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Harvesting can increase severity of wildlife disease epidemics

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Theoretical studies of wildlife population dynamics have proved insightful for sustainable management, where the principal aim is to maximize short-term yield, without risking population extinction. Surprisingly, infectious diseases have not been accounted for in harvest models, which is a major oversight because the consequences of parasites for host population dynamics are well-established. Here, we present a simple general model for a host species subject to density dependent reproduction and seasonal demography. We assume this host species is subject to infection by a strongly immunizing, directly transmitted pathogen. In this context, we show that the interaction between density dependent effects and harvesting can substantially increase both disease prevalence and the absolute number of infectious individuals. This effect clearly increases the risk of cross-species disease transmission into domestic and livestock populations. In addition, if the disease is associated with a risk of mortality, then the synergistic interaction between hunting and disease-induced death can increase the probability of host population extinction.

Keywords: wildlife disease dynamics; harvesting; seasonality; density-dependence; compensation; emerging infectious diseases

1. INTRODUCTION

The consequences of harvesting on the dynamics of wild animal populations have been studied quite extensively since the first development of optimal harvest theory (see, e.g. Getz & Haight 1989). However, it is only recently that the interaction between harvesting, density dependent population processes and seasonality have been studied in any detail (Kokko & Lindström 1998; Jonzén & Lundberg 1999; Boyce *et al.* 1999; Xu *et al.* 2005). In temperate regions, births of most wild animal species are seasonal and each year many more individuals are produced than will be able to survive until the following year, because of limited resources. This annual surplus, referred to as 'doomed surplus' by Errington (1934), allows the population to tolerate additional mortality. It has been demonstrated, both theoretically and in the field (Kokko & Lindström 1998; Jonzén & Lundberg 1999; Boyce *et al.* 1999; Xu *et al.* 2005 and references therein), that harvesting can thus be partially, fully, or even over-compensated by density-dependence. Full-compensation means that the population size at a given season of the year (e.g. post-breeding season) is not affected by hunting, whereas over-compensation means that the population size is actually increased by hunting (Murton *et al.* 1974). Although, the consequences of harvesting on the dynamics of wildlife populations have been studied in detail, no investigations, to our knowledge, have been carried out on the consequences of harvesting on the dynamics of the diseases that thrive in those wildlife populations (McCallum *et al.* 2005). This is all the more regrettable since epidemiological studies have demonstrated how the demography of a host population can influence the frequency and intensity of recurrent epidemics (Fine & Clarkson 1982; Anderson & May 1991; Keeling & Grenfell 1997; Rohani *et al.* 1999; Earn *et al.* 2000; Grenfell *et al.* 2001; Jansen *et al.* 2003; Davis *et al.* 2004).

The only hints that hunting could potentially disrupt the dynamics of wildlife diseases ironically come from attempts to control these pathogens. Indeed, the most common means of fighting wildlife diseases is the old age practice of culling animal reservoirs. Such policies, however, have been shown to be inefficient in numerous situations. Recent examples include the inability to eradicate tuberculosis in badger populations in the UK despite widespread culling (Donnelly *et al.* 2003) and rabies in fox populations in Europe (Woodroffe *et al.* 2004). It has been suggested that social and physiological plasticities of such animal populations confer them with a remarkable capacity to recover from control and that such a response to culling could actually increase the supply of susceptibles to the disease (Woodroffe *et al.* 2004). However, such potential perverse effects have not been investigated within a quantitative framework.

In this paper, we propose a simple general continuous-time model that accounts for the dynamics of a directly transmitted infectious disease in a harvested population. The disease confers permanent immunity to its host after recovery. The host population is naturally regulated by density dependent demographic processes, and births are assumed to be seasonal. We show that harvesting can increase disease prevalence and mortality, causing potential serious threats in terms of wildlife conservation. Moreover, the interaction between seasonality and density-dependence can increase the host population birth rate. In such situations, not only the prevalence but also the absolute number of infectious individuals is increased. Special attention is given to the effect of the timing of

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the harvest season on the dynamics of both the host population and the disease. These results are discussed in terms of wildlife management and prevention of epizootics.

2. THE MODEL

The general framework we introduce here is intended to mimic the dynamics of a directly transmitted microparasitic disease in a temperate population of game with a strong density dependent demography (intended to mimic, for example, wild boar or waterfowl). The general structure of the model, inspired from the classical *SEIR* framework (Anderson & May 1991), reads

$$\frac{\mathrm{d}S}{\mathrm{d}t} = \varphi(t)N(t)B(N) - [D_S(N) + \psi(t)q_S H_S + \lambda(t)]S(t), \tag{2.1}$$

$$\frac{\mathrm{d}E}{\mathrm{d}t} = \lambda(t)S(t) - [D_E(N) + \psi(t)q_E H_E + \sigma]E(t), \tag{2.2}$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \sigma E(t) - [D_I(N) + \psi(t)q_I H_I + \gamma + v]I(t), \tag{2.3}$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = \gamma I(t) - [D_R(N) + \psi(t)q_R H_R]R(t), \tag{2.4}$$

where the state variables S, E, I and R represent the numbers of susceptible, exposed (infected but not yet infectious), infectious, and recovered individuals, respectively. The term $B(N)$ is the *per capita* density-dependent birth rate, and $D_i(N)$ is the *per capita* density-dependent natural death rate in compartment i ($i \in \{S, E, I, R\}$). The size of the total host population is given by $N(t) = S(t) + E(t) + I(t) + R(t)$, and may vary. Parameters q_i ($0 \leq q_i \leq 1$) and H_i are, respectively, the catchability and the harvest effort in compartment i, where $i \in \{S, E, I, R\}$ (Kot 2001). Newborns are assumed susceptible and their exposure to disease is determined by the force of infection $\lambda(t)$ (see equation (2.7)). Recovereds stay immune for life. We assume that the mean durations of the exposed and infectious periods are $1/\sigma$ and $1/\gamma$, respectively. The disease can induce additional mortality on the infectious individuals, with a rate v. Seasonality in births (Macdonald 1984) is accounted for by the function $\varphi(t)$. In wildlife management, the most common practice is to permit hunting only during a specified season, usually short compared to the rest of the year (Xu *et al.* 2005). This seasonality in harvesting is rendered by the function $\psi(t)$.

