NATURAL RESOURCE MODELING
Volume 20, Number 4, Winter 2007

JOINTLY-DETERMINED ECOLOGICAL THRESHOLDS AND ECONOMIC TRADE-OFFS IN WILDLIFE DISEASE MANAGEMENT

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ABSTRACT. We investigate wildlife disease management, in a bioeconomic framework, when the wildlife host is valuable and disease transmission is density-dependent. Disease prevalence is reduced in density-dependent models whenever the population is harvested below a *host-density threshold*—a threshold population density below which disease prevalence declines and above which a disease becomes epidemic. In conventional models, the threshold is an exogenous function of disease parameters. We consider this case and find a steady state with positive disease prevalence to be optimal. Next, we consider a case in which disease dynamics are affected by both population controls and changes in human-environmental interactions. The host-density threshold is endogenous in this case. That is, the manager does not simply manage the population relative to the threshold, but rather manages both the population and the threshold. The optimal threshold depends on the economic and ecological trade-offs arising from the jointly-determined system. Accounting for this endogeneity can lead to reduced disease prevalence rates and higher population levels. Additionally, we show that ecological parameters that may be unimportant in conventional models that do not account for the endogeneity of the host-density threshold are potentially important when host density threshold is recognized as endogenous.

1. Introduction. A large number of human, livestock, and companion animal diseases have their origins with wildlife (Cleaveland

Received by the editors on June 23, 2005, and in revised form on March 17, 2006.

et al. [2001]). Many of these diseases have the potential to inflict large damages on society, but management may be costly (Daszak et al. [2000], Wobeser [2002] and Leighton [2002]). Wildlife managers therefore are faced with making trade-offs when determining how many resources to invest in disease management.

The solution to wildlife disease problems might be straightforward if it were easy to distinguish between healthy and infected individuals, so that infected animals could simply be culled (albeit at a cost). However, healthy and infected animals are often indistinguishable prior to harvest and postmortem testing is required, as the outward signs of an illness often take a long period of time to manifest (Lanfranchi et al.). In other words, harvests are necessarily nonselective with respect to disease status. This feature, which is ubiquitous in ecological models of wildlife disease, e.g., see Smith and Cheeseman [2002], does not necessarily eliminate harvests as a valuable management tool, however. A disease can be eradicated if the aggregate wildlife population is harvested below a *host-density threshold*—a threshold population density below which disease prevalence declines and above which a disease becomes epidemic—provided a threshold exists, McCallum et al. [2001]. A host-density threshold can only exist when disease transmission is density-dependent, because reducing the population in such cases reduces the number of infected contacts. When the population is below this threshold, the number of infectious contacts is sufficiently reduced that the disease dissipates naturally.[1]

The ecological literature on wildlife disease typically has not been concerned with how humans affect infectious wildlife disease dynamics apart from population control (Wobeser [2002]). Indeed, most studies focus primarily on identifying the level of human initiated population control to eradicate disease (Barlow [1991b], Barlow [1996], Wolfe et al. [2004], Caley and Ramsey [2001], Smith and Cheeseman [2002]). But human-environmental interactions apart from population control may greatly affect the disease transmission process (Daszak et al. [2000] and Wobeser [2002]). Human-environmental interactions can be large-scale landscape changes such as deforestation, which may have large impacts on wildlife disease emergence (Daszak et al. [2001]), or they can be smaller scale human-environmental interactions that alter habitat and wildlife behavior such as supplemental feeding programs. Feeding wildlife has been implicated as a key factor in the outbreak of disease

among garden birds (Hartup et al. [2000]) and wild deer (Schmitt et al. [2002]). In particular, supplemental feeding of deer has been shown to change deer behavior (Grenier et al. [1999]) and contact rates between individuals (Garner [2001]).

The primary purpose of this paper is to investigate the disease management problem, in a bioeconomic framework, when the wildlife host is of recreational value and the disease dynamics can be affected by both population controls and changes in human-environmental interactions (specifically, changes in supplemental feeding). The model is applied to the case of bovine tuberculosis (bTB), *Mycobacterium bovis*, in Michigan white-tailed deer, *Odocoileus virginianus*. We find that human-environmental interactions make the host-density threshold endogenous, whereas it is an exogenous function of disease parameters in conventional models that do not account for these interactions (McCallum et al. [2001]).

Ecologists and economists generally accept that economic and ecological systems are jointly determined, that is, human choices affect the state of ecological systems, and the state of ecological systems in turn affect the incentives that humans face for exploiting or conserving ecosystems (Tschirhart [2000], Shogren et al. [1999], and Sanchirico and Wilen [2001a, b]). Recognizing these linkages and system feedbacks is important for developing wildlife disease control strategies, yet the theme of jointly determined ecological-economic systems is only beginning to emerge in the wildlife disease literature, e.g., Bicknell et al. [1999] and Horan and Wolf [2005], and furthermore there has been no recognition that the host-density threshold might be endogenous.

For instance, Horan and Wolf [2005] explore the social planner's problem for managing bTB in Michigan white-tailed deer, where the planner chooses the economically optimal level of deer harvests and supplemental feeding. Supplemental feeding in their model, as in ours, boosts deer productivity but also increases the rate of disease transmission and reduces the rate of disease mortality. However, there is no host-density threshold in their model because they do not model density-dependent disease transmission. This means that population controls do not affect disease prevalence in Horan and Wolf's [2005] model. Rather, in their model it is only possible to reduce disease prevalence by keeping feeding at a low level for a sufficient period of

time (or by eradicating the deer herd, which would eliminate the disease and a valuable resource).

Horan and Wolf's [2005] use of a nondensity-dependent disease transmission function is consistent with some research on the Michigan bTB problem (McCarthy and Miller [1998]), but others say that density-dependent transmission is important based on theoretical considerations (Hickling [2002]). Model selection based on data is problematic because the data often do not exist for a range that enables robust model selection (Roberts [1996]). Therefore, the selection of the form of the transmission function needs to balance both empirical evidence and theoretical principles (Begon et al. [2002], McCallum et al. [2001], Roberts [1996]). In this paper we adopt a transmission function that exhibits a degree of density-dependence and has been suggested as superior to more extreme approaches (Roberts [1996]). This results in significantly different management options than were available in the Horan and Wolf model, as both population management and feeding have an impact on disease levels in the present model. Moreover, the level of feeding affects the host-density threshold and hence the degree to which population controls must be used to manage the disease. In other words, the endogeneity of the host-density threshold presents managers with the problem of managing the threshold in addition to the host population density.

The implications reach beyond wildlife disease problems, as ecological thresholds are important in other settings such as the establishment of invasive species (With [2004]) and hysteresis problems in ecological processes such as nutrient dynamics in lakes (Mäler et al. [2003]). This work also expands the growing bioeconomic literature on managing ecologically interdependent species, e.g., Bulte and van Kooten [1999], Brock and Xepapadeas [2002], Bulte and Damania [2003], Mesterton-Gibbons [1987, 1996] and Finnoff and Tschirhart [2003], which primarily focus on population management. In our model (with the interdependent species being the wildlife host and the pathogen), we also consider the role of human activities that influence species interactions.

A second contribution of the current paper is to show that ecological parameters that may be unimportant in conventional models having an exogenous host-density threshold are potentially important when the threshold is endogenous. Specifically, we consider the role of the pseudo-vertical transmission rate, i.e., transmission between mothers

and offspring, which some have said is unimportant for the predictive ability of disease models (Barlow [1993]). We verify that this parameter is unimportant when feeding does not enter into the model, but we illustrate its potential importance when feeding plays a role.

A third and final contribution is an analysis of a special case of the model in which feeding does not play a role. Here we characterize the economically optimal dynamics for a conventional model of wildlife disease, that is, one incorporating density-dependent transmission (unlike Horan and Wolf's [2005] model). In contrast to the ecological literature that focuses mainly on disease eradication, e.g., Barlow [1991b, 1996], Wolfe et al. [2004], Caley and Ramsey [2001], Ramsey et al. [2002], Roberts [1996], Smith and Cheeseman [2002], and Smith et al. [2001], we find that eradication may not be optimal. Rather, a unique steady state having a positive disease prevalence rate might exist.

We begin this paper by revisiting disease ecology theory and incorporating the effects of supplemental feeding on host population and disease dynamics. In Section 3 we discuss the endogeneity of the host-density threshold. Then in Section 4 we develop a bioeconomic model and describe the economic and ecological trade-offs that characterize an economically optimal solution. This is followed by a numerical model of bTB in Michigan white-tailed deer. Section 6 provides the results of some sensitivity analysis. The final section concludes.

