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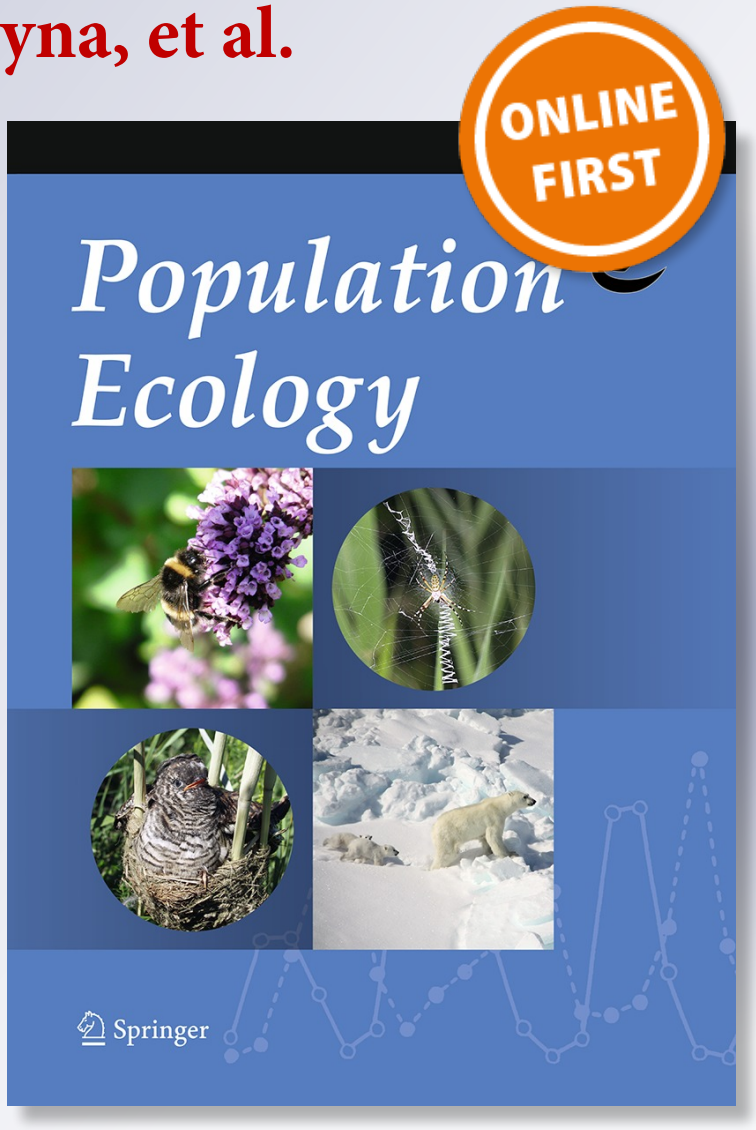

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ORIGINAL ARTICLE

Experimental study on the effect of cover and vaccination on the survival of juvenile European rabbits

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Abstract In Mediterranean ecosystems, the European rabbit is a keystone species that has declined dramatically, with profound implications for conservation and management. Predation and disease acting on juveniles are considered the likely causes. In the field, these processes are managed by removing predators, increasing cover to reduce predation risk and by vaccinating against myxomatosis. These manipulations can be costly and, when protected predators are killed, they can also be damaging to conservation interests. Our goal was to test the effectiveness of cover and vaccination on juvenile survival in two large enclosures, free of mammalian predators, by adding cover and vaccinating juveniles. Rabbit warrens were our experimental unit, with nine replicates of four treatments: control, cover, vaccination, and cover and vaccination combined. Our results showed that improved cover systematically increased juvenile rabbit survival, whereas vaccination had no clear effect and the interactive effect was negligible. Our experimental data suggest that improved cover around warrens is an effective way of increasing rabbit abundance in Mediterranean ecosystems, at least when generalist mammalian predators are scarce. In contrast the vaccination programme was of limited benefit, raising questions about its efficacy as a management tool.

