Prolonged decline in the abundance of wild European rabbits Oryctolagus cuniculus and high immunity level over three years following the arrival of rabbit haemorrhagic disease

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Prolonged decline in the abundance of wild European rabbits *Oryctolagus cuniculus* and high immunity level over three years following the arrival of rabbit haemorrhagic disease

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A free-living population of European rabbits *Oryctolagus cuniculus* was monitored over three years, from 1996 to 1998, following the arrival of rabbit haemorrhagic disease (RHD) in 1995. The survey was based on nocturnal observations of individually marked rabbits to determine population size and on blood collection to determine whether rabbits carried RHD antibodies. We directly confirmed RHD outbreaks by examination of dead rabbits and detection of RHD virus (RHDV). Only one further recurrence of RHD was detected by this method, in 1996. Contrary to the initial outbreak, this second occurrence did not result in a major decrease in population size. No direct evidence of other outbreaks was detected in 1997 and 1998. However, the sparse data did not enable us to conclude that RHD was absent. Indeed, seroconversions in individual rabbits were noticed throughout the three years and the level of population immunity remained high, proving that either RHDV or RHD-like viruses were present in the population. The influence of age, year and quarter on the serological status of the rabbits were analysed using a log-linear model. The selected model showed that the proportion of rabbits with RHD antibodies varied with year and period of the year. Three years after the first occurrence of RHD, the population had not recovered to its level prior to the first outbreak. We assumed that predation by red foxes could be partly responsible for keeping the population low.

Key words: epidemiology, foxes, France, immunity, Oryctolagus cuniculus, population size, predation, rabbit haemorrhagic disease

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Rabbit haemorrhagic disease (RHD) was first reported in China in 1984 (Liu, Xue, Pu & Quian 1984) and in Europe in 1988. Several studies have described its impact during a first outbreak on free-living populations of rabbits *Oryctolagus cuniculus* in Spain (Villafuerte, Calvete, Gortazar & Moreno 1994), France (Marchandeau, Chantal, Portejoie, Barraud & Chaval 1998a) and Australia (Mutze, Cooke & Alexander 1998, Saunders, Choquenot, McIlroy & Packwood 1998). However, there is little information on the

continued effect of RHD on populations after these first outbreaks. The main questions concern the evolution of the disease and changes in the size of the populations. Will RHD become endemic and repeatedly severely affect the populations? Will host populations recover to their initial level or will they repeatedly suffer major decreases due to successive outbreaks?

It is now known that RHD has been responsible for significant decreases in population size during initial outbreaks on free-living populations. In Australia, Mutze et al. (1998) recorded a mean mortality rate of 91%. Saunders et al. (1998) studied a first outbreak of RHD in three study areas. In two of them, the mortality rates were between 68 and 91%. In Europe, mortality rates were estimated at 55% in Spain by Villafuerte et al. (1994) and at about 45% in France by Marchandeau et al. (1998a), who recorded an annual mortality rate of 88% in adults in a population exposed to both RHD and myxomatosis. In this latter study the pattern of mortality was unusual in so far as (i) mortalities occurred throughout the year and were not confined to a relatively short period as commonly described elsewhere, and (ii) most rabbits previously had myxoma and RHD antibodies and were theoretically protected against both diseases. The authors assumed that the immunosuppressive characteristics of myxoma virus could be responsible for this particular pattern of mortality, inducing high mortalities by RHD in spite of the high immunity levels. However, a first outbreak of RHD does not necessarily induce a decrease in population size. Indeed, Saunders et al. (1998) noticed an increase in population size of 87% in a third study area and assumed that these differences in mortality rates could be due to time of arrival of RHD. When the outbreak occurs outside the breeding period, RHD only affects adults or subadults and the mortality is high. When it occurs during the breeding period, the mortality could be lower because juveniles less than two months old are resistant to the disease (Morisse, Le Gall & Boilletot 1991). In Italy, Capucci, Fusi, Lavazza, Pacciarini & Rossi (1996) isolated a non-pathogenic calicivirus related to the RHD virus (RHDV) that induce the production of RHD antibodies. The presence of such non-pathogenic RHDV-like viruses was already suggested by Rodak, Smid, Valicek, Vesely, Stepanek, Hampl & Jurak (1990) and Rodak, Smid & Valicek (1991), who detected RHD antibodies in rabbit sera collected in the Czech and Slovak Federal Republic before the first documented occurrence of RHD in Europe. It could also explain both the high levels of immunity recorded in numerous wild populations in Europe (von Maess, Ryll, Keyserlingk, Wenk & Pohlmeyer 1991, Trout, Chasey & Sharp 1997, Simon, Ortega, Maynar, Muzquiz, De Blas, Girones, Alonso & Sanchez 1998, Marchandeau, Ricci & Chantal 1998b, Marchandeau & Boucraut-Baralon 1999) and also the relatively low mortality rates due to RHD recorded in Spain and France. Only Saunders et al. (1998) have published results on the continuing impact of RHD on rabbit populations beyond the first occurrence of RHD. They showed that the two populations severely affected by RHD had not recovered to their initial level 12 months after the arrival of RHD.