2. A model of wildlife disease with human-environmental interactions.

2.1. *Background on modeling approach and motivating example.* While the purpose of this paper is to illustrate the potential importance of human-environmental interactions for wildlife disease management problems, there is no general way in which such interactions affect disease dynamics because there are many different ways in which these interactions can occur (as mentioned in the Introduction). We must therefore focus on a particular type of problem. Accordingly, the analytical and numerical models are developed to focus on the Michigan white-tailed deer case, where supplemental feeding is considered an important contributing factor in disease transmission. This example is not entirely case-specific, however, as supplemental feeding is an im-

portant issue in many wildlife and disease settings, and the types of impacts that supplemental feeding has on the host population and disease dynamics, i.e., increasing wildlife productivity, increasing disease transmission and reducing disease mortality, would be indicative of a broader class of problems involving human-environmental interactions.

Bovine TB among Michigan white-tailed deer is primarily concentrated in a four-county area in the northeastern part of the lower peninsula, formally designated as deer management unit (DMU) 452 (see Hickling [2002], MDA [2002], Schmitt et al. [2002]). This is the only known area in the United States where bTB has become established in a wild deer population, and conventional wisdom held that the disease was not self-sustaining in wild deer populations (Hickling [2002]). Indeed, while a few cases of infection have been found beyond this area, the disease does not appear to be sustainable outside DMU 452. This has led many to speculate that unique, area-specific features such as human-environment interactions, particularly feeding programs that encourage deer to congregate, have enabled the disease to become endemic (Hickling [2002]). These feeding programs have been sponsored by several hunt clubs in DMU 452. The historic density of deer in the area is estimated to have been seven to nine deer per square kilometer (O'Brien et al. [2002]). The hunt clubs, desiring greater density, began aggressive deer feeding programs to encourage herd growth resulting in deer density increasing to an estimated 25 deer/km^2 by the mid-1990s.

The disease has spread from the deer herd to local livestock, and so Michigan agriculture is concerned about disease-related costs and supports culling the deer population 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.

2.2. *Mathematical details of the ecological model.* Assume the aggregate host population, N, is closed and exists on a fixed land area. This is a reasonable assumption for the Michigan white-tailed deer case, as the deer migrate very little (Garner [2001], Hickling [2002]) and the region is bordered by major roadways to the south and west, and by Lake Huron to the east and north. Indeed, the Michigan Department of Natural Resources manages deer in the infected region as a unique population, and it estimates little risk of spread (Hickling [2002]). The

host population consists of susceptible (S) and infected (I) individuals, i.e., $N = S + I$. There is no recovered population, as bTB and many other wildlife diseases are chronic with no recovery (Barlow [1991b]).

Changes in S and I are written as

$$\dot{I} = G(I, N, f) + T(S, I, f) - A(I, f) - hI/N \tag{1}$$

$$\dot{S} = Z(S, I, N, f) - T(S, I, f) - hS/N \tag{2}$$

where G and Z are density-dependent growth functions and G includes pseudo-vertical transmission, i.e., mother-to-offspring transmission before or shortly after birth. T represents horizontal transmission (transmission not passed from mother to off-spring), A represents disease-induced mortality, h is aggregate harvest and f is supplemental feeding. Equations (1) and (2) are standard (Smith and Cheeseman [2002]), except for the role of f.

Define G to be a modified form of logistic growth: $G = I(vb - \delta)[1 - (N/k)(1 - \tau f)]$. The first modification relative to standard logistic growth involves pseudo-vertical transmission applied to offspring of infected hosts (Barlow [1991a]). To model this, the intrinsic growth rate, r, is first split into the per-capita birth rate, b, and per-capita mortality rate, δ (as $r = b - \delta$). Next, the birth rate is multiplied by the rate of pseudo-vertical transmission, v. Pseudo-vertical transmission is not modeled explicitly in many disease models, and Barlow [1993] states that v has little affect on the predictive ability of disease models. We model v to show that it can matter when supplemental feeding is incorporated, making the host-density threshold endogenous.

The second modification to the growth function involves the effects of feeding on the carrying capacity. Carrying capacity depends on resource availability, where only the most limiting resource constrains population growth. Specifically, the carrying capacity is defined as $\min\{k_1, \ldots, k_n\}$, where k_i represents the carrying capacity associated with the ith resource, e.g., food, cover, water. These carrying capacities can be increased by human investments that make the resources less limiting. In the case of food availability, the carrying capacity is an increasing function of supplemental feeding: $k_i = k_i(f)$ with $k_i'(f) > 0$. Assume food is the most limiting resource for $f < f^{\max}$, and denote the effective carrying capacity by $k/(1 - \tau f)$, where k is the carrying

capacity when $f = 0$ and $t < 1/f^{\max}$ is a parameter. Supplemental feeding therefore increases the effective carrying capacity in a manner consistent with Walters [2001]. As feeding is costly and only provides productivity benefits for $f < f^{\max}$, the upper bound on feeding will be $f^{\max}$.[2]

Similarly, define Z to be the following modified logistic form: $Z = [rS+bI(1-v)][1-(N/k)(1-\tau f)]$. Z differs from G only in the net birth term, $[rS + bI(1 - v)]$. Specifically, rS accounts for the fact that all births to susceptible animals are also susceptible, and the term $bI(1-v)$ represents the offspring of infected animals that escape pseudo-vertical transmission.

Next consider the transmission function, T. Following conventional models (Diekmann and Heesterbeek [2000] and Heesterbeek and Roberts [1995]), transmission is defined as

$$T(S, I) = C(N)\beta SI \tag{3}$$

where β represents the conditional probability of infection in a susceptible individual given contacts between infectious and susceptible individuals, and $C(N)$ represents these contacts (hence, $\beta C(N)$ is the probability that a susceptible individual becomes infected). $C(N)$ and β are generally modeled as deterministic. In particular, we define C to be a modified form of the function proposed by Roberts [1996]

$$C = \frac{(1 - \varepsilon + \varepsilon N)}{N}. \tag{4}$$

Here $\varepsilon \in [1, 0]$ is a parameter that indicates the degree of density dependence in horizontal disease transmission. When $\varepsilon = 1$, then $C = 1$ and T simplifies to the classic density-dependent model of transmission (McCallum et al. [2001]). When $\varepsilon = 0$, then $C = 1/N$ and T represents frequency-dependent (nondensity-dependent) transmission. Reality probably lies somewhere in between these two extremes (Schauber and Woolf [2003]), with $\varepsilon \in (0, 1)$ so that there is a degree of density-dependence in transmission.[3]

We include feeding in the contact function to account for Schmitt et al.'s [2002] observation that feeding alters social interaction and generally concentrates deer, also see Grenier et al. [1999]. Specifically,

assume feeding enters C so that transmission becomes

$$T(S,I,f) = \frac{(1-\varepsilon+\varepsilon N)(1+\omega f)}{N}\,\beta SI, \tag{5}$$

where ω is a parameter.

Now consider mortality due to the disease. We define $A = \alpha(1-\chi f)I$, where α is the disease-induced mortality rate and χ is a parameter. Changes to the environment, such as feeding, may decrease the effective mortality rate by lowering the energy requirements to find food (other types of habitat change may have the reverse effect).

Finally, consider the harvest terms in equations (1) and (2). It is often difficult or impossible to identify infected wildlife prior to harvest (Lanfranchi et al. [2003]). Harvests are therefore nonselective with respect to disease status, as is the case of bTB in Michigan (O'Brien et al. [2002]) as well as other wildlife disease situations (Smith and Cheeseman [2002]). Assuming the disease is uniformly distributed among the population, this results in the number of deer harvested in a particular health class being equal to the proportion of deer in that health class multiplied by the total harvests, h. The assumption of nonselective harvesting, modeled in this manner, is standard in virtually all ecological models of wildlife disease, e.g., Smith and Cheeseman [2002], Barlow [1991b].

Given the model specification, it is intuitively easier and mathematically more convenient to work in (N,θ) space, where $\theta = I/N$ is the disease prevalence rate. Noticing that and $\dot{N} = \dot{S} + \dot{I}$ and $\dot{\theta}/\theta = \dot{I}/I - \dot{N}/N$, the system of equations (1) and (2) can be written as

$$\dot{N} = rN\left(1 - \frac{N(1-\tau f)}{k}\right) - \alpha(1-\chi f)\theta N - h \tag{6}$$

$$\begin{aligned}\dot{\theta} = {} & b(1-v)\left(\frac{N(1-\tau f)}{k} - 1\right)\theta \\ & + [\beta(1+\omega f)(1-\varepsilon+\varepsilon N) - \alpha(1-\chi f)](1-\theta)\theta.\end{aligned} \tag{7}$$

Equation (6) says that net changes in the aggregate population are due to surplus growth, less disease-related mortality, less harvests. Equation (7) says that net changes in disease prevalence arise due to pseudovertical transmission, plus the net difference between horizontal transmission and disease-related mortality. Conventional wildlife disease

models do not include the pseudo-vertical transmission term, e.g., McCallum et al. [2001], either because v has been set equal to unity (so that the term vanishes) or else because pseudo-vertical transmission is not explicitly considered (in which case v implicitly equals unity). Finally, note that h does not affect $\dot{\theta}$ directly. However, h does affect $\dot{\theta}$ indirectly through its affects on N, provided $v = 1$ and $\varepsilon = 0$ do not both hold, in which case $\dot{\theta} = \beta(1 + \omega f)(1 - \theta)\theta$ such that neither N nor h influences $\dot{\theta}$.

