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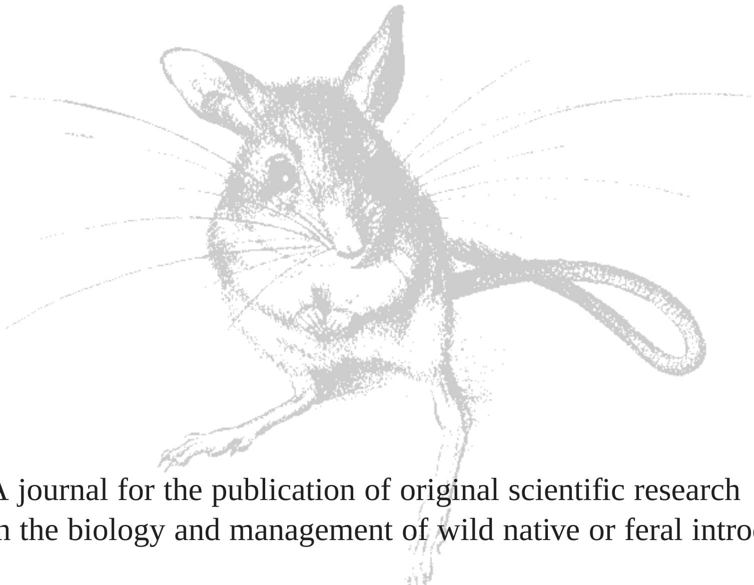

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Initial effects of rabbit haemorrhagic disease on free-living rabbit (*Oryctolagus cuniculus*) populations in central-western New South Wales

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Abstract

Quarterly spotlight counts of rabbits were conducted at three sites in central-western New South Wales. These counts commenced two years before the arrival of rabbit haemorrhagic disease (RHD) in the winter of 1996. The existing data on quarterly rates of change in rabbit abundance for the three populations provided a unique opportunity to study the effects of RHD on rabbit demography. Prior to the arrival of RHD, all three populations underwent phases of sequential increase and decrease in each year. On the basis of these patterns, RHD had a variable influence on the demography of the three rabbit populations. In 1996–97, the density of two populations declined over an expected period of increase, while at the third site the density increased as expected from pre-RHD patterns. Twelve months after their failure to generate expected positive rates of increase the two affected populations had returned to the normal sequence of increases and decreases in density although still at comparatively low numbers.

Introduction

Wild rabbit (*Oryctolagus cuniculus*) populations have a negative impact on agricultural production and conservation values in Australia (Williams *et al.* 1995). This impact is mostly experienced by pastoral industries. Rabbit haemorrhagic disease (RHD) (otherwise known as rabbit calicivirus disease or RCD in Australia) is potentially the most important rabbit-control agent to be made available to landholders in this country since the advent of myxomatosis. RHD was first described in China in 1984 from where it spread rapidly across Europe and other parts of the world (Ohlinger and Thiel 1991). In September 1995 the disease escaped from Australian quarantine facilities on Wardang Island in Spencer Gulf where it was being evaluated as a potential biological control agent (Cooke 1996). From there it quickly spread to mainland South Australia and neighbouring states at a rate of up to 18 km day^{-1} (Cooke 1996). Anecdotal or unpublished reports on the effectiveness of RHD during its initial spread in Australia suggest that declines in rabbit population densities were variable, particularly in the more temperate areas.

At the time of the escape of RHD, studies were being conducted in the central-western area of New South Wales involving long-term and regular measurements of rabbit population densities at three different locations. The purpose of the studies (and locations) were:

(i) To identify and demonstrate optimum rabbit management strategies for grazing enterprises in central-western New South Wales (Bathurst and Euchareena), and
(ii) To examine the potential of fertility control on fox populations (Lake Burrendong).

The eventual confirmation of RHD at these study sites gave us the opportunity to report on initial population mortality rates and subsequent recovery from the disease. Such information is essential if RHD is to be used as a long-term management tool for the reduction of rabbit impact in Australia.

