# Does Conventional Control of Rabbits ‘Re-set’ the Efficacy of RHD at Sites Where This Biocontrol Is Failing?

John Parkes






Landcare Research
Manaaki Whenua

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John Parkes

Landcare Research
PO Box 40, Lincoln 7640
New Zealand

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Marlborough District Council and Environment Southland
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| Reviewed by: | Approved for release by: |
| --- | --- |
| Bruce Warburton<br>Scientist<br>Landcare Research | Aaron McGlinchy<br>Science Team Manager<br>Wildlife Ecology & Epidemiology |

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## 1. Introduction

Landcare Research investigated whether conventional control by poisoning of most rabbits will reset the biocontrol at sites where RHD has failed. This work was carried out between July 2007–June 2009 for Marlborough District Council and Environment Southland under an Envirolink medium advice grant from the Foundation for Research, Science and Technology.

## 2. Background

The rabbit biocontrol agent RHD was released in 1998 in New Zealand and typically killed c. 80% of the rabbits in these initial epidemics. Its efficacy has been declining in recent years and, in general, two scenarios appear to be emerging:

Scenario 1: Sites where epidemics continue to suppress rabbit numbers but where the efficacy of RHD as a biocontrol is slowly declining.

In some areas (e.g. the Mackenzie Basin) RHD still kills most new rabbits each year but more of these young-of-the-year rabbits are left immune after epidemics (Fig. 1), which typically occur each autumn. Initially, less than 20% of young rabbits were immune after an epidemic and now over 50% are routinely seropositive when tested after the autumn epidemic (Parkes et al. 2008).

Of course older rabbits have survived at least two annual epidemics and indeed most are immune (nearly all amongst the really old age classes). This is as expected when the disease infects most rabbits but does not kill about 10–20 % of those infected (Parkes et al. 2002).

This ‘accumulation’ of immunity among older age classes is not a symptom of changing epidemiology. However, the increasing trend in the proportion of young with antibodies is a symptom of some change – what we do not know. It might be a change in the virus, evolution of resistance in the rabbit, or some epidemiological consequence of parental immunity.

The trend in immunity leads to slowly increasing rabbit numbers (Fig. 2), although only at a rate about 10% of what would occur in the absence of RHD. This suggests that RHD is working as a biocontrol but not as well as it did initially.

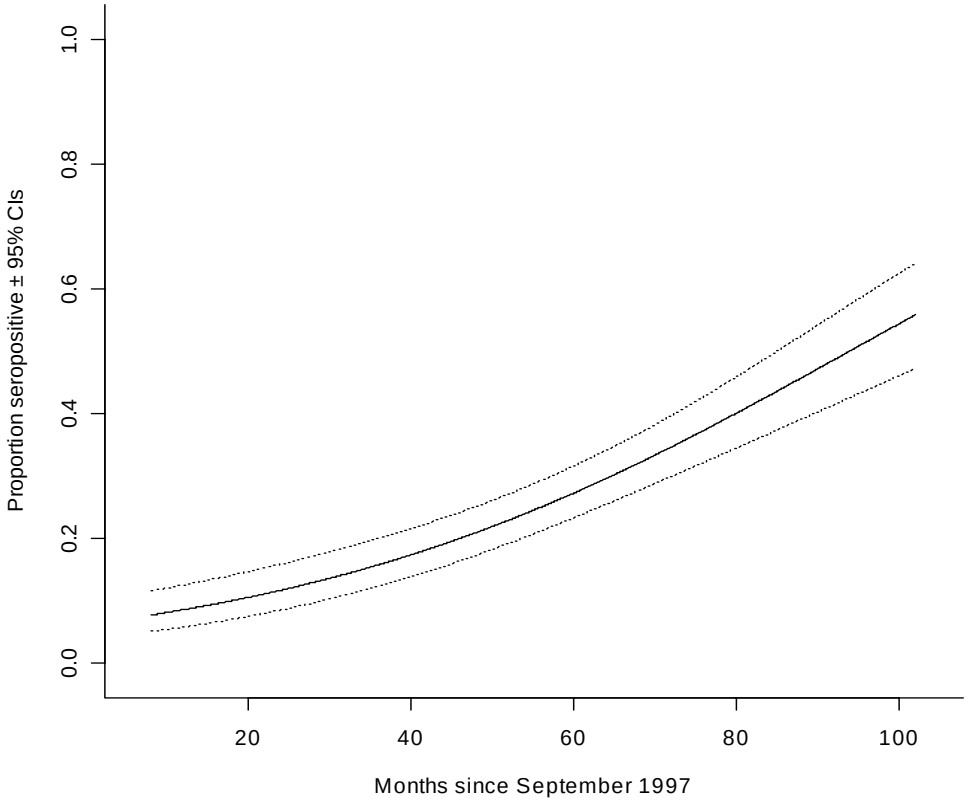


**Fig. 1** Trend in the proportion of young rabbits tested between February and May each year since 1998 that are immune to RHD for four sites in the Mackenzie Basin (after Parkes et al. 2008).

