Bull Math Biol (2017) 79:88–116
DOI 10.1007/s11538-016-0228-3

Society for Mathematical Biology

ORIGINAL ARTICLE

Optimal Culling and Biocontrol in a Predator–Prey Model

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Received: 17 July 2016 / Accepted: 18 October 2016 / Published online: 31 October 2016

Abstract Invasive species cause enormous problems in ecosystems around the world. Motivated by introduced feral cats that prey on bird populations and threaten to drive them extinct on remote oceanic islands, we formulate and analyze optimal control problems. Their novelty is that they involve both scalar and time-dependent controls. They represent different forms of control, namely the initial release of infected predators on the one hand and culling as well as trapping, infecting, and returning predators on the other hand. Combinations of different control methods have been proposed to complement their respective strengths in reducing predator numbers and thus protecting endangered prey. Here, we formulate and analyze an eco-epidemiological model, provide analytical results on the optimal control problem, and use a forward–backward sweep method for numerical simulations. By taking into account different ecological scenarios, initial conditions, and control durations, our model allows to gain insight how the different methods interact and in which cases they could be effective.

Keywords Eco-epidemiology · Biocontrol · Optimal control · Invasive pest · Predator–prey

Mathematics Subject Classification 34D20 · 49K15 · 92D30 · 92D40

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1 Introduction

In USA, cats are the most popular companion animal with more than 80 million individuals living in peoples' homes. The number of feral cats is unknown but estimated to range from 60 to 80 million (Loyd and Miller 2010). The feral domestic cat (*Felis catus*) is an opportunistic predator, eating what is most easily available, switching prey according to their relative spatial and temporal availability (Fitzgerald and Turner 2000). Among the most notorious and harmful introduced predators are feral cats. Cats have often been introduced on islands in attempts to control rats, which get to the shore from hitching a ride from sealing or whaling boats or from shipwrecks (Moors and Atkinson 1984). Feral cats are predatory invasive species with negative effect on wildlife and pose significant threat to tree- and ground-nesting birds, herpetofauna, and small mammals they prey upon (Loyd and Miller 2010). The native prey attacked by invasive cats often lack evolved anti-predation mechanisms, e.g., seabirds, which have to return to land to raise their young, after nesting on islands (Moors and Atkinson 1984; Diamond 1989). On remote oceanic islands, introduced feral cats pose particularly devastating threats on the native fauna. For example, five cats introduced on Marion island in 1949 resulted in a population of more than 2000 cats some 25 years later, depleting some 500,000 common diving petrels and severely affecting hole-nesting petrels (van Aarde 1980; van Rensburg and Bester 1988). At this same time, five cats introduced on the Kerguelen islands grew to several tens of thousands and are estimated to kill more than three million seabirds every year (Chaphuis 1995).

In an attempt to conserve the population of native birds, the eradication of invasive cat populations on islands has been the goal of many control programs (Courchamp et al. 2003; Robertson 2008; Lavers et al. 2010). While there are a number of successful eradications, the majority of them took place on small islands (Nogales et al. 2004). Eradication remains notoriously difficult, time-consuming, and costly—especially on remote and larger islands. Actually, eradication is not always the goal of management programs, because cats have become part of the ecosystem and their extirpation may lead to undesirable consequences such as the mesopredator release effect (Courchamp et al. 1999; Rayner et al. 2007; Russell et al. 2009).

In this paper, we use optimal control theory to find optimal control programs that (1) maximize the bird population size, while minimizing (2) the cat population size and (3) the intervention costs. To our knowledge, this is a novel application of optimal control theory in endangered species protection.

The most common methods of controlling feral cats on islands are trapping and hunting, i.e., "mechanical" forms of control, followed by poisoning (see Nogales et al. 2004). There are also two cases of releasing viral diseases as biocontrol agents that led to successful cat eradications on sub-Antarctic Marion Island (Bester et al. 2002) and central-Pacific Jarvis Island (Rauzon 1985). The combination of different control methods has been identified as an "important management consideration" (Dobson 1988, p. 35) and suggested as the best way to deal with mammalian introductions (Courchamp et al. 2003). In practice, however, different forms of control often occur in separate, consecutive phases (e.g., Bester et al. 2002). In the theoretical literature, the joint interplay of combined control methods has been rarely investigated.

However, using a mathematical model of ordinary differential equations, Courchamp and Sugihara (1999, p. 121) suggest that culling is "more efficient when held during simultaneous virus introduction."

Here, we consider the combined use of different intervention methods, namely mechanical control (culling) and biological control (infectious diseases). Culling can be in the form of trapping or hunting and is modeled by an additional time-dependent mortality rate of the cat population. For the biological control, we consider the feline immunodeficiency virus (FIV), which has been suggested and studied for controlling cats on oceanic islands (Courchamp and Sugihara 1999; Courchamp et al. 1998, 2000; Oliveira and Hilker 2010). FIV is a retrovirus inducing acquired immunodeficiency syndrome (AIDS) in cats. Its advantages as a potential biological control agent are its high host specificity and low prevalence (Courchamp and Pontier 1994; Hartmann 1998). The former makes mutation and spread to other species less likely (Cleaveland et al. 1999), while the latter is considered beneficial for ongoing transmission (Dobson 1988).

We consider three different strategies of implementing the combined control. First, we assume that FIV has already been introduced in the cat population by releasing a given number of infected individuals (henceforth called *fixed initial release, FIR*). Then, the optimal control problem consists of finding the optimal culling rate as a function of time. In the second strategy, we can choose the number of released infected cats to achieve our objectives (henceforth called *optimal initial release, OIR*). That is, we optimize also over this initial value in addition to optimizing over the culling rate. The third strategy takes into account another way of infecting cats, by which cats are trapped, 'manually' infected and then returned (henceforth called *trap–infect–return, TIR*). This is in addition to the second strategy which optimizes over the culling rate and the number of initially released infected cats.

While the initial release of infected cats is a one-time action, the manual infection of trapped cats as well as the culling can take place continuously in time. Therefore, our optimal control problem incorporates both scalar and time-dependent controls. The scalar control is the initial release of infected cats (via a parameter in the initial conditions of the differential equations). The time-dependent controls are the culling rate on the one hand and the TIR rate on the other hand (both via rates with time-dependent coefficients in the differential equations). The combination of both the scalar and time-dependent controls is interesting from the mathematical perspective as it poses a challenge in the optimization process. We approach this by optimizing first over the time-dependent controls and then over the scalar control.

The mathematical model underlying the optimal control problem is an eco-epidemiological one, as it takes into account the predator–prey interactions between cats and birds as well as the disease spread within the cat population. We introduce this eco-epidemiological model in Sect. 2 and present steady states, their stability, and reproduction numbers in Sect. 3. We then formulate optimal control problems for different strategies in Sect. 4, viz fixed initial release and optimal culling, optimal initial release (or parameter optimization) and optimal culling, and optimal initial release, infection rate and culling. Necessary conditions, characterization, and uniqueness results are established. Numerical simulations of all three strategies, using a

forward–backward numerical method, are presented in Sect. 5. We discuss our results and arrive at conclusions in Sect. 6.

2 Eco-epidemiological Model

In this section, we formulate the eco-epidemiological model that will be the basis of the optimal control problems in the following sections. The model needs to keep track of the cat and the bird population on an island situation as well as their ecological interactions. At the same time, the model needs to incorporate the disease dynamics, as the transmission of the biocontrol agent, FIV is part of the management strategies to be investigated. In addition, the model incorporates a culling rate of the cat population.

Eco-epidemiology is a branch of mathematical biology that deals with ecological and epidemiological aspects simultaneously. The interaction between ecological and epidemiological processes can become complex and lead to emergent dynamic phenomena (e.g., Anderson and May 1986; Hadeler and Freedman 1989; Bate and Hilker 2013). There are a number of papers modeling diseases in predators (e.g., Xiao and Van Den Bosch 2003; Hilker and Schmitz 2008; Kooi et al. 2011), and some are specifically motivated by using FIV to control cats (Courchamp et al. 2000; Courchamp and Sugihara 1999; Courchamp et al. 1998; Oliveira and Hilker 2010). Here, we build upon the model by Oliveira and Hilker (2010), but extend it by a culling rate and an alternative food source of the cats to account for their generalist hunting behavior.

Let $N(t)$ denote the density of prey (birds) at time t and $P(t)$ denote the density of predators (cats) at time t. FIV infection leads to life-long carriers, and there is no recovery or immunity to FIV (Courchamp and Sugihara 1999). We therefore divide the cat population into the two classes of susceptibles (S) and infecteds (I), so that $P(t) = S(t) + I(t)$ is the total population of predators at time t. The basic eco-epidemiological model reads

$$\frac{\mathrm{d}N}{\mathrm{d}t} = rN(t)\left(1 - \frac{N(t)}{K}\right) - aN(t)(S(t) + I(t)), \tag{1}$$

$$\frac{\mathrm{d}S}{\mathrm{d}t} = (b + \varepsilon_1 aN(t))(S(t) + I(t)) - \frac{\Phi(P(t))S(t)I(t)}{P(t)} - (m + h(t) + u(t))S(t), \tag{2}$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \frac{\Phi(P(t))S(t)I(t)}{P(t)} - (m + h(t) + \mu)I(t) + u(t)S(t), \tag{3}$$

with initial conditions

$$N(0) = N_0, \quad S(0) = S_0, \quad I(0) = I_0. \tag{4}$$

We assume that in the absence of predators, the prey population grows logistically with intrinsic per capita growth rate $r > 0$ and environmental carrying capacity $K > 0$. Both susceptible and infected cats prey on birds with a linear functional response and

an attack rate a per prey and predator. Parameter ε_1 is the trophic conversion efficiency and m the per capita natural mortality rate of cats. Infected cats suffer an additional disease-induced per capita mortality μ. Other than that, there is no difference between susceptible and infected cats (Courchamp et al. 1995). This is also the reason why we consider nonselective culling as a control with a per capita rate of $h(t) \geq 0$. Besides having an initial amount of infected cats I_0 as a potential scalar control, we also incorporate FIV infectivity in the model by trapping and infecting a fraction of susceptible predators in the population and releasing them (denoted by TIR control). Thus, the TIR control function, $u(t)$, is the effort in trapping and infecting susceptible predators and returning them to the population.

There is no vertical transmission of FIV, i.e., offspring of infected cats are disease-free, which is why both S and I reproduce into the susceptible class. FIV infection is thought to occur via direct contact through bites during fights for female monopolization or for territorial defense (Courchamp et al. 1998). The term $\Phi(P)$ is the direct transmission rate from susceptible to infected predators, which could be density dependent with $\Phi(P) = \beta_{\text{dd}} P$, if the contact rate between individuals increases linearly, or frequency dependent with $\Phi(P) = \beta_{\text{fd}}$, if the contact rate between individuals is constant. The former is suitable for cat populations in urban habitats with more than 1000 individuals per km^2 or rural and suburban habitats with 10–100 individuals per km^2, whereas the latter is suitable for populations in rural/suburban habitats with cat densities between 100 and 1000 per km^2 and less than 10 individuals per km^2 in nonanthropized areas (Fromont et al. 1998).