While our model is deliberately kept as generic as possible, the range of the parameter values used in the numerical analyses are intended to correspond to a classical swine fever-like disease in a wild boar-like host population.

(a) *Density-dependence*

In numerous game species, including large herbivores, it is well documented that both birth and death are density-dependent (Gaillard *et al.* 2000). For simplicity, we assume that these two rates are linearly related to population density such that

$$B(N) = b - \theta N(t-\tau), \tag{2.5}$$

$$D_i(N) = d_i + \mu_i N(t), \tag{2.6}$$

where b is the maximum per-capita birth rate, d_i is the minimum per-capita death rate in compartment i ($i \in \{S, E, I, R\}$), and θ and μ_i determine the strength of the density-dependence in birth and death rates, in compartment i ($i \in \{S, E, I, R\}$). The time-delay τ reflects the duration of gestation between the mating and birth seasons.

(b) *Frequency-dependence*

For directly transmitted diseases, the force of infection can be approximated as proportional to the frequency of infectious individuals (Anderson & May 1991; McCallum *et al.* 2001):

$$\lambda(t) = \beta \frac{I(t)}{N(t)}, \tag{2.7}$$

where the parameter β is the product of the contact rate between two individuals and the probability of disease transmission upon a contact between an infectious and a susceptible individuals. In §5, we speculate on the consequences of alternative formulations of $\lambda(t)$.

(c) *Seasonalities*

The two seasonality functions—$\varphi(t)$ affects the birth rate and $\psi(t)$ influences harvesting—are modelled by a simple periodic square function:

$$\varphi(t) = \mathbb{1}_{S_B}(t \bmod T), \tag{2.8}$$

$$\psi(t) = \mathbb{1}_{S_H}(t \bmod T), \tag{2.9}$$

where $T = 1$ yr is the period of the seasonal functions, and $S_B = [b_1, b_2]$ and $S_H = [h_1, h_2]$ are the periods of time corresponding to the birth and harvest seasons, respectively. The beginning of a year is defined arbitrarily and its specific definition has no influence on the model predictions.

3. PROPERTIES AT EQUILIBRIUM

In this section, we are interested in the total host population size N^* and the disease prevalence I^*/N^* at equilibrium, as functions of the harvest effort H. To ease analytical tractability, we make here some simplifying assumptions on the model parameters. We use the same density-dependence d for the per-capita birth and death rates: $d = \theta = \mu_S = \mu_E = \mu_I = \mu_R$. The minimum death rates are null ($d_S = d_E = d_I = d_R = 0$), and the maximum birth rate b is re-defined such that the carrying capacity K of the host population in the absence of harvesting and disease-induced mortality is constant:

$$b \equiv 2Kd. \tag{3.1}$$

These parameter choices and this scaling of the maximum birth rate allows one to isolate and investigate the effect of the strength of the density-dependence on the model outcomes. We consider that harvest effort and host catchability are not specific to any clinical status of the host: $H_S = H_E = H_I = H_R = H$ and $q_S = q_E = q_I = q_R = q$. Finally, we do not consider any of the seasonality functions ($\varphi(t) = \psi(t) = 1, \forall t$). The results of this section are derived analytically and compared to numerical solutions in figures 1 and 2, for which the following parameter values are used: $K = 10^5$ ind, $q = 0.5$, $\beta = 2000\ \mathrm{yr}^{-1}$, $\sigma = 1/8\ \mathrm{d}^{-1}$ and $\gamma = 1/5\ \mathrm{d}^{-1}$.

First, consider dynamics in the absence of disease-induced mortality ($v = 0$). Here, the effect of harvest effort

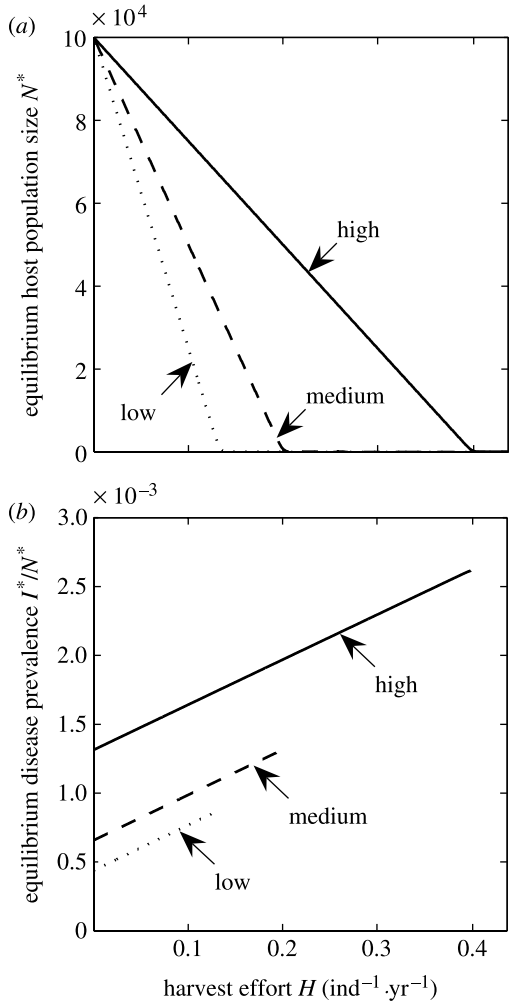

Figure 1. Effect of the harvest effort H on (*a*) the host population size N^* and (*b*) the disease prevalence I^*/N^* at equilibrium, for different strength of the density-dependence: high ($d \simeq 2.7397 \times 10^{-9}$, full line), medium ($d \simeq 1.3699 \times 10^{-9}$, dashed line) and low ($d \simeq 9.1324 \times 10^{-10}$, dotted line). The lines show numerical solutions which are exactly the same as the analytical solutions of equation (3.3). The minimum per-capita death rates are null ($d_S = d_E = d_I = d_R = 0$) and the maximum birth rate b is adjusted so that the carrying capacity in the absence of harvesting is $K = 10^5$ ind, whatever the strength of the density-dependence (see equation (3.1) in the main text). We thus have $b = 0.20$ ind^{-1} yr^{-1} (full line), $b = 0.10$ ind^{-1} yr^{-1} (dashed line) and $b \simeq 0.04$ ind^{-1} yr^{-1} (dotted line). Other parameter values are $q_S = q_E = q_I = q_R = 0.5$, $\beta = 2000$ yr^{-1}, $\sigma = 1/8$ d^{-1}, $\gamma = 1/5$ d^{-1} and $v = 0$. The value of H varies from 0 to 0.4745, every 365×10^{-6} and, for each H value, the equilibrium values of the dynamics are sampled on the 137 yr that followed a burn-out period of 1370 yr.

