

## MODELING THE BIOLOGICAL CONTROL OF AN ALIEN PREDATOR TO PROTECT ISLAND SPECIES FROM EXTINCTION

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**Abstract.** Introduced feral cat (*Felis catus*) populations are an important threat to many island vertebrate populations and to bird species in particular. Elimination of feral cat populations is desirable in most of these ecosystems. Release of a parasite species in these mostly immune-naïve populations is thought to be an efficient eradication measure. Such an approach is theoretically investigated here, using a mathematical model that describes the effects of introducing a virus into the cat population on population dynamics of both the cat and its prey. We studied the effects of two types of introduced feline viruses: Feline Immunodeficiency Virus and Feline Leukemia Virus, both of which are good candidates for eradicating a cat population. Results show that eradication is possible with Feline Leukemia Virus, if natural immunity is sufficiently low. Feline Immunodeficiency Virus cannot fully eradicate cat populations, but can be an effective agent for long-term control of cat populations on islands where total cat eradication is not possible (e.g., there is a high likelihood of continued introduction of cats) or not desirable (e.g., when rats are present). Culling, which by itself would require a very prolonged and logistically demanding effort to eliminate cat populations, may be more efficient when applied simultaneously with virus introduction.

**Key words:** alien species; biological control; bird protection; conservation biology; Feline Immunodeficiency Virus; FeLV; Feline Leukemia Virus; FIV; introduced feral cat; oceanic island; population model; virus introduction.

### INTRODUCTION

Since its domestication, the domestic cat (*Felis catus* L.) has been accidentally or deliberately introduced by humans to most parts of the world, including at least 65 major island groups (Atkinson 1989) and many remote oceanic islands, both inhabited and uninhabited (Fitzgerald 1988). The domestic cat is very adaptable (Konecny 1983, Apps 1986, van Aarde 1986). On the Kerguelen Islands, cats live in the burrows of rabbits (*Oryctolagus cuniculus*) and burrowing petrels (Procellariidae), and have developed a large layer of fat and hair that protects them against the cold (Derenne 1976). In the San Cristobal Islands, cats are found in the most arid and inhospitable regions and use lava folds as shelters (Tabor 1983). On these islands, moreover, cats have neither predators nor competitors and are infected with few parasites, which may lead to increased life expectancies (Derenne 1976, Konecny 1983) and higher population growth rates (Derenne 1976, van Aarde 1978, Pascal 1980). In fact, the only limiting factor may be food supply, but prey are usually numerous on these islands and often possess limited

antipredation behavior (e.g., Stone et al. 1994, Blázquez et al. 1997).

Thus, on many of these islands, cats were established and became feral, usually to the detriment of indigenous species. The cat is an opportunistic predator, and its diet on oceanic islands may include large proportions of reptiles (e.g., Konecny 1987, Bamford 1995), birds (e.g., Fitzgerald 1988, Rodriguez-Estrella et al. 1996), or mammals (Derenne and Mougin 1976, Jones 1977), according to the prey's relative abundance in different seasons and regions. In these closed ecosystems, introduced cats are known to be the direct cause of severe reduction or extinction of numerous populations of local vertebrate species (Iverson 1978, Taylor 1979, Moors and Atkinson 1984, King 1985). For example, five domestic cats introduced on Marion Island in 1949 resulted in a population 25 yr later of >2000 cats that killed nearly half a million burrowing petrels per year (van Aarde 1980). They drove at least one species to local extinction and threatened several others (van Rensburg and Bester 1988). On Kerguelen Islands, five founders in the same period led, in the early 1990s, to several tens of thousands of cats, which kill more than 3 million seabirds each year (Chapuis 1995).

Even now, the recovery of most of these island bird populations should be possible with the elimination of the introduced cat populations (van Rensburg and Bes-

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ter 1988, Cooper et al. 1995). Control of feral cat populations has been attempted on a number of oceanic islands, but eradication has rarely been achieved (Bloomer and Bester 1992). Culling and trapping programs can be very expensive, time-consuming, and labor-intensive, especially in removing the last individuals (Bester and Skinner 1991).

Introduction of pathogens is an alternative technique for the eradication of cats from islands. However, the introduction of pathogens to ecosystems requires careful feasibility studies to insure that the pathogen will not infect native species and will result in eradication or significant control of target species. The potential harm of micro- and macroparasites often depends on the immune state of the host individual and, thus, at least partially depends on the genetic diversity of the host population. Island feral cat populations are generally thought to originate from a small founding group (Dreux 1974, Cooper 1977). These few individuals, sometimes five or less (Derenne 1976, van Aarde 1979), certainly brought with them only a small subsample of the total feline parasite fauna. The total number of parasite species in an island population might be further reduced, relative to a mainland population, by selection against more virulent species, which require large host populations to maintain themselves (Dobson 1988). Thus, when compared to mainland populations, island cat populations should harbor fewer and less virulent parasite species. Consequently, island cat populations should be susceptible to most feline pathogens.

Introduction of a new feline parasite could thus be an efficient way to eradicate introduced feral cats and to protect the endangered indigenous species they prey upon. Feline Panleucopenia Virus (FPV) was used to eliminate introduced cats from Marion Island (van Rensburg et al. 1987). However, this virus was only partly successful, because many years of hunting have been required to complete the eradication of cats. In this study, we analyze the potential of feline retroviruses as control agents, because they present a number of advantages over FPV. We will focus on two major feline retroviruses: Feline Immunodeficiency Virus (FIV) and Feline Leukemia Virus (FeLV).