Keywords Capture-mark-recapture · Habitat management · Mediterranean ecosystems · Myxomatosis · *Oryctolagus cuniculus* · Predation risk

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Introduction

Disease and predation can profoundly affect animal populations (e.g., Sinclair and Arcese 1995; Connors et al. 2010). Studies of their impact on fitness have tended to focus on one process, yet in reality both processes can operate simultaneously, and may well interact in the field. On one hand, disease is known to increase the likelihood of animals being killed by predators (Temple 1987; Møller and Erritzoe 2000); whilst on the other hand, the risk of predation can have severe sub-lethal effects, affecting the incidence of disease and long-term survival and fitness (e.g., Navarro et al. 2004; Sheriff et al. 2011). An understanding of the relative importance of such population processes is crucial to develop effective management strategies aimed at species conservation and recovery.

In Mediterranean ecosystems of southwest Europe, European rabbits (*Oryctolagus cuniculus*) are considered a keystone species, mainly because they represent an important prey for nearly 40 predator species (Villafuerte 1994; Delibes-Mateos et al. 2007). Rabbits are also an important small-game species in Spain, being hunted in over 30,000 private hunting estates covering more than 70 % of the country (Villafuerte et al. 1998). Yet rabbit populations have declined dramatically in recent decades with consequences for conservation and hunting (Angulo and Villafuerte 2003). Declines have generated expensive game management efforts to stabilize and increase populations, often with little supporting evidence for their efficacy (Delibes-Mateos et al. 2008).

Viral diseases, such as myxomatosis, and predation are thought to have played a major role in rabbit population declines (Villafuerte et al. 1994; Angulo 2003; Moreno et al. 2007; Cotilla et al. 2010). Both of them operate primarily on juvenile rabbit survival (Smith and Trout 1994; Villafuerte 1994; Calvete et al. 2002; Angulo and Villafuerte 2003; Cotilla et al. 2010). In the wild, the pattern of myxomatosis outbreaks is closely related to the recruitment of susceptible juvenile rabbits during the breeding season (Calvete et al. 2002). Juvenile rabbits are virtually all infected in their first year of life and they either sucumb or survive to the disease (e.g., through maternal conferred immunity). Hence, the epidemiological pattern of myxomatosis is characterized by a rapid increase of antibodies in juvenile rabbits as a consequence of the annual outbreak, resulting in a high prevalence of antibodies in adult rabbits (Calvete et al. 2004). Similarly, predation acts predominantly on the younger age classes (Villafuerte 1994; Cotilla and Villafuerte 2007; Tablado et al. 2012). This predation pressure on juvenile rabbits is imposed mainly by raptors during winter and spring, potentially causing the loss of over 60 % of the reproductive potential of the population (Villafuerte 1994). The impact of avian predation is therefore a major conservation concern in southern Europe (Viñuela and Villafuerte 2003).

Predation and disease are also known to interact in lagomorphs (Tablado et al. 2012). For example, diseases may make rabbits more vulnerable to predation and high predation risk may influence physical condition, compromising immunity and making rabbits more vulnerable to disease (Dunsmore et al. 1971; Villafuerte et al. 1997; Moreno et al. 2007; Sheriff et al. 2011; Tablado et al. 2012).

Attempts to reduce levels of predation focus primarily on the direct legal control of predators and, indirectly, on the increase of the extent of available cover, or on the illegal killing of protected species (e.g., Moreno et al. 1996; Villafuerte and Moreno 1997; Villafuerte et al. 1998; Lombardi et al. 2003). Management to reduce the impact of diseases focuses on vaccination campaigns using commercial vaccines (Calvete et al. 2004; Guitton et al. 2008). These commercial vaccines succeed in immunizing domestic rabbits, but they appear to be less effective in the field (Ferreira et al. 2009). Rabbit management can be very costly (e.g., Delibes-Mateos et al. 2008) and, in the case of illegal predator control, it can have important conservation implications (Villafuerte et al. 1998). Yet little attempt has been made to understand the relative influence of both processes (predation and disease) on the effectiveness of legal forms of management.

