Bulletin of Mathematical Biology (2010) 72: 444–468
DOI 10.1007/s11538-009-9454-2

ORIGINAL ARTICLE

Modelling Disease Introduction as Biological Control of Invasive Predators to Preserve Endangered Prey

Nuno M. Oliveira[a,b], Frank M. Hilker[c,d,e,*]

[a] *Instituto Gulbenkian de Ciência, Apartado 14, 2781-901 Oeiras, Portugal*
[b] *Departamento de Biologia Animal, Faculdade de Ciências, Universidade de Lisboa, Campo Grande, 1749-016 Lisboa, Portugal*
[c] *Centre for Mathematical Biology, Department of Mathematical and Statistical Sciences, University of Alberta, Edmonton, AB, Canada T6G 2G1*
[d] *Centro de Matemática e Aplicações Fundamentais, Universidade de Lisboa, Complexo Interdisciplinar – Avenida Prof. Gama Pinto 2, 1649-003 Lisboa, Portugal*
[e] *Present address: Department of Mathematical Sciences, University of Bath, Bath, BA2 7AY, UK*

Received: 3 December 2008 / Accepted: 14 August 2009 / Published online: 29 September 2009

Abstract Invasive species are a significant cause of bio-diversity loss particularly in island ecosystems. It has been suggested to release pathogenic parasites as an efficient control measure of these mostly immune-naïve populations. In order to explore the potential impacts of such bio-control approach, we construct and investigate mathematical models describing disease dynamics in a host population that acts as a predator embedded in a simple food chain. The consequences of Feline Immunodeficiency Virus (FIV) introduction into a closed ecosystem are addressed using a bi-trophic system, comprising an indigenous prey (birds) and an introduced predator (cats). Our results show that FIV is unlikely to fully eradicate cats on sub-Antarctic islands, but it can be efficient in depressing their population size, allowing for the recovery of the endangered prey. Depending on the ecological setting and disease transmission mode (we consider proportionate mixing as well as mass action), successful pathogen invasion can induce population oscillations that are not possible in the disease-free predator–prey system. These fluctuations can be seen as a mixed blessing from a management point of view. On the one hand, they may increase the extinction risk of the birds. On the other hand, they provide an opportunity to eradicate cats more easily in combination with other methods such as trapping or culling.

Keywords Eco-epidemiological model · Bio-control · Invasive species removal · Island ecosystem restoration · Seabird conservation · Pathogen release · Reproduction number · Forward and backward Hopf bifurcations

*Corresponding author.
E-mail addresses: noliveira@igc.gulbenkian.pt (Nuno M. Oliveira), f.hilker@bath.ac.uk (Frank M. Hilker).

1. Introduction

Invasive species are a leading threat to bio-diversity (Lockwood et al., 2007; Wilcox and Donlan, 2007). Together with habitat degradation and human-driven atmospheric and oceanic alterations, biotic invasions are seen as major agents of global change (Mack et al., 2000; Chornesky and Randal, 2003; but see Gurevitch and Padilla, 2004 and Didham et al., 2005 for a different view).

Islands and insular-type of environments in particular are highly susceptible to invasion (Sax and Brown, 2000). Many introduced species are extremely adaptable and often find exceptionally good conditions on islands, where they are normally free from their natural competitors, predators, parasites, and pathogens (Dobson, 1988; Sax and Brown, 2000; Bax et al., 2001). In face of invaders, native species may be put at danger and are frequently driven to extinction (Moors and Atkinson, 1984), as they are not likely to have evolved defences against mainland predators and grazers accidentally or deliberately introduced (Krajick, 2005). More than 90% of species extinctions since 1600 have been island forms (Atkinson, 1989).

Control programmes, and eradication whenever possible, are largely regarded as the favoured way to solve the problems posed by invaders and to restore the ecosystem (Atkinson, 1988; Zavaleta et al., 2001). Biological control measures, and the introduction of pathogens in particular, are viewed as promising alternatives to time-consuming, costly and usually labour-intensive classical methods, such as culling and trapping programmes, especially on remote or uninhabited islands (Courchamp and Sugihara, 1999; Cleaveland et al., 1999). However, the introduction of pathogens in an ecosystem requires accurate impact studies, in order to insure that the pathogen (i) will not have adverse effects (like spreading to natives) and (ii) will result in the removal or at least significant control of the target species. The choice of suitable pathogens is of crucial importance, calling for both empirical and theoretical studies.

This paper is motivated by Courchamp and Sugihara (1999). They aim to model the effectiveness of virus introduction as biological control of invasive cats. Domestic cats are among the most notorious and damaging predators introduced in insular ecosystems (Nogales et al., 2004). For example, five cats introduced to Marion Island in 1949 resulted in a population of more than 2,000 cats 25 years later, depleting nearly half a million burrowing petrel per year. They caused the extinction of the Common Diving Petrel (*Pelecanoides urinatrix*) and severely affected some species of hole-nesting petrels (*Procellariidae*) (Van Aarde, 1980; Van Rensburg and Bester, 1988; Nogales et al., 2004). In the same period, a population of five cats introduced to the Kerguelen Islands grew to several tens of thousands, and is now estimated to kill more than three million seabirds per year (Chaphuis et al., 1994). The control of invasive cats therefore is a particular concern.

The model formulation in Courchamp and Sugihara (1999), however, is problematic, which can invalidate the authors' conclusions (cf. Section 2). We therefore develop new mathematical models that describe virus spread in a specialist predator host population and the potential impact on both predator and prey species. Cats and seabirds are used as predator–prey examples, but other interactions could be chosen instead, since our models are simple and general. Throughout this paper, we will use the terms predators and cats as well as prey and birds interchangeably.

We will focus on the Feline Immunodeficiency Virus (FIV) and its potential effects in a simple bi-trophic food chain of invasive cats and native seabird populations. The risks and limitations of using pathogens as bio-control agents are well recognised (Strong and Pemberton, 2000). However, FIV introduction seems to be a suitable and promising measure for several reasons. First, FIV is highly host-specific. Pathogen mutation and spread to other species thus seem relatively unlikely (Cleaveland et al., 1999). Second, FIV has a low virulence. This is thought to be better for ongoing transmission of the control agent within the host population (Dobson, 1988; Courchamp and Sugihara, 1999). A highly virulent virus, meaning that it kills the host rapidly, could become self-limiting, as observed on Marion Island. Feline Panleucopaenia Virus (FPLV) was introduced in 1977, upon which the cat population decreased from an estimated 3,409 to 615 cats in 1982 (Van Rensburg et al., 1987). However, at that time FPLV was no longer spreading effectively, and the population managed to recover. A complement of intensive trapping and poisoning was necessary to fully eradicate the cat population (Bester et al., 2002). FIV, on the contrary, persists for a long time before killing its host, allowing for multiple virus transmissions during the host's lifetime. As a consequence, the impact of this virus is expected to be higher on islands where cats may live 2–3 times longer than mainland populations (Courchamp et al., 2000). Third, FIV infects and kills predominantly individuals with high reproductive rates and survival probabilities (Courchamp et al., 1998), suggesting that it might be very efficient in controlling host population growth. The reasons pointed out make FIV a likely candidate for control programmes of invasive cats.

In comparison with the original study by Courchamp and Sugihara (1999), we use a substantially different approach in predator modelling, based on the principle of trophic coupling. Moreover, the functional response does depend on the prey density. This resolves the flaws of the original model, but keeps the new models still simple. We can find estimates for most of the parameters in the literature; the remaining ones are varied continuously to explore their potential impact. In the original study, some choices of parameter values appear arbitrary. Our results will demonstrate the possibility that pathogen release may destabilise the populations. This scenario was not recognised in the original model and we will discuss it in depth with related models considering disease spread in predator populations.

In addition, we take into account two different disease transmission patterns between individuals. Transmission is the driving force behind the dynamics of any infectious disease (Begon et al., 2002). The incidence depends on the spatial and social structure of the host, since it is a relation between population size, measured as a number or a density, and the rate of contact leading to disease transmission (Fromont et al., 1998). The dynamics of models developed to design strategies for managing disease threats to humans or animals, is thus greatly affected by the way in which transmission between infected and susceptible hosts is modelled (McCallum et al., 2001). We employ two different incidence functions that have been traditionally used. The first one assumes that the contact rate between hosts is constant (proportionate mixing transmission, also called frequency-dependent transmission or standard incidence), and the other one assumes that it increases directly with host population size (mass action transmission, also called density-dependent transmission). Both assume that the host population is homogeneously mixed. However, they are considered to be two modelling extremes in a continuum of possibilities (McCallum et al., 2001).

The remainder of this paper is organised as follows. Section 2 presents a brief overview of previous theoretical models on this subject. Section 3 introduces our models and their assumptions. In Sections 4 and 5, we present the results of this study, first the equilibria of each model and their local stability, and then the impact analysis of virus introduction. Finally, in Section 6, we discuss our models and results and draw conclusions important for alien species control and management.

2. Existing mathematical models

Mathematical modelling has much to offer to biological control. Models appear as useful and valid alternatives to risky and time-consuming field experiments. They allow the identification of critical processes and parameters that drive the dynamics of species interactions. Their broad and often simple, yet insightful, conclusions can be of great value in planning future biological control programmes (Waage and Greathead, 1988). In the past, theoretical models have focused on the use of arthropod natural enemies, especially parasitoids. Then models for the use of pathogens have been explored. In the 1980s, modelling efforts toward the understanding of the dynamics of insect–pathogen interactions developed (e.g. Anderson and May, 1981; Anderson, 1982; May and Hassell, 1988). Recent theoretical advances show how useful ecological theory can be in biological control (Murdoch and Briggs, 1996).

