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THE ECONOMICS OF MANAGING WILDLIFE DISEASE*

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THE ECONOMICS OF MANAGING WILDLIFE DISEASE

Abstract

The spread of infectious disease among and between wild and domesticated animals has become a major problem worldwide. Upon analyzing the dynamics of wildlife growth and infection when the diseased animals cannot be identified separately from healthy wildlife prior to the kill, we find that harvest-based strategies alone have no impact on disease transmission. Other controls that directly influence disease transmission and/or mortality are required. Next, we analyze the socially optimal management of infectious wildlife. The model is applied to the problem of bovine tuberculosis among Michigan white-tailed deer, with non-selective harvests and supplemental feeding being the control variables. Using a two-state linear control model, we find a two-dimensional singular path is optimal (as opposed to a more conventional bang-bang solution) as part of a cycle that results in the disease remaining endemic in the wildlife. This result follows from non-selective harvesting and intermittent wildlife productivity gains from supplemental feeding.
Introduction

The spread of infectious disease among and between wild and domesticated animals has become a major problem worldwide. The examples are almost too numerous to count, but a number involving wildlife as the primary vector of disease transmission are provided in Table 1. These examples highlight the diversity of disease concerns (most of which are distributed globally), particularly the risks of disease transmission to livestock and humans (see any recent issue of the Journal of Wildlife Disease to get a feel for the extent of potential problems). Biodiversity is also cited as a potential concern, particularly when threatened or endangered species are at risk (Simonetti 1995). Ultimately, these concerns all have economic dimensions.

The economic costs of wildlife-related disease can be substantial. For instance, a recent outbreak of foot and mouth disease in Europe led to the cull of millions of domestic and wild animals, the imposition of trade restrictions, and significant investments in biosecurity measures – both in Europe and abroad. The U.S. is currently considered free of foot and mouth disease, but the USDA estimates that any outbreak could cost billions of dollars the first year alone (USDA-APHIS 2002). For this and other diseases, economic losses occur at the farm level when domesticated livestock, such as cows or swine become infected. This is because some animals die or become less productive due to the disease, because the demand for livestock products is diminished, and/or because of strict regulations imposed when a herd becomes infected (e.g., depopulating a farmer’s herd). These costs are not limited to a particular farm that is infected. Trade sanctions are often imposed on entire counties, states, or even countries where the disease is present. For instance, in 2000 Michigan lost its status of being accredited tuberculosis (TB)-free following outbreaks in cattle herds in the Northeast part of the lower peninsula. As a result,
all cattle producers have faced stringent regulations and relating to the transfer or trade of livestock and associated products (MDA 2002; USDA-APHIS 1999).

Of course human health is an important concern as Table 1 indicates that humans are susceptible to a variety of wildlife-transmitted diseases. Chronic wasting disease (CWD) is the cause of recent concerns in North America. CWD is endemic in deer and elk populations in Wyoming and Colorado, with recent occurrences found in Wisconsin deer (Bishop 2002; Williams et al 2002; Wolfe et al. 2002). This has caused significant concern because CWD is a variant of Creutzfeldt-Jakob (C-J) disease of humans. There is currently no evidence that CWD can be transmitted to humans in the form of C-J disease. However, concern has developed because exposure to the related bovine spongiform encephalopathy (BSE, or ‘mad cow disease’) among the British and other Europeans has resulted in approximately 117 cases of the variant C-J disease as of April 2002 (Williams et al. 2002).

A disease outbreak may also impose costs on those who value wildlife products and/or services. For instance, the spread of an infectious disease within a deer population could lead to a loss of surplus to hunters who place a premium on healthy deer. Costs may also arise as infected or even healthy populations in close proximity to an outbreak are culled to reduce the threat of additional spread. The costs could be greater for wildlife species that are considered threatened or endangered. In particular, endangered species are often protected in parks that may not be large enough to support a viable population. As members of the population wander outside protected areas, the risk of infection increases – both for wandering individuals and for those in protected areas. Conservation measures must therefore be taken with disease control in mind (Simonetti 1995).
There has been relatively little research in the area of the economics of disease control among wildlife populations. Most work has estimated the costs to farmers and consumers under alternative control strategies, with little regard given to the wildlife dimension (e.g., Mahul and Gohin 1999; Kuchler and Hamm 2000; McInerney 1996; Ebel et al., 1992; Dietrich et al. 1987; Liu 1979). An exception is Bicknell et al. (1999), who developed a model to analyze the private incentives that a New Zealand farmer would have to undertake disease control measures for the case of bovine TB, which is spread by Australian brushtailed possums to dairy herds.

Following prior work on the spread of the disease among possums (Barlow 1991a,b, 1993), Bicknell et al. (1999) develop a bioeconomic model involving healthy and infected possum populations and also dairy cow populations. They then explore optimal disease control strategies for a farmer, including testing at the farm level, and hunting possums off the farm.

In this paper we depart from the Bicknell et al. analysis in three important ways. First, we consider the socially optimal management of wildlife and the disease as opposed to controls taken by a single farmer. We take this approach because wildlife disease problems affect many people (e.g., many landowners and hunters), and so an individual farmer would tend to under-invest in disease control investments relative to those investments that would maximize economic surplus for society. This is because the control actions taken by an individual farmer provide a public good (i.e., disease control also benefits the farmer’s neighbors although these neighbors do not compensate the farmer for this benefit). A second departure is that we assume infected wildlife cannot be identified without error, rendering it impossible to selectively harvest from the infected stock (whereas Bicknell et al. assumed selective harvesting was possible). It is not always possible to identify infected animals until after they are killed and examined (Williams et al. 2002). Accordingly, any off-take of infected animals is likely to be accompanied
by healthy animals that may be economically valuable. This brings us to our final departure, which is that healthy wildlife are considered to be economically valuable. In contrast, the possums in Bicknell et al.’s analysis had no positive *in situ* or *ex situ* economic values – only the negative values associated with the disease. It is certainly true that many disease-carrying animals such as deer or lions are economically valued.

The model is applied to the case of bovine TB among white-tailed deer in Michigan. The issue of bovine TB is Michigan is an important concern for its potential impact on livestock populations and productivity since bovine TB is transmitted among and between white-tailed deer and dairy cows and captive cervids in Michigan. TB was responsible for more livestock deaths than all other diseases combined at the turn of last century (MDA 2002). Moreover, the presence of TB among Michigan farms may lead to stringent regulations and trade restrictions. The USDA awarded Michigan TB accredited-free status in 1979 (MDA 2002). This important accreditation prevents other states from imposing trade restrictions on Michigan livestock and livestock products. But in the early to mid-1990s signs of bovine TB started to re-emerge both in the wild deer population and also among some small farms.\(^1\) In fact, Michigan is the only known area in North America where bovine TB has become established in a wildlife population.