H on the total population size $N(t) = N(t-\tau)$ can be studied by summing equations (2.1)–(2.4), which gives:

$$\frac{\mathrm{d}N}{\mathrm{d}t} = (b - qH)N - 2\mathrm{d}N^2. \tag{3.2}$$

For sufficiently small values of the harvest effort H, the non-trivial equilibrium population size N^* can then be expressed by setting equation (3.2) to 0, which yields

$$N^* = K - \frac{q}{2d}H. \tag{3.3}$$

This result exemplifies the tendency of the natural density-dependence d of the host population to compensate the harvest pressure H. This is consistent with the numerical solutions of figure 1*a* where, for a given harvest effort H, the higher the density-dependence d, the higher the equilibrium population size N^*.

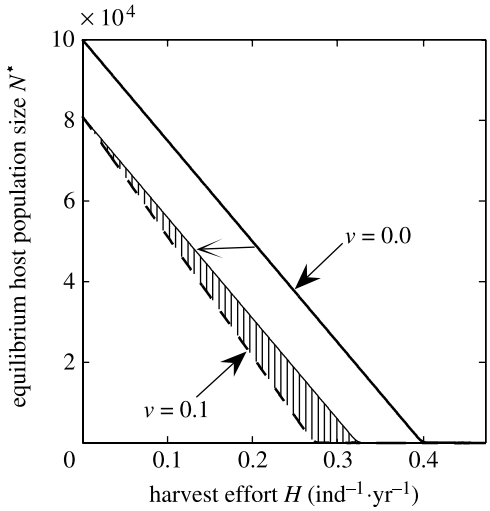

Figure 2. Effect of the harvest effort H on the host population size $N^\star$ in presence of disease-induced mortality rates v. The thick full line corresponds to the full line of figure 1(*a*) (i.e. $d \simeq 2.7397 \times 10^{-9}$ and $v = 0.0$). The dashed line is the same but with $v = 0.1$. The lines show numerical solutions which are undistinguishable from the analytical approximations of equation (3.9). The hatched area corresponds to the mortality due to the synergistic interaction between harvesting and disease-induced mortalities (see fourth term in the r.h.s. of equation (3.9)). Other parameter values are as in figure 1. The value of H varies from 0 to 0.4745, every 365×10^{-6} and, for each H value, the equilibrium values of the dynamics are sampled on the 137 yr that followed a burn-out period of 1370 yr.

The effect of harvest effort H on the equilibrium prevalence I^*/N^* can be derived by replacing $N(t)$ by N^* (equation (3.3)) in equations (2.1)–(2.4), equating them to zero, solving the system to get I^*, and dividing it by N^*. A linear approximation of the disease prevalence at equilibrium then reads as (see appendix A for approximation details):

$$\frac{I^*}{N^*} \simeq K\frac{d}{\gamma} + \frac{q}{2\gamma}H + o(H). \tag{3.4}$$

Not surprisingly, the equilibrium disease prevalence depends on the mean duration $1/\gamma$ of the infectious period and the strength d of the density-dependence in the host population. For a fixed carrying capacity, as assumed here, the strength of density-dependence reflects the population turn-over rate, and thus the rate of susceptible recruitment, known as a major determinant of the dynamics of strongly immunizing diseases (Earn *et al.* 2000). Interestingly, here, density-dependence d does not interact with the harvest effort H. This approximate result of equation (3.4) is consistent with the numerical solutions of figure 1*b*, where only the y-intercepts, and not the slopes, depend on the value of the density-dependence d.

When the harvest effort H increases, the equilibrium population size N^* decreases (equation (3.3), figure 1*a*) and the equilibrium disease prevalence I^*/N^* increases (equation (3.4), figure 1*b*). Above a harvest threshold $H^\star$, the equilibrium population size N^* reaches zero (figure 1*a*) and the equilibrium disease prevalence I^*/N^* tends

towards its maximum value $(I^*/N^*)^{\max}$ (figure 1*b*). Replacing N^* by zero in equation (3.3), we can express the threshold harvest value $H^\star$ which depends on the density-dependence d:

$$H^\star = 2K\frac{d}{q}. \tag{3.5}$$

Substituting H by $H^\star$ in equation (3.4), we get the maximum value of the disease prevalence at equilibrium:

$$\lim_{H\to H^\star}\left(\frac{I^*}{N^*}\right) = \left(\frac{I^*}{N^*}\right)^{\max} = 2K\frac{d}{\gamma}. \tag{3.6}$$

Interestingly, this means that the harvest pressure H can make the equilibrium disease prevalence I^*/N^* vary between its value in the absence of hunting (i.e. Kd/γ, see first term in the right-hand side of equation (3.4)) and twice this value (i.e. $2Kd/\gamma$, see equation (3.5)). This approximate result is consistent with the numerical solutions of figure 1*b*, where the maximum values are about twice the values of the y-intercepts.

