Virus-vectored immunocontraception to control feral cats on islands: a mathematical model

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Summary

1. Feral cats *Felis catus* introduced onto oceanic islands pose a major ecological threat to endemic vertebrates, but their control is difficult. Immunocontraception has not been considered previously as a method for their control or eradication, and therefore we used a modelling approach to assess whether virus-vectored immunocontraception (VVIC) might be effective.

2. We compared the relative efficiency of cat control/eradication using immunocontraception and three different disseminating techniques, i.e. baits, genetically modified viral vectors, or both. We accounted for several forms of dynamic compensation likely to arise in a population with artificially reduced fertility.

3. We conclude that, under the assumptions of our model, immunocontraception can control or eradicate feral cats on oceanic islands. VVIC was found to be a more efficient dissemination technique than baits, but an integrated method involving viral-infected baits was the most likely to lead to eradication.

4. We advocate field trials of this VVIC technique, when available, under island conditions where any risks to non-target fauna would be minimal.

Key-words: baits, biological control, *Felis catus*, introduced predator, island conservation, mammal pest, VVIC.

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Introduction

Mammalian pests pose major ecological and/or economic problems (Lever 1994; Cowan & Tyndale-Biscoe 1997; Sinclair 1997). Although their control has been performed for centuries, traditional methods (culling, trapping, poisoning) are seldom cost-efficient (Sinclair 1997). A promising alternative to these traditional methods is the use of lethal species-specific pathogens (Anderson 1982; Dobson 1988). Theoretically, this form of biological control may be more cost-effective, but there are ethical and conservation implications of releasing lethal pathogens into ecosystems. As an alternative, immunocontraception is based on reducing birth rates (Alexander & Bialy 1994; Tyndale-Biscoe 1994). Immunocontraception is a process by which the immune system of an individual is made to attack its own reproductive cells and hence lead to sterility. This is achieved by infecting individuals using a gamete protein that triggers an immune response; the resulting antibodies bind to these proteins and block fertilization (Bradley, Hinds & Bird 1997). Infection occurs by injection (e.g. for large mammals; Kirkpatrick *et al.* 1997), bait (e.g. for small carnivores; Bradley, Hinds & Bird 1997) or living vectors (e.g. for small herbivores; Tyndale-Biscoe 1994; Cowan 1996; Rodger 1997; Smith, Walmsley & Polkinghorne 1997). Virus-vectored immunocontraception (VVIC), for example, utilizes a species-specific virus to disseminate this vaccine through a pest population by placing the gene encoding the reproductive protein into the genome of the virus (Tyndale-Biscoe 1994). This potentially powerful new technique has generated a significant impetus of research effort into feasibility.

There are many advantages of immunocontraception for biological control. First, it is considered humane by the public and wildlife welfare organizations (Loague 1993; Cowan 1996). Secondly, it is environmentally benign. Thirdly, it is likely to be cheaper than traditional control methods, because it is to an extent self-disseminating. Therefore, VVIC could be used to treat large inaccessible areas at a minimal cost (Chambers, Singleton & Hood 1997). Finally, despite concern over release of genetically modified pathogens (Beringer 2000), this method is

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likely to be one of the most specific control methods (at least family specific; McCallum 1996). VVIC will be unlikely to cross the species barrier (Tyndale-Biscoe 1994) because reproductive cells (i.e. sperm), pathogens and pathogen transmission modes are often specific. Disadvantages include irreversibility of the process (Nettle 1997), development of host resistance, need for the engineering of a genetically modified vector (Bradley, Hinds & Bird 1997), slow response (McCallum 1996), difficulty of controlling vectors once released, and the risks of irreversible genetic alterations of the population/species through selection (Table 1).

Immunocontraception has been considered for many mammals. These include species for which problems are localized (Turner, Liu & Kirkpatrick 1996; FayrerHosken *et al.* 1997; Kirkpatrick *et al.* 1997; McShea *et al.* 1997; Turner *et al.* 1997; Heilmann *et al.* 1998) and those in which major threats

to the ecosystem have resulted from invasion: mice *Mus musculus* Rutty (Shellam 1994; Chambers, Singleton & Hood 1997), possums *Trichosurus vulpecula* Kerr (Cowan 1996; Ji, Clout & Sarre 2000), grey squirrels *Sciurus carolinensis* Gmelin (Moore, Jenkins & Wong 1997), rabbits *Oryctolagus cunniculus* L. (Holland & Jackson 1994; Robinson *et al.* 1997) and foxes *Vulpes vulpes* L. (Bradley, Hinds & Bird 1997; Pech *et al.* 1997). It is surprising that domestic cats *Felis catus* L., which are threatening many species in Australia, New Zealand and on many smaller oceanic islands over the world (Lever 1994; Dickman 1996), have not yet been considered for immunocontraceptive control.

