



OXFORD CENTRE FOR COLLABORATIVE APPLIED MATHEMATICS

Report Number 12/14

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by

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## Abstract

Disease control by managers is a crucial response to emerging wildlife epidemics, yet the means of control may be limited by the method of disease transmission. In particular, it is widely held that population reduction, while effective for controlling diseases that are subject to density-dependent transmission, is ineffective for controlling diseases that are subject to frequency-dependent transmission. We investigate control for horizontally transmitted diseases with frequency-dependent transmission where the control is via nonselective (for infected animals) culling or harvesting and the population can compensate through density-dependent recruitment or survival. Using a mathematical model, we show that culling or harvesting can eradicate the disease, even when

transmission dynamics are frequency-dependent. Eradication can be achieved under frequency-dependent transmission when density-dependent population regulation induces compensatory growth of new, healthy individuals, which has the net effect of reducing disease prevalence by dilution. We also show that if harvest is used simultaneously with vaccination and there is high enough transmission coefficient, application of both controls may be less efficient than when vaccination alone is used. We illustrate the effects of these control approaches on disease prevalence using assumed parameters for chronic wasting disease in deer where the disease is transmitted directly among deer and through the environment.

Key words: disease modeling, disease management, chronic wasting disease, frequency-dependent transmission

Control of any disease is related to thresholds, either in parameters or in population size. For example, a disease may be unable to spread if the population is below some critical size or the proportion of immune individuals is greater than a certain level. Such thresholds can be characterized by the basic reproduction number  $R_0$  [1-3]: if  $R_0 > 1$  the disease can spread. For wildlife diseases a manager's set of possible control actions is limited: reduction in population density, removal of infected individuals, or a vaccination of susceptibles. In the case of population reduction, a threshold must exist with respect to the population size at which the disease will die out. Population thresholds depend upon the disease transmission mechanism [4]. Typical mechanisms involve either density-dependent (DD) or frequency-dependent (FD) transmission [5,6]. In the former, the number of per capita contacts grows with population size due to increased contact rates with infected individuals. In FD case the number of per capita contacts is constrained to be independent of population size, such as when contacts occur in social groups, and group size is independent from the overall population size [5]. In the case of DD, population reduction can eliminate disease spread by reducing the population below a critical population threshold. In contrast, a population threshold is not exhibited in the case of FD, so population reduction, by itself, is not considered to be appropriate for disease management, see e.g. [7]. Disease transmission mechanisms other than DD or FD are possible [5-9], but FD and DD mechanisms are the most commonly assumed in disease modeling.

In this paper we present an SI model for the infection dynamics of a disease that has FD transmission mechanisms that occur through direct and environmental contact and is

coupled to density-dependent population birth and survival rates and no recovery from the disease. We describe the model in terms of population size  $n$  and disease prevalence  $i$ . We show that for the model there exists a population threshold in spite of FD disease transmission. It arises due to density-dependent birth that allows populations to (1) withstand culling/harvesting at levels sufficient to remove diseased individuals before they, on average, infect new susceptibles, and (2) effectively dilute disease prevalence with new, uninfected individuals. On the other hand, in populations exhibiting density-dependent mortality non-selective harvest may not increase removal of diseased individuals, just fewer of them die from natural causes. For this reason, density-dependent birth or recruitment appears to be critical for harvest control of a disease that has FD transmission.

We illustrate these effects by modeling chronic wasting disease (CWD) in deer [10]. CWD is a prion disease, and, to date, it is not known how to control the disease. Our previous modeling work [11] showed evidence that CWD transmission may result primarily from FD mechanisms related to deer social organization for both direct and environmental transmission [12]. In case when the disease prevalence grows significantly slower than the rate at which prions decay or become inaccessible to deer [13], both mechanisms can be described within the framework of SI-type model without explicit environmental compartment: due to the difference in the rates the environmental prion content is approximately proportional to the current number of infected individuals (see Supplementary Materials for the details of this approach). Deer also show density-dependent recruitment of new adults to the population [14]. Assuming FD transmission,

we investigate disease dynamics, and whether culling/harvesting results in disease extinction via a parameter-based threshold. Finally, we consider the possibility of vaccination as an alternative strategy that can be coupled to control via culling/harvesting to control disease and estimate levels needed to control the disease.

