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BIOECONOMIC MODELS OF BOVINE TUBERCULOSIS IN MICHIGAN WHITE-TAILED DEER: AN ANALYSIS OF ECOLOGICAL THRESHOLDS AND ECONOMIC TRADEOFFS IN WILDLIFE DISEASE MANAGEMENT

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has been accepted towards fulfillment
of the requirements for the

Masters of Science degree in Agricultural Economics

Major Professor's Signature

8-9-2005

Date

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A THESIS

Submitted to
Michigan State University
in partial fulfillment of the requirements
for the degree of

MASTER OF SCIENCE

Department of Agricultural Economics

2005

ABSTRACT

BIOECONOMIC MODELS OF BOVINE TUBERCULOSIS IN MICHIGAN WHITE-TAILED DEER: AN ANALYSIS OF ECOLOGICAL THRESHOLDS AND ECONOMIC TRADEOFFS IN WILDLIFE DISEASE MANAGEMENT

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Wildlife diseases threaten human and domestic animal health, natural resource-based recreation, and conservation of biodiversity. Yet knowledge of wildlife disease management is limited and the few options available for disease control are nonselective with respect to infected animals. The ecological literature has focused on identifying a host population density threshold (exogenously determined by ecological parameters) below which a disease naturally dissipates, and suggests using population controls to achieve that density. But human actions that influence wildlife habitats can also affect disease spread. There are likely tradeoffs between the two types of controls.

Bioeconomic models are useful for assessing economic and ecological tradeoffs associated with different management choices. A bioeconomic model, using bovine tuberculosis (*Mycobaterium bovis*) in Michigan white-tailed deer (*Odocoileus virginianus*) as case study, is developed to examine the use of population density and environmental controls. The host-density threshold is shown to be endogenously determined by both ecological and economic forces. However, the disease is not optimally eradicated, due to costs associated with the nonselective nature of the controls. This model is then expanded to allow targeting by sex, with males assumed to be the "risker" subpopulation. This improved target leads to more control over the host-density threshold, resulting in lower control costs and the optimal eradication of the disease.

ACKNOWLEDGMENTS

A number of people helped me with my research and the writing of this thesis. I am extremely grateful for the continued help of my major professor, Dr. Rick Horan, his mentoring, hours of answer questions, and the funding he provided me with. Dr. Christopher Wolf and Dr. Graham Hickling, the rest of my committee, have also been extremely helpful, making themselves accessible, answering questions, and providing addition tools and resources for my research. I would also like thank Dr. Clifford Weil and Dr. Jack Cortney for there help programming and troubleshooting my Mathematica code. Comments provided by Dr. Jean Tsao and Dr. Jim Bence also added to this work.

More generally, the above listed people, and the faculty and graduate students in the agricultural economics and fisheries and wildlife departments made the time spent on this work much more enjoyable.

Also, I would not have succeeded without the support of my wife, Tonka. Her continued support, tolerance, and interest in what I have spent my time doing was a great help in completing this work.

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KEY TO ABBREVIATIONS AND SYMBOLS

ABBREVIATIONS

Abbreviation	Full text
bTB	bovine tuberculosis
SI	susceptible-infected
s.t.	subject to
MRAP	most rapid approach path
LHS	left-hand-side
RHS	right-hand-side

SYMBOLS

Symbol	Description
N	Used to denote a population or stock of wild animals
G	Is a growth function (usually the logistic growth function)
t	Represents time
r	Represents the intrinsic rate of growth
k	Represents carrying capacity
h	Is a harvest of wildlife (fish or deer)
p	Is the value of harvested unit of wildlife
C	Is a cost function (chapter 1) and Is a contact function (chapters 2-3)
ρ	Is the discount rate
H	Is a Hamiltonian
λ	Is a co-state variable
Φ	Is a function
*	Represents an equilibrium solution
S	Is a susceptible population
I	Is an infected population
R	Is a recovered population
Z	Is a growth function (usually the logistic growth function)
T	Is a transmission function
A	Is a function representing disease induced mortality
f	Represents alterations to wildlife habitat, specifically in the case studies supplemental feeding
D	Is a function that describes damage to the livestock sector by infected wildlife
SNB	Is a social net benefits function
τ	Parameter associated with feedings effect on carrying capacity
b	Represents the per-capita birth rate
v	Represents the (pseudo-)vertical

δ	Represents the per-capital natural mortality rate
β	Is the transmission coefficient
ε	Is a shifting parameter that describes transmission dynamics
ω	Is a parameter associated with the effect of feeding on disease transmission
α	Is a the disease induced mortality rate
χ	Is a parameter associated with the effect of feeding on disease induced mortality
θ	Is the prevalence of disease in a population
$\hat{N}$	Is the host-density threshold
c	Is the per unit cost of harvesting
q	Is the catachablity coefficient
w	Is the per unit cost of supplemental feeding
μ	Is a co-state variable
f^{max}	Is the maximum amount of feeding feasible (this is an exogenous upper bound on feeding)
Γ	A function representing a golden rule
Λ	A function representing a golden rule
M	Denotes the male subpopulation
F	Denotes the female subpopulation
i	Indexes M and F
ϕ	Is the proportion of offspring that are male
Ψ	A function
Φ	A function
j	Indexes M and F

CHAPTER 1

INTRODUCTION

1.1 The Wildlife-Human-Disease System

Concerns over wildlife disease traditionally have focused on risks to human and livestock health, with disease management often being conducted at the expense of involved wildlife populations (Daszak et al. 2000; Wobeser 2002; Leighton 2002). Indeed, the views that wildlife species are simply a reservoir or vector for livestock and human diseases, that people have little direct impact on these risks apart from wildlife population control or, in some cases, *in situ* vaccination or treatment, and that such risks should be completely eliminated is pervasive in the veterinary literature (Leighton 2002; Artois et al. 2001; Lanfranchi et al. 2003). These views have spread to the ecological and economic literatures on wildlife disease and, while Peterson (1991) questioned these views in the early 90's, others seldom have followed his lead.

The ecological literature on wildlife disease has typically focused on infectious disease dynamics *sans* humans, and has not been concerned with how humans affect the system (Wobeser 2002). The few analyses that have included human actions often focus on determining what is required for disease eradication (Barlow 1991b; Barlow 1996; Smith et al. 2001), regardless of whether such a strategy is economically justifiable. Analogously, economic studies traditionally dealing with problems of disease in livestock populations usually have disregarded the wildlife vector in cases where one exists – an important omission because the majority of emerging diseases involve wildlife (Cleaveland et al. 2001). Such economic studies traditionally have focused on

eradication strategies, typically estimating the private costs (including those to consumers) of alternative on-farm control strategies (e.g. Mahul and Gohin 1999; McInerney 1996; Ebel et al. 1991; Dietrich et al. 1987; Liu 1979). Other studies incorporate the wildlife component, but focus on measuring the costs of *in situ* disease eradication programs without accounting for economic efficiency (Barlow 1991b; Wolfe et al. 2004).

Measures to eradicate a disease in a wildlife population seem reasonable when control efforts (such as culling) are not more costly than the benefits they generate; however being 'reasonable' is not the same as being economically optimal. Such a relationship is only likely to hold under limited conditions, for instance if the host species is a pest, such as rats or possums, and generate only costs without offsetting benefits. Even so, the direct costs of host control may be large. Moreover, host populations that may be more easily controlled may be highly valued, and this increases the opportunity costs of a cull-based disease control strategy.

Infected host species may be highly valued for recreation/hunting opportunities and/or their contribution to biodiversity and ecosystem function. This is certainly the case for Michigan white-tailed deer, *Odocoileus virginianus,* (infected with bovine tuberculosis (bTB; *Mycobacterium bovis*), lions, *Panthera leo*, in Krueger Park (also infected with bTB), and bison, *Bison bison*, in Yellowstone Park (infected with brucellosis). Vaccines often are unavailable, not feasible, or their effectiveness is unknown and carries risks of it own (Nishi et al. 2002).

Moreover, infected wild animals are often indistinguishable from healthy animals (Lanfranchi et al. 2003). Therefore control strategies involve non-selectively reducing

the aggregate population below some threshold level so that the disease cannot persist (McCallum et al. 2000). Such strategies can impose high costs when valuable, healthy animals are culled and population depletion impedes conservation efforts or increases hunting costs. Such a trade-off may be especially costly if the disease carries a low risk of causing the host population to become extinct, to lose genetic diversity, or to suffer losses in productivity (Peterson 1991). These costs may not be outweighed by the benefits associated with reduced risks to human and livestock health.

Few economists would be surprised by such a result, as it is well known that reducing environmental risks to zero is often not economically optimal (Hanley et al. 1997). For instance, work on invasive species has shown that oftentimes containment as opposed to eradication may better allocate resources, especially when the invader is already well-established (Sharov and Liebhold 1998).

It may also be difficult and costly to eradicate a wildlife disease if unregulated human decisions contribute to disease spread. Risk, to some extent, is generally understood to be an endogenous function of human choices. This means that human activity influences the likelihood that wildlife, livestock, and humans become infected. One human activity that may affect disease spread is habitat alteration. Intentional human alterations to habitats have played a key role in causing changes in deer behavior leading to an outbreak of bTB in Michigan (Schmitt et al. 2002), and unintentional habitat changes have lead to herpesvirus outbreaks in pilchards (Daszak et al. 2001). Human-wildlife interactions may limit or contribute to disease persistence. Hunting is often used to control ungulate disease, while backyard feeders have helped sustain various garden bird diseases (Hartup et al. 2000). Humans can also influence risk to

themselves and domestic animals through behavior, biosecurity measures, and farm management practices.

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; Sanchirico and Wilen 2001). Recognizing these linkages and system feedbacks is important for developing wildlife disease control strategies. Indeed, many authors have called for interdisciplinary approaches to disease management (Wobeser 2002; Daszak et al. 2001; Artois et al. 2001). Yet the theme of jointly determined ecological-economic systems is only now beginning to emerge in the wildlife disease literature.

A limited number of studies have looked at more general integration of wildlife disease systems and economics. Bicknell et al. (1999) examined the incentives for farmers to engage in control of Australian brushtailed possums (*Trichosurus vulpecula*) infected with bTB. These possums transmit bTB to dairy herds. In their paper, a model is developed whereby an individual farmer attempts to maximize discounted net revenue by choosing a cattle-management strategy, a testing strategy, and a possum harvesting strategy. Bicknell et al.'s (1999) model accounts for the fact that the farmer has some control over the transmission of disease from wildlife to cattle by directly controlling wildlife. The authors follow disease ecology theory and employ a simple model from the class of susceptible-infected models (*SI* models, which will be explained in detail below). Bicknell et al. (1999) show that farmers' wildlife harvesting decisions depend on the state of the system and that it is unlikely for farmers, who attempt to maximize

their individual welfare, to employee adequate effort to eradicate the disease. They also find that it is unlikely that the disease will be eradicated by individual farmers' efforts given "sharply increasing marginal costs."

Bicknell et al. (1999) focus mostly on private incentives, but it is state or national agencies that typically address management of wildlife resources in order to address broader social goals. Such social goals may include reducing the chance of disease spread to other farms and wildlife populations as well as welfare impacts on other farms, hunters, and groups interested in biological resources and public health. Horan and Wolf (2005) develop a model to examine the maximization of social net economic welfare from the management of bTB in white-tailed deer. Specifically, they solve the social planner's problem, which may be thought of as a benevolent dictator attempting to maximize discounted net benefits to society. The solution provides a benchmark for evaluating the efficiency of alternative policies that may be implemented in a decentralized system.

In the case of managing bTB in free-ranging Michigan deer, managers have proposed two main policy options: i) limiting or banning supplemental feeding of deer, and ii) culling the herd. Both of these options are based in scientific theories of disease and deer ecology. It is often believed that the greater the density of a host population the greater the number of new infections per area per unit time (Diekmann and Heesterbeek 2000). The deer herds in the infected region seem to be stable and move little (Garner 2001), making it possible for a cull to reduce the density. Furthermore, supplemental feeding programs are believed to concentrate deer and increase the effective density, and the additional food also is believed to increase productivity increasing the actual density

of the herd (Schmitt et al. 2002). Therefore, while feeding is often thought to benefit the deer population and hunters, it may also provide costs in the form of increased decreased prevalence that may ultimately result in livestock damage and lost hunting opportunities. Managers may be able to regulate deer density by managing the number or proportion of the herd harvested and the amount of supplemental feed provided. Culls, in the form of recreational hunting or otherwise, may at first seem to provide benefits to hunters by allowing more animals to be taken. However, there are costs associated with such a strategy since a reduction in the deer population today will leave fewer deer to reproduce resulting in a smaller herd tomorrow. Moreover, finding and harvesting a deer is not costless and the costs may increase as the deer population declines.

The problem facing the social planner is to maximize the discounted social economic surplus by choosing the level of supplemental feeding and harvesting of deer. In the Horan and Wolf (2005) model, deer transmit bTB to cattle causing damages to the livestock industry, although the management of cattle is not considered explicitly. An important difference between the Horan and Wolf (2005) and Bicknell et al. (1999) papers is that deer are a highly valued species whereas possums are an exotic pest that provide no benefits aside from a low pelt value (Barlow 1991b). Another difference is that, while Horan and Wolf (2005) also use a simple model from the *SI* class of disease transmission models to capture ecological effects, simply reducing density will not result in disease eradication. Rather, it is only possible to eradicate the disease by eradicating the deer herd or keeping feeding at a low level for a prolonged period of time. Under such conditions, Horan and Wolf (2005) find that it may be suboptimal to eradicate the disease from a societal perspective because it is costly to eliminate the stock and

reducing feeding leads to a large reduction in deer productivity. This implies that society must forgo valuable deer hunting opportunities to eradicate the disease. Horan and Wolf (2005) find a cyclical management strategy to be optimal. This cycle includes periods of relatively low deer population and relatively low disease prevalence followed by periods of relatively high deer population and relatively high disease prevalence. The solution also involves the periodic culling of the herd.

The purpose of this thesis is to illustrate how economic analysis can be incorporated within an interdisciplinary framework to more fully address wildlife disease management questions. The Bicknell et al. (1999) and Horan and Wolf (2005) papers make use of very simple disease ecology models. These models provide introductory insight, but are naive in the sense that they largely ignore potential complexities of human actions on disease ecology. Hence these models may be thought of as a blunt approach to managing the wildlife resource and disease simultaneously. Therefore, disease management efforts are not very direct and can be costly in terms of the unintended impacts on healthy populations.

Opportunities to target factors affecting the risk of disease transmission (risk factors) may be discovered by considering human-wildlife interactions in a more comprehensive fashion. By making greater use of the scientific understanding of ecological processes, it may be possible to tailor management programs to better target disease transmission. If the costs of targeting are low, then disease eradication is more likely to be the optimal outcome.

The remainder of this chapter provides an introduction to the theories and concepts that will be used and expanded upon later in this thesis. Specifically,

bioeconomic modeling is introduced along with background information on wildlife disease ecology. This is then used to develop a general framework for modeling wildlife disease dynamics in the presence of management.

1.2 Bioeconomics

Aldo Leopold, the farther of wildlife science, wrote in the 1948 Foreward [*sic*] to Sand County Almanac, "We abuse land because we regard it as a commodity belonging to us. When we see land as a community to which we belong, we may begin to use it with love and respect." Leopold argued that there is a need for a shift from thinking of natural resources as simple commodities for humans to trade to understanding that humans are indeed members of the natural communities those resources comprise. This argument is as true today as it was during Leopold's time. Leopold's arguments are philosophical, however a growing number of ecologists and economists recognize that understanding the earth's systems in this fashion is vital to ecological sustainability and the maximization of long-run social welfare. Indeed, the joint determination of economic and ecological systems is a theme of increasing importance in the economics of the management of wildlife resources (Shogren 1998; Shogren and Crocker 1999; Shogren et al. 1999; Brock and Xepapadeas 2002; Sanchirico and Wilen 2001). Bioeconomic models can help us understand and analyze this joint determination.

A fundamental principle of ecology is that organisms react to stimuli from their environment. Further, many complex human decisions are based on reactions to stimuli in the form of economic signals (e.g., prices and costs). These signals often arise outside of traditional markets and are influenced by environmental conditions and ecological

relationships. Of course, human actions influence ecological systems, creating dynamic feedback responses. Managing natural resources often involves ecological and economic trade-offs. Integrating economic and ecological systems into dynamic, bioeconomic models enables these trade-offs to be assessed. While some may be uncomfortable with dollars as the units of measure, such valuation may be thought of as simply ranking alternative opportunities (Tschirhart 2000).

Bioeconomic models may be framed within the context of systems modeling. However, unlike standard economic or biological models, a bioeconomic model attempts to capture human and ecosystem dynamics as well as the interaction between them. Such models exist at various levels of integration. Some models include economic parameters (i.e., prices) in dynamic ecological simulation models (i.e., Liu et al. 1994). Others use static optimization models where ecological objectives are maximized subject to economic constraints (i.e., Ando et al. 1998). Yet a third type of bioeconomic model combines dynamic simulation with optimization. The advantage to this type of model is that the model accounts for the trade-offs between competing objectives endogenously. Of course, there are trade-offs between model types that involve the level of model complexity and tractability. Ultimately, a model type must be chosen based on the modeling objectives. In this thesis, I focus on the third type of bioeconomic model.

Dynamic optimization of economic objectives subject to the constraints imposed by ecological systems requires that a simulation model of the ecological system be constructed. The nature of this model will impact the results. To illustrate how such a model may be constructed I use the classic and simple bioeconomic-fisheries model (Conrad 1999). Define $N(t)$ to be a naturally reproducing population of fish and $G((N(t))$

to be a density-dependent growth function that describes the net growth of the fish stock given a stock N at instant t. [1] In the absence of harvests, the fish population changes over time according to

$$(1.1) \quad \frac{dN}{dt} = \dot{N} = G(N)$$

where the time index is dropped for simplicity. The logistic growth function, $rN(1-N/k)$, is commonly used for G to model population dynamics, though there are many other possible forms. It is worth taking a moment to discuss some of the characteristics of logistic growth (other candidate growth functions are often constructed in a similar fashion). A simple logistic growth function assumes a fixed maximum per capita growth rate, r, known as the intrinsic rate of growth. Specifically, this is defined as the per capita birth rate minus the per capita death rate. This is the maximum rate of growth a population may achieve if scarce resources do not limit growth. However, as density increases resources per individual may become increasingly scarce. The degree of scarcity of these resources is often modeled indirectly using a parameter k, for carrying capacity. Simply put, the carrying capacity is the largest number of individuals the ecosystem can support. Carrying capacity may be understood by thinking of a wildlife population as a good that is produced according to a production function where the factor inputs are things such as food and shelter, given a level of wildlife stock. Assume that the production function is Leontief so that trade-offs between inputs may not be made in the production of new individuals. The carrying capacity is often assumed to be the maximum number or animals that can be supported by the most restricted resource.

[1] Fish stocks are often thought of as the reproductive capacity of a population, for simplicity stock and population may be thought of as synonymous, though this need not be the case.