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\(^1\) Bovine TB was discovered in wild deer in 1994, and in a captive deer operation in 1997. The first tuberculous cow was detected in 1998 in a beef herd. Following the detection of several more herds the following year, Michigan lost its bovine TB accredited-free status in June 2000. The result of this loss in status was a required testing program for all Michigan cattle, goats, bison, and captive cervids. In addition, other states could place movement restrictions on income Michigan livestock at their discretion. While the state covered the direct testing costs (e.g., lab tests and veterinary visits), farmers incurred the incidental testing costs (e.g., labor and lost performance) as well as increased transportation and trade requirements. The total losses to livestock agriculture were estimated at around $12 million per year (Wolf and Ferris 2000). At the time of this writing (May 2003) a total of 31 infected herds (29 beef and 2 dairy) had been discovered.
A model of infectious disease: A single population on a fixed land area

Before considering management of the disease, it is instructive to understand disease and wildlife population dynamics in the absence of an optimal management regime.

Case I: no harvesting

Consider a wildlife population that grows unexploited on a fixed land area. The aggregate wildlife population, $N$, consists of two sub-populations: a healthy but susceptible stock, denoted by $s$, and an infected stock, denoted by $z$. In the absence of exploitation or disease, the susceptible stock grows according to the logistic growth function, $rs(1-N/k)$, where $r$ is the intrinsic growth rate and $k$ is the carrying capacity. Following Barlow (1991a), the density-dependent part of this equation, $(1-N/k)$, depends on the aggregate population because susceptible and infected wildlife compete for the same habitat. This growth is reduced as members of the susceptible stock become infected, which occurs when a susceptible animal comes into contact with an infected animal. The $z$ infected animals make on average $\beta z$ contacts in each time period, with $s/N$ contacts being with susceptible animals (assuming wildlife is uniformly distributed across the land area). The equation of motion for the susceptible stock is (Barlow 1991a; Heesterbeek and Roberts 1995)

$$\dot{s} = rs(1 - N/k) - \beta z s/N$$  

(1)

The infected stock also grows according to the logistic growth function (assuming infected mothers pass the disease to their young, either in utero or shortly after birth through contact; this would be common among mammalian, avian, and marsupial wildlife), although the disease increases mortality by a rate of $\alpha$. The only other difference with (1) is that the infected stock increases when susceptible animals become infected. The equation of motion for the infected stock is (Barlow 1991a; Heesterbeek and Roberts 1995)
Can an interior equilibrium involving both stocks arise? The answer is no, as there are no positive values of \(s\) and \(z\) that solve both (1) and (2) when these expressions are set equal to zero (except for the special case in which \(\beta = \alpha\), the likelihood of which is essentially zero). Three corner solutions do exist: (a) \(s=k, z=0\), (b) \(s=0, z=k(r-\alpha)/r\), and (c) \(s=z=0\). The local stability properties of each equilibrium are given by the eigenvalues associated with the linearized forms of (1) and (2) (see Conrad and Clark 1987). The eigenvalues for the three equilibria are given by
(a): \(R_1^a = \beta - \alpha\) and \(R_2^a = -r < 0\), (b) \(R_1^b = \alpha - \beta\) and \(R_2^b = \alpha - r\), and (c) \(R_1^c = r > 0\) and \(R_2^c = r - \alpha\).

First consider the case in which \(\beta > \alpha\) and \(r > \alpha\). In this case \(R_1^a, R_2^a > 0\) and \(R_1^b, R_2^b < 0\). Equilibrium (a) is a saddle, equilibrium (b) is a stable node, and equilibrium (c) is an unstable node. Equilibrium (b) is for all intents and purposes globally stable in this case. Even though (a) is a saddle, which is conditionally stable, there will only be a single path to this equilibrium and there is zero likelihood that the initial values of \(s\) and \(z\) will be on this path in this autonomous model. The disease will not be eradicated.

The second case to consider is when \(\beta > \alpha\) and \(r < \alpha\). All three equilibria are saddles in this case. None of the equilibria will be pursued directly, but eventually the system will settle at either equilibrium (a) or (c). This is because there are depletion forces working on both stocks. The infected stock depletes the susceptible stock due to the relatively large value of \(\beta\) while the infected stock is being depleted due to the relatively large value of \(\alpha\). If the susceptible stock is eradicated while an infected stock remains, then the model is one of a single state variable and the origin becomes a stable node: all the wildlife die. If the infected stock is eradicated first, then the susceptible wildlife are no longer susceptible and they will approach their carrying capacity.
Finally, consider the case where $\beta<\alpha$. Equilibrium (a) is a globally stable node in this case, and so the disease cannot persist naturally: any exogenous influx of the disease would be short-lived.

Case II: non-selective harvesting

Now consider what happens when harvesting activities are undertaken to control the disease. Vaccination is not considered because for many diseases, such as bovine TB in wild deer, there are currently no effective vaccines (MDA 2002). The disease could be expediently controlled or even eradicated (albeit at possibly high cost) if the manager could selectively harvest from the infected stock. But selective harvesting may not be an option for it is often difficult to identify which individuals are infected prior to the kill; outward signs of an illness may take a while to manifest (MDA 2002; Williams et al. 2002). Harvesting will therefore include both healthy and infected individuals, which could be costly for species that are highly valued for recreational purposes (such as deer) or that are endangered.  

Given non-selective harvesting, a manager can only choose the aggregate harvest, $h$, with the harvest from each stock depending on the proportion of animals in that stock relative to the aggregate population. That is, $h_s = hs / N$ and $h_z = hz / N$, where $h_i$ denotes the harvest from population $i$. Given this specification, the equations of motion for the two stocks are written as

$$\dot{s} = rs(1 - N / k) - \betazs / N - hs / N$$

(3)

$$\dot{z} = rz(1 - N / k) - \alpha z + \betazs / N - hz / N$$

(4)

2 Non-selectivity is not unique to the current situation. For instance, hunters/fishermen cannot selectively harvest from different cohorts within exploitable populations of many species (Reed 1980; Clark 1990), and by-catch of non-targeted species is often a problem in fisheries.
As above, this system does not allow for an interior equilibrium involving both stocks. While equilibrium (c) is unaffected, the equilibrium values \( s^* \) and \( z^* \) will change in equilibria (a) and (b) due to the inclusion of harvests. The eigenvalues for equilibrium (a) become: \( R_1^a = \beta - \alpha \) (as before) and \( R_2^a = -r + 2h/s^* \). Harvests increase the value of \( R_2^a \), making eradication of the susceptible stock more likely when \( \beta > \alpha \) and less likely when \( \beta < \alpha \).