Now, consider the possibility of disease-induced mortality ($v>0$). Adding a constant mortality rate v in equation (3.2) would decrease the equilibrium population size (N^*, equation (3.3)) by $v/(2d)$ individuals. As disease-induced mortality actually affects only the infectious individuals, this number should be multiplied by the equilibrium disease prevalence I^*/N^* (equation (3.4)) to yield the expression of the total number D^* of individuals dying from the disease at equilibrium:

$$D^* = \frac{v}{2d}\frac{I^*}{N^*} \simeq K\frac{v}{2\gamma} + \frac{qv}{4d\gamma}H + o(H). \tag{3.7}$$

We can then express the equilibrium population size $N^\star$ in the presence of disease-induced mortality as

$$N^\star = N^* - D^* \simeq K\left(1-\frac{v}{2\gamma}\right) - \frac{q}{2d}\left(1+\frac{v}{2\gamma}\right)H + o(H). \tag{3.8}$$

Note that when $v=0$ in equation (3.8), we obtain the exact result of equation (3.3). Equation (3.8) shows that, in the absence of harvesting, disease-induced mortality decreases the equilibrium population size by a factor $v/(2\gamma)$ and, in the presence of harvesting, disease-induced mortality mitigates the compensatory effect of density-dependence by a factor $1+v/(2\gamma)$. Developing equation (3.8) lists all the causes of mortality:

$$N^\star \simeq K - \frac{K}{2\gamma}v - \frac{q}{2d}H - \frac{q}{4\gamma d}vH + o(H). \tag{3.9}$$

Thus, $Kv/(2\gamma)$ individuals die from the disease, $qH/(2d)$ individuals die from hunting, and $qvH/(4\gamma d)$ individuals die from the synergistic interaction between disease-induced mortality and harvesting. Natural mortality is included in the expression of the carrying capacity K. This approximate result is consistent with the numerical solutions of figure 2 which shows the equilibrium population size without disease-induced mortality (full line, which is the same as the full line in figure 1*a*), and with disease-induced mortality ($v=0.1$, dashed line). The hatched area corresponds to the disease-induced mortality increased by harvesting (fourth term in the r.h.s. of equation (3.9)). Taking the ratio of the third and fourth terms of the r.h.s. of equation (3.9), we come up with the conclusion that when X individuals are harvested, an additional $(v/2\gamma)X$ individuals die from harvest-induced increase in disease-induced mortality. Not surprisingly, the longer the infectious period (i.e. the lower γ), the stronger the effect of disease-induced mortality. Interestingly, the additional proportion of individuals dying from the disease as a side effect of harvesting ($v/(2\gamma)$) does not depend on the strength of the density-dependence d in the host population.

4. DYNAMICAL PROPERTIES

In §3, we studied the static properties of the model of equations (2.1)–(2.4), and investigated the potential of the host population density-dependence d to partially compensate for the harvest pressure H, both in the absence and in the presence of disease-induced mortality v. In this section, we focus on the dynamics of the model to explore how the interaction between density-dependence and seasonality can alter our conclusions. Indeed, whereas, at equilibrium, harvest toll on the host population cannot be more than partially compensated by density-dependent population processes, it has been shown that the interaction between density-dependence and seasonality has the potential to fully compensate and even over-compensate harvesting (Kokko & Lindström 1998; Boyce *et al.* 1999; Jonzén & Lundberg 1999; Xu *et al.* 2005 and references therein). We thus reintroduce here the seasonality functions $\varphi(t)$ (birth seasonality) and $\psi(t)$ (harvest seasonality). The introduction of these time-dependent terms renders the system analytically intractable, and we will rely, in the following, only on numerical solutions. For simplicity, numerical solutions are sought in the absence of disease-induced additional mortality ($v=0$). The effect of disease-induced additional mortality on the results is then discussed.

It has been documented for a wide variety of vertebrate species that the density-dependence on the per-capita natural mortality rate is much stronger in the younger age classes than in the adults, for which it is often negligible (Ekman 1984; Clutton-Brock *et al.* 1985, 1997; Skogland 1985; Hudson 1992; Gaillard *et al.* 2000). In consequence, we will assume, in the following, a constant mortality rate for the recovered class of individuals: $d_R>0$ and $\mu_R=0$. Indeed, in the case where the age at maturity (and/or the force of infection λ) is low, and the immunity lifelong, we can reasonably consider that all recovered individuals are adults (Anderson & May 1991).

Let us focus first on the dynamics of the total host population $N(t)$. Figure 3*a* shows 2 years of the total host population stationary dynamics in the presence (full line) and in the absence (dashed line) of harvesting. The harvest season (noted 'H' above the graph) begins 5 days after the end of the birth season (which corresponds to the increase in the host population size), and lasts for 15 days. In the absence of harvesting, the host population dynamics are clearly annual, driven by the birth pulses. In the presence of harvesting, hunting reduces quickly and dramatically the number of individuals in the population, and the consequences are twofold. First, the number $N(t-\tau)$ of individuals during the mating season is decreased, which tends to increase the future per-capita birth rate B (see density-dependent response in equation (2.5)). Second, the per-capita death rate is decreased