We use a simple population model, deriving conditions for disease eradication, in a manner that makes analysis transparent and shows the role of culling/harvesting and density-dependent deer recruitment and survival in disease management. We consider three adult disease classes: susceptible  $S$ , infected  $I$ , and immune after vaccination  $V$  with the total population size being  $n=S+I+V$ . The per capita recruitment of young into the populations,  $b(n)$ , is assumed to be a nonincreasing function of  $n$ , and the per capita natural mortality,  $m(n)$ , is assumed to be nondecreasing function of  $n$ . In other words, both may be density-dependent, with no Allee effects present. The increase of mortality rate due to infection is denoted by  $\mu$ , and hence the mortality rate for diseased individuals is  $m(n)+\mu$ . Here  $(m(n)+\mu)^{-1}$  is the average duration of infection prior to death. The disease transmission function is of a general form  $\beta(n)(I/n)S$ , where  $\beta(n)=\beta_{DD}n$  for DD transmission and  $\beta(n)=\beta_0$  for FD transmission, (note that the units for  $\beta_{DD}$  and  $\beta_0$  differ). Susceptible individuals become immune at per capita rate  $\gamma$ , which accounts both for vaccination intensity and vaccine efficiency. Finally, susceptibles, immune and infected individuals are culled or harvested at the same rate  $h$ , i.e., animals are non-selectively removed from the population.

The model takes the form:

$$\frac{dS}{dt} = b(n)(S + I + V) - m(n)S - \beta(n)(I/n)S - \gamma S - hS \quad (1)$$

$$\frac{dI}{dt} = \beta(n)(I/n)S - (m(n) + \mu)I - hI, \quad (2)$$

$$\frac{dV}{dt} = \gamma S - m(n)V - hV, \quad (3)$$

For the subsequent analysis it is convenient to rewrite the system in terms of population size  $n$ , disease prevalence  $i=I/n$  and immune fraction  $v=V/n$ . (see e.g. [15,16]). Then  $S=(1-i-v)n$ ,  $I=in$  and  $V=vn$ , and, after some transformations,

$$\frac{dn}{dt} = [b(n) - m(n) - \mu i - h]n \quad (4)$$

$$\frac{di}{dt} = iF(n, v, i), \quad F = \beta(n)(1 - v - i) - \mu(1 - i) - b(n) \quad (5)$$

$$\frac{dv}{dt} = \gamma(1 - v - i) - (b(n) - \mu i)v \quad (6)$$

(Supplementary Material). This new form yields interesting insights: (a) culling or harvesting does not directly influence disease prevalence  $i$  — culling/harvesting intensity  $h$  does not enter into (5) because nonselective harvest takes equal proportion out of all classes (see similar conclusion in [16]); and (b) culling/harvesting drives down the population size  $n$  and affects the disease prevalence indirectly by modifying the density-dependent contact rate  $\beta(n)$  and birth rate  $b(n)$ . In turn, the density-dependent contact rate and birth rate can play a major role in determining the disease prevalence  $i$  in (5).



Basic reproduction number  $R_0$  for this system can be obtained by standard methods at disease free equilibrium  $n = n_0, i = 0, v = v_0$  (see Supplementary Material), and condition for the disease persistence is

$$R_0 = \frac{\beta(n_0)(1 - v_0)}{\mu + m(n_0) + h} > 1. \quad (7)$$

We interpret it as the rate of production of new infectives  $\beta(n_0)(1 - v_0)$  times the average life span of infective individuals  $\tau_I = (\mu + m(n_0) + h)^{-1}$ . Vaccination increases  $v_0$  and hence decreases the rate of new infections; selective harvest increases  $\mu$  and decreases  $\tau_I$ . Both methods, when available, work regardless of the details of the disease and population dynamics. However, when there is no vaccine and infected individuals are hard to find or to distinguish from healthy ones, the only practical measure is nonselective population harvest  $h$ . Nonselective harvesting can influence  $n_0, v_0$  and  $\tau_I$ , and its effect depends on the details of disease transmission and population self-regulation.