The eigenvalues for equilibrium (b) become: \( R_1^b = \alpha - \beta \) (as before) and \( R_2^b = \alpha - r + 2h/s^* \). Harvests increase the value of \( R_2^b \), possibly changing its sign for sufficiently large values of \( h \). Clearly, a large harvest will eradicate both the susceptible and infected stocks. But what about more moderate values of \( h \)? Using (3) and (4) we obtain the following condition:

\[
\frac{\dot{s}}{s} - \frac{\dot{z}}{z} = \alpha - \beta
\]

Assuming that \( \beta > \alpha \) and given that \( s \) is diminishing in this case, condition (5) is consistent with two possibilities: (i) \( z \) increases over time while \( s \) decreases over time and eventually vanishes, and (ii) \( s \) and \( z \) both decrease over time, with \( z \) decreasing at a faster proportional rate. If \( s \) and \( z \) are both decreasing at each point in time, one stock eventually vanishes. In this case, condition (5) is only satisfied when \( s \to 0 \). Otherwise, if \( z \to 0 \) then \( s \to N<k \) and \( \dot{z}/z > 0 \) by L’Hopital’s rule. The reverse holds when \( \beta < \alpha \).

The effects of non-selective harvesting can be summarized as follows. If \( \beta < \alpha \), then the disease cannot persist even without harvesting, although harvests can expedite matters. In contrast, non-eradicative harvest policies have no positive effect on disease control when \( \beta > \alpha \). The only effective harvest-based option for disease eradication in this case is extermination of all wildlife. The only other potentially effective policies would be those that have the effect of
increasing $\alpha$ or decreasing $\beta$. For instance, some hunters bait wildlife with small amounts of food (baiting) and others actually provide large amounts of food to feed wildlife (feeding) throughout the year. Such practices might have the effect of both decreasing $\alpha$ (because infected animals are better nourished) and increasing $\beta$ (because individual animals come into close contact as they feed). So a prohibition on feeding and/or baiting could lead to disease eradication. Similarly, some wildlife such as deer gain access to livestock operations (e.g., at night) where they congregate and consume leftover food and water. This increases spread of the disease among wildlife, and also from wildlife to livestock and vice versa. Investments in on-farm biosecurity measures can therefore decrease $\beta$. Finally, infecting the population with a more lethal strain of the disease would have the effect of increasing $\alpha$, which could possibly make the disease die out naturally before all animals are infected. We now turn to the economic question of how much control is warranted.

3 We have only been considering disease spread and control within a single population inhabiting a fixed land area. But in reality wildlife may be spread across a large region hosting many sub-populations, with migrations being commonplace. Consider exogenous immigration from a disease-free area at a rate of $m$. The immigrants are not infected and so they become part of the susceptible population, modifying equation (3) in the following manner:

$$\dot{s} = rs(1 - N / k) - \beta s / N - hs / N + m$$

With this modification, the possibility for an interior equilibrium does arise as long as $\beta > \alpha$. This interior solution is stable but exists only if $m$ is sufficiently small. If $m$ is sufficiently large then a corner solution arises with $z^* = 0$. If $m/s > \beta - \alpha$, then we find immigration reverses the results derived in case II: the infected population goes extinct. This is because the migratory influx increases competition for food and habitat, crowding out the existing infected population. This is essentially a form of the competitive exclusion principle in ecology (McGehee and Armstrong 1977). An implication is that spatial management may be important because eradication may otherwise be the only economically rational option for an infected area (e.g., if measures are unavailable to sufficiently reduce $\beta$ or increase $\alpha$ or if such measures are prohibitively costly). If it is costly to control the disease when focusing only on the infected area, then a more economical plan might be to control population growth both within and outside the infected area and to possibly take additional steps to limit the spread of the disease within the infected area. On the other hand, if control within the infected area is not too costly, then it may not make sense to significantly alter management choices in nearby non-infected zones. In this paper we assume the efficiency loss from focusing control efforts entirely on the infected area is not too great. This is probably a reasonable assumption for the case of bovine tuberculosis in Michigan white-tailed deer.
Wildlife management and disease control for Michigan white-tailed deer

Bovine tuberculosis 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 or less-formally as the ‘core’ (see Figure 1). There is some limited infection beyond this area but the disease does not appear to be sustainable outside the core, leading many to speculate that the core exhibits unique features that have enabled the disease to become endemic (Hickling 2002). These features include human-environment interactions, with feeding programs being a particular concern.

Several hunt clubs in the core sponsor feeding programs that sometimes even dump tractor-trailer loads of food in the woods and fringe areas. These massive piles of food can be seen from the air along with the tracks of thousands of congregating deer. There are economic reasons for providing this food, including increasing the carrying capacity of deer in the core. But such practices could also lead to increased transmission of the disease as deer congregate, and the supplementary food could also reduce the mortality rate of the disease by supporting sick animals. Denote $f$ as food provided by feeding programs. Increased food availability reduces the density-dependent component of growth by a factor $(1-\tau f)$, increases the disease transmission coefficient by a factor $(1+\upsilon f)$, and decreases mortality due to the disease by a factor $(1-\delta f)$, where

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4 Conventional wisdom held that the disease was not self-sustaining in wildlife populations (Hicking 2002). In fact, prior to 1995, only eight cases of bovine TB had ever been reported in wild deer from North America (Schmitt et al. 1997).

5 The many hunt clubs in this area primarily exist to facilitate deer hunting. Originating in the late 1800’s and early 1900’s, these clubs purchased large amounts of land in the area for members from southern Michigan on which to hunt. This land was desirable for the clubs as it was easily accessible from highways and, as it consisted of generally poor soil for agronomic purposes, the land was inexpensive (Hickling 2002). 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). This low carrying capacity was not conducive to easy hunting so the hunt clubs began aggressive deer feeding programs to encourage deer herd growth. The feeding programs were quite successful in increasing deer density with the density estimated at around 25 deer km$^2$ by the mid-1990’s. As hunting is the highest valued use of land in the infected region, whole-sale changes to existing regulations and property-rights are not popular.
τ, υ, and δ are parameters. The disease could be endemic in the core if β(1+υf)>α(1-δf), and it would necessarily be endemic in this case if it were also true that r>α, as is widely believed. If β>α, then the disease will persist regardless of feeding or hunting choices (apart from wildlife eradication). In that case, migration or some other effort to reduce disease transmission will be required to eradicate the disease. But if β<α, then the disease would be eliminated by setting f<\(\frac{α-β}{βυ+αδ}\) for some time. A smaller f means the disease is eliminated sooner but at an interim cost of lost deer productivity. Of course, it is important to consider whether eradicating the disease is even an optimal policy.

Wildlife managers have two objectives when dealing with the disease: reduce the number of diseased animals and control the spread of the disease. To accomplish these goals, the choice variables under consideration are harvest levels and the amount of food provided by feeding programs (Hickling 2002).