Methods

Study sites

The three sites (Fig. 1) are on the Great Dividing Range in central-western New South Wales (Bathurst, 33º24′S, 149º21′E; Euchareena, 32º58′S, 149º13′E; and Lake Burrendong, 32º40′S, 149º10′E). The study

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sites are mostly used for fine wool, fat lamb and cattle production. The Bathurst and Euchareena sites are properties used purely for agricultural production while the Burrendong site consists of dam foreshores that are managed for water catchment. This includes the use of grazing stock. Topography ranges from undulating to hilly lowland with elevations of 450–800 m. Average monthly rainfall and temperature statistics for the study sites are presented in Table 1. Remnant vegetation consists of dry sclerophyll forest and woodland. All sites are in the Macquarie River Valley – Lake Burrendong and Euchareena are on the river itself and Bathurst is approximately 8 km from the river.

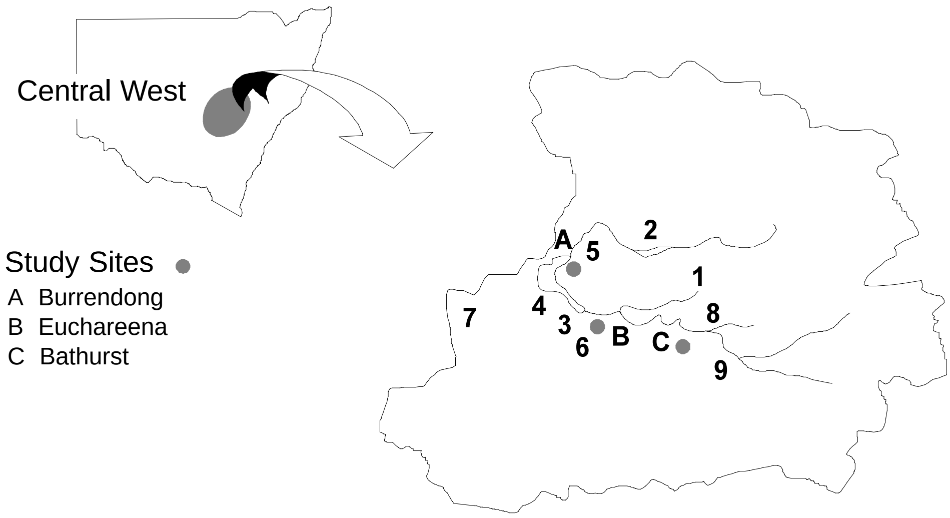

Fig. 1. Location of study sites and sequential arrival (1 to 9) of RHD in central-western New South Wales.

Table 1. Average monthly temperature and rainfall for study sites
Indicative temperatures for Burrendong and Euchareena could be obtained only from Wellington weather station

		Jan.	Feb.	Mar.	Apr.	May	Jun.	Jul.	Aug.	Sep.	Oct.	Nov.	Dec.
Bathurst	Max. temp. (°C)	27.7	26.9	24.3	20.3	15.7	12.1	11.1	12.8	15.7	19.6	22.6	26
	Min. temp. (°C)	13.2	13.0	10.4	6.2	3.5	1.5	0.1	1.1	2.9	5.9	8.3	11.0
	Rainfall (mm)	71.9	62.1	55.1	54.8	54.3	67.0	67.1	71.3	59.5	75.5	62.6	69.9
Euchareena	Max. temp. (°C)	31.1	30.4	27.4	23.4	18.6	14.9	14.1	15.6	18.7	22.8	26.2	29.7
	Min. temp. (°C)	17.4	17.4	14.8	11.0	7.6	4.3	3.3	4.3	6.4	9.9	12.7	15.6
	Rainfall (mm)	68.4	56.9	55.5	51.1	64.2	43.8	64.4	67.3	58.4	66.0	53.4	59.9
Burrendong	Max. temp. (°C)	31.1	30.4	27.4	23.4	18.6	14.9	14.1	15.6	18.7	22.8	26.2	29.7
	Min. temp. (°C)	17.4	17.4	14.8	11.0	7.6	4.3	3.3	4.3	6.4	9.9	12.7	15.6
	Rainfall (mm)	69.0	62.2	50.3	43.8	49.5	39.9	46.8	49.0	44.0	62.4	55.3	50.2