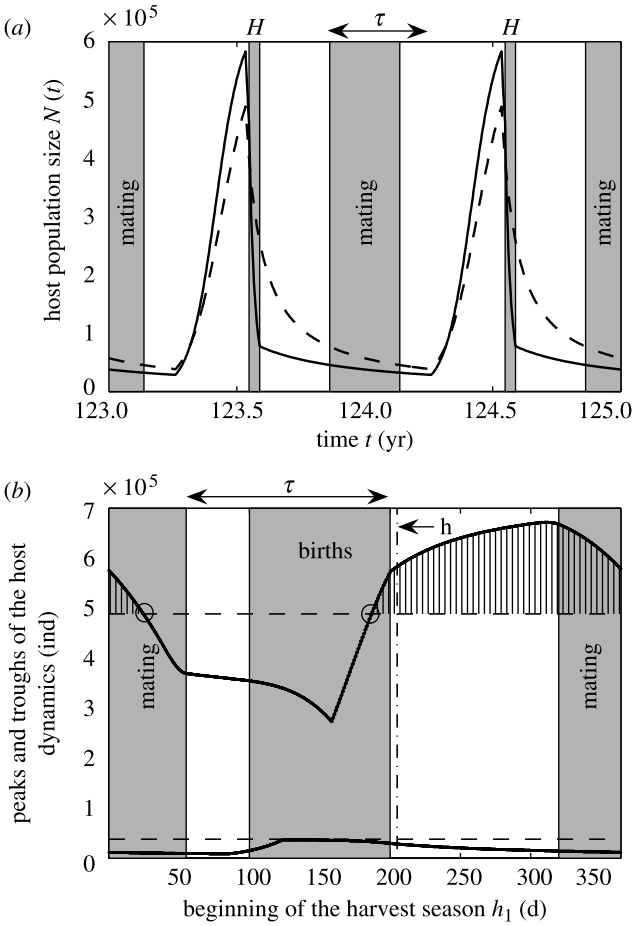

Figure 3. Effect of harvesting on the total host population size $N(t)$. (*a*) The figure shows numerical solutions of 2 years of the model's dynamics, after stationary dynamics has been reached. The periods of mating are represented on the graph. The harvest periods are indicated by a 'H' above the graph, and the duration τ of the gestation (between matings and births) is represented by the length of the double arrow above the graph. The dashed curve represents the total host population size $N(t)$ in the absence of harvesting ($H=0$) and the full line curve represents the dynamics of the total host population size $N(t)$ in the presence of harvesting ($H=0.2$). The harvest season begins at $h_1=205$ d, and ends at $h_2=h_1+15$ d. (*b*) The figure shows the peaks and the troughs of the dynamics as a function of the beginning time h_1 of the harvest season. As for (*a*), $h_2=h_1+15$ d. The thick full lines represent the peak and trough values in the presence of harvesting ($H=0.2$) and the horizontal dashed lines represent the peak and trough values in absence of harvesting ($H=0$). The mating and birth seasons are represented on the graph and the duration τ of the gestation is indicated by the length of the double arrow above the graph. The vertical dash-dotted line arrowed by a 'h' represents the value of h_1 used in (*a*). Circles show cases of full-compensation and hatched areas highlight cases of over-compensation. 100 yr of the model's dynamics have been numerically solved to reached stationarity, and then the peak and trough values have been sampled on the following 35 yr of the model's dynamics. The value of h_1 varies from 0 to 364.9 d, every 0.1 d. For (*a*) and (*b*), other parameter values are $K=10^5$ ind, $b\simeq 0.1461$ ind^{-1} d^{-1}, $d_S=d_E=d_I=d_R=0$, $\theta\simeq 4.5662\times 10^{-7}$, $\mu_S=\mu_E=\mu_I=\mu_R\simeq 9.1324\times 10^{-8}$, $\tau=145$ d, $b_1=100$ d, $b_2=200$ d, $q_S=q_E=q_I=q_R=0.5$, $\beta=365$ yr^{-1}, $\sigma=1/8$ d^{-1}, $\gamma=1/5$ d^{-1} and $v=0$.

(see density-dependent response in equation (2.6)), which keeps the population size N before the birth season close to its size in the absence of harvesting. The combination of the two effects—increased per-capita birth rate B and unchanged population size N before the birth season—is responsible for an increase in the number $B\times N$ of individuals born during the birth season. This is a case of over-compensation where the effect of hunting is, paradoxically, to increase the host population size after the birth season (compare the two curves on figure 3*a*).

Since reproduction is seasonal, the exact timing of the harvest season is expected to be a strong determinant of the magnitude of compensation (Kokko & Lindström 1998). Figure 3*b* explores the effect of harvest timing h_1 on the peak and trough values of the host population dynamics. The thick full lines represent the peak and trough values in the presence of hunting and should be compared with the horizontal dashed lines, which represent the peak and trough values in the absence of hunting. Consider first the peak values. The intersection between the thick full and the horizontal dashed lines (see circles on figure 3*b*) are cases of full-compensation, where harvesting has no effect on the host population size at the end of the birth season. Whenever the thick full line is above the horizontal dashed line, there is over-compensation, where harvesting actually increases the host population size at the end of the birth season (hatched area on figure 3*b*). Lastly, when the thick full line is below the horizontal dashed line, partial compensation cannot be distinguished from pure additive effects. Thus, figure 3*b* shows that over-compensation occurs whenever the hunting season is between the birth season and the next mating season. In this period of time, the later the hunting season, the stronger the over-compensation. Over-compensation decreases sharply as soon as the hunting season occurs during the mating season. Finally, note that the timing of the hunting season has also an effect on the trough values of the dynamics, even if lower than its effect on the peak values. In particular, it appears that hunting during the mating season could potentially increase the probability of extinction of the host population.

We have shown in §3 that one side-effect of harvesting is an increase in the disease prevalence. Moreover, as harvesting can also lead to over-compensation on the total host population size (see figure 3), we can expect hunting to increase, in some situations, the absolute number of infectives in the host population. Figure 4*a* shows an annual epidemic in the presence (full line) and in the absence (dashed line) of harvesting. Again, the dynamics of the disease, both in the presence and in the absence of hunting, are strongly annual and driven by the birth pulses. Comparing the two curves, it appears that hunting can alter both the amplitude and the timing of the epidemics. The effects of harvest timing h_1 on the amplitude of the epidemics are explored systematically in figure 4*b*.

Figure 4*b* has been drawn in a similar way as figure 3*b* but considers the dynamics of the infected individuals $I(t)$ only, instead of the total host population $N(t)$. Moreover, as the trough values in the presence and in the absence of harvesting are very low and almost confounded, they are not represented here. The amplitude of the epidemics is barely affected when hunting occurs between the mating