First, we assume that  $v_0$  is fixed, then changes in  $R_0$  may be only due to  $\beta(n_0)$  and  $\tau_I$ . Harvest decreases  $n_0$ , and in case of DD transmission  $\beta(n) = \beta_{DD}n$  it reduces the rate of new infections as well. In case of FD transmission  $\beta(n) = \beta_0$  harvest does not change the rate of new infections, which for some models implies no disease control [4].

Dependence of  $\tau_I$  on  $h$  is determined by population self-regulation mechanism. There are two extreme cases (e.g. [17]):

146 a) density-dependent birth (DB) and density independent mortality:  $m$  is  
 147 independent from  $n$ , and  $b(n)$  is a decreasing function of  $n$ ;  
 148 b) density-independent birth and density dependent mortality (DM):  $b$  is  
 149 independent from  $n$  and  $m(n)$  is an increasing function of  $n$ .

150 At disease-free equilibrium ( $i = 0$ ) the following equality always holds,

$$151 \quad b(n_0) = m(n_0) + h, \quad (8)$$

152 see (4). Therefore in case (a) we have  $b(n_0) = m + h$ , that is an increase in  $h$  is  
 153 compensated by an increase in births of new healthy individuals. Total mortality  $m + h$   
 154 increases, which causes decrease of  $\tau_i$  and decrease of  $R_0$ . Therefore, there exists  
 155 population threshold below which the disease cannot persist provided the population  
 156 survives harvest of the required intensity.

157

158 In case (b) we have  $b = m(n_0) + h$ , that is an increase in harvest reduces equilibrium  
 159 population size  $n_0$  and natural mortality  $m(n_0)$ , but does not change total mortality, which  
 160 means more individuals die of harvest, but fewer die of natural causes. The average life  
 161 span of infective individuals  $\tau_i$  also remains unchanged.

162

163 Therefore we have four combinations of disease transmission and population self-  
 164 regulation: DD+DB, DD+DM, FD+DB, FD+DM. Only in the latter case there is no  
 165 population threshold due to nonselective harvest because both the rate of new infection  
 166 and average life span of infective individuals are independent from population size  $n_0$ .

167

168 Taking in account (8), we can rewrite (7) as

$$R_0 = \frac{\beta(n_0)(1-v_0)}{\mu + b(n_0)} > 1, \quad (9)$$

which makes the above statements more obvious. Note that the same condition for disease persistence  $\beta(n)(1-v) > \mu + b(n)$  can be obtained from (5) as a requirement of positive prevalence growth rate  $F(n, v, i)$  at  $i=0$ , but (5) does not require the system to be at equilibrium, and hence (9) is valid even when the population is in a transient regime after some perturbations. In particular, it shows that local culling events that lower population size enough to decrease  $\beta(n_0)$  or increase  $b(n_0)$  can temporarily slow down the disease progression.

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From now on we consider only the case of density-dependent birth (DB) for population regulation with  $m$  being constant. One of the forms of DB dependence has been used in [17],

$$b(n) = b_0 \left( 1 - (n/n_c)^\theta \right) \quad m = \text{const}, \quad (10)$$

where  $\theta$  takes values between 1 and 7, depending on species, Fig. 1 (note that  $n_c$  is greater than carrying capacity). In case  $\theta = 1$  it is possible to obtain analytical estimates for population thresholds  $n_{0T}$  corresponding to  $R_0=1$  (9) or

$\beta(n_{0T})(1-v_0) = \mu + b_0(1 - n_{0T}/n_c)$  in DD and FD cases:

DD transmission:  $\beta(n) = \beta_{DD}n$  and  $n_{0T} = (\mu + b_0)/(\beta_{DD}(1-v_0) + b_0/n_c)$ .

FD transmission:  $\beta(n) = \beta_0$  and  $n_{0T} = n_c(\mu + b_0 - \beta_0(1-v_0))/b_0$ .