The limiting resource is commonly thought to be food, but this need not be the case. Figure 1.1 illustrates the population dynamics that result from the logistic growth function. In the absence of harvesting there are two equilibria: $N = 0$ and $N = k$. The $N = k$ node is globally stable, as indicated by the phase arrows, while the $N = 0$ node is unstable.

Now assume that fish harvests are valued for consumption or recreation. If harvests, h, are included in the model, then equation (1.1) may be modified to equation (1.2).

(1.2) $\dot{N} = G(N) - h$

When harvesting is included, an infinite number of potential equilibria emerge, depending on the value of h. The system will be in equilibrium when the stock does not change, $\dot{N} = 0$. Such a condition will be satisfied whenever $G(N) = h$. This is illustrated by figure 1.2 where the curve indicates the locus of points where net growth and harvests are equal. Stock-harvest combinations along the $\dot{N} = 0$ may be sustained. Harvests above the isocline reduce the stock, and harvests below the isocline allow the stock to increase, as is indicated by the phase arrows.

A management agency may be faced with the problem of determining the appropriate level of harvest. In the bioeconomic literature, the criterion for evaluating alternative harvest plans is the maximization of discounted economic surplus, sometimes called welfare. Assume that the planner may not affect a constant price, p, of a unit of harvested fish (i.e., the fishery in question is a small supplier of fish in the market). The costs of harvesting are given as $C(h,N)$, where C_h, $C_{hh} > 0$ and $C_N < 0$, $C_{NN} > 0$. In this

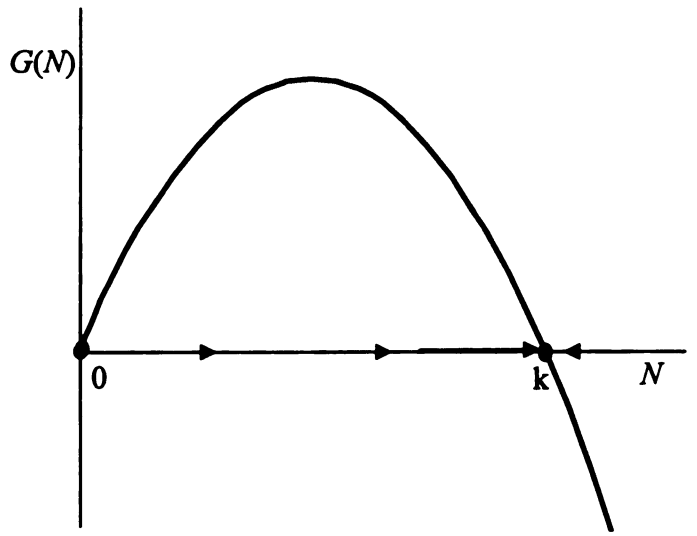

Figure 1.1. The logistic growth function in the absence of harvesting.

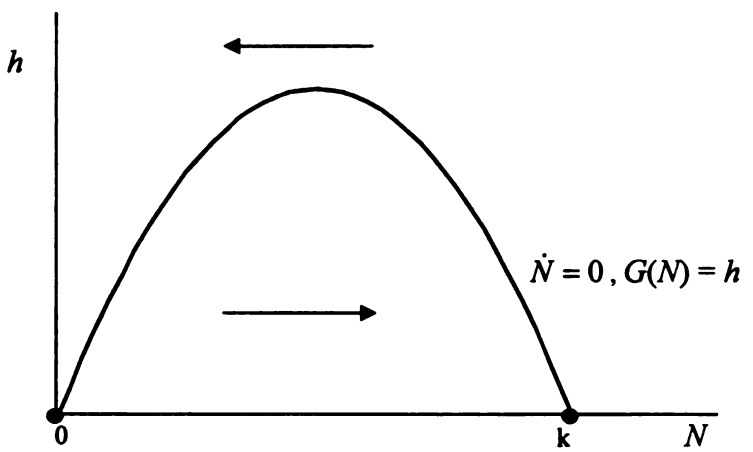

Figure 1.2. Phase-plane diagram illustrating the effects harvesting has on a population that grows according to the logistic growth equation.

case net economic surplus in time t is defined by firm quasi-rents, ph-$C(h,N)$. The manager will often be concerned with some planning horizon, T, which is taken here to be infinite since the fish population could be sustainably managed in perpetuity. People often show a preference for benefits today as opposed to the future (Conrad 1999) so a discount rate, ρ, is applied to future benefits. This results in the social planner's problem being defined as

$$(1.3) \quad \max_{h} \int_{t=0}^{\infty} [ph - C(h,N)]e^{-\rho t}dt$$

$$\text{s.t. } \dot{N} = G(N) - h, \qquad N(0) \text{ is given}$$

Notice that (1.3) defines a constrained optimization problem. The constraint is called the "equation of motion" because it describes the "motion" in time of the state variable. It is possible use Pontryagin's maximum principle to find the optimal harvest (Conrad and Clark 1987). The first step to apply the maximum principle is to define the Hamiltonian. The current value Hamiltonian for problem (1.3) is

$$(1.4) \quad H = ph - C(h,N) + \lambda(G(N) - h)$$

where λ is the co-state variable. The co-state variable represents the shadow value of the resource. This may be thought of as the value of having an additional unit of fish stock at the margin or the intertemporal opportunity cost of harvesting an additional fish today.

Assuming an interior solution, there are three necessary conditions for optimization of the Hamiltonian. These conditions, which define the optimal solution, are the equation of motion (equation 1.2) and

$$(1.5) \quad \frac{\partial H}{\partial h} = 0 = p - \frac{\partial C(h,N)}{\partial h} - \lambda$$

$$(1.6) \quad \dot{\lambda} = \rho\lambda - \frac{\partial H}{\partial N} = \rho\lambda + C_N(h,N) - \lambda G_N(N)$$

Condition (1.5) implies the following condition must hold at each point in time.

$$(1.7) \quad \lambda = p - \frac{\partial C(h,N)}{\partial h}$$

This condition states that in the optimum the marginal intertemporal cost of harvesting (λ) is equal to the current period marginal net benefits from harvesting.

Condition (1.6) is known as the adjoint equation. This condition ensures that the planner will be indifferent at the margin between reallocations of harvests across time; in essence it is an intertemporal arbitrage condition. Upon further manipulation it can be shown that condition (1.6) may be expressed as a condition defining the stock's optimal rate of return.

$$(1.8) \quad \rho = \frac{\dot{\lambda}}{\lambda} + G_N - \frac{C_N}{\lambda}$$

This is known as the "golden rule" of renewable resource management. The left-hand-side (LHS) is simply the discount rate representing the opportunity cost of holding the fish stock *in situ*, as the fish stock could otherwise be harvested and the proceeds invested elsewhere in the economy. The right-hand-side (RHS) represents the rate of return from holding the stock *in situ*. The first term simply represents capital gains (and will be zero at a steady state), while the second is the stock's marginal growth. The third term represents the cost savings effect due to a larger stock; specifically it accounts for the earlier assumption that $C_N < 0$ or that it is less costly to catch fish when there are

more fish to catch, and implies that there are incentives to retain a larger stock at the margin to take advantage of this cost savings effect.

Conditions (1.2), (1.6), and (1.7) form a system of three equations and three unknowns, h, N, and λ. From condition (1.7) take the time derivative of λ. Then use this relationship along with the relationship in (1.7) to substitute for λ and $\dot{\lambda}$ in equation (1.6) and solve for $\dot{h}$.

(1.9) $\dot{h} = \Phi(N, h)$

Equations (1.2) and (1.9) form a system of differential equations that may be solved simultaneously for the optimal time paths of $h(t)$ and $N(t)$, given any known point on the path. While $N(0)$ is known and fixed, the manager is free to choose $h(0)$. It is known that optimal stock and optimal harvest in long-run will be an equilibrium that may be determined, where $\dot{N} = \dot{h} = 0$. Indeed, it is well-established for this simple model that the equilibrium is a unique point that is conditionally stable, a saddle point (Conrad and Clark 1987).[2] When the equilibrium is conditionally stable there is a unique path that will lead to the equilibrium saddle point, and this path is known as the saddle path or separatrix (Figure 1.3). It is possible to find the path by starting near the saddle point and simulating the system backwards in time according to $\dot{N}$ and $\dot{h}$, until the point $N(0)$, $h(0)$ is determined. Again note that the $N(0)$ is given but that the manager is free to choose $h(0)$ to guarantee the system proceeds along the saddle path.

When problem (1.3) is linear in harvest, i.e. $C(h,N) = C(N)h$, a special case of the problem arises. In this case, the marginal effect of h on the Hamiltonian is

[2] It is optimal to move to equilibrium assuming there is only one and it is possible to do so (i.e. moving towards on unstable equilibrium would not be optimal).

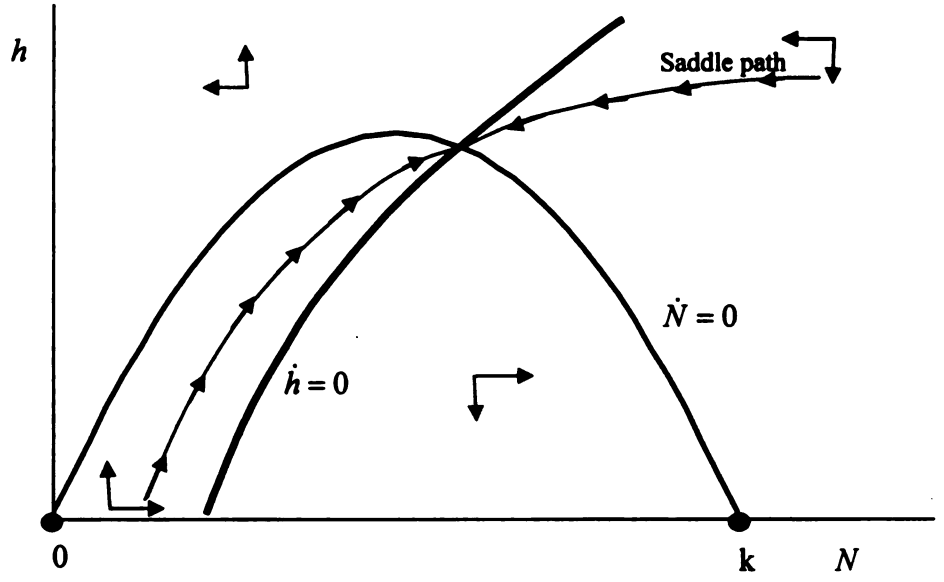

Figure 1.3. Phase-plane diagram illustrating the economically optimal harvest - a saddle path.

$$(1.10)\quad \frac{\partial H}{\partial h} = p - C(N) - \lambda$$

Clearly, this condition is independent of h. Assuming that the optimal point is interior, in the long-run condition (1.10) must equal zero, but there is no reason to expect this to be true at a given point in time. Moreover, values in condition (1.10) may not be freely chosen to force condition (1.10) to equal zero. If $\partial H/\partial h > 0$, then an increase in h increases the value of the Hamiltonian; hence h should be set as large as possible, h^{max}. This situation implies that at the margin a fish is more valued as a commodity than as a factor of fish production (i.e., for its reproductive potential). Conversely, if condition $\partial H/\partial h < 0$, then a decrease in h results in an increase in the value of the Hamiltonian; hence all harvesting should stop, $h = 0$. This may be interpreted as the value of the reproductive output of the first fish to be harvested is greater then that fish's value as a commodity.

An important case arises when $\partial H/\partial h = 0$. This outcome is called the singular solution. The necessary conditions for an optimum also include an arbitrage condition similar to (1.6). Therefore the singular solution involves, the equation of motion (equation 1.2) and

$$(1.11)\quad p - C(N) = \lambda$$

$$(1.12)\quad \dot{\lambda} = \rho\lambda + C_N h - \lambda G_N$$

The optimal path may be identified using a similar procedure as the nonlinear case. Take the time derivative of equation (1.11), $\dot{\lambda} = C_N(G(N) - h)$, and substitute this and the definition of λ from equation (1.11) into equation (1.12). This results in

$$(1.13)\quad \rho = G_N - \frac{C_N}{p - C(N)}$$

Condition (1.13), the "golden rule" expression, yields a unique value $N = N^*$ as the singular solution. Therefore, at the optimal equilibrium point $\dot{N} = 0$ and $h^* = G(N^*)$. The optimal harvest level is given as a feedback rule dependent on the state of the stock.

$$h(t) = \begin{Bmatrix} 0 & if & N(t) < N^* \\ h_{max} & if & N(t) > N^* \\ h^* = G(N^*) & if & N(t) = N^* \end{Bmatrix}$$

This is called a feedback rule, because the choice of h depends on the state variable N. In contrast, the solution to the nonlinear problem is a function of time, i.e., $h(t)$. According to the feedback rule, if the system is not at the steady-state, then controls should be set at extremes to achieve the steady-state as quickly as possible. When the control variable is set at the extreme value, the system is said to follow the most rapid approach path (MRAP) also commonly called a "bang-bang" solution. For a single state and control variable, a problem linear in the control variable is both necessary and sufficient for a "bang-bang" solution (Clark and Conrad 1987).

The above-illustrated models historically have been used to better understand single species management. Bioeconomic models first appeared in the fisheries management literature (Gordon 1954) and have subsequently become more complex, leading to common use in managing marine resources (Quinn and Deriso 1999, p 446) and terrestrial systems (Rondeau and Conrad 2003; Horan and Bulte 2004).

Traditionally, the biological models used as constraints in bioeconomic models have often been simple and focused on single species with a single impact on human

welfare. As computing technology increases, making these problems more tractable, it is important to relax simplifying assumptions and explore more realistic problems. Recently, there has been particular interest in systems where wildlife affects human welfare in multiple ways, creating both benefits and damages (Bhat et. al. 1996; Zivin et al. 2000; Rondeau and Conrad 2003; Horan and Bulte 2004). Also, ecological interdependencies have been included explicitly into bioeconomic models (Von Dem Hagen and Wacker 2001), and the bioeconomic approach has been extended to multi-species ecosystem management (Brock and Xepapadeas 2002).

1.3 A Conceptual Framework of a Wildlife-Human-Disease System

1.3.1 Disease ecology

In this thesis, bioeconomic models will be applied to management problems involving wildlife disease. Disease management problems involve multiple populations (e.g., infected and susceptible wildlife, domestic animals, humans, and the disease-causing parasites) interacting in multiple ways. Much could be learned by expanding on disease ecology concepts and incorporating human actions into wildlife-disease systems. Before developing the mathematical details, it is useful to illustrate a conceptual framework of a human-wildlife-disease system. Figure 1.4 illustrates such a framework.

Much work for various disease systems has gone into describing the shaded portion of the Figure 1.4 (see Grenfell and Dobson (1995) for a full review), which represents the ecological component of the disease system. Various authors have developed models of wildlife disease of varying complexity, and have addressed different objectives, answered different questions, and described different disease

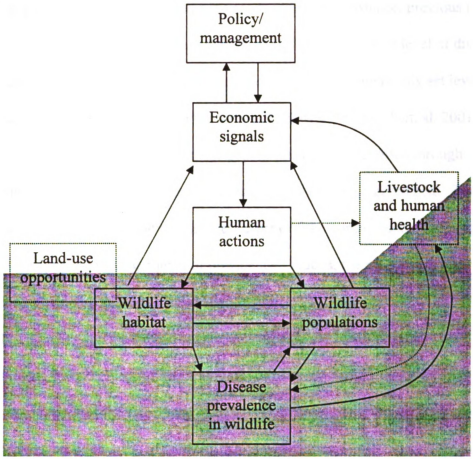

Figure 1.4. A conceptual framework for understanding a human-wildlife-disease system. The grey region on the bottom is the part of the system where biologist have traditionally focused their effort, and the white, upper region is where economist have traditionally focused their effort. Notice that there a number of connections between these to regions, indicated by arrows and boxes that overlap both regions. Relationships represented by solid lines are considered in this thesis, while relationships represented by dotted lines are left for future consideration.

systems (for examples of varying complexity see McCallum et al. 2001; Fulford and Roberts 2002; and Caraco et al. 2002). These studies have helped characterize disease systems and define potential management opportunities. For instance, previous models of wildlife disease management have largely assumed a target chosen level of disease prevalence (often zero) to be the management goal and have exogenously set levels of management action to achieve it (Barlow 1991b; Barlow 1996; Smith et. al. 2001; Smith and Cheeseman 2002). The focus of these papers was to analyze, often through simulation, the ability of different control methods at achieving a given goal. These papers did not consider how the technical efficiency of the control methods might influence the appropriate target level of prevalence. Indeed, the scope of most prior studies has remained confined to the shaded region.

The area outside the shaded region, and specifically how it interacts with a disease system, has received limited attention. Arrows leaving the shaded region have been taken to represent the effects the disease system has on human health, domestic animal health, and other areas of concern, but these areas of concern have been assumed not to provide feedback to the disease system. Similarly, arrows entering the shaded region are often assumed to represent relationships between the system and exogenous driving variables that affect the disease system. Yet, it is clear from figure 1.4 that the arrows leaving and entering the shaded area do not connect exogenous driving variables to the system, but rather complete endogenous feedback loops.

1.3.2 The effects of disease on economic values

It is important to recognize that the arrows leading out of the shaded region of figure 1.4

point to other components of a larger system, directly or indirectly connecting the ecological system to other variables that drive decision-making processes. Management agencies and society tend to be concerned with how disease impacts humans, property (i.e., livestock), and wildlife species that are valued for consumptive or non-consumptive purposes.

The relationship between wildlife disease and livestock or human health is often an area of high concern for management agencies (Cleaveland et al. 2001; Artois 1997). Human health is often of primary concern, however the ways of interpreting the relationships between wildlife and human health, and wildlife and livestock health are similar. Therefore, this thesis will focus on the wildlife-livestock relationship because this is better suited for the case study in the following chapters.

Traditionally, wildlife diseases, such as bTB in badgers, possums, and deer, are of concern primarily because of spillover to livestock. There are two ways to explore this relationship. The first, and more traditional approach, is to focus on management of the wildlife host and model a fixed relationship between the infected wildlife population, I, and the rate at which livestock become infected. This is often done as a fixed proportional damage function (Smith et al. 2001; Bicknell et al. 1999; Horan and Wolf 2005). The second option is to model a manageable relationship between wildlife hosts and livestock (i.e., investments in biosecurity), this places a "valve" in the "flow" of disease from wildlife to livestock (or visa-versa). In such a model human choices affect the relationship between the number of infected wildlife hosts and the rate at which livestock becomes infected. Recently, authors have begun looking at the effects of human actions, specifically livestock management practices and biosecurity investment,

(Scantlebury et al. 2004; Horan et al. 2004). There are modeling trade-offs between these two methods and they allow different insights to be gained.

The effect diseases have on wildlife populations may also be a concern (Daszak 2000; Dobson and Foufopoulos 2001; Miller 2003) to the extent that people value these populations. When these populations decline due to disease there is a loss in value to society.