The original model by Courchamp and Sugihara (1999) is based on an underlying predator–prey model. In the absence of disease, it can be described by the following pair of ordinary differential equations:

$$\frac{\mathrm{d}N}{\mathrm{d}T} = r_N N\left(1 - \frac{N}{K}\right) - \mu P, \tag{1}$$

$$\frac{\mathrm{d}P}{\mathrm{d}T} = r_P P\left(1 - \frac{P}{(N/\mu)}\right), \tag{2}$$

where $N(T)$ and $P(T)$ denote the number of individuals at time T in the prey and predator populations, respectively; K is the carrying capacity of the prey's environment (measured in individuals); r_N and r_P are the intrinsic growth rates of the prey and predators, respectively (measured in time^{-1}); and μ is the predation rate. The model is flawed for the following reasons:

- First of all, the model is dimensionally incorrect. Parameter μ is measured in time^{-1} in Eq. (1) and is dimensionless in Eq. (2).
- Moreover, for $N \equiv 0$, the time–derivative describing the rate of change of the prey population (Eq. (1)) becomes negative when predators are introduced ($P > 0$). This leads to negative population sizes, which does not make biological sense. Also, note that Eq. (2) is not defined for $N \equiv 0$.

Hence, despite its heuristic value, the model is ill-posed and the conclusions based upon it cannot be taken for granted. Furthermore, the model has the following drawbacks:

(1) The functional response of the predators (the number of prey each predator consumes per unit time) is constant. Each predator is assumed to consume a fixed number of prey

per unit time—regardless of the size of the prey population, which appears unrealistic. This and the following point have also been remarked by Fan et al. (2005).

(2) The model precludes the possibility of oscillatory dynamics. Although we are not aware of oscillations in cat or bird populations on oceanic islands, regular fluctuations are often observed in predator–prey communities (e.g. Turchin, 2003), and their absence may be viewed as a model limitation.

(3) The type of predator–prey relationship violates the biomass conversion principle, which states that the rate of reproduction of predators is uniquely determined by their functional response (Ginzburg, 1998; Turchin, 2003).

The model most similar to the one presented here is by Hilker and Schmitz (2008). Their starting point is the classical Rosenzweig–MacArthur (1969) model for the underlying predator–prey system, which they couple with a simple disease model that has been suggested for FIV spread (Courchamp et al., 1995). The functional response of the predators is of Holling-type II, which resolves all the drawbacks (1)–(3) listed above. Other predator–prey models accounting for disease spread in the predator population can be found in Anderson and May (1986), Venturino (1994), Han et al. (2001), Venturino (2002), Xiao and Van Den Bosch (2003), Fenton and Rands (2006), Haque and Venturino (2007) and Auger et al. (2009); see also the review by Hatcher et al. (2006).

In this paper, we choose an underlying predator–prey model of Lotka–Volterra type. The advantage is that this model has one parameter less (namely the prey-handling time). It is difficult to find estimates of this parameter in the literature. Therefore, our "step back" to the Lotka–Volterra predation submodel allows us to bypass this problem and to use parameter values that can be considered realistic (cf. Section 3.2). While the Lotka–Volterra model does not allow for predator–prey oscillations, we will find that the introduction of disease can trigger population cycles.

As already mentioned, Courchamp et al. (1995) construct a model to study FIV circulation in populations of domestic cats. Other viruses such as the Feline Leukaemia Virus (FeLV) and FPLV are also the subject of theoretical studies on the dynamics of disease spread among feline hosts (Fromont et al., 1998 and Berthier et al., 2000, respectively). Regarding the parvovirus FPLV, the latter authors conclude that it is unable to eradicate an exponentially growing population of cats with the characteristic of the Marion Island population, which is in agreement with field observations. Neither of these models, however, considers the impact of the bio-control agent introduction on a prey population.

3. Model descriptions

3.1. Eco-epidemiological models

This section introduces the mathematical models, which combine two sub-models, namely one describing the predator–prey dynamics of cats and birds and the other one describing disease spread within the cats. These types of coupled models are also called eco-epidemiological models. Figure 1 shows a corresponding transfer diagram.

We begin with the simple Lotka–Volterra predation model (Volterra, 1931)

$$\frac{\mathrm{d}N}{\mathrm{d}T} = rN\left(1 - \frac{N}{K}\right) - \mu NP, \tag{3}$$

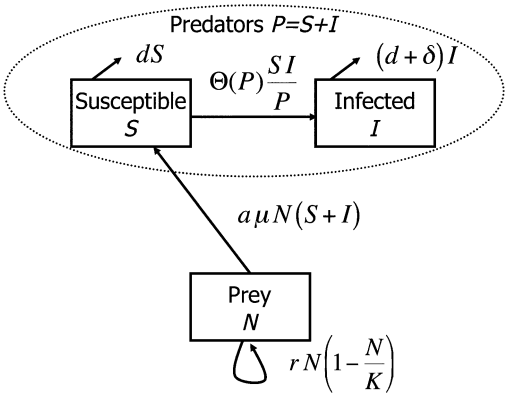

Fig. 1 Transfer diagram of the eco-epidemiological model in terms of susceptible and infected predators and the prey, cf. equations (3), (5) and (6).

$$\frac{\mathrm{d}\,P}{\mathrm{d}\,T} = a\mu N P - d P, \tag{4}$$

where $N(T)$ and $P(T)$ denote the population size of the prey (birds) and predators (cats), respectively, at time T. The prey population has an intrinsic per-capita growth rate r and a carrying capacity K. The functional response of predators is linear with predation rate μ per capita and unit prey. The predator population has a per-capita mortality rate d and a trophic conversion efficiency a.

For the spread of the infectious disease within the predator population, we assume that FIV splits the cats into a susceptible (S) and an infected (I) part without recovery or immunity, i.e. $P = S + I$. This follows Courchamp et al. (1995), where there is only one stage of illness combining all five stages of FIV: acute, seropositivity, PGL, ARC, and AIDS stages. Infected individuals suffer an additional disease-induced death rate δ (also referred to as virulence) and their predatory behaviour is not altered due to disease. There is no vertical transmission. Hence, the predator Eq. (4) is replaced by the following two differential equations:

$$\frac{\mathrm{d}\,S}{\mathrm{d}\,T} = a\mu N P - d S - \Theta(P)\frac{S I}{P}, \tag{5}$$

$$\frac{\mathrm{d}\,I}{\mathrm{d}\,T} = \Theta(P)\frac{S I}{P} - d I - \delta I. \tag{6}$$

$\Theta(P)$ is the effective contact rate leading to disease transmission. If the contact rate between predator individuals is constant ($\Theta(P) = \beta_{\mathrm{PM}}$), the incidence is of proportionate mixing type. If the contact rate between predator individuals increases linearly with population size ($\Theta(P) = \beta_{\mathrm{MA}} P$), the incidence is of mass action type. The former has been suggested in the original FIV model by Courchamp et al. (1995), whereas there are conditions in which the latter is appropriate (Fromont et al., 1998; Hilker et al., 2009).

It can be advantageous to rewrite the model equations for S and I in P and i state variables, with i being the prevalence of the disease, i.e. $i = I/(S + I)$. This allows to distinguish disease-induced extinction from the trivial equilibrium and to deal with the

singularity at $S = I = 0$ (the denominator of the PM transmission term becomes zero) as well. Moreover, this choice of state variables usually preserves a handy equation for the total host population.

Introducing dimensionless variables, the number of parameters can be reduced from seven to four, thus simplifying analytical investigations. Choosing $B = N/K$, $C = P\mu/r$ and $t = rT$, we obtain

$$\frac{\mathrm{d}B}{\mathrm{d}t} = B(1 - B) - BC, \tag{7}$$

$$\frac{\mathrm{d}C}{\mathrm{d}t} = eBC - mC - \alpha Ci, \tag{8}$$

$$\frac{\mathrm{d}i}{\mathrm{d}t} = \big(\big[\Gamma(C) - \alpha\big][1 - i] - eB\big)i, \tag{9}$$

where

$$\Gamma(C) = \frac{\beta_{\mathrm{PM}}}{r} = \sigma_{\mathrm{PM}} \quad \text{and} \quad \Gamma(C) = \frac{\beta_{\mathrm{MA}}}{\mu}C = \sigma_{\mathrm{MA}}C \tag{10}$$

for proportionate mixing and mass action transmission, respectively, and

$$e = \frac{a\mu K}{r}, \qquad m = \frac{d}{r}, \qquad \alpha = \frac{\delta}{r}. \tag{11}$$

Our models are described by the three Eqs. (7), (8), and (9). We will refer to the system with proportionate mixing and mass action transmission as PM and MA model, respectively.

3.2. Parameter values for the birds–cats–FIV system

We have used numerical values for the parameters that appear to be biologically acceptable, even though somewhat arbitrary. Our goal in the equilibria and stability analysis (Section 4) is to capture major dynamical features. However, when addressing the potential impact of FIV introduction in simple island ecosystems (Section 5), we resort to parameter values published in the literature. Since this work is not concerned with seasonal fluctuations, short-term behaviour or variation over space, we focus on intermediate values in the range of estimates made available in the literature, in order to represent average behaviour across time and space. Table 1 summarises the values used for the dimensional parameters, along with the corresponding references. Note that the dimensionless parameters can be obtained according to Eqs. (10) and (11).

There are several references to the biology of the domestic cat on sub-Antarctic islands such as Marion Island and Kerguelen Islands (e.g. Van Aarde, 1979; Van Rensburg et al., 1987; Say et al., 2002). However, due to the high variability of these parameters, in particular the cats' natural death rate d, we employ an averaged value previously used elsewhere (Courchamp et al., 1995), which was obtained from monitoring rural cat populations in France. Estimates for the virulence α and transmissibility σ_{PM} of FIV within cats come from Courchamp et al. (1995, 2000) and Courchamp and Sugihara (1999).

The literature on the biology of seabirds is vast (e.g. Williams et al., 1975, 1979; Siegfried, 1978; Berruti et al., 2000; Rounsevell and Copson, 1982; Schramm, 1986;

Table 1 Population and disease parameters for sub-Antarctic islands

Parameter	Estimated value	Reference
r, per-capita growth rate of birds	0.1	Rounsevell and Copson (1982)
K, carrying capacity of birds	–	
μ, predation rate	–	
a, trophic conversion efficiency	0.03	Pimm (1982)
d, natural per-capita death rate of cats	0.6	Courchamp and Sugihara (1999)
β_{PM}, contact rate	1.5	Courchamp et al. (2000)
β_{MA}, contact rate	–	
δ, virulence	0.2	Courchamp et al. (1995)

The impact of parameters K, μ and β_{MA} is investigated by varying dimensionless parameters e and σ_{MA}, using additional information on the intrinsic growth rate of cats on oceanic islands (cf. Section 3.2).