The aim of this study was to investigate theoretically the potential of immunocontraception for the control of feral cats introduced onto oceanic islands. First, we argue that the possible downsides of VVIC on oceanic islands are small and few (Table 1). Sec-

Table 1. General disadvantages of virus-vectored immunocontraception (VVIC) and relevance for the control of domestic cats *Felis catus* introduced onto islands

Disadvantages of VVIC	Relevance for domestic cats on islands
Lack of control of the vector	
Low public acceptance of release of genetically engineered organisms.	Public acceptance may be higher on remote and uninhabited islands if species protection is the issue.
Risk of vector transfer to pet cats that would be in contact with the target population.	Many islands free of human inhabitants and their pets.
Risk of virus spread into nearby, untargeted populations of the target species.	Control is desired for feral cat populations on remote islands.
Risk of vector transfer to closely related species (as in the case of the dog or the dingo for fox control).	Most islands free of such species - risk of virus spread out of remote uninhabited islands very low.
Risk of transfer of the vector to other species (lack of specificity, or recombinant virus losing species specificity with time).	Most islands free of species closely related to domestic cats - many islands free of endemic mammals.
Risk of accidental or criminal export of the vector from the target ecosystem.	Risk less likely for feral cats on islands.
No possibility of control once released: irretrievability, irreversibility.	Few cases where such reversibility would be desired.
Risks of population/species alteration	
Risk of genetic loss or alteration within the target population; risk of population/subspecies/ species extinction (as in possums in New Zealand and Australia).	Cat population conservation not desired on islands.
Risk of selection for the immunologically weakest individuals of the population.	Only likely to increase success of control.
Technical disadvantages	
Competition with existing strains in the population (as in myxomatosis for rabbits).	Not the case for cats introduced onto islands.
Slow response (for definitive result as well as for progress monitoring).	Too rapid eradication of cats may lead to release of introduced prey species such as rats and rabbits; these would need to be controlled simultaneously.
Low knowledge of potential candidate for vector.	Pathogens of domestic cat among the best known for animals.
Requires engineering of a genetically modified vector.	Domestic cat excellent model for laboratory studies; its pathology, physiology, genetics and endocrinology among the best known.

ondly, we present models of the effect of immuno-contraception on cat populations. We compare two different disseminating systems (baits and VVIC) and introduce a third possibility, where a self-disseminating virus could concurrently be disseminated through baits. We take into account two possible dynamic compensatory mechanisms in the host population, representing two extremes: increased reproduction and increased survival.

Methods

In the absence of immunocontraception, population growth is assumed to be logistic with the intrinsic growth rate (r) of the population equal to birth rate (b) – mortality (m). The carrying capacity is noted K. The logistic equation offers a good compromise between realism and simplicity (Berryman 1992) and it embodies compensatory density-dependence: the carrying capacity represents population regulation due to density-dependent effects. If the population density is decreased due to some kind of control, a compensatory reaction from all or part of the population occurs due to increased access to resources (Sinclair 1997). In this way, density-dependent effects can act both on the birth rate and the death rate, and the logistic equation makes no distinction between them. Because we were interested in a case where a fraction of the population is made unable to reproduce normally (by immunocontraception), we needed to separate these two contributions. For a population with a birth rate B and a death rate M, a logistic growth rate could arise, where $B - M = r(1 - N/K)$, in which N is the total population density. Birth rate B and death rate M can be expressed as:

$$\begin{cases} B = b - \dfrac{\varepsilon r N}{K} \\ M = m + \dfrac{(1 - \varepsilon) r N}{K}. \end{cases} \qquad \text{eqn 1}$$

The case where density-dependent effects act on birth alone corresponds to $\varepsilon = 1$, whereas when $\varepsilon = 0$ only mortality is affected. The cases $\varepsilon < 0$ and $\varepsilon > 1$ are biologically unrealistic, because they correspond, respectively, to cases where fecundity increases and mortality decreases as population density increases.