Since cats are opportunistic and generalist predators, we assume that they have alternative food sources on which they grow with per capita rate b. This also reflects that cats have a high reproductive capacity and are sexually mature by 5–6 months of age, so that they can sustain high numbers even with high mortality rates (Nutter et al. 2004). If cats depend solely on birds, then $b = 0$, otherwise, $b > 0$.

Table 1 gives an overview of the model parameters along with the values used in the numerical simulations later on as well as their sources. The predation (or attack) rate per prey and predator, a, and density-dependent transmission rate, β_{dd}, are calculated as follows: on Kerguelen Islands, 3500 cats consumed approximately 1.2 million seabirds per year in 1977 (Pascal 1980). Later on, Chaphuis et al. (1994) estimate that cats kill more than 3 million seabirds a year. So if aNP = 1,200,000 or aNP = 3,000,000, with P = 3,500, and the total population of seabirds is approximated at N = 2,000,000, then the attack rate per prey and predator is $a = 0.00017$ or $a = 0.00043$, respectively. In our numerical simulations, we use $a = 0.00017$. On the other hand, Courchamp et al. (2000) estimate the frequency-dependent transmission rate of cats as $\beta_{\text{fd}} = 1.5$, and since the direct transmission rate from susceptible to infected predators is $\Phi(P) = \beta_{\text{dd}} P$ for density-dependent transmission or $\Phi(P) = \beta_{\text{fd}}$ for frequency-dependent transmission, we approximate the density-dependent transmission rate as $\beta_{\text{dd}} = \frac{\beta_{\text{fd}}}{S_0 + I_0}$, where $S_0 = 1173$ is the population of susceptible cats at equilibrium and $I_0 = 100$ is the initial population of infectious cats (assumed). This gives $\beta_{\text{fd}} = 0.0012$. Later on, we will use scalar optimization to determine I_0.

Table 1 Parameters of the eco-epidemiological model (1)–(3)

Par.	Description	Units	Value/range	References
r	Recruitment rate of birds	year^{-1}	0.1–0.5	Fan et al. (2005), Oliveira and Hilker (2010), Rounsevel and Copson (1982)
a	Predation rate of cats on birds	$\text{cat}^{-1}\,\text{year}^{-1}$	0.00017	Calculation
ε_1	Trophic conversion efficiency	cat bird^{-1}	0.01–0.03	Oliveira and Hilker (2010), Pimm (1982)
m	Natural death rate of cats	year^{-1}	0.6	Courchamp and Sugihara (1999), Fan et al. (2005)
μ	Disease-induced mortality of cats	year^{-1}	0.2	citeCourchamp3
b	Birth rate of cats	year^{-1}	0.61	Assumed
h	Culling rate of cats	year^{-1}	0–1	Vary
β_{fd}	Frequency-dependent transmission	year^{-1}	1.5	Courchamp et al. (2000)
β_{dd}	Density-dependent transmission	$\text{cat}^{-1}\,\text{year}^{-1}$	0.0012	Calculation
K	Carrying capacity of birds	Birds	2×10^6	Assumed

3 Reproduction Numbers, Steady States and Stability Analysis

Oliveira and Hilker (2010) investigated the equilibrium solutions and stability analysis of system (1)–(3) when $h(t) \equiv 0$, $u(t) \equiv 0$ and $b = 0$. In this section, we study the model when $h(t) \equiv h > 0, u(t) \equiv 0$ and $b > 0$ (due to sustainability of the population of cats without birds). First, we change variables to nondimensionalize system (1)–(3) and thus ease the stability analysis of steady states. We introduce the following nondimensional variables and parameters:

$$x = \frac{N}{K}, \quad y = \frac{S}{S_0}, \quad z = \frac{I}{S_0}, \quad \tau = rt, \quad \alpha = \frac{aS_0}{r},$$
$$\beta = \frac{\beta_{\text{dd}} S_0}{r}, \quad \delta = \frac{b}{r}, \quad \xi = \frac{a\varepsilon_1 K}{r}, \quad e = \frac{m}{r}, \quad \gamma = \frac{\mu}{r}, \quad \theta = \frac{h}{r}.$$

This leads to the following nondimensionalized system:

$$\frac{\mathrm{d}x}{\mathrm{d}\tau} = x(1-x) - \alpha x(y+z), \tag{5}$$
$$\frac{\mathrm{d}y}{\mathrm{d}\tau} = \delta(y+z) + \xi x(y+z) - \beta yz - (e+\theta)y, \tag{6}$$

$$\frac{\mathrm{d}z}{\mathrm{d}\tau} = \beta yz - (e + \theta + \gamma)z. \tag{7}$$

We list, without proof, the existence of equilibria:

Theorem 1 *System (5)–(7) has five possible equilibria:*

(i) *the trivial equilibrium point* $(x_1^*, y_1^*, z_1^*) = (0, 0, 0)$,
(ii) *the cat-free steady state* $(x_2^*, 0, 0) = (1, 0, 0)$,
(iii) *the predator–prey coexistence steady state in the disease-free subsystem*

$$(x_3^*, y_3^*, 0) = \left(\frac{e + \theta - \delta}{\xi}, \frac{\delta + \xi - e - \theta}{\alpha\xi}, 0\right),$$

which is biologically feasible if $\delta < e + \theta$ *and* $\delta + \xi > e + \theta$,
(iv) *the infected predator steady state in the prey-free subsystem*

$$(0, y_4^*, z_4^*) = \left(0, \frac{e + \theta + \gamma}{\beta}, \frac{(\delta - e - \theta)(e + \theta + \gamma)}{\beta(e + \theta + \gamma - \delta)}\right),$$

which is biologically feasible if $\delta > e + \theta$ *and* $e + \theta + \gamma > \delta$,
(v) *the infected predator–prey coexistence equilibrium* (x_5^*, y_5^*, z_5^*), *where*

$$x_5^* = \frac{\xi + \gamma + e + \theta - \delta - \sqrt{D}}{2\xi}, \quad y_5^* = \frac{e + \theta + \gamma}{\beta},$$
$$z_5^* = \frac{\beta(\xi + \delta) - (e + \theta + \gamma)(\beta + 2\alpha\xi) + \beta\sqrt{D}}{2\alpha\beta\xi},$$

with $D = (\delta + \xi - (e + \theta + \gamma))^2 + \frac{4\alpha\gamma\xi}{\beta}(e + \theta + \gamma) > 0$; x_5^* *and* z_5^* *are positive if* $(e + \theta + \gamma)\left(1 - \frac{\alpha\gamma}{\beta}\right) > \delta$ *and* $\beta(\xi + \delta) + \beta\sqrt{D} > (e + \theta + \gamma)(\beta + 2\alpha\xi)$, *respectively.*

Using the next-generation method (Diekmann et al. 1990, 1991, 2010, 2013; Diekmann and Heesterbeek 2000; Driessche and Watmough 2002), we obtain the following demographic reproduction number, $\mathcal{R}_D$, and basic reproduction number, $\mathcal{R}_0$, of cats in the presence of culling, evaluated at the cat-free equilibrium and at the predator–prey coexistence steady state in the disease-free subsystem, respectively:

$$\mathcal{R}_D = \frac{\delta + \xi}{e + \theta} \quad \text{and} \quad \mathcal{R}_0 = \frac{\beta(e + \theta)(\mathcal{R}_D - 1)}{\alpha\xi(e + \theta + \gamma)}. \tag{8}$$

The demographic reproduction number gives the expected number of offspring of a predator individual in its lifetime, with the assumption that the prey population is at carrying capacity. On the other hand, the basic reproduction number, $\mathcal{R}_0$, only makes sense if $\mathcal{R}_D > 1$. If $\mathcal{R}_D > 1$, predators are sustained, while the disease establishes itself in the population if $\mathcal{R}_0 > 1$. These reproduction numbers give

insight into the existence and stability of the cat-free steady state and the predator–prey coexistence equilibrium in the disease-free subsystem. The stability analysis of equilibria is summarized in Theorem 2.

Theorem 2 (i) *The trivial extinction point* $(0, 0, 0)$ *is unstable.*
(ii) *The cat-free steady state* $(1, 0, 0)$ *is stable if* $\mathcal{R}_D < 1$ *and unstable if* $\mathcal{R}_D > 1$.
(iii) *The predator–prey coexistence steady state in the disease-free subsystem* $(x_3^*, y_3^*, 0)$ *exists if* $\mathcal{R}_D > 1$ *and is stable if* $\mathcal{R}_0 < 1$.
(iv) *The infected predator steady state in the prey-free subsystem* $(0, y_4^*, z_4^*)$ *exists if* $\mathcal{R}_D > 1$ *and is stable if*

$$\frac{\gamma(e+\theta)(\mathcal{R}_D - 1)}{\xi\mathcal{R}_0(e+\theta+\gamma-\delta)} > 1.$$

Proof The stability analysis of the nondimensionalized model (5)–(7) is governed by the Jacobian matrix

$$J(x, y, z) = \begin{pmatrix} 1 - 2x - \alpha(y+z) & -\alpha x & -\alpha x \\ \xi(y+z) & \eta + \xi x - \beta z & \delta + \xi x - \beta y \\ 0 & \beta z & \beta y - \omega \end{pmatrix}, \tag{9}$$

where $\eta = \delta - e - \theta$ and $\omega = e + \theta + \gamma > 0$.