Ryan, 1987; Brooke et al., 1988; Cooper and Brown, 1990; Cooper et al., 1995; Sæther and Engen, 2002; Granadeiro et al., 2006). These references provide evidence of large ecological variability within and between species. For the intrinsic growth rate r, we therefore use the estimate from King Penguin growth, one of the seabird species of Marion Island with lowest growth rate, aiming to model an extreme situation. Estimates for trophic conversion efficiencies of vertebrates are not easy to obtain. Birds and mammals, being endotherms, have additional requirements of keeping warm when compared to ectotherms, and thus their trophic efficiency e is thought to be less than 3% (Pimm, 1982).

The birds' carrying capacity K and the predation rate μ are the parameters for which we could find least information or the largest variability and uncertainty. Estimates for the transmissibility β_{MA} (which has a different dimension than β_{PM}) remain vague as well. Their impact will be investigated by varying dimensionless parameters $e = \frac{a}{r}\mu K$ and $\sigma_{\mathrm{MA}} = \beta_{\mathrm{MA}}/\mu$. The maximum per-capita net growth rate of cats ($\frac{a\mu K - d}{r}$ in the dimensional and $e - m$ in the dimensionless model) as well as the estimated transmissibility (σ_{PM}) define landmarks at which to orient the parameter variation for island ecosystems (Van Aarde, 1983; Courchamp and Sugihara, 1999). This will be done continuously in Section 5. The aim of the next section is to investigate determinants of community composition and to explore possible dynamical behaviour.

4. General model behaviour

In the first subsection, we introduce biologically plausible threshold quantities, namely epidemiological and demographic/ecological reproduction numbers. They determine the persistence and extinction of the disease and the predators. As shown in Hilker and Schmitz (2008), they can completely explain the entire community composition—before as well as after disease introduction. Moreover, they confirm the results from linear stability analysis of equilibria $\boldsymbol{E} = (B^*, C^*, i^*)$ (summarised in Appendix A). The advantage of reproduction numbers is the imminent biological insight they provide. The second subsection makes use of numerical bifurcation and continuation analysis. The focus will be on population oscillations induced by dynamic instabilities. Although we are considering epizootiological systems, we loosely employ notations from standard mathematical epidemiology and refer to epidemic and endemic rather than epizootic and enzootic terms.

4.1. Invasion, persistence and extinction—reproduction numbers

The ecological *basic reproduction number of the predators in the absence of disease* is

$$R_{1,0}^C = \frac{e}{m}.$$

Biologically, $R_{1,0}^C$ gives the expected number of offspring (eB^*) of an average predator individual in its lifetime ($1/m$), assuming that the prey population is at carrying capacity ($B^* = 1$). Hence, if $R_{1,0}^C > 1$, the predators are sustained by the prey; otherwise, they go extinct. The subscripts indicate that the prey is at carrying capacity 1 and the disease prevalence is 0.

The *basic reproduction number of the disease* is

$$\mathcal{R}_0 = \frac{\Gamma(C^*)}{m+\alpha}.$$

$\mathcal{R}_0$ can be defined as the number of secondary disease cases ($\Gamma(C^*)$) produced by an infectious individual during its infectious lifetime ($\frac{1}{m+\alpha}$) after its introduction into an entirely susceptible population. C^* is the cat population size at disease-free equilibrium $\boldsymbol{E}_{BC}$, i.e. $C^* = C_4^*$, cf. Appendix A. If $\mathcal{R}_0 < 1$, the infection cannot establish and the fate of the predator population depends on $R_{1,0}^C$. If $R_{1,0}^C > 1$, it will settle at the disease-free equilibrium, whereas if $R_{1,0}^C < 1$ the predators go extinct with only the prey remaining. If $\mathcal{R}_0 > 1$, the disease establishes in the predator population, and the prevalence of infected individuals stabilises to a positive value. In this case two scenarios can happen depending on the *predators' basic reproduction number in the presence of disease*

$$R_{1,i}^C = \frac{e}{m+\alpha i^*}.$$

$R_{1,i}^C$ is a threshold that takes into account the additional mortality rate α of the infected population fraction i^*. Note that the subscript 'i' in $R_{1,i}^C$ refers to the presence of disease in the predator population. If $R_{1,i}^C > 1$, the disease remains endemic in the host population and all three species coexist at the aforementioned equilibrium.

For the MA model, one can show that always $R_{1,i}^C > 1$. Hence, the disease cannot drive the predator population to extinction, which is described by $R_{1,i}^C < 1$. In the PM model, host extinction is possible, and the system then tends to either $\boldsymbol{E}_{Bi}$ or $\boldsymbol{E}_B$. In both cases, the predators disappear, but in the process of vanishing predators the prevalence either remains positive or approaches zero, respectively. The former case corresponds to disease-induced extinction. In the latter case, the disease goes extinct before the predators do. These two scenarios can be distinguished by the *prevalence reproduction number*

$$\mathcal{R}_i = \frac{\sigma_{\mathrm{PM}} - \alpha}{e} = \frac{\mathcal{R}_0}{R_{1,i}^C}, \tag{12}$$

which is defined only for the PM model (as there is no disease-induced extinction in the MA model). $\mathcal{R}_i$ corresponds to the number of secondary infections discounted by the fact that the total host population and the infecteds change differently. Consider the situation, in which the prevalence is small compared to the host population. Then the infected part

approximately decays at a rate $\sigma_{\mathrm{PM}} - \alpha - m$. The total host population, however, decays at an approximate rate of $e - m$. The difference between these two rates determines whether the prevalence grows ($\mathcal{R}_i > 1$) or decays ($\mathcal{R}_i < 1$) and therefore makes up the value of $\mathcal{R}_i$ in (12). The prevalence reproduction number can also be obtained mathematically by setting the prevalence equation (9) to zero and solving for zero-growth conditions.

Note that $\mathcal{R}_i$ can be substituted by the ratio of $\mathcal{R}_0$ and $R^C_{1,i}$. The persistence of all three species involved can therefore be characterised by three reproduction numbers for both the MA and the PM model. Three different outcomes in terms of community composition are possible: (i) prey alone, (ii) prey and predators, and (iii) prey, predators, and disease. The prey can always survive, hence the equilibria $\boldsymbol{E}_0$ and $\boldsymbol{E}_i$ are always unstable. Since the predator is a specialist, it always needs the prey to persist. Similarly, the disease needs the predators as a host.

4.2. Oscillations and stability—numerical continuation of bifurcations

While the reproduction numbers are useful in categorising the persistence or extinction of populations, they do not give information about the stability of equilibria. This will be the subject in the remainder of this section. Tables 2 and 3 summarise the community

Table 2 Community composition and stability of equilibria $\boldsymbol{E} = (B^*, C^*, i^*)$ in the PM model

	$R^C_{1,0} > 1$			$R^C_{1,0} < 1$	
	$\mathcal{R}_0 < 1$	$\mathcal{R}_0 > 1, R^C_{1,i} > 1$	$\mathcal{R}_0 > 1, R^C_{1,i} < 1$	$\mathcal{R}_i < 1$	$\mathcal{R}_i > 1$
$\boldsymbol{E}_B = (1, 0, 0)$	Unstable	Unstable	Unstable	Stable	Unstable
$\boldsymbol{E}_{Bi} = (1, 0, i^*_6)$	–	–/unstable	Stable	–	Stable
$\boldsymbol{E}_{BC} = (B^*_4, C^*_4, 0)$	Stable	Unstable	Unstable	–	–
$\boldsymbol{E}_{BCi} = (B^*_5, C^*_5, i^*_5)$	–	Stable/cyclic	–	–	–
	Birds–cats	*Birds–cats–FIV*		*Birds only*	

$R^C_{1,0}$ and $R^C_{1,i}$ are the demographic reproduction numbers of the disease-free and infected predator population, respectively. $\mathcal{R}_0$ is the endemic reproduction number of the disease. $\mathcal{R}_i$ is the reproduction number of the disease prevalence within the predator population and can be substituted by $\mathcal{R}_0/R^C_{1,i}$. Details of the stationary states are in appendix A. '–' indicates that an equilibrium does not exist or is unfeasible. 'Cyclic' means that an equilibrium is unstable and surrounded by a limit cycle. The equilibria $\boldsymbol{E}_0 = (0, 0, 0)$ and $\boldsymbol{E}_i = (0, 0, 1)$ are always unstable and therefore omitted.

Table 3 Community composition and stability of equilibria $\boldsymbol{E} = (B^*, C^*, i^*)$ in the MA model

	$R^C_{1,0} > 1$		$R^C_{1,0} < 1$
	$\mathcal{R}_0 < 1$	$\mathcal{R}_0 > 1$	
$\boldsymbol{E}_B = (1, 0, 0)$	Unstable	Unstable	Stable
$\boldsymbol{E}_{BC} = (B^*_4, C^*_4, 0)$	Stable	Unstable	–
$\boldsymbol{E}_{BCi} = (B^*_5, C^*_5, i^*_5)$	–	Stable/cyclic	–
	Birds–cats	*Birds–cats–FIV*	*Birds only*

Equilibria $\boldsymbol{E}_0 = (0, 0, 0)$ and $\boldsymbol{E}_i = (0, 0, 1)$ are always unstable and therefore omitted. All other notations as in Table 2.

composition and the stability properties (as determined by reproduction numbers, linear stability and numerical bifurcation analysis) for the PM and MA model, respectively.

Both systems admit a positive periodic solution, where birds, cats and infection coexist in an oscillatory regime. Figures 2a and 2b present bifurcation diagrams for the PM and MA model, respectively. The bird population size is shown for varying transmissibilities. The cats are assumed to exist on the birds in the absence of disease. For low transmissibility values, both systems stabilise at $\boldsymbol{E}_{BC}$. As long as $\mathcal{R}_0 < 1$, the disease cannot invade, and the bird and cat populations remain constant since the predator–prey subsystem does not depend on disease parameters.