## METHODS

### *The viruses*

FIV is a retrovirus inducing Acquired Immunodeficiency Syndrome (AIDS) in cats and is thought to be transmitted by bites during fights. FIV infection leads to lifelong virus carriers. The clinical staging of FIV infection is very similar to that of the Human Immunodeficiency Virus infection (HIV), with a short acute stage, a long asymptomatic period (lasting up to several years, and in which the cat is healthy and may reproduce), a Persistent Generalized Lymphadenopathy, an AIDS-Related Complex associated with chronic infec-

tions, and finally AIDS. As in humans infected by the homologous HIV, feline AIDS is characterized by a loss of immunological defenses and subsequent opportunistic infections. The cat is infectious during all these five stages of infection, which have been estimated to last an average of 5 yr (Pedersen and Barlough 1991). We will use this estimate in the model (the mortality rate due to FIV infection will thus be 0.2). There is no recovery or immunity to FIV, either natural or artificial, although a few cats have failed to show any symptoms after 8 yr under laboratory conditions (M. Pistello, *personal communication*). Antibodies to FIV have been found in at least 16 species of felids so far (Carpenter and O'Brien 1995) and several strains of FIV have been isolated in different species of felids. A more complete description of various aspects of FIV biology is given by Bendinelli et al. (1995).

FeLV is a retrovirus that leads to immunosuppression in infected cats, with clinical features comparable to those of FIV infection. This virus is transmitted via "amicable" contact (by saliva, through licking, maternal grooming, and food sharing) and also through mating or biting. Infection is followed by a temporary viremic stage, which is generally asymptomatic, and then by two possible outcomes. A certain proportion of the infected individuals will develop natural immunity and recover from infection. These naturally immunized cats are not infectious, have a normal life expectancy, and are believed to be immunized for life. Individuals that do not become naturally immunized become persistently viremic and die within an average of 2 yr from various proliferative or immunosuppressive disorders. This mortality rate (0.5) will be used in this model (Fromont et al. 1997). As for FIV, FeLV infects other felids, but never nonfelid species (Jessup et al. 1993). A more complete description of this virus and its properties is given by Hardy (1993).

There are three main reasons why these two retroviruses could be more efficient than FPV as control agents. First, unlike FPV, both FIV and FeLV persist for a long time before killing their host, allowing multiple transmissions of the viral strain during the life of the host. Moreover, the impact of these retroviruses should be higher on islands, where cats may live longer, on average (Derenne 1976, Konecny 1983), allowing more transmissions than in natural mainland conditions where life expectancy is short compared to infection length (Courchamp et al. 1995a). To control or eradicate a host population, a low-to-moderate virulence is said to be better than a highly virulent pathogen (Dobson 1988). A highly virulent virus (i.e., one that kills most infected hosts very rapidly) introduced onto an island could become self-limiting because of the short course of the induced disease and the accordingly restricted period of virus transmission (Howell 1984), as observed on Marion Island. Moreover, the highly virulent pathogens require a minimum host density to maintain themselves, which might not be maintained

as the virus reduces the population size (Anderson and May 1985). In general, the higher the virulence of a virus, the larger the population size required for the disease to persist (Price et al. 1988).

Second, in many ecosystems, juvenile mortality is the key factor regulating population density. Pathogens that kill mainly juveniles, like FPV, have a limited impact in these populations, because the virus kills individuals that would have died anyway and spares those that would have survived. A virus such as FIV, which predominantly infects and kills individuals with high survival probabilities (dominant individuals; Courchamp et al., 1999a), would have higher predicted efficiency.

Finally, environmentally transmitted infections, such as FPV infection, are less successful at low density (e.g., after the first outbreak; Howell 1984). This is not the case with FIV and FeLV, which are both transmitted behaviorally and, thus, are efficient as long as cats are present. FIV is transmitted by bites during fights, and it is likely that island populations have social structures similar to those in comparable low-density rural habitats. In these rural populations, numerous fights occur between males, but also between males and females (Natoli and De Vito 1991). Indeed, several long-term studies have highlighted frequent cat fights in some oceanic island populations (Pascal 1980, Brothers et al. 1985). Similarly, a low density does not preclude amicable social contacts, which are the basis of FeLV transmission. For these three reasons at least, FIV and FeLV are excellent candidates for eradication programs for introduced cats, which motivates our efforts to test their potential utility through mathematical modeling.

Our approach is to model the coupled dynamics of prey (e.g., a bird species) and predator (cat) populations, and to analyze the effect of introducing different kinds of viruses into the predator population. We construct a very simple prey–predator model, which we combine with a host–parasite model to study the indirect effect of the parasite introduction on the prey population size. For all parameters, we use realistic values obtained from the literature or from previous host–parasite models (Courchamp et al. 1995b, Fromont et al. 1997). In this paper, we use the epidemiological definition of the term “endemic”; constant prevalence rate in time and space (by opposition to epidemic).

#### The models

*Before virus introduction.*—We first define a set of coupled differential equations that describe the linked dynamics of the prey (e.g., bird) and predator (e.g., cat) populations, with the explicit aim of keeping the model as simple as possible. For both the prey and predator species, we assume that the population dynamics are well characterized by the classical logistic equation (Verhulst 1838). Introduced domestic cats may indeed exhibit logistic population dynamics in such a predator-,

competitor-, and (nearly) parasite-free environment. In this case, cat population dynamics can be characterized by the intrinsic growth rate and the carrying capacity of the environment. The prey population may be influenced by a higher number of inter- and intraspecific interactions, such as competition, predation, and parasitism. Prior to introduction of the predator, the prey population is assumed, however, to have reached a stable equilibrium state, given by the carrying capacity of the habitat. The population dynamics of native oceanic island bird species prior to cat introduction are still poorly documented; we believe that assuming an equilibrium population level is the most reasonable approach, in the absence of better information.

We link the predator and prey populations by supposing that the carrying capacity of the predator is a function of prey population size. In addition to being a biologically realistic hypothesis, this allows us to minimize the number of parameters used in the model. Specifically, we assume that the predator capacity is given by the prey population size divided by the yearly individual predation rate, which corresponds to the maximum number of predators that can feed upon the prey population at time  $t$ . In this model, we thus assume that the carrying capacity of the predator population depends only on food availability, without considering other aspects (such as density). Indeed, cat populations have been shown to reach very high densities when food is not limited (Liberg and Sandell 1988). This term is similar to the pioneer predator–prey ratio model of Leslie (1948). We chose to use predator–prey ratio instead of prey density in the model, because the induced slanting predator isocline solves the paradoxes of enrichment and biological control (see Arditi and Berryman 1991). Moreover, this kind of model has both very plausible mechanisms and biological justification (e.g., Berryman et al. 1995) and is said to describe and explain real data more satisfactorily (e.g., Akçakaya et al. 1995). We did not include realistic predator functional response, because it would unnecessarily complicate these models.