The goal of this study was to experimentally manipulate cover and susceptability to disease through vaccination and test their effectiveness at improving juvenile rabbit survival. Here we focus on juvenile rabbit survival, as an indicator of population quality and a crucial parameter for population persistence (Smith and Trout 1994; Angulo and Villafuerte 2003; Cotilla and Villafuerte 2007). We worked in large enclosures, where mammalian predators were excluded, as is the case in many managed hunting estates, and where there was grass, but little other cover. We increased cover around rabbit warrens and manipulated susceptability to disease by vaccinating juvenile rabbits against myxomatosis using a standard, commercial vaccine.

We expected that cover would improve juvenile survival directly by reducing predation by raptors, and indirectly by reducing the impact of myxomatosis. We anticipated that myxomatosis would outbreak half way through the experiment. We expected that vaccination would improve juvenile survival directly by reducing the impact of myxomatosis, and indirectly by reducing the levels of predation. Specifically, our predictions were that: (1) rabbits in control plots would always have lower survival; (2) rabbits in control plots would have higher survival before than after the disease outbreak; (3) cover would always improve juvenile survival relative to control plots, equally before and after the outbreak; (4) vaccination would increase

survival only after the outbreak, being similar to control before the outbreak; and (5) combined cover and vaccination treatments would have similar survival to that of cover alone before the outbreak, but the highest survival after the outbreak.

Methods

Study area

The study area (Los Melonares) is situated in the south of the Sierra Norte Natural Park of Seville, Sierra Morena, SW Spain. It is characterised by a typically Mediterranean climate, with hot, dry summers and temperate, wet winters. The area consists mainly of grassland and scrubland including *Cistus ladanifer*, *Pistacia lentiscus, Myrtus communis*, *Lavandula stoechas* and *Retama sphaerocarpa*. The subspecies of wild rabbit occurring in the study area is the *O. cuniculus algirus*. Eleven species of raptors are known to nest in the area, many of which preyed extensively on rabbits (Delibes-Mateos et al. 2007).

Experimental design

In 2002, four 200 × 200 m experimental plots were built, approximately 1 km from each other in the grassland area, in the context of a rabbit recovery program (see Rouco et al. 2008, 2011 for more details). No natural or artificial warrens were previously present in any plot. Two of these plots were provided with an exclusion fence to prevent the entry of terrestrial mammalian predators. Fenced enclosures are an increasingly used management technique in southwest Europe, to support high densities of rabbits and provide food for threatened populations of birds of prey (Ferreira and Delibes-Mateos 2010; Ferrer et al. 2013; Guerrero-Casado et al. 2013). Additionally, these are convenient systems to simulate legal predator control, one of the most important management measures implemented in this region to boost rabbit populations (Angulo 2003). For this reason, in this paper we focus on the two fenced plots only. Fences were 3 m tall and 1 m underground (4 × 4 cm mesh), with an electric wire at the top, to prevent mammalian predators entering. Small terrestrial predators were excluded by attaching another fence of smaller mesh size at the base (120 cm tall, 1.5 × 1.5 cm mesh).

In each plot 18 artificial warrens were built and were regularly distributed in four alternate lines of four or five warrens approximately 40 m apart (Rouco et al. 2011). Two different warren sizes were built: large (6 per plot) and small (12 per plot). Large warrens were exactly four times bigger than the small ones. Each warren was constructed using wooden pallets, wood, stones and soil (Rouco et al. 2008) and surrounded with a wire net (approximately 1 m high, 0.5 m underground, 1.5 cm mesh). Three rabbit traps were placed around the small warrens and five around the large ones. Rabbits could only leave or enter warrens by passing through these traps. Food and water were provided ad libitum next to each warren in both plots throughout the experiment ensuring that these resources were never limiting. Rabbits were live-trapped in all warrens in the two plots over 2–3 consecutive nights every month (usually the last week of each month) from March to October 2007. At their first capture, animals were marked with individually numbered ear tags and measured (sex, weight, tarsus and ear length).