(i) At the trivial extinction point $(x_1^*, y_1^*, z_1^*) = (0, 0, 0)$, the eigenvalues of (9) are $\lambda_1 = 1 > 0$, $\lambda_2 = \delta - e - \theta$ and $\lambda_3 = -(e + \theta + \gamma) < 0$. Hence, the trivial steady state $(0, 0, 0)$ is unstable.
(ii) At the cat-free steady state $(x_2^*, 0, 0) = (1, 0, 0)$, the eigenvalues of (9) are $\lambda_1 = -1 < 0$, $\lambda_2 = (e + \theta)(\mathcal{R}_D - 1)$ and $\lambda_3 = -(e + \theta + \gamma) < 0$. Hence, the cat-free steady state $(1, 0, 0)$ is stable if $\mathcal{R}_D < 1$ and unstable if $\mathcal{R}_D > 1$.
(iii) At the predator–prey coexistence steady state in the disease-free subsystem $\left(\frac{e+\theta-\delta}{\xi}, \frac{\delta+\xi-e-\theta}{\alpha\xi}, 0\right)$, one of the eigenvalues of the Jacobian matrix (9) satisfies

$$\lambda_1 = \beta\left(\frac{\delta + \xi - e - \theta}{\alpha\xi}\right) - (e + \theta + \gamma) \equiv (e + \theta + \gamma)(\mathcal{R}_0 - 1),$$

and the other two eigenvalues, $\lambda_{2,3}$, satisfy the quadratic equation

$$\lambda_{2,3}^2 - \left(\frac{\delta - e - \theta}{\xi}\right)\lambda_{2,3} - \frac{(\delta - e - \theta)(\delta + \xi - e - \theta)}{\xi} = 0.$$

In this case, $\eta = \delta - e - \theta < 0$. This gives

$$\lambda_2 = \frac{\eta}{2\xi} + \frac{1}{2}\sqrt{\left(\frac{\eta}{\xi}\right)^2 + \frac{4\eta(e+\theta)(\mathcal{R}_D - 1)}{\xi}},$$

$$\lambda_3 = \frac{\eta}{2\xi} - \frac{1}{2}\sqrt{\left(\frac{\eta}{\xi}\right)^2 + \frac{4\eta(e+\theta)(\mathcal{R}_D - 1)}{\xi}}.$$

Thus, λ_2 and λ_3 are real and negative roots or complex roots with negative real parts. Hence, the predator–prey coexistence steady state in the disease-free subsystem is stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.

(iv) At the infected predator steady state in the prey-free subsystem $(0, y_4^*, z_4^*) = \left(0, \frac{e+\theta+\gamma}{\beta}, \frac{(\delta-e-\theta)(e+\theta+\gamma)}{\beta(e+\theta+\gamma-\delta)}\right)$, one of the eigenvalues of the Jacobian matrix (9) satisfies

$$\begin{aligned}\lambda_1 &= 1 - \frac{\alpha(e+\theta+\gamma)}{\beta} - \frac{\alpha(\delta-e-\theta)(e+\theta+\gamma)}{\beta(e+\theta+\gamma-\delta)} \\ &= 1 - \frac{\gamma(e+\theta)(\mathcal{R}_D-1)}{\xi\mathcal{R}_0(e+\theta+\gamma-\delta)},\end{aligned}$$

and the other two eigenvalues, $\lambda_{2,3}$, satisfy the quadratic equation

$$\lambda_{2,3}^2 - \left(\frac{\delta(e+\theta-\delta)}{e+\theta+\gamma-\delta}\right)\lambda_{2,3} + (\delta-e-\theta)(e+\theta+\gamma) = 0.$$

In this case, $\eta = \delta - e - \theta > 0$. This gives

$$\begin{aligned}\lambda_2 &= -\frac{\delta\eta}{2(e+\theta+\gamma-\delta)} + \frac{1}{2}\sqrt{\left(\frac{\delta\eta}{e+\theta+\gamma-\delta}\right)^2 - 4\eta\omega}, \\ \lambda_3 &= -\frac{\delta\eta}{2(e+\theta+\gamma-\delta)} - \frac{1}{2}\sqrt{\left(\frac{\delta\eta}{e+\theta+\gamma-\delta}\right)^2 - 4\eta\omega}.\end{aligned}$$

Thus, λ_2 and λ_3 are real and negative roots or complex roots with negative real parts. Hence, the steady state $(0, y_4^*, z_4^*)$ is stable if $\frac{\gamma(e+\theta)(\mathcal{R}_D-1)}{\xi\mathcal{R}_0(e+\theta+\gamma-\delta)} > 1$ and unstable if $\frac{\gamma(e+\theta)(\mathcal{R}_D-1)}{\xi\mathcal{R}_0(e+\theta+\gamma-\delta)} < 1$.

Finally, we examine the stability of the infected predator–prey coexistence equilibrium, using the Routh–Hurwitz conditions (Allen 2007; Kot 2001; Murray 1993).

Theorem 3 *If* $\frac{(\beta+\alpha\xi)\sqrt{D}}{\alpha\xi} > \xi+\gamma-e+\theta+\delta$, $2e\xi > (1+\xi)\delta$, $\xi < 1$ *and* $\frac{(\gamma+1)}{2\xi}(\delta+\xi-(e+\theta+\gamma)+\sqrt{D}) > 1+\frac{\alpha\gamma}{\beta}(e+\theta+\gamma)$, *then the infected predator–prey coexistence equilibrium,* $\left(x_5^*, \frac{e+\theta+\gamma}{\beta}, z_5^*\right)$, *is stable.*

Proof Eigenvalues of the Jacobian matrix (9) at the point $\left(x_5^*, \frac{\omega}{\beta}, z_5^*\right)$, where $\omega = e+\theta+\gamma$, satisfy

$$0 = \left(1 - \frac{\alpha\omega}{\beta} - 2x_5^* - \alpha z_5^* - \lambda\right)\left[-\lambda(\delta - e + \xi x_5^* - \beta z_5^* - \lambda)\right.$$
$$\left. - \beta z_5^*(\delta - \omega + \xi x_5^*)\right] - \alpha x_5^*\left(\frac{\xi\omega}{\beta} + \xi z_5^*\right)\lambda$$
$$- \alpha\beta x_5^* z_5^*\left(\frac{\xi\omega}{\beta} + \xi z_5^*\right).$$

This leads to the characteristic equation

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0, \tag{10}$$

where

$$a_1 = -\left(1 - \frac{\alpha\omega}{\beta} + \delta - e + (\xi - 2)x_5^* - (\alpha + \beta)z_5^*\right),$$
$$a_2 = \left(1 - \frac{\alpha\omega}{\beta} - 2x_5^* - \alpha z_5^*\right)(\delta - e + \xi x_5^* - \beta z_5^*)$$
$$+ \alpha x_5^*\left(\frac{\xi\omega}{\beta} + \xi z_5^*\right) - \beta z_5^*(\delta - \omega + \xi x_5^*),$$
$$a_3 = \beta z_5^*(\delta - \omega + \xi x_5^*)\left(1 - \frac{\alpha\omega}{\beta} - 2x_5^* - \alpha z_5^*\right) + \alpha\beta x_5^* z_5^*\left(\frac{\xi\omega}{\beta} + \xi z_5^*\right).$$

With the assumptions in Theorem 3, the characteristic equation (10) satisfies the following Routh–Hurwitz conditions: $a_1 > 0, a_3 > 0$ and $a_1 a_2 > a_3$. Thus, the eigenvalues of equation (10) have negative real parts, and hence, the infected predator–prey coexistence equilibrium is stable.

We are finished with the nondimensionalized system and will return to system (1)–(3). In a situation requiring control of the cat population, we will formulate an optimal control problem and investigate harvesting and disease-related control strategies.

4 Optimal Control Formulation and Analysis

Besides two time-varying controls, $h(t)$ and $u(t)$, the scalar, I_0, is also taken as a control, meaning that the initial infected predator population is to be chosen. Therefore, using system (1)−(3), we minimize the objective functional

$$J(I_0, h, u) = A_3 I_0^2 + \int_0^{t_1} (A_1(S(t) + I(t)) - A_2 N(t))\mathrm{d}t$$
$$+ \int_0^{t_1} (ch(t)(S(t) + I(t)) + \varepsilon h(t)^2)\mathrm{d}t$$
$$+ \int_0^{t_1} (B_1 u(t)S(t) + B_2 u(t)^2)\mathrm{d}t, \tag{11}$$

over time-dependent controls $h(t)$ and $u(t)$, and scalar control $I(0) = I_0$. Weight constants, A_1, A_2, A_3, B_1, B_2, c, and ε are nonnegative constants that balance the relative importance of terms in J. The terms $\int_0^{t_1}(A_1(S(t)+I(t))\mathrm{d}t$ and $\int_0^{t_1} A_2 N(t)\mathrm{d}t$ in the objective functional give the respective numbers of cats and birds over the time period t_1 being modeled. The term $h(S+I)$ represents the total number of cats culled, where h represents the per capita rate of culling cats from the population, and c is the cost per cat culled. Thus, $\int_0^{t_1}(ch(t)(S(t)+I(t))+\varepsilon h^2(t))\mathrm{d}t$ gives the cost of culling cats from the population. The coefficient B_1 converts the total number of susceptible cats trapped and infected with FIV to the cost of infecting susceptible cats, so that $B_1 uS + B_2 u^2$ represents the total cost of trapping and infecting susceptible cats in the population. The term $A_3 I_0^2$ represents a cost to have initial infected predator population, I_0. As the costs in an objective functional are frequently nonlinear functions of the control actions (Gaff and Schaefer 2009; Lenhart and Workman 2007), we chose quadratic terms for our costs (with B_2 and ε). The optimal control formulation for our problem involving culling, infection rate, and parameter optimization (optimal initial release) is: Find $(I_0^*, h^*, u^*) \in \mathcal{U}$ such that

$$J(I_0^*, h^*, u^*) = \inf_{I_0}\left(\inf_{h,u} J(I_0, h, u)\right) \tag{12}$$

subject to the state system defined in Eqs. (1)–(3), where the objective functional is given by equation (11), and the set of all admissible controls is

$$\begin{aligned}\mathcal{U} = \{(I_0, h, u) \in M \times (L^\infty([0,t_1]))^2 |\ h : [0,t_1] \\ \to [0, h_{max}],\ u : [0,t_1] \to [0, u_{max}]\},\end{aligned}$$

with a finite set $M \subset \mathbb{N}$, the set of natural numbers.

In order to prove the existence of an optimal control problem, we require the state functions of the eco-epidemiological model to be bounded. The positivity and boundedness results below follow from the structure of the system.

Theorem 4 *Given the state equations for N, S, and I defined in Eqs. (1)–(3) with initial conditions (4), and $N_0 \geq 0$, $S_0 \geq 0$, $I_0 \geq 0$, there exist constants C_1, C_2, $C_3 > 0$ such that $0 < N(t) \leq C_1$, $0 < S(t) \leq C_2$ and $0 \leq I(t) \leq C_3$, for all $t \in [0, t_1]$.*

One reasonable approach to optimize over both parameter and time-dependent controls is to start with the time-dependent controls and incorporate the parameter optimization afterward. To use Pontryagin's maximum principle (Pontryagin et al. 1967) on the time-dependent controls, we first need existence of a triple of optimal controls, and characterize the time-dependent controls and adjoint equations for system (1)–(3), when density- and frequency-dependent transmission rates are studied.