In both cases the threshold is a decreasing function of transmission coefficient. However, in DD case the threshold always exists, while in FD case it exists only in case of moderate transmission coefficient or large enough  $b_0$ , i.e., maximum population growth

potential. Below we analyze conditions for  $b_0$  in more detail. We also point out that in the case of DD, dependence of  $b$  on  $n$  decreases the threshold, which agrees with [18].

The above analysis has been done under the assumption that  $v_0$  is fixed. A more realistic assumption is that the vaccination rate  $\gamma$  is fixed and simultaneous use of both vaccination and harvest causes their interaction. On the one hand, nonselective harvest may hinder the disease spread, but on the other hand it removes a part of vaccinated individuals and hence may facilitate the disease. At the disease-free equilibrium proportion of immune individuals is  $v_0 = \gamma / (\gamma + m + h)$ . We characterize competition of harvest and vaccination by the value of vaccination rate  $\gamma_{\text{er}}(h)$  needed to make  $R_0=1$  and eradicate the disease at the given intensity of harvest  $h$ . In the case of FD transmission, there is a critical value of the transmission coefficient  $\hat{\beta}_0 = (m + \mu)^2 / \mu$ , such that for  $\beta_0 < \hat{\beta}_0$   $\gamma_{\text{er}}(h)$  is strictly decreasing, and an increase in harvest means that vaccination efforts can be reduced (Fig. 2). However, for  $\beta_0 > \hat{\beta}_0$   $\gamma_{\text{er}}(h)$  is increasing for small  $h$ , reaches its peak value at  $h = \sqrt{\mu\beta_0} - m - \mu$ , and only then it starts to decrease. We conclude that there are parameter regions where combinations of two control measures may be less effective than vaccination alone. In the case of DD transmission the qualitative behaviour of  $\gamma_{\text{er}}(h)$  is similar to FD transmission, although expressions become more complicated.

If harvest is the only available measure, assuming no vaccination ( $\gamma=0$ ,  $v=0$ ), it can be applied as a control measure only if the population were not to go extinct due to it. The ability of the population to survive harvest of the given intensity  $h$  depends on its

maximum possible growth rate  $b_0$  (10). We estimate the maximum possible effect of  
 harvest in case of frequency-dependent disease transmission  $\beta(n) = \beta_0$  and identify  
 conditions for population collapse, disease control, and disease eradication in terms of  $b_0$ .  
 Our analysis does not require an exact form of density-dependent recruitment; we focus  
 only on some of the qualitative properties of the effect. We assume that  $b(n)$  is  
 qualitatively similar to (10), i.e., it has a maximum  $b_0$  at  $n=0$  and decreases  
 monotonically until it equals zero at  $n=n_c$ , in particular  $\theta$  can take any positive value. This  
 is sufficient to apply phase plane analysis to the system represented by Eqs. (4), (5)  
 (Supplementary Material). Its results can be presented as a bifurcation diagram that plots  
 the equilibrium disease prevalence  $i^*$  as a function of the maximum recruitment rate  $b_0$   
 and the culling/harvesting rate  $h$  (Fig. 3). Three qualitative outcomes pertain. Low  
 maximum recruitment rates  $b_0 < b_{0\text{coll}}$ ,  $b_{0\text{coll}} = (\beta_0 - \mu)(m + \mu) / \beta_0$ , results in population  
 collapse even without harvest. Intermediate recruitment rates  $b_{0\text{coll}} < b_0 < b_{0\text{elim}}$ ,  
 $b_{0\text{elim}} = \beta_0 - \mu$ , allow for reduction of disease prevalence via culling/harvesting.  
 Prevalence can be maintained between  $i_{\text{max}}^* = 1 - (m + \mu) / \beta_0$  and  $i_{\text{min}}^* = 1 - b_0 / (\beta_0 - \mu)$ ,  
 further harvest increase above  $h_{\text{max}} = b_0 \beta_0 / (\beta_0 - \mu) - m - \mu$  causes population collapse.  
 Finally, if  $b_0 > b_{0\text{elim}}$ , recruitment allows for complete disease eradication via  
 culling/harvesting at harvest intensity  $h_e = \beta_0 - m - \mu$ .