Rather than trying to estimate the value of ε, we chose instead to study only the extreme cases $\varepsilon = 0$ and $\varepsilon = 1$. The stable population density for a model with any given value of ϵ will, however, always lie between the values calculated for the extreme cases $\varepsilon = 0$ and $\varepsilon = 1$. We assumed that the effect of immunocontraception was to prevent reproduction of a subclass of the population, whereas fertile individuals reproduce normally. While this is a crude approximation, we believe that it captures the essen-

tial features of the biological reality. We divided the population into subpopulations of fertile (F) and sterile (S) individuals, which were described by equations based on the standard models for disease–host interactions (Anderson & May 1991):

$$\begin{cases} \dfrac{\mathrm{d}F}{\mathrm{d}t} = F(B - M) - i \\ \dfrac{\mathrm{d}S}{\mathrm{d}t} = -SM + i \end{cases} \qquad \text{eqn 2}$$

where B and M are the birth and mortality rates described in equation 1, i is the infection rate (rate at which individuals pass from the fertile to the sterile class) and $F + S = N$.

In our model we assumed that, in response to control, any demographic compensation by the population takes place through the density dependence of the birth and death rates. In other words, the only mechanism whereby birth and death rates alter is via the availability of resources, which depends upon the total population size (if sterile and fertile individuals need the same amount of resources). The combination of equations 1 and 2 gives different models of demographic compensation in response to control, according to the value of the compensation parameter ε. We have assumed that the mortality rate of sterile individuals is the same as for fertile ones. However, the equations would not change if the parameter ε only described the density dependence of the sterile individuals because we have assumed underlying logistic dynamics at the outset. The dynamics of the fertile individuals do not depend upon how the logistic term breaks down into birth and death rates. This yields the following two models, representing the two extremes of 'mortality compensation' alone and 'recruitment compensation' alone.

If $\varepsilon = 0$, then one has:

$$\begin{cases} \dfrac{\mathrm{d}F}{\mathrm{d}t} = rF\left(1 - \dfrac{(F + S)}{K}\right) - i \\ \dfrac{\mathrm{d}F}{\mathrm{d}t} = rS\left(1 - \dfrac{(F + S)}{K}\right) - bS + i \end{cases} \qquad \text{eqn 3}$$

From equation 1, we see that only the death rate is density dependent, so we refer to this as 'mortality compensation'.

If $\varepsilon = 1$, then:

$$\begin{cases} \dfrac{\mathrm{d}F}{\mathrm{d}t} = rF\left(1 - \dfrac{(F + S)}{K}\right) - i \\ \dfrac{\mathrm{d}S}{\mathrm{d}t} = -mS + i \end{cases} \qquad \text{eqn 4}$$

Here density dependence acts on birth rate alone, so we refer to this model as 'recruitment compensa-

tion'. In both cases, i is the rate at which fertile individuals become sterile.

Model applications

We applied the models under different systems of immunocontraception. The first involved immunocontraception by bait delivery, because it is the traditional method most employed (Artois *et al.* 1993) and progress has recently been made in the development of cat attractants (Clapperton *et al.* 1994; Moodie 1995). In this case, the infection rate is $i = \mu F$, where μ is the baiting rate (with time^{-1} as a dimension).

Our second approach involved a self-disseminating microparasite. We consider a virus that is transmitted horizontally by direct contact (i.e. not from mother to kitten), with no resistance for the disease, a sterility efficiency of 100%, no recovery, and only two pathological states, uninfected (fertile) and infected (sterile). There are two possible transmission terms in classical epidemiological models, the proportionate mixing (PM) model and mass action (MA) model (Busenberg & Cooke 1993), also called 'true mass action' and 'pseudo mass action', respectively (de Jong, Diekmann & Heesterbeek 1995). Because natural populations of domestic cats display a great deal of variation in their social and spatial structures (Liberg & Sandell 1988; Natoli & De Vito 1988), the incidence term can, depending on spatial or temporal effects, take either form (or more realistically an intermediate form). The PM model assumes that the rate at which individuals come into contact is a constant, ρ. Then, a proportion equal to $S/(F + S)$ of all the contacts by a single susceptible individual is with infected individuals. Thus the rate at which contacts between susceptible and infected individuals occur is equal to $\rho SF/(F + S)$. If the transmission efficiency is α, then the force of horizontal transmission is $\rho\alpha SF/(F + S)$, i.e. $\beta FS/(F + S)$ if $\rho\alpha = \beta$. For the MA model, the contact rate is proportional to the number of individuals ($F + S$), and the force of transmission is βFS. Because of the nature of the models, β has a different dimension for each model, and we use $\beta = \sigma$ for the PM model and $\beta = \gamma$ for the MA model. In our numerical results, we have assumed $\sigma = \gamma K$, which means that the force of infection is the same for the two models when the population equals the carrying capacity. The PM transmission is more appropriate to large heterogeneous stable populations of cats, whereas the MA transmission applies rather to smaller, homogeneous and less dense populations. Therefore, cat populations may be better defined by the MA model at the early stage of introductions, and by the PM model when they have been established. If the virus is self-disseminating, we consider three possible forms for i of equations 3 and 4:

$$\begin{cases} i_{MA} = \beta FS \\ i_{PM} = \beta \dfrac{FS}{(F + S)} \\ i_f = \dfrac{\beta FS}{1 - f + f(F + S)} \end{cases} \qquad \text{eqn 5}$$

where i_{MA} is the incidence term for the MA model, i_{PM} is the incidence term for the PM model, and i_f is the incidence term for an intermediate model, with $0 \le f \le 1$. If $f = 0$, then $i_f = i_{MA}$, and if $f = 1$, then $i_f = i_{PM}$. In this paper we only consider the extreme cases MA and PM, but we include the intermediate form as a possible avenue for future research.

Our last step was to use an integrated control strategy, assessing the effect of a virus-vectored immunocontraceptive whose spread is ensured both by its natural transmission mode and by bait dissemination. We assume that using a bait as the transmission medium is technically no less feasible for the transmission of a self-disseminating virus than for the transmission of a vaccine. With this method, cats infected by baits could then infect other cats. Thus, baits would be acting as an additional (and controllable) transmission rate. If the virus is spread both by baits and by contact, $i = \mu F + \gamma FS$ with an MA incidence term, and $i = \mu F + \sigma \dfrac{FS}{(F + S)}$ with a PM incidence term.
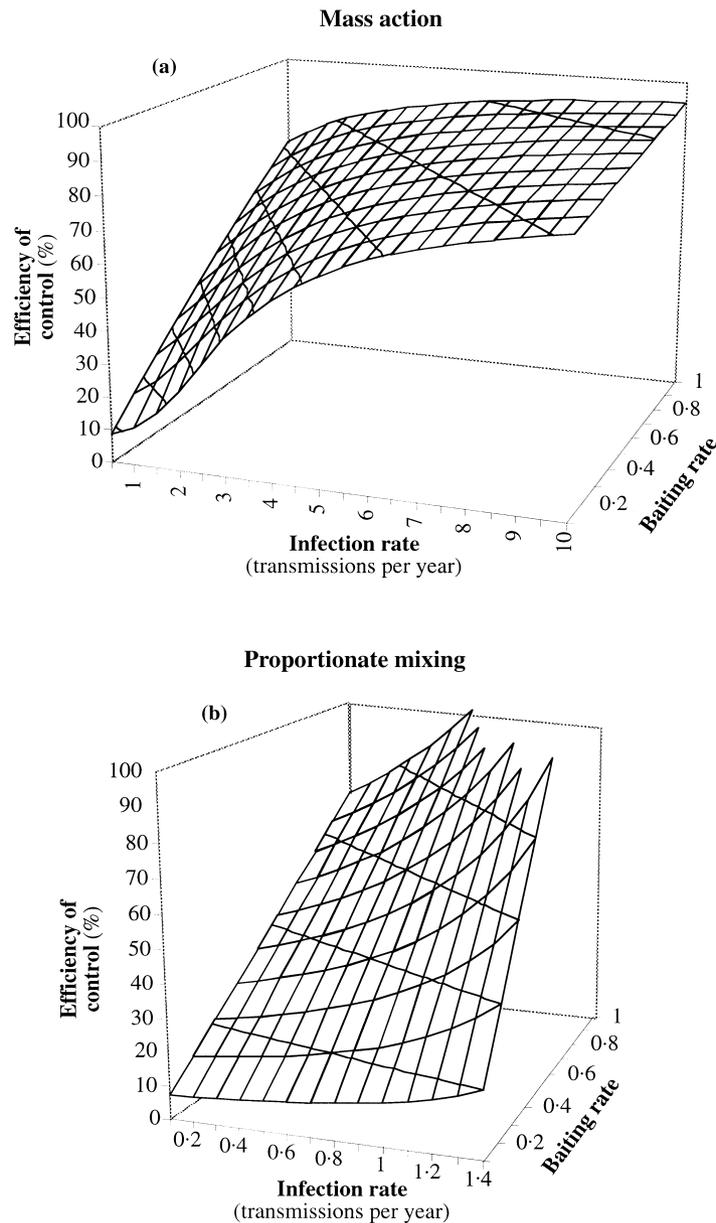
Results