Survey techniques

Quarterly spotlight counts were undertaken at all sites commencing in July 1994. Set transects were driven at uniform speed and all animals seen were counted. Counts were conducted over three consecutive nights, commencing at approximately the same time each night. Although counts were conducted quarterly they did not necessarily coincide in time at each site. Transect lengths were 7.5 km at Bathurst, 7.4 km at Euchareena and 23.8 km at Lake Burrendong. Data from these counts are expressed as the quarterly mean number of rabbits counted per transect-kilometre.

History of RHD in New South Wales

After the initial escape of the disease, the spread of RHD in rabbit populations throughout New South Wales was closely monitored. Confirmation of the disease required submission of blood and/or tissue (mostly liver) samples from rabbits for laboratory analysis using antigen-capture ELISA tests. The disease was first detected in the far west of the State around Broken Hill in November 1995 (T. Korn, personal communication). From there it spread to the south-west of New South Wales in April 1996 and eastwards to Cooma by early May. The next confirmed case of RHD in New South Wales was in the central-west at Sofala (Point 1 in Fig. 1) on 28 May (390 km from Cooma) followed by Stuart Town (Point 2) (near Lake Burrendong and Euchareena sites) (3 June) and through to Bathurst (Point 9) by late July. Anecdotal evidence suggests that, in some cases, human intervention was involved in the spread of the disease. By the end of July 1996, there were nine confirmed cases of RHD in the central-west (Fig. 1). A common feature of most of these initial outbreak sites in the central-west was proximity to the main river systems of the area. The spread of RHD slowly continued throughout the State until October 1996, when the first of the authorised, State-wide introductions of the disease commenced.

Laboratory confirmation of the presence of RHD on or adjacent to the study sites was first confirmed at Lake Burrendong on 12 June, at Euchareena on 4 September and at Bathurst on 19 September 1996. Because of the rapid spread of the disease around the central-west of the State following its first detection in late May, these dates are only indicative. Given the early cluster of RHD outbreaks in the vicinity of Lake Burrendong and its upper reaches of the Macquarie River and the observed decline of rabbit populations in this general area, we believe the disease arrived on the Euchareena site around the same time as the confirmed Burrendong outbreak (June). Because the Bathurst site was being closely monitored throughout this period (blood sampling and spotlight counts) and with the later occurrence of confirmed outbreaks nearby, arrival of RHD appears to have been some months after the other sites. Although there was one confirmed sero-positive case in September no further cases were detected until January 1997, when both sero-positive and antigen-positive animals were detected. We therefore assume that RHD arrived at the Bathurst site at the latter time.

Estimating changes to rabbit populations

The irregular timing of the spotlight counts conducted on each site meant that density estimates could not be consistently matched to the annual cycle of breeding and recruitment. Hence, sequential seasonal peaks and troughs in rabbit density were identified from population estimates and used to derive monthly exponential rates of change in abundance ($((\ln N_{t+1} - \ln N_t)/ t)$) as populations increased to their annual maximum density over spring/summer and declined to annual minimum densities over autumn/winter. In this way, an increase and decrease phase for each population could be identified every 12 months and an associated rate of change in abundance estimated. Because the rabbit populations at Lake Burrendong and Euchareena did not go through an increase following the arrival of RHD in 1996, changes in abundance were estimated for the corresponding period of increase at the Bathurst site. In the absence of an estimate of summer density for Lake Burrendong in 1994–95, rates associated with the increase phase in 1994–95 or the decrease phase in 1995 could not be estimated for this site.