The aim of the present work was to monitor a rabbit population during three years following the first occurrence of RHD (Marchandeau et al. 1998a) to determine whether a severely affected free-living population would recover to its initial level and whether RHD might become endemic. Therefore, we studied this population using capture and marking to record observations on changes in rabbit population size, occurrence of new RHD epizootics and changes in the serological status of the population. However, because RHD and RHD-like viruses induce the production of the same antibodies, hereafter called RHD antibodies, the presence of antibodies does not necessarily indicate an epizootic but expresses the immunity of the population, the antibodies being protective (Parra & Prieto 1990, Chasey, Trout & Edwards 1997).

Material and methods

Study area and data collection

The Chèvreloup arboretum (48°50'N, 02°06'E) is located close to Paris, France. The climate is oceanic with a continental influence. Mean annual rainfall is 606 mm and mean annual temperature is 10.3°C. It is a closed 200-ha park managed by the Museum National d'Histoire Naturelle. In this park, a 5-ha area was chosen on account of its high density of rabbit warrens and clearly defined as the study site.

Rabbits were caught at the warrens of the study site in wire cage-traps as described by Biadi & Le Gall (1993). Trapping sessions were carried out every three weeks from January to September in 1996 and 1997. In 1998, they were carried out every five weeks from January to September. In October of

each year, one capture operation of each warren was conducted using ferrets *Mustela furo*. At each capture, each rabbit caught was weighed and a blood sample was taken on a strip of blotting paper (Gilbert, Picavet & Chantal 1989, Chantal & Gilbert 1996). In addition, at its first capture, the sex of each animal was determined and they were individually marked with ear tags covered with reflective paper of different distinctive colour combinations suitable for long-distance identification. Nocturnal resightings using a 100-W halogen spotlight and binoculars were conducted every two months in 1996 and 1997 and every three months in 1998. During each resighting session we searched for marked rabbits on three nights over the entire rabbit foraging area around the study site. Moreover, the number of red foxes *Vulpes vulpes* encountered each night was also recorded. A red fox abundance index was defined as the mean number of foxes recorded nightly during the resighting session.

Antibody detection and post-mortem examinations

Rabbits with RHD die quickly and without developing readily detectable external signs of disease. Therefore, it is not possible to detect an outbreak from examination of captured rabbits. The only way to document an occurrence of RHD is to collect dead rabbits and to carry out post-mortem examinations to determine the cause of their death. Corpses collected during our study were examined by the Departmental Veterinary Laboratory of Maine et Loire, France. RHDV were detected using HA tests (Marchandeau et al. 1998a). In addition, the examination of the serological history of some rabbits caught repeatedly during the study enabled detection of seroconversions, indicating the rabbits' likely exposure to RHDV or RHDV-like viruses. Thus, it was possible to determine periods when RHDV or RHDV-like viruses were circulating in the population.

The detection of RHD antibodies was carried out at the Associated Laboratory of Molecular Microbiology, Institut National de la Recherche Agronomique - Ecole Nationale Vétérinaire de Toulouse using ELISA (Enzyme Linked Immunosorbent Assay) techniques developed by Laurent, Vautherot, Madelaine, Le Gall & Rasschaert (1994).

Data analysis

We assessed the changes in population size by combining the capture and resighting data following an enumeration method, the population size at any time being estimated as the number of rabbits known to have been alive at that time. A rabbit was considered to be alive up to the time of its last capture or resighting (Krebs 1966, Wood 1980). Indeed, it was not possible to use capture-mark-recapture models because of a strong heterogeneity in recapture histories of individual rabbits (Lebreton, Burnham, Clobert & Anderson 1992).

The statistical tests were performed using the SPSS program. The proportion of rabbits with RHD antibodies was analysed using log-linear models under multinomial assumption. The effects of year, period of the year (quarters) and body mass were used as explanatory variables. Quarters were arbitrarily chosen as January-March, April-June, July-September and October-December, and two age-classes were defined, adults and juveniles. Rabbits were considered to be juveniles during the year of their birth. When rabbits were captured several times in any given year, we considered only the antibody status of the rabbit at its first capture to ensure that samples were statistically independent. We used the screening effects method described by Brown (1976). In a first step, the interactions between the studied variables and the presence of antibodies were analysed following two methods. The first was conducted on the whole table to test the interactions with a partial association. Each interaction was tested by the difference of fitting between a model including all the interactions of equal rank to the tested interaction and the model including all but the tested interaction. The second was conducted on marginal tables to test the interactions with a marginal association. The interactions were tested by the difference of fitting between