Predation and disease were manipulated as follows. Warrens were randomly allocated to one of the following four treatments: control (no treatment), cover, vaccination, or both cover and vaccination. In total, there were nine warrens (3 large and 6 small) in each treatment split between the two plots. The impact of raptor predation was manipulated by adding cover to the surroundings of the appropriate warrens (e.g., Richardson and Wood 1982). Cover was added in February 2007 and consisted of six wooden pallets (2 × 1 m) placed in the immediate vicinity of the warren exits. These provided cover for rabbits to move to and from their feeding areas. To manipulate the impact of myxomatosis, all juvenile rabbits (weight <900 g; Soriguer 1981; Villafuerte 1994) were either injected with 0.5 ml of a commercial vaccine against myxomatosis (POX-LAP from OVEJERO Laboratories, León, Spain), or a 0.5 ml saline control solution, at their first capture. Myxomatosis was known to be consistently present in the population with typical annual outbreaks in the summer (Villafuerte et al. 1994; Calvete et al. 2002; Rouco et al. 2008), in contrast to RHD (Rabbit Hemorrhagic Disease) for which outbreaks are extremely irregular. In 2007, the myxomatosis peak was detected in July when nearly 50 % of juvenile rabbits showed symptoms of the disease, regardless of treatment (Ferreira et al. 2009), and so, for analyses purposes, we considered this month to represent the disease peak. Finally, blood samples were collected in two occasions (April and October 2007, pre- and post-outbreak periods, respectively) to detect antibodies against myxomatosis in juvenile rabbits as a way to check immunization against myxomatosis, since seropositivity would indicate which animals could potentially survive after the disease outbreak. The details on the seroprevalence analysis and results are throroughly presented in Ferreira et al. (2009).

Capture-mark-recapture survival analysis

We used capture-mark-recapture techniques (Lebreton et al. 1992) to test our predictions about the effects of cover

and vaccination on juvenile survival. First we built an initial capture history database spanning all sampling occasions (March to October 2007) with all juvenile rabbits grouped by treatment in order to test that the dataset met the assumptions underlying capture-mark-recapture analyses (Lebreton et al. 1992). We tested these assumptions by applying the goodness-of-fit tests available in the program U-CARE 2.3 (Choquet et al. 2005). Then we modified the structure of the capture-recapture dataset according to the biases detected, and performed further goodness-of-fit tests of dispersion in MARK 6.0 (White and Burnham 1999). Once we had a suitable general starting model that fitted the data adequately, we incorporated plot, warren size and time varying age (since some juveniles became adults during the experiment) as covariates of both survival and detection probability.

Subsequently we used MARK 6.0 to model survival and detection probability, using the Akaike's Information Criterion modified for small sample sizes (AIC_c) in order to assess model fit (Burnham and Anderson 2002). We started by investigating the influence of covariates, infection period and experimental treatment on detection probability. Models accounting for infection period were designed to fit a hypothetical difference in estimates before and after the outbreak of myxomatosis in July. This was achieved by merging pre-outbreak (March–June) and post-outbreak (July–October) time dependent parameters separately. We then investigated the influence of covariates, infection period and experimental treatment in survival rates.

To test for an effect of myxomatosis on juvenile survival, we assessed whether infection period explained a significant part of the temporal variation in survival using an ANODEV test (Grosbois et al. 2008). The test included a model with constant survival for all treatments, a model with time dependent survival for all treatments, and a model where pre-/post-outbreak survival parameters differed additively for all treatments.