We use the following notation:  $N$  is the number of prey,  $r_n$  is the intrinsic growth rate of the prey population,  $K$  is the equilibrium prey population carrying capacity,  $\mu$  is the annual individual intake of prey per individual predator,  $C$  is the number of predators, and  $r_c$  is the intrinsic growth rate of the predator population.

We thus have the following simple set of equations to describe the system:

$$\frac{dN}{dt} = r_n N \left(1 - \frac{N}{K}\right) - \mu C \quad (1)$$

$$\frac{dC}{dt} = r_c C \left(1 - \frac{C}{(N/\mu)}\right). \quad (2)$$

*Introduction of feline immunodeficiency virus.*—We first analyze the effect of the introduction of FIV, a virus with dynamics that can be modeled in a relatively

straightforward fashion. Model analysis is similar to that of a previously published FIV dynamics model (Courchamp et al. 1995b). Development and analyses of this model, based on Anderson and May's work (e.g., 1991), are available elsewhere and will not be described here in detail. The main assumptions, in addition to those used to describe the population dynamics in the absence of disease, are as follows.

There is no immunity or recovery. Infected individuals developing immunodeficiency are assumed to die too rapidly and to be too weak to interact with other individuals, and are not considered here. Thus, there is only one stage of the disease (asymptomatic seropositivity), which lasts a constant time (i.e., infected individuals die at a constant rate from disease) during which individuals display normal behavioral and reproductive characteristics. There are, therefore, only two classes of individuals: susceptible and infected. The virus is not transmitted vertically (from mother to kitten), and infected individuals are able to reproduce (thus giving birth to susceptible individuals). The force of infection term is of the Proportionate Mixing type (the number of new infections is proportional to the proportion of infected individuals in the population). We call  $\sigma$  the virus transmission coefficient, and  $\alpha$  the disease-induced mortality rate. The predator birth and death rates are termed  $b$  and  $m$ , respectively. Following conventional notation,  $X$  gives the number of susceptible individuals and  $Y$  the number of infected individuals, with the total population  $C$  being  $C = X + Y$ .

The system may now be described by Eqs. 1–2, where

$$\frac{dC}{dt} = \begin{cases} \frac{dX}{dt} = b(X + Y) - mX - r_c X \frac{(X + Y)}{(N/\mu)} \\ - \frac{\sigma XY}{(X + Y)} \\ \frac{dY}{dt} = \frac{\sigma XY}{(X + Y)} - mY - r_c Y \frac{(X + Y)}{(N/\mu)} - \alpha Y. \end{cases} \quad (3)$$

Analysis of the system (Eqs. 3–4) indicates that three equilibrium points may arise (for more details, see the mathematical analysis of a similar model in Courchamp et al. 1995b). These points correspond to the vanishing of the disease in the predator population, the vanishing of the predator population, or a stable infection prevalence within the predator population. Each of these three equilibrium points is controlled by two major epidemiological parameters:  $R_0$ , the reproductive rate of the disease and  $R_1$ , the “net reproductive coefficient of the infected population when the disease is endemic” (e.g., Anderson and May 1980; see also Courchamp et al. 1995b), with

$$R_0 = \frac{\sigma}{\alpha + b} \quad (5)$$

$$R_1 = \frac{b}{m + \alpha y^*} \quad (6)$$

where

$$y^* = 1 - \frac{b}{\sigma - \alpha}. \quad (7)$$

$R_0$  can be defined as the number of secondary cases produced by an infectious individual in the population. When  $R_0 < 1$ , the epidemic disappears and the predator population will settle at its disease-free equilibrium size (Anderson et al. 1981). When  $R_0 > 1$ , the prevalence of infected individuals stabilizes to  $y^*$ . In this case, the predator population becomes extinct (and the prey population thus reaches the predator-free carrying capacity of the habitat) if  $R_1 < 1$ , or the disease remains endemic in the system if  $R_1 > 1$  (Table 1).

*Introduction of feline leukemia virus.*—Our next step is to analyze, in the same manner, the effects of introducing a virus with a different pathological pattern. In this model, a certain proportion of individuals is naturally immunized against the virus' pathogenic effects. These individuals do not die from the virus and are not able to transmit the virus. For this model, we will use as an example the FeLV. FeLV dynamics that have been studied previously through a model similar to the one presented in the previous section (Fromont et al. 1997); therefore, we use the detailed mathematical analyses given therein.

The main assumptions, in addition to those used to describe population dynamics in the absence of disease, are as follows.

The transmission rate is of the Proportionate Mixing type and the transmission coefficient is  $\sigma$ . A proportion  $\pi$  of infected individuals will develop symptoms and will die with a rate of mortality  $\alpha$ , with no possibility of recovery. A proportion  $1 - \pi$  of infected individuals will develop natural immunity to the disease and will not suffer from FeLV pathogenic effects, nor will these individuals be infectious. There is only one pathological stage. The result is three pathological classes: susceptible, infected, and immune. The virus is not transmitted vertically and immune individuals are able to reproduce (giving birth to susceptible individuals), whereas infectious individuals are considered too sick to reproduce (Hoover et al. 1983). The notation is the same as in the FIV model, with  $Z$  being the number of immune individuals: the total population is given by  $C = X + Y + Z$ .