One source of value is recreational hunting. Diseases, such as chronic wasting disease (CWD), that infect species valued for hunting may create concern due to their effects on recreational opportunities in addition to the standard health concerns. In the United States, hunting is a valuable industry. In 2001, hunting contributed approximately $671,660,644 to Michigan retail sales, provided 12,144 jobs, and over $8 million in state tax revenue (IAFWA 2003). If diseases cause wildlife populations to decline or if infected animals are less desirable, then hunters may reduce or move effort elsewhere, or incur greater costs when hunting.

Many wildlife populations provide important ecosystem services and have high existence values. Therefore, wildlife disease has also become a major concern for conservation. Infectious wildlife diseases may drastically increase the costs of species conservation or increase the risk of extinction (Haydon et al. 2002). Wildlife diseases also present significant barriers to the recovery of some species. Cattle diseases such as bTB, anthrax, and bovine brucellosis have been cited as a major barrier to the recovery of endangered woodland bison, *Bos bison*, in Canada (Mitchell and Gates 2002). Introduced avian malaria is the primary barrier preventing the recover of populations of Hawaiian low elevation bird species (Jarvi et al. 2001). Infectious disease is a constant

threat to populations existing at low levels, such as outbreaks of canine distemper in the Ethiopian wolf (Haydon et al. 2002). Additionally, introduced diseases have been implicated as the cause of decline in species ranging from amphibians to penguins (Duignan 2001; Muths et. al. 2003). As the value society places on conservation and endangered species increase, infected endangered species are likely to send stronger economics signals altering the way disease management is approached.

1.3.3 The link between economic signals and human responses

Human behaviors can impact disease systems, intentionally or unintentionally. Human actions are components of complex feedback loops, whereby ecological outcomes affect economic values that in turn induce human actions and affect ecological outcomes. Attempting to choose actions without accounting for these kinds of dynamic feedback effects can cause unintended consequences, cause management actions to fail to achieve broader resource management goals, and misallocate scare resources.

The first response in managing a wildlife disease is often to manage the host population, and wildlife disease management is often only feasible at the population level (Wobeser 2002). Such management schemes often result from the ecological threshold implication of certain transmission processes, but do not account for economic tradeoffs. Rather, most wildlife disease management has grown out of farm veterinary medicine and this may not be appropriate (Nishi et al 2002). The response of many game management agencies is to increase hunter permits for wildlife populations containing infected wildlife (Van Deelen and Etter 2003). Of course, such strategies would be inappropriate for threatened and endangered species. Some researchers have

investigated fertility control and vaccination (Smith and Cheeseman 2002; Barlow 1991b). However, these kinds of programs have been relatively unsuccessful and are often expensive (a notable exception is the oral vaccine program for raccoon rabies - this program is often considered effective but it is still expensive (MacInnes and LeBer 2000).

Alternatively, it may be possible to alter host habitat. Alterations in wildlife habitat have been implicated in the emergence of new diseases (Daszak 2001; Dobson and Foufopoulos 2001), but habitat management may also present an opportunity for managing disease emergence (Wobeser 2002). While changes in habitat are often thought of as deforestation, other changes also alter wildlife behavior and can lead to the emergence of diseases. For example, feeding wildlife has received considerable attention due to its role in garden bird disease (Hartup 2000) and bTB in wild deer (Schmitt et al. 2002). Indeed, the effects of feeding on other systems likely have gone underestimated (Daszak et al 2000). Therefore, human actions must also be taken into consideration when selecting transmission functions for modeling wildlife disease dynamics. Such consideration is presently lacking in the literature.

Finally, it is possible to alter human and livestock behavior. Changes in livestock behavior may affect opportunities for infection. Scantlebury et al. (2004) showed that changing livestock grazing patterns could lower the likelihood of bTB transmission to cattle from badgers. The choice to take action, and then which action to take, is ultimately based on economic signals.

1.4 A Mathematical Model

1.4.1 Understanding the biological system

It is important to understand the basic mathematical models of disease *sans* economics and human action and some of their implications to help assess the relevant components for a model of wildlife disease management. The following can be thought of as an overview of simple models that can capture the possible interactions within the shaded region of figure 1.4.[3] This discussion is expanded and these models explored in greater depth throughout this thesis.

Most wildlife-disease system models are based on the relationship between the number of susceptible (*S*), infected (*I*), and recovered (*R*) individuals in the host population (McCallum et al. 2001; Diekmann and Heesterbeek 2000). These models are known as *SIR* models. *SIR* models involve a set of three differential equations describing the growth of the *S*, *I*, and *R* populations. Many wildlife diseases are chronic and there is no recovered population (Barlow 1991b). Models developed for this kind of disease system need only account for the changes in the *S* and *I* populations and are therefore referred to as *SI* models. This thesis focuses on diseases that may be modeled using *SI* models. In the absence of management, the change in *S* and *I* may be written as

$$\dot{I} = G(I,N) + T(S,I) - A(I) \tag{1.13}$$

$$\dot{S} = Z(S,I,N) - T(S,I) \tag{1.14}$$

[3] For a short and thorough discussion see McCallum et al. (2001) and for a more in depth discussion consult Grenfell and Dobson (1995).

Functions G and Z are growth functions, with G possibly including some proportion of new offspring becoming infected and infectious from their mother.[4] The T function represents horizontal transmission, all transmission not passed from mother to offspring. Finally, the A function captures disease induced mortality.[5]

Human actions may affect all the terms in equations (1.13) and (1.14) and may also introduce new terms. Consider how the human actions of supplemental feeding, f, representing habitat manipulation, and harvesting, h, representing direct population management affect disease dynamics.[6] Harvesting clearly adds a term to equations (1.13) and (1.14). However, infected wildlife are often difficult, impossible, or very costly to identify in the field (Lanfranchi et al. 2003) making any harvest non-selective with respect to disease status. Harvests from each subpopulation are therefore assumed to proportional to the relative abundance of the sub-population, so that infected harvests equal hI/N and susceptible harvest equal hS/N. In contrast feeding may affect each of the ecological functions directly. Feeding may affect the growth function by altering carrying capacity (Walters 2001). Feeding also may affect transmission (as noted above by altering deer social behavior, causing deer to congregate and come into more frequent contact), and may lessen the effects of disease induced mortality. Equations (1.13) and (1.14) may be rewritten with human actions as

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[4] This may occur two ways *in utero* (vertical transmission) and through contact between mother and offspring after birth (pseudo-vertical transmission).

[5] $N = S+I$, in SI models, N is included separately to emphasis that the growth function is density-dependent.

[6] In the later chapters of this thesis supplemental feeding programs are used as an example of the way that humans interact with wildlife habitat to change animal behavior and disease dynamics, and harvests are used as an example of a direct impact on the host population. An explanation of why these two control variables are chosen is provided in the case studies, however for notational consistency I use them here as well.

$$\dot{I} = Z(I,N,f) + T(S,I,f) - A(I,f) - h\frac{I}{N} \quad (1.15)$$

$$\dot{S} = G(S,I,N,f) - T(S,I,f) - h\frac{S}{N} \quad (1.16)$$

Of particular interest may be the way that T is specified because the particular specification of T implies particular management opportunities (Schauber and Woolf 2003). Later chapters of this thesis explore how changes in T affect management trade-offs and ecological thresholds. However, it is important to understand that some specifications of T imply a host-density threshold (a density of wildlife) below which the disease will not persist and the system will converge to a point where $S > 0$, and $I = 0$. This type of argument is often put forth in support of culling programs. However, as noted above it is not possible only to cull the I population, so any cull will impose a cost associated with a potential excess harvest of healthy individuals. For some other specifications of T, no host-density threshold exists.

This simple framework for modeling wildlife disease without considering trade-offs may be expanded to include multiple species and populations or demographic components of a particular population. In general, the change over time of each subpopulation or species may be represented by a separate differential equation. This is demonstrated in Chapter 3.

1.4.2 The planner

It is often useful to begin an analysis of resource allocation by examining the social planner's problem in order to provide a benchmark that defines economically optimal allocation of resources. The process of identifying this benchmark highlights economic

and ecological tradeoffs and provides insights into policy opportunities, as well as insights into important variables and parameters that should be considered in future research. Moreover, the planner's problem provides a useful point of comparison for (sub-optimal) policy choices made in a decentralized setting.

Economists often assume that the goal of a social planner is to maximize discounted economic surplus. Economic surplus is often defined as the benefits from the system minus the costs. These costs and benefits include both benefits and cost generated by the system *in situ* and management. Explicit mathematical formulation of benefits and costs are delayed until the case studies of later chapters. For now, define social net benefits at time t as $SNB(h, f, S, I)$. Assuming the social planner is interested in an infinite planning horizon, and that a discount rate of ρ is applied to take time preference into account, the planner's problem becomes

$$\max_{h,f} \int_0^{\infty} SNB(h, f, S, I) e^{-\rho t} dt \tag{1.17}$$

s.t. equations (1.15) and (1.16), and $I(0)$ and $S(0)$

The problem now looks similar to the problem (1.3).

As with problem (1.3), the social planner must simultaneously decide how to allocate resources across at least four competing opportunities, taking into account the dynamic interactions between and among the pathogen, the wildlife host, livestock, and humans. First, the manager must decide how many resources to allocate to managing host density. Second, the manager must decide how many resources to allocate to habitat manipulations that affect behavior, transmission opportunities, and host

productivity. Third, the manager may allocate resources to biosecurity or altering human and livestock behavior to limit opportunities for transmission to valued species (including humans). Finally, as with other wildlife management programs, the manager and society must balance the resources allocated to manage wildlife disease with forgone opportunities elsewhere (Shogren et al. 1999). In order to do this optimally, the manager must recognize all of the interactions – not just ecological or economic relationships.

Ultimately, allocating resources involves making trade-offs. To understand these trade-offs it is important to consider all of figure 1.4. Increasing the detail within the boxes in figure 1.4 creates more relationships and more opportunities. One way the planner may increase management opportunities, considered in this thesis, is to identify a risk factor that may be targeted. If the population may be divided along another axis (other than health status) that is correlated with disease, then additional management opportunities that may lower costs may emerge. The ability to target "at risk" members of the human population has been widely used to manage human disease (just look at any disease awareness brochure). Yet, targeting has received little attention in wildlife disease management. Indeed, it is not possible to target infected wildlife so for a targeting strategy to work there must be an identifiable risk factor that is connected to health status.[7]

The complexity of the resource allocation problem rapidly increases as the opportunities increase. Therefore, it is vital for interdisciplinary teams to make qualitative trade-offs at different levels to identifying opportunities and relationships so

[7] Issues of targetablity are not unique to wildlife disease management and have also arisen in fisheries management and bycatch issues (Clark 1990). However, issues of targetablity greatly increase model complexity (Clark 1990; Mesterton-Gibbions 1996).

that insight may be gained from modeling. It is also important to gain inference from simpler models to aid in solving more complex models. Specifically, as the sub-model becomes more complex, model predictions may improve but the system as a whole may become intractable making it difficult to improve management. Conversely, oversimplified sub-models may cause inferences about the larger system to be irrelevant. Balancing these modeling trade-offs requires a solid understanding of the subsystems, clear identification of modeling objectives, intuition gained from simpler models, and cooperation and cross-education between experts in various fields.

1.5 Outline of this Thesis

The purpose of this chapter has been to provide an overview of wildlife disease and wildlife disease management issues, and also to introduce how bioeconomic modeling may be used in analyzing disease management. In the second and third chapters I present a case study based on the Michigan bTB-deer system. This case study is particularly useful because of the clear benefits from deer, in the form of hunting, and the clear costs of disease to the livestock sector that allow us to focus on increasing ecological complexity without increased economic complexity. In part, the motivation for these analyses is in response to Dasgupta and Mäler's (2003) call for economists to examine models with more ecological complexity without necessarily making the economics more complex. There are many unknowns about the dynamics of bTB in wild deer, however compared to other wildlife diseases, it is relatively well understood. Furthermore, the particular case study is geographically isolated making geographic spread of the disease of little concern (Garner 2001).

In the case, study supplemental feeding of deer in Michigan is used to increase deer productivity for hunting benefits. However, supplemental feeding alters deer behavior and fundamentally changes disease transmission dynamics, serving as one of the many sources of tradeoffs to be examined.

In Chapter 2 such behavioral changes are accounted for to examine how human decisions affect ecological thresholds that may lead to disease eradication, and the economic tradeoffs associated with managing the disease as well as the thresholds. This chapter advances earlier work in which human choices did not affect transmission (Bicknell et al. 1999) or in which ecological thresholds were not present (Horan and Wolf in press).

In Chapter 3, the model is extended to model how supplemental feeding alters contact rates between and among male and female deer, and sex-based management is explored. O'Brien et al. (2002) note that disease prevalence differs by sex. Therefore, explicit consideration of sex may allow one sex to be identified as "the riskier sex" and allow better targeting of disease control measures. Economic theory predicts that improved targeting should reduce control costs. On one hand, this could make eradication more likely to be optimal, but on the other hand, it could make it less costly to manage the disease endemically. Chapter 3 analyzes these tradeoffs. The fourth chapter provides a brief conclusion.

CHAPTER 2

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

2.1 Introduction

A large number of human, livestock, and companion animal disease have their origins with wildlife (Cleaveland et al. 2001). Many of these diseases have the potential to inflict large damages on society, but management may be costly. Wildlife managers are often faced with making trade-offs when determining how many resources to invest in disease management. Analysis of these trade-offs must span traditional disciplinary bounds, as a number of authors have already suggested (Wobeser 2002; Daszak et al 2001; Artois et al 2001).

The ecological literature on wildlife disease typically has focused on infectious disease dynamics *sans* humans, and has not been concerned with how humans affect the system (Wobeser 2002). Analyses that have included human actions often have focused on determining what is required for disease eradication (Barlow 1991b; Barlow 1996; Smith et al. 2001) often based on achieving some host population density threshold – an ecological threshold below which the disease naturally dissipates. These studies have not considered whether the goal of eradication meets broader social objectives, especially when it is costly to eradicate a disease. And even if eradication is optimal, is a strategy of immediately culling the stock to the ecological threshold necessarily the most efficient way to manage a disease system?

Evaluating such trade-offs is traditionally the role of economists. But, until recently, economic studies of disease in livestock or human populations usually

disregarded wildlife vectors and hosts. Such studies also tended to focus on eradication strategies, typically estimating the private costs to farmers and consumers under alternative on-farm control strategies (e.g., Mahul and Gohin 1999, McInerney 1996, Ebel et al. 1991, Dietrich et al. 1987 and Liu 1979). Some management-minded ecologists have included some costs of potential management strategies into their studies, but they have continued to concentrate on *in situ* disease eradication (Barlow 1991b and Wolfe et al. 2004), usually done through managing host population density to achieve the ecological threshold (Barlow 1991b, Barlow 1996, Caley and Ramsey 2001, Ramsey et al. 2002, Roberts 1996, Smith and Cheeseman 2002, Smith et al. 2001). These studies have not necessarily accounted for the full range of opportunity costs that such a strategy implies.

The focus on eradication in the literature may result from the fact that many wildlife management programs have explicitly stated goals of eradicating wildlife disease (Wobeser 2002). This probably results from the fact that wildlife disease management has grown out of on-farm livestock disease management (Nishi et al. 2002). The appropriateness of eradication goals for wildlife disease has rarely been questioned, in part because it is implicitly assumed that pursuing eradication results in the loss of few opportunities. However, trade-offs are made implicitly through political and budget allocation processes, and placing wildlife management decisions in the context of a bioeconomic model increases transparency and may result in more efficient allocation of resources (Shogren et al. 1999). The use of such analysis not only aids managers in deciding how much disease management to undertake, but also which avenues likely provide the best returns.

Besides the role of population density on disease transmission, the way humans affect the environment of wildlife may greatly affect the disease transmission process (Daszak et al. 2000; 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). Specifically, supplemental feeding of deer has been shown to change deer behavior (Grenier et al. 1999) and contact rates between individuals (Garner 2001). This may have a substantial effect on host-density thresholds. This presents managers with the problem of managing the host-density threshold in addition to the host population density.

In this chapter we develop a continuous-time, deterministic bioeconomic model of wildlife disease and management that incorporates human environment-interactions, focusing on a case study of bovine tuberculosis (bTB), *Mycobacterium bovis*, in Michigan white-tailed deer, *Odocoileus virginianus*. We begin by revisiting disease ecology theory to incorporate the effects of human-environmental interactions. We show how these actions potentially affect ecological thresholds. Then we solve for the management regime that maximizes economic welfare, so as to tailor management to better allocate scarce resources.

2.1.2 Study area

Michigan is the only known area in the United States where bTB has become established in a wild deer population. In the mid-1990s signs of bTB started to re-emerge in the wild deer population, and by the end of the decade some farms were also becoming infected. Michigan's bTB accredited-free status was revoked in June 2000 and the state was required to adopt a testing program for all Michigan cattle, goats, bison, and captive cervids. In addition, other states gained the freedom place movement restrictions on Michigan livestock (MDA; USDA-APHIS 1999).

In 2004, Michigan received "split state" status for bTB, resulting in two disease management zones having separate requirements for animal movement, identification and testing. This status came about after extensive testing found that the bTB outbreak was confined to the northeast corner of Michigan's Lower Peninsula. Regulatory costs are now primarily confined to this area. Michigan agriculture industry is concerned about the costs of disease and disease control, and therefore supports culling the deer population to eradicate the disease. However, such measures could be costly to recreational hunters, particularly since deer hunting is arguably the highest-valued use of the land in the infected region (Horan and Wolf 2005).

Bovine TB among Michigan white-tailed deer is primarily concentrated in a four-county area in the northeastern part of the Lower Peninsula. This area was designated deer management unit (DMU) 452 and is less-formally known as the 'core' (see Hickling 2002). While infected deer have been found beyond this area, the disease does not appear to be sustainable outside the core. This has led many to speculate that unique, core-specific features such as human-environment interactions – particularly

feeding programs that encourage deer to congregate – have enabled the disease to become endemic (Hickling 2002). Indeed, prior to 1995, only eight cases of bTB had been reported in wild deer from North America, and conventional wisdom held that the disease was not self-sustaining in wild deer populations (Schmitt et al. 2002).

Several hunt clubs in the core have sponsored feeding programs to increase deer density. Originating in the late 1800's, these clubs purchased large amounts of core area land on which they could restrict access, allowing only their members to hunt. This land was desirable for hunting because 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). The hunt clubs, desiring greater density, began aggressive deer feeding programs to encourage herd growth. At times, these programs have included dumping tractor-trailer loads of food in the woods and edge areas, with the resulting massive food piles being visible from the air along with the tracks of many congregating deer (Hickling 2002). As a result, the deer density increased in the core area to an estimated 25 deer/km^2 by the mid-1990's (O'Brien et al. 2002).

