Gender-Based Harvesting in Wildlife Disease Management

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Wildlife disease management strategies traditionally focus on lowering aggregate host density below a host-density threshold, reducing infectious contacts (when transmission is density-dependent) to reduce prevalence. The focus on aggregate host density is because controls such as harvests are typically nonselective with respect to disease status. Such nontargeted strategies increase control costs and may not optimally lead to eradication. We consider targeting an observable trait correlated with infection—gender. Two endogenous host-density thresholds emerge, in contrast to the exogenous thresholds arising in the ecological literature on multiple-host-pathogen problems. The ability to manage these thresholds reduces control costs and makes eradication optimal.

Key words: bioeconomics, bovine tuberculosis, epidemiology, host-density threshold, targeting, white-tailed deer.

The spread of wildlife diseases is a major threat to agricultural livestock, human health, natural resource-based industries, and biodiversity conservation (Lanfranchi et al. 2003). Yet our knowledge of how to best manage wildlife diseases is limited. The solution might be straightforward if the health status of individual wildlife was easily observable, enabling a cull of infected animals (albeit at a cost). However, health status is often unobservable prior to harvest and postmortem testing (Lanfranchi et al. 2003).

Two approaches to disease control have been advocated when wildlife harvests are nonselective with respect to disease status. The first approach, which is reliant on density-dependent disease transmission, requires harvesting the aggregate population below an exogenous host-density threshold (Bicknell, Wilen, and Howitt 1999; Barlow 1991; McCallum, Barlow, and Hone 2001). This is the population density level at which infectious contacts are reduced enough that the disease begins to dissipate. But prolonged disease control, or eradication, can be costly when nonselective harvesting is the only management option or the host-density threshold occurs at a low density.

The second approach is to manipulate environmental or habitat conditions to affect disease dynamics (Horan and Wolf 2005). As with harvests, habitat controls may be nonselective with respect to health status. Horan and Wolf (2005) analyzed a model with density-independent (or frequency-dependent) transmission where supplemental feeding of wildlife (an effective change in habitat) led to artificially high infectious contacts. There is no host-density threshold under frequency dependence, but there is a feeding threshold such that disease prevalence diminishes if feeding is kept below this value. Horan and Wolf (2005) considered the case of bovine tuberculosis (bTB) (Mycobacterium bovis) in Michigan white-tailed deer (Odocoileus)

2 Other control methods (e.g., vaccination) have also been investigated, but most studies favor harvests (Smith and Cheeseman 2002). In particular, effective vaccines do not exist for some diseases, such as bovine tuberculosis—our focus here (MDA 2002).

3 Frequency dependent transmission occurs when the contact rate is independent of host density—that is, a susceptible individual makes the same number of contacts with other animals regardless of the host population density (McCallum, Barlow, and Hone 2001).
and found eradication might not be optimal: the benefits of maintaining sufficiently small feeding levels, in terms of reduced infectious contacts, may not outweigh the costs associated with reducing in situ reproduction of healthy animals.

A combination of harvests and habitat manipulation can be used to improve disease management when disease transmission is density-dependent. Fenichel and Horan (2008) develop a density-dependent model of bTB transmission in Michigan white-tailed deer, whereby prevalence can decline via both harvests and habitat controls. They show that the host-density threshold becomes endogenous when habitat controls also influence transmission. Managers therefore do not simply manage the population relative to the threshold; rather, they manage both the population and the threshold. Fenichel and Horan (2008) find that prevalence rates are optimally reduced when two controls are used instead of a single control, but that eradication may still not be optimal.

In each case, the nonselective nature of harvest and habitat controls may drive the results that eradication is not optimal, as any solution derived under this feature can only be second-best. First-best solutions require controls that are selective according to health status, while nonselective controls create excessive control costs: for instance, unintended harvests of healthy animals impose costs on future generations, and reduced feeding lowers the in situ productivity of both infected and healthy animals.

Although the use of nonselective controls is unavoidable (since the health-status of individual animals is unobservable), there may be ways to better target the controls. This would increase efficiency, thereby reducing control costs and making the optimality of disease eradication more likely. The purpose of this article is to examine the use of controls that indirectly target infected wildlife. This is accomplished by targeting an observable trait correlated with infection. Gender is the most basic and often (though not always) observable difference arising within a wildlife population. Moreover gender-specific physiological and behavioral traits can differentially affect disease transmission and hence prevalence (Smith, Cheeseman, and Clifton-Hadley 2001). In the case of bTB in Michigan white-tailed deer, estimated bTB prevalence differs significantly between males (8%) and females (2%) (O’Brien et al. 2002). Targeting harvests by gender could reduce overall disease prevalence, even though gender-specific harvests remain nonselective with respect to disease. Big game managers traditionally establish gender-based hunting regulations, but their goals have not focused on disease control.

We analyze gender-based harvest strategies using a bioeconomic model having greater biological detail than the single-host-pathogen (i.e., genderless, aggregated population) models typically used (this is consistent with recent bioeconomic trends of adding greater biological detail; e.g., Bulke and van Kooten 1999; Brock and Xepapadeas 2002; Bulke and Damania 2003; Finnoff and Tscharnkt 2003). The biological component can be viewed as a multiple-host-pathogen model, with each gender being a different host. The ecological literature has focused primarily on single-host models, but Dobson (2004) identifies the development of multiple-host models as a priority. A major focus of the emerging literature in this area is to identify the minimum amount of control effort required to eradicate the pathogen. Diekmann, Heesterbeek, and Metz (1990) derived the basic reproductive ratio for a pathogen (R0) as a function of control effort, and showed how a constant (i.e., time-invariant) effort level could be chosen to eradicate the disease. Roberts and Heesterbeek (2003) criticized this approach because the control efforts would have to be administered uniformly across hosts. They responded by deriving minimum required control efforts targeted to individual host populations. Control efforts for one population were chosen independently of management applied to other hosts. Roberts and Heesterbeek (2003) did not recommend specific effort levels other than minimums, and they did not consider if eradication was a socially desirable objective.

We expand the ecological literature on multiple-host systems by recognizing that the economic and ecological systems are jointly-determined, and by considering economic and ecological tradeoffs when designing time-variant control strategies. A key result is that the host-density thresholds are endogenous, whereas the ecological literature treats them as exogenous (Dobson 2004; Roberts and Heesterbeek 2003).

The model we present also expands the bioeconomic literature. In contrast to Fenichel and Horan’s (2008) single-host model, we find that the host-density threshold is endogenous, even without habitat management. The multiple-host setting provides managers with
improved targeting opportunities and greater flexibility to manage the thresholds within one or both subpopulations. The manager’s ability to manage the disease is improved, and eradication is more likely to be optimal.

Finally, we consider the impacts of habitat controls on disease dynamics, which Dobson (2004) and Daszak, Cunningham, and Hyatt (2001) stress could be important. We find improved targeting of harvest efforts reduces reliance on habitat controls.