To show how these theoretical results can be applied to a specific disease of great  
 concern, we consider CWD in white-tailed deer. In the case of CWD, we use a SI-type  
 model for a prion disease that can spread among individuals or through the environment.

We assume a rate of decay in prion availability obtained in [13] is faster than the rate of CWD prevalence growth, and hence the amount of prion in the environment is proportional to the current number of infected individuals deposition. Details of this approach are given in the Supplementary Materials). Deer population is primarily regulated by density-dependent juvenile mortality, i.e. density-dependent recruitment of new adults [14]. There is no current vaccination for CWD but one is anticipated. Transmission coefficients for this species in Wisconsin have been estimated by Wassenberg et al. [19]. For purposes here, if we assume that CWD transmission is FD and using the estimate  $\beta_0 \approx 1.64$  infections/year from [19], and the estimate of  $\mu \approx 0.57 \text{ year}^{-1}$  as measured in captive mule deer [13] we obtain the condition of disease persistence (9) for a completely susceptible population as

$$1.64 > 0.57 + b(n) \quad \text{or} \quad b(n) < 1.07 \text{ year}^{-1}$$

assuming there is no difference in CWD duration between species. Because CWD is mostly evident in adult deer [20], we can interpret  $b$  as recruitment of new adults, which accounts for both birth rate and survival of juveniles during their first year. White-tailed deer have high fertility and on average adult females bear close to 2 or more fawns each year. Assuming buck:doe ratio as approximately 1:3, 2 fawns per year per adult female resulting in 2 fawns per 4/3 adults or 1.5 fawns/adult. However, typically only about 40% of the fawns survive until adulthood [21], which gives  $b(n) \approx 0.6 \text{ year}^{-1} < 1.07 \text{ year}^{-1}$ .

Therefore, our analysis shows that, according to condition (9), CWD prevalence should increase among free-ranging white-tailed deer under these conditions and no harvest.

Using the values of  $\beta_0$  and  $\mu$  used in [19] we obtain  $b_{0elim} = 1.07 \text{ year}^{-1}$ . To estimate the collapse threshold we need deer natural mortality rate  $m$ . In [19] the estimate of survival is 0.97 (per half a year?), which corresponds to  $m=0.06$ , and gives  $b_{0coll} = 0.41 \text{ year}^{-1}$ .

With this information it is possible to determine the effect of harvest on CWD prevalence, once the maximum recruitment rate  $b_0$  is known (Fig. 3). At present there are limited data on  $b_0$  for white-tailed deer. However, there is evidence that juvenile survival can increase by  $0.16 \text{ year}^{-1}$  in population with reduced density [22], which would increase the above estimate for  $b(n)$  to  $0.76 \text{ year}^{-1}$ , so we may assume it as a low estimate of  $b_0$ . An upper estimate of  $b_0$  should correspond to the highest possible survival of fawns. For mule deer the highest registered fawn survival rate is close to 0.8 [23], so we can assume that  $b_0 < 1.20 \text{ year}^{-1}$ . Therefore most probably  $b_0 > b_{0coll}$ , and deer would not die out due to CWD. An assumption  $b_0 > b_{0elim}$  does not seem unrealistic, but practical use of this inequality would require an increase of juvenile survival to about 0.72 via release from density-dependence. Such high survival is unlikely to occur except in very productive environments where deer densities are kept far below food-based carrying capacity by harvest and in years of mild environmental conditions. Most plausible assumption is that  $b_{0coll} < b_0 < b_{0elim}$ , and we conclude that under these conditions the modelled deer population is in the middle domain in Fig. 3 where harvest can reduce the disease prevalence, but it cannot eradicate the disease. Therefore, disease eradication would require vaccination as well as harvest.