The system may now be described by the following set of equations:

$$\frac{dN}{dt} = r_n N \left( 1 - \frac{N}{K} \right) - \mu C \quad (1)$$

$$\frac{dC}{dt} = r_c C \left( 1 - \frac{C}{(N/\mu)} \right) - (b + \alpha) Y \quad (8)$$

with

$$\begin{cases} \frac{dX}{dt} = b(X + Z) - mX - r_c X \frac{(X + Y + Z)}{(N/\mu)} \\ \quad - \frac{\sigma XY}{(X + Y + Z)} & (9) \\ \frac{dY}{dt} = \frac{\sigma XY}{(X + Y + Z)} \pi - mY \\ \quad - r_c Y \frac{(X + Y + Z)}{(N/\mu)} - \alpha Y & (10) \\ \frac{dZ}{dt} = \frac{\sigma XY}{(X + Y + Z)} (1 - \pi) - mZ \\ \quad - r_c Z \frac{(X + Y + Z)}{(N/\mu)}. & (11) \end{cases}$$

Following the same algebraic developments given previously, the analysis of the system (Eqs. 9–11) leads to the same conclusion as for the FIV model (a detailed mathematical analysis of a similar model is given in Fromont et al. 1997). Three equilibrium points may arise: extinction of the disease in the predator population, extinction of the predator population, or a stable equilibrium of the disease prevalence within the predator population, where

$$R_0 = \frac{\sigma\pi}{\alpha + b} \quad (12)$$

$$R_1 = \frac{b}{m + (b + \alpha)y^*} \quad (13)$$

and

$$y^* = \frac{b(\sigma\pi - b - \alpha)}{(b + \alpha)(\sigma - b - \alpha)}. \quad (14)$$

As for the previous model, when  $R_0 < 1$ , the disease dies out and the predator population reaches the disease-free carrying capacity of the habitat. Provided that  $R_0 > 1$ , when  $R_1 < 1$ , the predator population becomes extinct; when  $R_1 > 1$ , the disease stabilizes at a unique endemic equilibrium (Table 1).

*Combined effects of culling and virus introduction.*— Virus introductions may be used in conjunction with conventional methods to ensure eradication success or to lower its cost for an increased efficiency. It is interesting to study simultaneous complementary actions such as culling (or trapping, poisoning, or several such actions simultaneously) of the population while a pathogen is causing it to decrease. The effect of culling in a disease-free population can be analyzed easily. The culling program will cause a decrease in the population if and only if the culling (or trapping or poisoning) rate  $\delta$  is such that  $\delta > r_c[1 - C/(N/\mu)]$ . Here, the duration of the culling program is proportional to the difference between these two terms.

In the case of simultaneous culling and virus introduction, let us assume that there is no difference be-

tween the rate of culling susceptible (and immune) and infected individuals. The population will then be eradicated if and only if the culling effort is maintained such as:

$$\delta > r_c \left( 1 - \frac{C}{N/\mu} \right) - \alpha y^* \quad (15)$$

for FIV-type viruses, and

$$\delta > r_c \left( 1 - \frac{C}{N/\mu} \right) - (\alpha + b)y^* \quad (16)$$

for FeLV-type viruses, where  $y^*$  is the prevalence rate of the virus within the population at equilibrium, as given in Eqs. 7 and 14 for FIV and FeLV, respectively.

## RESULTS

### *Before virus introduction*

Three possible outcomes can arise from the system described in Eqs. 1 and 2: both populations go extinct (at equilibrium, the final number of prey  $N^* = 0$  and the final number of predators  $C^* = 0$ ); only the predator population goes extinct ( $N^* = K, C^* = 0$ ); or both populations reach a steady equilibrium state ( $N^* = K[(r_n - 1)/r_n]$ ;  $C^* = K[(r_n - 1)/(\mu \times r_n)]$ ). Simulations shown in Fig. 1 demonstrate the existence of the third equilibrium point, for different parameter values (A), or for different initial conditions (B).

The impact of predator introduction on prey population size is defined simply by the difference between the prey population size prior to predator introduction ( $K$ ) and the population size at equilibrium once the predator is established ( $N^*$ ). According to this model, the impact of the predator on the prey population is solely a function of predation rate, and not of initial predator population size or its intrinsic growth rate (Fig. 1A, B). The predator population size at equilibrium, however, will depend on both the intrinsic prey growth rate ( $r_n$ ) and the predation rate ( $\mu$ ). We can, therefore, define the maximum impact of the introduction of a predator species on a single prey species population by considering only the basic prey population parameters (intrinsic growth rate,  $r_n$  and carrying capacity of the habitat,  $K$ ). This impact will be maximal at equilibrium and will have the value  $K[1 - (r_n - 1)/r_n]$ .

We can now analyze the impact of a virus introduced into the predator population on the predator population dynamics and, hence, on the prey population. We can also deduce from this analysis the threshold value of some key parameters leading to the extinction of the predator population.

### *Introduction of FIV*

In this paper, we will use only cat population parameter values estimated from island ecosystems, i.e.,  $r_c = 0.43$ – $0.55$  (Derenne 1976) or  $r_c = 0.233$ – $1.171$  (van Aarde 1978, 1983). With a FIV transmission coefficient of 3.5 and a disease-induced mortality rate of 0.2

TABLE 1. Different outcomes of the predator population and of the whole system following the introduction of the two feline viruses, according to  $R_0$  and  $R_1$ .

| $R_0$ | $R_1$ | Introduction of FIV          |   | Introduction of FeLV         |   |
|-------|-------|------------------------------|---|------------------------------|---|
|       |       | Predator fate                | System fate   | Predator fate                | System fate   |
| $<1$  |       | Vanishing of the disease     | $\begin{cases} N^* = K \left( \frac{r_n - 1}{r_n} \right) \\ C^* = K \left( \frac{r_n - 1}{\mu \times r_n} \right) \end{cases}$   | Vanishing of the disease     | $\begin{cases} N^* = K \left( \frac{r_n - 1}{r_n} \right) \\ C^* = K \left( \frac{r_n - 1}{\mu \times r_n} \right) \end{cases}$   |
| $>1$  | $<1$  | Extinction of the population | $\begin{cases} N = K \\ C = 0 \end{cases}$  | Extinction of the population | $\begin{cases} N = K \\ C = 0 \end{cases}$  |
| $>1$  | $>1$  | Stable equilibrium state     | $\begin{cases} N^* = K \left[ 1 - \frac{1}{r_n} \left( 1 - \frac{\alpha y^*}{r_c} \right) \right] \\ C^* = \frac{N^*}{\mu} \left( 1 - \frac{\alpha y^*}{r_c} \right) \end{cases}$ | Stable equilibrium state     | $\begin{cases} N^* = K \left[ 1 - \frac{1}{r_n} \left( 1 - \frac{(b + \alpha)y^*}{r_c} \right) \right] \\ C^* = \frac{N^*}{\mu} \left( 1 - \frac{(b + \alpha)y^*}{r_c} \right) \end{cases}$ |