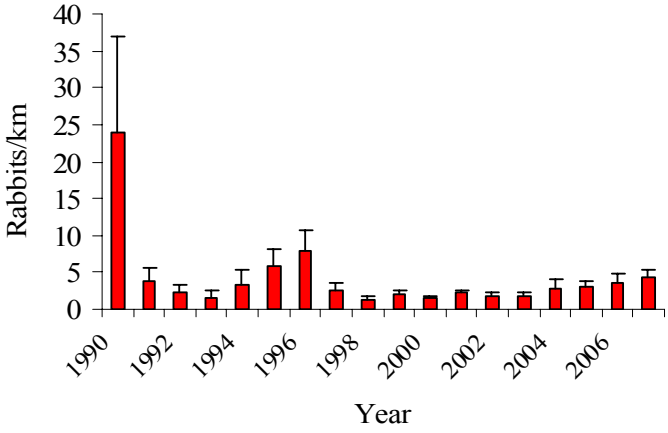


**Fig. 2** Trend in rabbit abundance indices along 33 spotlight routes in the Mackenzie Basin, 1990 to 2007 (after Parkes et al. 2008). Rabbits were controlled under the Rabbit and Land Management Programme in 1990 and RHD arrived in 1997.

Scenario 2: Sites where RHD persists but where rabbit abundance has exceeded tolerable levels and where most rabbits that survive epidemics are immune.

In other areas rabbits have reached densities at which conventional control is triggered in regional pest management strategies, and where most rabbits have antibodies to RHD and are thus immune.

Regional Council Pest Management Strategies require landowners to control rabbits when they exceed set densities (often measured by an index called the McLean Scale). When the area to be treated is substantial, aerial baiting with 1080 or pindone is the usual control method, and this is increasingly planned for such 'failed' sites and should result in a 90% plus reduction in rabbit numbers.

*Question 1*: Will the sites where rabbit abundance and immunity in the young rabbits are still at low-medium levels but trending upwards become problem areas and require conventional control?

*Question 2*: Will poisoning or other conventional control such as shooting of most rabbits at the 'failed' sites reset the biocontrol and give farmers many years grace before they have to spend large sums of money again. Or, will RHD always fail at these places leading to high proportions of immune rabbits especially in the youngest age classes, and rabbits rapidly recover to their pre-RHD levels? This is the question addressed (in part) in this project.

## 3. Objective

- To compare the serological status of rabbits at sites where RHD has failed before and after population reduction by conventional control to assess whether this control reduces the prevalence of antibodies to RHD, particularly among young rabbits

## 4. Methods

### 4.1 Experimental design

The experiment to give an empirical answer (without any explanation of cause) is simple. Most of the rabbits at the 'failed' site were killed (using 1080 or pindone baits), and two response variables were measured:

- Rates of population recovery at these places were measured from standard spotlight counts taken each autumn for a variable number of years before the site was poisoned and then each year afterwards.
- Changes in the proportions of young rabbits (usually born each spring) with antibodies when tested after the usual autumn epidemics.

More generally, if the trial is to be extended to other sites, poisoning is usually done between March and July (depending on the bait used) but before rabbits start to produce independent young in about August, and RHD epidemics usually occur between October and May with

apparent peaks in autumn. So, annual counts (using standard spotlight routes) in say April, followed soon afterwards by annual shot samples to look at the serology and by inference the immune status by age class of the rabbits in mid-autumn, will provide data on trends in rabbit numbers and trends in their immune status (Fig. 3). It is often clear by the fresh rabbit carcasses seen in the field when RHD is active. The spotlight counts and serological sampling should occur a few weeks after these signs are noticed to give the epidemic time to impose its effects.

