive at both Otago sites in the spring of 1998. The rates remained positive at Bendigo despite evidence of an epidemic (see Table 2), but were negative between November 1998 and January 1999 (days 401 to 488 after RHD) at Earnscleugh (Fig. 2).

Serological results (see Table 2) show that RHD broke out again at both Otago sites between June and September 1998, although we cannot date this accurately as, unlike the initial epidemic, no fresh carcasses were noted at these sites. Elsewhere, reports of fresh carcasses suggest the disease was also active in the Mackenzie Basin, North Canterbury, and Marlborough during spring of 1998, a conclusion confirmed by serological tests which showed some young rabbits born in 1998/99 had been challenged by the virus (J. Parkes, unpubl. data). Despite these second epidemics, rabbit densities increased at most sites we monitored (Table 1). Rabbit numbers increased at all Mackenzie Basin sites by an average of 71% (range 8% to 1400%) at the pastoral sites, albeit from very low densities. Numbers remained unchanged at a single North Canterbury site measured, but continued to decline by an average of 33% (range -100% to +300%) at five Marlborough sites.

TABLE 2: Proportion of three cohorts of rabbits sampled over 18 months that had antibodies to RHD at natural epidemic (Earnscleugh) and biocided (Bendigo) sites from Central Otago. * Some spot-baiting was done at this site before we sampled the "pre-RHD" rabbits. Cohort 1 were rabbits born in the 1996/97 breeding season and earlier. Cohort 2 were born in 1997/98, and cohort 3 were born in the 1998/99 breeding season (July to March).

Date sampled	No. rabbits in cohort 1	% with antibodies	No. rabbits in cohort 2	% with antibodies	No. rabbits in cohort 3	% with antibodies
Earnscleugh						
8/9/97	60	0	0		0	
25/11/97	35	31.4	0		0	
8/12/97	27	29.6	0		0	
14/2/98	28	21.4	2	0	0	
8/6/98	28	7.1	2	0	0	
7/9/98	28	28.6	3	33.3	0	
16/11/98	29	58.6	1	100	0	
4/2/99	22	9.1	1	0	7	14.3
Bendigo						
9/9/97	60	13.3	0		0	
21/11/97	54	48.1	6	0	0	
12/2/98	18	44.4	12	0	0	
2/6/98	17	23.5	13	0	0	
9/9/98	19	47.4	11	9.1	0	
22/11/98	4	75.0	6	0	21	19.0
2/2/99	2	50	5	20	23	17.4

Field epidemiology

The disease was active at our two Otago sites in September through to November 1997. Judging by the presence of fresh carcasses counted along our survey routes,

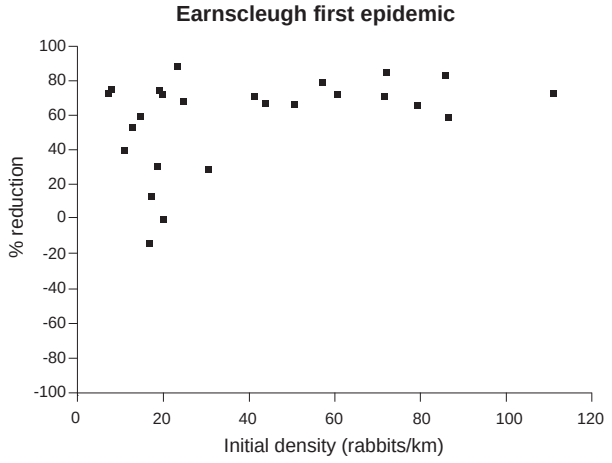
the epidemic at Bendigo lasted about 40 days with peak mortality at day 3. The natural epidemic at Earnscleugh lasted 80 days with peak mortality at about day 20 (Parkes, *et al.,* 1999).

Table 2 shows the proportion of rabbits in each age class that were seropositive in samples shot at the two Otago sites since September 1997. Four results are apparent from these data:

- Biociding may have left more of the adult survivors seropositive and immunised than the natural epidemic as shown across other sites in Oatgo (O'Keefe *et al.,* 1998). In samples shot soon after each epidemic, 48% and 30% of 62 and 54 adult rabbits were seropositive at Bendigo and Earnscleugh, respectively (χ^2 = 3.7, d.f. = 1, P = 0.054).
- The proportion of the cohort born in 1996/97 and earlier and surviving the 1997 epidemic that were seropositive declined from an average of 39% in the samples (pooled from both sites) shot in November/ December 1997 (immediately after the initial epidemics), to an average of 30% in February 1998, to only 13% in June 1998. This pattern of antibody loss was apparent in all populations sampled by regional councils and in longitudinal pen studies on individual rabbits (J. Parkes, *unpubl. data*).
- None of the 32 rabbits born during the 1997/98 breeding season that were sampled before September 1998 were seropositive, i.e., these young animals escaped infection during the initial epidemic, but not the second.
- A second epidemic of RHD had broken out at both sites by September 1998, judging by the increase in seropositive adults and juveniles of both the 1997/98 and 1998/99 cohorts.