Motivating Example

The models in this article are based on the Michigan white-tailed deer example analyzed by Horan and Wolf (2005) and Fenichel and Horan (2008). The example is relevant due to the large differences in male and female prevalence rates, and it allows for comparison with prior work. Moreover, several features of the problem facilitate the analysis. First, the population is basically closed within a four-county area in the northeastern part of the lower peninsula, known as deer management unit (DMU) 452 (Hickling 2002; Schmitt et al. 2002). Tracking studies indicate the deer migrate little even within DMU 452 (Garner 2001), and the Michigan Department of Natural Resources (MDNR) manages deer in DMU 452 as a unique population and estimates little risk of spread (Hickling 2002).

Second, human-environmental interactions, in the form of supplemental feeding, play a major role in the problem. DMU 452 is the only known area in the United States where bTB has established in wild deer, and conventional wisdom held that the disease was not self-sustaining in wild deer populations (Hickling 2002). It is believed that area-specific features—particularly feeding programs that encourage deer to congregate—have enabled the disease to become endemic (Hickling 2002). Additionally, deer density in the region has been elevated about three and a half times historic densities by hunt club-sponsored feeding programs (O’Brien et al. 2002).

The disease has spread from the deer herd to local livestock, and so Michigan agriculture is concerned about disease-related costs and supports culling deer to eradicate the disease. However, such extreme measures could be very costly, particularly since deer hunting is arguably the highest-valued use of the land in the infected region.

Ecological Thresholds in Prior Single-Host Models

We begin with a single-host (genderless) wildlife disease model with density-dependent disease transmission, to provide background into the relevant issues. Consider a closed deer population, \( N \), on a fixed land area. \( N \) consists of susceptible (S) and infected (I) individuals, i.e., \( N = S + I \), so that the disease prevalence rate is \( \theta = I/N \). There is no recovered population, as bTB and many other wildlife diseases are chronic with no recovery (Barlow 1991). We adopt Fenichel and Horan’s (2008) model (except we assume perfect vertical transmission to simplify the algebra)

\[
\dot{N} = rN \left(1 - \frac{N(1 - \tau f)}{k} \right) - \alpha (1 - \chi f) \theta N - h
\]

\[
\dot{\theta} = [\beta (1 + \omega f) (1 - \epsilon + \epsilon N)] - \alpha (1 - \chi f) \theta (1 - \theta) \theta.
\]

The controls in equations (1) and (2) are harvests, \( h \), and supplemental feeding, \( f \). The remaining terms are parameters: \( r \) is the intrinsic growth rate of the population \( N \), \( k \) is the carrying capacity, \( \tau \) reflects the role of \( f \) in increasing the effective carrying capacity, \( \alpha \) is the disease mortality rate when \( f = 0 \), \( \chi \) reflects the role of \( f \) in reducing disease mortality, \( \beta \) is the rate at which a contact with an infectious individual causes infection, \( \omega \) reflects the role of \( f \) on increasing infectious contacts, and \( \epsilon \) defines the degree of density dependence in transmission (see Fenichel and Horan [2008] for details). Horan and Wolf (2005) model the special case where \( \epsilon = 0 \), i.e. frequency-dependent transmission.

The primary implication of density-dependent transmission (\( \epsilon > 0 \)) is that \( h \) can indirectly influence changes in \( \theta \) via its impact on \( N \), whereas there is no such impact in a frequency-dependent model. Define \( \hat{N} \) to be the value of \( N \) that solves \( \hat{\theta} = 0 \)

\[
\hat{N}(f) = \frac{k [\alpha (1 - \chi f) + \beta (\epsilon - 1) (1 + \omega f)]}{\beta k \epsilon (1 + \omega f)}
\]

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4 The multiple-host problem is readily extendable to spatially explicit problems, because a metapopulation model is a special case of the multiple-host model.
\( \hat{N}(f) \) is a host-density threshold such that disease prevalence is increasing \((\theta > 0)\) for values of \( N > \hat{N}(f) \), and prevalence is decreasing \((\theta < 0)\) for values of \( N < \hat{N}(f) \).

Most ecological studies focus on deriving the constant harvest effort level required to maintain \( N \) below \( \hat{N} \) (Heesterbeek and Roberts 1995). These studies ignore how human choices such as \( f \) impact habitats, thereby implicitly holding human choices fixed (although such impacts are increasingly acknowledged to influence disease dynamics; see Daszak, Cunningham, and Hyatt 2000; Dobson 2004). From equation (3), holding \( f \) fixed implies that \( \hat{N}(f) \) is a vertical line in \((N,\theta)\) space—the threshold is constant for all time, and a constant effort plan must simply maintain the population below this value.

There are several limitations with this approach. First, assuming \( f \) is fixed, the approach derives the minimum effort level required to eventually eradicate the disease—it does not identify a “best” effort level, nor does it address the question of whether eradication is worthwhile. Second, fixing \( f \) imposes an unnecessary constraint. When this constraint is relaxed, both \( f \) and the host-density threshold become endogenous. Accordingly, the disease manager’s problem is not simply to manage \( N \) in relation to \( \hat{N} \), but rather to manage \( N \) and \( \hat{N} \) simultaneously.

Fenichel and Horan (2008) consider optimal management for the model described above. They first consider the case where \( f \) is held fixed, and they find that an interior steady state \((\theta > 0)\) is optimal. When \( f \) is endogenous and chosen optimally, then \( f \) is set according to a feedback rule of the form \( f(N, \theta) \).

The host-density threshold in this case is backward bending in \((N, \theta)\) space—that is, the optimal threshold depends on the current value of \( \theta \). The optimal dynamics are cyclical as in Horan and Wolf (2005): investing in the deer population is optimal when both \( N \) and \( \theta \) are low (as the opportunity cost of foregone deer productivity is high), and investing in reduced prevalence is optimal when \( N \) and \( \theta \) are high. However, the system exhibits smaller prevalence rates and larger population levels than in the Horan and Wolf (2005) model or in the density-dependent model in which \( f \) is held fixed. The reason is that access to two controls to manage \( \theta \) (as opposed to one in either of the other cases) reduces disease management costs.

### Ecological Thresholds in Prior Multiple-Host Models

Multiple equations of motion, \( \theta_i \) \((\text{where} \ i \ \text{indexes host populations}) \), govern disease dynamics in the multiple-host case. Disease dynamics are dependent across populations, and so the host-density threshold for population \( i \) \((\text{derived by setting} \ \theta_i = 0)\) depends on the population sizes and prevalence rates of all other \((j \neq i)\) populations. This means that the threshold for a particular population is state-dependent, even before economic choices such as feeding are considered. Still, the ecological literature concentrates on the threshold arising at one particular state of the world—the point of eradication (Diekmann, Heesterbeek, and Metz 1990; Roberts and Heesterbeek 2003; Dobson 2004).