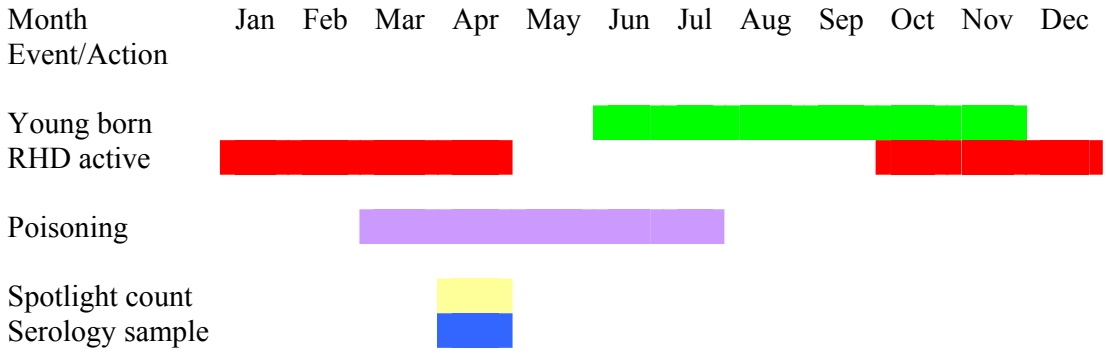


**Fig. 3** Suggested annual timetable for sampling around key events

### 4.2 Study sites

Three sites where RHD had failed and where conventional control was imposed were monitored (Table 1).

**Table 1** Study sites where RHD had 'failed' and where conventional control was conducted. Note: additional sites from the Canterbury Region will be included in later analyses.

| Site | Poisoned (winter of) | Pre-poison rabbit abundance (rabbits/km) | Pre-poison prevalence (%) of antibodies in young/old rabbits | Council |
|---|---|---|---|---|
| Honeymoon | 2007 | 62.9 | 56/44 | Marlborough |
| Tone | 2007 | 27.5 | 56/50 | Marlborough |
| Five Rivers | 2008 | 143.8 | 71/77 | Southland |

## 5. Results

### 5.1 Honeymoon

RHD had reduced rabbit numbers in the wider area (Muller No. 1: Blicks) from around 24 rabbits/km before RHD arrived in spring 1997 to as low as only 1–2 rabbits/km in 2001 (Marlborough District Council, unpubl. data). Spotlight counts on the area to be poisoned had increased by 2005 with over 60 rabbits/km recorded in 2007, and the area was baited with pindone baits in the winter of 2007, which resulted in a reduction in the index of 97% (Fig. 4).

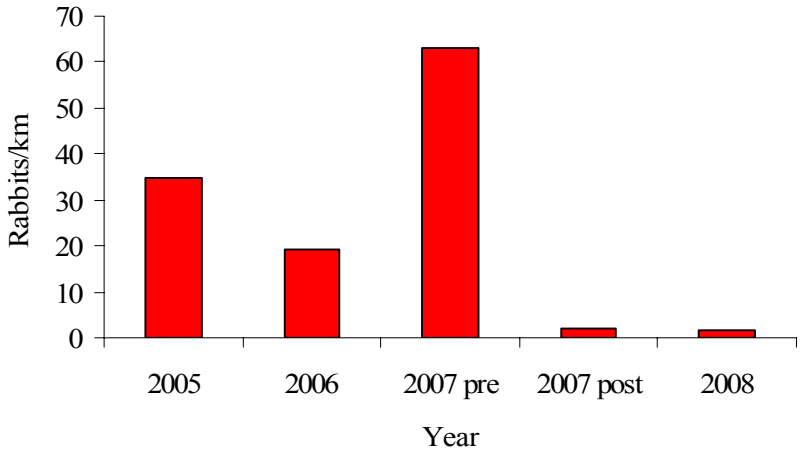


**Fig. 4** Changes in rabbit density indices at the Honeymoon block, 2005–2008.

The percentage of young rabbits with antibodies to RHDV at this site after the initial epidemics in the area in 1997 is unknown but assumed to be around 20% – as typically occurred elsewhere in New Zealand (Parkes et al. 2008). The percentage of young rabbits ($n$ = 74) with antibodies in the three years (2005–2007) before conventional control was 53% and this had fallen to 38% in the 53 young rabbits sampled in 2008–2009 after the population was poisoned (Fig. 5). The decrease was not significant ($\chi^2 = 2.12$, $P = 0.145$).

The percentage of adult rabbits with antibodies remained high before and after the population was poisoned ($n$ = 28, 60.7%; $n$ = 17, 70.6%, respectively).


**Fig. 5** Percentage of young rabbits with antibodies to RHDV at the Honeymoon site, 2005–2009.

3.2 Tone

By 2005, RHD had begun to fail at the Tone site with the spotlight index showing rabbit densities were approaching pre-RHD levels. Pindone baiting in the winter of 2007 reduced the population by about 71% (Fig. 6).