Upon disease establishment ($\mathcal{R}_0 > 1$), cats decrease in population size (not shown) which indirectly leads to the continuous increase of the bird population. For further increasing values of the transmissibility, the species start to oscillate with increasing amplitudes. However, the amplitudes of the cycles start to shrink again, and the oscillations eventually stabilise. Mathematically, the onset and disappearance of these limit cycles correspond to forward and backward Hopf bifurcations, respectively. For even larger transmissibilities, the disease continues to depress cat population size and ultimately drives it to extinction in the PM model. The birds then reach their carrying capacity (Fig. 2a). In the MA model, cat eradication is not possible and all three species remain at positive levels for large transmissibilities (cf. Fig. 2b).

A two-parameter continuation analysis is shown in Fig. 3 with virus transmissibility (σ_{PM} or σ_{MA}) and the predators' trophic conversion efficiency (e) as bifurcation parameters. Four different scenarios can be distinguished in the PM model (Fig. 3a). Parameter region 1 corresponds to the disease-free equilibrium ($\boldsymbol{E}_{BC}$) being stable, region 2 corresponds to the stable coexistence state ($\boldsymbol{E}_{BCi}$), region 3 corresponds to the unstable coexistence state, characterised by cyclic dynamics (stable limit cycles), and region 4 corresponds to the stable disease-induced extinction state ($\boldsymbol{E}_{Bi}$). The MA model allows for three different parameter regions, as there is no disease-induced extinction (Fig. 3b). Cat eradication in the PM model is possible for highly transmissible viruses and small trophic conversions. Limit cycle oscillations require a minimum efficiency of trophic conversion and intermediate ranges of transmissibility values.

5. Impact analysis for sub-Antarctic ecosystems

The previous section identified invasion, persistence and extinction conditions as well as potential dynamical behaviour. This section is concerned with the consequences of FIV release on oceanic islands. We therefore use existing parameter values and vary unknown parameters in ranges that can be regarded as plausible for sub-Antarctic islands (cf. Section 3.2). The aim is to investigate the impact of pathogen introduction on the host (predator) population as well as on the prey population. To measure the effect of biological control, we consider the following quantity to which we shall refer as perturbation of the cat population by FIV introduction:

$$\frac{C^{\bullet} - C^{\circ}}{C^{\circ}} \times 100.$$

$C^{\bullet}$ and C° denote the equilibrium values of the cat population after and before disease introduction, respectively. Note that $C^{\bullet} = C_5^*$ if the disease successfully invades or $C^{\bullet} =$

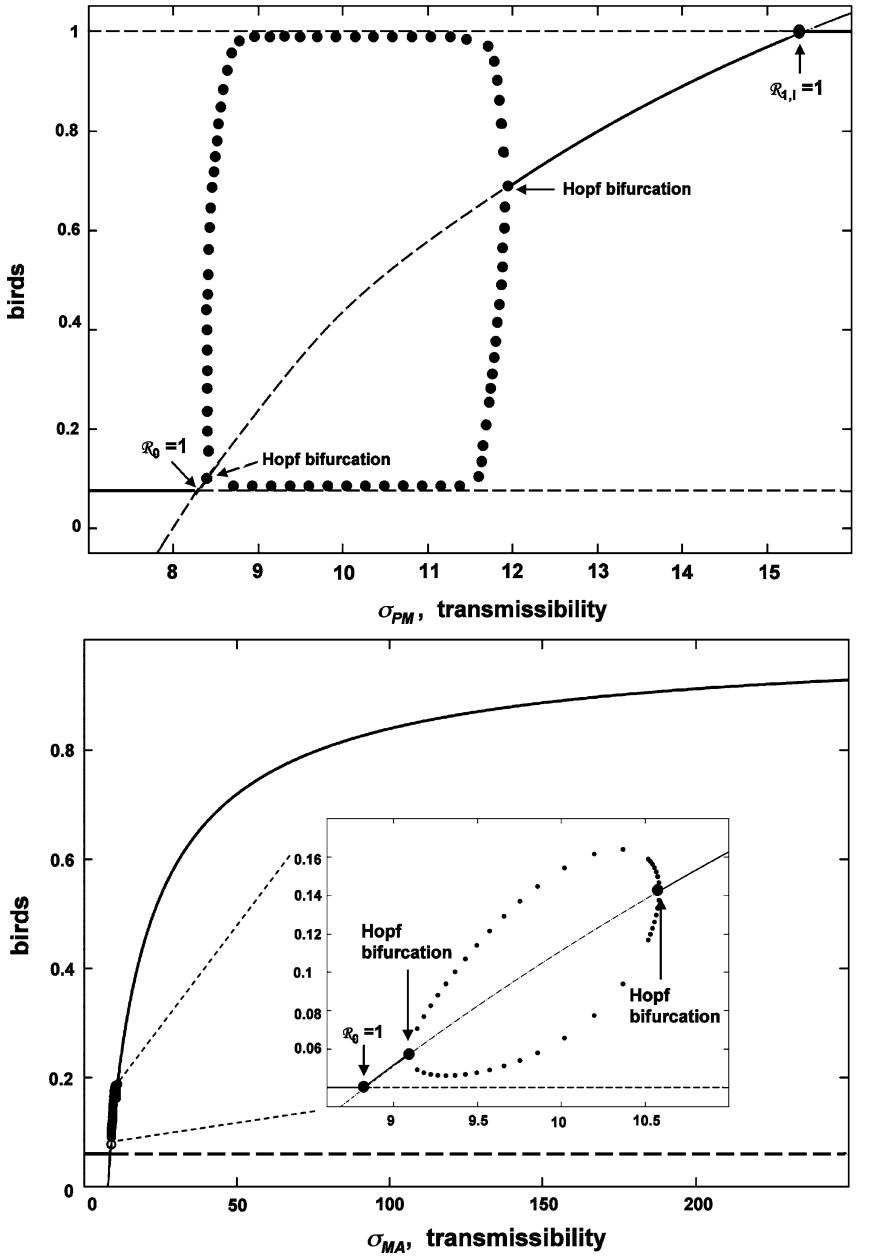

Fig. 2 Bifurcation diagram generated by *XPPAUT* for the bird population in the PM model (top panel) and the MA model (bottom panel). Solid (dashed) lines correspond to stable (unstable) equilibria. Filled circles indicate the maximum and minimum amplitudes of stable limit cycle oscillations. Zero solutions are not shown. Parameter values: $m = 0.3$, $\alpha = 8$, $e = 4$ (top) and $e = 5$ (bottom).

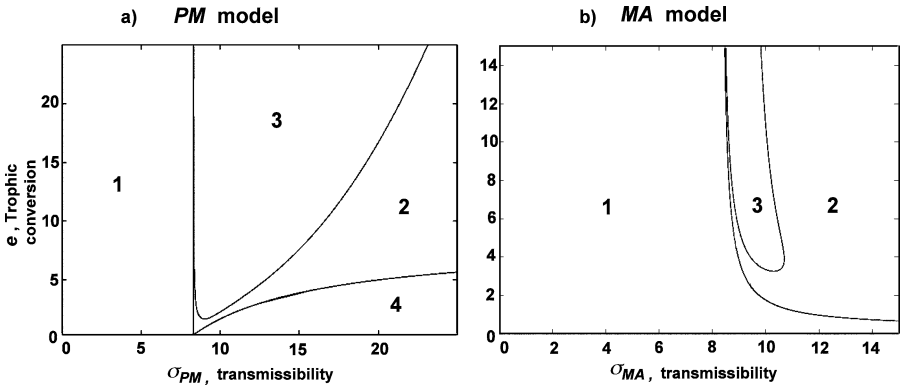

Fig. 3 Classification of the dynamic model behaviour. The two-parameter diagrams are obtained from *XPPAUT* by continuing each bifurcation (transcritical and Hopf bifurcations). Possible outcomes: (1) disease cannot establish; (2) endemic coexistence; (3) cyclic endemic coexistence; and (4) disease-induced predator extinction. $\mathcal{R}_0 = 1$ corresponds to the transcritical bifurcation line separating regions 1 and 2. $R^C_{1,i} = 1$ corresponds to the transcritical bifurcation line separating regions 2 and 4. The Hopf bifurcation line between regions 2 and 3 indicates the (dis-)appearance of limit cycle oscillations. Fixed parameter values: $m = 0.3$ and $\alpha = 2$.

C^*_4 otherwise (cf. Appendix A). For the disease-free value, $C^\circ = C^*_4$ or $C^\circ = 0$ depending on whether the predators can exist on the prey. The perturbation of the bird population is defined analogously. Thus, the perturbation measure basically is the percentual variation of the abundance of each species after virus introduction. If the model exhibits cyclic dynamics, we rather use the mean population size averaged over an oscillation.

Figure 4 illustrates the potential impact of introducing FIV in a cat population with either PM or MA transmission, for both the host and its prey. First of all, one can clearly see the minimum transmissibility required for disease establishment (i.e. $\mathcal{R}_0 > 1$). In the PM model, this is a constant. In the MA model, this varies with trophic conversion efficiency. Otherwise, if $\mathcal{R}_0 < 1$, the disease does not invade (i.e. $C^\bullet \equiv C^\circ$, $B^\bullet \equiv B^\circ$) and the perturbations of cats and birds remain zero. One can also clearly identify the critical value of trophic conversion efficiency e, for which the predators' disease-free basic reproduction number is equal to one. For $R^C_{1,0} < 1$, the predators cannot exist even in the absence of disease, i.e. $C^\bullet \equiv C^\circ \equiv 0$. The perturbation of cats is not defined in this parameter range. The birds remain at their carrying capacity ($B^\bullet \equiv B^\circ \equiv 1$), hence there is no impact on them.