2.2. A Model of Wildlife Disease with Human-Environmental Interactions

We begin by revisiting the basic mathematical models of disease *sans* economics and human action and some of the implications of these models to help understand how incorporating human actions affects outcomes. We adopt the basic *SI* model described in Chapter 1, although we now add specificity to the model. Let S be the susceptible

population and I be the infected population, with the aggregate population being $N = S + I$. Assuming the population is closed and exists on a fixed land area, population size and density can be shown to be interchangeable. Changes in S and I are written as

$$\dot{I} = G(I,N,f) + T(S,I,f) - A(I,f) - hI/N \quad (2.1)$$

$$\dot{S} = Z(S,I,N,f) - T(S,I,f) - hS/N \quad (2.2)$$

where G and Z are density-dependent growth functions, and G includes some proportion of new off-spring becoming infected and infectious before or shortly after birth from their mother (i.e., vertical or pseudo-vertical transmission). T is a function representing horizontal transmission (all transmission not passed from mother to off-spring), A is an additive mortality function capturing disease-induced mortality, h is harvest, and f is supplemental feeding.[8]

Define G to be the following modified form of the logistic growth function: $G = I(vb-\delta)[1-(N/k)(1-\tau f)]$. The first modification relative to the standard logistic function involves pseudo-vertical transmission applied to the births attributed to infected individuals (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 parameter v, which may be thought of as the probability of an infected mother transmitting bTB to an offspring.[9] The second modification involves the effects of feeding on the carrying capacity, k. Assume that feeding increases the effective carrying capacity in a similar manner as Walters (2001). Denote the effective

[8] N is identified separately in G and Z because the growth function is assumed to be density-dependent, and by including N we aim to emphasize that this density dependence relates to the entire population.

[9] Pseudo-vertical transmission in the model should not be confused with *in utero* vertical transmission nor is pseudo-vertical restricted to transmission through lactation. In the model pseudo-vertical is included to take account of the observation that related deer are more likely to be in the same health class (Blanchong 2003).

carrying capacity by $k/(1-\tau f)$, where τ is a parameter. As $f \rightarrow 1/\tau$, the carrying capacity is effectively eliminated so that the deer population grows at its maximum rate.[10]

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

Next consider the specification of the transmission function, T. Disease modeling theory states that the force of infection governs disease dynamics. The force of infection is defined as the probability of a susceptible individual becoming infected per unit time (Diekmann and Heesterbeek 2000). Assuming the force of infection is proportional to the number of infected individuals, I, then this probability is defined as I times the conditional probability of infection in a susceptible individual given contacts between infectious and susceptible individuals, β, times the number of contacts, $C(N)$ (Diekmann and Heesterbeek 2000; Heesterbeek and Roberts 1995). $C(N)$ and β are generally modeled as deterministic. Given this specification, T may be defined as

$$T(S,I) = C(N)\beta SI \tag{2.3}$$

where β represents the conditional probability of infection in a susceptible individual given contacts between infectious and susceptible individuals, and $C(N)$ represents these

[10] Carrying capacity is a complex concept involving more than food. It is known that increasing available food relaxes the carrying capacity "constraint" on growth therefore $\tau > 0$, but we also know that food can not relax the carrying capacity "constraint" completely, as another resource will eventually limit population growth after a certain amount of food is provided. This, along with the cost associated with supplemental feeding implies that there is an upper-bound to f, with the maximum upper bound being $1/\tau$. This upper bound is made explicit in our simulation.

contacts (hence, $C(N)\,\beta$ represents the probability that a susceptible individual becomes infected at any point in time). $C(N)$ and β are generally modeled as deterministic.

The contact function, C, is defined to be a modified form of the contact function proposed by Roberts (1996)

$$(2.4) \quad C = \frac{(1-\varepsilon+\varepsilon N)}{N}$$

Here $\varepsilon \in [0,1]$ is a shifting or contact parameter. Assumptions about the value of ε have important implications for management. When $\varepsilon = 1$, the contact function takes a value of one. In this case, T simplifies to the classic mass-action or density-dependent model of disease transmission, with recruitment from the S population to the I population depending entirely on the host population density (McCallum et al. 2001). Density-dependent disease transmission is often applied in theoretical models, but may not hold up when tested empirically (McCallum et al. 2001). As we show below, density-dependent transmission allows for a population density threshold below which prevalence begins to decline, and this has been the rationale for many culling or density management programs.

When $\varepsilon = 0$, the contact function becomes $1/N$, and T represents frequency-dependent transmission. Frequency-dependent models are often employed to model sexually transmitted diseases. McCallum et al. (2001) argue that frequency-dependent form is appropriate in such a case because mating contacts may be roughly constant, so that the rate of infection does not change with total host density. The frequency-dependent form has been shown to fit data in certain case studies better than the density-dependent form (Begon et al. 1998; Begon et al. 1999). The frequency-dependent form

is often used when there is reason to believe that transmission is not directly proportional to host density. Unlike the density-dependent transmission function, the frequency-dependent transmission function allows the transmission rate to be independent of host density (McCallum et al. 2001). We illustrate this below.[11] This observation emphasizes the fact that culling to reduce host density will not result in the eradication of a disease under frequency-dependent transmission.

Reality probably lies somewhere in between these two extremes (Schauber and Woolf 2003), with $\varepsilon \in (0,1)$. Furthermore, human-environmental interactions that alter habitat and animal behavior almost certainly affect the degree to which contacts are density-dependent or frequency-dependent. For instance, deer, which typically segregate by sex (O'Brien et al. 2002), relax social boundaries and increase between-sex contacts around supplemental feed piles (Grenier et al. 1999). This implies that, under natural conditions, contacts for the total population should not be fully density-dependent due to avoidance behavior, but feeding may cause social barriers to break down so that the system moves closer to full density dependence.

Feeding is included in the contact function to account for changes in social interaction and the assumption that feeding generally concentrates deer (Schmitt et al. 2002). Assume feeding enters the contact function linearly so that transmission becomes

$$(2.5) \quad T(S,I,f) = \frac{(1-\varepsilon+\varepsilon N)(1+\omega f)}{N}\beta SI$$

where ω is a parameter.

Now consider mortality due to the disease. In the absence of supplemental

[11] The result that transmission is independent of host density in frequency-dependent model creates a theoretical problem, namely that transmission is positive even when density is zero (Roberts 1996).

feeding, the disease-related mortality function A is specified simply as αI, where α is a disease-induced mortality rate. However, 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). Total mortality due to the disease is therefore specified as $\alpha(1-\chi f)I$, where χ is a parameter.

Finally, consider the harvest terms in equations (2.1) and (2.2). 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. Assuming the disease is uniformly distributed among the population (a potentially strong assumption), 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.

Given the specification of the model, it is intuitively easier and mathematically more convenient to work in N-θ space, where $\theta=I/N$ is the disease prevalence rate. The system of equations (2.1) and (2.2) can therefore be written as

$$\dot{N} = rN\left(1-\frac{N(1-\tau f)}{k}\right)-\alpha(1-\chi f)\theta N - h \tag{2.6}$$

$$\dot{\theta} = b(1-v)\left(\frac{N(1-\tau f)}{k}-1\right)\theta+[\beta(1+\omega f)(1-\varepsilon+\varepsilon N)-\alpha(1-\chi f)](1-\theta)\theta \tag{2.7}$$

Equation (2.7) illustrates our earlier claim that the horizontal transmission rate is independent of host density under frequency dependency. To see this, set $\varepsilon = 0$ and notice that the horizontal transmission term becomes $\beta(1+\omega f)(1-\theta)\theta$, which is independent of N.

2.3. Ecological Thresholds

The goal of many wildlife disease management programs, eradication, motivates a discussion of the implications of human-environmental actions. Often programs aim to manage the host population at or below an ecological threshold. How do human-environmental interactions affect this threshold? The discussion revolves around equation (2.7). Note that h does not affect $\dot{\theta}$ directly, but it does affect $\dot{\theta}$ indirectly through its affects on N (provided $v = 1$ and $\varepsilon = 0$ do not both hold, in which case N does not influence $\dot{\theta}$). For given values of f and θ, the $\dot{\theta} = 0$ isocline can be solved for

$$(2.8) \quad \hat{N}(f,\theta) = \frac{(1-v)kb + k[\alpha(1-\chi f) + \beta(\varepsilon - 1)(1+\omega f)](1-\theta)}{(1-v)(1-\tau f)b + \beta k\varepsilon(1+\omega f)(1-\theta)}$$

which represents a host-density threshold as a function of f and θ. In the simple case in which $v = 1$ (which we focus on in the numerical example), the $(1-\theta)$ terms cancel and $\hat{N}$ is only a function of f. In this restricted case, holding f fixed, the $\dot{\theta} = 0$ isocline drawn in (N,θ) space is a vertical line at $\hat{N}(f)$. Disease prevalence is increasing ($\dot{\theta} > 0$) for values of $N > \hat{N}(f)$, and prevalence is decreasing ($\dot{\theta} < 0$) for values of $N < \hat{N}(f)$. Hence, disease prevalence will decline towards zero if the population is kept below the threshold, $\hat{N}(f)$.

Of course, the value of f is not necessarily fixed. For the simple case of $v = 1$, it is easily verified that $\partial\hat{N}/\partial f < 0$: 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, when $v = 1$, feeding increases the rate of change in prevalence by increasing transmission while decreasing disease-related mortality. However, if $v < 1$ the

relationship between f and $\hat{N}$ becomes 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 2.1. Using the parameter values from Table 2.1 and setting θ=0.025, we find that $\partial\hat{N}/\partial f < 0$ for all values of v. However, the marginal effect of changes in f on $\hat{N}$ are much greater when v is larger, as might be expected due to the counteracting fertility effect when v is small.[12] Although we concentrate on the case of $v = 1$ in what follows, we do explore the effect of changes in v in the sensitivity analysis section of this chapter.

It is helpful to understand how assumptions about v affect the impact of different management options, specifically feeding, as this will influence the trade-offs that could emerge. *Ceteris paribus*, increases in feeding results in a decrease in the threshold if the prevalence is kept fixed (Figure 2.1). Yet, the effect of feeding on the threshold is smaller for lower values of v. If v is small a reduction in feeding may have small effect on the host-density threshold, but if v is large a reduction in feeding may greatly increase the host-density threshold. The effect of a change in v is further explored in the sensitivity analysis section of this chapter.

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

[12] From (2.8), it should be clear that the fertility effect is larger when θ is larger, and that $\partial\hat{N}/\partial f > 0$ will result as $\theta \to 1$. However, the value of θ at which the slope changes signs is much larger than the values that optimally arise in our bioeconomic analysis.

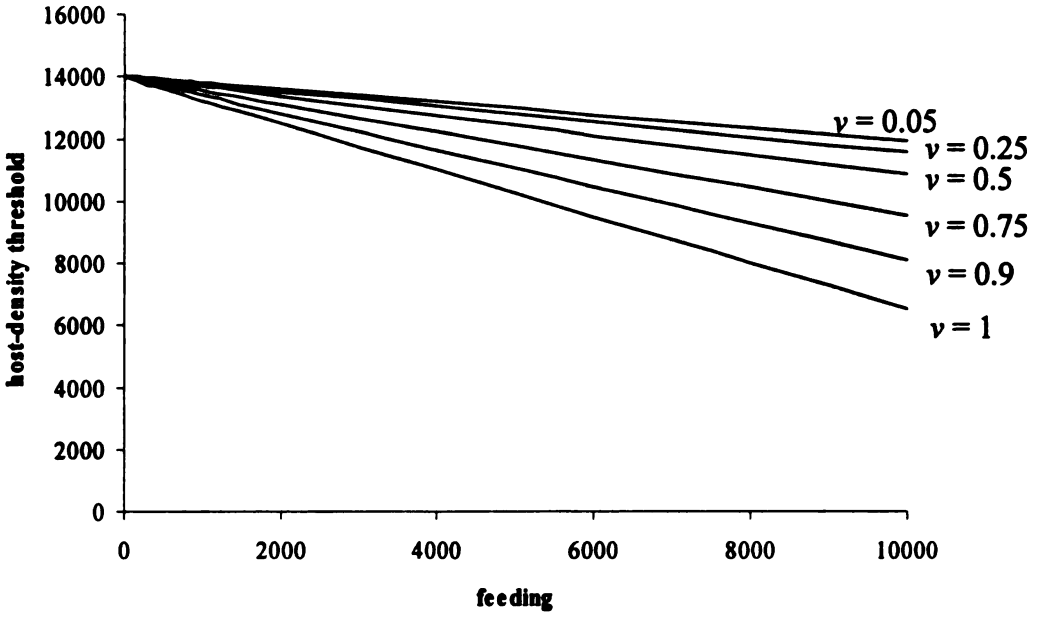

Figure 2.1. The relationship between the host-density threshold and feeding for a prevalence of 0.025.

Table 2.1. Parameter values and descriptions. Methods of calculation are described in Appendix I.

Parameter	Description	Value
N_0	initial population size	13,298
θ_0	initial prevalence	0.0023
r	intrinsic rate of growth	0.5702
δ	per-capita mortality rate	0.3623
k	carrying capacity	14,049
τ	coefficient for feeding effect on k	8.0×10^{-5}
β	transmission coefficient	3.39×10^{-5}
ω	coefficient for feeding effect on β	2.64×10^{-6}
ε	contact coefficient	0.75
v	rate of pseudo-vertical transmission	1
α	disease induced mortality rate	0.3556
χ	coefficient for feeding effect on α	5.32×10^{-5}
p	value of harvested healthy deer	1270.80
c/q	marginal harvesting cost / catchablity coefficient	231,192
w	unit cost of feeding	36.53
D	marginal damages to the livestock sector	5491
ρ	discount rate	0.05
f^{max}	maximum feeding level	10,000

this imply different economic and ecological tradeoffs, and a planning agency interested in managing the disease and hunting interests must assess the trade-offs that emerge. We now turn to a bioeconomic model to evaluate these tradeoffs in order to choose a socially desirable management strategy.

2.4. A Bioeconomic Model

2.4.1 Economic Specification and Optimality Conditions

The economic specification of the model is essentially the same as that of Horan and Wolf (2005). Suppose that 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 minus the damage costs associated with infections to the livestock sector. Hunting provides utility through the process of shooting deer as well as the act of 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., $p \geq p_I$. For simplicity, we set $p_I = 0$ so that harvests of infected animals yield no benefits.[13] The benefits from hunting are therefore $phS/N = p(1-\theta)h$.

Let harvests occur according to the Schaefer harvest function (although in general this specification is not required), and that the unit cost of effort, c, be 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.

[13] This assumption should not affect the qualitative nature of the results, but it may affect the trajectories in the numerical exercise.

Finally, the costs of the bTB infection to the livestock sector must also be considered. Denote the variable economic damages caused by infected deer as $D(I)$ (with $D(0) = 0, D' > 0, D'' \geq 0$). Lost stock, increased testing, and business interruption losses due to infections in the cattle herd result in variable damage costs.[14] We use a linear damage function in the numerical example, $D(I)=DI$, where D is a parameter representing marginal damages (although in general this specification is not required).

Given this specification of the model, and assuming a discount rate of ρ, the social planner's problem is[15]

$$\max_{h,f} \int_0^\infty [ph(1-\theta) - \frac{ch}{qN} - wf - D(\theta N)]e^{-\rho t} dt$$
$$s.t. \quad (7),(8); \quad N(0) \text{ and } \theta(0) \text{ given} \tag{2.9}$$

Problem (2.9) is a dynamic optimization problem that may be classified as a linear control problem, since the objective function and constraints are all linear in the control variables, h and f.

To solve problem (2.9), we first define the current value Hamiltonian

$$H = ph(1-\theta) - \frac{ch}{qN} - wf - D(\theta N) + \lambda \dot{N} + \mu \dot{\theta} \tag{2.10}$$

where λ and μ are the co-state variables associated with the host population, N, and

[14] Trade restrictions and federally-mandated testing programs also result in significant lump sum damages. These lump sum costs are primarily policy-induced and, when significantly large, may affect the optimal management trajectory. Analysis is restricted an optimal plan in the absence of these lump sum costs, so that the solution is efficient from Michigan's point of view. Automobile accidents and damage to agricultural crops are also deer induced costs and would be required for an optimal deer management plan (Rondeau 2001; Rondeau and Conrad 2003). These other costs are ignored in order to focus on the impacts of disease.

[15] This problem is identical to the one analyzed by Horan and Wolf (2005), except that the equations of motion are different. In this problem the biological constraints allow for ecological thresholds and for $v < 1$.

disease prevalence, θ, respectively. The marginal impact of harvests on the Hamiltonian is given by

$$(2.11)\quad \frac{\partial H}{\partial h} = p(1-\theta) - \frac{c}{qN} - \lambda$$

The right-hand-side (RHS) of expression (2.11) is the linear coefficient of harvests in the Hamiltonian. If this expression is positive so that marginal rents, $p(1-\theta) - c/(qN)$, 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. 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.

Now consider the marginal impacts of feeding on the Hamiltonian

$$(2.12)\quad \frac{\partial H}{\partial f} = -w + \left\{\lambda N\left(\frac{rN\tau}{k} + \alpha\chi\theta\right) + \mu\frac{-b(1-v)\theta N\tau}{k}\right\} + \mu\left((1-\varepsilon+\varepsilon N)\beta\omega + \alpha\chi\right)(1-\theta)\theta$$

The RHS of expression (2.12) 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 (2.12) 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, representing 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).

An optimal solution also requires two adjoint equations.

$$\dot{\lambda} = \rho\lambda - \frac{\partial H}{\partial N} \tag{2.13}$$

$$\dot{\mu} = \rho\mu - \frac{\partial H}{\partial \theta} \tag{2.14}$$

These conditions prevent intertemporal arbitrage opportunities: if they were not satisfied, then gains could be made from reallocating harvests of 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.

$$\rho = \frac{2rN(\tau f - 1) + k(r + \alpha(\chi f - 1)\theta)}{k} + \frac{\dot{\lambda}}{\lambda} + \frac{ch}{\lambda q N^2} - \frac{D\theta}{\lambda} + \frac{\mu\left[k\alpha(1 - \chi f) + (b(1 - v + (-1 + v)\tau f) + k\beta\varepsilon(1 + \omega f))\theta N^2 - k\beta\varepsilon(1 + \omega f)\theta N^2\right]}{\lambda k N^2} \tag{2.15}$$

$$\rho = \left\{\frac{b(v-1)(k + (\tau f - 1)N)}{k} - (1 + \omega f)(1 - \varepsilon + \varepsilon N)\beta(1 - 2\theta) - 2\alpha(\chi f - 1)\theta\right\} + \frac{\dot{\mu}}{\mu} - \frac{ph + DN + \alpha(1 - \chi f)N\lambda}{\mu} \tag{2.16}$$

Consider condition (2.15). The left-hand-side (LHS) of conditions (2.15) 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 (2.15) is $\partial\dot{N}/\partial N$, or the stock's own marginal growth. The second RHS term of conditions (2.15) represents the capital gains to holding the stock *in situ*, i.e. the rate of growth in

the marginal value of the stock. The other terms in (2.15) account for other costs and benefits from the deer population. These include marginal savings in harvesting costs (as deer are less costly to find when they are more abundant), marginal damages (as the infected stock is expected to increase along with the aggregate population), and marginal costs of increased transmission (as more deer leads to more infectious contacts).

Condition (2.16) is also a "golden rule" expression, although it has slightly different interpretation. The discount rate now 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.