Notes:  $R_0$  is the reproductive rate of the disease, and  $R_1$  is the net reproductive coefficient of the population when the disease is endemic.  $N^*$  and  $C^*$  are the number of prey and number of predators, respectively, at equilibrium;  $r_n$  and  $r_c$  are the intrinsic growth rates of prey and predator populations, respectively.  $K$  is the equilibrium prey population carrying capacity,  $b$  is the predator birth rate,  $\mu$  is the annual individual intake of prey per individual predator,  $\alpha$  is the disease-induced mortality rate, and  $y^*$  is the prevalence rate of the virus within the population at equilibrium.

(Courchamp et al. 1995b), we find that both  $R_0$  and  $R_1$  are always higher than 1, indicating that the disease will always remain within the predator population.

The impact ( $I$ ) of the virus on the predator population size may be defined as the difference between the predator population size at equilibrium in the absence of the virus (the carrying capacity of the habitat) and the number of individuals at equilibrium when the disease is endemic:  $I = (N/\mu) - C^*$ , which is  $(N^*/\mu) \times (\alpha y^*/r_c)$ . Similarly, the effect of virus introduction on the prey population is given by an increase of  $(K/r_n) \times (\alpha y^*/r_c)$  prey in the population.

We can also define the values of the predator population parameters for which the virus successfully eradicates the population. We previously saw in Eqs. 5 and 6 that the conditions are  $R_0 > 1$  and  $R_1 < 1$ . This is equivalent to  $\sigma - \alpha - b > 0$  and  $\alpha[1 - b/(\sigma - \alpha)] - r_c > 0$ . With values of  $\sigma$  and  $\alpha$  previously defined (3.5 and 0.2, respectively; Courchamp et al. 1995b), FIV can eradicate the cat population, provided that its intrinsic growth rate  $r_c$  is such that  $0 < r_c < 0.17$ . Thus, the dynamic characteristics of a cat population in island ecosystems provide an indication of the potential of FIV for eradicating the cat population. However, the maximum possible value of  $r_c$  allowable under this scenario is low compared to most estimates in island ecosystems ( $\geq 0.43$ , e.g., Derenne 1976, van Aarde 1983), and it is unlikely that FIV would be able to fully eradicate most introduced cat populations (Fig. 1C).

*Introduction of FeLV*

When using the previous biological values for the cat population parameters and the value of the FeLV parameters used previously ( $\pi = 0.33$ ,  $\alpha = 0.53$ , and

$\sigma = 10$ ; Fromont et al. 1997), we again find that, provided that  $R_0 > 1$ , we always have  $R_1 > 1$ . That is, if the virus does not vanish, then the disease will spread without causing the extinction of the predator population. The impact ( $I$ ) of the virus on the predator population size is given here by  $I = (N^*/\mu) \times [(b + \alpha)y^*/r_c]$ . Similarly, the effect on the prey population of virus introduction is an increase in the number of individuals at equilibrium, given by  $(K/r_n) \times [(b + \alpha)y^*/r_c]$ .

As with the previous model, we can also identify values of the cat population parameters that lead to its eradication by the introduction of FeLV. We previously saw (Table 1) that the conditions are  $R_0 > 1$  and  $R_1 < 1$ . This is equivalent to  $\sigma\pi - \alpha - b > 0$ , and  $(b + \alpha)y^* - r_c > 0$ . For FeLV, for which a rather high proportion of the cat population is naturally immunized in mainland populations (up to two-thirds; Fromont et al. 1997), the effect of a virus introduction also depends on the actual proportion  $\pi$  that would suffer from FeLV infection in the particular ecosystem. Relatively few individuals are responsible for the founding of the present populations on several islands. Thus, it is reasonable to hypothesize that the natural immunity to FeLV is not present in the insular population at the same level as in other populations. The small number of founders of several island populations could even have led to some totally susceptible populations. To evaluate the eradication potential of FeLV, we therefore must account for different values of the proportion of naturally immunized individuals.

The range of values of the proportion of non-immunized individuals,  $\pi$ , required for FeLV to eradicate its host population is given in Fig. 2 as a function of the intrinsic growth rate ( $r_c$ ) of the predator population.