The equations of motion for the infected and susceptible stocks, (3) and (4), must be modified to account for the impacts of feeding. The equation of motion for the susceptible stock becomes

\[
\dot{s} = rs[1 - (N/k)(1 - ψ')] - (1 + υf)βs / N - hs / N
\]

Equation (4) would be modified in a similar way to account for f. It will be more intuitive and mathematically more convenient, however, to work in terms of the variable N instead of s, and

---

6 The disease would not be sustainable outside the core if β_o<α_o, where β_o and α_o represent parameter values outside the core area. These parameters may differ from β and α due to human-environment interactions apart from feeding.

7 The state of Michigan announced a goal of eradicating the disease by 2010. To that end, the wild white-tailed deer population in the area was to be decreased through hunting programs that sold increased licenses. In addition, the practice of legally feeding deer in the infected area was ended and the practice of baiting was temporarily ended.
the variable $\theta = z/N$ instead of $z$ (where $\theta$ represents the infected proportion of the population).

The relations $s = N-z$ and $z=\theta N$ can be used to substitute for $s$ and $z$ in equations (4) and (6), and without loss we can focus on the following equations of motion instead of (4) and (6)

\begin{align}
\dot{N} &= rN[1-(N/k)(1-\theta')] - \alpha(1-\delta\theta)\theta N - h \\
\dot{\theta} &= \beta(1+\theta f) - \alpha(1-\delta\theta)(1-\theta)\theta 
\end{align}

Economic specification

Consider the economic side of the model. Hunters gain utility from the actual process of shooting wildlife and/or consuming meat and other wildlife products. The (constant) marginal utility from harvesting healthy wildlife is denoted $p$, which is not less than the (constant) marginal utility from harvesting infected wildlife, $p_z$, i.e., $p \geq p_z$. For simplicity and without loss, we set $p_z = 0$ so that harvests of infected animals yield no benefits. The benefits from hunting are therefore $phs/N = p(1-\theta)h$.

Assume harvests occur according to the Schaefer harvest function, and that the unit cost of effort, $c$, is constant. 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 economic damages caused by infected wildlife by $D(z)$ (with $D(0)=0, D', D'' > 0$).\footnote{The Schaefer specification is not necessary, but it does simplify the exposition and it is consistent with the numerical analysis to follow. \footnote{The imposition of trade restrictions in response to the disease may result in a significant lump sum damage component, which if large enough could affect the optimal plan. Deer are also important causes of automobile accidents and damage to agricultural crops (Rondeau 2001; 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 could be important.}}
Given the discount rate $\rho$, an economically optimal allocation of harvests and feeding solves

$$\text{Max } \int^\infty_0 \left[ p(1-\theta)h - (c/q)(h/N) - wf - D(\theta N) \right] e^{-\rho t} dt$$

subject to the equations of motion (7) and (8).\(^{10}\) The current value Hamiltonian is

$$H = p(1-\theta)h - (c/q)(h/N) - wf - D(\theta N)$$
$$+ \lambda[rN(1-(N/k)(1-\theta))] - \alpha(1-\theta)\theta N - h] + \phi[\beta(1+vf'] - \alpha[1-\delta f'])(1-\theta)\theta]$$

where $\lambda$ and $\phi$ are the co-state variables associated with $N$ and $\theta$, respectively.

The marginal impact of harvests on the Hamiltonian is given by

$$\partial H / \partial h = p(1-\theta) - c/(qN) - \lambda$$

If this expression is positive so that marginal rents exceed the marginal user cost, then harvests should be set at their maximum levels. If this expression is negative then no harvesting should occur. The singular solution is pursued when marginal rents and the marginal user cost are equated. This is the standard condition for linear control problems involving renewable resources (e.g., Clark 1990), except for two important differences. First, marginal rents are reduced by $p\theta$ because not all harvested animals are valued (as some are infected). Second, because harvests of $N$ are non-selective, the marginal user cost of $N$ can be positive or negative, i.e., the sign of $\lambda$ is ambiguous. It is easy to show that the following relation must hold:

$$\lambda = (\partial SNB / \partial s)(1-\theta) + (\partial SNB / \partial z)\theta,$$

where $\partial SNB / \partial s < 0$ and $\partial SNB / \partial s > 0$ or $< 0$ depending on whether increases in $s$ only end up fueling the growth of a larger infected population $z$. If

\(^{10}\) It is implicitly assumed that $h,f \geq 0$ and that $f \leq \min(1/\delta, 1/\tau)$. A value of $f > 1/\delta$ would result in a negative mortality rate due to the disease, which is not possible. A value of $f > 1/\tau$ would result in a negative density dependence factor, which also does not seem realistic. In our numerical example these assumptions are explicit.
\(\theta=1\), then all additions to \(N\) only add to the infected stock so that \(\lambda<0\), and vice versa when \(\theta=0\). There must exist a set of values for the state variables such that \(\lambda=0\), other sets such that \(\lambda>0\), and still others such that \(\lambda<0\). A potential non-convexity therefore emerges, with the possibility of multiple optimality candidates (Rondeau 2001; see also Tahvonen and Salo 1996; Huffaker and Wilen 1994; Maler et al. 2003). The potential for non-convexities does not arise when harvests can be made selectively.

Now consider the marginal impacts of feeding on the Hamiltonian

\[
\frac{\partial H}{\partial f} = -w + \lambda \left[ r \left( \frac{N^2}{k} \right) \tau + \alpha \delta \theta N \right] + \phi \left[ \beta \nu + \alpha \delta \right] (1 - \theta) \theta
\]

Feeding can be thought of as an investment in both the productivity of the resource and of the disease. As we show below, the solution has similarities but also important differences than when investments are made in harvesting capital (see Clark et al. 1979). The singular solution should be followed whenever the unit cost of feeding equals the \textit{in situ} net marginal value of feeding on the two state variables. The \textit{in situ} net marginal value is the difference between the marginal benefits of feeding on the overall stock (which includes increased productivity and decreased mortality, and which may be negative when \(\lambda<0\)) 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). If the marginal \textit{in situ} values exceed the unit cost, then feeding should proceed at some maximum rate. If the unit cost exceeds the \textit{in situ} value then feeding should optimally cease. It must be the case that \(\phi<0\), for disease is never beneficial. Equation (12) therefore implies that \(\lambda>0\) must hold along a singular path, and so non-convexities can only emerge along a non-singular feeding path.
The necessary arbitrage conditions for an optimal solution are given by

\[
\dot{\lambda} = \rho \lambda - \frac{\partial H}{\partial N} = \rho \lambda - ch/(qN^2) + D' \theta \\
- \lambda [r - 2r(N/k)(1 - \sigma') - \alpha (1 - \sigma') \theta]
\]

\[
\dot{\phi} = \rho \phi - \frac{\partial H}{\partial \theta} = \rho \phi + ph + D'N + \lambda \alpha (1 - \sigma') N \\
- \phi [\beta(1 + \psi') - \alpha (1 - \sigma') ](1 - 2 \theta)
\]

Conditions (13) and (14) reflect intertemporal changes in optimal marginal resource values.