2.4.2 Characterizing the double-singular solution

The overall solution to the problem 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 (2.11) and (2.12) 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 (2.11) or (2.12) 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; Clark 1990). Blocked intervals will be shown to introduce some interesting complexities into the model. A potential third type of solution is a fully constrained solution when neither condition (2.11) or (2.12)

vanishes. However, it is first necessary to understand the fully unconstrained solution (i.e. the double-singular solution, which arises within a free interval – a period of time during which neither control is blocked) to understand how the solution transitions between solutions and between blocked and free intervals. Additionally, it is known that "the optimal path must always lie as close as possible to the [double] singular path" (Clark 1990).

Condition (2.11) and (2.12) both equal zero for a double singular solution. These conditions may therefore be used to solve for λ and μ, and can then be substituted into the "golden rule" conditions (2.15) and (2.16). Moreover, conditions (2.11) and (2.12) 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 the differential equations (2.7) and (2.8) 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.[16] The double-singular path is state-dependent, meaning

[16] 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 (2.11) and (2.12) equal to zero and noticing that the coefficient matrix for the vector [λ μ] 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.

that it depends on the initial state of the world. Therefore, different singular paths will arise for different starting values. This differs from most linear control problems, for which a singular path (or point, as in Chapter 1) is uniquely defined and "bang-bang" controls are needed to jump to the singular solution along a MRAP when the system is not initially on the singular path (Clark and Conrad 1987).

2.4.3 Characterizing the partial-singular solution

It is possible that the feedback rules just derived will yield values of $f > f^{max}$, $f < 0$, 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 (2.12) will no longer vanish, and f must be set to either its minimum or maximum value. Moreover, this means that (2.12) 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 (2.11) to solve for λ and $\dot{\lambda}$ and substitute these expression into (2.15), 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 (2.16). 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.

2.5. 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.

The data used to parameterize the model are listed in Table 2.1. We have used the best available data for the Michigan bTB case, however research on this system is still evolving at 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.

2.5.2 Characterizing the phase plane

We begin our analysis by drawing the phase plane (Figure 2.2) associated with the solution to problem (2.9). First, we determine the feedback rules for the double-singular solution and determine the loci of points for which $f(N, \theta) = 0$ and $f(N,\theta) = f^{max}$ (the case of $h(N, \theta)=0$ is unapproachable in the resulting dynamic system, so it is ignored). These loci of points, plotted as dotted lines, determine boundaries that divide the state

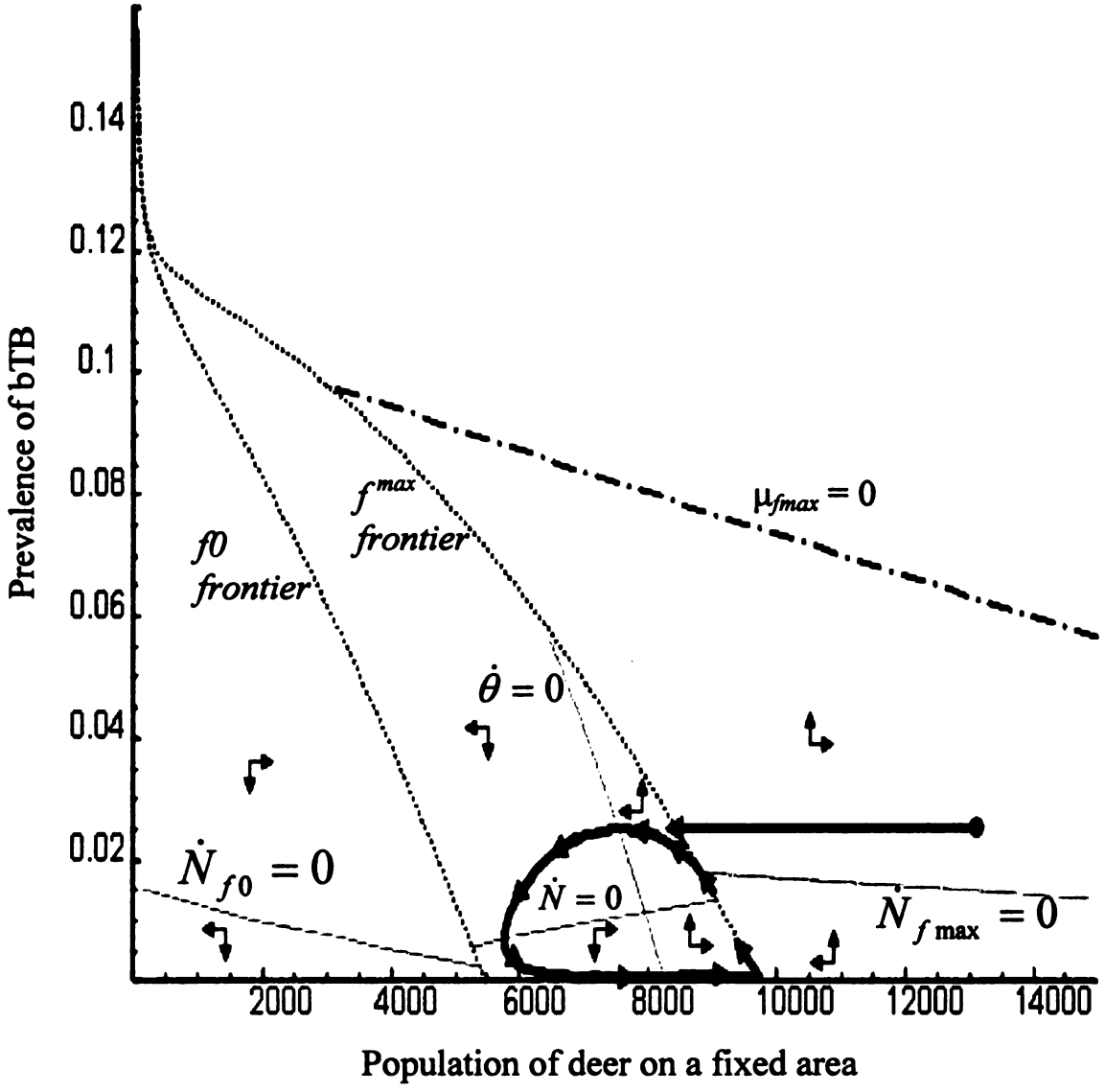

Figure 2.2. Phase-plane diagram illustrating dynamics and the simulated optimal trajectory, see detailed explanation in text.

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. Also, a boundary for $\mu(N, \theta) = 0$, given $f=f^{max}$, is presented as a broken line in Figure 2 and labeled $\mu_{fmax} = 0$.[17] Only below this boundary will μ be less then zero (implying that the disease is socially costly), as is required for an optimal solution. Paths leading to this boundary are deemed sub-optimal.

Next, we determine the $\dot{N} = 0$ and $\dot{\theta} = 0$ isoclines within each region. Figure 2.2 illustrates that 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.

2.5.3 The optimal path

Given the phase plane, 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 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

[17] There also exist $\mu = 0$ boundaries for the unconstrained region and the $f = 0$ region; however, given starting values in the range believed to exist for this case study, these boundaries do not influence the optimal path.

considered. The phase arrows indicate that pursuing the partial-singular solution will lead to the $\mu_{fmax} = 0$ boundary and is therefore infeasible. The only feasible 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 a feasible path in one of the other regions. A feasible double-singular path emerges at the $f=f^{max}$ frontier and heads into the interior region. The optimal trajectory is governed by the local dynamics, indicated by the phase arrows. Given that the intersection of the $\dot{\theta} = 0$ and $\dot{N} = 0$ isoclines 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.[18] 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 2.3, 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, as does the sensitivity analysis in section 2.6.

[18] Due to the deterministic nature of the problem an arbitrary cutoff must be used to say exactly when the disease is eradicated.

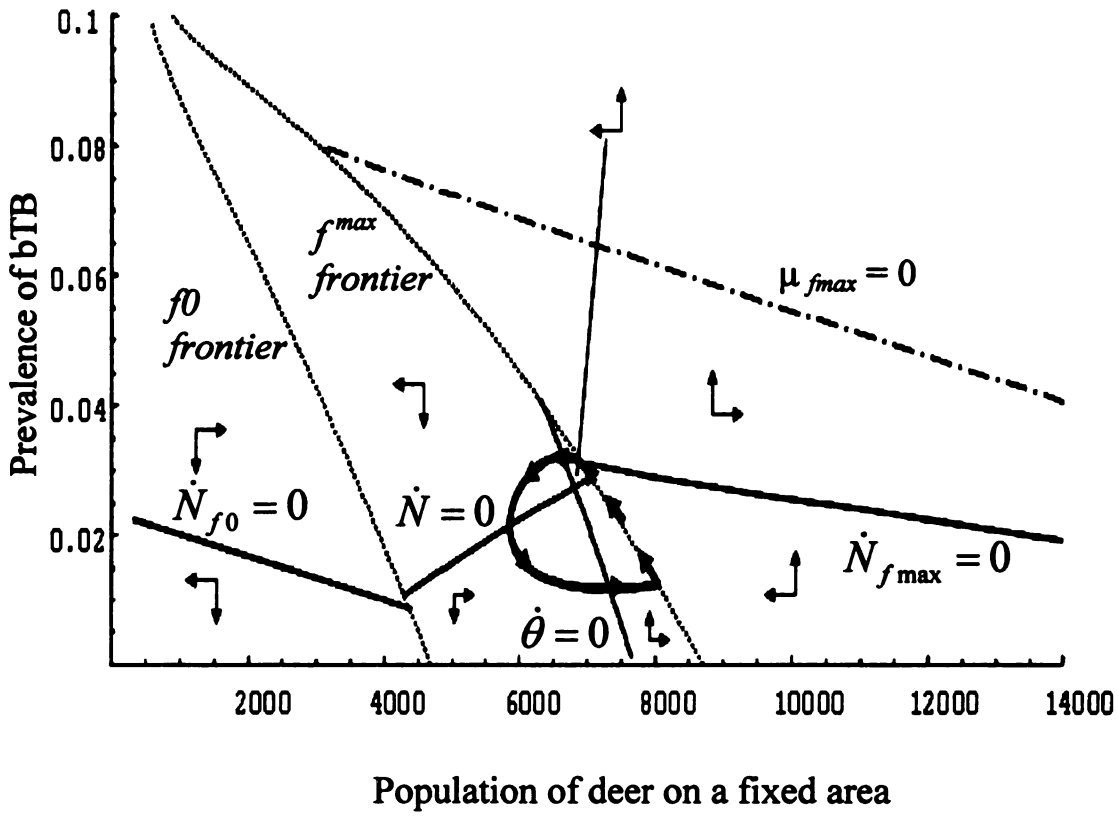

Figure 2.3. This phase-plane shows the optimal interior cycle when the discount rate is increased to 15%, holding all other parameters constant. On comparing the results with Figure 2.2, it is clear that by changing this one parameter the optimal disease prevalence does not approach zero and the optimal trajectory remains well above the x – axis.

After intersecting the $f=f^{max}$ frontier, the optimal path travels along the f^{max} frontier by "chattering" between the constrained and unconstrained regions.[19] 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 2.2).

The final part of the optimal path that must be determined is the initial cull. The "premature switching principle" suggests that it is optimal 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 wildlife 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 herd under much more controlled conditions (Frye 1995).

2.5.4 Endogenous ecological and economic thresholds

Note that prevalence is increasing to the right of the $\dot{\theta} = 0$ isocline, while

[19] 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 Swallow's (1990) solution for a problem depended on the current state of two state-variables. This problem also appears to have the potential for chattering for a sub-set 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.

prevalence is decreasing to the left of the $\dot{\theta} = 0$ isocline. Hence, the $\dot{\theta} = 0$ isocline represents the optimal host-density threshold – the value $N = \tilde{N}(\theta)$ below which the disease dissipates, given a value of θ. The expression for $\tilde{N}(\theta)$ will differ from the expression for $\hat{N}(\theta, f)$, the host-density threshold defined by equation (2.8) (although the *values* of these two expressions will be equivalent if $\hat{N}$ is evaluated at the optimal value of *f*). The reason is that $\hat{N}(\theta, f)$ is an ecologically determined threshold, given values of θ and *f*. In contrast, $\tilde{N}(\theta)$ reflects both ecological and economic considerations, as it is endogenously determined based on the optimal choice level of feeding, *f*(*N*, θ). Indeed, $\tilde{N}(\theta)$ is determined by plugging *f*(*N*, θ) into the expression $\dot{\theta} = 0$ and solving for *N*. Because the feedback rule *f*(*N*, θ) is derived based on economic-ecological tradeoffs, the optimal host-density threshold, $\tilde{N}(\theta)$, also reflects these tradeoffs. Other choices of feeding would produce different host-density thresholds, but these thresholds would be suboptimal.

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. The intuition for the cyclical path is essentially the same as the intuition behind Horan and Wolf's (2005) optimal solution under the special case of strict frequency-dependent

transmission (for which the optimal host-density threshold has no strictly ecological component, but rather is based entirely on economic-ecological tradeoffs affecting the optimal choice of f). That is, 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 dis-investment of the disease is warranted (i.e., the population is reduced below the host-density threshold, a process which is aided by a concomitant reduction in feeding). 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. However, unlike the Horan and Wolf (2005) model, the ecological threshold in the present model does not depend solely on changes in feeding – there is also a purely ecological component due to some degree of density-dependent disease transmission. This implies lower control costs in the present model, which translates into much lower prevalence rates than in the model developed by Horan and Wolf (2005).

2.6. 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 analyses 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 one biological parameter where empirical and theoretical knowledge is significantly lacking: the rate of pseudo-vertical transmission, v.[20] 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 should be qualitatively similar to those differences that would arise for the present model.[21]

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 then sets of unrelated animals, as is the case for

[20] 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 a lower level, and the deer population is maintained at a higher level. Furthermore, there is no need for a periodic call after the initial reduction in population.

[21] 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 (also see Figure 2.3). Since a larger discount rate means a less balanced weighting of near and far term benefits, feeding is increased and larger population with a larger disease prevalence is maintained. Higher long-term damages are traded off for increased near-term productivity. Horan and Wolf (2005) find similar results for larger marginal damages, feeding costs, and the disease induced mortality rate.

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 ecological model, it is possible that the pseudo-vertical transmission rate may have significant impacts on the optimal management strategy.

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 2.4), indicating that disease prevalence is optimally diminished at larger values of N. The reason is that a smaller v reduces vertical and, hence, overall transmission. The ecological threshold, $\hat{N}(\theta, f)$ 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 2.3). A decrease in the value of v causes the $\dot{N} = 0$ isocline to rotate upwards. This indicates that, at 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.

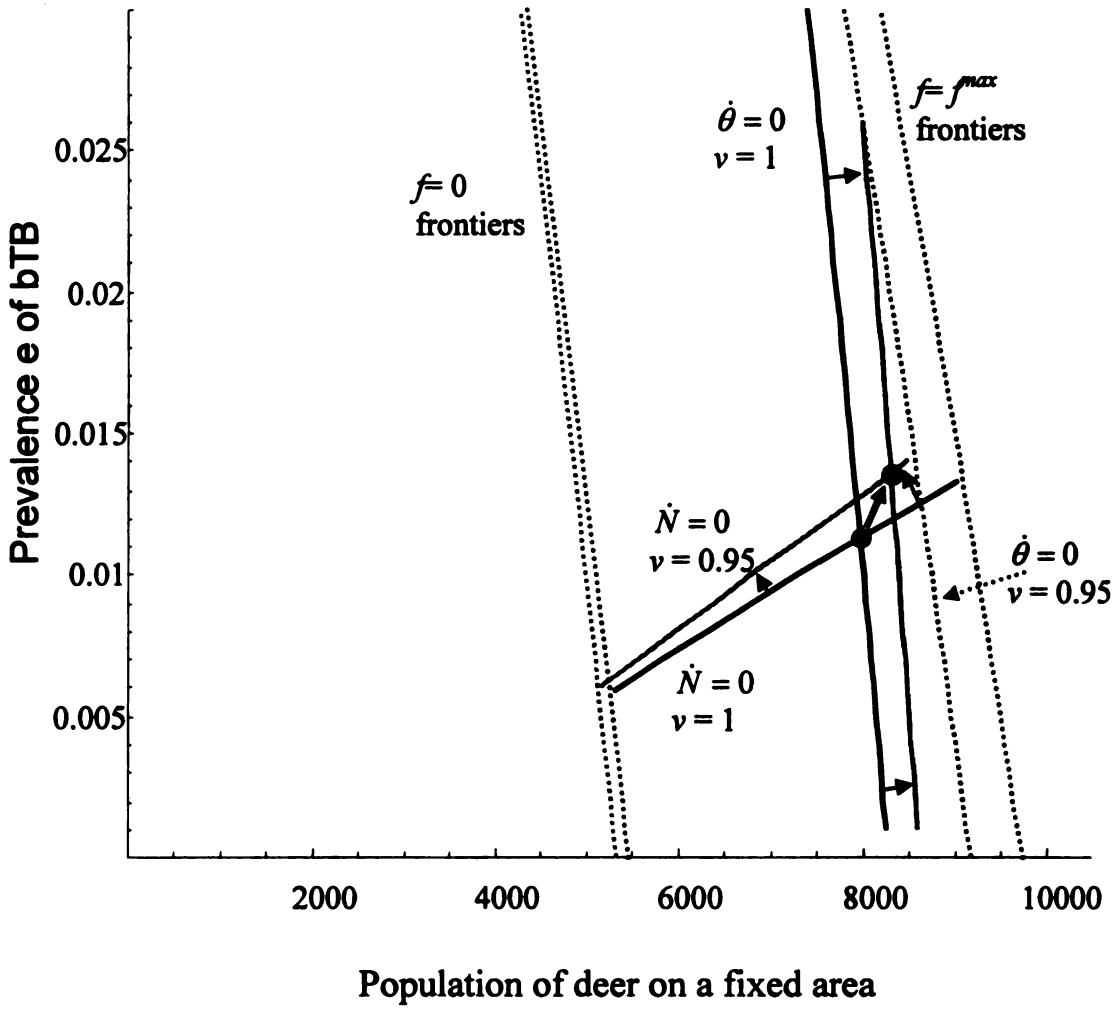

Figure 2.4. Phase plane showing how the $f = 0$ and $f = f^{max}$ frontiers, and the $\dot{N} = 0$ and the $\dot{\theta} = 0$ isocline change when ν is decreased from 1 (black) to 0.95 (gray). The phase dynamics are the same as those in figure 2.2. The solid single arrows indicate how the isoclines shift, and solid double arrow indicates how the focus point shifts with a decrease in ν.

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).

2.7. Discussion and Conclusion

Concern over wildlife disease continues 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, humans, and domesticated animals. Yet, there is surprisingly little research on the management of wildlife diseases, particularly on how changes to the environment influence opportunities for disease management. Indeed, much of the extant literature on wildlife disease management focuses on disease eradication via reducing population levels below some ecological host-density threshold, which is defined exogenously of human-environmental interactions. In this chapter, we have explored how human-environmental interactions affect ecological thresholds (specifically the interaction between supplemental feeding and host-density threshold for the persistence of bTB in Michigan white-tailed deer), and the implications for efficient management given that the economic and ecological systems are jointly determined. Specifically, we showed that addressing the problem efficiently requires management of both the deer population and the ecological threshold.