Successful disease introduction, in general, leads to a reduction in predator population size, indirectly promoting an increase in the prey. We now consider the two different incidence terms separately. In the PM model, cats appear to be not that much affected by FIV for large values of the trophic conversion efficiency (Fig. 4a). Increased consumption rates balance the disease-related mortality. For small conversion efficiencies, however, FIV is capable of reducing its host significantly. The disease can even eradicate the cats ($R^C_{1,i} < 1$, not marked in Fig. 4a), provided the trophic conversion of the host is sufficiently low. Despite the lack of knowledge regarding real numerical values for this parameter, such low values do not seem realistic (Section 3.2). Thus, our results suggest that FIV cannot eradicate cat populations and depresses them only marginally in ecological settings similar to sub-Antarctic islands. However, it can be effective in allowing the

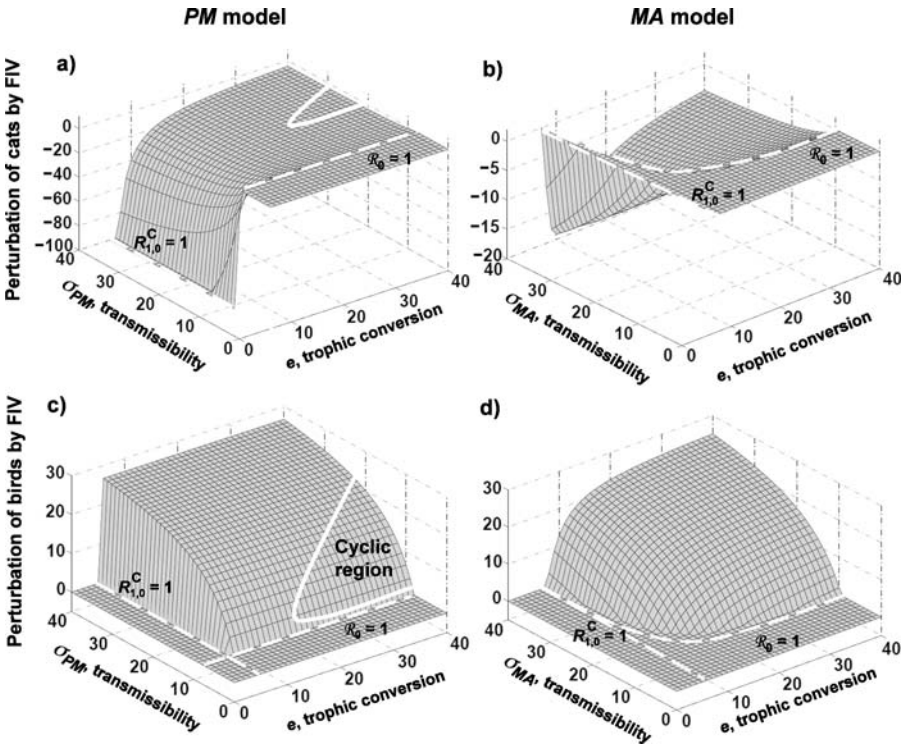

Fig. 4 Impact of FIV introduction on cats and birds, plotted over parameter ranges that can be considered biologically plausible. The perturbation is defined as the percentual change of the mean population values before and after virus release. An impact of -100% corresponds to extinction. The solid white lines in panels (a) and (c) separate parameter regions with cyclic dynamics. Parameter values: $m = 6$ and $\alpha = 2$.

endangered prey to recover. The gain of the bird population after FIV introduction increases with transmissibility (Fig. 4c). Trophic conversion efficiency does not affect the disease impact on the birds. Instabilities in form of population cycles have no effect on the average impact of pathogen release. This is because the mean population sizes during an oscillatory period is equal to the population size of the unstable stationary state (cf. Appendix B).

In the MA model, cats eradication is not possible, because disease transmission vanishes for small host population sizes. The impact on cats therefore increases with transmissibility values (Fig. 4b). Even very small trophic conversion efficiencies are not dangerous for the cats in the MA model, because the disease-related deaths remain limited in small host population sizes. The recovery of the bird population after FIV introduction increases with both transmissibility and trophic conversion efficiency (Fig. 4d). A larger conversion efficiency of the predators means that the cats have a larger population in which the disease can spread more quickly. In a biologically plausible parameter range, MA transmission does not lead to population oscillation (although they are possible in principle as we showed in the previous section).

6. Conclusions and discussion

We have presented a theoretical study of the potential effects of introducing a retrovirus into an ecosystem for biological control purposes. In particular, the effects of releasing Feline Immunodeficiency Virus (FIV) in a cat population that preys exclusively on island bird species have been addressed. Our results suggest that FIV has the potential to depress feral cats by lowering their population equilibrium level. For both transmission modes, the reduction in cat population size due to disease introduction gives endangered bird species a chance to recover. The models thus reveal that pathogens can be used as bio-control agents not only for their host, but also for other trophic levels. This is in accordance with previous work demonstrating that infectious diseases have regulatory effects also on other species their host interacts with (e.g. Anderson and May, 1986).

The regulatory competence of FIV, however, depends strongly on ecological details within the system considered. The social and spatial organisation structure of cat populations is highly variable as is the population dynamics (Berthier et al., 2000; Liberg et al., 2000). Therefore, we considered two models of virus spread that represent the classic extremes of disease transmission, proportionate mixing (PM model), and mass action (MA model).

If FIV is transmitted by mass action, the disease cannot lead to host extinction. This is because pathogen transmission vanishes for small host population sizes (e.g. de Castro and Bolker, 2005), provided that the host does not experience an Allee effect which could drive the population extinct in a joint interplay with the disease (Hilker et al., 2009). Depressing the population size of cats is possible—but not their eradication. If the objective of a control programme is host eradication and if transmission is of mass action type, disease introduction alone is not to be recommended. Other methods, such as trapping and culling, despite their already mentioned disadvantages, might have more success.

With proportionate mixing, pathogen transmission is ongoing even in vanishing populations. Disease-induced extinction can occur under certain conditions, e.g. if the transmissibility is sufficiently large (Fig. 2a) and the trophic conversion efficiency of the host sufficiently low (Fig. 4a). Specific parameter estimates for the conversion efficiency are lacking, but such extreme values of both conversion efficiency and transmissibility appear to be unrealistic (cf. Section 5). Although disease-induced eradication therefore seems to be unlikely for proportionate mixing, FIV might prove an effective agent for long-term control with moderate population reduction. Given the low impact FIV is thought to have on cats (Courchamp et al., 1995), this is a plausible result.

In general, we can distinguish the following scenarios, which are showcased in Fig. 5:

1. disease-induced extinction of the predators (Fig. 5a)
2. long-term control of the predators (Fig. 5b)
3. system destabilisation, meaning that disease introduction induces limit cycle oscillations (Fig. 5c)

Note that all three outcomes imply a recovery of the bird population, but the actual extent depends on the parameter values. According to the ecological context of sub-Antarctic Islands, scenarios (b) and (c) appear to be realistic for the PM model. For the MA model, scenario (b) is likely to occur since (a) is impossible and (c) unrealistic.

Scenarios (a) and (b) are similar to the conclusions from an earlier, but ill-posed model (Courchamp and Sugihara, 1999). The destabilisation of community dynamics

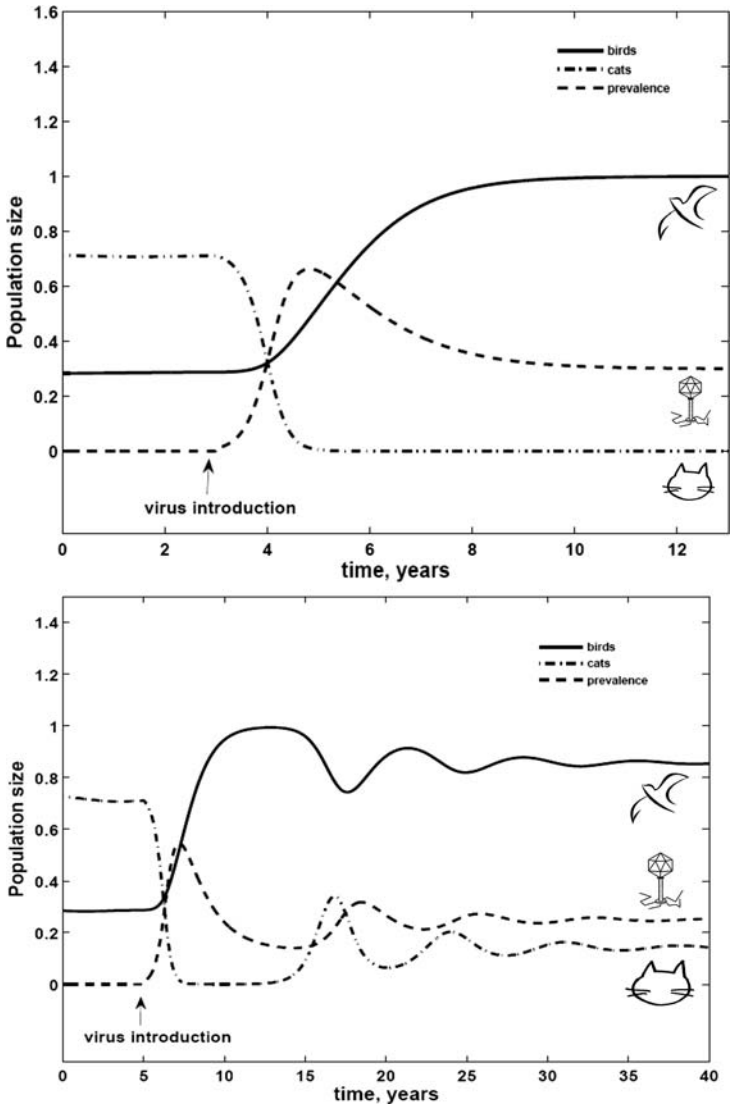

Fig. 5 Potential outcomes of FIV introduction into a birds–cats food chain. (a) Disease-induced extinction of the predators; (b) long-term depression of the host: FIV introduction is not able to eradicate cats, but reduces their population significantly; (c) the disease destabilises the system, inducing limit cycle oscillations. According to the ecological context of sub-Antarctic islands, scenarios (b) and (c) are possible for the PM model and only scenario (b) seems likely for the MA model. All three time-plots are obtained from the PM model with the following parameter values: $e = 3.5, m = 1, \alpha = 8$ and (a) $\sigma_{\mathrm{PM}} = 13$, (b) $\sigma_{\mathrm{PM}} = 12$ and (c) $\sigma_{\mathrm{PM}} = 10$. Note that time on the horizontal axis is dimensional in this plot.