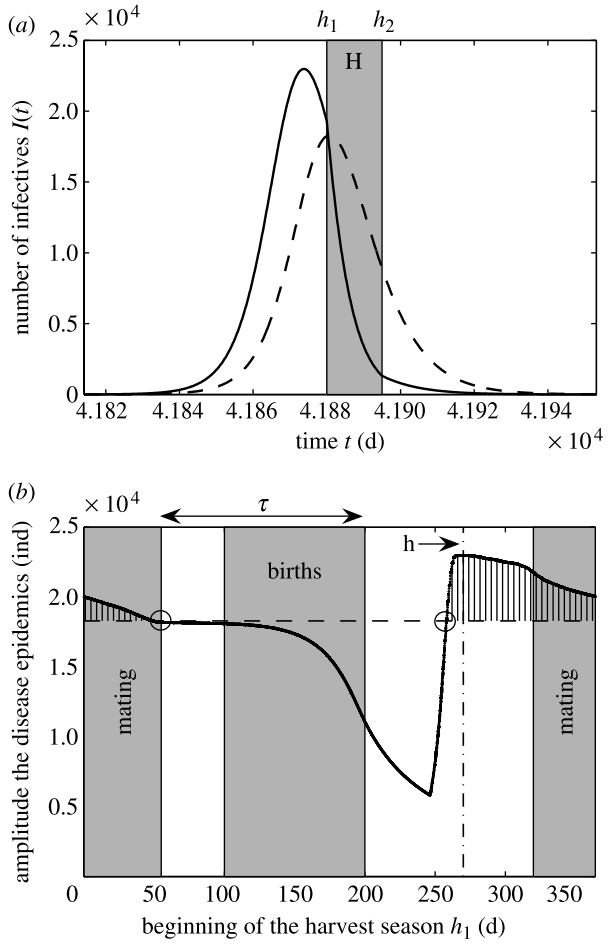

Figure 4. Effect of harvesting on the dynamics of infectious individuals $I(t)$. (*a*) The figure shows examples of annual epidemics, after stationary dynamics has been reached. The dashed curve represents the disease dynamics in the absence of harvesting ($H=0$) and the full line curve represents the disease dynamics in the presence of harvesting ($H=0.2$). The harvest season (indicated by 'H' on the graph) begins at $h_1=270$ d, and ends at $h_2=h_1+15$ d. (*b*) The figure shows the amplitude of the disease dynamics as a function of the beginning time h_1 of the harvest season. As for (*a*), $h_2=h_1+15$ d. The thick full line represents the amplitude of the disease epidemics in the presence of harvesting ($H=0.2$), and the horizontal dashed line represents the amplitude of the disease epidemics in absence of harvesting ($H=0$). The mating and birth seasons are represented on the graph and the duration τ of the gestation is indicated by the length of the double arrow above the graph. The vertical dash-dotted line arrowed by 'h' represents the value of h_1 used in (*a*). Circles show cases of disease full-compensation and hatched areas highlight cases of disease over-compensation. 100 yr of the model's dynamics have been numerically solved to reached stationarity, and then the peak and trough values have been sampled on the following 35 yr of the model's dynamics. Values of h_1 varies from 0 to 364.9 d, every 0.1 d. For (*a*) and (*b*), other parameter values are the same as for figure 3.

and the birth seasons; it starts to decrease when hunting occurs during the birth season. In this latter case, the later the hunting season starts, the higher the decrease in amplitude. The most dramatic decrease in epidemic amplitude is observed when the hunting season starts just before the epidemic peak is reached, somewhere between the birth season and the next mating season. Around the corresponding value of h_1, the amplitude of the disease epidemic becomes strongly sensitive to the exact timing of the harvest season. Indeed, in a window of only 20 days for h_1, the effect of harvesting on the amplitude of the epidemics can go from a strong decrease (if the harvest season occurs just before the epidemic peak) to a substantial increase (if the harvest season occurs just after the epidemic peak). After this critical period of 20 days, the amplitude of the epidemic keeps being increased by hunting, until h_1 reaches the end of the mating season. In this period of time, the later the hunting season starts, the smaller the increase in epidemic amplitude due to harvesting.

5. DISCUSSION

(a) *Harvesting can increase disease prevalence and mortality*

It has been documented for a large number of species that wild animal populations are regulated by density-dependent birth and death processes (Ekman 1984; Clutton-Brock *et al.* 1985, 1997; Skogland 1985; Hudson 1992; Gaillard *et al.* 2000). The analytical study of our model highlights the potential of such density-dependence to compensate for harvesting (equation (3.3), figure 1*a*). This well-known phenomenon lies at the core of sustainable management, where the essential aim consists in maximizing the short-term yield, without threatening the population of extinction in the long-term (Hilborn *et al.* 1995). However, current harvest models have not considered the potential effects of infectious diseases on the hunted population. Our model shows that the density-dependent compensation of harvesting tends—by stimulating the birth rate—to increase the prevalence of a disease which confers lifelong immunity to their host (equations (3.4) and (3.6), figure 1*b*). If the disease induces additional mortality to the infectious individuals, then the density-dependent compensation of harvesting can result in an increase of the number of disease-induced deaths (equation (3.8), hatched area on figure 2). Such a synergistic interaction between harvesting and disease-induced mortality implies that more individuals actually die than when hunting and disease-induced mortality are supposed independent (equation (3.9), hatched area on figure 2). This harvesting side-effect is predicted to be particularly severe for diseases with high basic reproduction ratio R_0 ($\propto 1/\gamma$), in host populations of relatively low density-dependence (equation (3.9)). The consequence is that optimal harvest efforts derived from population models that do not account for disease dynamics can be over-optimistic and lead to unexpected population extinction.

Our conclusions are based on the classical assumption that disease-induced mortality acts on infectious individuals (Anderson & May 1991), such that in equation (2.3) we have:

$$\frac{\mathrm{d}I}{\mathrm{d}t}=\cdots-\gamma I-vI.$$

(Note that here, to ease analytical tractability, we chose the *rate* instead of the probability formulation—similar results are obtained with either formulation.) One consequence of this assumption is that the effective mean duration of the infectious period is reduced by disease-induced mortality, because some infectious individuals die before

transmitting the disease. However, for some diseases, disease-induced mortality mainly occurs once the infected individual is not actively infectious (Keeling & Rohani in press). This may happen either when mortality results from pathogen-related complications (such as encephalitis or hypoxia) once infected individuals are in an advanced diseased state or following opportunistic infections (such as secondary lung infections). In this case, the model equations (2.3) and (2.4) would be changed to

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \cdots - \gamma I,$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = (1-\nu)\gamma I - \cdots,$$

where, here, the parameter ν represents the *probability* of succumbing to infection. In this representation, the mean infectious period is not affected by disease-induced mortality. Using this formulation, we unsurprisingly find that the effects reported in figure 2 become even more pronounced (results not shown).