3. Ecological thresholds. Assuming $\varepsilon > 0$, changes in disease prevalence will depend on the host population level, and a host-density threshold exists and may have important management implications. To analyze this threshold, first consider the conventional case in which $v = 1$ and $f = 0$ (as this is consistent with situations where the level of feeding is not explicitly considered). The host-density threshold, denoted $\widehat{N}$, is then determined by solving the $\dot{\theta} = 0$ isocline for N: $\widehat{N} = k[\alpha + \beta(\varepsilon - 1)]/\beta k\varepsilon$, which is independent of θ. In this restricted case, the $\dot{\theta} = 0$ isocline drawn in (N, θ) space is a vertical line at $\widehat{N}$. Disease prevalence is increasing ($\dot{\theta} > 0$) for values of $N > \widehat{N}$, and prevalence is decreasing ($\dot{\theta} < 0$) for values of $N < \widehat{N}$. Hence, disease prevalence will decline towards zero if the population is kept below the threshold, $\widehat{N}$. This has been the rationale for many culling or density management programs.

Now consider the case where $f > 0$ but $v = 1$ still holds (the focus of our numerical example), so that the threshold becomes $\widehat{N}(f) = k[\alpha(1 - \chi f) + \beta(\varepsilon - 1)(1 + \omega f)]/[\beta k\varepsilon(1 + \omega f)]$. Now $\widehat{N}$ is a function of f, which is not necessarily fixed. For this simple case it is easily verified that $\partial\widehat{N}/\partial f < 0$: an increase in f reduces the host-density threshold so that a smaller population is required for the disease to die out. This results because feeding increases the rate of change in prevalence by increasing transmission while decreasing disease-related mortality. Since supplemental feeding affects the host-density threshold, the disease manager's problem is not simply to manage the population in relation to the threshold, but rather to manage the population and the threshold simultaneously. Different strategies for doing this imply different economic and ecological trade-offs, and a planning agency

interested in managing the disease and hunting interests must assess the trade-offs that emerge.

Before turning to a bioeconomic model to evaluate these trade-offs, we consider one last specification: $v < 1$. In this case, the host-density threshold is defined in its most general form as

$$\widehat{N}(f,\theta) = \frac{(1-v)kb + k[\alpha(1-\chi f) + \beta(\varepsilon - 1)(1+\omega f)](1-\theta)}{(1-v)(1-\tau f)b + \beta k\varepsilon(1+\omega f)(1-\theta)}, \tag{8}$$

which is now a function of both f and θ. The relationship between f and $\widehat{N}$ is now analytically ambiguous due to a fertility effect that at least partially counteracts the horizontal transmission and mortality effects. The reason is that a lower v reduces vertical transmission so that increased feeding increases the recruitment of healthy animals by more than that of infected animals, resulting in a negative impact on prevalence.

An example is illustrated in Figure 1. Using the parameter values from Table 1 and setting $\theta = 0.025$, we find that the host-density threshold is almost unique when $f = 0$, for any value of v. That is, the value of v does not influence the threshold when $f = 0$, supporting Barlow's [1993] statement that v does not matter.

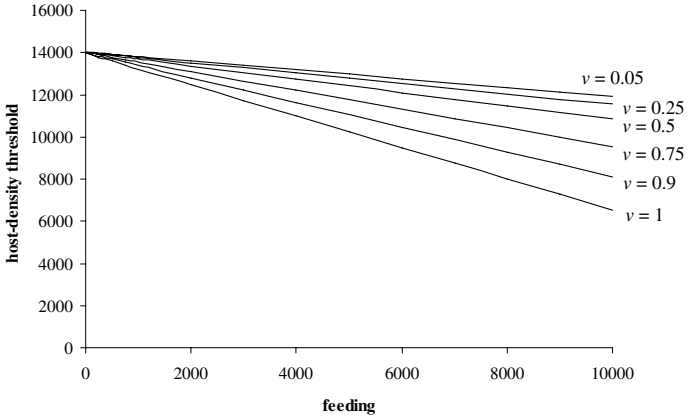

FIGURE 1. The relationship between the host-density threshold and feeding when $\theta = 0.025$.

TABLE 1. Parameter values and sources.[a]

Parameter	Description	Value	Source
N_0	initial population size	13,298	Hill [2002]
θ_0	initial prevalence	0.023	O'Brien et al. [2002]
r	intrinsic rate of growth	0.5702	Rondeau & Conrad [2003]
δ	per-capita mortality rate	0.3623	Sitar [1996]
k	carrying capacity	14,049	Miller et al. [2003]; O'Brien et al. [2002]
τ	coefficient for feeding effect on k	8.0×10^{-5}	Miller et al. [2003]
β	transmission coefficient	3.39×10^{-5}	Miller & Corso [1999]; McCarty & Miller [1998]
ω	coefficient for feeding effect on β	2.64×10^{-6}	Miller et al. [2003]
ε	contact coefficient	0.75	assumption
v	rate of pseudo-vertical transmission	1	assumption
α	disease induced mortality rate	0.3556	Hill 2002; Miller et al. [2003]
χ	coefficient for feeding effect on α	5.32×10^{-5}	Hill 2002; Miller et al. [2003]
p	value of harvested healthy deer	1270.80	Boyle et al. [1998]; Frawley [1999]; U.S. DOI-FWS
y	proportional reduction in the value of infected deer relative to healthy deer	1	assumption
c/q	marginal harvesting cost/ catchablity coefficient	231,192	Rondeau & Conrad [2003]
w	unit cost of feeding	36.53	Miller et al. [2003]
D	marginal damages to the livestock sector	5491	Wolf & Ferris [2000]
ρ	discount rate	0.05	assumption

[a] Some values are derived based on data presented in the original source. See Horan and Wolf [2005] for details on many of the derivations.

However, $\partial \widehat{N}/\partial f < 0$ for all values of v, with the marginal effect of changes in f on $\widehat{N}$ being much greater when v is larger, as might be expected due to the counteracting fertility effect when v is small.[4] Hence, v matters when $f > 0$, and it matters more the larger is f. Although we concentrate on the case of $v = 1$ in the numerical example, we explore the effect of changes in v in the sensitivity analysis section of the paper.

4. A bioeconomic model.

4.1. *Economic specification and optimality conditions.* Suppose a manager wants to control wildlife population levels and disease prevalence rates in a manner that maximizes the discounted net economic benefits to society. These net benefits include net benefits to hunters less the damage costs associated with infections to the livestock sector. Hunters gain utility from the actual process of shooting deer and/or consuming meat and other deer products. Given readily accessible substitutes, i.e., healthy deer, in other nearby regions, the (constant) marginal utility from harvesting healthy deer is denoted p, which is not less than the (constant) marginal utility from harvesting infected deer, p_I, i.e., $p \geq p_I$. Although harvests are nonselective because infected animals cannot be identified prior to the kill, economic values do depend on health status because infected animals can be identified after the kill, e.g., from lesions present inside the carcass or else from an examination of the tonsils, which has been required for all deer harvests in the infected region in Michigan. For simplicity, let $p_I = (1-y)p$, where y is the proportional loss in value due to the disease. The benefits from hunting are therefore $phS/N + (1-y)phI/N = p(1-y\theta)h$.

Assume harvests occur according to the Schaefer harvest function (although in general this specification is not required), and that the unit cost of effort, c, is constant.[5] Then total harvesting costs, restricted on the *in situ* stocks, are $(c/q)h/N$, where q is the catchability coefficient. The unit cost of food is w.

Finally, the costs of the disease, particularly to farmers and related agribusinesses, must also be considered. Denote the variable economic damages caused by infected deer by $D(I)$. These variable damages are due to infections in the cattle herd that result in lost stock,

increased testing, and business interruption loss.[6] We use a linear damage function in the numerical example, $D(I) = DI = D\theta N$, where D is a parameter representing marginal damages (although in general this specification is not required).

Assuming a discount rate of ρ, the social planner's problem is

$$\max_{h,f} \int_0^\infty \Big[ph(1-y\theta) - \frac{ch}{qN} - wf - D\theta N\Big] e^{-\rho t}\, dt \tag{9}$$
$$\text{subject to (7), (8);} \quad N(0) \quad \text{and} \quad \theta(0) \quad \text{given.}$$

Problem (9) is a linear control problem since the objective function and constraints are all linear in the control variables, h and f.