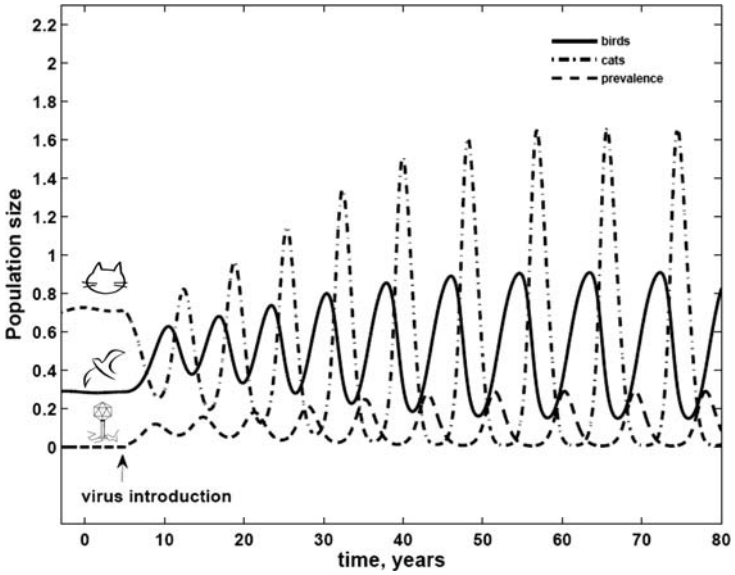

Fig. 5 (*Continued.*)

(c) is a potential effect of FIV introduction that has not been addressed before and that we shall discuss in the remainder in some detail. Regular limit cycle oscillations may appear under both types of virus transmission. The underlying predator–prey model of Lotka–Volterra type, however, does not allow for population cycles (e.g. Kot, 2001). The sustained fluctuations can therefore be clearly attributed to the introduction of disease agents. The emergence of cycles in simple predator–prey models of Lotka–Volterra type with infected predators has been reported before (e.g. Anderson and May, 1986). Such eco-epidemiological models have some structural similarity to intra-guild predation models, for which dynamic instabilities leading to oscillations and chaos have been observed as well—even when population interactions are bi-linear (Holt and Polis, 1997; Tanabe and Namba, 2005). One could think of infected predators as top-predators "consuming" both susceptible predators and prey. However, the infected predators reproduce into the susceptible compartment as there is no vertical transmission of FIV. Hence, the analogy of our models with intra-guild predation food webs is limited.

In the disease-free system, there is a unique and stable equilibrium of prey and predators. Consider a perturbation of this equilibrium in the form that the prey population is increased. Then the consumption rate of predators increases linearly, thus reducing the prey and leading to an approach of the equilibrium in damped oscillations. In the presence of disease, however, susceptible predators become infected and, moreover, suffer from an additional disease-related death rate. Infected predators continue to consume prey with the same rate as susceptible predators, but the disease transmission and mortality induce a kind of time lag, which allows the prey to continue growing. Only when the prey has become abundant in large numbers, the predation rates increase sufficiently to rebound the predators and depress the prey. Next, when the prey population becomes too small,

the predators decline and suffer again from additional disease mortality, which allows the prey to take off and start the cycle again.

Interestingly, disease-induced destabilisation is possible for both transmission modes. Since mass action transmission depends on host population size, one can imagine that it might be more prone to fluctuations than proportionate mixing. In fact, MA transmission admits oscillations in a simple SI model with Allee effect (Hilker et al., 2009), whereas PM transmission in the same model does not exhibit fluctuations (Hilker et al., 2007). PM could have therefore be seen as more stabilising.

The key ingredient to induce oscillations appears to be disease-related host mortality, for both PM and MA transmission. The onset of cycles occurs when the disease burden is sufficiently large, which requires large enough transmissibilities, for example. This explains the first (forward) Hopf bifurcation point in Fig. 2. The oscillations disappear again for further increasing transmissibilities in a second (backward) Hopf bifurcation. If disease burden is too large, the predator population is reduced to such a level that the density-dependence felt by the prey becomes strong. This is a well-known stabilising mechanism and has been identified to stabilise endogenous predator–prey cycles as well (Hilker and Schmitz, 2008).

The interaction of the two (forward and backward) Hopf bifurcations leads to a "bubbling" phenomenon: oscillations appear and their amplitudes increase in extent until they reach some maxima and decrease before the cycles eventually disappear (cf. Fig. 2). A similar bubbling has been observed in few models only (Xiao and Van Den Bosch, 2003; Fenton and Rands, 2006). Our models appear to be structurally simpler and allow to identify and explain the underlying mechanisms.

The possibility of population oscillations is very interesting from a biological control point of view. When populations cycle, they periodically undergo peaks and troughs. This can be a double-edged sword for management measures. On the one hand, if bird populations become very small in such troughs, they become even more prone to extinction than they were at equilibrium. This is a negative aspect of the cyclic regime and why it should be avoided. On the other hand, there could also be a positive aspect if the cat population becomes very small. In this case, successful eradication seems more likely as only a much smaller population size would need to be removed. This could be of advantage in the planning of control actions, for example in combination with culling. These considerations are especially relevant for drastic oscillations with extreme amplitudes (e.g., Fig. 2a). Taking into account stochastic effects, which are abundant in nature, one can expect that the species is driven to extinction.

In summary, before any control attempt through the introduction of a pathogen is made, the relevant eco-epidemiological parameters involved should be investigated. Our models confirm that the impact of a parasite may vary widely according to host population characteristics (Grenfell and Dobson, 1995), thus stressing the need of detailed field studies before engaging into large-scale control programmes. Any such action must require a careful preliminary study before a pathogen is introduced into an ecosystem; otherwise, the consequences of such measure can be devastating. In particular, host–parasite interactions can be substantially different in their organisational structure and dynamics; a single model may therefore be insufficient to explore the potential impacts of bio-control agents. Mathematical models can only be one source of guidance for biological control programmes. Other sources of information will be crucial and are required for more refined parameterisations and validations of the models developed. They include, amongst

others: the natural history; the natural enemy's suitability to local climate and environment; the phenotypes of pest and enemy; their habitat requirements; and the danger of attack by the enemy on non-target species. The continual testing of models with data in different circumstances seems to be a promising strategy to approach a solution which is as good as possible (Le Corre, 2008).

The possibility of introducing pathogens in an ecosystem for biological conservation purposes implies potentially killing animals. One should also keep in mind that these animals have become part of the ecosystem as well and fulfill certain roles (e.g., cats preying on rodents). Even though our paper is theoretical in nature, both ethical concerns and philosophical questions demand caution before putting theory into practice.

Acknowledgements

Alessandro Margheri pointed out the relation described in Appendix B, which is due to him. He also provided valuable comments and suggestions, as did Manuel Gomes. Ricardo Águas is gratefully acknowledged for his support in computational issues. The authors are thankful for fruitful discussions with the research groups of Mark Lewis (Edmonton, Alberta) and Gabriela Gomes (Oeiras/Lisbon). NMO was funded by IGC/PFE-EA-UE and supported in part by the European Commission, grant MEXT-CT-2004-14338 to Gabriela Gomes, and the Centre for Mathematical Biology at the University of Alberta. FMH is an Alberta Ingenuity, Honorary Killam and Ciência 2007 fellow.

Appendix A: Linear stability analysis

This Appendix outlines the linear stability analysis of the PM and MA models. Conditions for the existence and local stability of equilibria will be presented as far as possible. The Jacobian of model system (7)–(9) is

$$\begin{pmatrix} 1-2B-C & -B & 0 \\ eC & eB-m-\alpha i & -\alpha C \\ -ei & (1-i)i\,\frac{\partial}{\partial C}\Gamma(C) & (\Gamma(C)-\alpha)(1-2i)-eB \end{pmatrix}.$$

A.1 PM model

System (7)–(9) with PM transmission has six possible equilibria: $\boldsymbol{E}_0$, $\boldsymbol{E}_i$, $\boldsymbol{E}_B$, $\boldsymbol{E}_{BC}$, $\boldsymbol{E}_{Bi}$ and $\boldsymbol{E}_{BCi}$, where the indices indicate which state variables are non–zero in the equilibrium. The stability conditions are as follows:

1. $\boldsymbol{E}_0 = (0, 0, 0)$. The trivial extinction state is always a saddle point, which means it is always unstable. The eigenvalues are

$$\lambda_1 = 1 > 0,$$
$$\lambda_2 = -m < 0,$$
$$\lambda_3 = \sigma_{\mathrm{PM}} - \alpha.$$

2. $\boldsymbol{E}_i = (0, 0, 1)$. The trivial extinction state due to disease is always a saddle point, which means it is always unstable. The eigenvalues are

$$\lambda_1 = 1 > 0,$$
$$\lambda_2 = -m - \alpha < 0,$$
$$\lambda_3 = -(\sigma_{\mathrm{PM}} - \alpha).$$

3. $\boldsymbol{E}_B = (1, 0, 0)$. The disease-free prey-only state is stable if the predators cannot establish ($R^C_{1,0} < 1$) and decay faster than the disease at small population size ($\mathcal{R}_i < 1$). The eigenvalues are

$$\lambda_1 = -1 < 0,$$
$$\lambda_2 = e - m \lessgtr 0 \quad \text{if } R^C_{1,0} \lessgtr 1,$$
$$\lambda_3 = \sigma_{\mathrm{PM}} - \alpha - e \lessgtr 0 \quad \text{if } \mathcal{R}_i \lessgtr 1.$$

4. $\boldsymbol{E}_{BC} = (B^*_4, C^*_4, 0)$ with $B^*_4 = \frac{m}{e}$ and $C^*_4 = \frac{e-m}{e}$. The predator–prey coexistence state in the disease-free subsystem exists if $R^C_{1,0} > 1$ and it is stable if $\mathcal{R}_0 < 1$. The eigenvalues are

$$\lambda_1 = \sigma_{\mathrm{PM}} - \alpha - m \lessgtr 0 \quad \text{if } \mathcal{R}_0 \lessgtr 1,$$
$$\lambda_{2,3} = \frac{1}{2e}\Big(-m \pm \sqrt{m\big(m + 4e[m - e]\big)}\Big) \quad \text{with Re } \lambda_{2,3} < 0.$$

5. $\boldsymbol{E}_{BCi} = (B^*_5, C^*_5, i^*_5)$ with $B^*_5 = \frac{(\sigma_{\mathrm{PM}}-\alpha)(m+\alpha)}{e\sigma_{\mathrm{PM}}}$, $C^*_5 = \frac{e\sigma_{\mathrm{PM}}-(\sigma_{\mathrm{PM}}-\alpha)(m+\alpha)}{e\sigma_{\mathrm{PM}}}$ and $i^*_5 = \frac{\sigma_{\mathrm{PM}}-\alpha-m}{\sigma_{\mathrm{PM}}}$. The non-trivial, endemic coexistence equilibrium exists if $\mathcal{R}_0 > 1$ and $R^C_{1,i} > 1$. The eigenvalues are too lengthy to present, but one can implicitly show that $\boldsymbol{E}_{BCi}$ is stable if

$$\sigma_{\mathrm{PM}} > \alpha i^*_5 - \frac{1}{2}\Big(B^*_5 - \sqrt{B^{*2}_5 + 4e\alpha C^*_5 i^*_5 - 4eB^*_5 C^*_5}\Big) \quad \text{and}$$
$$\sigma_{\mathrm{PM}} < \alpha i^*_5 - \frac{1}{2}\Big(B^*_5 + \sqrt{B^{*2}_5 + 4e\alpha C^*_5 i^*_5 - 4eB^*_5 C^*_5}\Big).$$

If these conditions are not fulfilled, $\boldsymbol{E}_{BCi}$ is unstable and numerical simulations reveal the existence of stable limit cycles.