In the absence of vaccination ( $v=0$ ) and low harvest, we obtained a value of basic reproduction number at disease-free equilibrium for CWD  $R_0 = \beta_0 / (\mu + b(n)) \approx 1.4$  (9), which agrees with the values reported by Miller et al. [13] for mule deer. Thus an effective immunity level of  $v_0 \approx 0.3$  yields a basic reproduction number of  $R_0 \approx 1$ . Hence immunity of about 1/3 of adult population would be necessary to stop CWD spread under the assumption of FD transmission with negligible vertical transmission, and for the assumed parameter values above. An estimate of the critical value of FD transmission coefficient in Fig. 1 is  $\hat{\beta}_0 \approx 1.75$ . Thus harvest may not increase the required vaccination efforts and a joint harvesting/vaccination program may be necessary for disease eradication.

The parameter estimates we are using are only preliminary and require additional and better data on deer and CWD [24] before they can be used to guide management, particularly studies of prion dynamics in the environment and their accessibility over time. To show that predicted outcomes of our modeling depend crucially upon parameter estimates, we considered a set of alternative parameters that may apply to white-tailed deer populations like in Wisconsin. If we take  $\beta_0 \approx 0.82$  infections/year (half of the above estimate, [e.g. due to smaller group sizes](#)),  $m=0.21 \text{ year}^{-1}$  (estimate of survival rate for deer in Northeastern Minnesota [25]),  $\mu \approx 0.33$  (assuming slower disease development due to lower contact rates in the wild than in penned deer), we obtain very different results:  $b_{0\text{coll}} \approx 0.32 \text{ year}^{-1}$ ,  $b_{0\text{elim}} \approx 0.49 \text{ year}^{-1}$ . These values are well below the estimated recruitment rate for white tailed deer  $b(n)=0.76$ , and this in turn is below the value  $b_0$ . For this scenario eradication via harvesting could be a possibility. Hence,



detailed information of parameters related to deer population dynamics and disease dynamics is required for determining efficient control measures for region-specific CWD management.

The effect of harvest on disease prevalence may be complicated by other factors as well. Choisy & Rohani [26] reported the disease prevalence increased with harvest for a disease with FD transmission. Their example had critical differences from our case: their work represented a highly contagious disease ( $\beta_0 = 2000 \text{ year}^{-1}$ ) with duration about 2 weeks, very high probability of recovery, very low or zero mortality increase, and immune recovered individuals. In other words, the main effect of the disease is “vaccination” of the population: equilibrium proportion of recovered individuals exceeds 96%. The effect of harvest slightly resembles the case of our Fig. 2 for  $\beta > \hat{\beta}$ , when the diminishing of proportion of immune individuals has stronger effect than the decrease of  $R_0$  (see Supplementary Materials). Bolzoni et al. [18] also observed an increase in disease prevalence with harvest, but in a model with DD transmission. In their model harvest of adults reduces juvenile mortality, and may even increase the total population and hence enhance the disease transmission. In [27,28] there are described other situations when harvest causes other density-dependent population responses, i.e., increased litter size, or change animal behaviour, as in case of badgers, which may also facilitate disease spread. While possible, no such factors have yet been identified for deer and CWD.

In this paper we have studied a model of fatal disease with frequency-dependent transmission. We have shown that non-selective population harvest (i.e., removal of infected and uninfected animals at equal rate) may still be a useful disease management tool even under FD transmission, when population self-regulates through density-dependent birth or recruitment, but it may not be possible when population self-regulates through density-dependent adult mortality. Under the former conditions, the most important population characteristic for applicability of the harvest control is its maximum recruitment rate at low population density. Harvest may both facilitate and impede disease management by vaccination, depending on the disease transmission coefficient. With high transmission coefficient the main effect of harvest is boosting the disease due to removal of immune individuals. With low transmission coefficient stronger effect has the decrease of secondary infections due to shorter lifespan of infected animals, so the optimal management policy depends on the disease. To the best of our knowledge, the harvest control of diseases with FD transmission has not been considered in detail previously, perhaps because it potentially can be applied only to species having the corresponding population regulation mechanism and high recruitment potential.