Consider harvesting and feeding choices along a singular path, so that conditions (11) and (12) both vanish. We refer to such a path, in which the solution is singular for both controls, as a dual singular path (solutions that are singular for only one control variable might also be possible, and sometimes these are the only feasible singular possibilities, e.g., see Clark et al. 1979). Differentiating condition (11) with respect to time, substituting the right-hand-side (RHS) of condition (13) in for \(\lambda\), and using (11) to substitute for the co-state variable \(\lambda\), we have the expression

\[
\rho = r - \frac{2rN}{k} (1 - \sigma') - \alpha (1 - \sigma') \theta + \frac{(c/qN^2)[rN[1 - (N/k)(1 - \sigma')] - \alpha (1 - \sigma') \theta N]}{p(1 - \theta) - c/(qN)} - D' \theta \\
- \frac{p[\beta(1 + \psi') - \alpha (1 - \sigma') ](1 - \theta) \theta}{p(1 - \theta) - c/(qN)}
\]

Equation (15) is a variant of the conventional “golden rule” for renewable resource management: the rate of return for holding the healthy stock in situ equals the marginal productivity of the stock, plus net marginal stock effects (i.e., the marginal cost savings that accrue as harvests come from a larger stock minus the marginal damages, normalized by marginal user cost), minus the (normalized) value of foregone revenues as some of the remaining healthy in situ stock will become infected and result in a larger proportion of infected deer in future harvests.
Equation (15) must hold at all times along either the dual singular path or a singular path involving only the harvest. In conventional autonomous renewable resource models, the singular path is a single point, $N^*$, because the golden rule is only a function of the stock and can be solved for a unique value of $N$. In contrast, condition (15) is a function of one of the control variables, $f$. If we solve (15) for $f$ as a function of the current state variables, $N$ and $\theta$, the result is a nonlinear feedback law along a two-dimensional singular arc (Bryson and Ho 1975). As we describe below, the existence of this feedback rule means that the dual singular solution will be a path and not simply a steady state point.\textsuperscript{11}

Now differentiate condition (12) with respect to time and substitute the right-hand-side (RHS) of condition (14) in for $\dot{\phi}$. Using (11) and (12) to substitute for the co-state variables, we get a golden rule expression for managing the proportion of infected wildlife. The explicit form of this expression is too complex to present here, but in implicit form it is written

$$\rho = F(N, \theta, h, f)$$ (16)

Equation (16) depends on both control variables, $h$ and $f$ (to see that it depends on $h$, note that (14) depends on $h$ and there is no chance of this term vanishing in the golden rule expression). This equation must also hold along the dual singular path. If we plug the feedback law for $f$ into this expression, it is possible to construct a feedback law for $h$.

The feedback laws $h(N, \theta)$ and $f(N, \theta)$ can be plugged into the differential equations (7) and (8) to solve for the optimal path along the singular arc. Because the singular arc is two-dimensional, the entire $(N, \theta)$ plane – or at least a subset of it – satisfies the necessary conditions

\textsuperscript{11} For the class of autonomous problems, such feedback laws seem to be more common in non-economic applications such as aerospace engineering. For instance, Bryson and Ho (1975) provide a famous example of optimal thrust programs for rockets. In the resource economics literature, Swallow (1990) illustrates a case in which a steady state does not always arise at the singular solution.
for the dual singular solution.\textsuperscript{12} Constraints on the controls may provide some additional restrictions that limit the space over which a dual singular solution may arise (and we explore this below). But assuming $f(N_0, \theta_0)>0$, the dual singular path can generally be found numerically by using the nonlinear feedback laws along with the equations of motion (7) and (8) and the initial states $N_0$ and $\theta_0$.

\textit{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. The data used to parameterize the model are described in the Appendix. While we have made every effort to calibrate the model realistically, research on the Michigan bovine TB problem is still evolving at a fairly early stage so knowledge of many parameters is somewhat limited. The following analysis is therefore best viewed as a numerical example rather than a true reflection of reality.

The numerical solution is presented in Figure 2 for the case of $\rho=0.1$. Although not presented, an interior solution arises at the point $(N=5,561, \theta=0.013)$, just northeast of the point $d$. This interior equilibrium point is an unstable focus that is not to be pursued. Instead, we find an interior cycle involving the paths 3, 4, 5, and 6 is optimal. We explain this cycle below. Note that $N=0$ is not an optimal steady state because the marginal cost of exterminating the wildlife population is infinite while the marginal benefits of extermination approach zero. Equilibria involving $\theta=0$ are not optimal either because it takes too long for the disease to die out naturally.

\textsuperscript{12} That (11) and (12) both vanish when the feedback rules are followed, for any state variable combination such that the non-negativity constraints are satisfied, is verified by setting equations (11) and (12) equal to zero and noticing that the coefficient matrix for the vector $[\lambda, \phi]$ for this system is not singular – thus a unique value of both $\lambda$ and $\phi$ satisfy the singular conditions for all relevant combinations of $N$ and $\theta$. 
We now turn to the optimal solution. Given \( N_0 \) and \( \theta_0 \), represented by point \( a \) in Figure 2, the dual singular path \( I \) is followed. This path spirals away from the interior equilibrium, increasing \( \theta \) while \( N \) is first increased and later decreased. The result that feeding should be initially encouraged runs contrary to Michigan’s current policy approach of banning feeding. Feeding represents an investment in stock productivity, initially increasing the stock while enabling large harvests along both the initial phase (the singular path \( I \)) and a second phase (discussed below) of the depletion path. The disease prevalence rate goes up along the singular path, but the increased damages are offset by the rewards of larger near-term harvests.

Feeding and also prevalence rates continue to grow along the path \( I \). Eventually \( f(N, \theta) = f^{\text{max}} = 10,000 \), represented by the boundary \( f = f^{\text{max}} \) in Figure 2. This boundary creates a blocked interval that prevents the state variables from following the dual singular path (Arrow 1964; Clark 1990, p. 56). The feedback solution is myopic, but the farsighted planner knows the boundary is approaching. So the singular path is abandoned (at least for the feeding control variable) prior to reaching the \( f = f^{\text{max}} \) boundary, for instance at the point \( b \), and an extremal value of \( f \) is chosen (Arrow 1964). Clark (1990, p. 57) refers to this result as the “premature switching principle”.

At the instant at which the dual singular path \( I \) is abandoned, say time \( T \), it becomes optimal to pursue the (non-dual) singular solution for \( N \) conditional on the extremal value of \( f \) (note there are two possible extremal values for \( f \): \( f^{\text{max}} \) and 0). This singular path is characterized by equation (15), holding \( f \) fixed at its constrained value. Given that \( f \) is constrained exogenously, equation (15) can be solved for \( N(\theta, f) \), with \( \theta \) moving exogenously.