6. $\boldsymbol{E}_{Bi} = (1, 0, i^*_6)$ with $i^*_6 = \frac{\sigma_{\mathrm{PM}}-\alpha-e}{\sigma_{\mathrm{PM}}-\alpha}$. This equilibrium exists if $\mathcal{R}_i > 1$ and is stable if $R^C_{1,i} < 1$. The eigenvalues are

$$\lambda_1 = -1 < 0,$$
$$\lambda_2 = \frac{e\sigma_{\mathrm{PM}}}{\sigma_{\mathrm{PM}} - \alpha} - m - \alpha \lessgtr 0 \quad \text{if } R^C_{1,i} \lessgtr 1,$$
$$\lambda_3 = -(\sigma_{\mathrm{PM}} - \alpha - e) \lessgtr 0 \quad \text{if } \mathcal{R}_i \gtrless 1.$$

There is one special case that occurs only for the particular parameter combination $\sigma_{\mathrm{PM}} = \alpha$. In this case, $\boldsymbol{E}_7 = (0, 0, i^*_7)$ with $i^*_7 \in (0, 1)$ is a continuum of stationary

states. These equilibria are unstable saddle points; their eigenvalues are $\lambda_1 = 1 > 0$, $\lambda_2 = -m - \alpha i_7^* < 0$, $\lambda_3 = (\sigma_{\mathrm{PM}} - \alpha)(1 - 2i_7^*)$. As the parameter condition is unlikely to be met exactly in nature, this special case is not considered in the main text.

A.2 MA model

System (7)–(9) with MA transmission has five possible equilibria: $\boldsymbol{E}_0$, $\boldsymbol{E}_i$, $\boldsymbol{E}_B$, $\boldsymbol{E}_{BC}$ and $\boldsymbol{E}_{BCi}$. The equilibrium corresponding to cat eradication with positive prevalence and bird persistence does not exist. The stability conditions are as follows:

1. $\boldsymbol{E}_0 = (0, 0, 0)$. The trivial extinction state is always a saddle point, which means it is always unstable. The eigenvalues are

$$\lambda_1 = 1 > 0,$$
$$\lambda_2 = -m < 0,$$
$$\lambda_3 = -\alpha < 0.$$

2. $\boldsymbol{E}_i = (0, 0, 1)$. This trivial disease-induced extinction state is always a saddle point, which means it is always unstable. The eigenvalues are

$$\lambda_1 = 1 > 0,$$
$$\lambda_2 = -m - \alpha < 0,$$
$$\lambda_3 = \alpha > 0.$$

3. $\boldsymbol{E}_B = (1, 0, 0)$. The disease-free prey-only state is stable if the predators are too weak to establish ($R_{1,0}^C < 1$). The eigenvalues are

$$\lambda_1 = -1 < 0,$$
$$\lambda_2 = e - m \lessgtr 0 \quad \text{if } R_{1,0}^C \lessgtr 1,$$
$$\lambda_3 = -e - \alpha < 0.$$

4. $\boldsymbol{E}_{BC} = (B_4^*, C_4^*, 0)$ with $B_4^* = \frac{m}{e}$ and $C_4^* = \frac{e-m}{e}$. The predator–prey coexistence state in the disease-free subsystem exists if $R_{1,0}^C > 1$ and it is stable if $\mathcal{R}_0 < 1$. The eigenvalues are

$$\lambda_1 = \sigma_{\mathrm{MA}} - \alpha - \frac{m(\sigma_{\mathrm{MA}} + e)}{e} \lessgtr 0 \quad \text{if } \mathcal{R}_0 \lessgtr 1,$$
$$\lambda_{2,3} = \frac{1}{2e}\left(-m \pm \sqrt{m^2 - 4em(e-m)}\right) \quad \text{with Re } \lambda_{2,3} < 0.$$

5. $\boldsymbol{E}_{BCi} = (B_5^*, C_5^*, i_5^*)$ with B_5^*, C_5^* and i_5^* being long and cumbersome expressions; they can be easily obtained by a computer algebra system and it can be checked that they are biologically feasible. The reasoning in Section 4 makes clear that $\boldsymbol{E}_{BCi}$ cannot exist if $\mathcal{R}_0 < 1$ and $R_{1,i}^C < 1$. The stability of the nontrivial equilibrium has been investigated by means of continuation analysis and numerical simulations. $\boldsymbol{E}_{BCi}$ can be stable or unstable. In the latter case, stable limit cycles exist.

Appendix B: Mean population sizes in cyclic regimes

We demonstrate analytically that, in the PM model, the average value $\overline{x}$ of a state variable in a cycle period T is equal to the value x^* of the corresponding unstable equilibrium.

Consider the first equation (7). Dividing by B yields

$$\frac{B'}{B} = 1 - B - C,$$

where the prime denotes the derivative with respect to time. Integrating in $[0, T]$ and taking into account that $B(0) = B(T)$, we get

$$\int_0^T \frac{B'(t)}{B(t)}\,\mathrm{d}t = \ln B(T) - \ln B(0) = 0 = \int_0^T 1\,\mathrm{d}t - \int_0^T B(t)\,\mathrm{d}t - \int_0^T C(t)\,\mathrm{d}t.$$

Dividing by T and recalling that $\overline{x} = \frac{1}{T}\int_0^T x(t)\,\mathrm{d}t$, we finally obtain:

$$0 = 1 - \overline{B} - \overline{C}.$$

Proceeding in a similar way with the other Eqs. (8) and (9), we see that the averages satisfy the entire system of differential equations and therefore correspond to the non-trivial equilibria.

References

Anderson, R.M., 1982. Theoretical basis for the use of pathogens as biological control agents of pest species. Parasitology 84, 3–33.

Anderson, R.M., May, R.M., 1981. The population dynamics of microparasites and their invertebrate hosts. Philos. Trans. R. Soc. Lond. B 291, 451–524.

Anderson, R.M., May, R.M., 1986. The invasion, persistence and spread of infectious diseases within animal and plant communities. Philos. Trans. R. Soc. Lond. B 314, 533–570.

Atkinson, I.A.E., 1988. Opportunities for ecological restoration. N.Z. J. Ecol. 11, 1–12.

Atkinson, I.A.E., 1989. Introduced animals and extinction. In: Western, D., Pearl, M.C. (Eds.), Conservation for the Twenty-First Century, pp. 54–75. Oxford University Press, Oxford.

Auger, P., Mchich, R., Chowdhury, T., Sallet, G., Tchuente, M., Chattopadhyay, J., 2009. Effects of a disease affecting a predator on the dynamics of a predator–prey system. J. Theor. Biol. doi:10.1016/j.jtbi.2008.10.030.

Bax, N., Carlton, J.T., Mathews-Amos, A., Haedrich, R.L., Howarth, F.G., Purcell, J.E., Rieser, A., Gray, A., 2001. The control of biological invasions in the world's oceans. Conserv. Biol. 15, 1234–1246.

Begon, M., Bennett, M., Bowers, R.G., French, N.P., Hazel, S.M., Turner, J., 2002. A clarification of transmission terms in host-microparasite models: numbers, densities and areas. Epidemiol. Infect. 129, 147–153.

Berruti, A., Griffiths, A.M., Imber, M.J., Schramm, M., Sinclair, J.C., 2000. Status of seabirds at Prince Edward Island. S. Afr. J. Antarct. Res. 10, 32–33.

Berthier, K., Langlais, M., Auger, P., Pontier, D., 2000. Dynamics of a feline virus with two transmission modes within exponentially growing host populations. Proc. R. Soc. Lond. B 267, 2049–2056.

Bester, M.N., Bloomer, J.P., van Aarde, R.J., Erasmus, B.H., van Rensburg, P.J.J., Skinner, J.D., Howell, P.G., Naude, T.W., 2002. A review of the successful eradication of feral cats from sub-Antarctic Marion Island, Southern Indian Ocean. S. Afr. J. Wildl. Res. 32, 65–73.

Brooke, R.K., Cooper, J., Hockey, P.A.R., Ryan, P.G., Sinclair, J.C., Suter, W., Tree, A.J., 1988. Distribution, population size and conservation of the Antarctic tern *Sterna vittata* in southern Africa. Cormorant 16, 107–113.