Theorem 5 *There exist optimal controls $(I_0^*, h^*, u^*) \in \mathcal{U}$ which minimize the objective functional, J, subject to the state system (1)–(3).*

Proof By the uniform boundedness of states and controls, the infimum is finite, and thus, there exist minimizing sequences $\{I_0^n\}$, $\{h_n\}$, $\{u_n\}$:

$$\lim_{n\to\infty} J(I_0^n, h_n, u_n) = \inf_{(I_0,h,u)\in\mathcal{U}} J(I_0, h, u).$$

Since the corresponding states N_n, S_n, and I_n are uniformly bounded for all n over the interval $[0, t_1]$ and from the structure of system (1)−(3), it follows that their derivatives are also uniformly bounded. Thus, N_n, S_n, and I_n are Lipschitz continuous with the same Lipschitz constant. Thus, the sequence $\{N_n, S_n, I_n\}$ is equicontinuous, and therefore, by Arzela–Ascoli theorem, there exists (N^*, S^*, I^*) such that on a subsequence,

$$(N_n, S_n, I_n) \to (N^*, S^*, I^*) \quad \text{uniformly on} \quad [0, t_1].$$

Also, the control sequences, h_n and u_n, are bounded for any n and t, so there exist subsequences $I_0^{n_k}$, h_{n_k} and u_{n_k}, and controls $(I_0^*, h^*, u^*) \in \mathcal{U}$ such that

$$I_0^{n_k} \to I_0^*, \quad \text{and} \quad h_{n_k} \rightharpoonup h^*, u_{n_k} \rightharpoonup u^* \quad \text{weakly in} \quad L^2([0, t_1]).$$

Using the lower-semicontinuity of L^2 norms with respect to weak convergence, we have

$$\begin{aligned} J(I_0^*, h^*, u^*) \le\ & A_3(I_0^*)^2 + \liminf_{n\to\infty} \int_0^{t_1} (A_1(S_n(t) + I_n(t)) - A_2 N_n(t))\mathrm{d}t \\ & + \liminf_{n\to\infty} \int_0^{t_1} (ch_n(t)(S_n(t) + I_n(t)) + \varepsilon h_n(t)^2)\mathrm{d}t \\ & + \liminf_{n\to\infty} \int_0^{t_1} (B_1 u_n(t) S_n(t) + B_2 u_n(t)^2)\mathrm{d}t \\ =\ & \inf_{(I_0,h,u)\in\mathcal{U}} J(I_0, h, u). \end{aligned}$$

Using the convergence of the state sequences and passing to the limit in the ODE system, we have that N^*, S^*, and I^* are the states corresponding to the controls I_0^*, h^*, and u^*. Note that the uniform convergence of states and the weak convergence of the controls are needed for the convergence of terms like $h_n S_n$. Since $I_0^* \in M \subset \mathbb{N}$, where M is finite, we conclude that (I_0^*, h^*, u^*) is a triple of optimal controls.

We characterize the time-dependent controls and the corresponding adjoint equations, when density- and frequency-dependent transmission rates are studied. We fix $I_0 \in M$ first and apply Pontryagin's maximum principle (Pontryagin et al. 1967) to our problem.

In finding $\min_{h,u} J(I_0, h, u)$, we use the Hamiltonian

$$\begin{aligned} H =\ & A_1(S + I) - A_2 N + ch(S + I) + \varepsilon h^2 + B_1 uS + B_2 u^2 \\ & + \lambda_N \left(rN\left(1 - \frac{N}{K}\right) - aN(S + I)\right) \end{aligned}$$

$$+\lambda_S\left((b+\varepsilon_1 aN)(S+I)-\frac{\Phi(P)SI}{P}-(m+h+u)S\right)$$
$$+\lambda_I\left(\frac{\Phi(P)SI}{P}-(m+h+\mu)I+uS\right),$$

where λ_N, λ_S, and λ_I are adjoint functions associated with the state functions N, S, and I, respectively. The following two theorems characterize optimal culling and infection rate when density- and frequency-dependent transmission rates are considered.

Theorem 6 *Given a fixed initial release, I_0, and for density-dependent transmission, with optimal controls $h^* = h^*(I_0)$ and $u^* = u^*(I_0)$, and corresponding states N^*, S^*, and I^*, there exist adjoint functions λ_N, λ_S, and λ_I satisfying the equations*

$$\lambda_N' = \left(-r+\frac{2rN^*}{K}+aP^*\right)\lambda_N - \varepsilon_1 aP^*\lambda_S + A_2, \tag{13}$$
$$\lambda_S'(t) = -(b+\varepsilon_1 aN^* - \beta_{\mathrm{dd}}I^* - (m+h^*+u^*))\lambda_S + aN^*\lambda_N$$
$$-(\beta_{\mathrm{dd}}I^*+u^*)\lambda_I - ch^* - B_1u^* - A_1, \tag{14}$$
$$\lambda_I'(t) = aN^*\lambda_N - (b+\varepsilon_1 aN^* - \beta_{\mathrm{dd}}S^*)\lambda_S$$
$$-(\beta_{\mathrm{dd}}S^* - (m+h^*+\mu))\lambda_I - ch^* - A_1, \tag{15}$$

with final time conditions

$$\lambda_N(t_1) = \lambda_S(t_1) = \lambda_I(t_1) = 0. \tag{16}$$

Furthermore, the optimal control characterization for the time-dependent controls, h^* and u^*, is

$$h^*(t) = \min\left\{h_{\max}, \max\left\{0, \frac{S^*(t)\lambda_S(t)+I^*(t)\lambda_I(t)}{2\varepsilon} - \frac{c(S^*(t)+I^*(t))}{2\varepsilon}\right\}\right\}, \tag{17}$$

$$u^*(t) = \min\left\{u_{\max}, \max\left\{0, \frac{S^*(t)(\lambda_S(t)-\lambda_I(t)-B_1)}{2B_2}\right\}\right\}. \tag{18}$$

Proof The adjoint equations are obtained from the partial derivatives of the Hamiltonian, H with respect to each state variable. That is,

$$\lambda_N'(t) = -\frac{\partial H}{\partial N}, \quad \lambda_S'(t) = -\frac{\partial H}{\partial S}, \quad \text{and} \quad \lambda_I'(t) = -\frac{\partial H}{\partial I}.$$

Using

$$\frac{\partial H}{\partial h} = c(S+I) + 2\varepsilon h - S\lambda_S - I\lambda_I = 0,$$
$$\frac{\partial H}{\partial u} = B_1S + 2B_2u - S\lambda_S + S\lambda_I = 0,$$

on the interior of the control set, the optimal control characterizations given in Eqs. (17) and (18) are obtained.

Theorem 7 *Given a fixed initial release, I_0, and for frequency-dependent transmission, with optimal controls $h^* = h^*(I_0)$ and $u^* = u^*(I_0)$, and corresponding states N^*, S^*, and I^*, there exist adjoint functions λ_N, λ_S, and λ_I satisfying the equations*

$$\lambda_N' = \left(-r + \frac{2rN^*}{K} + aP^*\right)\lambda_N - \varepsilon_1 aP^*\lambda_S + A_2, \tag{19}$$

$$\begin{aligned}\lambda_S' = {} & aN^*\lambda_N - \left(b + \varepsilon_1 aN^* - \beta_{\text{fd}}\left(\frac{I}{P}\right)^2 - m - h^* - u^*\right)\lambda_S \\ & - \left(\beta_{\text{fd}}\left(\frac{I}{P^*}\right)^2 + u^*\right)\lambda_I \\ & - ch^* - B_1u^* - A_1,\end{aligned} \tag{20}$$

$$\begin{aligned}\lambda_I' = {} & aN^*\lambda_N - \left(b + \varepsilon_1 aN^* - \beta_{\text{fd}}\left(\frac{S^*}{P^*}\right)^2\right)\lambda_S \\ & - \left(\beta_{\text{fd}}\left(\frac{S^*}{P^*}\right)^2 - m - h^* - \mu\right)\lambda_I - ch^* - A_1,\end{aligned} \tag{21}$$

with final time conditions (16)*, and optimal control characterizations as in* (17) *and* (18) *of Theorem* 6*.*

Proof Follows as in Theorem 6.

Remark The adjoint systems in Theorems 6 and 7 are linear in λ_N, λ_S, and λ_I. Since we have a linear system in finite time with bounded coefficients, it follows that λ_N, λ_S, and λ_I are uniformly bounded. Using the boundedness of state and adjoint functions, it can be shown that the solution of the optimality system is unique for t_1 small.

5 Numerical Simulations

In this section, we investigate different control strategies for the model with density-dependent transmission in numerical simulations. The three management programs considered are: (i) a fixed initial release of infected cats; (ii) an optimal initial release of infected cats; and (iii) a trap–infect–return control program. All of these controls are combined with optimal culling, but we will also consider the case without culling.

We start by investigating three reference scenarios, consisting of birds and cats population dynamics in the absence of any control. They correspond to the three types of qualitative behavior identified in the stability analysis of Sect. 3. When investigating the control strategies, we will consider the impact of different initial conditions, the time horizon over which control can be implemented, and different cost parameterizations. Before doing so, we describe the numerical details.

Table 2 Weight constants and upper bounds of culling and TIR-infection rates

Parameter	A_1	A_2	A_3	B_1	B_2	c	ε	$h_{\max}$	$u_{\max}$
Value	1	1	0.1	1	200	1	100	0.3	0.2

5.1 Numerical Integration Method and Parameter Values

The optimality system is solved using an iterative scheme. A forward–backward sweep method (Lenhart and Workman 2007), using the fourth-order Runge–Kutta, is used to solve for the state and adjoint equations. Starting with an initial condition for the state functions and an initial guess for the control, a forward sweep with fourth-order Runge–Kutta is used to obtain an approximate solution to the state equations. Using this estimate and the final time conditions, the solution to the adjoint system is approximated using a backward sweep with fourth-order Runge–Kutta method. The control is updated by using an average of its previous values and values from the control characterization (Lenhart and Workman 2007). Previous steps are repeated until consecutive iterates of controls, states, and adjoints are sufficiently close. The convergence of the forward–backward sweep method is based on the work by Hackbusch (1978).

The parameter values used in our numerical simulations, unless stated otherwise, are presented in Tables 1 and 2. The TIR control may be difficult to implement at a high rate, as trapping, infecting, and returning of cats are costly and challenging. Therefore, we use a smaller number for the upper bound of the TIR rate $u(t)$, and $B_2 > \varepsilon$. Irrespective of the duration of control at the present level of culling ($h_{\max} = 0.3$) and TIR rate ($u_{\max} = 0.2$), in the simulations performed the optimal TIR rate lasts for a shorter period of time at its maximum level compared to the optimal culling rate.

5.2 Reference Scenarios

We consider three reference scenarios in which there is no control implemented yet. That is, there is no culling and FIV. In each case, we assume initial values of 2 million birds and 3500 cats.

In the first reference scenario, cats depend solely on birds for survival, i.e., $b = 0$. Figure 1a, b shows that the cats initially increase in population size but cannot persist on the birds alone and eventually go extinct. This allows the birds to recover. Henceforth, we will refer to this scenario as *cat extinction scenario*.