A “next-generation” matrix, \( K \), derived from the system of disease dynamics, is typically used to measure a pathogen’s capability of invading a noninfected multiple-host system (Diekmann, Heesterbeek, and Metz 1990). \( K \) can be used to calculate each subpopulation’s host-density threshold, evaluated at the point of eradication (Roberts and Heesterbeek 2003). For our example of one male and one female population, \( K \) accounts for both within-gender and between-gender transmission, taking the form

\[
K = \begin{bmatrix}
K_{MM} & K_{MF} \\
K_{FM} & K_{FF}
\end{bmatrix}.
\]

Each element \( K_{ji} \) \((\text{of} \ K)\) represents the expected number of secondary cases of infection in host type \( j \) that would arise from an initial infection within the susceptible population of host type \( i \), assuming that the population is at equilibrium in the absence of disease (Diekmann, Heesterbeek, and Metz 1990). Accordingly, each element \( K_{ji} \) depends on the (steady state) equilibrium population densities arising in the absence of disease.

The dominant eigenvalue of \( K \), denoted \( R_0 \), is the basic reproduction ratio of the pathogen—the expected number of secondary cases arising from a primary case in a susceptible population (Dobson 2004; Roberts and Heesterbeek 2003). If control efforts can reduce \( R_0 \) below unity, then the pathogen will eventually die out. But this criterion does not provide any indication as to how to target efforts differentially across host types. Roberts
and Heesterbeek (2003) use $K$ to determine a host-specific disease reproduction ratio, $R_{0i}$, which is the expected number of all secondary cases resulting from a primary case in susceptible host $i$. They then use $R_{0i}$ to determine host-density thresholds and the constant levels of host-specific control that can lead to eradication. But, as in the single-host case, these are minimum required efforts—they are not optimal levels derived according to a decision model. Optimal effort levels reflect tradeoffs between an objective function and the system dynamics, and these effort levels likely vary over time.

Using $K$ to develop management recommendations would be problematic even if $K$ was to be incorporated into a decision model. The primary limitation of $K$ is that its elements are exogenously-determined, based on the assumption of a predisease steady state. Accordingly, $K$ ignores important dynamic and endogenous aspects of the problem. If $K$ is used to calculate the required level of control, then this level is also exogenously-determined, based on ecological relations and based on the predisease steady state. Moreover, any host-density thresholds derived from $K$ will be independent of all other states (as these are held constant when $K$ is derived). A bioeconomic approach, in which management strategies are chosen with consideration given to the full system dynamics and associated economic-ecological feedbacks, would be preferred from an economic standpoint. In the following sections, we develop a multiple-host, gender-based model of disease transmission and population dynamics. We then construct a bioeconomic model to explore economically optimal management.

A Multiple-Host, Gender-Based Model of Infectious Disease Transmission

Suppose that the deer population, $N$, is partitioned along two dimensions—health status and gender. In that case, the aggregate deer population consists of four subpopulations. As above, the first dimension, indexed by $z$, divides the deer population into healthy animals ($z = S$) and infected animals ($z = I$). The second dimension, gender, is indexed by $i$ and divides the deer into males ($i = M$) and females ($i = F$). Denote the total male and female populations by $N_M = S_M + I_M$ and $N_F = S_F + I_F$, respectively, with $N = N_F + N_M$.

Four processes affect the growth of each subpopulation $i$: (i) recruitment via births, $B_{iz}$, (ii) natural mortality, $M_{iz}^N$, (iii) harvests, $h_{iz}$, and (iv) new infections via transmission from subpopulation $i$ to subpopulation $j$, $T_{ij}$. Infected populations are also affected by an additional component: mortality due to the disease, $M_{iz}^D$. Specifically, growth of the subpopulations takes the general forms

$$\dot{S}_i = B_{iS} - M_{iS}^N - \sum_{j=M,F} T_{ji} - h_{iS} \quad i = M, F$$

$$\dot{I}_i = B_{iI} - M_{iI}^N - M_{iI}^D + \sum_{j=M,F} T_{ji} - h_{iI} \quad i = M, F.$$ 

For aggregated population models in the disease ecology literature (e.g., Roberts 1996; Barlow 1991), birth and mortality processes are commonly combined into a single net growth or surplus production function—most often the logistic or modified logistic growth function, i.e., $B - M_N = rN(1 - (N/k)^\zeta)$, where $r$ is the intrinsic growth rate, $k$ is the carrying capacity, and $\zeta$ is a parameter that affects the symmetry of the growth function. The intrinsic growth rate represents the maximum growth rate of the stock in the absence of resource competition (i.e., for food), and equals the birth rate, $b$, minus the natural mortality rate, $\delta$. The term $(1 - (N/k)^\zeta)$ is the density-dependent component of net growth that tempers growth in response to resource competition driven by the habitat’s carrying capacity. Standard logistic growth (when $\zeta = 1$) is symmetric around the population value $N = k/2$. A value of $\zeta > 1$ ($\zeta < 1$) shifts the peak of the growth function to the right (left). We follow the convention of using the standard logistic model ($\zeta = 1$) to capture the effects of density-dependent, compensatory growth (other values of $\zeta$ are explored in the sensitivity analysis). However, we separate the birth and mortality components to model recruitment into the different subpopulations.

Total births are $bN_F$, where $b$ is the birth rate per female.$^5$ Fawns produced by healthy females will all be healthy, with a proportion, $\phi$, being male. Fawns produced by

$^5$ Assume the male population is large enough to avoid an Allee effect (the number of males does not constrain female fecundity). This is a common assumption in populations modeling, especially for polygamous species such as deer (Casewell 2001, p. 570).
infected females may or may not be infected. Denote $v$ to be the rate of pseudo-vertical transmission—the proportion of fawns that are infected either in utero or after birth through maternal contact. Given this specification, total births of healthy females is $b(1 - \phi)[S_F + I_F(1 - v)]$, total births of infected females is $bv(1 - \phi)I_F$, total births of healthy males is $b\phi [S_F + I_F(1 - v)]$, and total births of infected males is $bv\phi I_F$. Natural mortality is given by the gender-specific rate $\delta_i (i = M, F)$.

Net growth is determined by multiplying the difference between births and natural mortality by the density-dependent term $(1 - (N/k)^\delta_i)$, modified by the impacts of feeding. Assume the effective carrying capacity is increased by feeding for $f < f^{\max}$—at that point another resource becomes limiting (Walters 2001). Denote the effective carrying capacity by $k/(1 - \tau f)$, where $f$ is supplemental feed and $\tau < 1/f^{\max}$ is a parameter. Supplemental feeding increases the effective carrying capacity in a manner consistent with Walters (2001). As feeding is costly and only provides productivity benefits for $f < f^{\max}$, $f^{\max}$ is an upper bound on feeding (this is made explicit in our simulation). Hence, net growth of the $S_F$ population is $(b(1 - \phi)[S_F + I_F(1 - v)] - \delta_F S_F)(1 - ((N/k) (1 - \tau f))^{\delta_F})$. Net growth is analogously derived for the other subpopulations.