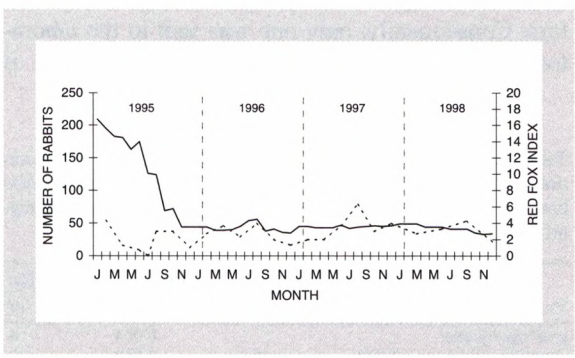

Figure 1. Number of rabbits in the population studied and the red fox index defined as the mean number of red foxes encountered per night during resighting sessions during 1995-1998. The first outbreak of RHD occurred in 1995. The solid line indicates the rabbit population size and the dashed line the red fox index.

Table 1. Quarterly proportion of adult and juvenile rabbits with RHD antibodies during 1996-1998. For rabbits caught several times a year, only serological data from first capture were considered. Numbers in parentheses indicate sample sizes.

	1996		1997		1998	
Quarter	Adults	Juveniles	Adults	Juveniles	Adults	Juveniles
January-March	71% (7)	100% (6)				
April-June	0 % (2)	17% (29)	100% (6)	70% (23)		
July-September	100% (1)	67% (15)	100% (2)	85% (13)	17% (6)	0% (2)
October-December	100% (12)	78 % (9)	100% (12)	100% (8)	100% (17)	100% (3)

the model including the tested interaction and the lower rank effects, and the model including only the lower rank effects. In a second step we selected the final model including all the significant interactions. The tested interactions without partial and marginal association were excluded, those with both partial and marginal associations were included and those with one association were tested by comparison of models using likelihood ratio tests.

Results

After the initial population decrease during 1995, the abundance of rabbits at Chèvreloup did not recover and remained at the low level recorded at the end of 1995 (Fig. 1). The marked population comprised 209 rabbits in January 1995 and only 30 rabbits in December 1998. The red fox index decreased with the number of rabbits during the first half of 1995, but then recovered to its level prior to the first RHD outbreak.

A second epizootic of RHD was recorded in 1996. A total of 19 dead rabbits were discovered between 30 September and 13 October on the overall 200-ha park. Six of these were discovered in the 5-ha study area. Most corpses were partly scavenged by predators. Consequently, only one was sent to the laboratory where analysis confirmed RHD as the cause of death. No new epizootic was detected in 1997 and 1998. Otherwise, 13 seroconversions to RHD were recorded; seven occurred during May-November 1996, three during June-December 1997 and two during August-November 1998. These data confirm the presence of RHDV or RHDV-like viruses throughout the three years of our study.

In 1996, antibody prevalence seemed to increase throughout the year for both adults and juveniles (Table 1). In 1997, the prevalence was high throughout the year. Data were too sparse to detect any trend in prevalence in 1998. Marginal and partial associations showed that year and quarter effects on seropositivity were highly significant (Table 2). Age and age*quarter effects were significant only for marginal association, which was insufficient to include them straight off in the model. We then compared three models, year + quarter, year + quarter + age and year + quarter + age + age*quarter to determine the final model. The likelihood ratio tests showed that neither the introduction of age ($G^2 = 3.088$, df = 1, $P > 0.05$), nor the introduction of age*quarter ($G^2 = 7.681$, df = 3, $P > 0.05$) into the model increased the significance of the adjustment. Therefore, the final model was year + quarter, showing that the proportion of rabbits having antibodies depended on year and on period of the year.

Table 2. Results of the screening for partial and marginal associations. There is no partial association for the highest rank association. * indicates test significance at $P \leq 0.05$; *** indicates test significance at $P \leq 0.001$.

	Partial association			Marginal association		
Variable	G^2		df	G^2		df
year*age*quarter				0.004		4
year*age	3.333		2	2.812		2
age*quarter	3.797		2	8.882	*	2
year*quarter	5.896		6	9.581		6
year	36.008	***	2	20.970	***	2
age	3.088		1	17.617	***	1
quarter	45.805	***	3	47.548	***	3

Discussion

Following a strong decrease in population size due to the first epizootic of RHD, the population at Chèvreloup had not recovered to its initial level three years later and remained at the low level reached at the end of this initial outbreak. The enumeration method used to estimate population size most likely provided an underestimate towards the end of the study, due to fewer data being available for the estimation. However, considering the difference in estimated population size between the beginning of 1995 and the end of 1998, this underestimation is insuffi-

cient to mask a recovery of the population. In Australia, Saunders et al. (1998) noticed that two populations severely affected by RHD had not recovered to their initial level 12 months after the arrival of RHD. Nevertheless, they retained the normal sequence of seasonal increases and decreases typical of population dynamics before the first outbreak of RHD. This suggests, as shown by our results, that RHD has a lower impact on populations when it occurs repeatedly after the initial outbreak.