The remaining reference scenarios take into account that cats are opportunistic predators and feed on alternative food sources, i.e., $b > 0$. In the second reference scenario, the supply of alternative food is so high ($b > m$) that cats can persist without birds. Figure 1c, d shows that the cat population size rapidly increases and extinguishes the birds, after which the cat population size grow at a slower rate. We will refer to this case as *bird extinction scenario*.

In the third reference scenario, the supply of alternative food is at some intermediate level ($0 < b < m$). This allows cats to persist, and predation pressure on birds is not too high, so that both species can coexist, cf. Fig. 1e, f. We will refer to this case as *coexistence scenario*.

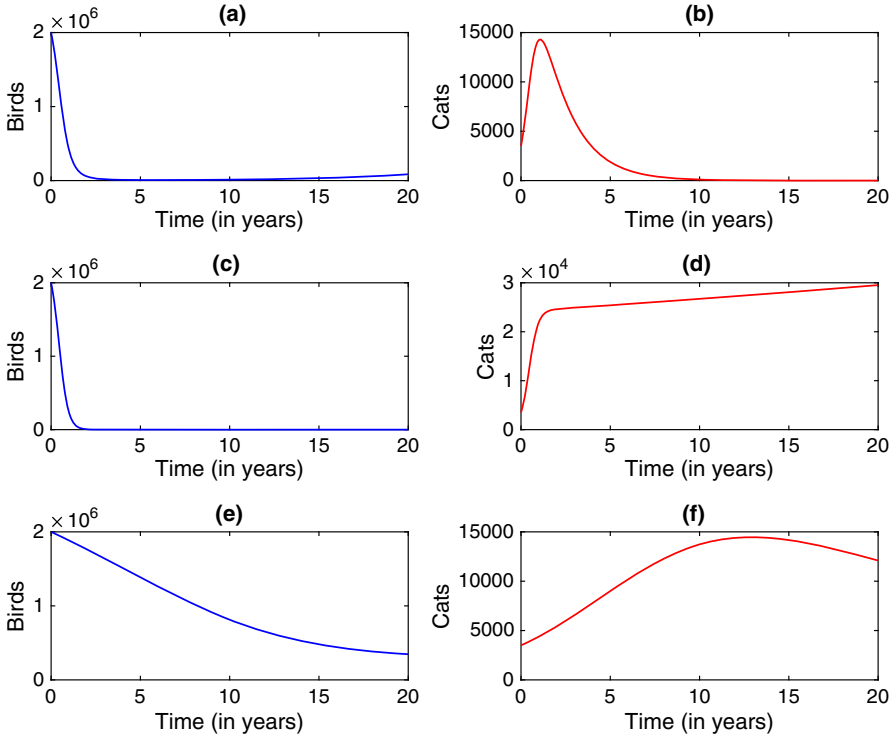

Fig. 1 Reference scenarios in the absence of any control. **a**, **b** cat extinction scenario, in which cats are specialist predators ($b = 0$). **c**, **d** bird extinction scenario, in which cats are generalist predators and $b > m$, with $b = 0.61$. **e**, **f** coexistence scenario, in which cats are generalist predators and $0 < b < m$, with $b = 0.5$. All other parameter values as in Table 1. Model (1)–(3) with $h(t) = u(t) = I_0 = 0$ and initial conditions $N_0 = 2 \times 10^6$ and $P_0 = 3500$

The three reference scenarios reflect the results from the stability analysis, in the absence of culling and disease control. If the cats' birth rate from alternative food sources is larger than their natural mortality ($b > m$ or, equivalently, if $\delta > e$ in terms of nondimensionalized parameters), the predator steady state in the prey-free subsystem is stable. On the other hand, if $b < m$, the predator–prey coexistence steady state in the disease-free subsystem is stable.

5.3 Fixed Initial Release (FIR) Control and Optimal Culling

We now assume an initial condition that is the equilibrium point of susceptible cats and birds in the absence of disease and culling. First, we consider the coexistence scenario and introduce FIV as a biological control agent. This is assumed as fixed initial release, i.e., with an initial condition fixed at $I_0 = 100$. Figure 2 shows trajectories for birds, susceptible cats, and infectious cats over time. We additionally consider optimal culling over a period of 4 years on the one hand and the absence of culling on the other hand.

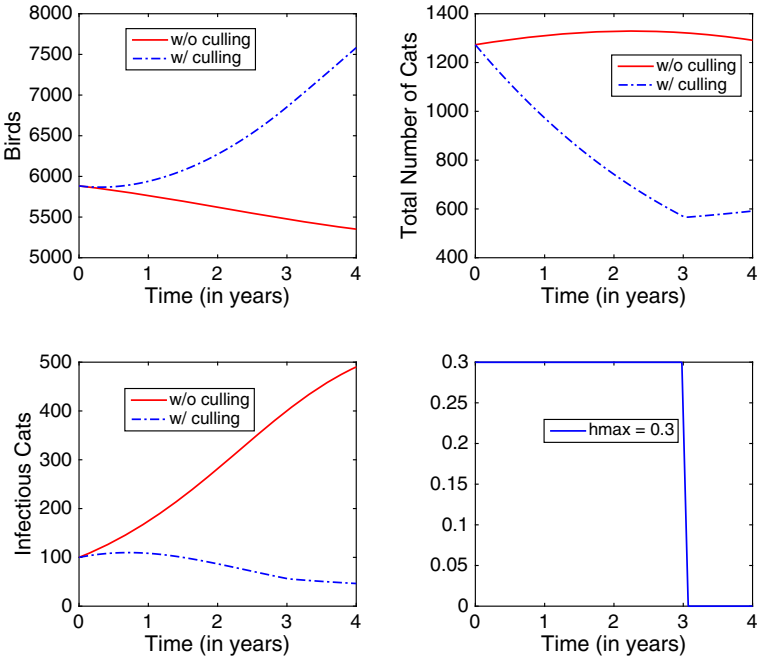

Fig. 2 Fixed initial release strategy and optimal culling in the coexistence scenario ($0 < b < m$) with the uncontrolled coexistence equilibrium as initial condition. $N_0 = 5883$, $S_0 = 1173$, $I_0 = 100$, $m = 0.61$, $b = 0.60$, and $\beta_{\text{dd}} = 0.0012$

In the absence of culling, the disease spreads within the cat population with the number of infectious cats rapidly increasing. The total cat population initially shows a slight increase to be followed by a slight decrease. Overall, the cats remain at a high level above their initial value. The bird population steadily declines.

In the presence of (optimal) culling, the decline of the bird population is reversed to an increase. The total population of cats decreases within the first 2.9 years, followed by a short slight increase. The culling effort is at its maximum value within the first 2.9 years, followed by a sharp decline between years 2.9 and 3.1, with no culling effort afterward. Of the 100 infectious cats initially introduced, approximately 50 remain at the end of the control period.

Even though the combination of biocontrol and optimal culling is efficacious in reducing the cat population and letting the birds recover, the results in Fig. 2 suggest that this control strategy is insufficient in controlling the population of cats, since the total cat population size shows an increasing trend at about 600 individuals toward the end of the control period. Thus, for the coexistence scenario in Fig. 2, if the birth rate of cats is smaller than their background mortality, then the fixed initial release strategy may not suffice as a control strategy in eradicating cats.

Second, we now investigate the situation where the birth rate of cats is greater than their background mortality, which corresponds to the bird extinction scenario. As initial condition, we assume the bird population at half their carrying capacity.

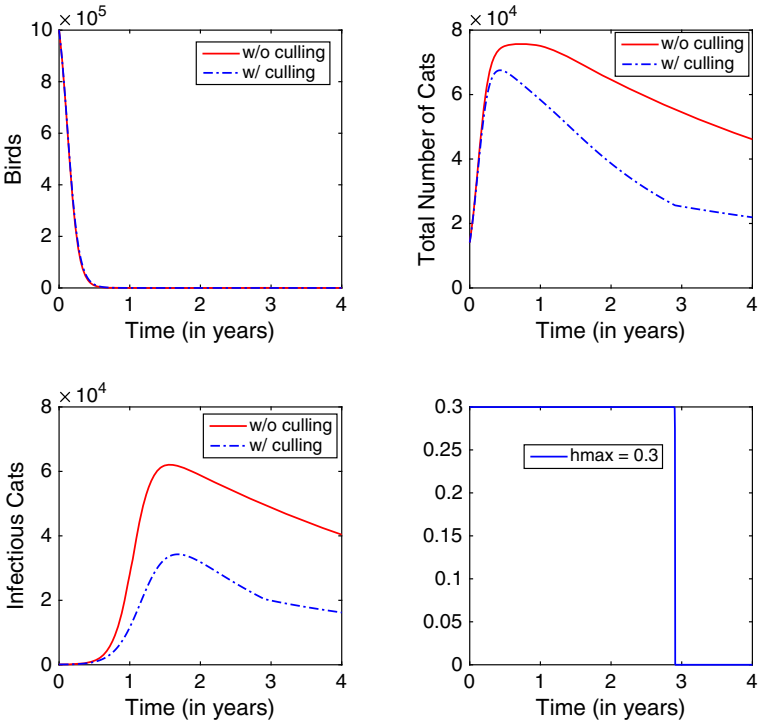

Fig. 3 Fixed initial release strategy and optimal culling in the bird extinction scenario ($b > m$) with birds at half their carrying capacity as initial condition. $N_0 = 1 \times 10^6$, $S_0 = 14000$, $I_0 = 100$, $K = 2 \times 10^6$, and $\beta_{dd} = 0.0001$

The initial population of susceptible cats corresponds to the population of cats at the time when the population of birds is one-half their carrying capacity. In the absence of culling, the population of cats rises sharply at first and decreases afterward (Fig. 3). In the presence of culling, there is still the sharp initial increase in the total population of cats; the following decrease is stronger, with fewer cats observed at the end of the control period. Infectious cats show a similar hump-shaped pattern in population size over time, but their peak growth period is delayed. The population of birds reduces for the first 6 months and eventually becomes extinct, both in the presence and absence of control.

The rapid bird extinction observed in Fig. 3 may be due to the high predation pressure caused by the initial condition. We therefore consider, third, an initial condition with low levels of cats and birds (Fig. 4), still in the bird extinction scenario. The bird population still decreases, but does not go extinct during the control period. Culling promotes the level of bird population. Biocontrol alone reduces the total cat population somewhat, but the joint control with culling reduces the cats to a much lower level. The culling effort is at its maximum for the first 2.5 years, followed by a steady decrease between years 2.5 and 3, and no culling effort afterward.