To solve the planner's problem, we first define the current value Hamiltonian

$$H = ph(1-y\theta) - \frac{ch}{qN} - wf - D\theta N + \lambda \dot{N} + \mu \dot{\theta} \tag{10}$$

where λ and μ are the co-state variables associated with the host population, N, and disease prevalence, θ, respectively. The marginal impact of harvests on the Hamiltonian is given by

$$\frac{\partial H}{\partial h} = p(1-y\theta) - \frac{c}{qN} - \lambda. \tag{11}$$

The right-hand side (RHS) of expression (11) is the linear coefficient of harvests in the Hamiltonian. If this expression is positive so that marginal rents, $p(1-y\theta) - c/(qN)$, exceed the marginal user cost, λ, then larger harvests only increase the value of the Hamiltonian; hence, harvests should be set at their maximum levels, $h^{\max}$. If this expression is negative, then harvests should not occur. The singular solution is pursued when the expression vanishes.

Now consider the marginal impacts of feeding on the Hamiltonian

$$\frac{\partial H}{\partial f} = -w + \left\{ \lambda N \left(\frac{rN\tau}{k} + \alpha\chi\theta \right) + \mu \frac{-b(1-v)\theta N\tau}{k} \right\} \tag{12}$$
$$+ \mu\big((1-\varepsilon+\varepsilon N)\beta\omega + \alpha\chi\big)(1-\theta)\theta.$$

The RHS of expression (12) is the linear coefficient of feeding in the Hamiltonian. If this expression is positive, then feeding should be set

at its maximum level, $f^{\max}$. If the expression is negative, then $f = 0$ is optimal. The singular solution for feeding should be followed whenever the RHS of condition (12) vanishes. To understand when this occurs, it is useful to think of feeding as an investment in both the productivity of the resource and of the disease. The singular solution should be followed whenever the unit cost of feeding equals the *in situ* net marginal value of feeding on the two state variables. The *in situ* net marginal value is the difference between the marginal benefits of feeding on the overall stock (the second term within the curly brackets; specifically, increased productivity, decreased mortality, and, when $v < 1$, a fertility-related reduction in θ when f is increased at the margin) and the marginal costs of feeding in terms of an increased proportion of infected animals (due to increased transmission and decreased mortality among the infected stock, the third term). Conventional models do not treat feeding as a choice variable, in which case f would be treated as fixed without consideration of the sign of condition (12). Such an outcome would necessarily be inefficient. In the numerical example below, we analyze a case in which f is fixed so as to better illustrate the implications of choosing f optimally (relative to more conventional models where this does not occur).

The overall solution to problem (9) will be a set of harvest and feeding choices over time, which in turn results in an optimal path for the state variables N and θ. Along the optimal path, three types of solutions might arise at different points in time. The first type is known as a double-singular solution, and it arises when conditions (11) and (12) simultaneously vanish, so that singular solutions arise for both control variables. The second type of solution is known as a partial-singular solution, which arises when only one of the conditions (11) or (12) vanishes, so that a singular solution only arises for a single variable. Partial-singular solutions arise as part of a blocked interval, a period of time during which one of the controls is "blocked" or constrained from following the double-singular path (Arrow [1968] and Clark [1990]). A potential third type of solution is a fully constrained solution when neither condition (11) or (12) vanishes.

Regardless of the type of solution, e.g., doubly or partially singular, an optimal solution also requires the following two adjoint equations to

be satisfied:

$$\dot{\lambda} = \rho\lambda - \frac{\partial H}{\partial N} \tag{13}$$

$$\dot{\mu} = \rho\mu - \frac{\partial H}{\partial \theta}. \tag{14}$$

These conditions prevent intertemporal arbitrage opportunities: if they were not satisfied, then gains could be made from reallocating harvests or feeding across time, in which case the solution would not be intertemporally optimal. These equations may be manipulated into two "golden rule" equations that must hold at each point in time:

$$\begin{aligned}\rho = {} & \left\{ r - \frac{2rN(1-\tau f)}{k} - \alpha(1-\chi f)\theta \right\} + \frac{\dot{\lambda}}{\lambda} + \frac{ch}{\lambda q N^2} - \frac{D\theta}{\lambda} \\ & + \frac{\mu}{\lambda}\left[b(1-v)\,\frac{(1-\tau f)\theta}{k} + \beta(1+\omega f)\,\varepsilon(1-\theta)\theta \right]\end{aligned} \tag{15}$$

$$\begin{aligned}\rho = {} & \left\{ b(1-v)\left(\frac{N(1-\tau f)}{k} - 1 \right) \right. \\ & \qquad \left. + [\beta(1+\omega f)(1-\varepsilon+\varepsilon N) - \alpha(1-\chi f)](1-2\theta) \right\} \\ & + \frac{\dot{\mu}}{\mu} - \frac{phy + DN + \alpha(1-\chi f)N\lambda}{\mu}.\end{aligned} \tag{16}$$

Consider condition (15). The left-hand side (LHS) is the discount rate, which represents the rate of return elsewhere in the economy, or the opportunity cost of leaving deer *in situ*. The first term on the RHS of condition (15) (in {}) is $\partial\dot{N}/\partial N$, or the stock's own marginal growth as a result of it being a reproducible asset. The second RHS term of conditions (15) represents the capital gains to holding the stock *in situ*, i.e., the rate of growth in the marginal value of the stock. The third RHS term is the marginal savings in harvesting costs from having more deer at the margin (as deer are less costly to find when they are more abundant). The fourth RHS term is the marginal damages associated with a larger deer population (as the infected stock is expected to increase along with the aggregate population). The final RHS term is the (imputed) marginal costs of increased transmission (as more deer leads to more infectious contacts). This term is zero in Horan and

Wolf's [2005] model with frequency-dependent transmission and $v = 1$. Relative to that model, the effect of this term is to reduce the RHS of (15) which, for a given discount rate, implies that N should be larger in the present model, everything else being equal.

Condition (16) is also a "golden rule" expression, although it has a slightly different interpretation. Here, the discount rate represents the opportunity cost of pulling resources from elsewhere in the economy and using them to manage the disease. The RHS represents the rate of return to controlling the disease. We again compare the results with Horan and Wolf's [2005] model with frequency-dependent transmission, i.e., $\varepsilon = 0$, and $v = 1$. As v is decreased, the RHS of (16) is decreased: less pseudo-vertical transmission means there are fewer gains to disease control investments. To maintain equality of (16) as v is reduced, other RHS terms must therefore increase. Other things being equal, we would expect an increase in θ when horizontal transmission outweighs mortality and vice versa when mortality outweighs horizontal transmission (to see this, totally differentiate (16) to obtain $d\theta/dv$). The opposite result occurs when ε is increased: increased density-dependent transmission means population management has greater impacts on disease control, thereby increasing the benefits of disease control.

Conditions (15) and (16) are used to derive the feedback rules that characterize the double-singular and partial-singular solutions. We denote the feedback rules for the singular values of the controls by $h(N, \theta)$ and $f(N, \theta)$, respectively, but note that the actual functions will generally differ depending on whether we are discussing a double-singular or partial-singular solution. The process for deriving these feedback rules is outlined in the Appendix.

5. Numerical example. We now examine the optimal solution numerically because the feedback rules and the differential equations that define the solution are too complex to analyze analytically. Moreover, the choice of whether to pursue a free interval solution or a blocked interval solution is inherently numerical (Arrow [1968]). The software package Mathematica 5.1 (Wolfram Research) was used to arrive at the numerical solution, using the data in Table 1 to parameterize the model. We have used the best available data for the Michigan bTB case, however research on this system is still evolving from a fairly

early stage and so knowledge of many parameters is somewhat limited. The following analysis is therefore best viewed as a numerical example rather than a prescription for optimal management of the Michigan bTB situation. Nonetheless, the results shed light on the economics of wildlife disease management in general and specifically on bTB in Michigan deer.

5.1. *Optimal management when supplemental feeding is fixed.* We begin with a special case in which f is held constant. This is interesting in its own right, but also provides a baseline for comparison with the case in which f is chosen optimally, so that we may highlight the role of feeding. We fix $f = f^{\max}$, as this represents a situation with substantial supplemental feeding activities, much like the situation in Michigan prior to the implementation of feeding controls. Also, the solution to the $f = f^{\max}$ case is equivalent to the partial singular solution arising when $f = f^{\max}$ in the more general model (in which f is chosen optimally), and so the current discussion will inform the analysis of the more general model.

Given the initial states of the world, N_0 and θ_0, the planner must choose whether to set $h = 0$, $h = h(N, \theta)$ (the singular value of h), or $h = h^{\max}$. This problem is similar to the one posed by Mesterton-Gibbons (MG) [1987], where nonselective harvest levels were chosen to optimally manage two noninteracting populations. A general version of this technique has been used to solve problems where species interact through competitive and predator-prey relationships (Mesterton-Gibbons [1996]). Our model involves two interacting populations, which could be thought of as bacteria and deer interacting in a host-parasite relationship.[7] We follow the basic approach outlined by MG and consider each solution type in turn. Trajectories for the different solution types are presented in Figure 2 as dark curves with arrows. Broken curves represent either isoclines or boundaries that pertain only to the singular solution, and these are presented in each panel to introduce a sense of scale relative to the singular solution.