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13 The continuation of the singular path \( I \) is not illustrated, but immediately after the boundary it changes direction and moves northeast: the larger harvests required to offset increased prevalence begin to deplete the stock.
through time as a function of the constrained value of $f$. The result is a singular path for $N$, referred to as the constrained singular path, which is essentially a non-autonomous singular path given the exogenous movement of $\theta$ (see Clark 1990 or Conrad and Clark 1987 for examples of non-autonomous singular paths) and is optimally approached along a most rapid approach path (MRAP).

As indicated above, there are two possible extremal values for $f$. Consider the singular path $N(\theta, f)$ that arises when $f$ is set at the extremal value $f_{\text{max}}$, denoted $N(\theta, f_{\text{max}})$ and labeled as path $3'$ in Figure 2. The path $N(\theta, f_{\text{max}})$ lies to the left of the $f=0$ curve (the $f=0$ curve is defined below; it has no meaning with respect to the path $N(\theta, f_{\text{max}})$ other than that this path lies to the left of the $f=0$ curve) and begins at the point $c'$ for the value of $\theta$ at time $T$. An immediate cull of the deer herd is required at time $T$ to move as fast as possible to this path. However, once at point $c'$ it would not be optimal to feed at a rate of $f_{\text{max}}$ because the state variable combination would still lie below the $f = f_{\text{max}}$ boundary. Rather, it would be preferable to follow a dual singular solution in which $f$ is not constrained but rather follows the feedback function $f(N, \theta)$ given the starting point $c$. But such a dual singular solution is infeasible at this point because the feedback function $f(N, \theta)$ is negative in the region to the left of the $f=0$ curve. So immediately after the cull occurs at $T$, the extremal value $f=0$ is optimal. Accordingly, it is optimal to pursue the constrained singular path $N(\theta)$ as derived from the following modified form of equation (15) with $f=0$

$$\rho = r - \frac{2rN}{k} - \alpha \theta + \frac{(c/qN^2)(rN[1-N/k] - \alpha \theta N) - D' \theta}{p(1-\theta) - c/(qN)} - \frac{p(\beta - \alpha)(1-\theta)\theta}{p(1-\theta) - c/(qN)}$$

It turns out this singular path coincides with the $f=0$ curve in the present model. Since this new singular path is pursued along a MRAP instantaneously after the cull at point $b$ (or time $T$), the
optimal choices at point $b$ are $f = f^{\max}$ and an immediate cull (represented by 2) to point $c$. The singular path $N(\theta)$, represented by 3, should then be followed.

Disease prevalence diminishes while wildlife stocks increase along the singular path 3, until point $d$ is reached. At point $d$, $f=0$ is no longer a binding constraint but rather the solution to the feedback function $f(N,\theta)$ along a dual singular path that moves northeasterly away from the $f=0$ boundary. At this point, it becomes optimal to again take advantage of enhanced productivity via supplemental feeding. The planner therefore moves off of the constrained singular path 3 and pursues the dual singular path that emanates from this point, labeled 4.

Note that continuation along the constrained singular path 3 would have led to an outcome with a disease-free wildlife stock (after which time feeding could be reintroduced without creating any disease problems). But that outcome is not pursued because the opportunity cost of waiting for the disease to die out is too high relative to the gains that can be made from re-investing in deer productivity at $d$. The marginal productivity impact of supplemental feeding depends on the size of the deer population. If the deer stock is relatively small, such as at point $c$, then feeding is costly: it results in only a small productivity boost while simultaneously causing increased disease prevalence. But when point $d$ is reached, feeding again becomes beneficial: small amounts of supplemental feeding can have a significant productivity boost while adding little to disease prevalence. This is reflected by the relatively flat slope of path 4 in the vicinity of $d$. Path 4 eventually turns around and heads back to the $f=0$ boundary. But knowing that this boundary is imminent, the planner culls the stock sometime prior to reaching

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14 Prior to point $d$, the $f=0$ boundary represents the solution to the feedback function $f(N,\theta)$ along a dual singular path that moves in a westerly direction away from the $f=0$ boundary, rendering such paths infeasible.
the boundary, jumping to a point such as e by way of path 5.\textsuperscript{15} Once at e, path 6 is pursued and the cycle 3-4-5-6 repeats. The disease is never eradicated because the deer are highly valuable and feeding intermittently becomes a good investment to boost productivity of the stock.

In many respects the optimal path is similar to that of Clark et al. (1979), who analyze irreversible investments in harvesting capacity for renewable resources. They find it is optimal to temporarily over-capitalize (relative to the steady state) prior to a stock-depletion phase. The reason is that the larger capital levels allow more harvesting early on, which generate greater near-term benefits prior to advancing to the steady state. Somewhat analogously in our model, we find that initial and intermittent future investments in resource productivity create opportunities for near-term gains. An important difference between our model and Clark et al.’s model is that a steady state is not optimal in our model. Unlike Clark et al., investment in our model (via feeding) produces adverse effects on resource dynamics: along with the productivity enhancing investments comes the unwanted side-effect of the disease, and sustained investment (feeding) would only lead to increasing disease prevalence. If allowed to continue unabated, this increasing prevalence eventually causes damages to swamp benefits. Therefore, intermittent dis-investment in the disease is warranted.

\textit{Extensions of the basic model}

We conclude our numerical example with a few small extensions designed to shed light on how certain key parameters influence the optimal solution. First, consider Figure 3, which illustrates the solution when $\rho=0.05$. There are two important differences between Figures 3 and 2. First,

\textsuperscript{15} The jump to e is drawn after path 4 crosses path 2 but we are not asserting this is necessarily optimal. To fully and accurately characterize the solution it is necessary to find the optimal time $T$ at which path 1 is abandoned and the optimal times at which path 4 is abandoned. This is beyond the scope of the current paper.
the dual singular path 1 is negatively sloped in Figure 3 but positively sloped in Figure 2. This difference arises because a smaller discount rate results in a more equal weighting of the near-term benefits that accrue from investing in *in situ* deer productivity (via feeding) and the costs that stem from the greater associated disease prevalence. Accordingly, feeding occurs at lower levels under smaller discount rates and hence provides a smaller productivity boost. A second difference is that the dual singular path 4 in Figure 3 has shifted down and exhibits a shorter upward path relative to its analogue in Figure 2. The shorter upward path results from the same sort of tradeoff influencing singular path 1. The downward shift results because the smaller discount rate reduces the opportunity cost of waiting for smaller disease prevalence levels relative to the benefits of feeding-induced productivity enhancements. In sum, the equilibrium cycle is shorter and occurs at lower disease prevalence rates for smaller discount rates.