With the initial condition in Fig. 4, and again for the bird extinction scenario, birds persist over the control period, while the stability analysis for this scenario

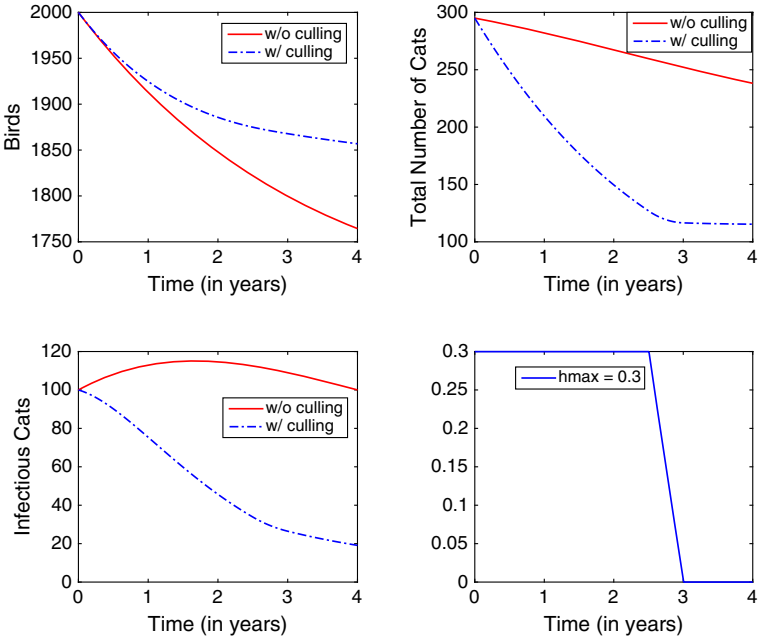

Fig. 4 Fixed initial release strategy and optimal culling in the bird extinction scenario ($b > m$) and with low levels of cats and birds as initial condition. $N_0 = 2000$, $S_0 = 195$, $I_0 = 100$, $K = 2000$, and $\beta_{\text{dd}} = 0.0051$. Here, the initial number of infected cats released is optimal, so this control corresponds to optimal initial release control

predicts asymptotic bird extinction. Both the results in Figs. 2 (coexistence) and 3 (bird extinction) corroborate the stability results in Theorem 2 within the time horizon considered.

Despite considering different scenarios and different initial conditions, the fixed release strategy with optimal culling did not achieve cat eradication or substantial bird recovery in the simulations performed. In the following, we incorporate the initial population of infectious cats as a scalar control and investigate the effect of combining optimal culling with the scalar optimization.

5.4 Parameter Optimization

For the OIR and TIR controls, we wish to determine the optimal parameter I_0^* for the initial release of infected cats. In order to do, we find the J values for each $I_0 \in M$, using the optimal harvest, $h^*(I_0)$, and optimal effort in trapping–infecting–returning susceptible predators, $u^*(I_0)$, in the objective functional given in Eq. (11). Thus, we find I_0^* such that

$$J(I_0^*, h^*(I_0^*), u^*(I_0^*)) = \min_{I_0 \in M} J(I_0, h^*(I_0), u^*(I_0)) \tag{22}$$

Table 3 Scalar optimization of (22) when $A_3 = 0.1$

I_0	Value of J for $t = 4$ years	Value of J for $t = 10$ years
10	367,750.5	504,261.2
20	363,143.7	497,749.7
30	360,680.3	494,263.9
50	358,016.0	490,430.7
75	356,488.4	488,094.2
100	355,943.1*	487,055.7
150	356,463.8	487,026.8*
200	358,335.9	488,640.0
300	364,784.0	494,968.9
400	374,034.8	504,363.8
600	399,596.6	530,572.1
1000	476,229.5	609,094.8

Asterisks indicate extremal values

numerically. We illustrate this idea using

$$M = \{10, 20, 30, 50, 75, 100, 150, 200, 300, 400, 600, 1000\}.$$

Table 3 gives values of the objective functional, J, evaluated at $h^*(I_0)$ and $u^*(I_0)$, for $I_0 \in M$, with $A_3 = 0.1$. The optimal parameter I_0^* is 100 infectious cats for a time horizon of 4 years, and I^* is 150 infectious cats for a time horizon of 10 years. When $A_3 = 0.01$ (meaning lower cost of this I_0 action), the optimal parameter I_0^* increases to 150 infectious cats for a time horizon of 4 years, and I_0^* increases to 200 infectious cats for a time horizon of 10 years.

5.5 Optimal Initial Release (OIR) Control and Optimal Culling

In the OIR control, we find the optimal scalar using the optimal culling, $h^*(I_0)$, but with no TIR control (i.e., $u(t) = 0$). For a control period of 4 years, the optimal parameter I_0^* is 100 infectious cats. This case is already shown in Fig. 4 for the bird extinction scenario with low levels of birds and cats as initial condition. That is, in this case FIR is the same as OIR when $I_0^* = 100$.

So far, we have been considered time horizons of 4 years for the control. If we allow control to take place over a period of 10 years, the results change to the ones shown in Fig. 5. Note that the optimal number of infectious cats correspondingly changes to $I_0^* = 150$.

Without any culling, the bird population continually declines, but with optimal culling the declining trend reverses halfway through the control period, and the bird population size increases even though it does not reach its initial value. The total cat population size continually decreases with or without culling. The optimal effort in

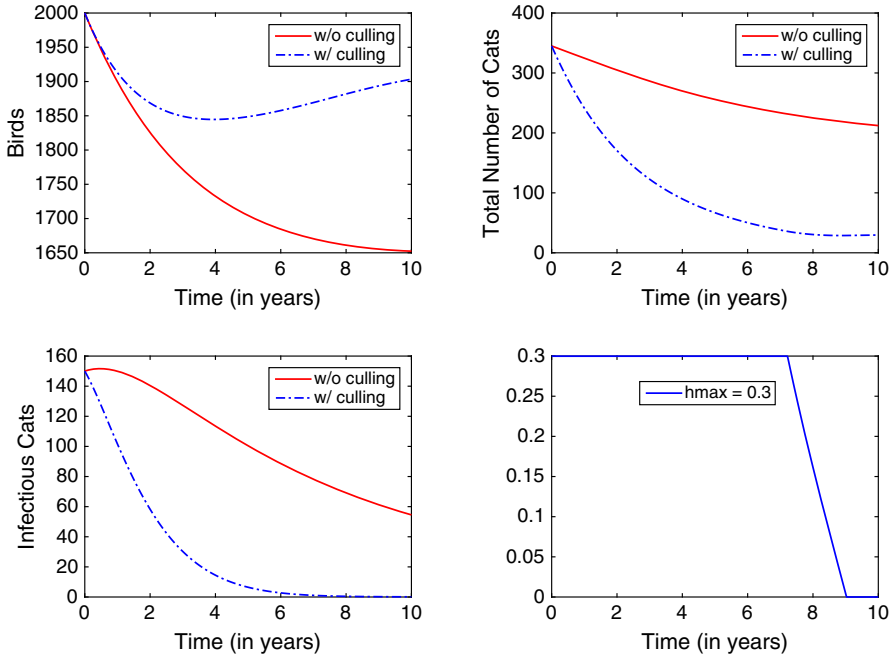

Fig. 5 Time horizon of $t = 10$ years. Optimal initial release strategy and optimal culling with $I_0^* = 150$ in the bird extinction scenario ($b > m$) and with low levels of cats and birds as initial condition. $N_0 = 2000$, $S_0 = 195$, $K = 2000$, and $\beta_{\text{dd}} = 0.0043$

culling is at its maximum level for approximately 7 years and decreases between 7 and 9 years. Within the last year, no culling effort is required.

Finally, we return to a 4-year control period and consider the coexistence scenario. With the uncontrolled coexistence equilibrium as initial condition, Fig. 6 shows that both optimal initial release and optimal culling increase the bird population size beyond its initial value. The total cat population reduces to approximately half its initial value. In the absence of culling, the bird population declines slightly, and the total number of cats remains roughly unchanged.

5.6 Trap–infect–return (TIR) Control with OIR and Optimal Culling

We now consider a control program that combines trapping, infecting, and returning cats with an optimal initial release of infectious cats as well as optimal culling of cats. Figure 7 shows the results for the bird extinction scenario. The TIR control rate, $u(t)$ in the population lasts for 2 years at its optimal level, followed by a decrease and eventually with no control effort afterward. The bird population survives and seems to approach a stationary value at the end of the control period. The quantitative level is similar to the control program without TIR, cf. Fig. 4. The time plot of the cat population shows a slightly different pattern, because the total population size continues to decline over almost the entire control period. This may be caused by the

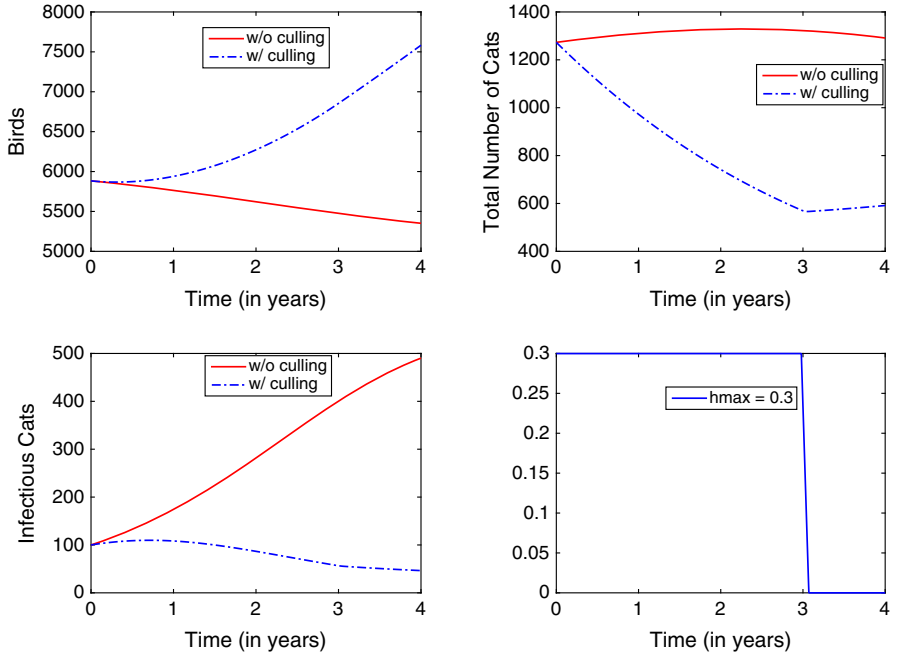

Fig. 6 Optimal initial release strategy and optimal culling with $I_0^* = 100$ in the coexistence scenario ($0 < b < m$) with the uncontrolled coexistence equilibrium as initial condition. $N_0 = 5883$, $S_0 = 1173$, $K = 2 \times 10^6$, $\beta_{dd} = 0.0011$, $m = 0.61$, and $b = 0.60$

culling that is at its maximum effort over almost the entire period (in contrast to the OIR program where culling ceases after 3 years; see Fig. 4).