(b) *Harvesting can increase the probability of disease cross-species transmission*

In temperate regions, the reproduction of most animal species is strongly seasonal, and the interaction between density-dependence and seasonality has been recognized to potentially over-compensate the harvest (see references in Kokko & Lindström 1998). Figure 3 illustrates this phenomenon and explores systematically the effect of the timing of the harvest season on the magnitude of compensation. The conclusion is that compensation is strongly sensitive to the exact timing of the harvest season and occurs mainly—but not solely—when the harvest season is scheduled between the birth season and the next mating season. This finding agrees with the results derived by Boyce *et al.* (1999) and Jonzén & Lundberg (1999) with discrete-time population models, by Kokko & Lindström (1998) with the continuous Beverton–Holt and Ricker models, and recently by Xu *et al.* (2005) with a continuous logistic model. When considering disease dynamics, our model shows that the combination of the harvest-induced increase in disease prevalence and the harvest over-compensation can increase the absolute number of infectious individuals. Although the mean annual number of infectious individuals is only slightly increased (less than 5% of increase in our numerical calculations), the peak values of the epidemics can be significantly increased (figure 4*a*). If the disease induces additional mortality, this could lead to a harvest-induced increase in disease-induced mortality of higher magnitude than derived at equilibrium (equation (3.9), figure 2), thus stressing further the risk of population extinction. If the disease does not induce additional mortality, then the increase in the epidemic amplitude can result in an increase, over a short period of time, of the probability of disease transmission to other species (Antia *et al.* 2003; Woolhouse *et al.* 2005). Such cross-species disease transmissions constitute real threats to wildlife biodiversity and conservation (McCallum & Dobson 1995; Cleaveland *et al.* 2002; Altizer *et al.* 2003), as well as to livestock economy or human populations (Morse 1995; Schrag & Wiener 1995; Daszak *et al.* 2000).

Figure 4*b* explores the effect of the timing of the harvest season on the epidemic amplitude. In particular, it shows that when the harvest season occurs during the epidemics, then the epidemic amplitude becomes very sensitive to the exact timing of the harvest season. Specifically, hunting just before the epidemic peak dramatically decreases the epidemic amplitude, whereas hunting just after the epidemic peak can substantially increase the epidemic amplitude. From a practical point of view, this result stresses the importance of carefully taking into account the dynamics of infectious diseases when managing wildlife populations. Indeed, in our numerical example, not accounting for the disease dynamics would have led to the conclusion that hunting between the birth season and the next mating season is safe for the population (figure 3*b*), whereas accounting for the disease dynamics shows that, between the birth season and the next mating season, the sooner the hunting season, the safer in terms of disease-associated risks (figure 4*b*).

(c) *Model hypotheses and applications*

Our conclusions result from three major ingredients in our model: (i) seasonal density-dependent regulation of the host population, (ii) seasonal constant-rate harvesting, and (iii) direct transmission of a disease that confers permanent immunity after recovery.

Density-dependence in the birth and death processes has been documented to be of strong magnitude for a variety of animal species, resulting in large seasonal variations in population densities (see, e.g. Ekman 1984; Clutton-Brock *et al.* 1985, 1997; Skogland 1985; Hudson 1992; Gaillard *et al.* 2000). Moreover, the strength of the density-dependence in the mortality rate is known to vary with both age and sex. Whereas our model does not include any structure other than the clinical status, we did try to account for the age effect by removing the density-dependence on the death rate in the recovered class—which, in the case of a disease conferring permanent immunity after recovery, consists essentially of adults (Anderson & May 1991). Relaxing this last assumption in our model would mitigate the observed effects of harvesting on the epidemic amplitude. Indeed, density-dependence is the mechanism that compensates for hunted individuals. Thus, the lower the strength of density-dependence on the death rate, the lower the compensation, and the stronger the population decrease due to harvesting. As, after an epidemic, the host population is essentially made up of susceptible and recovered individuals, a reduced density-dependence on the death rate of the recovered class means that compensation occurs mainly in the susceptible class, thus increasing the number of susceptibles in the host population. For particular host-disease associations, it is also possible that sex-specific density-dependence on the death rate would lead to slightly different results, unless the density-dependence on the birth rate is strong enough to compensate for the difference of density-dependence between the male and the female death rates. Lastly, our model is based on a simple linear form of density-dependence. Other types of density-dependence would probably change the results quantitatively, but likely not qualitatively. Moreover, it appears from precise measures of birth and death rates that, for most animal species, density-dependence would actually be close to linear (Gaillard *et al.* 2000).

A constant-rate harvest effort was used in our model. This corresponds to most hunting practices and

contrasts with natural predation where the specific functional responses usually include one form of density-dependence (Kot 2001). Moreover, for natural predation, there is no pre-defined 'predation season', and the seasonality in predation is generally a consequence of the predator functional response (Turchin 2003). The predation pressure would thus most likely occur just after the birth season, where, from our model predictions, the risks associated with disease-induced mortality are minimized. Another difference between predation and harvesting concerns the prey catchabilities q_i and harvest pressures H_i. In the case of predation, prey catchability tends to be higher for younger and inexperienced individuals than for adults. In contrast, human hunting efforts tend to concentrate on the older age classes. In our model, neither catchability nor harvest pressure are age-structured. However, we can predict that, in figure 4*b*, including higher catchability for the younger age classes than for the older ones would shift the curve downwards, thereby increasing the beneficial effect of harvesting in diminishing the risk associated with disease mortality or cross-species transmission. On the contrary, including higher harvest pressure for the older age classes than for the younger ones would shift the curve upwards, thereby increasing the harmful effect of harvesting in increasing the risk associated with disease mortality or cross-species transmission.