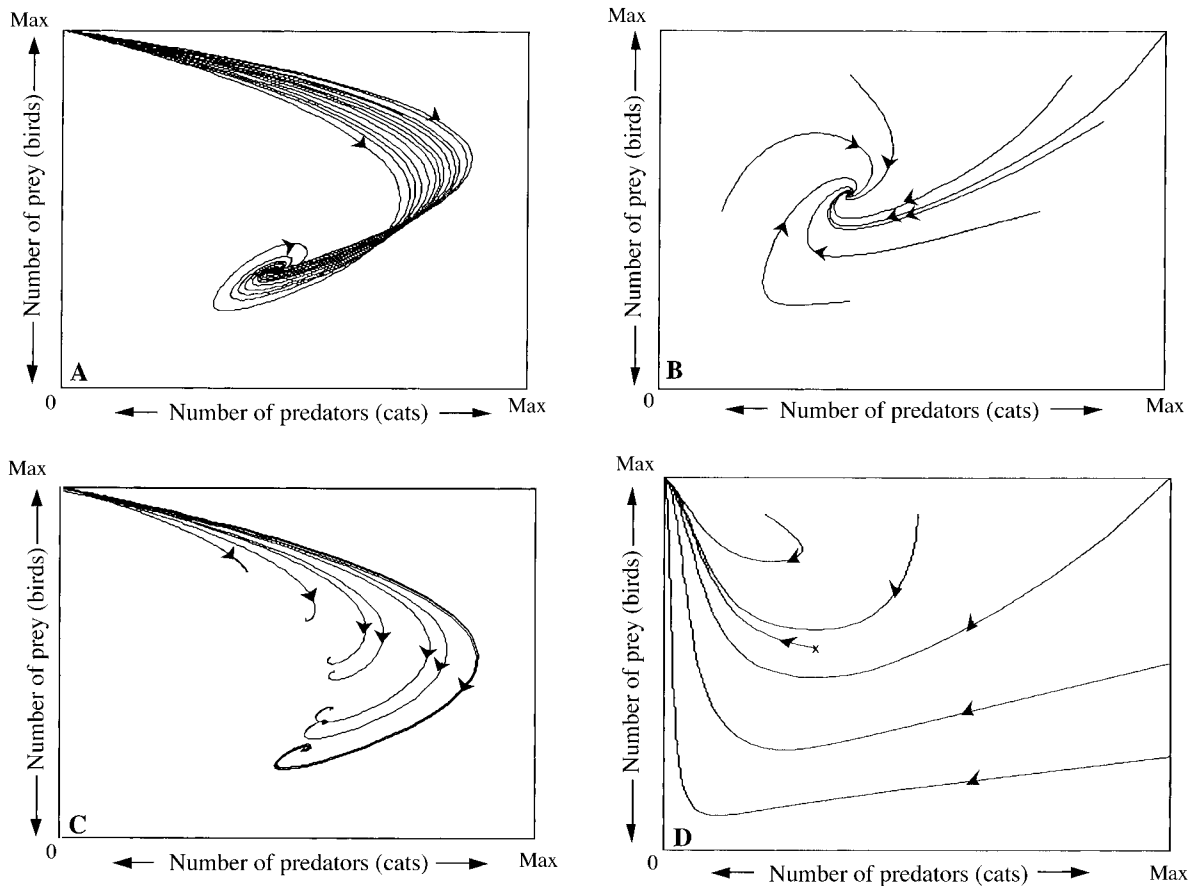


FIG. 1. Phase portraits of prey and predator population dynamics (A) in the absence of feline virus and as a function of different intrinsic growth rates for the predator population, for one unique initial population size (prey population at maximum,  $1 \times 10^7$ , and 10 introduced predators); and (B) in the absence of virus, for different initial population sizes. Whatever the predator population growth rate and the initial conditions, the system reaches a stable equilibrium point. (C) Effect of introducing a FIV-like virus in the predator population. The bottom bold curve represents population dynamics in the absence of viruses for the same initial population sizes as in (A). Upper curves show the effect on both populations of introducing the virus, with different values of transmission coefficient and disease-induced mortality rate. Whatever those values (and the initial conditions and intrinsic growth rate, not shown), an equilibrium state is reached between the predator and prey populations. However, increased values for these two viral parameters induce an increase in the prey population. (D) Effect of introducing a FeLV-like virus in the predator population, shown for different initial population sizes. Whatever these initial conditions ("x" indicates an initial condition corresponding to the disease-free equilibrium), the predator population goes extinct, and the prey population reaches its predator-free equilibrium size, provided the natural immunity rate is lower than previously defined.

The values of  $r_c$  that are available in the literature for several islands where cats are threatening birds species (e.g., Derenne 1976, Van Aarde 1983) are within the range of those for which the model predicts eradication of the cat population by the introduction of FeLV (Fig. 1D). The feasibility of eradicating the feral cat population of a given island by introduction of FeLV thus requires a determination of the proportion of naturally immunized individuals in the population, in addition to the populations' vital rates. Fig. 2 shows that no culling effort is necessary to eradicate the population if the proportion of non-immunized individuals is high enough. It also shows that eradication occurs for high values of  $\pi$  and for low values of  $r_c$ ; all other parameters being equal, it is easier to eradicate a population with

a low intrinsic growth rate and/or with a high susceptibility level.

#### *Combined effects of culling and virus introduction*

We can estimate the minimal culling effort ( $\delta$ ) required to eradicate a predator population with a given growth rate when a virus is introduced. Fig. 3 provides the range of values of  $\delta$  and  $r_c$  for which introduction of FIV, in addition to culling, results in eradication of the predator population. This figure shows that eradication is possible for lower intrinsic growth rates and/or for higher culling efforts. Eradication also depends on  $C/N$ , the ratio of the number of cats to the number of birds. The lower the value of  $C/N$ , the more difficult eradication is. This is because the number of birds de-

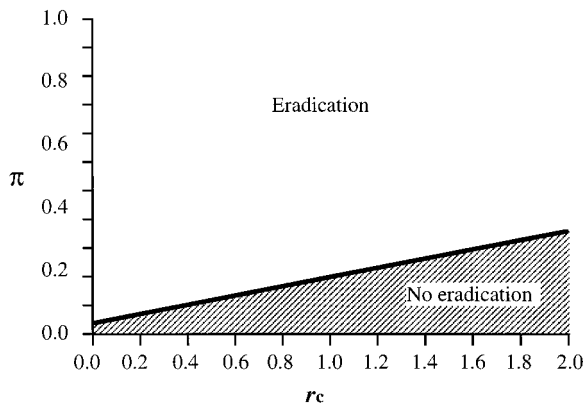


FIG. 2. Limit (bold line) between possible eradication (white area) and no eradication (gray area) of the predator population by virus introduction, given by different values of the population growth rate,  $r_c$ , and FeLV susceptibility rate,  $\pi$  (the natural immunity rate is  $1 - \pi$ ). Populations with higher  $\pi$  and/or lower  $r_c$  are more likely to be eradicated.