Results and Discussion

Fig. 2 shows the variation in rabbit abundance over the duration of the study at the three study sites and indicates those estimates used to derive sequential rates of change in rabbit density (Fig. 3). Prior to the arrival of RHD in 1996, all three populations appeared to undergo a regular pattern of increasing density over spring and summer and decreasing density over autumn and winter. The data presented in Fig. 3 show that the sequential increase and decrease phases for the Bathurst population appeared unaffected by the presence of RHD. In contrast, the 1996–97 increase phase failed completely for both the Lake Burrendong and Euchareena populations. The subsequent decrease phase (1997) was similar for all three populations, as was the increase phase for 1997–98. Hence, the only apparent effect of RHD on rates of change for the three populations monitored in this study was the continued decline in the abundance of the Lake Burrendong and Euchareena populations following the arrival of the disease in 1996. Given previous patterns of variation in rabbit abundance on these two sites, an increase phase over this period would have been reasonably expected.

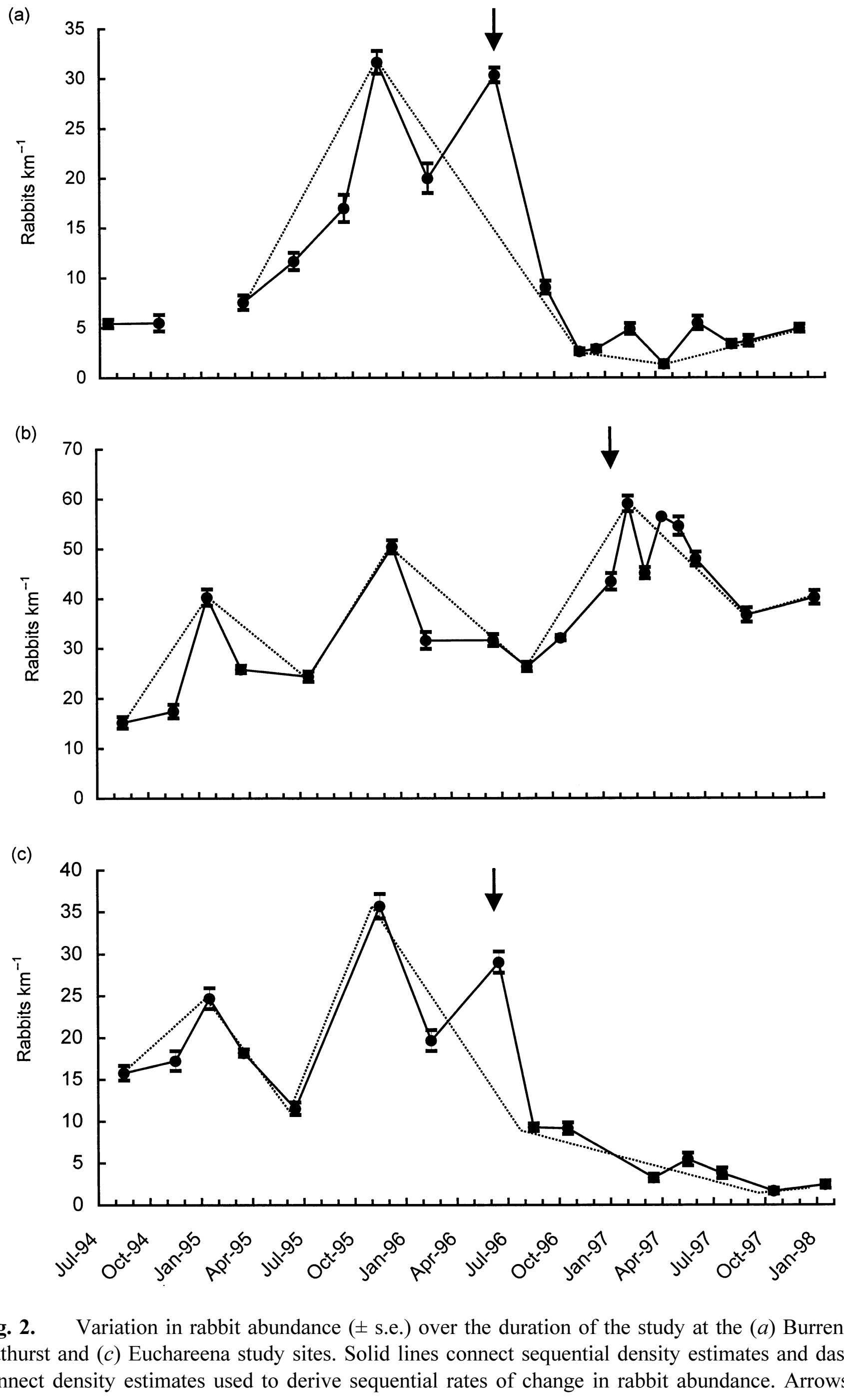

Fig. 2. Variation in rabbit abundance (± s.e.) over the duration of the study at the (*a*) Burrendong, (*b*) Bathurst and (*c*) Euchareena study sites. Solid lines connect sequential density estimates and dashed lines connect density estimates used to derive sequential rates of change in rabbit abundance. Arrows indicate arrival of RHD.