The first possibility we consider is to set $h = h^{\max}$ until an equilibrium is reached. The trajectories for this case, which are illustrated in Figure 2a, all lead to extinction of the deer. Such a strategy would be infinitely costly given our cost function, and would therefore be dominated by any outcome in which welfare is finite.

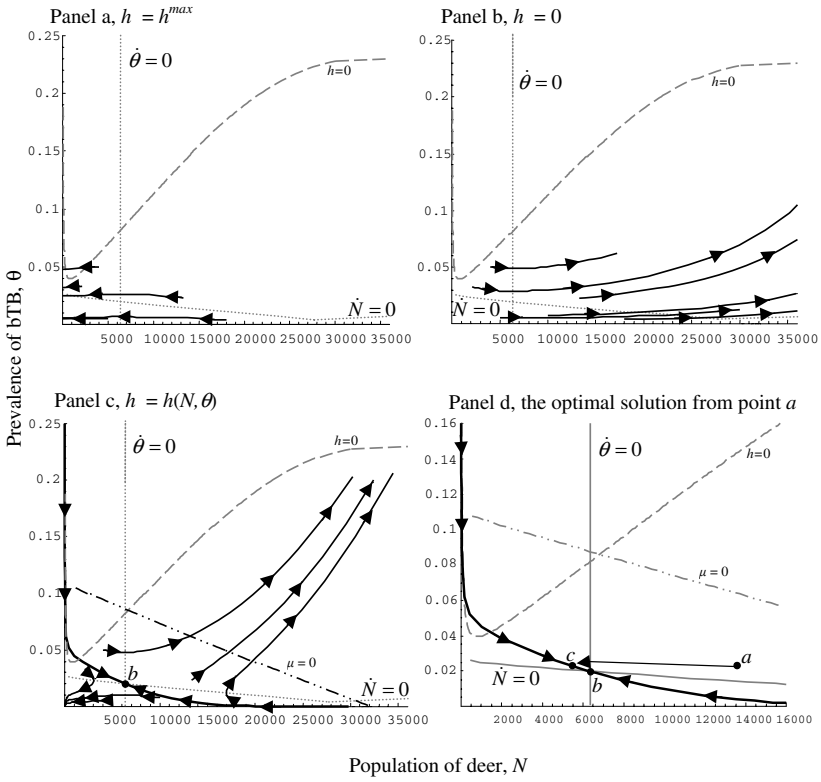

FIGURE 2. The trajectories given the application of different harvest rules when $f = f^{\max}$ and is not managed.

The second possibility is to set $h = 0$. The trajectories for this case, given different starting values, are illustrated in Figure 2b. With no harvests, but with positive disease prevalence levels, discounted net benefits will be negative along any of these trajectories. Moreover, the ultimate outcome is the steady state point $N = k/(1 - \tau f)$, $\theta = 1$, which does not satisfy the necessary conditions for optimality. Evaluating equation (15) at this point and setting $\dot{\lambda} = 0$, we can solve for $\lambda = -D/[\rho - r + \alpha(1 - \chi f^{\max})] < 0$. Then imposing $\partial H/\partial h < 0$ (which must hold along an optimal path when $h = 0$), we find that the following condition must hold along an optimal path,

$D/[\rho - r + \alpha(1-\chi f^{\max})] < c(1-\tau f^{\max})/k$. This condition is not satisfied numerically (for any reasonable parameter values), however, so it would be inefficient to remain on an $h = 0$ trajectory into perpetuity. Indeed, once $\theta = 1$, it can be verified numerically that the associated singular solution coincides with a steady state value of N that is less than the carrying capacity value, and that a jump to this point from any other value of N will be optimal.

Now consider the singular solution $h = h(N, \theta)$, trajectories which are illustrated in Figure 2c. Two separatrices lead away from unstable equilibria (one arising where $\theta = 1$ and another where $\theta = 0$) to an interior, saddle point equilibrium of $N = 6,355$, $\theta = 0.02$, defined by point b. Above this separatrix, trajectories lead past the $\mu = 0$ boundary (so that greater disease prevalence counter-intuitively has positive social value at the margin) and ultimately to the $h = 0$ boundary, beyond which $h = 0$ always holds. Our discussion above, as well as our intuition that μ should be nonpositive, indicates that it is not optimal to stay on such a trajectory. Similarly, it is not optimal to follow trajectories that lie below the separatrices, as these all lead to extinction.

An optimal plan is therefore similar to the one described by MG (who provides a rigorous proof for a similar class of problems), and illustrated in Figure 2d. For initial points above the separatrices, set $h = h^{\max}$ until the separatrix is reached and then follow the separatrix to the steady state. This is consistent with the strategy described above when $\theta = 1$. For initial points below the separatrices, set $h = 0$ until the separatrix is reached and then follow the separatrix to the steady state. Given our starting values $N(0) = 13,298$ and $\theta(0) = 0.023$, as defined by point a, the optimal solution is to jump to point c and the follow the separatrix to the steady state b.

The result that a positive level of disease prevalence is optimal runs counter to the disease eradication focus of the ecological literature. The economic intuition behind this result is that it is costly, in terms of foregone deer productivity, to maintain the deer population below the exogenously determined host-density threshold (the isocline) for a sufficiently long time as to let disease prevalence fall to zero.

In contrast, disease eradication is optimal for the opposite case, when $f = 0$. In this case, the host-density threshold shifts to the right, so

that disease prevalence falls for a larger range of population levels. In turn, the opportunity cost of waiting out the disease is reduced since the disease can be eliminated at larger population levels. However, the full opportunity cost of this strategy is not really being accounted for since feeding is being held at a fixed level.

5.2. *Optimal management when harvests and supplemental feeding are chosen optimally.* Now consider the optimal management strategy when both harvests and feeding levels are choice variables. The phase dynamics for the double-singular solution are illustrated in Figure 3 between the $f = 0$ and $f = f^{\max}$ boundaries, where these boundaries are defined as the loci of points for which $f(N, \theta) = 0$ and $f(N, \theta) = f^{\max}$. These loci of points, plotted as dotted lines, determine boundaries that divide the state space into three regions in which double and partial-singular solutions will emerge: $f = 0$ partial-singular solutions arise to the left of the $f = 0$ frontier; $f = f^{\max}$ partial-singular solutions arise to the right of the $f = f^{\max}$ frontier; and double-singular solutions arise in the interior region.

Next, we determine the $\dot{N} = 0$ and $\dot{\theta} = 0$ isoclines within each region. The isoclines in the $f = f^{\max}$ region are the same as those presented in Figure 2. These isoclines are shifted within the double-singular region because feeding is adjusted in response to the current states of N and θ. Note in particular the $\dot{\theta} = 0$ isocline in the double-singular region. This isocline, which represents the host-density threshold, is no longer vertical. Rather, it is a curve that reflects endogenous economic and ecological trade-offs. We expand on this in the following section.

The isoclines for the double-singular solution intersect in the interior of the double-singular region. This intersection defines an interior equilibrium at the point $N = 7,962$ and $\theta = 0.0113$. The eigenvalues of the differential equation system, linearized at the equilibrium point, are complex with positive real parts. This indicates that the equilibrium is an unstable focus, see Conrad and Clark [1987]. This means that it is only optimal to be at this point if the system starts at this point. Otherwise, it is optimal to spiral away from this point. There are no equilibria in any of the constrained regions.

We can now determine the optimal path given the starting values $N(0) = 13,298$ and $\theta(0) = 0.023$. The feedback rule associated with a

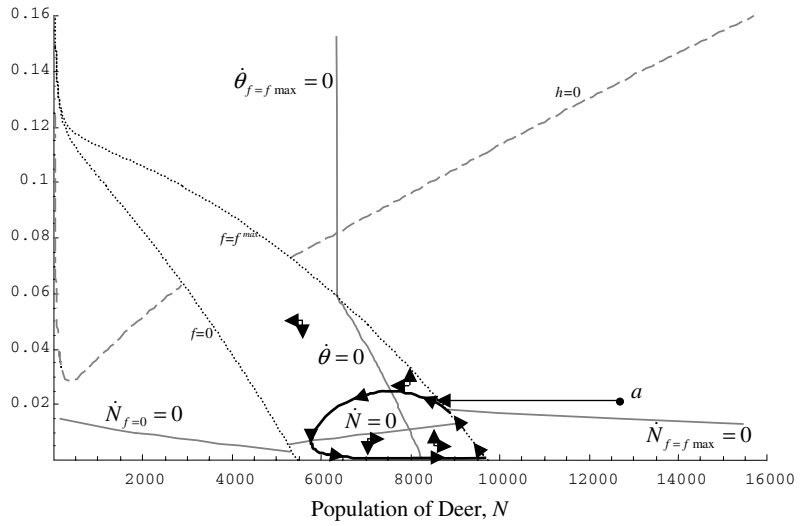

FIGURE 3. Phase-plane diagram illustrating the simulated dynamics and optimal trajectory form starting point a.

double-singular solution at these starting values results in $f > f^{\max}$. The system therefore begins in a constrained region, and so the partial-singular solution for $f = f^{\max}$ must be considered, as in Figure 2d. The initial point lies above the separatrix associated with the partial-singular solution for $f = f^{\max}$, and so a westward jump to the separatrix would be optimal if such a path led to the steady state associated with this partial singular solution. But it does not, as the separatrix disappears at the $f = f^{\max}$ boundary. Upon crossing the boundary, feeding is no longer constrained and so a double-singular solution becomes optimal. The optimal solution, therefore, is a "bang-bang" control with respect to the harvest—an instantaneous cull of the deer population that allows us to jump to the double-singular path that exists in the interior region.