A model of bTB transmission in Michigan white-tailed deer with two control variables (a level of supplemental feeding and a level of harvests) was constructed. This model differed from the earlier work of Horan and Wolf (2005) by allowing for an ecological threshold that was defined by host density. Below this threshold disease prevalence declines naturally.

There are three main results that come from this work. First, the ecological threshold for an optimally managed disease system is endogenously determined. 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 chooses for feeding exist, and all of these lead to other thresholds, but such thresholds would be suboptimal and waste resources that could be used elsewhere more efficiently. It is also shown that economic thresholds interact with ecological thresholds so that society may benefit more from containing the disease by following a cyclical path rather than investing heavily in disease eradication.[22]

This leads to the second result; eradication may not be optimal. Economic and ecological tradeoffs 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., such as 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 ecological thresholds has two major drawbacks. First, if there are human-environmental actions affecting the disease

[22] Sharov and Liebhold's (1998) also find that containment of invasive species may be economically superior to eradication.

transmission process and these are not accounted for, the computed host-density threshold is likely to be wrong and will likely be a "moving target." Secondly, not considering economic feedback causes trade-offs to be evaluated elsewhere often in a less transparent way.

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 due to random mixing, and the relationship between individuals matters. Altering v may account for this, and the value v takes may impact the model in a qualitative way, altering the tradeoffs that a planner faces. This is true even if disease prediction models may be less sensitive to assumptions about v. Lower levels of pseudo-vertical transmission 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 the 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).

There is a clear need to move from a solely ecological understanding of wildlife disease to an interdisciplinary understanding of wildlife disease management. This chapter shows human-environmental interactions affect ecological thresholds, but the

economic drivers of these actions are also affected by the ecological system jointly determining efficient management. This chapter emphasis the need to consider both ecological thresholds and economic signals when developing wildlife disease management plans.

CHAPTER 3

SEXUAL DISCRIMINATION IN WILDLIFE DISEASE MANAGEMENT

3.1 Introduction

The results presented in Chapter 2 indicated that a management strategy that allows the disease to remain endemic might be socially optimal. This solution is conditioned upon that fact that both controls in the model – feeding and harvesting – were non-selective with respect to infected deer because infection is an unobservable trait. But what if a control could indirectly target infected deer by targeting an observable trait that is correlated with the probability of infection, i.e., disease risk factors? If so, this could reduce the costs of disease management, possibly making it optimal to eradicate the disease. The purpose of this chapter is to explore how increased targetablity of control might affect the socially optimal outcome.

Sex is the most basic and often observable difference arising in wildlife populations.[23] Sexual dimorphism allows for low cost identification of subpopulations by sex. This ability may allow wildlife disease managers to take advantage of physical, physiological, genetic, and behavioral differences between different subpopulations. In particular, these differences may lead to different levels of disease transmission and susceptibility between the sexes (Smith et al. 2001). In the case of white-tailed deer in Michigan, it has been suggested that males play a greater per capita role in transmission (O'Brien et al. 2002). This is emphasized by the current estimates of bTB prevalence in white-tailed deer, which are about eight percent in males and two percent in females

[23] Of course, not all animals may be distinguished by sex easily in the field. Some may be distinguished by age easily and other species may not be able to be divided into easily identified subpopulations at all.

(O'Brien et al. 2002). Targeting harvests on the basis of sex could increase the likelihood of reducing disease prevalence. Although harvest remains non-selective with respect to disease, harvest may be made selectively with respect to sex, which is clearly an important disease risk factor. This enhanced ability to selectively target a risk factor improves the manager's ability to manage the disease, and highlights the more general theme that improved management could result from incorporating greater biological realism into bioeconomic models (e.g., Bulte and van Kooten 1999; Brock and Xepapadeas 2002; Bulte and Damania 2003; Finnoff and Tschirhart 2003).

Big game managers traditionally establish differential hunting regulations based on animal sex, but their goals have focused mainly on the sustainability of wildlife populations for harvesting and recreation, and not disease control. Important economic tradeoffs emerge from a sex-based management approach when disease control becomes an additional objective. A specific facet of sex-based management is that males and females influence demographic change differently. Differentially harvesting males and females affects disease prevalence levels and the makeup of both the current stock and future harvests (Jensen 2000), the latter being important in part because males and females of many species are valued differently.

3.2 A Model of Infectious Disease Transmission

Consider a closed deer population, N, on a fixed land area. The aggregate deer population, when partitioned along two dimensions – health status and sex, consists of four sub-populations. The first dimension, health status with relation to bTB, divides the deer population into healthy (but susceptible) animals, S, and infected animals, I.

Assume that bTB is a chronic disease with no recovery and no immunity, so that the entire population can be classed as infected or susceptible, with all infected individuals also being infectious. This assumption may not perfectly fit bTB dynamics in wild deer, but it allows for mathematical simplification that improves tractability, and it is unlikely to qualitatively impact results. The second dimension, sex, is indexed by i and divides the deer population into male ($i=M$) and female ($i=F$) subpopulations. Denote the total male and female populations by $N_M = S_M + I_M$ and $N_F = S_F + I_F$, respectively.

Four processes affect the growth of each sub-population: (i) recruitment via births, (ii) natural mortality, (iii) harvests, and (iv) new infections. Infected populations are also affected by an additional component: mortality due to the disease. For aggregated population models, it is common to combine the birth and mortality processes into a single net growth or surplus production function – most often the logistic growth function $rN(1-N/k)$, where k is the carrying capacity and r is the intrinsic growth rate (e.g., Clark 1990). The intrinsic growth rate represents the maximum growth rate of the stock in the absence of competition for limited resources (i.e., food), and equals the birth rate, b, minus the natural mortality rate, δ. The term $(1-N/k)$ is the density-dependent component of net growth, which tempers the rate of growth in response to resource competition driven by the habitat's natural carrying capacity. We follow the convention of using the logistic model as a way of capturing the effects of density-dependent, compensatory growth. However, we separate the birth and mortality components because these will generally differ by sub-population.

Total births are given by the birth rate per female (the fecundity rate), *b*, multiplied by the number of females.[24] Fawns produced by healthy females will all be healthy, with a proportion, ϕ, being male. Fawns produced by infected females may or may not be infected. Denote v to be the proportion of fawns that are infected either *in utero* or after birth through maternal contact.[25] Given this specification, total births of healthy females are $S_F b(1-\phi) + I_F b(1-v)(1-\phi)$, total births of infected females are $I_F bv(1-\phi)$, total births of healthy males are $S_F b\phi + I_F b(1-v)\phi$, and total births of infected males are $I_F bv\phi$. Natural mortality is allowed to differ by sex, with the rate being defined by δ_i (i=*M,F*).

Net growth is determined by multiplying the difference between births and natural mortality by the density-dependent term (1-*N*/*k*). For instance, under natural environmental conditions the net growth of healthy females is given by $\left(S_F b(1-\phi) + I_F b(1-v)(1-\phi) - \delta_F S_F\right)(1-N/k)$, and the net growth of healthy males is defined analogously by $\left(S_F b\phi + I_F b(1-v)\phi - \delta_M S_M\right)(1-N/k)$. However, we make one final modification to the density-dependent term to reflect the fact that humans may alter environmental conditions. Specifically, hunt club-sponsored supplemental feeding programs have been used intensively to artificially raise the carrying capacity in the infected core area. Denote the effective carrying capacity "constraint" by $k/(1-\tau f)$, where f is supplemental feed and τ is a parameter. As $f \to 1/\tau$, the carrying capacity is

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[24] Assume that the male population is large enough to avoid an Allee effect, such that the number of males is not a constraint on the fecundity of females. This is a common assumption in populations modeling, especially for models of polygamous species such as deer (Casewell 2001 p. 570).

[25] When mothers transmit the disease to off-spring through contact after birth this is known as pseudo-vertical transmission. Bovine TB in white-tailed deer is not known to be transmitted *in utero*, but is suspected to be transmitted pseudo-vertically.

effectively eliminated so that deer grow at their maximum rates. Carrying capacity is a complex concept involving more than food. It is known that increasing available food relaxes the carrying capacity "constraint" therefore $\tau f > 0$, but we also know that food can not relax the carrying capacity "constraint" completely and therefore $\tau f < 1$. Given this modification, the net growth of healthy females becomes $(S_F b\phi + I_F b(1-v)\phi - \delta_F S_F)(1-(N/k)(1-\tau f))$. Net growth is analogously derived for the other sub-populations.

Harvests are assumed to reduce the stock after net growth has occurred. Harvests are selective with regard to sex, as the sex of an individual deer is observable, but harvests are non-selective with regard to health status.[26] Given non-selective harvesting, a manager can only choose the aggregate harvest for each sex class, h_i, with the harvest from each health class depending on the proportion of animals in that stock relative to the aggregate sub-population $N_i = S_i + I_i$. That is, harvests of healthy deer from sex class *i* are $h_{iS} = h_i S_i / N_i$, and harvests of infected deer from sex class *i* are $h_{iI} = h_i I_i / N_i$.

Disease transmission is assumed to alter a population in a similar fashion as harvesting – after density-dependent growth and mortality has occurred. Three types of contacts among deer can transmit disease, mother to offspring (pseudo-vertical transmission, described above), within-sex (male-male or female-female), and cross-sex (male-female or female-male). Transmission between adult animals is broken into two types because, under natural conditions, white-tailed deer segregate by sex and live apart

[26] Non-selectivity is not unique to the current situation. For instance, hunters and 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.

for most of the year, except for the rut (mating season) and yarding (congregation to keep warm during severe winters) (Sitar 1996; O'Brien et al. 2002).

For the within-sex and cross-sex cases we adopt the following transmission function that is based on the one proposed by McCallum et al. (2001) and employed by Roberts (1996) (also see Chapter 2)

(3.1) $(1-\varepsilon_{ij}+\varepsilon_{ij}N_i)(1+\omega f)\beta_{ij}S_iI_j/N_i \quad i,j\in(M,F)$

where β_{ij} is the contact rate per infectious deer, ε_{ij} and ω are parameters (with $i=j$ for within-sex transmission and $i\neq j$ for cross-sex transmission), and $N_i = N_i$ for within-sex transmission and $N_i = N$ for cross-sex transmission. Suppose there is no supplemental feeding, i.e., $f = 0$. If $\varepsilon_{ij} = 1$, then (3.1) is a mass action or density-dependent transmission function. That is, the contact rate is directly proportional to density (McCallum et al. 2001). If ε_{ij}=0, then (3.1) is a frequency-dependent or density-independent transmission function. Here, transmission depends on the proportion of infected individuals as opposed to total density. Values of ε_{ij} in the interval (0,1) imply that transmission dynamics lay somewhere on the spectrum between density-dependence and independence.

Disease transmission has traditionally been modeled with the density-dependent model, but McCallum et al. (2001) note this model may not hold up empirically. One important assumption of the density-dependent model is that the population mixes homogeneously, but by introducing sex we introduce heterogeneity into the population. Heterogeneity within the population could be one reason why some authors have argued that frequency-dependent transmission in certain situations has fit the data better for

diseases such as cowpox in bank voles and wood mice (Begon et al. 1998; Begon et al. 1999) and brucellosis in Yellowstone bison (Dobson and Meagher 1996). Moreover, it will be shown below that this heterogeneity, even under a frequency-dependence (if the two subpopulations are assumed to have different contact parameters or prevalence rates) can result is thresholds. Reality probably lies somewhere in between for most cases (Schauber and Woolf 2003). The major difference between the two extreme transmission functions, from a management perspective, is that reducing the aggregate wildlife population (via harvesting) does not affect prevalence under frequency dependency while it reduces prevalence under density dependency.[27]

Due to sexual segregation, the density-dependent assumption would probably only hold for within-sex transmission.[28] In contrast, frequency dependence is more likely for cross-sex transmission. Bovine TB is transmitted through close contact, and so, under natural conditions, cross-sex transmission is hypothesized to be limited to the breeding season for species that exhibit sexual segregation (Ramsey et al. 2002). Therefore, a model for sexually transmitted diseases may be more appropriate for cross-sex transmission, though the disease is not truly transmitted via sexual contact. The density-dependent transmission model is generally inadequate for modeling sexually transmitted disease because the number of sexual partners is not dependent on density (McCallum et al. 2001; Caley and Ramsey 2001). Rather, the number of sexual partners

[27] This is particularly important because for many diseases, such as bovine TB in wild deer, there are currently no effective vaccines (MDA 2002).

[28] Males form herds and intermix regularly making this assumption valid. Females have a complex social networks – forming family groups and these groups often have territories that do not overlap with other family groups (Oyer and Porter 2004). This makes true density-dependent transmission among females unlikely, but this is the best available way to model within female transmission. Models that have looked to integrate population dynamics and disease transmission have made similar generalizations (Haydon et al. 2002).

per animal is fixed (McCallum et al. 2001), so that sexually transmitted diseases depend on the proportion of infected individuals (McCallum 2000).

Supplemental feeding acts on the transmission rate, β, by attracting more animals into a smaller area, effectively increasing the density of the herd. In the case of density-dependent within-sex transmission, supplemental feeding effectively increases the transmission rate. This adjustment is required because populations in our model are defined in terms of the number of animals over a fixed area as opposed to local densities.

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

The equations of motion for infected males, infected females, susceptible males, and susceptible females respectively are determined by combining the components described above

$$\begin{aligned}\dot{I}_M &= (I_F b v\phi - \delta_M I_M)(1-(N/k)(1-\tau f)) - \alpha_m(1-\chi f)I_m + \\ &(1-\varepsilon_{MM}+\varepsilon_{MM}N_M)(1+\omega f)\beta_{MM}S_M I_M/N_M + \\ &(1-\varepsilon_{FM}+\varepsilon_{FM}N)(1+\omega f)\beta_{FM}S_M I_F/N - h_M I_M/N_M\end{aligned} \tag{3.2}$$

$$\begin{aligned}\dot{I}_F &= (I_F(1-\phi)vb - \delta_F I_F)(1-(N/k)(1-\tau f)) - \alpha_F(1-\chi f)I_F + \\ &(1-\varepsilon_{FF}+\varepsilon_{FF}N_F)(1+\omega f)\beta_{FF}S_F I_F/N_F + \\ &(1-\varepsilon_{MF}+\varepsilon_{MF}N)(1+\omega f)\beta_{MF}S_F I_M/N - h_F I_F/N_F\end{aligned} \tag{3.3}$$

$$\begin{aligned}\dot{S}_M &= (S_F b\phi + I_F b(1-v)\phi - \delta_M S_M)(1-(N/k)(1-\tau f)) - \\ &(1-\varepsilon_{MM}+\varepsilon_{MM}N_M)(1+\omega f)\beta_{MM}S_M I_M/N_M - \\ &(1-\varepsilon_{FM}+\varepsilon_{FM}N)(1+\omega f)\beta_{FM}S_M I_F/N - h_M S_M/N_M\end{aligned} \tag{3.4}$$

$$\dot{S}_F = \left(S_F b(1-\phi) + I_F(1-\phi)(1-v)b - \delta_F S_F\right)\left(1-(N/k)(1-\tau f)\right) -$$
$$(1-\varepsilon_{FF}+\varepsilon_{FF}N_F)(1+\omega f)\beta_{FF}S_F I_F/N_F - \tag{3.5}$$
$$(1-\varepsilon_{MF}+\varepsilon_{MF}N)(1+\omega f)\beta_{MF}S_F I_M/N - h_F S_F/N_F$$

It is more intuitive and mathematically convenient to work in terms of the variables N_i and θ_i instead of S_i and I_i, where θ_i is the infected proportion of sub-population of i. Substituting the relations $S_i = N_i - I_i$ and $\theta_i = I_i/N_i$ into the equations of motion, we can instead focus on the following equations of motion

$$\dot{\theta}_M = (\theta_F v - \theta_M)b\phi(N_F/N_M)\left(1-(N/k)(1-\tau f)\right) - \alpha_M(1-\chi f)\theta_M(1-\theta_M) +$$
$$(1-\varepsilon_{MM}+\varepsilon_{MM}N_M)(1+\omega f)\beta_{MM}(1-\theta_M)\theta_M + \tag{3.6}$$
$$(1-\varepsilon_{FM}+\varepsilon_{FM}N)(1+\omega f)\beta_{FM}(1-\theta_M)\theta_F N_F/N$$

$$\dot{\theta}_F = (1-\phi)b\theta_F(v-1)\left(1-(N/k)(1-\tau f)\right) - \alpha_F(1-\chi f)\theta_F(1-\theta_F) +$$
$$(1-\varepsilon_{FF}+\varepsilon_{FF}N_F)(1+\omega f)\beta_{FF}(1-\theta_F)\theta_F + \tag{3.7}$$
$$(1-\varepsilon_{MF}+\varepsilon_{MF}N)(1+\omega f)\beta_{MF}(1-\theta_F)\theta_M N_M/N$$

$$\dot{N}_M = (N_F\phi b - \delta_M N_M)\left(1-(N/k)(1-\tau f)\right) - \alpha_M(1-\chi f)\theta_M N_M - h_M \tag{3.8}$$

$$\dot{N}_F = (N_F(1-\phi)b - \delta_F N_F)\left(1-(N/k)(1-\tau f)\right) - \alpha_F(1-\chi f)\theta_F N_F - h_F \tag{3.9}$$

3.3 Ecological Thresholds

Models of disease transmission that include some degree of density-dependent transmission may be solved for a host-density threshold. As described in Chapter 2, this ecological threshold defines a critical host density below which disease prevalence declines and may ultimately dissipate naturally, leaving only a healthy population. The thresholds associated with this sex-specific model are more complex than the host-density threshold presented in Chapter 2, which depended only on feeding and prevalence (under certain circumstances this could be further simplified). In the sex-

specific model, the host-density threshold $\hat{N}_i$ is always conditional on the prevalence of both sexes and the population of the other sex, i.e.,

(3.10) $\hat{N}_i = \Phi(f, \theta_F, \theta_M, N_j)$

The dependence on state variables related to the other sex makes tradeoffs associated with holding populations below the threshold levels significantly more complex.

3.4 Economic Specification

The economic specification is similar to that of Horan and Wolf (2005) and Chapter 2, with a few important changes. Not all animals are equally valued. Hunters value male white-tailed deer more highly than female white-tailed deer (Wenders 1991; Loomis et al. 1989).[29] Larger average size, scarcity, and trophy value may be contributing factors to this difference in value. The value placed on sex *i* is denoted p_i. For all animals this is not less than the constant marginal utility from harvesting infected wildlife, p_I, i.e., $p_i \geq p_I$. For simplicity, we set $p_I = 0$ so that harvests of infected animals yield zero benefits. The total value of harvests are therefore $p_M h_M (1-\theta_M) + p_F h_F (1-\theta_F)$.