To assess differences in juvenile survival between experimental treatments, we used treatment contrasts, where model fit was assessed using AIC_c. Differences between pairs of treatments were assessed by comparing models with (1) equal survival parameters for the pair of treatments, (2) with different parameters before and after the outbreak of myxomatosis, (3) with different parameters only before the outbreak, or (4) with different parameters only after the outbreak. In addition, similar contrasts were used to assess whether cover and vaccination had an additive or interactive effect in the combined treatment before and/or after the outbreak. Because transience in juvenile survival was detected (see Electronic Supplementary Material [ESM]), estimates reported in the results and discussion sections refer to the non-transient class. Estimates for the transient class are provided in ESM. In order to account for model uncertainty, parameter estimates reported in this manuscript are model averaged across the best set of models with refined detection probability and survival rates (Burnham and Anderson 2002). Results and contrasts reported are based on differences on the logit scale, since we used the logit link throughout the analysis in MARK (White and Burnham 1999).

Results

Between March and October 2007, 1312 juveniles were live-trapped, 595 of which corresponded to new captures (details in Table S1 in ESM). The mean number of juvenile rabbits captured per warren per month was 16.60 ± 1.300 (standard error). Initial models suggested that neither treatment nor infection period affected probability of detecting rabbits (Table S4 in ESM). Refinement of survival parameterisation indicated a large influence of plot and age, and a small influence of warren size on juvenile survival (models 10–14, Table 1). Accounting for treatment improved model fit (compare models 17 and 20, 19 and 22, 15 and 18, Table 1).

Model fit was not improved by accounting for infection period (models 20–22, Table 1). However, examination of monthly survival estimates from an additive time dependent model (model 17 in Table 1) showed a marked decrease in juvenile survival in August, suggesting that the impact of a July outbreak of myxomatosis might be reflected with 1 month delay in juvenile survival (Table S3 in ESM). Thus, we fitted a further set of models with a delayed impact of the outbreak in survival, e.g., where the pre-outbreak period ran from March to July (instead of June as previously considered), while the post-outbreak period ran from August to October. This set of models showed a better fit than previous models (compare models 15, 16 and 18 with their time varying/infection period equivalents, Table 1).

The ANODEV test indicated that myxomatosis explained a significant part of the time variation in survival ($F_{1,22} = 18.90$, $P < 0.001$), causing a substantial reduction in mean survival rates across all treatments (Fig. 1). Between treatment contrasts (Table 2) suggested that cover improved juvenile survival compared to control, especially before the outbreak (estimates for non-transients rabbits pre-outbreak cover = 0.939, 95 % CI [0.848, 0.977], control = 0.907 [0.807, 0.958]; post-outbreak cover = 0.325 [0.211, 0.464], control = 0.250 [0.163, 0.380]). However, vaccination did not improve juvenile survival in relation to controls, (pre-outbreak vaccinated = 0.906 [0.809, 0.957]; post-outbreak vaccinated = 0.271 [0.180, 0.387]). In the combined treatment, juvenile survival was similar to control pre-outbreak (0.903 [0.790, 0.958]) and higher than control