Harvests reduce the stock after net growth has occurred. Harvests are only selective with regard to gender: a manager can only choose the aggregate harvest for each gender class, $h_i$. The harvest from each health class depends on the proportion of animals in that stock relative to the aggregate subpopulation $N_i$. That is, harvests from gender class $i$ and health class $z$ are $h_{iz} = h_i z_i/N_i$.

Disease transmission also alters a population after density-dependent growth and mortality has occurred. Three types of contacts can transmit disease: mother to offspring (pseudo-vertical transmission, described above), within-gender (male–male or female–female), and cross-gender (male–female or female–male). Transmission between adults is broken into two types because, under natural conditions, white-tailed deer segregate by gender and live apart for most of the year, except for the rut (mating season) and yarding (congregation to keep warm during severe winters) (Sitar 1996; O’Brien et al. 2002).

For the within-gender and cross-gender cases we adopt a modified version of Roberts’ (1996) transmission function (this specification also underlies equation (2) for the genderless case, where subscripts $i, j$, and $l$ are dropped)

$$(7) \quad T_{ij} = (1 - \epsilon_{ij} + \epsilon_{ij} N_i) \times (1 + \omega f)\beta_{ij} S_i I_j/N_i$$

where $N_l = N_i$ for within-gender transmission, and $N_i = N$ for cross-gender transmission. Transmission per healthy deer, $T_{ij}/S_i$, depends on a contact function (defining the rate at which deer contact each other), $(1 - \epsilon_{ij} + \epsilon_{ij} N_i)(1 + \omega f)$, multiplied by the proportion of contacts with infectious individuals, $I_j/N_i$, multiplied by the rate at which healthy deer acquire infection after contacting infectious deer, $\beta_{ij}$. The parameters $\epsilon_{ij}$ and $\omega$ are as defined for the genderless case in equation (2): $\omega$ reflects the role of $f$ on increasing infectious contacts, and $\epsilon_{ij}$ defines the degree of density dependence in transmission. Proportional density-dependent transmission is implied by $\epsilon_{ij} = 1$, frequency-dependent transmission is implied by $\epsilon_{ij} = 0$ (i.e., $N$ does not influence the contact function), and values of $\epsilon_{ij} \in (0, 1)$ imply transmission dynamics somewhere on the spectrum between proportional density-dependence and density-independence (or frequency-dependence) transmission (Roberts 1996). In frequency-dependent, genderless models (e.g., Horan and Wolf 2005; McCallum, Barlow, and Hone 2001), reducing the host density has no effect on prevalence rates. In the present gender-based model, this feature does not arise because relative densities and prevalence rates matter for cross-gender transmission.

Some observations can be made about the contact and transmission parameters in the present case. First, some degree of density dependence is likely for within-gender transmission (i.e., $\epsilon_{ii} > 0$) (Schauber and Woolf 2003). In contrast, frequency dependence is more likely for cross-gender transmission (i.e., $\epsilon_{ij} = 0$) due to sexual segregation among deer. This is because cross-gender infectious contacts are primarily limited to the breeding season for species such as deer that exhibit sexual segregation (Ramsey et al. 2002), and frequency-dependence is generally used to model sexually transmitted diseases (McCallum, Barlow, and Hone 2001). We therefore assume frequency-dependent cross-gender transmission, even though bTB is not truly sexually transmitted. Second, larger...
home ranges, sparring, and physiological stress make males more susceptible to bTB infection (O’Brien et al. 2002), implying that $\beta_{MM} > \beta_{FF}$ and $\beta_{FM} > \beta_{MF}$. This explains why males currently have a higher prevalence rate.

The final component of population growth is disease-induced mortality, which only affects infected subpopulations. Denote this mortality rate by $\alpha_i$. Supplemental feeding may decrease the effective mortality rate. Total mortality due to the disease is therefore specified as $\alpha_i(1 - \chi_f I_i)$, where $\chi_i$ is a parameter.

It is more intuitive and mathematically convenient to work in terms of the variables $N_i$ and $\theta_i$ instead of $S_i$ and $I_i$, where $\theta_i = I_i / N_i$ is the infected proportion of subpopulation of $i$. Noticing that $N_i = S_i + I_i$ and $\theta_i / \theta_i = I_i / I_i - N_i / N_i$, and given the specification described above, the system of equations (5) and (6) can be written as

$$
\dot{N}_M = (N_F \phi b - \delta_M N_M)(1 - [(N/k)(1 - \tau_f)]^i) \\
- \alpha_M(1 - \chi_M f)\theta_M N_M - h_M
$$

(8)

$$
\dot{N}_F = (N_F(1 - \phi)b - \delta_F N_F) \\
	imes (1 - [(N/k)(1 - \tau_f)]^i) \\
- \alpha_F(1 - \chi_F f) \\
	imes \theta_F N_F - h_F
$$

(9)

$$
\dot{\theta}_M = (\theta_F v - \theta_M)\phi(1 - [N_F/N_M]) \\
\times (1 - [(N/k)(1 - \tau_f)]^i) \\
- \alpha_M(1 - \chi_M f)\theta_M (1 - \theta_M) \\
+ (1 - \varepsilon_{MM} + \varepsilon_{MM} N_M)(1 + \omega_f)\beta_{MM} \\
\times (1 - \theta_M)\theta_M + (1 - \varepsilon_{FM} + \varepsilon_{FM} N) \\
\times (1 + \omega_f)\beta_{MF}(1 - \theta_M)\theta_F N_F / N
$$

(10)

$$
\dot{\theta}_F = (1 - \phi)b\theta_F (v - 1) \\
\times (1 - [(N/k)(1 - \tau_f)]^i) \\
- \alpha_F(1 - \chi_M f)\theta_F (1 - \theta_F) \\
+ (1 - \varepsilon_{FF} + \varepsilon_{FF} N_F)(1 + \omega_f)\beta_{FF} \\
\times (1 - \theta_F)\theta_F + (1 - \varepsilon_{MF} + \varepsilon_{MF} N) \\
\times (1 + \omega_f)\beta_{MF}(1 - \theta_F)\theta_M N_M / N.
$$

(11)

As with equation (2), $h_i$ does not affect $\dot{\theta}_F$ or $\dot{\theta}_M$ directly, but indirectly affects prevalence changes through its affects on $N$. For given values of $f$ and the state variables, the $\hat{\theta}_i = 0$ isoclines can be solved for

$$
\dot{N}_i = \Phi(f, \theta_F, \theta_M, N_i) \\
i = F, M, \quad j \neq i
$$

(12) which represents a host-density threshold as a function of feeding, both prevalence rates, and the other gender’s population level. Disease prevalence is increasing ($\dot{\theta}_i > 0$) for values of $N_i > \hat{N}_i$, and prevalence is decreasing ($\dot{\theta}_i < 0$) for values of $N_i < \hat{N}_i$. Disease eradication will result if the population $N_i$ is kept below the threshold for long enough, but the threshold will vary over time as it depends on both state and control variables. In this sense, the threshold for subpopulation $i$ is endogenous because it depends on feeding as well as the size of subpopulation $j$ (which can be affected by harvests) and the prevalence rates of both subpopulations (each of which are indirectly influenced by harvests of both subpopulations and feeding). Moreover, unlike thresholds arising in genderless models, as defined by equation (3), the thresholds defined by equation (12) are endogenous even in cases where feeding is not a choice variable because harvests can still influence the magnitude of subpopulation $j$ as well as both prevalence rates.