Finally, the model presented here makes the assumptions that the disease is directly transmitted and confers permanent immunity after recovery. Whereas it is unlikely that our conclusions would have been altered if indirect transmission was considered, it is important to recognize that permanent immunity is the key trait for our model outcomes. Some degree of partial immunity would certainly mitigate the observed effects of hunting on the disease dynamics. Permanent and/or partial immunity characterizes a large variety of highly contagious and potentially fatal microparasitic wildlife diseases. Lastly, the model assumes a frequency-dependent transmission of the disease, which appears the most appropriate for gregarious host populations (McCallum *et al.* 2001). However, since hunting increases not only the disease prevalence (figure 1*b*) but also the host population size (figure 3*b*), a density-dependent transmission of the disease would have produced even more pronounced results.

Since a major concern today in Europe and North America is the veterinary control of wildlife diseases, we hope that the simple and general framework presented here will serve as a basis for more complex and specific models in decision making in wildlife management. Examples that would fulfil our general model's assumptions include the classical swine fever and Aujeszky disease that infect wild boars in Europe, rabies that infects various species of wild canids worldwide, bovine tuberculosis that infects badger populations in the UK, myxomatosis that infects rabbits. Our model could also potentially be applied to emerging infectious disease like the avian flu that currently threatens waterfowls.

6. CONCLUSION

The aim of wildlife sustainable management is to design a harvesting policy that optimizes the short-term yield without driving the population to extinction in the long-term. This challenge has been the inspiration of a huge variety of models in the ecological literature of the last several decades. However, none of these models account for the dynamics of diseases that may thrive on these harvested populations. This is regrettable given the recent accumulation of evidences that diseases can substantially affect the population dynamics of their hosts. The model presented here shows that the complex dynamical interactions between seasonal density-dependence and harvesting can produce unexpected and unwanted effects such as an increase in epidemic amplitude. This result helps to understand the failure of widespread culling policies to eradicate tuberculosis in badger populations in the UK (Donnelly *et al.* 2003) and rabies in fox populations in Europe (Woodroffe *et al.* 2004): as suggested by Woodroffe *et al.* (2004), the demographic plasticity of such animal populations confers them with a remarkable capacity to recover from control, and such a response to culling can actually increase the supply of susceptibles to the disease. Moreover, from a conservation biology point of view, our result warns that harvesting policies which do not account for wildlife diseases are likely to underestimate the extinction probability of hunted populations. Besides purely dynamical properties, there are other aspects that appear worth taking into account for an efficient wildlife and wildlife disease management. One of them is behavioural ecology, the effects of which have been barely addressed theoretically, despite its evidenced key role in the dynamics of diseases in wild vertebrate populations (Donnelly *et al.* 2003; Hosseini *et al.* 2004; Woodroffe *et al.* 2004). Theoretical investigations further in this direction are promising in yielding results of valuable application in wildlife disease management.

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APPENDIX A: LINEAR APPROXIMATION OF THE DISEASE PREVALENCE AT EQUILIBRIUM

Here, we detail the calculations done to reach the analytical approximation of equation (3.4). Replacing $N(t)$ by N^* (equation (3.3)) in equations (2.1)–(2.4), equating them to zero, solving the system to get I^*, and dividing this number by N^* (equation (3.3)) gives:

$$\frac{I^*}{N^*} = \frac{\begin{array}{r}(2dK+Hq)\{d(2dK+Hq)(2dK+Hq+2\gamma)\\ +2\sigma[2d^2K+Hq\beta+d(Hq-2K\beta+2\gamma)]\}\end{array}}{\beta(Hq-2dK)(2dK+Hq+2\gamma)(2dK+Hq+2\sigma)}, \quad \text{(A 1)}$$

where the asterisks refer to equilibrium values. This is a polynomial fraction of H where the degrees of the numerator and the denominator are equal to 3. In the following, we will consider a linear approximation of this expression, for small values of H, by approximating it with

a polynomial of degree 1:

$$\frac{I^*}{N^*} = A + BH + o(H). \tag{A 2}$$

Equating H with 0 in equation (A 1) gives the expression of A in equation (A 2):

$$A = \frac{8\mathrm{d}^2K[\mathrm{d}K(\mathrm{d}K+\gamma)+\sigma((d-\beta)K+\gamma)]}{2\beta\mathrm{d}K(2\mathrm{d}K+2\gamma)(2\mathrm{d}K+2\sigma)}. \tag{A 3}$$

In the denominator, note that $\mathrm{d}K \ll \sigma$ and $\mathrm{d}K \ll \gamma$. Thus, the denominator can be approximated by $8\beta\mathrm{d}K\sigma\gamma$. Developing the numerator, we get

$$8(\beta\sigma\mathrm{d}^2K^2 - \gamma\sigma\mathrm{d}^2K - \gamma\mathrm{d}^3K^2 - \sigma\mathrm{d}^3K^2 - \mathrm{d}^4K^3)$$

$$= 8\beta\sigma\mathrm{d}^2K^2 + o(\mathrm{d}^2K^2), \tag{A 4}$$

the dominant term of which being the one in d^2K^2. In consequence,

$$A \simeq \frac{\mathrm{d}K}{\gamma} + o(\mathrm{d}K). \tag{A 5}$$

Now, let find the coefficient B of equation (A 2). When $H>0$, the parameter d becomes negligible before any other parameter. Thus, the dominant term of the numerator of equation (A 1) becomes $-2q^2\beta\sigma H^2$, and the dominant terms of the denominator of equation (A 1) become

$$-4q\beta\sigma\gamma H - 2q^2\beta(\sigma+\gamma)H^2 - q^3\beta H^3 = -4q\beta\sigma\gamma H + o(H), \tag{A 6}$$

the dominant term of which being the one in H. In consequence, neglecting the parameter d in equation (A 1) approximates it to

$$\frac{I^*}{N^*} \simeq \frac{qH}{2\gamma} + o(H), \tag{A 7}$$

which gives $B = qH/(2\gamma)$. Using the expressions of parameters A (equation (A 5)) and B (equation (A 7)) in equation (A 2) yields the linear approximation of I^*/N^* of equation (3.4):

$$\frac{I^*}{N^*} \simeq K\frac{d}{\gamma} + \frac{q}{2\gamma}H + o(H). \tag{A 8}$$

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