The optimal trajectory in the interior region is governed by the local dynamics, indicated by the phase arrows. Given that the $\dot{\theta} = 0$ and $\dot{N} = 0$ isoclines intersect to form an unstable focus, the optimal trajectory must first move into the northeast quadrant of the interior region and then rotate around the focus point to intersect either the

$f = 0$ frontier, the N-axis, or the $f = f^{\max}$ frontier. If the optimal path intersects the N-axis when $N > 0$, the disease is eradicated and a healthy deer population remains. However, in the numerical example this does not occur. Rather, the optimal path misses the N-axis and swings back around to intersect the $f = f^{\max}$ frontier (at the point $N = 9,720$, $\theta = 3.7 \times 10^{-4}$), nearly but not fully eradicating disease. This result is highly parameter-dependent. Eradication may arise for some parameter combinations, while prevalence may remain significantly larger than zero for other parameter combinations. Figure 4, derived using a larger discount rate (so that less value is placed on future damages relative to the near-term productivity benefits of feeding), illustrates the latter case.

The optimal path travels along the $f^{\max}$ frontier by "chattering" between the constrained and unconstrained regions.[8] Chattering ceases once the system crosses the $\dot{N} = 0$ isocline (at the point $N = 8,827$, $\theta = 0.018$), sending the system back into the interior and resulting in a cyclical path, Figure 3.

The final part of the optimal path that must be determined is the initial cull. The "premature switching principle" suggests that it is optimal to cull directly to a point lying on the optimal cyclical path. Given the initial value of $\theta = 0.023$, this results in an initial cull of 5031 deer so that $N = 8,267$ (just to the left of the $f = f^{\max}$ threshold). It is interesting to note that a single cycle takes > 50 years in the simulation, indicating that optimal disease management likely involves a long-term commitment. This is not surprising given that it took 62 years to previously eliminate the disease in cattle herds under more controlled conditions, Frye [1995].

5.3. *Endogenous ecological and economic thresholds.* Recall the $\dot{\theta} = 0$ isocline represents the optimal host-density threshold, the value $N = \widetilde{N}(\theta)$ below which the disease dissipates, given values of θ. The expression for $\widetilde{N}(\theta)$ will differ from the expression for $\widehat{N}(\theta, f)$, the host-density threshold defined by equation (8) (although the *values* of these two expressions will be equivalent if $\widehat{N}$ is evaluated at the optimal value of f). The reason is that $\widehat{N}(\theta, f)$ is an ecologically determined threshold, given values of θ and f. In contrast, $\widetilde{N}(\theta)$ reflects both ecological and economic considerations, as it is endogenously determined based on the optimal choice level of feeding, $f(N, \theta)$.

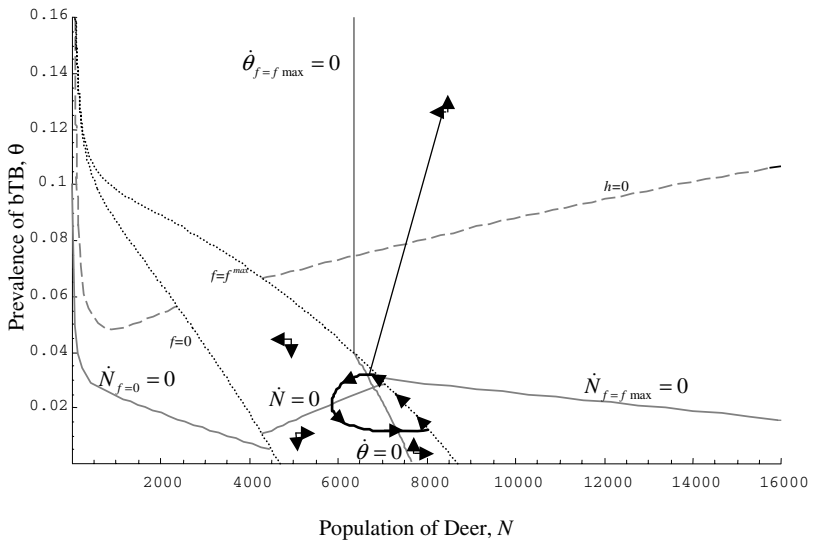

FIGURE 4. The optimal interior cycle when the discount rate is increased to 15%, holding all other parameters constant.

Indeed, $\widetilde{N}(\theta)$ is determined by plugging $f(N,\theta)$ into the expression $\dot{\theta} = 0$ and solving for N. Because the feedback rule $f(N,\theta)$ is derived based on economic-ecological trade-offs, the optimal host-density threshold, $\widetilde{N}(\theta)$, also reflects these trade-offs. Other choices of feeding would produce different host-density thresholds, but these thresholds would be suboptimal.

The endogeneity of the threshold can be seen by comparing the shapes of the $\dot{\theta} = 0$ isoclines in Figures 2 and 3. In contrast to the vertical isocline in Figure 2, the $\dot{\theta} = 0$ isocline is negatively sloped in Figure 3, as the value of f differs at each point on this curve. Specifically, the value of f on this curve is smaller for smaller values of θ and larger values of N, reflecting the control the manager has over the threshold as well as the trade-offs the manager makes between feeding and population controls. At higher prevalence rates, the manager prefers to reduce prevalence through population controls, as this has a secondary effect of significantly reducing damages at the margin while at the same time providing significant near-term harvesting benefits (which are heightened due to substantial rates of supplemental feeding). At

lower prevalence rates, population controls have little marginal impact on reducing damages and so reducing feeding becomes the preferred approach to further reducing prevalence.

Somewhat analogous to the endogenous host-density threshold is an endogenous, economic-based prevalence threshold. Specifically, the $\dot{N} = 0$ isocline within the double-singular region defines an economic-based prevalence threshold, $\theta = \hat{\theta}(N)$, below which it becomes optimal to increase feeding (and above which feeding is optimally declining).

Together, these two thresholds govern the cyclical management of the disease. Horan and Wolf [2005], in their model of frequency-dependent disease transmission, also find that an optimal path involves cyclical fluctuations in the deer population and disease levels. The intuition behind these fluctuations is the same in both models. Specifically, initial and intermittent future investments in deer productivity (via feeding) create opportunities for near-term gains. However, the investments also provide the unwanted side-effect of increased disease prevalence. Eventually, the damages due to increased prevalence would swamp the benefits from investment; therefore intermittent disinvestment of the disease is warranted. Of course, this also carries a cost in terms of lost productivity. So, after prevalence is reduced below the economic-based prevalence threshold, the benefits from investing in deer productivity again outweigh the costs of increased prevalence. Accordingly, feeding increases along with the deer population, and eventually prevalence follows so that the disease is not eradicated.

But while the intuition is the same in both models, the paths do differ as a result of the different disease transmission functions and the implications these have for management. In Horan and Wolf's [2005] model, feeding was eliminated along much of the optimal path because reductions in feeding were the only way to reduce prevalence. In the present model, a reduction in feeding also reduces prevalence but it has an additional effect: it increases the host-density threshold so that fewer population controls are needed to reduce prevalence. This additional effect helps to lower disease management costs, so that we would expect lower prevalence rates. Similarly, harvests create an additional benefit in the present model relative to the frequency-dependent model: they create future benefits in terms of disease reduction. These benefits are also expected to lower disease control costs, as well as reduce the pressure to limit feeding activities. The net result is that feeding and

population levels are both larger while disease prevalence is smaller in the present solution than in Horan and Wolf's solution.