**Economic Specification**

The economic specification is a modified version of Horan and Wolf’s (2005) model. Recreational hunters value wildlife harvests, but males are valued more highly than females (Loomis, Updike, and Unkel 1989). Larger average size, scarcity, and trophy value may be contributing factors to this difference in value. The value placed on healthy animals of gender $i$ is denoted $p_i$. For all animals this is not less than the constant marginal utility from harvesting infected wildlife, $p_{iI}$, i.e., $p_i \geq p_{iI}$. Although harvests are nonselective, harvest values depend on health status because infected animals are identified through postmortem testing, required in Michigan (e.g., from lesions inside the carcass or examination of the tonsils). Let $p_{iI} = (1 - y_i)p_i$, where $y_i$ is the proportional loss in value due to the disease. The total value of harvests is $p_M h_M (1 - y_M \theta_M) + p_F h_F (1 - y_F \theta_F)$.

Assume harvests occur according to the Schaefer harvest function and that the unit cost of effort, $c$, is constant and independent of the targeted gender. Then total harvesting costs for gender $i$, restricted on the in situ
stocks, are \((c/q_i)h_i/N_i\), where \(q_i\) is the catchability coefficient. Supplemental feed is taken to have a constant per unit cost, \(w\).

Finally, consider the damage costs of the disease to the livestock sector. Denote the variable economic damages caused by infected deer as \(D(\theta_F N_F + \theta_M N_M)\), where \(D\) is a parameter defining marginal damages. These variable damages are due to infections in the cattle herd that result in lost stock, increased testing, and business interruption loss.6

### Optimal Management

We analyze management from the perspective of a social planner, such as MDNR. It is worth noting that MDNR utilizes ecological models to develop management strategies, and has shown concern over livestock-sector damages. MDNR employed McCarty and Miller’s (1998) model of bTB in Michigan deer to predict future bTB prevalence under alternative management scenarios, and estimated regression models to predict impacts to cattle (Hickling 2002). Such models are generally used to search for disease eradication strategies. In contrast, bioeconomic models are used to develop strategies that maximize economic surplus, and inform managers if eradication is socially desirable.

MDNR has focused on harvests and feeding as its primary control variables (Hickling 2002), having implemented a feeding ban and encouraged higher harvest rates (particularly among female deer) in DMU 452. We adopt these choice variables, but we relax current policy prescriptions so that we may derive the solution is efficient from Michigan’s point of view in the absence of regulatory impositions. Ignoring these costs only slows eradication.

subject to the equations of motion (8)–(11).7

The current value Hamiltonian is

\[
H = \sum_{i=M,F} \left[ p_i (1 - y_i \theta_i) h_i - c h_i/(q_i N_i) \right] - w f - D \left( \sum_{i=M,F} \theta_i N_i \right) + \sum_{i=M,F} \left[ \lambda_i N_i + \mu_i \theta_i \right]
\]

where \(\lambda_i\) and \(\mu_i\) are costate variables associated with \(N_i\) and \(\theta_i\), respectively.

The marginal impact of harvests by gender \(i\) on the Hamiltonian is

\[
\partial H/\partial h_i = p_i (1 - y_i \theta_i) - c/q_i N_i - \lambda_i \quad i = M, F.
\]

The RHS of expression (15) is the linear coefficient of \(i\)th gender harvests in the Hamiltonian. When this expression is positive so that marginal rents exceed marginal user cost, \(h_i\) should be set at its maximum. No harvests should be undertaken when the expression is negative. The singular solution is pursued when this expression equals zero, so that marginal rents and marginal user costs are equated. These tradeoffs are identical to those arising in genderless models, except that there are now two conditions instead of one. Conditions (15) may therefore be singular or nonsingular for both genders at once, or singular for one gender and not the other.

The marginal impact of feeding on the Hamilton is

\[
\partial H/\partial f = -w + \sum_{i=M,F} \lambda_i \partial N_i/\partial f + \sum_{i=M,F} \mu_i \partial \theta_i/\partial f.
\]

The RHS of expression (16) is the linear coefficient of feeding in the Hamiltonian. The first RHS term is the marginal cost of providing supplemental feed. The second RHS term is the marginal benefit of feeding due to its role in increasing deer productivity. The third term is the marginal cost of feeding

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6 Treating damages as an endogenous function of the livestock sector would add state variables and unnecessarily complicate the analysis. Also, we ignore lump sum costs from federal and interstate regulatory impositions in response to the disease (but see Horan and Wolf 2005), so the solution is efficient from Michigan’s point of view in the absence of regulatory impositions. Ignoring these costs only slows eradication.

7 Following Swallow (1990), Brock and Xepapadeas (2002), and others dealing with multistate variable problems, problem (13) is constructed as a linear control problem to simplify the exposition and analysis. Bounds are required for the linear processes. We implicitly assume that \(f \leq \min(1/\lambda, 1/\sigma, 1/\omega)\); this is explicit in the numerical example.
due to its role of increasing disease prevalence. Specifically, increased feeding reduces the host-density threshold for each population, resulting in increased prevalence *ceteris paribus*. Hence, supplemental feeding can be viewed as an investment in both the productivity of deer stock and of the disease.

If the RHS of expression (16) is positive, then feeding is optimally set at its maximum level, \( f^\text{max} \). If the expression is negative, then \( f = 0 \) is optimal. The singular solution for \( f \) should be followed whenever the RHS of condition (16) vanishes, thereby equating the marginal benefits and marginal costs of feeding. Like harvests, supplemental feeding is nonselective with respect to health status. Unlike harvests, supplemental feeding is nonselective with respect to gender. Condition (16) differs from condition (15) in that it is not gender-specific, and therefore takes a similar form to the related condition in genderless models (Fenichel and Horan 2008; Horan and Wolf 2005).