ACKNOWLEDGEMENTS. This work has been supported by Alberta Prion Research Institute and Alberta Innovation through grants (E.Merrill: RES0004230), Natural Sciences and Engineering Research Council of Canada Discovery Grants (EM, MAL) Canada Research Chair (MAL), NSERC Accelerator Grant (MAL) and Research Fellowship from Oxford Centre for Collaborative and Applied Mathematics supported by

346 Award No KUK-CI013-04 made by King Abdullah University of Science and  
347 Technology (KAUST) (MAL). We thank reviewers for helpful suggestions.  
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## Figure Legends

Fig. 1. Examples of dependencies  $b(n) = b_0 \left(1 - (n/n_c)^\theta\right)$  for three values of  $\theta$ . For  $\theta=2$  the disease-free equilibrium calculation is shown, when recruitment compensates for mortality and harvest.

Fig. 2. Examples of vaccination rate necessary to eradicate the disease  $\gamma_{er}(h)$  as a function of harvest rate  $h$  under frequency-dependent disease transmission and density-dependent recruitment. For the values of transmission coefficient  $\beta_0 > \hat{\beta}_0 = (m + \mu)^2 / \mu$  there is an interval of  $h$  values where joint use of two controls is worse than vaccination alone.

Fig. 3. Minimum equilibrium disease prevalence  $i_{*min}(b_0)$  that can be achieved by population harvest as a function of maximum population fecundity or recruitment  $b_0$  (dotted line), and the required harvest rate  $h$  (dashed line) to achieve it for the case of FD transmission. For too small  $b_0$  population collapses after disease introduction; at medium  $b_0$  values population harvest can only reduce the prevalence, and too intensive harvest also causes population collapse; at high  $b_0$  values harvest allows to eradicate the disease at  $h = h_c$ .

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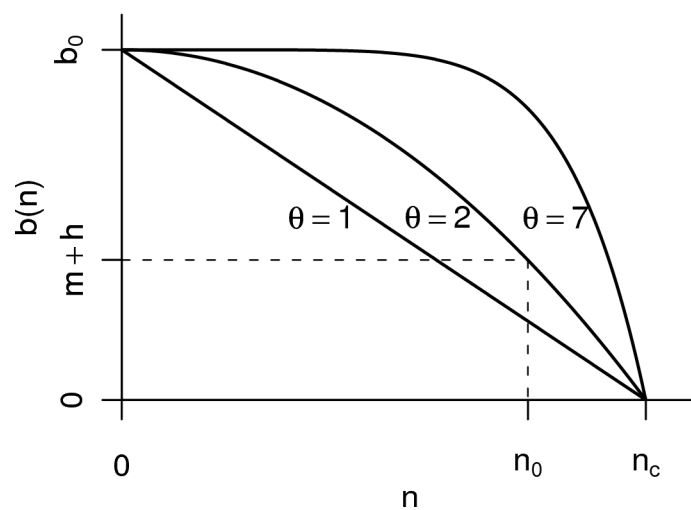
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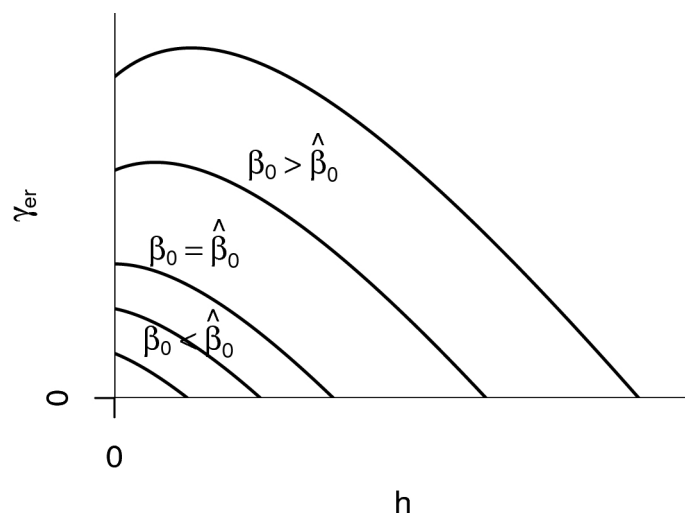
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Fig. 1

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Fig. 2

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