Table 1 Summary of the model selection process

Modelling step	Model number	Model specification	AIC_c
General starting model	1	Φ (a2 + t) + Age + Plot + WSize, $p(t)$ + Age + Plot + WSize	2,861.77
Detection probability covariates	2	Φ (a2 + t) + Age + Plot + WSize, $p(t)$ + Age + Plot	2,859.99
	3	Φ (a2 + t) + Age + Plot + WSize, $p(t)$ + Age + Plot + WSize	2,861.77
	4	Φ (a2 + t) + Age + Plot + WSize, $p(t)$ + Age + WSize	2,867.41
	5	Φ (a2 + t) + Age + Plot + WSize, $p(t)$ + Plot + WSize	2,947.49
	6	Φ (a2 + t) + Age + Plot + WSize, $p(t)$	2,950.61
Detection probability, infection period and treatment	7	Φ (a2 + t) + Age + Plot + WSize, $p(t)$ + Age + Plot	2,859.99
	8	Φ (a2 + t) + Age + Plot + WSize, $p(t$ + treat) + Age + Plot	2,861.57
	9	Φ (a2 + t) + Age + Plot + WSize, $p(t$ + inf) + Age + Plot	2,870.68
Survival rates covariates	10	Φ (a2 + t) + Age + Plot + WSize, $p(t)$ + Age + Plot	2,859.99
	11	Φ (a2 + t) + Age + Plot, $p(t)$ + Age + Plot	2,861.44
	12	Φ (a2 + t) + Age + WSize, $p(t)$ + Age + Plot	2,863.71
	13	Φ (a2 + t) + Plot + WSize, $p(t)$ + Age + Plot	2,871.40
	14	Φ (a2 + t), $p(t)$ + Age + Plot	2,877.53
Survival rates, infection period and treatment	15	Φ (a2 + delayinf + treat) + Age + Plot + WSize, $p(t)$ + Age + Plot	2,855.96
	16	Φ (a2 + delayinf × treat) + Age + Plot + WSize, $p(t)$ + Age + Plot	2,856.06
	17	Φ (a2 + t + treat) + Age + Plot + WSize, $p(t)$ + Age + Plot	2,856.83
	18	Φ (a2 + delayinf) + Age + Plot + WSize, $p(t)$ + Age + Plot	2,857.52
	19	Φ (a2 + inf + treat) + Age + Plot + WSize, $p(t)$ + Age + Plot	2,859.63
	20	Φ (a2 + t) + Age + Plot + WSize, $p(t)$ + Age + Plot	2,859.99
	21	Φ (a2 + inf × treat) + Age + Plot + WSize, $p(t)$ + Age + Plot	2,863.02
	22	Φ (a2 + inf) + Age + Plot + WSize, $p(t)$ + Age + Plot	2,863.40
	23	Φ (a2 + treat) + Age + Plot + WSize, $p(t)$ + Age + Plot	2,889.66

For every successive modelling step, the complete set of models considered is given, with decreasing level of support based on Akaike's Information Criterion (AIC_c) scores

Note: Symbols: Φ survival rate, p detection probability, *treat* treatments (categorical: control, cover, vaccination, or both cover and vaccination), *a2* time since marking subclasses (categorical: 1 month since marking vs >1 month), *inf* myxomatosis infection periods (categorical: pre-outbreak vs. post-outbreak), *delayinf* infection period with one-month delay in the impact of myxomatosis, t = time dependent parameter, *Age* time-varying covariate age, *Plot* covariate experimental plots (fenced enclosures), *WSize* covariate warren size

post-outbreak (0.326 [0.189, 0.502]). Estimates above show that survival in the combined treatment was similar to control and vaccination treatments before the outbreak and similar to that of cover after the outbreak.

Discussion

This experiment demonstrated that, in the absence of mammalian predators, juvenile rabbit survival was highest in warrens with additional cover. However, the level of improved survival was relatively modest in the pre-outbreak period (with a 3.5 % increase relative to controls) but rather important during the post-outbreak phase (26.3 % increase relative to controls). In contrast, vaccination had no measurable effect on juvenile survival, despite the fact that the myxomatosis outbreak had a large impact on juvenile survival across all treatments.

The unexpected observation that vaccination did not improve juvenile survival could be related to different causes. For example, it has been shown that vaccination can have adverse effects on rabbit physiology (Peeters et al. 1995; Twigg et al. 1997). Some secondary effects include mild fevers (Marlier et al. 2000) and lethargy, making juveniles less responsive and more vulnerable to predation or even death. There is also the possibility that vaccination failed to immunize juvenile rabbits or failed to improve the survival of this age class at the population level. The fact that in a previous study (Ferreira et al. 2009) the proportion of juveniles seropositive to myxomatosis was similar between vaccinated vs. non-vaccinated, both before and after the disease outbreak, may corroborate the first possibility. In fact, in the post-outbreak period (October 2007), all of the juveniles sampled in Ferreira et al. (2009) were seropositive to the disease regardless of whether they had been vaccinated or not against