6. Sensitivity analysis. Sensitivity analyses are commonly used to examine how changes in one or more parameters affect the solution. There are many parameters in the present model, and a sensitivity analysis could be performed for each of them. However, a new phase plane would have to be presented and examined for each new parameter scenario, and there are many potential scenarios that could be considered. Rather than working through changes for every parameter, we focus on two parameters. The first is a biological parameter where empirical and theoretical knowledge is significantly lacking: the rate of pseudo-vertical transmission, v. The second is the economic parameter, y.[9] Horan and Wolf [2005] explore changes in discount rates and economic parameters, and differences between the results of their base model and the alternative scenarios are qualitatively similar to the differences that would arise for the present model.[10]

The importance of the vertical or pseudo-vertical transmission rate has at times been downplayed. Barlow [1993] states that the pseudo-vertical transmission parameter has little affect on the predictive ability of a model of disease spread. This has led to a wide range of values used for parameters in bTB and other disease models. Indeed, authors have used rates spanning the unit interval, often due to a lack in data (Barlow [1991a, 1993, 1996], Roberts [1996], Fulford et al. [2002], and Smith and Cheeseman [2002]). One reason for including high rates of pseudo-vertical transmission is that sets of related animals are more likely to be infected than sets of unrelated animals, as is the case for deer with bTB (Blanchong [2003]). But still the actual rate is unknown. And while the choice of v may have only a small impact on the predictive ability of the model when the host-density threshold is exogenous, it is possible that the pseudo-vertical transmission rate may have significant impacts on the optimal management strategy if the endogeneity of the host-density threshold is recognized.

In order to gauge the potential impact of pseudo-vertical transmission on the optimally determined host-density threshold, the parameter v was reduced to $v = 0.95$. A decrease in v causes the optimal host-density threshold (the $\dot{\theta} = 0$ isocline) to shift to the right and to rotate slightly, Figure 5, indicating that disease prevalence is optimally

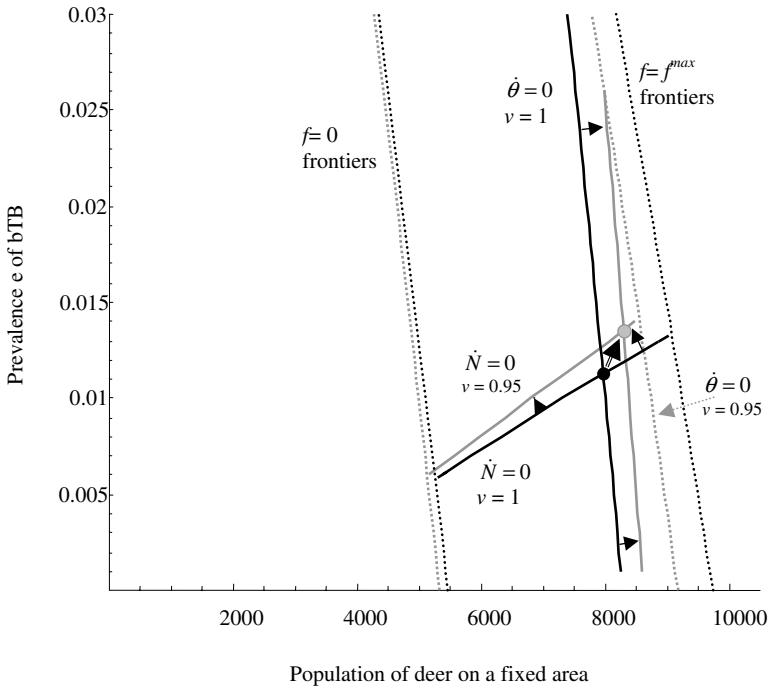

FIGURE 5. Phase plane showing how the $f = 0$ and $f = f^{\max}$ frontiers, and the $\dot{N} = 0$ and $\dot{\theta} = 0$ isoclines change when v is decreased from 1 (black) to 0.95 (gray). The phase dynamics are the same as those in Figure 2. The solid single arrows indicate how the isoclines shift, and the solid double arrow indicates how the focus point shifts with a decrease in v.

diminished at larger values of N. The reason is that a smaller v reduces vertical and, hence, overall transmission. The ecological threshold, $\widehat{N}(\theta, f)$, is therefore increased at each prevalence rate, for any value of f. This increase is offset somewhat (but not entirely) by an increase in supplemental feeding, $f(N, \theta)$, since the disease-related costs of feeding (in terms of increasing population growth and hence the number of infected offspring) are reduced along with vertical transmission. The net effect is therefore an increase in the optimal host-density threshold.

The value of v also impacts the economic threshold that defines when feeding should be increasing or decreasing, illustrated by the $\dot{N} = 0$ isocline, Figure 5. A decrease in the value of v causes the isocline

to rotate upwards. This indicates that, for lower values of v, feeding should begin to increase at a higher level of prevalence for a given population density (again, because disease-related costs of feeding, in terms of increasing population growth and hence the number of infected offspring, are reduced), and this effect is greater for larger values of N. The effect is to increase investment in deer productivity over a larger range of θ.

The net effect of a reduction in v is an increase in both the population and prevalence of disease at the unstable equilibrium. In turn, this should shift the equilibrium cycle upwards, reducing the likelihood that eradication will be optimal (because the costs of managing the disease have been reduced).

Now consider the implications of reducing y. So far, we have assumed $y = 1$: that infected animals yield no benefits to hunters. This would be reasonable if hunting was mainly for meat and if the disease made the meat unsafe to handle or consume. However, in the case of bTB in Michigan deer, hunters also gain utility from the recreational value of hunting. Therefore, we conducted sensitivity on the parameter y by first decreasing its value to 0.5 and then reducing it to 0.05, such that infected deer retain more of their recreational hunting value. In the interest of space we do not present the phase planes for these analyses. Suffice to say, in each case the entire phase plane (and optimal path) shifts slightly up and to the right, in accordance with the fact that there are fewer damages to hunters in these scenarios and hence fewer incentives for the planner to reduce disease prevalence. However, the effect on the optimal path is small because the proportion of infected deer is small relative to the total harvest in this region.

7. Discussion and conclusion. It may be expected that concern over wildlife disease will continue to grow as human encroachment into wild lands intensifies, stressing ecological systems and making them more susceptible to both infection and the severe adverse consequences of infection, i.e. extinction in the case of threatened or endangered species, (Daszak et al. [2001]). Such changes may also lead to more opportunities for close contact between wildlife and humans and domesticated animals. Yet, there is surprisingly little research on the management of wildlife diseases, particularly how changes to the environment influence opportunities for disease management.

The purpose of this paper is to show the need to move from a solely ecological understanding of wildlife disease to an interdisciplinary understanding of wildlife disease management, one that incorporates human behavior. There are three main results that come from this work. First, we showed that the ecological threshold for an optimally managed disease system is endogenously determined when habitat management is accounted for. In this model, the host-density threshold is a function of prevalence and feeding, but feeding is optimally a function of the current level of population and prevalence. A variety of suboptimal choices for feeding exist, all of which lead to other thresholds, but such thresholds would be suboptimal and waste resources that could be used elsewhere more efficiently.

This leads to the second result: eradication may not be optimal. Horan and Wolf [2005] have previously reported this result, but for the case of frequency-dependent transmission in which there is no host-density threshold. When this threshold does exist, then economic and ecological trade-offs must be accounted for, and the active eradication of disease carries with it the direct costs of management as well as foregone opportunities, e.g., foregone hunting benefits when wildlife populations are at low levels and growing slowly, that need to be accounted for when planning a disease management program. A narrow focus on eradication based solely on exogenous ecological thresholds will be inefficient and possibly ineffective. When human-environmental actions affect disease transmission and these are not accounted for, any target host-density threshold is likely to be wrong and, moreover, endogenous human responses may alter transmission dynamics and result in an unanticipated change in the host-density threshold.

Finally, as in all modeling efforts, assumptions may mislead the manager when the model is extended beyond its intended purpose. The sensitivity analysis shows that assumptions about pseudo-vertical transmission, v, can be important for management, especially when these assumptions are made in an *ad hoc* fashion. Blanchong [2003] shows that transmission does not happen solely due to random mixing, and the relationship between individuals matters. Altering v may account for this, but the value v takes may impact the model in a qualitative way and alters the trade-offs that a planner faces, even though disease prediction models may be less sensitive to assumptions about v. Lower levels of pseudo-vertical make maintaining an endemic level of disease

less costly, and this reduces the likelihood that eradication would be an optimal solution. Furthermore, the specific role of inter-generational transmission has been downplayed in the literature but is likely to be important given length of time (and, hence, multiple wildlife generations) needed to manage wildlife diseases. In our numerical example optimal management results in long cycles lasting > 50 years. Disease control programs that have been considered "successful" have also required long-term commitments (Caley et al. [1999]).

Acknowledgments. We gratefully acknowledge funding provided by the Economic Research Service-USDA cooperative agreement number 43-3AEM-3-80105 through ERS' Program of Research on the Economics of Invasive Species Management (PREISM). The views expressed here are the authors and should not be attributed to ERS or USDA. Thanks to two anonymous reviewers for helpful comments and suggestions. Any remaining errors are our own.