termines the carrying capacity of cats, and cat dynamics will be the strongest when cats are far from the carrying capacity. As the population is being eradicated, the number of cats decreases and the number of birds increases. As  $C/N$  decreases, eradication becomes increasingly difficult. The fewer cats there are, the more difficult it is to eradicate them, and the culling rate must therefore be increased until complete eradication. Fig. 4 provides the range of  $\delta$ ,  $r_c$ , and  $\pi$  for which the introduction of FeLV, complemented by a culling program, can eradicate the predator population. This figure shows that eradication becomes possible as the proportion of susceptibles ( $\pi$ ) increases ( $\pi$  may be seen as the degree of weakness of the cat population toward the virus), the cat intrinsic growth rate ( $r_c$  decreases ( $r_c$  may be seen as the degree of natural vigor of the cat population), and/or the culling effort ( $\delta$ ) increases ( $\delta$  may be seen as the degree of constraint applied to the cat population). The difference between the two areas highlights the effect of  $C/N$ : the lower this ratio, the more difficult the eradication becomes.

DISCUSSION

We have presented a theoretical investigation of the effect of introducing a retrovirus into feral cat populations that prey on island bird species. Two retroviruses were used as examples, but the models are general enough to be transferable to other candidate viruses. The parameter values for the viruses were taken from previous studies from which the host-parasite part of the models is derived (Courchamp et al. 1995b, Fromont et al. 1997). The parameter values for prey and predator populations have been taken from field studies of subantarctic and tropical islands (Derenne 1976, Pascal 1980, 1983, and Weimerskirch et al. 1988 for Kerguelen; Rauzon 1985 for Jarvis; Jones 1977, and Brothers et al. 1985 for Macquarie; van Aarde 1978, 1983, and Cooper and Brown 1990 for Marion). Some

of the parameter values were collected at different times. However, it is believed that most of them stay within a range that leads to similar results. The aim of this study was to investigate the feasibility of eradicating feral cat populations from island ecosystems by introducing a retrovirus and to motivate new studies in this area, rather than to provide exact parameter values for such an eradication. Neither the nature of the model nor the state of current knowledge in conservation biology makes such exact values possible.

Any such program would require a very careful preliminary study before a pathogen is introduced into a fully susceptible ecosystem. Paradoxically, if the introduction of an alien species (microparasites) is proposed to solve the problem of the introduction of another alien species (predators), one should not forget that cats were introduced to many of these islands to solve a previous problem of alien species introduction of mice, rats, or rabbits (Moors and Atkinson 1984). Moreover, dramatic results of deliberate introductions of pathogens in fully susceptible ecosystems, such as myxomatosis in Europe and in Australia (reviews in Fenner 1994, Sumption and Flowerdew 1985) remind us that microparasite ecology is still not understood fully.

The first precaution is to assess the potential success of eradication or control using an introduced virus, including the viability of potential vectors. Introduction of myxomatosis on the Kerguelen Islands to control

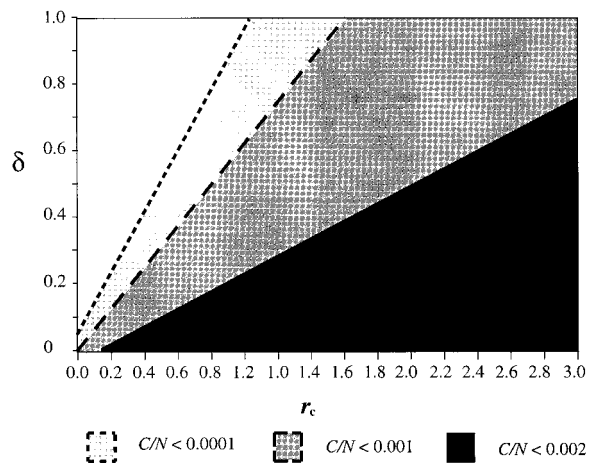


FIG. 3. The different lines represent the limit between possible persistence (darker area) and eradication (lighter area) of the predator population by simultaneous FIV introduction and continuous culling, given by different values of culling effort,  $\delta$ , and of population growth rate,  $r_c$ . For a population with a given growth rate, increasing the culling effort can lead to eradication. Similarly, for a given culling effort, eradication is more likely if the growth rate is lower. Each line is for a different value of  $C/N$ , the ratio of the number of predators to the number of prey. These values were obtained from the literature. The lower this ratio (the fewer cats there are), the more intense the culling must be to eradicate the cat population, for a given growth rate, and the lower the growth rate must be for a given culling effort.