The conditions for an interior optimal solution involve four adjoint equations that may be expressed as “golden rule” equations that must hold at each point in time:

\[
\rho = \left( \frac{\partial \hat{N}_i}{\partial N_i} + \frac{\lambda_i \partial \hat{N}_j}{\lambda_i N_i} \right) + \left[ \frac{\mu_j \partial \hat{\theta}_i}{\lambda_i \lambda_j N_i} + \frac{\mu_j \partial \hat{\theta}_j}{\lambda_j} \right] + \frac{\hat{\lambda}_i}{\lambda_i} + \frac{1}{\lambda_i q_i N_i^2} \frac{D\theta_j}{\lambda_i} \quad i, j = M, F, \quad i \neq j.
\]

Equation (17) equates the opportunity cost of holding the deer *in situ* (\( \rho \), rate of return from elsewhere in the economy) to the rate of return from holding deer in subpopulation \( i \) *in situ* (the RHS). The first RHS term (in parentheses) is the marginal productivity effect of subpopulation \( i \) on the growth of both subpopulations. The second RHS term (in brackets) is the impact of a larger subpopulation \( i \) on changes in disease prevalence in both subpopulations: a larger subpopulation \( i \) creates more opportunities for within-gender and cross-gender infectious contacts. Alternatively, a larger subpopulation \( i \) represents an adverse change relative to its own host-density threshold, *ceteris paribus*, and it has the adverse effect of reducing the host-density threshold for gender \( j \). This endogenous-threshold effect does not arise in genderless models. The remaining RHS terms represent, respectively, the additional marginal benefits of investing in a larger subpopulation \( i \) (i.e., the capital gain and marginal cost savings from having a larger subpopulation \( i \)) less the additional marginal costs (i.e., marginal livestock damages).

Equation (18) is the adjoint condition associated with disease prevalence within subpopulation \( i \). This condition equates the marginal benefit from investing elsewhere in the economy (\( \rho \)) to the marginal cost of redirecting resources away from disease control (the RHS). The first RHS term is the marginal impact of increased prevalence within subpopulation \( i \) on the growth of subpopulation \( j \) (note there is no impact on \( j \)). The second RHS term (in brackets) is the marginal impact of increased prevalence within subpopulation \( i \) on changes in disease prevalence within both subpopulations: a larger prevalence within subpopulation \( i \) creates more opportunities for infectious within-gender and cross-gender contacts. An alternative way to view the cross-gender term is that a larger prevalence within subpopulation \( i \) has the adverse effect of reducing the host-density threshold for gender \( j \). Finally, the remaining RHS terms represent, respectively, the capital loss from having greater prevalence among subpopulation \( i \), and marginal damages to hunters and the livestock industry resulting from greater prevalence.

The adjoint equations reflect the same trade-offs as in the genderless case, with two exceptions. First is the fact that there are now four states to manage (and hence four adjoint equations) as opposed to two in the genderless model. The second exception is the addition of the cross-gender marginal effects in the current framework. The cross-marginal impacts on changes in prevalence are particularly important because these reflect how changes in the state variables associated with one subpopulation impact the host-density threshold associated with the other subpopulation. These differences, relative to genderless models, yield more flexibility in controlling the disease.

The optimal solutions for control variables in a linear control problem are feedback rules,
with the optimal values at each point in time depending on the current state (Conrad and Clark 1987), i.e., \( h_i = h_i(N_M, N_F, \theta_M, \theta_F) \) and
\[
f = f(N_M, N_F, \theta_M, \theta_F). \]
From (12), this means the optimal host-density thresholds are also state-dependent, in contrast to thresholds derived from the next-generation matrix \( K \).

The optimal feedback rules can involve various combinations of singular or nonsingular controls, depending on the current state. A fully unconstrained, or triple-singular, solution is pursued when both (15) and (16) vanish. Partial-singular solutions emerge when one or two control variables are constrained, that is one or two of the conditions (15) and (16) vanish. Partial-singular solutions arise within a blocked interval, a period of time when one or more of the controls is “blocked” or constrained from following the triple-singular path (Arrow 1968; Clark 1990). Finally, a fully constrained or “bang–bang” solution is pursued when all control variables are constrained.

With three control variables, there are too many combinations of potential partial and fully singular solutions to analytically derive each solution type here (see Fenichel and Horan [2008] for the procedure in a genderless model). Moreover, the overall solution may involve a series of free and blocked intervals over time, with each interval corresponding to a different type of partial or fully singular solution. The potential combinations of possibilities renders analytical analysis intractable, particularly since analysis of when to pursue blocked or free intervals is inherently numeric (Arrow 1968).

Numerical Example

We now illustrate the Michigan bTB problem numerically. The parameterization is described in the Appendix. While we have made every effort to calibrate the model realistically, research on this problem is evolving. As knowledge of many parameters is limited, the following analysis is best viewed as a numerical example rather than a case study. Nonetheless, the results shed light on the economics of wildlife disease control. Furthermore, this example allows us to make qualitative comparisons to prior work based on a genderless host population (Horan and Wolf 2005; Fenichel and Horan 2008).

A discrete-time approximation was used to solve problem (13) numerically. The discrete-time model is specified identically to the continuous-time model, except that the discrete-time form of the cost function is
\[
(c_i/q_i)\ln[N_i/(N_i - h_i)] \quad \text{(Conrad and Clark 1987)}.
\]
While this function is nonlinear in harvests, results based on this specification are known to approximate those of linear, continuous-time models as in problem (13) (Clark 1976).

The discrete-time problem was solved using the AD Model Builder software (Otter Research). The optimization was run for a 150-year planning horizon. Initially, no steady state assumptions were imposed so that the system could progress to either an interior (disease) solution or eradication. We found the disease is optimally eradicated for our base case scenario. To verify these results, we resolved the model using the procedure outlined by Conrad (1999), in which final period welfare equals the net present value of a steady state in which each subpopulation’s harvests and net growth are equated. The results are essentially the same as in the unconstrained case, except social net benefits are slightly higher (as expected) when the steady state is accounted for.

Figure 1 shows the optimal time paths of the state variables and host-density thresholds, until a disease-free equilibrium is attained. Figure 2 shows the optimal time paths of the control variables over the same interval. An initial feeding pulse coincides with pulse harvests of both subpopulations (with males experiencing two pulse harvests). These initial pulses (Phase I) are analogous to most rapid approach paths (along a blocked interval) to a singular solution (Phase II), which would emerge in a continuous-time version of the model. The “singular” value for each subpopulation is not unique, however, and changes over time as the other states change (see Horan and Wolf 2005).

The initial reduction in population levels, in conjunction with reduced feeding, leads to reduced prevalence in both subpopulations (Phase II). This trend generally continues for much of the first decade. Female prevalence drops quickly and smoothly as \( N_F \) remains well below its optimal host-density threshold, \( \hat{N}_F \), during this phase. In contrast, male prevalence falls in an oscillatory fashion during the first decade as \( N_M \) fluctuates around its optimal host-density threshold, \( \hat{N}_M \). Specifically, \( N_M < \hat{N}_M \) whenever \( \theta_M > \theta_F \) over the first decade. Starting from such a point, \( \theta_M \) is reduced below \( \theta_F \) in the next period, and \( \hat{N}_M \) is
Figure 1. Optimal time series for populations, $N_i$, optimal host-density thresholds, $\hat{N}_i$, and prevalences, $\theta_i$.