Comparison of the initial local density of rabbits with the percent killed at these places in natural epidemics clearly shows a density dependent relationship. At densities above about 25 rabbits/km the disease was always effective, below that density changes were variable (Fig. 3).

FIGURE 3: Effect of local rabbit density along a 14 km spotlight route on the percent of rabbits killed in a natural epidemic in 1997 at Earnscleugh, Central Otago.



Benefits of RHD

Rabbits at our Earnscleugh site declined from an index of 71/km in spring 1994 to about 5/km in spring of 1998 (Fig. 2). Pasture production in 1998 had been reduced by the drought to about a third of the 1994 levels but the reduction in rabbits also reduced their grazing effects (Table 3). In summary, an extra 71kg/ha of dry matter was produced after RHD, a 1.5-fold increase in production or a 77% reduction in grazing impact due to RHD.

TABLE 3: Summary of results from a grazing trial at Earnscleugh using exclosed and open plots.

Year	Rabbit density (rabbits/spotlight km)	Pasture biomass (kg/ha)
1994	0	853
1994	30 to 76	139
1998	0	302
1998	3 to 6	210

DISCUSSION

To date RHD has been effective at reducing rabbit numbers only in some places in New Zealand , but it has clearly persisted and caused a second epidemic in the spring of 1998 at the two Otago study sites (Table 2). Serological data from sites monitored by regional councils show similar evidence of new epidemics in 1998/99 (J. Parkes, *unpubl. data*). Despite this second epidemic, most rabbit populations increased during the 1998/99 breeding season. The causes of these variable outcomes are unknown, but some possible reasons include :

- The use of poor-quality baits and virus in most biocide attempts in 1997 in Otago (O'Keefe *et al.*, 1998), although biociding in the Mackenzie Basin and at our Bendigo site achieved good results.
- Most epidemics require host density thresholds to sustain disease. Good kills were achieved in low-density rabbit populations that were biocided, i.e., the transmission rates probably depended on bait density rather than rabbit density. However, there was a clear indication of a density dependent effect in the initial natural epidemics, with a threshold of about 25 rabbits/km at our Earnscleugh site.
- The age structure of rabbit populations may affect the success of RHD. The 1998 epidemic occurred at both our Otago sites, but further reduced rabbit numbers only at the Earnscleugh site where the population was comprised largely of old rabbits (see Table 2). At the Bendigo site the rabbits were almost all young (<2 years old) (see Table 2) and recruitment exceeded mortality during the 1998 epidemic. If age does affect how well RHD works, this will have implications for how conventional rabbit control is applied (because ongoing conventional control maintains a young age structure).
- The role of vertebrate and invertebrate vectors may vary between sites. We speculate that effective spread

of the disease may require the presence of vertebrate scavengers and predators (hawks, ferrets, and cats) to open carcasses and expose virus to insect vectors. The vertebrates are probably everywhere, but we do not know which species of insects might be effective vectors or how they are distributed in space and time. Insects are known to carry the disease and infect rabbits (Barratt *et al.*, 1998).

The decline in the proportion of seropositve rabbits between the 1997 and 1998 epidemics may be due to loss of antibodies in infected survivors, to increased mortality in infected survivors, or both. Longitudinal studies of infected survivors in pen trials shows these individuals do lose antibodies over time unless reinfected, but do not lose immunity (J. Parkes, unpubl. data). If loss of antibodies in individuals is the cause of the declining proportion of seropositive rabbits in our cross-sectional samples, it leaves us with a diagnostic problem (seronegative rabbits that are nevertheless immune). It also leaves managers with a problem because, if they sampled rabbits during winter to see if was worthwhile trying to biocide to start an epidemic, they would underestimate of the percent of the population that is immune.

An ideal biological control agent should reduce the target pest numbers sufficiently to remove or reduce the adverse impacts of the pest in the long-term for the single cost of releasing the agent. Evidence of the success of RHD in controlling rabbits should therefore be seen in improved pasture biomass or condition, and ultimately in livestock production, and should be indexed by fewer rabbits and lower costs of conventional control. The results of the unreplicated trial we conducted on grazing effects summarised in this paper cannot be extrapolated.

Returning to the first question posed in the introduction, can we understand and predict the behaviour of RHD? At present we cannot. Although the disease has persisted and returned to areas a year after the first epidemic, our understanding of the critical epidemiological parameters (transmission rates, modes of persistence, and effects of rabbit densities and population structure) remains hazy. However, we predict that the disease will persist, but that it will not do so or be effective at every place each year.

The second question (what is the relationship between rabbit densities and their impacts on production (and environmental) values?) also remains largely unanswered by our unreplicated pasture biomass trial, in which we tested only three rabbit densities tested and were confounded by the drought in 1998. We have some good experimental data and models from Australia to show us how to answer this question (Choquenot *et al.,* 1998), but the work needs to be done under New Zealand conditions. Only then can we answer the third question: what is the optimal strategy to add conventional control to RHD? The costs of this control are well understood, the benefits are not known.

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