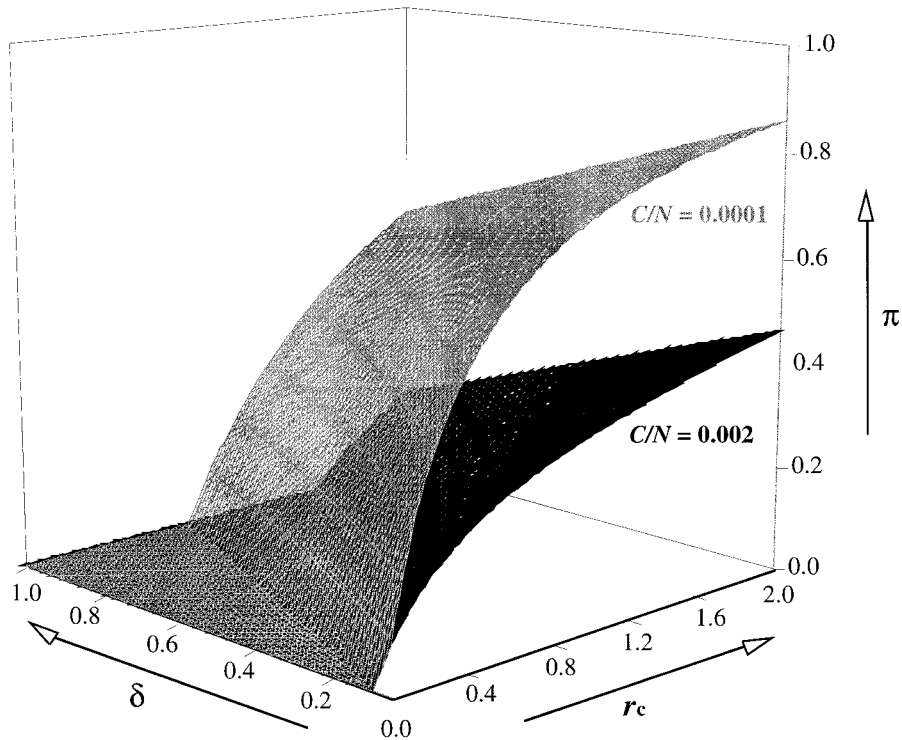


FIG. 4. The areas represent the limit above which the eradication of a cat population becomes possible, according to its intrinsic growth rate,  $r_c$ , FeLV susceptibility rate, ( $\pi$ ), and culling rate, ( $\delta$ ), when both culling and FeLV introduction are used simultaneously. In this case, increasing the value of the culling effort or of the susceptibility rate, and/or decreasing the value of the intrinsic growth rate, may make eradication possible. The upper area is for  $C/N = 0.002$ , and the lower area is for  $C/N = 0.0001$ . As  $C/N$  decreases, the eradication of a population with a given growth rate is possible with higher values of  $\delta$ . This means that the culling rate must be constantly increased until there is complete eradication.

introduced rabbit populations failed due to the unsuccessful adaptation of the insect vector to the subarctic climate (Chapuis et al. 1994). The second precaution is to assess the susceptibility of all nontarget species. It seems unlikely that birds would be susceptible to feline viruses, but at least some feline macroparasites have infected some bird species (Clark and McKenzie 1982). Moreover, several marine mammal species are often present in these insular ecosystems and little is known of their epidemiology. The introduction of such viruses on an island could potentially cause more harm than benefit to biodiversity.

We have seen that, according to our model, FeLV can totally eradicate a cat population, given a putative lower degree of natural immunity of island cats due to a founder effect. FeLV cannot eradicate cat populations with the immunity rate values that exist on continents. Similarly, FIV is unable to eradicate cat populations introduced onto islands. This is not an unexpected result, because it seems logical that pathogens would not evolve toward a total eradication of their host population if the pathogens were to survive (in our model, little distinguishes these populations from continental ones). Indeed, although it is possible that pathogens evolve increased virulence (Ewald 1994), other param-

eters such as increased transmission must then change to ensure the pathogen's survival.

FIV could thus, at best, control a feral cat population. Control should not be preferred to eradication when eradication is possible and when control decreases the probability of future successful eradication by selecting individuals who are difficult to kill using that control method. However, we argue here that, in some cases, control of the feral cat population might be preferable to total eradication. Indeed, two types of problems preclude a successful low-cost eradication of cat populations on many islands. The first concerns the vulnerability of the population itself. On the Kerguelen Islands, for example, the problems of accessibility to most parts of the main island are sufficient to preclude a reasonably simple and fast eradication of cats. As a comparison, it took 3880 person-days and 4 yr to rid the Little Barrier Island, New Zealand, of "just" 150 cats (Veitch 1985); the main island of Kerguelen is >250 times larger, most of it is barely accessible, and it harbored an increasing population of >10 000 cats in 1980 (Pascal 1983). Vulnerability is also a problem when humans live on or near the island, because it increases the likelihood of repeated reintroductions of cats.

The second problem is the presence of other introduced mammals, such as rats. Indeed, rats are notoriously harmful to most bird species. They are predators of eggs and chicks of many birds species, including some that are too large or that use burrows too narrow to be preyed upon by cats (Derenne and Mougin 1976, Karl and Best 1982). Rats may constitute a large portion of cat diets, even on islands where cats are threatening bird or reptile populations (Karl and Best 1982, Apps 1986, Henderson 1992). We have shown (Courchamp et al. 1999b) that the eradication of cats on islands where rats are present could result in a "mesopredator release" (Soulé et al. 1988). This sudden burst of mesopredators (rats) could be more harmful to indigenous bird and reptile populations than cat predation itself. Thus, on islands where total eradication of the feral cat population is estimated to be unfeasible or undesirable, introduction of FIV could be an alternative for a low-cost and efficient method of long-term cat population control.

In any case, culling programs are shown here to be more efficient when held during simultaneous virus introduction. Cats have been extirpated by culling from several islands (Fitzgerald and Veitch 1985, Rauzon 1985, Veitch 1985, Domm and Messersmith 1990). Virus introduction, followed by culling, has also been applied successfully to eradicate the feral cat populations of subantarctic Marion Island (Bloomer and Bester 1992). This population, which consisted of >3400 cats in 1977, was eradicated (Cooper 1995) by the introduction of Feline Panleucopenia Virus (van Rensburg et al. 1987) and culling, followed by trapping when the cat population decrease had begun to slow (Bloomer and Bester 1991). Once FPV had dramatically reduced the cat population, it lost its effectiveness, because cat population density was too low for transmission. The remaining individuals (~620 cats) became very difficult to remove (Bester and Skinner 1991), with removal effort inversely proportional to the number of remaining individuals. This difficulty is not taken into account in the model; accordingly, the value of  $\delta$  might have to be increased. Although the FPV alone did not eradicate cats from Marion Island, it was believed to have been an indispensable technique, without which culling likely would have failed.

Few three-species interaction models of population dynamics have yet been published. We believe that, even when they are very simple, such models may provide important information about the mechanisms of multispecies interactions (Anderson and May 1986). Our current work on the advantages of introducing two viruses simultaneously for biological control should provide further insight in this area. The importance of modeling studies is particularly clear in studies of relatively simple systems, such as the almost closed island ecosystems. This kind of theoretical work should be valuable in conserving island biodiversity, where rep-

licated conditions are rare and experimental work is seldom possible.

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