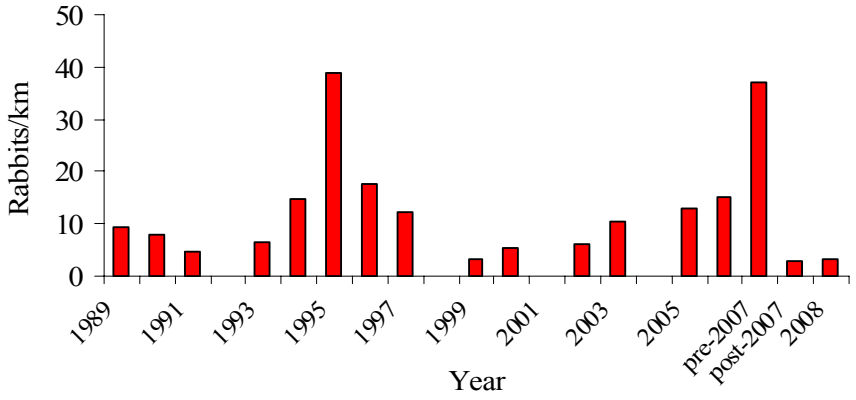


**Fig. 6** Changes in rabbit densities indices at the Tone site since 1988.

Again there are no site-specific data on the percentages of young rabbits that had antibodies after the first epidemics in 1997 so the 20% baseline is assumed. The percentages of young rabbits ($n$ = 100) with antibodies in the three years before the population was poisoned and in the two years after ($n$ = 36) were 52% and 42%, respectively (Fig. 7). This is not a significant change ($\chi^2$ = 0.94, $P$ = 0.33).

The percentage of adult rabbits ($n$ = 14 and 22, respectively) with antibodies was very high over the three years before control (78.6%) and remained so after control (72.7%).

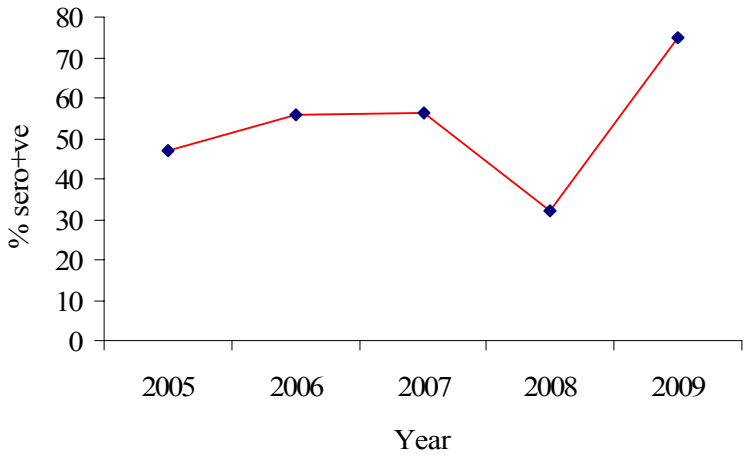


**Fig. 7** Percentages of young rabbits with antibodies to RHDV at the Tone site, 2005–2009.

### 5.2 Five Rivers

Rabbit densities were very high at this site in June 2008 (144 rabbits/km) leading to control in July 2008. A high kill was achieved (judging by the difficulty in shooting enough rabbits for the serological samples in 2009). In April 2008, 70.6% of the 17 young rabbits had antibodies to RHDV and in April 2009 only 28.6% of the seven young rabbits sampled had antibodies. Fisher's exact test shows no significant difference in the percentages before and after the control.

A high percentage of adult rabbits had antibodies before ($n$ = 13, 77%) and after ($n$ = 10, 70%) the control.

### 5.3 All sites pooled

If the data from all three sites are combined and tested using a general linear model there is a significant effect of the treatment (poisoning) in that significantly fewer young rabbits have antibodies after the control was imposed than before ($\chi^2$ = 5.78, $P$ = 0.02).

## 6. Conclusions

The results are equivocal. Taken site by site it appears that the problem of high immunity levels is not resolved by reducing the populations. However, the generally small sample sizes for the juvenile rabbits make any effect difficult to demonstrate at a site.

If we pool all sites, there is an effect that suggests population reduction may 'reset' the efficacy of the biocontrol to some extent at least. The average percentage of immune young rabbits before the control was 54% and after the control was 39%. The 'expected' value would be around half this if the system was reset to similar levels experienced in the initial epidemics in 1997.

So, assuming the pooled results do suggest the seroprevalence among juvenile cohorts of populations can be reset, then what might be the mechanism? The percentage of adult rabbits with antibodies remained high among the survivors of poisoning, so presumably the fall in juvenile seroprevalence is not due to some transmission from mother to offspring – otherwise the percentage would remain unchanged.

What has changed is the density of rabbits. So we might speculate that this results in a lower abundance of virus in the environment and so a lowered force of infection for the young rabbits.

**Recommendation**

- It is recommended that the three sites be monitored for at least another year, and that additional sites from Canterbury be added to the analysis.

## 7. Acknowledgements

I thank the council staff in Marlborough and Southland who collected the data from the study sites. Funding for this work was provided by the Foundation for Research, Science and Technology under Envirolink medium advice grant MLDC32.

## 8. References

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