The possible outcomes of the three different control methods in different types of population are given in Table 2. They show that control is possible when immunocontraception delivered through baits is involved. In addition, eradication is in theory possible with all methods, except dissemination with VVIC alone in an MA system. The value of the impact of the integrated control method is illustrated in Fig. 1 and Fig. 2. The efficiency of the integrated control method is given in Fig. 1 as a function of both the baiting rate (μ) and the virus transmission rate (γ or σ) for the case of mortality compensation. For the MA model, an increase in the transmission rate has more effect than an increase in the baiting effort when the latter is low. Control efficiency increases more markedly with baiting rate than with infection rate in the PM model. There is eradication of the cat population for high values of the baiting rate in the PM model, which is never achieved with the MA model, despite high control efficiency (*c.* 95%). Note that the PM model is not the most appropriate for low-density populations, which may modify our conclusions.

Baiting alone appeared to be the least efficient method, even with variation in baiting rate, and VVIC with a PM transmission force had more impact than with an MA transmission force (Fig. 2). The VVIC method with PM transmission is not

F. Courchamp &
S.J. Cornell

Table 2. Summary of the different control methods, with the two transmission models and associated infection rates, with the two types of compensation from the cat population, and results obtained by these different methods: condition for failure, eradication or control and, in that last case, quantitative efficiency of control

System description			Results			Impact of control (as a fraction of K)
Method	Infection rate (I)	Compensation (ε)	Failure	Eradication	Control	
Baits	μF	Any	Never	$\mu \geq r$	$\mu < r$	$\frac{\mu}{r}$
MA	γFS	Mortality	$K\gamma \leq b$	Never	$K\gamma > b$	$1 - \frac{b}{K\gamma}$
	"	Recruitment	$K\gamma \leq m$	Never	$K\gamma > m$	$\frac{\gamma K - m}{r + K\gamma}$
PM	$\sigma \frac{FS}{(F+S)}$	Mortality	$\sigma < b$	$\sigma \geq b$	Never	
	"	Recruitment	$m \geq \sigma$	$\sigma \geq b$	$m < \sigma < b$	$\frac{(\sigma - m)}{r}$
Baits + MA	$\mu F + \gamma FS$	Mortality	Never	$\mu \geq r$	$\mu < r$	$\frac{1}{2}\left(1 - \frac{b}{K\gamma} + \sqrt{\left(1 - \frac{b}{K\gamma}\right)^2 + \frac{4\mu b}{rK\gamma}}\right)$
	"	Recruitment	Never	$\mu \geq r$	$\mu < r$	$\frac{1}{2(r+\gamma K)}\left(\mu + \gamma K - m + \sqrt{(\mu + \gamma K - m)^2 + 4\mu m\left(1 + \frac{\gamma K}{r}\right)}\right)$
Baits + PM	$\mu F + \sigma \frac{FS}{(F+S)}$	Mortality	Never	$\sigma \geq b\left(1 - \frac{\mu}{r}\right)$	$\sigma < b\left(1 - \frac{\mu}{r}\right)$	$\frac{\mu b}{r(b - \sigma)}$
	"	Recruitment	Never	$\sigma \geq b\left(1 - \frac{\mu}{r}\right)$	$\sigma < b\left(1 - \frac{\mu}{r}\right)$	$\frac{1}{2r}(\mu + \sigma - m + \sqrt{(\mu + \sigma - m)^2 + 4\mu m})$

Mass action

**(a)**

Proportionate mixing

(b)

Fig. 1. The effect of baiting and transmission rates on the efficiency of the integrated control (i.e. bait and VVIC concurrently) for (a) the mass action model (MA) and (b) the proportionate mixing model (PM). Both are shown for a population with mortality compensation of cats in response to control. The value of the parameters used for this graph are $b = 2.1$, $m = 0.6$ and $K = 100$ so that results are expressed in percentages of the carrying capacity.

shown for $\epsilon = 0$ because this method does not allow control of the cat population (Table 2). The integrated method was the most efficient disseminating system for control by immunocontraception when VVIC is with PM transmission. Recruitment compensation by the cat population ($\epsilon = 1$) in each case allowed a better control than mortality compensation ($\epsilon = 0$). Interestingly, the impact of control using an integrated approach was sometimes greater than the sum of both methods separately (Fig. 3). This was always the case for the PM model, but occurred also for the MA model when the transmission rate was low. This is due to the low transmis-

sion rate of an MA-type infection term at low population densities.