Reduced so that $\hat{N}_M < N_M$. Accordingly, $\theta_M$ increases above $\theta_F$ in the next period and the oscillations continue (but with diminished magnitude). Mathematically, the oscillations are due to the pseudo-vertical transmission term in equation of motion (10): the coefficient $(\theta_F v - \theta_M)$ is positive when female prevalence is relatively higher and negative otherwise. Pseudo-vertical transmission plays an important role here because there is significant year-to-year turnover within the male population since $N_M$ is maintained at such low levels. Indeed, a high rate of male turnover is common in deer populations as few bucks survive past 18 months of age (O’Brien et al. 2002). Sensitivity to changes in $v$ is examined in the next section.

Although prevalence declines over the first decade, it is too costly to continue to eradication because both subpopulations would have to remain at low levels, resulting in small and costly harvests. Moreover, the lower prevalence rates reduce livestock-sector damages. It
therefore becomes optimal to increase feeding to boost both subpopulations, which enables greater harvests and reduces hunting costs to increase near-term hunting benefits. Around year twelve, the system enters a period where both subpopulations exceed their host-density thresholds, and prevalence rates slowly rise (Phase III).

At first, it is optimal to allow increased prevalence. However, as prevalence continues to increase it becomes optimal to again reduce feeding and population levels (Phase IV) to reduce damages (although damage reductions are offset somewhat as prevalence continues to rise). The reductions in feeding and population continue until prevalence starts to decline around year 25 (Phase V). In contrast to Phase II, prevalence in both subpopulations is reduced in a relatively smooth fashion until it is eradicated. The smooth reduction in $\hat{N}_M$ is facilitated by choosing control variables to maintain $\hat{N}_M$ at levels greater than, but very close to, $N_M$ (figure 1(A)). This tempers downward adjustments in male prevalence and maintains $\theta_M > \theta_F$ (figure 1(C)), thereby eliminating the “male turnover” effect described above. The female population is also managed closer to its host-density threshold, $\hat{N}_F$, during this phase (figure 1(B)). Two things happen, relative to Phase II, to bring $N_M$ and $N_F$ closer together. First, $N_M$ is increased due to increased feeding, enabling greater harvests and reducing hunting costs to increase hunting benefits. Second, $\hat{N}_F$ is reduced due to both increased feeding and prior management. Managing populations and host-density thresholds closer together means disease eradication takes longer, but the cost associated with this is more than offset by the increased harvests made possible by feeding-induced increases in deer productivity.

Note that it is optimal to differentially target each subpopulation, as each has a distinct role in transmission risks and each is valued differently. Males are almost always targeted much more stringently than females because males are both more valuable and more susceptible to disease. The degree of male targeting is limited by costs, however. In this regard, targeting females generates an important indirect benefit associated with $\hat{N}_M$. The male host-density threshold is increased when there are fewer females, *ceteris paribus*. Therefore, reducing $N_F$ reduces the pressure to harvest males, as the disease can dissipate at a larger

Figure 2. Optimal time series for male harvests, $h_M$ (black line, Panel A), female harvests, $h_F$ (gray line, Panel A), and feeding, $f$ (Panel B)
male population and at lower male harvesting costs. Of course reducing the number of males has a similar effect on the female host-density threshold, and this is taken into account when determining relative control levels. However, reducing females to increase the male threshold generates larger marginal benefits given the large harvesting costs associated with the smaller male stock.

The disease eradication result differs from results of genderless models, in which the optimal solution is an interior cycle in \( N \theta \) space (Horan and Wolf 2005; Fenichel and Horan 2008). However, our results prior to Phase V are similar to those of prior work, particularly Fenichel and Horan (2008). The optimal path in \( (N_F, \theta_F) \) space is presented in figure 3 (a similar graph can be constructed in \( (N_M, \theta_M) \) space, but the path is more chaotic due to the oscillations described above). The curve \( f = f^{\text{max}} \), which is interpolated from the numerical results, represents a boundary to the right of which feeding is optimally constrained at \( f^{\text{max}} \). Similar boundaries are illustrated by Horan and Wolf (2005) and Fenichel and Horan (2008), except the boundary in figure 3 is conditional on \( N_M \) and \( \theta_M \) (not shown) and is therefore not stationary. The present \( f = f^{\text{max}} \) boundary is best viewed as a heuristic device for comparing results across the different models.

The optimal path in figure 3, as well as in Fenichel and Horan (2008), begins with a population cull to jump out of the region of constrained feeding (Phase I). Next, a curved path is followed along which prevalence is first reduced (Phase II) and then increased along with the population (Phase III). This leads back to the \( f = f^{\text{max}} \) boundary, which is followed before heading back to an interior solution (Phase IV). Prevalence increases briefly before the second disease control phase (V) is pursued.

The qualitative results of the two models diverge at this point. Fenichel and Horan’s (2008) optimal path cycles back to a stationary \( f = f^{\text{max}} \) boundary, resulting in a stable cycle. In contrast, Phase V in figure 3 leads to eradication. Eradication is pursued because gender-specific harvests allow greater (although not perfect), and hence less costly, control over the levels of the subpopulations, their respective host-density thresholds, and prevalence levels. By the start of Phase V these values are in position to make eradication optimal. Note that eradication was not optimal during Phases I and II due to the imperfect control of the thresholds. Those phases can be viewed as a case of overshooting: there were benefits to quickly reducing prevalence, but accomplishing this resulted in a low male threshold and low deer productivity. Continued prevalence reduction under these circumstances was too costly, and so an additional adjustment period was required to put the pieces into place to ease the burden of eradication.

Finally, our result that feeding levels remain significant during Phase V contrasts with the results of Horan and Wolf’s (2005) genderless model (and with the current ban on feeding). Horan and Wolf (2005) find optimal feeding levels are zero or very small whenever

![Figure 3. “Phase plane” of the female stock and female disease prevalence, with heuristic \( f^{\text{max}} \) boundary](http://ajae.oxfordjournals.org/)

Downloaded from http://ajae.oxfordjournals.org/ at Michigan State University on April 17, 2014
prevalence declines because reduced feeding is the only way to reduce prevalence in that (frequency-dependent) model. The current result, however, lends further support to the results of Fenichel and Horan (2008), who find reliance on feeding controls is reduced (so that feeding optimally increases) when population controls are an additional tool to indirectly affect prevalence (under density-dependence). Here we find improved population targeting requires even less reliance on habitat management: more feeding occurs over most of the time path in the present case than in Fenichel and Horan (2008) because feeding provides \textit{in situ} productivity benefits, while damages may be managed more effectively through targeted population controls.

\textbf{Sensitivity Analysis}

We now investigate the sensitivity of our results to values of initial states and parameters. Results are summarized in table 1. To begin, we note that our result of an eradication path involving more stringent controls of males is independent of initial population values. An example involving a significantly larger initial male population is given in table 1. In this case, the larger male subpopulation reduces the marginal benefits of feeding on population growth, but it increases the marginal disease impacts of feeding. In the context of figure 3, the $f = f_{\text{max}}$ boundary shifts to the right and the system starts with less feeding, reducing welfare. However, less initial feeding results in a smaller initial adjustment and much less overshooting: the system proceeds directly to eradication, increasing welfare. Indeed, the eradication time is cut by 61% and there is a small (5%) increase in welfare (table 1).