There are few reported rates for RHD-induced declines in density in free-living rabbit populations. Two studies in Spain identified population reductions of 75% (Piero and Seva 1991) and 55% (Villafuerte *et al.* 1994). In commercial rabbit farms, mortality rates have been

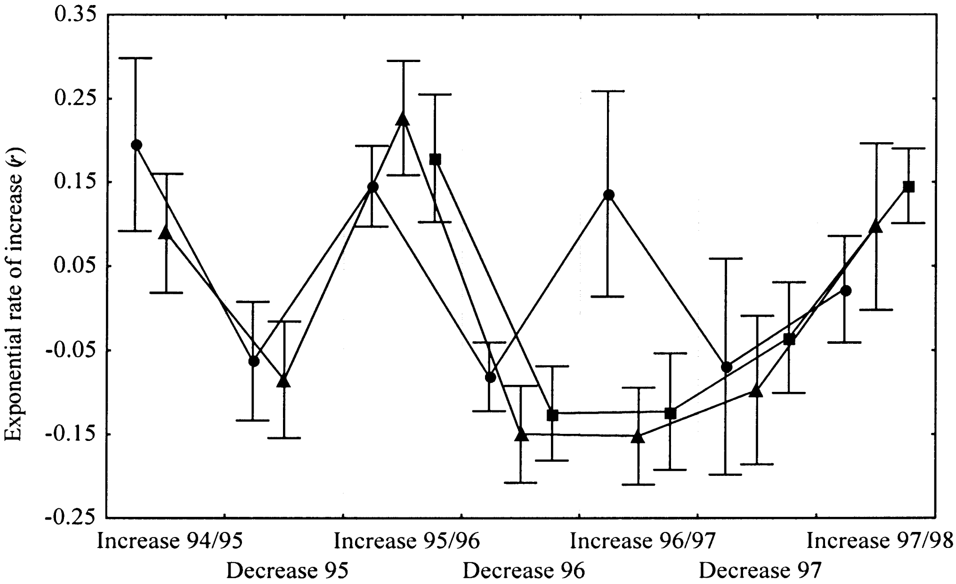

Fig. 3. Variation in monthly exponential rates of increase (± s.e.) for rabbit populations at Euchareena (▲), Burrendong (■) and Bathurst (●) sites. Because rabbit densities did not increase at Euchareena and Burrendong over the summer of 1996–97, peak densities could not be identified. Rates of change for these sites were calculated from estimates of rabbit density corresponding to the period over which rabbits on the Bathurst site increased to peak densities.

variable: after first reports of the disease in China and later in Europe, mortalities amongst adult farmed rabbits greater than 80% were reported (Capucci *et al.* 1996) while in two outbreaks in the United Kingdom the mortality was 15 and 71% (Fuller *et al.* 1993). Rates of decline in rabbit density observed here are similarly variable. For the critical periods immediately before and after the arrival of RHD, measured declines in rabbit population densities (rabbits counted per transect kilometre) were 91% at Lake Burrendong and 68% at Euchareena. In contrast, the population at Bathurst increased by 87% above the density observed prior to the arrival of RHD. Interpretations based only on these estimates of decline or increase should be made with caution. In some instances the effect of RHD could be hidden by normal changes in rates of increase. The true variability in effect of RHD is more accurately represented by the method depicted in Fig. 3.