Elasticity of the model parameters reflects the sensitivity of the outcomes of the model to the value of these parameters. The lower the elasticity of the main parameters, the more robust the model. Elasticity of the level of control was very low when the level of control was high. When only baits were used, the elasticity with respect to baiting rate was equal to one. In the absence of baiting, the elasticity of the impact of control diverged when the transmission parameter was close to the borderline between failure and control, and was low far from this criti-
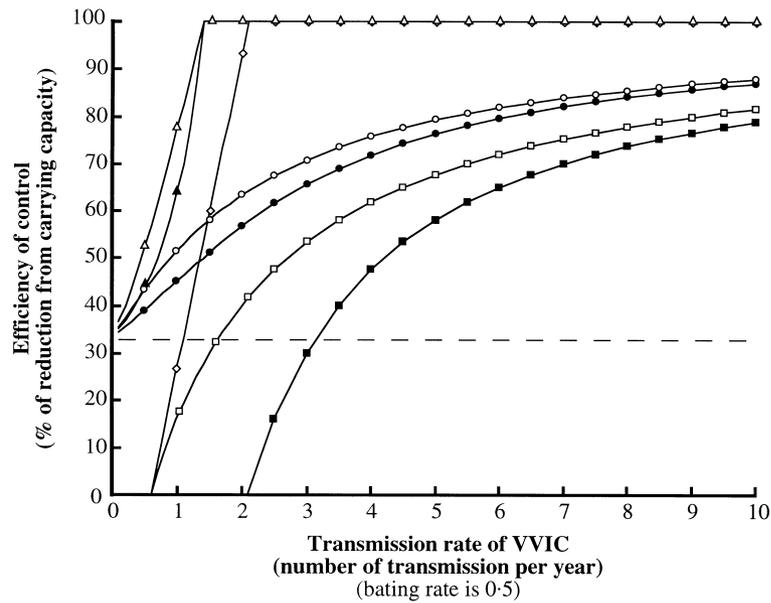
Fig. 2. Impact of the control, as a function of the VVIC infection rate, for five different disseminating systems: baits (dashed line); VVIC with a mass action transmission (MA; black square = mortality compensation, white square = recruitment compensation); VVIC with a proportionate mixing transmission (PM; diamond = recruitment compensation; PM with mortality compensation does not allow control, see Table 2); or by both baits and VVIC, with MA (black circles = mortality compensation, white circles = recruitment compensation) and PM (black triangles = mortality compensation, white triangles = recruitment compensation) transmissions, respectively. The value of the parameters used for this graph are $b = 2.1$, $m = 0.6$, baiting rate $\mu = 0.5$, and results are expressed in percentages of the carrying capacity. An impact of 100% represents eradication.
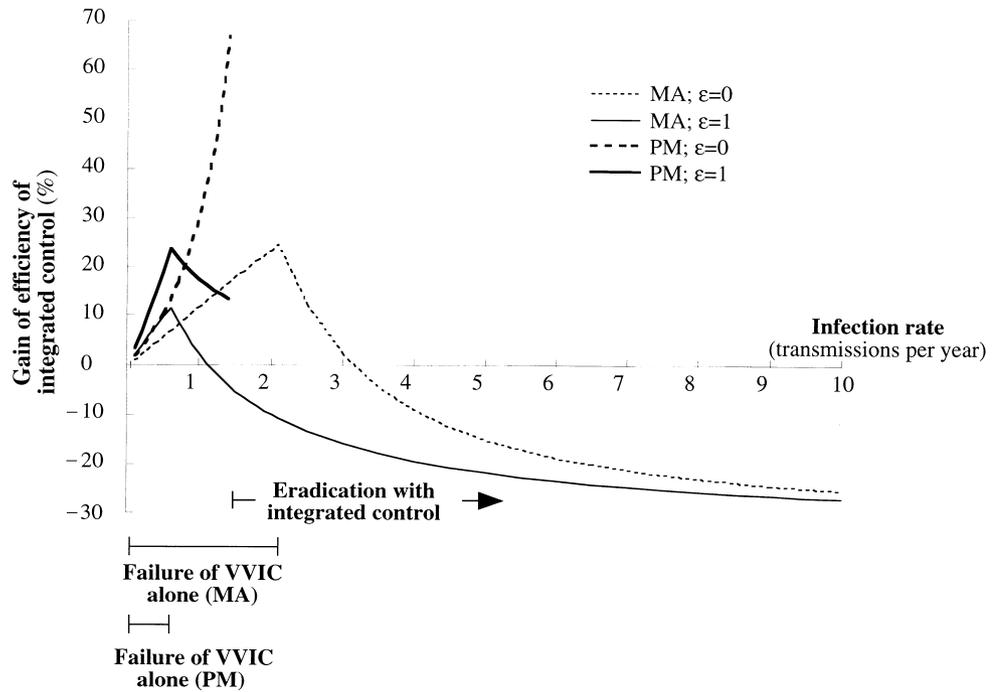
Fig. 3. Relative efficiency of integrated control against alternative methods. The lines are the difference between the integrated method and the sum of the two alternative methods (baits + VVIC control alone). The lines represent the presence (positive values) or absence (negative values) of a synergetic effect: there is a synergetic effect when the efficiency of the integrated control is higher than the sum of alternative methods. Termination of the lines indicates eradication by the integrated control. Drops in the lines are explained by a failure of one method alone (low transmission rates).
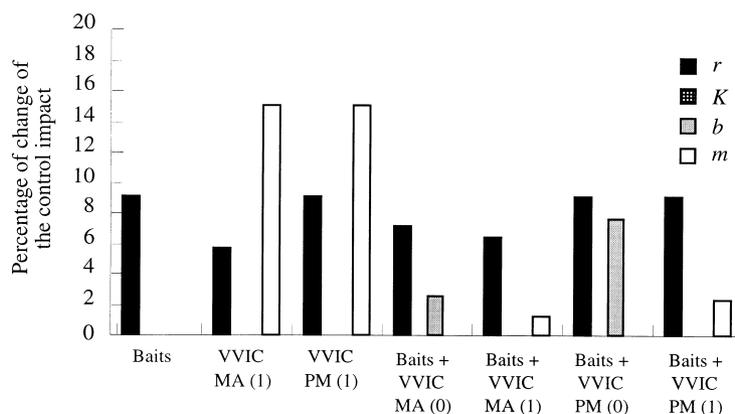
Fig. 4. Sensitivity of the parameters for the different models, represented by the quantitative effect of a 10% increase of each parameter on the value of the impact of the control (as a percentage). The effect of changes in K is always zero here, because the impact of the control is given as a percentage of the carrying capacity. The value of ε is given in parentheses.