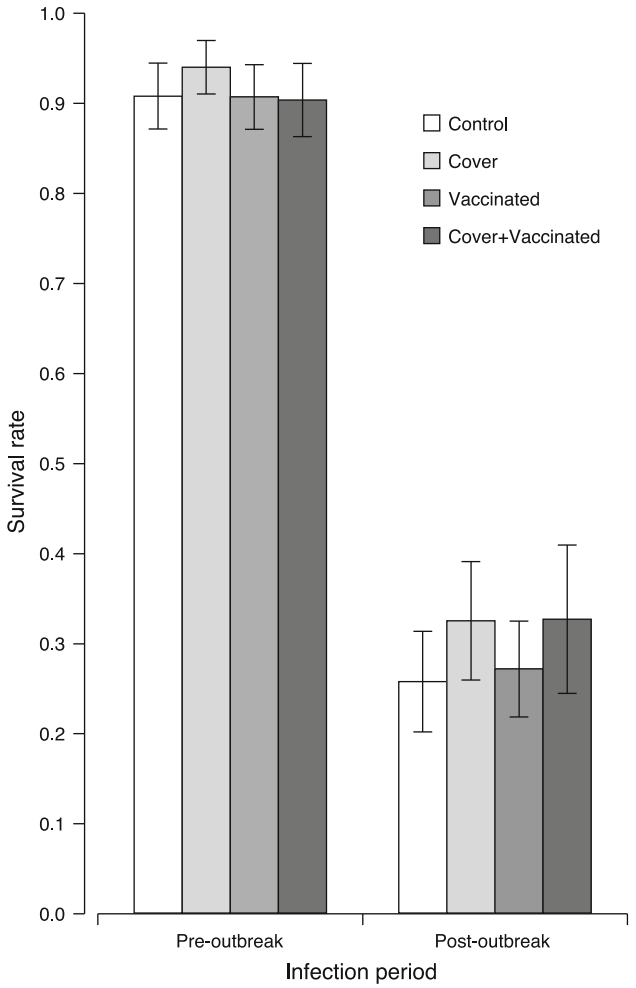

Fig. 1 The combined effect of treatment and infection period (pre-outbreak: March–July and post-outbreak: August–October) on juvenile survival across the whole experiment and obtained from Time Since Marking (TSM) models. The graph shows model averaged survival mean estimates (±SE) for non-transient juveniles

myxomatosis prior to the outbreak, which suggests that, in our experiment, vaccinating against myxomatosis was redundant. Vaccination campaigns in the field can additionally be influenced by the highly variable spatial–temporal pattern exhibited by the virus (Villafuerte et al. 2000), which is a function of a variety of factors such as the virulence of circulating strains or population density (Arthur and Louzis 1988). It is therefore possible that the vaccine we used (developed for domestic rabbits), which is the only one available against myxomatosis (regardless of the source laboratory of production), might be ineffective at protecting wild specimens against all the strains of the virus occurring in the wild. The latter is supported by the report of cases where highly virulent strains have decimated even vaccinated rabbits in rabbitries, e.g., in Greece (Kritas et al. 2008). Whatever the mechanism it seems clear that vaccination programmes in wild populations are likely to be costly (e.g., average 4,790 euros/year per 2,000 ha; Angulo 2003) and potentially ineffective (Ferreira et al. 2009).

Our results clearly show that cover improves juvenile rabbit survival in areas where raptors are their main predators. Avian predation is particularly heavy on juveniles up to 3 months of age (Villafuerte and Viñuela 1999) and this could explain the success of the cover treatment in our study. Cover is fundamental for juvenile rabbits as a resource that increases refuge opportunities from predators (Moreno et al. 1996), decreases the need for group vigilance (Villafuerte 1994), and reduces individual distances to forage (Villafuerte and Moreno 1997).