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 and independent of the targeted sex. Then total harvesting costs for sex *i*, restricted on the *in situ* stocks, are $(c/q_i)h_i/N_i$, where q_i is the catchablity coefficient. Supplemental feed is taken to have a constant per unit cost, *w*.

[29] For the purposes of the model all animals are assumed to be adults. An age structured model may provide further insight, but would make the analysis significantly more complex.

Finally, the costs of the disease, particularly to farmers and related agribusiness, must also be considered.[30] Denote the variable economic damages caused by infected deer by $D(\theta_F N_F + \theta_M N_M)$ where $D(0) = 0$, $D' > 0$, $D'' \geq 0$. These variable damages are due to infections in the cattle herd that result in lost stock, increased testing, and business interruption loss.[31]

3.5 Optimal Management

Wildlife managers have two objectives when dealing with disease: reduce the number of diseased animals and control the spread of the disease. To accomplish these goals, Michigan wild deer managers have focused on harvest levels and the amount of food provided by feeding programs as their primary choice variables (Hickling 2002). Given the discount rate, ρ, an economically optimal allocation of harvests and feeding solves

$$(3.11)\quad \underset{h_M, h_F, f}{Max}\ SNB = \int_0^{\infty} \left(\begin{array}{l} p_M(1-\theta)h_M + p_F(1-\theta)h_F \\ -\dfrac{ch_M}{q_M N_M} - \dfrac{ch_F}{q_F N_F} - wf - D(\bullet) \end{array} \right) e^{-\rho t} dt$$

[30] 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.

[31] The damage function is taken to be fixed and exogenous. Horan et al. (2004) develop a sexless model of deer and livestock, where damage to livestock is an endogenous function. In the present two-sex model, incorporating the livestock sector would add several more state variables to the model and unnecessarily complicate the analysis. In the conclusion, however, we do discuss some implications of endogenizing the damage function. Finally, the imposition of trade restrictions and Federally-mandated testing requirements in response to the disease may also result in a lump sum damage component. Such lump sum damages are primarily policy-induced and, if large enough, could affect the optimal plan. For simplicity, we focus on 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 (but see Horan and Wolf (2005) for an analysis of the impact of regulatory-based lump sum costs). If these costs were included, their only impact would be to shorten the time frame for disease eradication.

subject to the equations of motion (3.6)-(3.9).[32] The current value Hamiltonian is

$$(3.12)\quad \begin{aligned} H = {} & p_M(1-\theta_M)h_M + p_F(1-\theta_F)h_F - ch_M/(q_M N_M) - ch_F/(q_F N_F) - \\ & wf - D(\theta_F N_F + \theta_M N_M) + \lambda_M \dot{N}_M + \lambda_F \dot{N}_F + \mu_M \dot{\theta}_M + \mu_F \dot{\theta}_F \end{aligned}$$

where λ_i and μ_i are co-state variables associated with N_i and θ_i respectively.

The marginal impact of harvests of sex i on the Hamiltonian is

$$(3.13_i)\quad \partial H/\partial h_i = p_i(1-\theta_i) - c/q_i N_i - \lambda_i, \qquad i = M, F$$

If this expression is positive so that marginal rents exceed marginal user cost, then harvests for a given sex should be set at their maximum levels. Conversely no harvest should be undertaken if the expression is negative. The singular solution is pursued when this expression equals zero, so that marginal rents and the marginal user costs are equated. Conditions (3.13_i) may be singular for both sexes simultaneously, singular for one sex and not the other, or non-singular for both sexes.

The marginal impact of feeding on the Hamilton is a complex function shown here in implicit form

$$(3.14)\quad \partial H/\partial f = \Psi(N_F, N_M, \theta_F, \theta_M, \lambda_F, \lambda_M, \mu_F, \mu_M)$$

The equation for the marginal impacts of feeding includes the shadow values associated with all the state variables, unlike the marginal impacts of harvests. This is related to the fact that feeding does not target a single stock while harvesting does. Of course, if

[32] Following the convention set by Swallow (1990), Brock and Xepapadeas (2002), and others dealing with multi-state variable problems, problem (10) has been constructed as a linear control problem to simplify the exposition and analysis, and because the relation between feeding and ecological processes is not well-understood. We do, however, recognize that bounds must be placed on the linear processes. It is implicitly assumed that $f \leq \min(1/\chi, 1/\tau, 1/\omega)$. A value of $f > 1/\chi$ would result in a negative mortality rate due to the disease, which is not possible. A value of $f > 1/\tau$ or $f > 1/\omega$ would result in negative density dependence factors, which also does not seem realistic. In our numerical example these assumptions are explicit.

$\partial H/\partial f > 0$ then feeding should be set at the maximum level and if $\partial H/\partial f < 0$ feeding should not occur. When condition (3.14) vanishes, the singular solution is pursued.

The conditions for an interior optimal solution also involve four adjoint equations. These conditions prevent intertemporal arbitrage.

$$(3.15)\ \dot{\lambda}_M = \rho\lambda_M - \frac{ch_M}{q_M N_M^2} - \lambda_M \frac{\partial \dot{N}_M}{\partial N_M} - \lambda_F \frac{\partial \dot{N}_F}{\partial N_M} - \mu_M \frac{\partial \dot{\theta}_M}{\partial N_M} - \mu_F \frac{\partial \dot{\theta}_F}{\partial N_M}$$

$$(3.16)\ \dot{\lambda}_F = \rho\lambda_F - \frac{ch_F}{q_F N_F^2} - \lambda_M \frac{\partial \dot{N}_M}{\partial N_F} - \lambda_F \frac{\partial \dot{N}_F}{\partial N_F} - \mu_M \frac{\partial \dot{\theta}_M}{\partial N_F} - \mu_F \frac{\partial \dot{\theta}_F}{\partial N_F}$$

$$(3.17)\ \dot{\mu}_M = \rho\mu_M + w - \lambda_M \frac{\partial \dot{N}_M}{\partial \theta_M} - \lambda_F \frac{\partial \dot{N}_F}{\partial \theta_M} - \mu_M \frac{\partial \dot{\theta}_M}{\partial \theta_M} - \mu_F \frac{\partial \dot{\theta}_F}{\partial \theta_M}$$

$$(3.18)\ \dot{\mu}_F = \rho\mu_F + w - \lambda_F \frac{\partial \dot{N}_M}{\partial \theta_F} - \lambda_F \frac{\partial \dot{N}_F}{\partial \theta_F} - \mu_M \frac{\partial \dot{\theta}_M}{\partial \theta_F} - \mu_F \frac{\partial \dot{\theta}_F}{\partial \theta_F}$$

Following Chapter 2, these equation may be algebraically manipulated into a series of "golden rule" equations that must hold at each point in time.

$$(3.19)\ \rho = \frac{\dot{\lambda}_M}{\lambda_M} + \frac{\partial \dot{N}_M}{\partial N_M} + \frac{1}{\lambda_M}\left(\frac{ch_M}{q_M N_M^2} + \lambda_F \frac{\partial \dot{N}_F}{\partial N_M} + \mu_M \frac{\partial \dot{\theta}_M}{\partial N_M} + \mu_F \frac{\partial \dot{\theta}_F}{\partial N_M}\right)$$

$$(3.20)\ \rho = \frac{\dot{\lambda}_F}{\lambda_F} + \frac{\partial \dot{N}_F}{\partial N_F} + \frac{1}{\lambda_F}\left(\frac{ch_F}{q_F N_F^2} + \lambda_M \frac{\partial \dot{N}_M}{\partial N_F} + \mu_M \frac{\partial \dot{\theta}_M}{\partial N_F} + \mu_F \frac{\partial \dot{\theta}_F}{\partial N_F}\right)$$

$$(3.21)\ \rho = \frac{\dot{\mu}_M}{\mu_M} + \frac{\partial \dot{\theta}_M}{\partial \theta_M} + \frac{1}{\mu_M}\left(-w + \lambda_M \frac{\partial \dot{N}_M}{\partial \theta_M} + \lambda_F \frac{\partial \dot{N}_F}{\partial \theta_M} + \mu_F \frac{\partial \dot{\theta}_F}{\partial \theta_M}\right)$$

$$(3.22)\ \rho = \frac{\dot{\mu}_F}{\mu_F} + \frac{\partial \dot{\theta}_F}{\partial \theta_F} + \frac{1}{\mu_F}\left(-w + \lambda_M \frac{\partial \dot{N}_M}{\partial \theta_F} + \lambda_F \frac{\partial \dot{N}_F}{\partial \theta_F} + \mu_F \frac{\partial \dot{\theta}_M}{\partial \theta_F}\right)$$

As noted in Chapter 2, equations (3.19) and (3.20) may be interpreted as equating the rate of return from other opportunities elsewhere in the economy to the rate of return

from holding the resource *in situ*. Equations (3.21) and (3.22) are also "golden rule" equations, but these are associated with disease and can be interpreted as equating the opportunity cost of re-directing resources from elsewhere in the economy to manage disease with the rate of return from controlling disease .

The optimal solution to problem (3.11) can involve various combinations of singular and non-singular solutions for each of the three control variables. Following Chapter 2, a triple-singular solution is a fully unconstrained solution. This happens when (3.13_i) and (3.14) all vanish. Partial-singular solutions emerge when one or more (but not all) control variables are constrained. This is the case when any one or two of conditions (3.13_i) and (3.14) vanish. Finally, there is the potential for a fully constrained or "bang-bang" solution, when all control variables are constrained. There are a total of 27 potential combinations of controls (Table 3.1) and the analytical solutions defining these combinations are too complex to present here. However, the solution algorithm is essentially the same as that presented in Chapters 1 and 2.

For instance, for the triple-singular solution set conditions (3.13_i) and (3.14) all equal to zero. Conditions (3.13_i) enable the co-state variables λ_M and λ_F to be defined as functions of only state variables. These may be substituted into the "golden rule" equations (3.19) - (3.20). Take time derivates of λ_M and λ_F and substitute these on the LHS of conditions (3.19) and (3.20). Then, conditions (3.19) and (3.20), the two "golden rule" equations associated with the male and female deer populations, are functions of all the state variables, the remaining co-state variables (μ_i), and feeding. However, these golden rules are non-traditional in the sense that they involve co-state and control variables. It is possible to solve equations (3.19), (3.20), and (3.14)

Table 3.1. This table illustrates all 27 possible combinations of controls. Max and min represent constrained controls at their maximum and minimum levels respectively. Singular represents when a control may follow a singular solution (the first order condition associated with it vanishes).

Solution number	**Control**			**Solution Type (number of solutions in this category)**
	harvest females	*harvest males*	*feeding*	
1	singular	singular	singular	Triple-singular solution (1)
2	singular	singular	max	partial-singular solution (18)
3	max	singular	singular	
4	singular	max	singular	
5	singular	max	max	
6	max	singular	max	
7	max	max	singular	
8	singular	singular	min	
9	min	singular	singular	
10	singular	min	singular	
11	singular	min	min	
12	min	singular	min	
13	min	min	singular	
14	singular	min	max	
15	min	singular	max	
16	min	max	singular	
17	singular	max	min	
18	max	singular	min	
19	max	min	singular	
20	max	max	max	Full constrained, "bang-bang" solution (8)
21	min	max	max	
22	max	min	max	
23	max	max	min	
24	min	min	max	
25	min	max	min	
26	max	min	min	
27	min	min	min	

simultaneously for the remaining co-state a variable (μ_i), and feeding.

$$(3.23_i)\quad \mu_i = \Theta_i\left(N_i, \theta_i, f\left(N_i, \theta_i\right)\right)$$

Conditions (3.23_i) may be substituted into the "golden rule" equations associated with disease, conditions (2.21)-(2.22). Furthermore, time derivates of conditions (3.23_i) may also be substituted into conditions (2.21)-(2.22) for $\dot{\mu}_i$. This results is a system of two equations containing only state variables and h_i. These two equations maybe solved simultaneously for h_i as functions of the state variables – feedback rules so that harvests are a function of the state of system.

Therefore, the singular-solutions for feeding and harvests are non-linear feedback rules depending on the endogenous state variables. Horan and Wolf (2005) and the second chapter of this thesis also find non-linear feedback rules to linear control problems (see Bryson and Ho 1975 for more on non-linear feedback rules in resulting from linear control problems).

Again, the triple-singular solution is only one of the 27 possible solution types that may arise along an optimal path, since the solution procedure does not endogenously account for boundary constraints placed on the control variables, specifically control variables must satisfy the conditions that $h_i \geq 0, f \geq 0$, and $f \leq f^{max}$. Indeed, the overall solution may involve different types of feedback rules, and possible jumps in control variables, along a series of free and blocked intervals (discussed in Chapter 2). Analysis of when to pursue blocked or free intervals is inherently numeric (Arrow 1968).[33]

[33] Additionally, pursuing the feedback rules is only feasible when $\mu_i < 0$, implying that disease can never be beneficial to society. Even if all control variable constraints are satisfied the additional condition that $\mu_i < 0$ must also be satisfied, for the solution to be optimal.

In Chapter 2, it was possible to identify the space occupied by potential free and blocked intervals in a semi-analytical fashion. However, the sex-specific model is so complex that the solution must be evaluated entirely through the use of numerical methods. To understand the complexities that arise here, consider that Clark et al. (1979) also confronted a problem of analytical complexity in their well-known paper on irreversible investment and quasi-malleable capital. They use a combination of phase-plane analysis and analytical reasoning to evaluate a problem with two control variables. To do so involved identifying two singular-solutions resulting in a potential for a double-singular solution, four partial singular solutions, and four fully constrained solutions for a total of nine solutions to evaluate at any point in time, and the potential to switch between these solutions over time (just as in Chapter 2 of this thesis). However, due to dimensionality constraints, phase-plane analysis can not be used to determine blocked interval frontiers in the problem presented here. Furthermore, this problem has three control variables resulting in 27 combinations of triple-singular, partial-singular, and fully constrained solutions at any point in time, with the potential to switch between solutions over time. Clearly, this many solutions render analytical analysis intractable.

A discrete time approximation (over a 100-year planning horizon) was used to understand how the inclusion of sex may affect optimal management. The discrete version of the model is specified identically to the continuous time model, with the exception of the cost function. Following Conrad and Clark (1987), the Schaffer function was modified to a discrete form so that the cost function becomes $\ln[N_t/(N_t - h_i)]c_i/q_i$. All other equations remained the same. The constrained optimization package

in the software GAUSS was used to numerically solve for harvest and feeding strategies that maximized discounted social welfare.

3.6 Numerical Example

While we have made every effort to calibrate the model realistically, research on the Michigan bTB problem is still evolving at 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 true reflection of reality. Nonetheless, the results shed light on the economics of wildlife disease management in general and specifically on bTB in Michigan deer. Furthermore, this example allows us to make qualitative comparisons to previous work that treat the host population as homogeneous (Horan and Wolf (2005); second chapter of this thesis). The data used to parameterize the model are described in the Table 3.2.

Results are presented in Figures 3.1 – 3.3. Figure 3.1 illustrates the general dynamics of the state variables subject to the feedback rules in N-θ space. This is not a true phase plane (as a true phase plane for this problem would necessarily be four-dimensional) but allows for easy comparison to the results presented in Chapter 2. Notice that initially both male and female populations are drastically and rapidly reduced (also see Figure 3.2). This likely corresponds to moving the system along a MRAP into a feasible region (see Chapter 2). Intuition may lead to the conclusion that increased control through targeting by sex would lead to eradication of the male population, especially given our assumption about the lack of Allee effects. Indeed, if there were no stock effects in the cost function this would be the case as can be seen from condition

Table 3.2. Parameter values and descriptions. Methods of calculation are described in Appendix II.

Parameter	Description	Value
N_0	starting population	13298
	sex ratio (females to males)	3.035
b	*per capita* birth rate	1.22
δ_M & δ_F	*per capita* mortality rate by sex	0.3623
ϕ	sex ratio at birth	0.5
k	carrying capacity	14,049
τ	coefficient for feeding effect on k	8.0×10^{-5}
β_{FF} & β_{MF}	female-female and male-female transmission coefficient	3.23×10^{-5}
β_{MM} & β_{MF}	male-male and female-male transmission coefficient	9.45×10^{-5}
ω	coefficient for feeding effect on β	2.64×10^{-6}
ε_{FF} & ε_{MM}	own-sex contact coefficient	1
ε_{MF} & ε_{FM}	cross-sex contact coefficient	0
v	rate of pseudo-vertical transmission	1
α_F	disease induced mortality (females)	0.339
α_M	disease induced mortality (males)	0.339
χ	coefficient for feeding effect on α	5.00×10^{-5}
p_F	value of harvested healthy female	935.72
p_M	value of harvested healthy male	1534
c/q	marginal harvesting cost/ catachablity coefficient	231,192
w	Unit cost of feeding	36.53
D	marginal damages to livestock sector	5491
ρ	discount rate	0.05
f^{max}	maximum limit for feeding	10,000

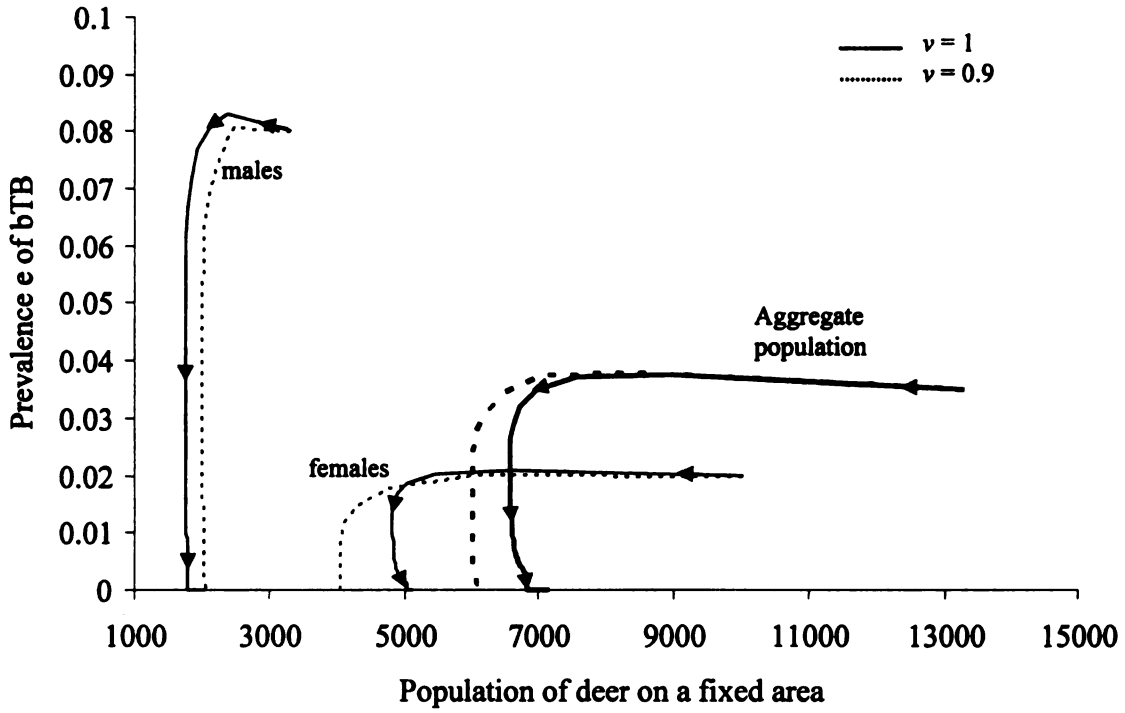

Figure 3.1. This figure illustrates the path resulting from following the optimal feedback rules in N-θ space by population and aggregated.