In Fig. 8, the control period is extended to 10 years. The TIR control reaches its maximum level for 6 years, i.e., for four more years in comparison with the shorter control period, before it decreases and ceases. The optimal culling takes on maximum values also over a longer time period in comparison with the 4-year control horizon. As a consequence, the bird decline can be reversed to increasing numbers in comparison with Fig. 7. The total cat population is considerably reduced at the end of the control period, and the infectious cats approach extinction.

In comparison with the 10-year management program without TIR (Fig. 5), the results have improved somewhat in terms of increased bird numbers and decreased cat numbers at the end of the control period. However, there is not only extra TIR control, but also the effort in culling is longer at its maximal level due to the TIR of susceptible cats in the population.

In the absence of both TIR and culling (red solid lines in Figs. 7 and 8), the results are by definition the same as for biocontrol with OIR only (red solid lines in Figs. 4, 5).

6 Discussion

Since invasive mammals are notoriously difficult to control, we have considered combined management strategies that integrate culling and biocontrol with a host-specific

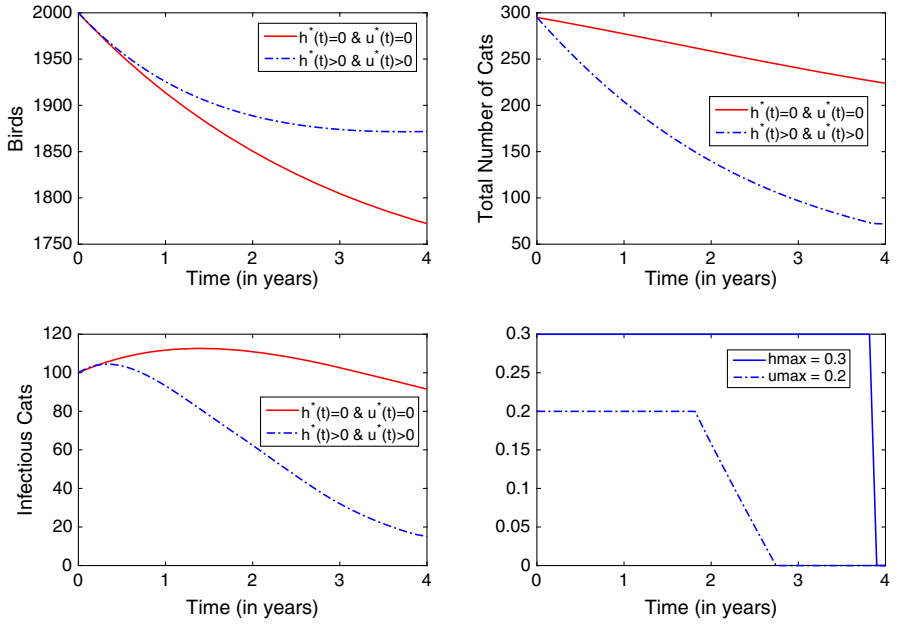

Fig. 7 Trap–infect–return (TIR) control, optimal initial release (OIR), and optimal culling with $I_0^* = 100$ in the bird extinction scenario ($b > m$) and with low levels of cats and birds as initial condition. $N_0 = 2000$, $S_0 = 195$, $K = 2000$, and $\beta_{\text{dd}} = 0.005$

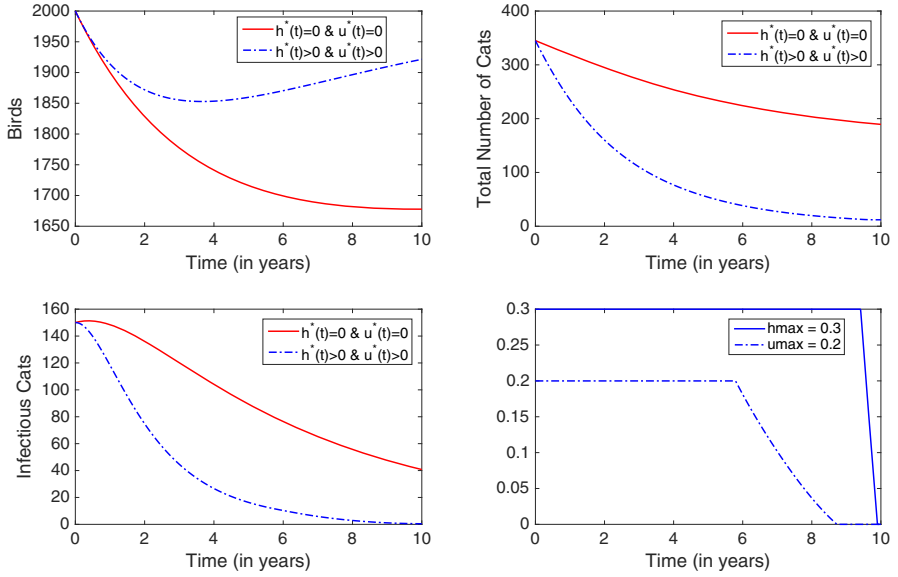

Fig. 8 Time horizon increased from $t = 4$ years to $t = 10$ years. Trap–infect–return (TIR) control, optimal initial release (OIR), and optimal culling with $I_0^* = 150$ in the bird extinction scenario ($b > m$) and with low levels of cats and birds as initial condition. Other parameter values as in Fig. 7

pathogen. Our models are motivated by invasive cats that threaten native birds on oceanic islands. A candidate biocontrol agent is feline immunodeficiency virus. There are two different ways of introducing the infectious disease. On the one hand, infected individuals can be released. This is modeled in form of an initial condition and enters the optimal control problem in form of a scalar control. On the other hand, cats can be trapped, infected, and then returned. This is modeled as a nonautonomous rate and thus enters the optimal control problem in form of a time-varying control. Culling is a time-varying control as well. Our optimal control problems therefore involve both scalar and time-dependent controls.

Overall, the results appear sensitive to a number of aspects, including the initial condition and the length of the control period. Moreover, they depend on a number of cost coefficients and weight constants. A thorough investigation of all influencing factors appears impossible, which is why we focus on the numerical simulations shown.

6.1 Effect of Combined Control

In the simulations performed, the combination of mechanical and biological control proves effective. In comparison with biocontrol only, additional optimal culling has the following two broad effects. First, it can reverse bird decline (in the absence of culling) to an increasing bird population (in the presence of culling). The population of cats is further reduced by culling. This can be observed for FIR and OIR management strategies in the coexistence scenario (Figs. 2, 6, respectively) and for 10-year control programs (Figs. 5, 8). Second, in the bird extinction scenarios, the additional optimal culling leads to the birds surviving at a higher quantitative level than in the absence of mechanical control. Similarly, the total cat numbers are controlled to a lower quantitative level than with biocontrol only. This can be observed for the management strategies of FIR/OIR (Figs. 4, 5) with optimal culling and for all three cases with TIR, OIR, and optimal culling (Figs. 7, 8).

In our simulation, we have focused on the bird extinction scenario as this seems the most pressing one. The only case in which the birds did not survive at the end of the control period was for an initial condition in which birds were at half their carrying capacity. The corresponding initial value of cats may be simply too large and cause a predation pressure that drives the birds extinct. This is also the only case in which total cat numbers were not reduced at the end of the control period in comparison with their initial value.

6.2 Impact of Initial Conditions

Clearly, the initial conditions seem to play a large role. This is not surprising, as we consider a limited time period and transients resulting from initial conditions are important. The importance of initial conditions is well known in ecological modeling (e.g., Hastings 2004), and in turn, these conditions affect the optimal control results. The simulations performed here were based on three different initial conditions. The first one corresponds to the coexistence equilibrium of the uncontrolled system. In this

case, the birds show a sharp rise in population size (Figs. 2, 6). In fact, these are the only cases where bird numbers have increased at the end of the control period.

Second, in the majority of simulations we have considered low levels of birds and cats. This initial condition showed the greatest variety in response. The bird numbers continually declined (Fig. 4); declined initially and approached a stationary value (Fig. 6); declined initially but started to rise (Figs. 5, 8). In any case, despite the control, the bird population size always decreased in comparison with their initial value.

The third initial condition is the one where birds are at half their carrying capacity. As already discussed, this is the only one leading to bird extinction despite control (Fig. 3).

6.3 Length of the Control Period

If the length of the control period increases from 4 to 10 years, the response of the bird population size to the control program is not monotonous anymore in our simulations. Instead, we observe in each case an initial decrease of bird numbers to be followed by an increase (Figs. 5, 8). Yet, the bird numbers are reduced at the end of the 10-year control period compared to their initial value.

In each of these cases, the total cat population continually declines and seems to approach a stationary value at low numbers. In fact, these are the smallest cat population sizes observed in all simulations.

In reality, the length of control programs varies considerably. Cat eradication techniques on Jarvis Island took place for less than a year (Chaphuis et al. 1994). A secondary shooting campaign on Kerguelen Islands lasted 7 years (Chaphuis et al. 1994). The full-scale implementation of hunting on Marion Island lasted 4 years, but the entire eradication program including preparation and test periods took 19 years.

6.4 Adaptive Management Programs

Rather than pursuing a certain control strategy over the entire time period, management programs in practice are often adaptive. That is, a control strategy can be applied for a short period of time and then be re-evaluated to determine how to continue with the strategy or if an alternative approach is required. To some extent, this is implicit in our optimal control problem, as the time-varying controls may rest at certain times. This can be observed in our simulations toward the end of the control periods, when optimal culling rates as well as optimal TIR rates become zero.

Management programs may also involve separate phases dedicated at monitoring population responses and testing control implementations. For instance, the 19-year cat eradication campaign on Marion Island comprised seven different phases (Bester et al. 2002).

6.5 Extinction of Infected Cats

With a control period of 10 years, the infected cat population goes extinct after about 7–9 years. In the case of OIR control, this seems to coincide with the point of time where culling effort becomes zero (Fig. 5). In the case of TIR control, it seems to happen when the TIR rate vanishes (Fig. 8). Therefore, once the infectious cats are extinct, the biocontrol is removed from the system and the only remaining control is culling.

The disappearance of FIV infection is noteworthy as one advantage ascribed to biocontrol is its self-persistence over a longer period of time (Courchamp et al. 2003). However, it is also well known that biocontrol becomes less effective after some time, which is why it may be backed up or followed by mechanical control (e.g., Dobson 1988; Bester et al. 2002).

The extinction of infectious cats is also noteworthy because it seems to be driven by the other form of control, namely culling. Since the goal was the combination of different control types, the disease extinction seems undesirable. Yet, it appears that this combination is effective as the extinction of infectious cats precedes the eventual cat eradication if time went on, see Figs. 5 and 8. The joint interplay of infection and culling has reduced the cat population to small numbers, but the disease does not seem effective in cat eradication, which is why this would remain the job of culling.

In these simulations, the disease transmission is density dependent. Therefore, infections vanish at small host population sizes, which is why cat eradication is not possible due to the disease only. The results might be different for frequency-dependent transmission, because disease transmission is ongoing even in small host populations. However, as this may be considered an artifact as the cats approach eradication, we have considered only the more conservative case of density-dependent transmission in our simulations.