cal value. In the case of integrated control, when the level of control was small, elasticity of the impact of control could be very sensitive to both the baiting effort and the transmission parameter. This is due to the high gain in efficiency shown in Fig. 3. For all models, the elasticity of the level of control was relatively low (Fig. 4), indicating the robustness of the model; small changes in the values of the parameters did not result in large changes in outcome.

Discussion

These simple mathematical models show the dynamics of populations in the presence of an immunocontraception vaccine. Although the models are general, we concentrate on the example of feral cats, which are a major threat to small vertebrate prey in many ecosystems (Moors & Atkinson 1984; Lever 1994). We considered scenarios where dynamic compensation in response to control either acted on mortality or recruitment. These simulations suggest that even when this biologically realistic hypothesis is taken into account, a population can be controlled and even eradicated by immunocontraception. The two types of compensation we study in this paper span a wide range of possible biological situations for pest mammals introduced onto islands. It is significant that both provide similar outcomes, which suggests there are robust. We would expect a real system to interpolate between the two cases. We assessed the potential of three disseminating methods for immunocontraception of cats introduced onto islands: by baits, by virus and by virus-infected baits.

Our first result is that immunocontraception disseminated by virus (VVIC) would almost always be more efficient than by baits. In reality there would

be other practical advantages to VVIC over bait delivery. For example, a spread of the vaccine by a biological vector on an island would in most cases be less labour intensive than continuous baiting (McCallum 1996; Cowan & Tyndale-Biscoe 1997). Moreover, a disseminating virus potentially induces a stronger immune response and a greater immunological memory than baits (Chambers, Singleton & Hood 1997). Furthermore, the species-specificity of viruses might be stronger than that of baits (Chambers, Singleton & Hood 1997; Hood, Chesson & Pech 2000).