Next consider the impacts of a larger disease mortality rate. When \( \alpha/\beta = 1.1 \), holding all other parameters from the basic model constant, we find the solution looks almost identical to Figure 3 but for different reasons. With larger disease mortality, infected deer die more rapidly. This means the productivity boost to the aggregate deer stock is not as great and the stock declines more rapidly for a given harvest level – hence the negatively sloped dual singular path 1 and the short upward portion of the dual singular path 4. The fact that infected deer die more rapidly also decreases the opportunity cost of waiting out reductions in disease prevalence. Hence the downward shift in singular path 4. Similar results occur for larger marginal damages, which reduces the opportunity cost of waiting for reduced prevalence, and for larger feeding costs, which reduces the marginal benefits of productivity investment via feeding and hence reduces the cost of waiting for reduced prevalence.
If marginal damages, feeding costs, or $\alpha/\beta$ is increased enough, we find that the opportunity cost of eradication of the disease becomes optimal in the long run. This process is presented in Figure 4. After an initial productivity investment there is a jump to singular path 3 along the $f=0$ curve. This singular path is optimally followed until $\theta=0$ and $N = N^*_{\theta=0,f=0}$, as there is no dual singular path that moves easterly out of the $f=0$ curve (coinciding with the fact that there is no interior focus point in this case). At this point, feeding again becomes optimal provided the costs are not too great (otherwise the system remains at $N^*_{\theta=0,f=0} = 5,921$ deer) because feeding will have no impact on $\theta$. It can easily be verified that the singular solution in this case involves equation (11) being satisfied as a strict equality and equation (12) as a strict inequality, so that feeding should be set at its maximum level. Equation (15) then uniquely determines the singular stock, $N^*_{\theta=0,f=f^{max}}$, which should be approached along a most rapid approach path, 4. This implies zero harvests until the stock has increased to the steady state value $N^*_{\theta=0,f=f^{max}}$, which equals 30,942 deer.

**Conclusion**

This paper represents a first step in understanding the economics of disease control in wildlife populations. We have formulated a general model of wildlife growth and disease transmission and found there are limitations to a harvesting strategy when harvests cannot be made selectively from the diseased population. Strategies to address disease prevalence must therefore focus on more than just the harvest, and can be particularly effective if they address disease transmission and mortality.
For our numerical example of bovine tuberculosis in Michigan deer populations, we find that eradication of either the disease is not likely to be optimal. It takes too long for the disease to dissipate naturally once supplemental feeding is halted, which is not surprising considering that it took sixty-two years to eliminate the disease in cattle herds under much more controlled conditions. It is also too difficult and costly to kill all the deer in the infected area, as managers in Michigan are currently discovering. Instead, it is optimal for the disease to remain endemic in the area at very low levels, with intermittent investments (via supplemental feeding) in in situ deer productivity. Of course an endemic disease is not always optimal. If marginal damages, feeding costs and/or disease mortality are large enough, we find that it may be optimal to delay feeding-induced productivity enhancements and in favor of disease eradication.

Although the model was applied to the specific case of bovine TB in deer herds, the model and results are likely to be applicable to other wildlife disease problems – even those problems where supplemental feeding is not an issue. Supplemental feeding decisions in our model represent the easiest method of controlling disease transmission for the Michigan case, and the control of disease transmissions would likely be a part of any wildlife disease management strategy. For other diseases, alternative environmental variables could be manipulated in ways that reduce disease transmission, and it is reasonable to believe that such actions might result in tradeoffs in in situ productivity (e.g., if contact is somehow reduced then fertility might also be expected to decline). Hence the current model provides a foundation for analyzing a range of wildlife disease problems.

Finally, an important caveat to our results is that the disease was assumed to be unsustainable beyond the core area. This is reasonable for the Michigan bovine TB problem, but it may not be the case for some other diseases. Rather, it might be possible for some other
diseases to spread among additional populations. Such a situation might imply greater marginal
damages due to the disease and hence more incentives to contain the disease. Additional
tradeoffs may also arise involving the management of spatially differentiated populations that
possibly interact through migratory processes. A spatially explicit analysis would be required in
such instances to fully assess the implications of spatial disease transmission.

Appendix

The model is calibrated using parameters obtained from a variety of sources. The initial number
of deer in the core area (deer management unit [DMU] 452), $N_0$, was estimated to be 13,298 in
Spring 2002 (after the previous winter mortality and prior to births) (Hill 2002). Disease
prevalence in the core since 1995 has fluctuated between 2.2 and 4.8 percent, averaging 2.3
percent during the period 1998–2000 (O’Brien et al. 2002). More recent estimates are
unavailable, but it is believed that prevalence rates have been fairly constant over the past few
years (Hickling 2002; O’Brien et al. 2002). We therefore adopt a value of $\theta_0=0.023$.

Core carrying capacity is taken to be 9 deer/km$^2$ (Miller et al. 2003; O’Brien et al. 2002),
which is the upper bound of estimates from the 1960s prior to extensive hunt club feeding
activities. This implies a value of $k=14,049$ for the 1561 km$^2$ core area. Extensive feeding has
increased the carrying capacity, with recent populations peaking around 19-23 deer/km$^2$
(O’Brien et al. 2002; Hickling 2002). Of course these populations have been subject to
significant exploitation and so we assume a slightly higher effective carrying capacity value of
27 deer/km$^2$, which translates to $k/(1-\tau_f)= 42,147$. Miller et al. (2003) report approximately 8212
kg/km$^2$ of fruits, vegetables, and grains being fed to deer in the core area. Although this is
probably an underestimate of actual feeding activities, we adopt this value for a lack of better estimates. Setting $f=8212$ we can solve for $\tau=0.00008$.

To calibrate the transmission of the disease, we use Miller and Corso’s (1999) reported rates of infected contact by sex, along with survival rates from the time of contact to that of infection. Using these values along with deer sex ratios reported by McCarty and Miller (1998), we derive $\beta(1+\upsilon_f)=0.346$. Miller et al.’s (2003) results are used to calibrate $\upsilon$. Assuming the number and size of feed sites are constants and that feed is applied fairly uniformly across sites, then an increase in total feed is an increase of food density at each site. Using a weighted average of feed-types and densities across sites, we find a base density of 0.118 kg/m$^2$. Miller et al. (2003) calculate an odds ratio of 2.8 for a 10kg increase in feed (fruit, vegetables and grain) per m$^2$, where this 10kg increase translates to a value of $f=704,144$. Although we have a deterministic model, the ‘likelihood’ of an infection can be thought of as the total new infections divided by the total susceptible. The odds ratio can be written in reduced form as $(1+\upsilon_{704144})/(1+\upsilon_{8212})=2.8$, which implies $\upsilon=2.64\times10^{-6}$. Given this value and $f=8212$, we can solve for $\beta=0.339$. Note that the Miller et al. value of $f=704,144$ is not assumed to be a reasonable value for management purposes. Rather, this is simply a scaled value that they used in order to present an odds ratio of reasonable scale. In our analysis, we set the maximum value of $f$ equal to $f^{\text{max}}=10,000$. This choice is somewhat arbitrary but it has little bearing on our qualitative graphical results.