6.6 Eradicating the Last Remaining Individuals

The preceding discussion points to the importance of removing the last few individuals of the cat population if the aim were eradication. There may be good reasons to control but preserve the cat population (e.g., Courchamp et al. 1999; Rayner et al. 2007; Russell et al. 2009), and the definition of our objective functional actually includes only the reduction in the cat population.

Nevertheless, it is worthwhile discussing the difficulty of removing the last individuals, because the effort in doing so may increase significantly or even drastically. We could include these considerations in our optimal control problem by defining corresponding cost terms in the objective functional. For instance, the cost of culling and/or TIR control could increase when cat numbers become small.

6.7 TIR Control

The TIR control program involves enormous expenditures as cats need to be trapped, infected, and returned. In our simulations, this occurs in addition to the initial release

of infected individuals and the culling of cats. Considering the extra expenditures, the effect of TIR in terms of bird and cat population size seems rather disappointing, as there is only little quantitative change (compare Fig. 5 with Fig. 8). In particular, it seems surprising that culling at maximal effort lasts longer in the presence of TIR. These observations may be caused by the particular choice of cost coefficients and could change if their values were varied.

6.8 Model Formulation and Analysis

We formulated a predator–prey model that includes culling and disease-related control, with the objective of reducing the cat population size and raising the population of birds. With a specialist predator model which assumes that cats depend solely on births for survival, cats exponentially decline in the absence of birds. This is not realistic, since cats are opportunistic predators, switching prey according to their spatial and temporal availability. Thus, we incorporated a birth term for the cats that models alternative food sources. Provided this birth term is greater than the death term, the cats will grow exponentially in the absence of birds and disease. In the presence of disease (and no birds), the cats are shown to approach a stable endemic equilibrium. This is an example of an infectious disease regulating unbounded host population growth.

We obtained the basic and demographic reproduction numbers for cats in the model and established conditions for the existence of various steady states. Their stability was studied as well, which served later on as reference scenarios for the optimal control simulations.

6.9 Conclusions

The combination of culling and biocontrol in an integrated management approach is a recurrent topic pertinent to invasive mammals and the harm they cause. Yet, there is little modeling insight. Here, we have studied an optimal control problem that brings both mechanical and biological control together.

The results appear to be influenced by a large number of factors. In addition to eco-epidemiological parameters (which determine the reference scenario), there are parameters related to the cost of control, their weighting in the objective functional, the length of the control period, and the initial condition. If the initial population of pest species is unknown, then optimal control tools can give an appropriate rule of thumb on how to proceed.

The impact of the management programs on the bird population seems to be mainly driven by the initial condition, while the near eradication of the cats seems to require a prolonged control period. More detailed conclusions and management recommendations will necessitate improved knowledge of parameter values or a systematic analysis of their uncertainty.

The current study has identified a number of factors that seem important in this respect and deserve further study. Moreover, our problem formulation and analysis

have provided a means to study optimal control with both scalar (number of infected cats released) and time-varying (culling and TIR rate) controls.

Acknowledgements This work was partially supported by the National Institute for Mathematical and Biological Synthesis, an Institute sponsored by the National Science Foundation through NSF Award #DBI-1300426, with additional support from The University of Tennessee, Knoxville. Lenhart's research was also partially supported by the University of Tennessee Boyd Center for Business and Economic Research.

References

Allen LJS (2007) An introduction to mathematical biology. Pearson Prentice Hall, New Jersey

Anderson R, May RM (1986) The invasion, persistence and spread of infectious diseases within animal and plant communities. Phil Trans R Soc Lond 314:533–570

Bate AM, Hilker FM (2013) Complex dynamics in an eco-epidemiological model. Bull Math Biol 75:2059–2078

Bester MN, Bloomer JP, van Aarde RJ, Erasmus BH, van Rensburg PJJ, Skinner JD, Howell PG, Naude TW (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

Chaphuis J, Bousses P, Barnaud G (1994) Alien mammals, impact and management in the French subantarctic islands. Biol Conserv 67:97–104

Chaphuis JL (1995) Alien mammals in the French Subantarctic Islands. In: Dingwall PR (ed) Progress in conservation of the subantarctic islands, conservation of the southern polar region, vol 2. The World Conservation Union, Paimpont, pp 127–132

Cleaveland S, Thirgood S, Laurenson K (1999) Pathogens as allies in island conservation? Trends Ecol Evol 14:83–84

Courchamp F, Pontier P (1994) Feline immunodeficiency virus: an epidemiological review. C R Acad Sci Paris Sér III Sci Vie 317:1123–1134

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 (1995) Population dynamics of feline immunodeficiency virus within cat populations. J Theor Biol 175:553–560

Courchamp F, Yoccoz NG, Artois M, Pontier D (1998) At-risk individuals in feline immunodeficiency virus epidemiology: evidence from multivariate approach in a natural population of domestic cats (feline catus). Epidemiol Infect 121:227–236

Courchamp F, Langlais M, Sugihara G (1999) Cats protecting birds: modelling the mesopredator release effect. J Anim Ecol 68:282–292

Courchamp F, Say L, Pontier D (2000) Transmission of feline immunodeficiency virus in a population of cats. Wildl Res 27:227–236

Courchamp F, Chapuis JL, Pascal M (2003) Mammal invaders on islands: impact, control and control impact. Biol Rev 78:347–383

Diamond J (1989) Overview of recent extinctions. In: Western D, Pearl MC (eds) Conservation for the twenty-first century. Oxford University Press, New York, pp 37–41

Diekmann O, Heesterbeek JAP (2000) Mathematical epidemiology of infectious diseases: model building, analysis and interpretation. Wiley, New York

Diekmann O, Heesterbeek JAP, Metz JAJ (1990) On the definition and the computation of the basic reproduction ratio in models for infectious diseases in heterogeneous populations. J Math Biol 28:365–382

Diekmann O, Dietz K, Heesterbeek JAP (1991) The basic reproduction ratio for sexually transmitted diseases: theoretical considerations. Math Biosci 107:325–339

Diekmann O, Heesterbeek JAP, Robert MG (2010) The construction of next-generation matrices for compartmental epidemic models. J R Soc Interface 9:873–885

Diekmann O, Heesterbeek H, Britton T (2013) Mathematical tools for understanding infectious disease dynamics. Princeton University Press, New Jersey

Dobson AP (1988) Restoring island ecosystems: the potential of parasites to control introduced mammals. Conserv Biol 3:31–38

Driessche PVD, Watmough J (2002) Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. Math Biosci 180:29–48
Fan M, Kuang Y, Feng Z (2005) Cats protecting birds revisited. Bull Math Biol 167:1081–1106
Fitzgerald BM, Turner DC (2000) Hunting behaviour of domestic cats and their impact on prey populations. In: Turner DC, Bateson P (eds) The domestic cat: the biology of its behaviour, 2nd edn. Cambridge University Press, Cambridge, pp 151–175
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
Gaff H, Schaefer E (2009) Optimal control applied to vaccination and treatment strategies for various epidemiological models. Math Biosci Eng 6:469–492
Hackbusch W (1978) A numerical method for solving parabolic equations with opposite orientations. Computing 20:229–240
Hadeler KP, Freedman HI (1989) Predator–prey populations with parasite infection. J Math Biol 27:609–631
Hartmann K (1998) Feline immunodeficiency virus infection: an overview. Vet J 155:123–137
Hastings A (2004) Transients: the key to long-term ecological understanding? Trends Ecol Evol 19:39–45
Hilker FM, Schmitz K (2008) Disease-induced stabilization of predator–prey oscillations. J Theor Biol 255:299–306
Kooi BW, van Voorn GAK, pada Das K (2011) Stabilization and complex dynamics in a predator–prey model with predator suffering from an infectious disease. Ecol Complex 8:113–122
Kot M (2001) Elements of mathematical ecology. Cambridge University Press, Cambridge
Lavers JL, Wilcox C, Donlan CJ (2010) Bird demographic responses to predator removal programs. Biol Invasions 12:3839–3859
Lenhart S, Workman JT (2007) Optimal control applied to biological models. Chapman and Hall, Boca Raton
Loyd KA, Miller CA (2010) Influence of demographics, experience and value orientation on preferences for lethal management of feral cats. Hum Dimens Wildl 15:262–273
Moors PJ, Atkinson IAE (1984) Predation on seabirds by introduced animals, and factors affecting its severity. In: Croxall PJ, Evans PGH, Schreiber RW (eds) Status and conservation of the world's seabirds, vol 2. ICBP Technical Publications, Cambridge, pp 667–690
Murray JD (1993) Mathematical biology, 2nd edn. Springer, Heidelberg
Nogales M, Martin A, Tershey BR, Donlan CJ, Veitch D, Puerta N, Wood B, Alonso J (2004) A review of feral cat eradication on islands. Conserv Biol 18:310–319
Nutter FB, Levine JF, Stoskopf MK (2004) Reproductive capacity of free-roaming domestic cats and kitten survival rate. J Am Vet Med Assoc 225:1399–1402
Oliveira NM, Hilker FM (2010) Modeling disease introduction as biological control of invasive predators to preserve endangered prey. Bull Math Biol 72:444–468
Pascal M (1980) Structure et dynamique la population de chats harets de l'archipel del kerguelen. Mammalia 44:161–182
Pimm SL (1982) Food webs. Chapman and Hall, London
Pontryagin Boltyanskii V, Gamkrelize R, Mishchenko E (1967) The mathematical theory of optimal processes. Wiley, New York
Rauzon MJ (1985) Feral cats on Jarvis Island: their effects and their eradication. Atoll Res Bull 282:1–32
Rayner MJ, Hauber ME, Imber MJ, Stamp RK, Clout MN (2007) Spatial heterogeneity of mesopredator release within an oceanic island system. Proc Natl Acad Sci USA 104:20,862–20,865
Robertson SA (2008) A review of feral cat control. J Feline Med Surg 10:366–375
Rounsevel DE, Copson GR (1982) Growth rate and recovery of a king penguin aptenodytes patagonicus population after exploitation. Aust Wildl Res 9:519–525
Russell JC, Lecomte V, Dumont Y, Le Corre M (2009) Intraguild predation and mesopredator release effect on long-lived prey. Ecol Model 220:1098–1104
van Aarde RJ (1980) The diet and feeding behaviour of feral cats, *Felis catus* at Marion Island. S Afr J Wild Res 10(3/4):123–128
van Rensburg PJJ, Bester MN (1988) The effect of cat, *Felis catus*, predation on three breeding Procellariidae species on Marion Island. S Afr J Zool 23(4):301–305
Xiao Y, Van Den Bosch F (2003) The dynamics of an eco-epidemic model with biological control. Ecol Model 168:203–214