Our second result is that VVIC would be more efficient if the transmission was characterized by proportionate mixing, i.e. in large, dense and heterogeneous populations, characteristic of the final stages of invasions. This result implies that VVIC would be an adequate method of cat control even on islands where cat populations have been established for decades. It is also worth noting that, as in Barlow's model (Barlow 1994), control with less than 100% efficiency would still allow a partial recovery of prey populations (Courchamp & Sugihara 1999).

The third result concerning disseminating systems is that a strategy involving both baiting and viruses running concurrently would be more efficient in most cases. Following these results, we propose an integrated biological control method involving a complementary and controllable virus transmission rate through additional baits containing the virus. This method would permit a unique opportunity to control the epidemiology of a virus. It could, for example, be used to link patches of infection, or to increase the infection rate if it became too low. This integrated method is, in theory, the most efficient even when a constant bait delivery rate is used.

Furthermore, using both methods concurrently may result in a better form of pest control than the sum of both methods individually (Fig. 3). Indeed, by contrast with the simple baiting method, cats infected by baits in this system will also transmit the virus, resulting in a higher sterility rate, and would maintain a high infection rate even when the population density gets very low.

The control of introduced cats is part of restoration policies of many disturbed oceanic islands, but despite a few successful examples (Rauzon 1985; Veitch 1985; van Rensburg, Skinner & van Aarde 1987; Domm & Messersmith 1990) the poor efficiency of current methods still prevents systematic eradication (Sinclair 1997), especially on large islands. For example, on Marion Island, cats were eradicated using feline panleucopenia virus (van Rensburg, Skinner & van Aarde 1987). However, this virus was only partly successful because many years of hunting have been required to complete the eradication of the cats. We propose here that VVIC, alone or together with immunocontraception disseminated by baits, has potential for the control or eradication of feral cats on islands. Most reservations about VVIC release in ecosystems do not apply to the situation in isolated habitats. For example, the lack of control of the virus (Chambers, Singleton & Hood 1997; Nettle 1997; Williams 1997) would not present the same problem on a remote island free of human inhabitants (Table 1). Also, these ecosystems would not be affected by the adverse impact of continuous immigration (Barlow 1994) or emigration (Tyndale-Biscoe 1994). The slower response of VVIC compared with classical methods (e.g. shooting) may also be an advantage in some cases. Rats are present on many islands, and it has been shown that the rapid eradication of cats could trigger an explosive increase in the rat population, called mesopredator release, which would be more detrimental for endemic small vertebrates (Courchamp, Langlais & Sugihara 1999). Unfortunately, island accessibility is an important factor in cases where a combination of control techniques is considered for introduced mammals. Obviously, in each case the different control options must be carefully considered. Competition with existing strains in the population (as in rabbit myxoma virus; Robinson *et al.* 1997; Williams 1997; Kerr *et al.* 1998) is less likely to occur because of the founder effect characterizing many introduced cat populations. For instance, five domestic cats that were introduced on Marion Island in 1949 resulted in a population of more than 2000 cats 25 years later (van Aarde 1980). This founder effect limits the biodiversity of cat pathogens present in these insular populations (Dobson 1988), making these naive populations more sensitive to a larger array of pathogens than continental populations. Furthermore, domestic cats are social felids (Liberg & San-dell 1988; Natoli & De Vito 1988) and hence physical (and potentially infectious) contacts between individuals are common in most populations even when density becomes low. As a consequence, the array of pathogen candidates for VVIC is broader in cats, including many diseases transmitted by direct contact (Moodie 1995). There are several pathogens whose biological and epidemiological characteristics could make them potential candidates for VVIC. For example, it would probably be interesting to test feline retroviruses more thoroughly (Courchamp & Sugihara 1999). Our current results only apply to vaccination via viral infection with neither resistance nor recovery, which is the case for some feline retroviruses (e.g. feline immunodeficiency virus). We now advocate a trial, using the methods outlined above when available, in an environment where potential biohazard would be naturally limited in space (an island). This could be a low risk opportunity to provide empirical support for the efficiency of VVIC at the scale of an ecosystem.

Acknowledgements

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