Appendix

8. Deriving feedback rules for the singular solutions.

8.1. *The double-singular solution.* A double-singular solution arises when conditions (11) and (12) simultaneously vanish. These conditions may therefore be used to solve for λ and μ and can then be substituted into the "golden rule" conditions (15) and (16). Moreover, conditions (11) and (12) may be differentiated with respect to time to solve for $\dot{\lambda}$ and $\dot{\mu}$, which may also be substituted into the "golden rule" conditions. After making these substitutions, the golden rule conditions depend only on state and control variables. These conditions can be solved simultaneously for the control variables as functions of the current states, resulting in nonlinear feedback rules for the controls, $h(N, \theta)$ and $f(N, \theta)$ (while explicit rules can be derived, they are too complex to present here; see Bryson and Ho [1975] for more on nonlinear feedback rules in the context of singular solutions). The feedback rules $h(N, \theta)$ and $f(N, \theta)$ can be substituted into equations (7) and (8) to (numerically) solve for the double-singular path, given the initial states, N_0 and θ_0, and assuming that the feedback rules satisfy feasibility conditions at these initial states.[11]

8.2. *Partial-singular solutions.* It is possible that the double-singular feedback rules will yield values of $f > f^{\max}$, $f < 0$, $h > h^{\max}$, or $h < 0$ for some states of the world, as these bounds were not explicit in the solution algorithm for the double-singular solution. When such situations arise, the solution becomes blocked and it is necessary to determine the partial-singular solution to the problem. In principle, the solution can be blocked with respect to f or h, but in our numerical example only f becomes blocked. Hence, we focus on this case.

When f is blocked, condition (12) will no longer vanish and so f must be set to either its minimum or maximum value. Moreover, this means that (12) cannot be used to solve for μ or $\dot{\mu}$. The solution procedure in this case proceeds as follows. First, set f equal to its constrained value (either 0 or $f^{\max}$). Next, use condition (11) to solve for λ and $\dot{\lambda}$ and substitute these expressions into (15), as in the procedure used to find the double-singular solution. The resulting golden rule can be written in implicit form as $\rho = \Gamma(N, \theta, \mu)$. Hence, we can solve for $\mu(N, \theta)$. Next, take the time derivative of $\mu(N, \theta)$ and substitute $\mu(N, \theta)$ and $\dot{\mu}(N, \theta)$ into condition (16). The resulting "golden rule" can be written in implicit form as $\rho = \lambda(N, \theta, h)$. This enables us to solve for $h(N, \theta)$, which is the feedback rule for the partial-singular solution.

ENDNOTES

1. Other population and disease control methods, such as contraception and vaccination, have also been investigated, but most studies favor harvests (Smith and Cheeseman [2002]). In particular, vaccination is not even feasible for some diseases like bovine tuberculosis (bTB), *Mycobacterium bovis*, the focus of this paper. Moreover, treatment involves a strict and lengthy regimen of antibiotics—something that would be impossible to administer effectively in the wild.

2. This upper bound is made explicit in our simulation.

3. Density-dependent disease transmission is often applied in theoretical models, but may not hold up when tested empirically (McCallum et al. [2001]). Frequency-dependent models are often employed to model sexually transmitted diseases (McCallum et al. [2001]), and this form has also been shown to fit data better than the density-dependent form in some other cases (McCallum et al. [2001], Begon et al. [1998] and Begon et al. [1999]). Begon et al. [2002] demonstrate how frequency and density dependent transmission are special cases of a more general transmission function. Unlike the density-dependent transmission function, the transmission rate is independent of host density under frequency-dependent transmission. This creates a theoretical problem for frequency-dependent transmission, namely that transmission is positive even when density is zero (Roberts [1996]).

4. From (8), the fertility effect is clearly larger when θ is larger, and $\partial \widehat{N}/\partial f > 0$ will result as $\theta \to 1$. However, the value of θ at which the slope changes signs is relatively large. So while, in principle, $d\widehat{N}/df$ could take on any sign, our solution will involve $d\widehat{N}/df < 0$ given the values of θ that arise along an optimal path in our model.

5. The cost parameter c accounts for the opportunity cost of effort involved in hunting, e.g., travel and search costs, hunting equipment, etc. It does not account for permit fees (which we should point out are generally small relative to other variable harvesting costs). Permits are generally charged by the Department of Natural Resources on a per deer basis, but this is part of a regulatory framework that is not being imposed in the planner's problem. Indeed, the purpose of the planner's problem is to define the socially efficient management decisions independent of any regulatory framework. Moreover, permit fees are simply a transfer payment from the hunter to the government which would cancel out of any measure of social net benefits. The only other fees that hunters might incur in this area of Michigan are access fees to hunt on some private lands (as such fees are not charged for access to public lands in the area). These are not generally charged on a per deer basis, and so they would not affect marginal hunting incentives.

6. Several caveats must be made about the damage function. First, the livestock sector response to changes in deer prevalence is taken as given. Wildlife and agriculture in Michigan (and many other states) are managed by separate agencies. So the planner in our model is the wildlife agency, e.g., the Department of Natural Resources, and the solution can be thought of as being optimal conditional on the livestock sector responses. Second, the imposition of trade restrictions and federally-mandated testing requirements in response to the disease may also result in a significant lump sum damage component. However, these lump sum costs are not contingent on the elimination of the disease in the wildlife herd. Rather, the lump sum costs are contingent on the elimination of the disease in the livestock herd (and this took about 62 years the last time livestock were infected with bTB in Michigan). So again these costs would not be directly relevant to the wildlife agency. Moreover, such lump sum damages are primarily policy-induced and, if large enough, could affect the optimal plan. We restrict our investigation to an optimal plan without these lump sum costs, as the solution is efficient from Michigan's point of view in the absence of exogenous regulatory impositions. Horan and Wolf [2005] discuss the variable and fixed components of the damage function, and they investigate the lump sum cost issue for the case of frequency-dependent transmission. Third, deer are also important causes of automobile accidents and damage to agricultural crops (Rondeau [2001] and Rondeau and Conrad [2003]). We ignore these other damages in order to focus on the impacts of disease, but we note that these other damages may be important for optimally managing a herd.

7. Alternatively, prior to the transformation of variables, we had two populations of deer (infected and susceptible) that interact via disease transmission and competition.

8. Chattering is rapid switching between two optimal control solutions or isosectors. Clark [1990] first discussed chattering in the context of multi-cohort fisheries management models where it was not possible to target individual cohorts. Clark [1990] explains that chattering emerges because there is no optimal control that leads to the optimal steady-state. In the model presented here an optimal control exists and chattering emerges because the $f^{\max}$ constraint can be considered "soft" and there are two optimal controls, one on either side of the $f^{\max}$ frontier.

Zelikin and Borisov [1994] recommend referring to problems like Clark's [1990] as "sliding control" problems, and reserving chattering for problems like ours, where a unique control does exist, but involves an infinite number of switches over a finite time interval. Our solution is likely related to Swallow's [1990] solution for a problem dependent on the current state of two-state variables. This problem also appears to have the potential for chattering for a subset of optimal paths (the optimal path is determined by starting values in this model). However, the solution to the model presented in this paper appears to be the first case of a chattering control between a double and partial-singular solution, along a frontier defining a blocked interval in the field of natural resource economics. It has been argued that the Clark [1990] example emerges due to instantaneous adjustment that may be infeasible and chattering may never be optimal for resource economics problems (Liski et al. [2001]). Zelikin and Borisov [1994] argue that chattering is likely a common occurrence for resource allocation problems. The existence of chattering solutions to natural resource problems merits further investigation.

9. Another parameter of interest is ε, the shifting parameter that defines the degree of density-frequency dependence. This parameter is often considered at the extreme values of zero and one, but values within this interval are more likely to be realistic and create additional management opportunities. Horan and Wolf [2005] examine the case where $\varepsilon = 0$. In the case presented here, disease is maintained at a lower level and the deer population is maintained at a higher level. Furthermore, there is no need for a periodic cull after the initial reduction in population.

10. Horan and Wolf [2005] examine adding fixed costs that vanish if the disease is eradicated. For the Michigan case they find that a $4 million lump cost would cause the interior cycle they find to be suboptimal and eradication is the optimal strategy. Given the lower costs of eradication in the model presented here, fixed costs likely increase the optimality of eradication. Furthermore, Horan and Wolf also investigate the effect of the discount rate. Since a small discount rate means a more balanced weighting of near and far term benefits, feeding is decreased and smaller population with a lower disease prevalence is maintained. Increased near-term productivity is traded off to lower long-term damages. Horan and Wolf [2005] find similar results for larger marginal damages, feeding costs, or disease induced mortality rate.

11. That (11) and (12) both vanish when the feedback rules are followed, for any state variable combination such that the nonnegativity constraints are satisfied, is verified by setting equations (11) and (12) equal to zero and noticing that the coefficient matrix for the vector $[\lambda\mu]$ for this system is not singular; thus, a unique value of both λ and μ satisfy the singular conditions for all relevant combinations of N and θ.

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