Chaphuis, J.L., Boussès, P., Barnaud, G., 1994. Alien mammals, impact and management in the French Subantarctic Islands. Biol. Conserv. 67, 97–104.
Chornesky, E.A., Randal, J.M., 2003. The threat of invasive alien species to biological diversity: setting a future course. Ann. M. Bot. Gard. 90, 67–76.
Cleaveland, S., Thirgood, S., Laurenson, K., 1999. Pathogens as allies in island conservation? Trends Ecol. Evol. 14, 83–84.
Cooper, J., Brown, C.R., 1990. Ornithological research at the sub-Antarctic Prince Edward Islands: a review of achievements. S. Afr. J. Antarct. Res. 20, 40–57.
Cooper, J.A., Marais, A.V.N., Bloomer, J.P., Bester, M.N., 1995. A success story: breeding of burrowing petrels (Procellariidae) before and after the eradication of feral cats Felis catus at subantarctic Marion Island. Mar. Ornithol. 23, 33–37.
Courchamp, F., Sugihara, G., 1999. Modeling the biological control of an alien predator to protect island species from extinction. Ecol. Appl. 9, 112–123.
Courchamp, F., Pontier, D., Langlais, M., Artois, M., 1995. Population dynamics of Feline Immunodeficiency Virus within cat populations. J. Theor. Biol. 175, 553–560.
Courchamp, F., Yoccoz, N.G., Artois, M., Pontier, D., 1998. At-risk individuals in Feline Immunodeficiency Virus epidemiology: Evidence from a multivariate approach in a natural population of domestic cats (Felis catus). Epidemiol. Infect. 121, 227–236.
Courchamp, F., Say, L., Pontier, D., 2000. Transmission of Feline Immunodeficiency Virus in a population of cats (Felis catus). Wildl. Res. 27, 603–611.
de Castro, F., Bolker, B., 2005. Mechanisms of disease-induced extinction. Ecol. Lett. 8, 117–126.
Didham, R.K., Tylianakis, J.M., Hutchinson, M.A., Ewers, R.M., Gemmell, N.J., 2005. Are invasive species the drivers of ecological change? Trends Ecol. Evol. 20, 470–474.
Dobson, A.P., 1988. Restoring island ecosystems: the potential of parasites to control introduced mammals. Conserv. Biol. 2, 31–39.
Fan, M., Kuang, Y., Feng, Z., 2005. Cats protecting birds revisited. Bull. Math. Biol. 67, 1081–1106.
Fenton, A., Rands, S.A., 2006. The impact of parasite manipulation and predator foraging behavior on predator-prey communities. Ecology 87, 2832–2841.
Fromont, E., Pontier, D., Langlais, M., 1998. Dynamics of a feline retrovirus (FeLV) in host populations with variable spatial structure. Proc. R. Soc. Lond. B 265, 1097–1104.
Ginzburg, L.R., 1998. Assuming reproduction to be a function of consumption raises doubts about some popular predator–prey models. J. Anim. Ecol. 67, 325–327.
Granadeiro, J.P., Dias, M.P., Rebelo, R., Santos, C.D., Catry, P., 2006. Numbers and population trends of Cory's Shearwater Calonectris diomedea at Selvagem Grande, Northeast Atlantic. Waterbirds 29, 56–60.
Grenfell, B.T., Dobson, A.P. (Eds.), 1995. Ecology of Infectious Diseases in Natural Populations. Cambridge University Press, Cambridge.
Gurevitch, J., Padilla, D.K., 2004. Are invasive species a major cause of extinctions? Trends Ecol. Evol. 19, 470–474.
Han, L., Ma, Z., Hethcote, H.W., 2001. Four predator prey models with infectious diseases. Math. Comput. Model. 34, 849–858.
Haque, M., Venturino, E., 2007. An ecoepidemiological model with disease in predator: the ratio-dependent case. Math. Methods Appl. Sci. 30, 1791–1809.
Hatcher, M.J., Dick, J.T.A., Dunn, A.M., 2006. How parasites affect interactions between competitors and predators. Ecol. Lett. 9, 1253–1271.
Hilker, F.M., Schmitz, K., 2008. Disease-induced stabilization of predator-prey oscillations. J. Theor. Biol. 255, 299–306.
Hilker, F.M., Langlais, M., Petrovskii, S.V., Malchow, H., 2007. A diffusive SI model with Allee effect and application to FIV. Math. Biosci. 206, 61–80.
Hilker, F.M., Langlais, M., Malchow, H., 2009. The Allee effect and infectious diseases: extinction, multistability, and the (dis-)appearance of oscillations. Am. Nat. 173, 72–88.
Holt, R.D., Polis, G.A., 1997. A theoretical framework for intraguild predation. Am. Nat. 149, 745–764.
Kot, M., 2001. Elements of Mathematical Ecology. Cambridge University Press, Cambridge.
Krajick, K., 2005. Winning the war against island invaders. Science 310, 1410–1413.
Le Corre, M., 2008. Cats, rats and seabirds. Nature 451, 134–135.
Liberg, O., Sandell, M., Pontier, D., Natoli, E., 2000. Density, spatial organisation and reproductive tactics in the domestic cat and other felids. In: Turner, D.C., Bateson, P. (Eds.), The Domestic Cat: The Biology of Its Behaviour, 2nd edn., pp. 119–147. Cambridge University Press, Cambridge.

Lockwood, J.L., Hoopes, M.F., Marchetti, M., 2007. Invasion Ecology. Blackwell Publishing, Oxford.
Mack, R.N., Simberloff, D., Lonsdale, W.M., Evans, H., Clout, M., Bazzaz, F.A., 2000. Biotic invasions: causes, epidemiology, global consequences, and control. Ecol. Appl. 10, 689–710.
May, R.M., Hassell, M.P., 1988. Population dynamics and biological control. Philos. Trans. R. Soc. Lond. B 318, 129–169.
McCallum, H., Barlow, N., Hone, J., 2001. How should pathogen transmission be modelled? Trends Ecol. Evol. 16, 295–300.
Moors, P.J., Atkinson, I.A.E., 1984. Predation on seabirds by introduced animals, and factors affecting its severity. In: Croxall, P.J., Evans, P.G.H., Schreiber, R.W. (Eds.), Status and Conservation of the World's Seabirds, vol. 2, pp. 667–690. ICBP Technical Publications, Cambridge.
Murdoch, W.W., Briggs, C.J., 1996. Theory for biological control: recent developments. Ecology 77, 2001–2013.
Nogales, M., Martín, A., Tershy, B.R., Josh Donlan, C., Veitch, D., Puerta, N., Wood, B., Alonso, J., 2004. A review of feral cat eradication on islands. Conserv. Biol. 18, 310–319.
Pimm, S.L., 1982. Food Webs. Chapman and Hall, London.
Rosenzweig, M.L., 1969. Why the prey curve has a hump. Am. Nat. 103, 81–87.
Rounsevell, D.E., Copson, G.R., 1982. Growth rate and recovery of a king penguin, Aptenodytes patagonicus, population after exploitation. Aust. Wildl. Res. 9, 519–525.
Ryan, P.G., 1987. The distribution, population size and foraging behaviour of Kerguelen terns at the Prince Edwards Islands. S. Afr. J. Antarct. Res. 17(2), 163–166.
Sæther, B.E., Engen, S., 2002. Pattern of variation in avian population growth rates. Philos. Trans. R. Soc. Lond. B 357, 1185–1195.
Sax, D.F., Brown, J.H., 2000. The paradox of invasion. Glob. Ecol. Biogeogr. 9, 363–371.
Say, L., Gaillard, J.M., Pontier, D., 2002. Spatio-temporal variation in cat population density in a Sub-Antarctic environment. Polar Biol. 25, 90–95.
Schramm, M., 1986. Burrow densities and nest site preferences of petrels (Procellariidae) at the Prince Edward Islands. Polar Biol. 6, 63–70.
Siegfried, W.R., 1978. Ornithological research at the Prince Edward islands: a review of progress. S. Afr. Antarktis Nave 8, 30–34.
Strong, D.R., Pemberton, R.W., 2000. Biological control of invading species—risk and reform. Science 288, 1969–1970.
Tanabe, K., Namba, T., 2005. Omnivory creates chaos in simple food web models. Ecology 86, 3411–3414.
Turchin, P., 2003. Complex Population Dynamics. A Theoretical/Empirical Synthesis. Princeton University Press, Princeton.
Van Aarde, R.J., 1979. Distribution and density of the feral house cat Felis catus at Marion Island. S. Afr. J. Antarct. Res. 9, 14–19.
Van Aarde, R.J., 1980. The diet and feeding behaviour of feral cats, Felis catus at Marion Island. S. Afr. J. Wildl. Res. 10(3/4), 123–128.
Van Aarde, R.J., 1983. Demographic parameters of the feral cat Felis catus population at Marion Island. S. Afr. J. Wildl. Res. 13(1), 12–16.
Van Rensburg, P.J.J., Bester, M.N., 1988. The effect of cat Felis catus predation on three breeding Procellariidae species on Marion Island. S. Afr. J. Zool. 23(4), 301–305.
Van Rensburg, P.J.J., Skinner, J.D., Van Aarde, R.J., 1987. Effects of feline panleucopenia on the population characteristics of feral cats on Marion Island. J. Appl. Ecol. 24, 63–73.
Venturino, E., 1994. The influence of diseases on Lotka-Volterra systems. Rocky M. J. Math. 24(1), 381–402.
Venturino, E., 2002. Epidemics in predator-prey models: disease in the predators. IMA J. Math. Appl. Med. Biol. 19, 185–205.
Volterra, V., 1931. Leçons sur la Théorie Mathématique de la Lutte pour la Vie. Gauthier-Villars, Paris.
Waage, J.K., Greathead, D.J., 1988. Biological control: challenges and opportunities. Philos. Trans. R. Soc. Lond. B 318, 111–128.
Wilcox, C., Donlan, C.J., 2007. Compensatory mitigation as a solution to fisheries bycatch-biodiversity conservation conflicts. Front. Ecol. Environ. 5, 325–331.
Williams, A.J., Burger, A.E., Berruti, A., Siegfried, W.R., 1975. Ornithological research on Marion Island 1974–1975. S. Afr. Antarktis Nave 5, 48–50.

Williams, A.J., Siegfried, W.R., Burger, A.E., Berruti, A., 1979. The Prince Edward Islands: a sanctuary for seabirds in the Southern Ocean. Biol. Conserv. 15, 59–71.
Xiao, Y., Van Den Bosch, F., 2003. The dynamics of an eco-epidemic model with biological control. Ecol. Model. 168, 203–214.
Zavaleta, E.S., Hobbs, R.J., Mooney, H.A., 2001. Viewing invasive species removal in a whole-ecosystem context. Trends Ecol. Evol. 16, 454–459.