If RHD was able to produce substantial population declines at two of the sites, why did rabbit numbers at the third site remain ostensibly unaffected despite the presence of the disease? The topography and climate of the sites were similar, initial rabbit densities prior to the arrival of RHD were similar and all sites were within the proximity of many other confirmed outbreaks. The only obvious difference was that RHD arrived at Euchareena and Lake Burrendong in June whereas it was delayed until the following December/January at Bathurst. That rabbits at the Bathurst site were able to produce a normal breeding response in 1996–97 may reflect a greater rate of survival of rabbits exposed to the disease or compensatory breeding that negated initial mortalities. These responses may have been possible only at Bathurst due to the later arrival of RHD at a time when breeding had already commenced. At two of the sites (Euchareena and Bathurst) live-trapping of rabbits (unpublished data) in May/June 1996 produced no kittens (<700 gm) (n = 48 and 209 respectively). However, a major breeding event occurred at the Bathurst site commencing in September/October 1996 (0.8% kittens in September, n = 127; and 35.7% kittens in October, n = 168). Young rabbits (<2 months of age) are known to be resistant to RHD, although the mechanism involved is unknown (Fuller *et al.* 1993). The newborn rabbits from the spring breeding pulse at Bathurst may still have been young enough to benefit from this natural protection when RHD arrived at the site.

The arrival and spread of RHD in the central-west of New South Wales highlights our lack of understanding of the disease, particularly in higher-rainfall (>300 mm) areas. Such variations in effectiveness of RHD have implications for the sustained, long-term use of the virus as a management tool; in particular, the importance of monitoring rabbit populations and follow-up control (Coman 1997). Continuing research aimed at determining population parameters that can influence the effectiveness of RHD will be essential.

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References

Capucci, L., Fusi, P., Lavazza, A., Pacciarini, M. L., and Rossi, C. (1996). Detection and preliminary characterization of a new rabbit calicivirus related to rabbit haemorrhagic disease virus but nonpathogenic. *Journal of Virology* **70**, 8614–8623.

Coman, B. (1997). 'Rabbit Control and Rabbit Calicivirus Disease: A Field Handbook for Land Managers in Australia.' (Gillingham Printers and Packaging: Sydney.)

Cooke, B. D (1996). Analysis of spread of rabbit calicivirus from Wardang Island through mainland Australia (Project CS.236). Meat Research Corporation.

Fuller, H. E., Chasey, D., Lucas, M. H., and Gibbens, J. C. (1993). Rabbit haemorrhagic disease in the United Kingdom. *Veterinary Record* **133**, 611–613.

Ohlinger, V. F., and Thiel, H.-J. (1991). Identification of the viral haemorrhagic disease virus of rabbits as a calicivirus. *Revue Scientifique et Technique (Office International des Epizooties)* **10**, 311–323.

Peiro, V., and Seva, E. (1991). Maladie hemorrhagique virale du lapin de la garenne (*Oryctolagus cuniculus*) au sud-est de l'Espagne. In 'XXth Congress of the International Union of Game Biologists, Godollo, Hungary, August 21–26, 1991'. Pp. 752–757.

Villafuerte, R., Calvete, C., Gortazar, C., and Moreno, S. (1994). First epizootic of rabbit haemorrhagic disease in free living populations of *Oryctolagus cuniculus* at Donana National Park, Spain. *Journal of Wildlife Diseases* **30**, 176–179.

Williams, C. K., Parer, I., Coman, B. J., Burley, J., and Braysher, M. L. (1995). 'Managing Vertebrate Pests: Rabbits.' (Bureau of Resource Sciences & CSIRO Division of Wildlife and Ecology, Australian Government Publishing Service: Canberra.)

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