Across the Iberian Peninsula rabbits seem to be recovering better in areas where several management activities have been carried out simultaneously and regularly (Delibes-Mateos et al. 2008). In particular, improved rabbit recovery has been observed in hunting estates where both mammalian predator control and habitat management are frequently applied (Angulo 2003; Delibes-Mateos et al. 2008). Conversely, rabbit populations did not change in places where restocking or vaccination were the main management activities (Delibes-Mateos et al. 2008). Rabbits are such an important component of Mediterranean ecosystems (Delibes-Mateos et al. 2007) that there is an urgent need to restore healthy, wild populations. This will benefit both conservation and human wellbeing and livelihoods. Whilst rabbit populations are at low density, protected species of predators are likely to continue to be vulnerable to direct or indirect killing by hunters (Márquez et al. 2013). Identifying the most effective management techniques to improve rabbit abundance is therefore urgently needed. The results from this study suggest that habitat management to improve cover is likely to be most effective at improving survival of juvenile rabbits. There is now a need to understand the optimum strategies for managing cover and other habitat features targeted at the European rabbit (Ferreira et al. 2013).

Despite clear results, caution needs to be taken before extrapolating them to natural populations, since they are based on only two enclosures studied over 8 months and during one single outbreak. Our rabbits were free from mammalian predators and were provided with ad libitum food and water at all times. They were therefore in good condition and may have higher survival than wild populations. For example, the average juvenile survival in Doñana National Park was 0.45 (Villafuerte 1994), which is considerably lower than in our study. Also the concomitant influence of RHD was not explored in our study, although this disease was not detected in our study area during 2007. Therefore, further research should explore (1)

Table 2 Results of contrasts fitted to assess differences in juvenile survival rate (Φ) between treatments

Contrast set	Model fitted	AIC_c interaction set	AIC_c additive set
Baseline model		2,856.06	2,855.96
cover vs. control	Φ cover = Φ control	2,858.91	2,859.61
	Φ cover ≠ Φ control both periods	2,856.06	2,855.95
	Φ cover ≠ Φ control pre-outbreak only	2,854.83	2,852.96
	Φ cover ≠ Φ control post-outbreak only	2,858.61	2,861.48
vaccinated vs. control	Φ vaccinated = Φ control	2,852.12	2,853.91
	Φ vaccinated ≠ Φ control both periods	2,856.06	2,855.95
	Φ vaccinated ≠ Φ control pre-outbreak only	2,854.18	2,855.97
	Φ vaccinated ≠ Φ control post-outbreak only	2,854.03	2,853.76
cv[a] vs. control	Φ cv[a] = Φ control	2,854.93	2,854.32
	Φ cv[a] ≠ Φ control both periods	2,856.06	2,855.95
	Φ cv[a] ≠ Φ control pre-outbreak only	2,856.87	2,855.68
	Φ cv[a] ≠ Φ control post-outbreak only	2,854.67	2,852.36
cv[a] vs. cover + vaccinated	Φ cv[a] = Φ cover + vaccinated	2,888.50	2,855.54
	Φ cv[a] ≠ Φ cover + vaccinated both periods	2,856.06	2,855.95
	Φ cv[a] ≠ Φ cover + vaccinated pre-outbreak only	2,854.15	2,855.68
	Φ cv[a] ≠ Φ cover + vaccinated post-outbreak only	2,880.75	2,852.36

To account for model uncertainty in the model selection process, two sets of models were fitted built upon general models with (i) an interactive effect of treatment and delayed myxomatosis (model 16 in Table 1), and (ii) an additive effect of treatment and delayed myxomatosis (model 15 in Table 1). For every contrast, models were fitted with same survival parameters for treatments under consideration, with different parameters in both pre-outbreak and post-outbreak periods, or with different parameters in only one period. Lower AIC_c score for every contrast and set indicate better fit

[a] Treatment with cover and vaccination combined parameterised as an interaction between those treatments

the effect of improving cover in open areas with mammalian predators, (2) optimum strategies for improving cover; and (3) alternative techniques to minimize the effects of diseases, including RHD, in the field.

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