Next, consider a 10\% increase in $\zeta$, so that deer productivity is everywhere increased and the peak of the growth curve moves right. This increases the marginal benefits of feeding, resulting in more feeding. Increased deer productivity also decreases the marginal cost of female population control, shifting population controls from males to females. Both effects increase welfare and the time to eradication (table 1).

Now consider a 10\% reduction in $\varepsilon_{FF}$, which reduces female contacts (male contacts are unaffected; it is more likely that contact rates among males would exceed those of females, O’Brien et al. 2002). This reduces the marginal disease impacts of feeding, resulting in more feeding. Without the increase in feeding, a smaller $\varepsilon_{FF}$ would reduce the eradication time. However, this is more than offset by the additional feeding. In addition, a smaller $\varepsilon_{FF}$ increases the female host-density threshold, leading to a shift in population controls from females to males. The net result is a 12\% increase in welfare and a 17\% increase in eradication time.

Changes in three economic parameters where also investigated. First, marginal damages where reduced by 20\%, reflecting a

\begin{table}[h]
\centering
\caption{Sensitivity Analysis Results}
\begin{tabular}{|l|c|c|}
\hline
Scenario & \% Change in Welfare & Years to Eradication \\
\hline
Baseline & - & 87 \\
$N_0$ increased to 5,296 & 5.39 & 34 \\
$v$ reduced 10\% & 10.65 & 82 \\
$\zeta$ increased 10\% & 5.89 & 96 \\
$\varepsilon_{FF}$ reduced 10\% & 12.35 & 102 \\
Damages reduced 20\% & 11.48 & 112 \\
$p_F$ reduced 20\% & -2.25 & 100 \\
y reduced 20\% & 9.93 & 120 \\
Feeding ban & -39.33 & 18 \\
\hline
\end{tabular}
\end{table}
situation where ranchers take actions to mitigate damages. This reduces disease control incentives, decreasing optimal host-density thresholds and also the incentives to reduce population levels relative to these thresholds. In particular, population controls shift slightly from males to females, increasing the time to eradication. The net result is a 29% increase in eradication time and an 11% increase in welfare (due to decreased damages; note that if $D$ is sufficiently reduced, eradication never occurs). Qualitatively similar results occur when $y$, which represents damages to hunters, is reduced by 20%. The third economic parameter we considered a 20% reduction in the marginal value of doe harvests. This reduces the marginal benefits of targeting does for disease control, reducing welfare and increasing the time to disease eradication.

Finally, suppose feeding is constrained at zero, reflecting the current feeding ban. Clearly it is optimal to eradicate the disease when there is no feeding, as eradication is optimal even when there is a higher level of feeding. However, welfare is substantially reduced given this constraint, even though the eradication time is significantly reduced (table 1).

**Discussion and Conclusion**

Game managers traditionally use a gender-targeted approach to managing wildlife production, and this could be extended to manage wildlife health. The wildlife-pathogen system in this context can be viewed as a multiple-host-pathogen system, in contrast to the single-host systems that have been the focus of prior bioeconomic work and much of the ecological literature. In a single-host context, management options are generally restricted to lowering aggregate host density, and an important implication is that disease eradication may not be optimal because the management tools being applied are highly inefficient. Conversely, in previous publications on multiple-host pathogen systems, eradication is assumed to be desirable, “targeting” is based only on whether a population is a disease reservoir, and the importance of benefit-cost tradeoffs are ignored (Roberts and Heesterbeek 2003).

In this article, we show how management options can be improved when the disease problem is viewed as a bioeconomic multiple-host system. Specifically, we examine the implications of targeting an observable trait correlated with infection (gender). The primary result is that multiple, endogenous host-density thresholds emerge in the multiple-host system case (in contrast to single-host systems). We find that harvesting males affects the degree to which females need to be harvested to reduce female disease prevalence, and vice versa. Hence, a manager is not simply managing a population in relation to its host-density threshold, as is suggested in the ecological literature, but rather manages both the populations and the thresholds simultaneously. This expands the disease management options, which improves efficiency relative to situations in which only the aggregate population is considered. The result is that disease eradication is optimal in the multiple-host example provided here, whereas it was not optimal in prior single-host models based on the same example.

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**References**


**Appendix: Model Calibration**

The following gender-independent parameters are taken from Horan and Wolf (2005): \(c/q = 231,192\), \(w = 36.53\), \(D = 5,491\), \(f_{\text{max}} = 10,000\), \(k = 14,049\), \(\tau = 8.0 \times 10^{-5}\), \(\omega = 2.64 \times 10^{-6}\), and \(N_0 = 13,298\) (the initial value of \(N\)). We also assume \(\phi = 0.5\), \(v = 1\), and \(\rho = 0.05\).

Gender-specific ecological parameters are computed as follows. The average gender ratio of deer in and around the core was estimated to be 3.035 females per male (Sitar 1996). Given this ratio and \(N_0\), we compute the initial subpopulation levels \(N_{M0} = 3,296\) and \(N_{F0} = 10,022\). Data on yearly birth rates (Sitar 1996) is used to set \(b = 1.22\). Natural mortality was computed using survival estimates (Sitar 1996) and hunting mortality (McCarty and Miller 1998) resulting in \(\delta_i = 0.3623\).

Estimates of initial disease prevalence are \(\theta_{F0} = 0.02\) and \(\theta_{M0} = 0.08\), which are believed to have remained fairly constant in recent years (O’Brien et al. 2002; McCarty and Miller 1998). We use Miller and Corso’s (1999) rates of infected contact by gender, along with survival rates from the time of contact to that of infection to obtain the effective contact rates \((1 - \varepsilon_{IM} + \varepsilon_{IM} N_i) \beta_{IM} (1 + \omega f) = 0.672\) and \((1 - \varepsilon_{IF} + \varepsilon_{IF} N_i) \beta_{IF} (1 + \omega f) = 0.1855\). Assume \(\varepsilon_{ii} = 1\) (density-dependent within-gender transmission) and \(\varepsilon_{ij} = 0\) (frequency-dependent cross-gender transmission). Then we can solve for \(\beta_{MF} = 0.1816\), \(\beta_{FF} = 1.186 \times 10^{-5}\), \(\beta_{FM} = 0.6577\), and \(\beta_{MM} = 1.996 \times 10^{-4}\). Using the procedure outlined by Horan and Wolf (2005), we derive \(\alpha_F = 0.3797\), \(\alpha_M = 0.8205\), \(\chi_F = 5.8 \times 10^{-5}\), and \(\chi_M = 9.0 \times 10^{-5}\). The relation \(\alpha_M > \alpha_F\) is consistent with O’Brien et al. (2002).

Now consider gender-specific economic parameters. Using Horan and Wolf’s (2005) marginal value of a harvested deer, \(p = $1,270.80\), and applying the relative values reported by Loomis, Updike, and Unkel (1987), we compute \(p_M = $1,534\) and \(p_F = $936\).