The intrinsic growth rate for white-tailed deer is taken to be $r=0.5703$ (Rondeau and Conrad 2003). This value is the natural birth rate less the natural mortality rate. But we also require the additional mortality rate due to the disease ($\alpha$). Hill (2002) estimates that 1340 deer out of an initial population of 20,418, or 6.56 percent, died from reasons other than legal hunting.
mortality in 2001. We need an estimate of natural mortality outside the core area to enable us to separate out the effects of natural and disease-based mortality. Hill’s outside core estimates vary considerably depending on the amount of snowfall received by various areas. Medium snowfall areas outside the core imply a natural mortality rate of only 5.6 percent. If we take this value to be the natural mortality rate for healthy deer inside the core, then we would expect 1117 natural deaths among 19,948 healthy deer, leaving 223 deaths for the remaining 470 infected deer. Some of these deaths were likely due to illegal hunting and other reasons unrelated to the disease. We therefore adopt an effective mortality of \( \alpha(1-\delta_f) = 0.2 \), which would account for slightly less than half of these other deaths. This rate does not imply that 20 percent of all infected deer die as a direct result of the disease, as few deer actually die from tuberculosis. Rather, the deer are weakened by their infection and ultimately die from something else. One more piece of information is still required to calibrate \( \alpha \) and \( \delta \). The unsustainable nature of the disease outside the core suggests that \( \alpha > \beta \). We have chosen \( \alpha/\beta = 1.05 \) because this value produces reasonable results relative to historical changes in disease prevalence when recent deer populations, disease prevalence rates, harvests and feeding choices are plugged into the model.

The price per harvested deer is \( p=\$1270.80 \), which is derived from various estimates of consumer’s surplus, hunting effort and expenditures provided by Boyle et al (1998), Frawley (1999), and U.S. DOI-FWS (1996). Scaled harvesting costs, \( c/q \), are taken from Rondeau and Conrad (2003) to be \$231,192. The price of feed is set at \( w=36.53 \), which is imputed from Miller et al.’s (2003) feed density rates for the core along with anecdotal evidence about feed expenditures. Finally, total damages are estimated to be \$12 million per year at current stocks and infection rates (Wolf and Ferris 2000). Using a linear damage function of the form \( \xi z \), this implies a value of \( \xi = 5491 \).
References


Hicking, G. J. Dynamics of bovine tuberculosis in wild white-tailed deer in Michigan, Michigan Department of Natural Resources Wildlife Division Report No. 3363. March 2002.


Table 1. Examples of disease transmission among and between wildlife and livestock

<table>
<thead>
<tr>
<th>Disease</th>
<th>Wildlife vector</th>
<th>Livestock at risk?</th>
<th>Humans at risk?</th>
<th>Disease</th>
<th>Wildlife vector</th>
<th>Livestock at risk?</th>
<th>Humans at risk?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Botulism</td>
<td>Birds, mammals</td>
<td>Yes</td>
<td>Yes</td>
<td>Q (Query)</td>
<td>Mammals, birds</td>
<td>Yes</td>
<td>Yes</td>
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<tr>
<td><em>Brucella abortus</em></td>
<td><em>Bison bison</em> and <em>Cervus elaphus</em></td>
<td>Yes</td>
<td>Yes</td>
<td>Rabies</td>
<td>Mammals (skunks, bats, raccoons, foxes are frequent hosts)</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Chronic wasting disease</td>
<td>Deer and elk</td>
<td>Yes</td>
<td>Uncertain</td>
<td>Raccoon roundworm</td>
<td>Raccoon</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Colorado tick fever</td>
<td>Rodents (esp. ground squirrels)</td>
<td>Yes</td>
<td>Yes</td>
<td>Relapsing fever</td>
<td>Rodents</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Cryptococcosis</td>
<td>Pigeons (feces)</td>
<td>Yes</td>
<td>Yes</td>
<td>Rickettsial pox</td>
<td>Mice</td>
<td>Yes</td>
<td>Yes</td>
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<tr>
<td>Encephalitis</td>
<td>Bats, birds, squirrels</td>
<td>Yes</td>
<td>Yes</td>
<td>Rocky Mountain spotted fever</td>
<td>Mammals</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Foot and mouth disease</td>
<td>Andean deer (<em>Hippocamelus bisulcus</em>), Deer</td>
<td>Yes</td>
<td>No</td>
<td>Salmonellosis</td>
<td>Rodents, birds</td>
<td>Yes</td>
<td>Yes</td>
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<tr>
<td>Histoplasmosis</td>
<td>Feces of especially birds and bats</td>
<td>Yes</td>
<td>Yes</td>
<td>Sarcosporidiosis</td>
<td>Cats and other Mammals</td>
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<tr>
<td>Leptospirosis</td>
<td>Rodents</td>
<td>Yes</td>
<td>Yes</td>
<td><em>Toxocara catti</em></td>
<td>Puda concolor</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Lyme disease</td>
<td>Mammals, birds, reptiles (especially white-footed mice)</td>
<td>Yes</td>
<td>Yes</td>
<td>Toxoplasmosis</td>
<td>Mammals, birds; main host cats</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Lymphocytic-Chloriomeningitis</td>
<td>Mice and rats</td>
<td>Yes</td>
<td>Yes</td>
<td>Tuberculosis</td>
<td>Brushtailed possum, Deer, coyotes, lions, other mammals</td>
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<td>Yes</td>
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<tr>
<td>New Castle's disease</td>
<td>Birds</td>
<td>Yes</td>
<td>No</td>
<td>Tularemia</td>
<td>Mammals (esp. rabbits and rodents)</td>
<td>Yes</td>
<td>Yes</td>
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<tr>
<td>Plague</td>
<td>Rodents</td>
<td>Yes</td>
<td>Yes</td>
<td>Typhus</td>
<td>Rodents (including rats, mice, squirrels)</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>

*a* Most often transmitted by contact from excretions left by infected animal.

*b* Caused by a tick after tick bites infected animal.

Figure 1. Locations of bovine TB-infected livestock and wild cervids (338 deer, 1 elk) identified from 1994 - 2000 within the counties of Michigan’s northeastern Lower Peninsula. Source: Hickling (2002)
Figure 2. Solution of the benchmark numerical example
Figure 3. Solution of the numerical example when $\rho=0.05$, *ceteris paribus*
Figure 4. Solution of the numerical example when feeding costs are increased tenfold