During our study, only one new outbreak was documented in 1996. It did not induce any further significant reduction in population size, which remained almost constant throughout the year. However, a lack of direct evidence of repeated outbreaks in 1997 and 1998 does not prove that RHD did not occur. In spite of a careful survey of the area, the disease could have remained undetected, and in fact seroconversions show that the RHDV or RHD-like viruses were present and active in the population.

Different hypotheses could explain the non-recovery of the population. Undetected outbreaks of RHD may have caused high mortalities in juveniles and reduced the recruitment of young rabbits maintaining the population at its low level. This could explain the absence of seasonal fluctuations of the population during the study. The predation by red foxes may also have had an important role. Indeed, we noticed that, throughout the three years, the red fox index did not decrease with the reduction in rabbit numbers. Consequently, the high number of foxes relative to the number of rabbits suggests that predation could have delayed the recovery of the rabbit population. Other studies have also suggested that predation might limit population recovery after a large decrease due to factors such as myxomatosis (Moore 1956, Jaksic & Yanez 1983), starvation during a hard winter (Erlinge, Göransson, Högstedt, Jansson, Liberg, Loman, Nilsson, von Schantz & Sylvén 1984) or food shortage (Gibb, Ward & Ward 1978, Newsome, Parer & Catling 1989). More generally, the influence of predation in preventing the increase of low density rabbit populations was pointed out by Gibb, Ward & Ward (1969), Lloyd (1981) and Trout & Tittensor (1989).

The event of RHD in 1996 did not induce a strong reduction of population size, as was observed in 1995. Saunders et al. (1998) reported that mortality rates varied among areas in Australia. They suggested that the level of mortality could depend on the period in which the disease appears: the mortality seemed lower when the outbreak occurred during the breeding season. Such outbreaks do not severely affect juveniles less than two months old as these are relatively resistant to RHD and suffer low mortality, yet still produce antibodies when they are exposed to the virus (Morisse et al. 1991). This period effect cannot explain the variation in mortality rates recorded during 1995 and 1996, as outbreaks were detected in both years in autumn, when there were few juveniles less than two months old (Marchandeau et al. 1998a). The difference in mortality rates could be related to the repetition of outbreaks. Indeed, rabbits that survived the first epizootic had antibodies and were protected when subsequent outbreaks occurred. They would also produce juveniles that would likely have a temporary protection conferred by maternal antibodies. Differences between the successive outbreaks recorded in our study area are also apparent from the evidence of mortality detected by the observers. In 1995, the mortality rate was high and Marchandeau et al. (1998a) estimated that 300 rabbits died throughout the year. However, only one was found, suggesting that most of them died in their warrens. In 1996, the mortality was lower, but 19 rabbits were found in the arboretum, and of those, six were found in the study area. These results suggest that the localities in which rabbits died from RHD may have differed between outbreaks, as was previously suggested by Marchandeau et al. (1998a), from comparative studies with those of Villafuerte et al. (1994) in Spain. Indeed, some studies show that many rabbits may die away from the warrens (Villafuerte et al. 1994) whereas others indicate that rabbits die inside the warrens (Marchandeau et al. 1998a, Mutze et al. 1998). RHD seems to have different expression in terms of the rabbits' behaviour at the time of death, which may be related to viral strain, environmental conditions or intrinsic characteristics of the population. Another factor affecting the pattern of mortality might be the interaction between RHDV and other pathogenic agents. In particular, the immunosuppressive effect of myxoma virus on nematode and cestode infections was reported by Boag (1988), and Marchandeau et al. (1998a) suggested it to be related to the extent of mortality caused by RHD during the first outbreak in our study area.

In spite of sparse data, we noticed that in 1997 both juveniles and adults were permanently protected against RHD, as shown by the serological status of the population, most rabbits carrying RHD antibodies throughout the year. In 1996, the proportion of

rabbits with RHD antibodies was rather low during the first half of the year and then increased. These observations were confirmed by the adjustment to a log-linear model that outlined the effects of the year and the quarter on the proportion of rabbits with antibodies. The low seroprevalence recorded in the first part of 1996 may have enabled the occurrence of RHD in 1996 and may also have affected the mortality level, which was lower than in 1995 but still high enough to be detected. On the other hand, the non-detection of RHD in 1997 could have been due to the high protection of the population conferred by the high antibody prevalence. Such protection may be related to the presence of RHD-like viruses, and may mean that RHD either does not occur or that it affects few rabbits and induces a low and undetectable mortality rate.

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