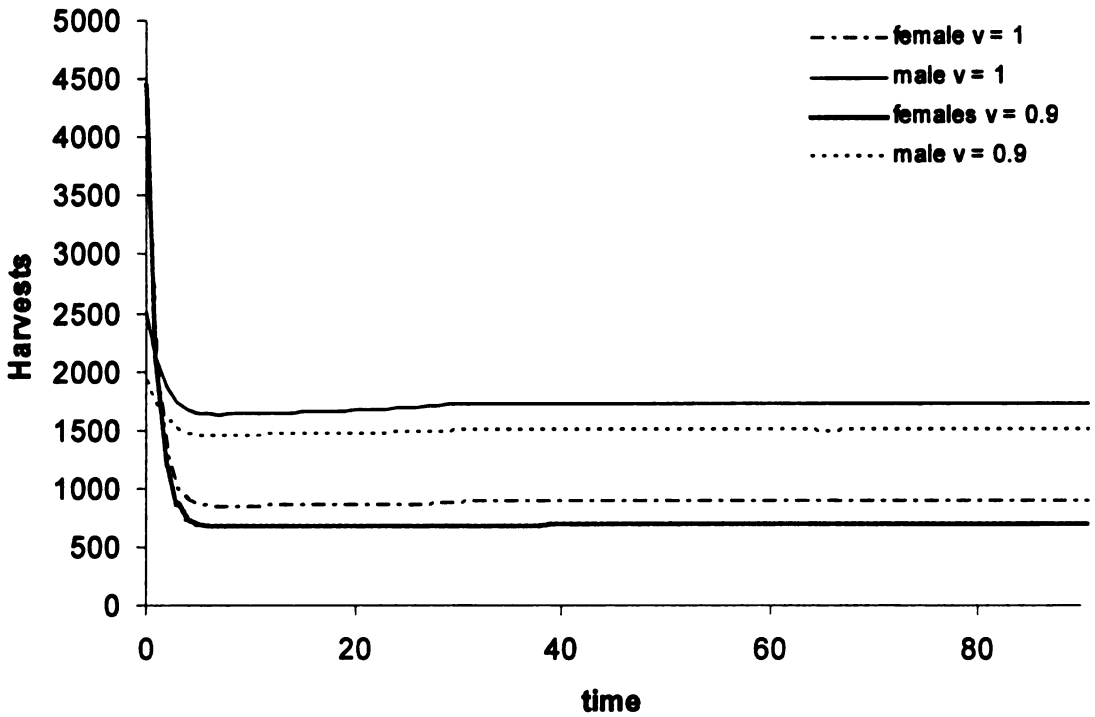

Figure 3.2. The optimal time path for harvests by sex under different assumptions about pseudo-vertical transmission.

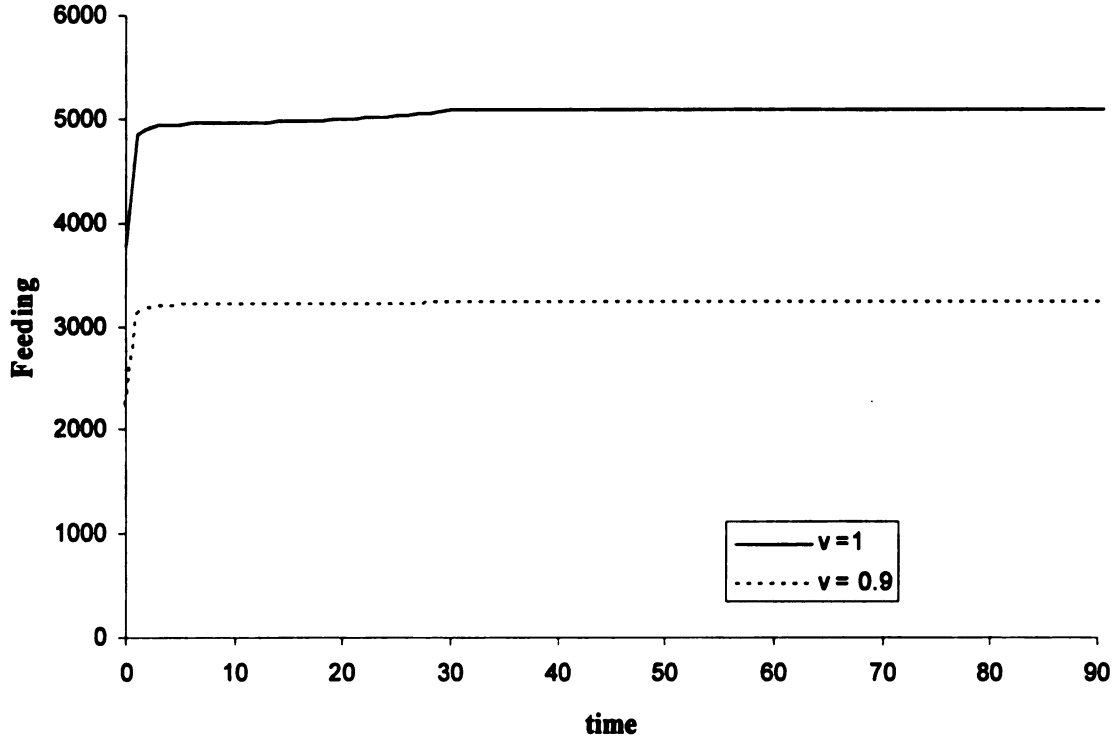

Figure 3.3. The optimal level of feeding under different assumptions about vertical transmission.

(3.15).[34] However, eradication of the male stock is infinitely costly given the specified cost function. The male population is therefore culled to just fewer than 1,800 individuals, and this level is essentially maintained until the disease dies out.

The female population is also culled initially, with the female population being reduced much more than the male population (in absolute terms). This may seem counter-intuitive since males appear to be the "risker" sex. However, reducing females is less costly given their larger initial numbers (even though few benefits are derived from female harvests). Moreover, reducing females creates direct and indirect disease prevalence-reducing benefits.

First, there is a direct own-sex benefit, whereby within-sex density-dependent horizontal transmission is reduced along with the reduction in female density. Second, a reduction in female prevalence creates direct inter-generational benefits associated with reduced pseudo-vertical transmission, which ultimately affects prevalence in both males and females. Third, there are direct cross-sex benefits, whereby cross-sex horizontal transmission is reduced along with the reduction in female density. This also creates an indirect benefit associated with the host-density threshold for males. The male host-density threshold is a function of the female population, and this threshold is increased

[34] With no stock effects, the second term on the RHS of (3.15) vanishes. It is known that μ_M and μ_F both must be negative as greater disease prevalence is never beneficial. We also know from (3.6)-(3.9) that $\partial \dot{N}_i / \partial N_M < 0$ and $\partial \dot{\theta}_i / \partial N_M > 0 \ \forall i$ (i.e., males do not affect fecundity, but they do compete for resources and create a larger pool for disease transmission). Assuming that $\lambda_M > 0$, then the right hand side (RHS) of (3.15) is always positive. This means that λ_M is always increasing. If λ_M were to grow without bound, then this would not be optimal: from (3.13_M) male harvests would eventually cease and the male population would grow with the effect of reducing *in situ* productivity and increasing disease transmission. If λ_M were to asymptotically approach a maximum value, then $\dot{\lambda}_M$ would approach zero. However, condition (3.15) could only hold in this case if $\lambda_M < 0$, which is a contradiction of our earlier assumption. So λ_M must be negative in each time period along an optimal path. If marginal rents of deer harvesting $(p_M(1-\theta_M) - c/q_M N_M)$ are positive or not too negative, then condition (3.13_M) implies that $\partial H / \partial h_M > 0$ so that h_M should be set at its maximum rate.

when there are fewer females. Therefore, reducing the number of females reduces the pressure to harvest males, as the disease can dissipate at a larger male population and at lower male harvesting costs.

Feeding levels are initially depressed to low levels ($f > 0$), but increase as disease prevalence declines. Feeding increases to an upper asymptote, approximately 5,000,

$f < f^{max}$ and is maintained at moderate levels (Figure 3.3). This is in contrast with the results in Chapter 2 where feeding levels decline throughout most of the prevalence-reduction stage of management. Improved targeting allows feeding to persist at moderate levels while disease prevalence is still declining increasing the likelihood of optimal disease eradication. Feeding is allowed to increase in the present case because it provides *in situ* productivity benefits, while damages associated with feeding may be managed more effectively through targeted population control.

3.7 Sensitivity Analysis

In the numerical analysis v was assumed to be one, implying that infected mothers have infected off-spring. On first principles this assumption seems logical since mothers likely have extensive close contract with their off-spring after birth. Blanchong (2003) reports that infected deer are more likely to be related to other infected deer then healthy deer. However, a number of authors consider pseudo-vertical transmission to play a minor part in bTB transmission (O'Brien 2002; Miller and Corso 1999). In the case of New Zealand possums, assumptions about v range from zero (Barlow 1996) to one (Roberts 1996). This variation may reflect confusion over difference between

modeling as relationship in a convenient way that approximates reality and a clinical definition of vertical transmission.

As in Chapter 2, a lower value of v ($v = 0.9$) is used to assess how assumptions about intergenerational transmission potentially affect optimal management. The dotted lines in Figures 3.1 – 3.4 show the changes that result from a decreased value of v. Figure 3.1 shows that a lower level of v results in fewer females but more valuable males being left in the population with the overall effect of a reduction in the total population.

The changes in the management paths are the result of two effects. First, a reduction in v increases the host-density threshold for females and males. However, an increase in the population of males decreases the host-density threshold of females (and visa-versa). Harvesting males is more costly due to small male population, so the increased threshold provides greater benefits to the management of males and allows for a larger population of males to be maintained. Second, because a larger population of males is maintained the female host-density threshold is lower and more females must be harvested (Figure 3.1). This may be interpreted as all or most of the benefits from lower pseudo-vertical transmission being allocated to manage the male population. Also notice that there is a smaller initial decrease in the male population and that it takes longer for disease to dissipate in the male population, but that the focus on females causes disease to dissipate faster in the female population (Figure 3.4).

Figure 3.3 indicates that the optimal level of feeding is lower when v is assumed to take smaller values. As v declines the benefits from sex-specific management also decline. This increases the role of feeding (or rather a reduction in feeding) in disease management.

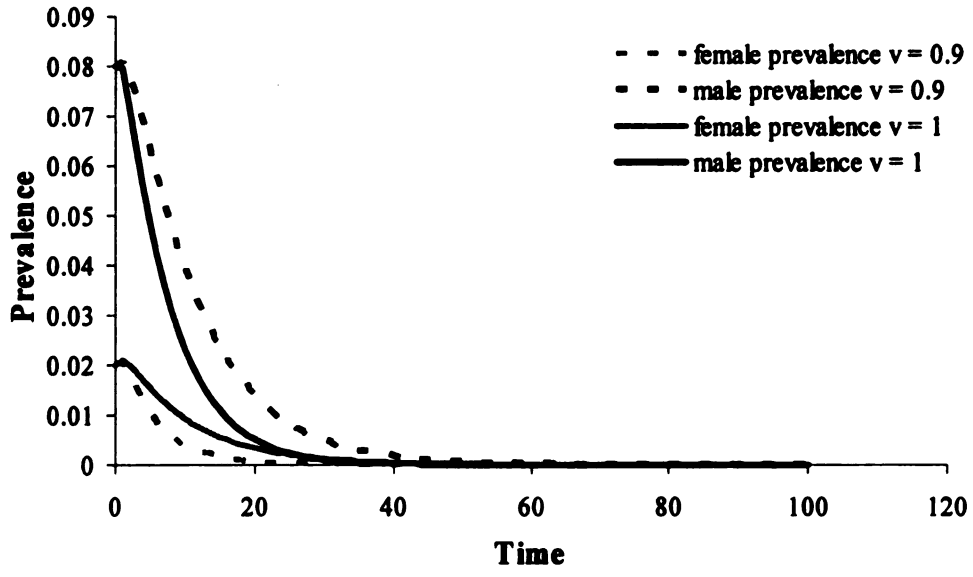

Figure 3.4. The time path of disease prevalence in males and females under differing assumptions about pseudo-vertical transmission.

Another area of interest is sensitivity to starting values. Chapter 2 showed that higher initial levels of prevalence may increase the chance that eradication will be optimal. Efforts were made to calibrate this model in a similar fashion to those in Chapter 2, however since parameters from various sources were used the aggregate prevalence rate in this model was slightly higher. When prevalence was scaled to a lower aggregate prevalence it was also optimal to eradicate disease.

3.8 Discussion and Conclusion

It can 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, as current approaches are rooted in those originally developed for livestock disease problems (Nishi et al. 2002). In this chapter, we build on earlier work to explore how the ability to target subpopulations affects tradeoffs in a jointly determined ecological and economic system.

The model presented in Chapter 2 is expanded into a sex-specific model that allows disease transmission to vary among and between male and female subpopulations. Furthermore, it was possible to discriminate between males and females so that harvesting effort is preferentially targeted.

By incorporating more ecological detail than previous models, we show how wildlife managers can target an observable risk factor to cost-effectively eradicate a wildlife disease. Indeed, prior work did not explicitly incorporated sex or other easily observed risk factors in analyses of wildlife disease control management plans.[35] Rather, the primary focus has been on lowering aggregate host densities. However, in this chapter we show that the host-density threshold can be significantly more complex when a population may be divided in subpopulations using demographic features such as sex. This creates additional tradeoffs and opportunities to manage an infected wildlife population for both wildlife benefits and disease control. These additional opportunities allow the population to be managed in a less costly manner than would be possible by just considering the aggregate population.

The results in this chapter may be compared to the results from the homogeneous population model in Chapter 2. In both cases, managers are faced with economic trade-offs to manage host density and the host-density threshold, at which the disease will dissipate naturally. However, these two models result in qualitatively different outcomes, specifically that when targeting is possible disease eradication is more likely to be optimal. This happens because the ability to target a specific risk-factor (sex) buffers the simple two-way trade-off between managing the aggregate host-density threshold and the aggregate host density. In the sex-specific model, a complex array of tradeoffs emerges and these may be exploited to make disease control and eradication

[35] Brooks and Lebreton (2001) incorporate age (stage which is observable as eggs, juveniles, and adults) in their management problem of yellow-legged herring gulls, *Larus cachinnans*, however they assume a stable population structure represents a steady state solution and the socially optimal stock of gulls. The gulls represent a nuisance but are protected so that there is a trade-off between harvesting and protecting the gulls. They do not carry a disease.

less costly. Moreover, disease prevalence is lowered faster and without forgoing as much *in situ* deer productivity.

CHAPTER 4

CONCLUSION

4.1 General Conclusions and Discussion

The emergence of new wildlife diseases threatens human and domestic animal health (particularly livestock), natural resource-based recreation, and conservation of biodiversity worldwide (Simonetti 1995), and managers have few options to control wildlife disease. If it were easy to identify and treat or remove infected wildlife, management would be straight forward. But this is not the case. Outwards signs of many wildlife diseases are rare and only appear in the final stages of infection (Williams et al. 2002; Lanfrachi et al. 2003).

Knowledge of wildlife disease management is limited. The ecological literature focuses on identifying a host-density threshold that can be used to guide population control approaches (such as harvesting, contraceptives, and vaccination – since vaccination eliminates animals form the susceptible population), as disease prevalence begins to decline for population densities below this threshold (e.g., Barlow 1991b; Roberts 1996; Smith and Cheeseman 2002). The host-density threshold in these studies is exogenously determined by ecological parameters, since population controls do not directly affect the disease transmission process. In addition to focusing on the host-density threshold, the ecological literature assumes eradication of the disease is an appropriate goal, and has ignored any resource allocation tradeoffs that this goal implies in world of scarce resources.

It is clear that the problem of managing wildlife disease is an economic problem subject to biological constraints. While others have identified the problem of wildlife disease management in this way (Bicknell et al. 1999; Horan and Wolf 2005), the first chapter of this thesis explicitly constructs a conceptual framework to identify interconnections and feedbacks among the ecological and economic systems. The potential for bioeconomic models to help identify economically efficient management solutions to wildlife disease problems is demonstrated. This framework also highlights the need for interdisciplinary cooperation in modeling wildlife disease management.

Harvest-based management options to control host density have limited effectiveness since harvests are non-selective with respect to disease status, and so it makes sense to explore whether other options may be of use. One option that has been proposed for use in conjunction with host-density control is environmental or habitat manipulation (Wobeser 2002). Indeed, it is clear that interactions between humans and wildlife habitat can influence disease in wildlife (Daszak 2001).

In Chapter 2, supplemental feeding is introduced as a human action that alters wildlife habitat, and the model is applied to the case of bovine tuberculosis in Michigan white-tailed deer. In order to investigate tradeoffs between environmental and host population controls, standard *SI* models were modified to include management actions other than harvesting. Horan and Wolf (2005) follow a similar procedure applied to the same case study, but they restrict their analysis to frequency-dependent transmission, thereby eliminating the possibility of an ecologically determined host-density

threshold.[36] When the opportunity set available to managers is expanded beyond population control to also include environmental control methods, it is shown that the host-density threshold is no longer determined only by exogenous ecological parameters, but also by endogenous human action. Therefore, incorporation of environmental controls makes the host-density threshold an endogenous economic-ecological threshold. Moreover, when this model is compared to the Horan and Wolf (2005) model, the inclusion of an ecological threshold effect allowed the disease to be managed at lower prevalence with a larger associated population.

On a more technical note, the non-selective nature of both control instruments (i.e., neither changes in supplemental feeding nor reduction of the overall population differentially target infected or susceptible animals) expands our knowledge of solutions to linear control models. A number of authors have noted that when control variables in a linear control model cannot be used to directly target the state variables (i.e., controls are imperfect), the solution may involve nonlinear feedback rules that may or may not require bang-bang controls (Mesterton-Gibbons 1996; Bhat and Bhatta 2004; Horan and Wolf 2005). Clark (1990) also noted that a lack of targetablity can lead to a phenomenon known as "chattering", which is a rapid switch between control rules. In this thesis, these observations are united by examining how non-linear feedback rules may interact with blocked intervals to create "chattering" between double-singular and partial-singular solution feedback rules. This happens along a blocked interval frontier. It is likely that such solutions will become increasingly common as more complex problems are analyzed that include multiple state variables that interact with a limited

[36] Upon further investigation it can be shown that when feeding is controlled in the Horan and Wolf (2005) model an economic-ecological threshold is created.

number of imperfectly-targeted control variables. This improved understanding of the nature of solutions to such problems will aid researchers investigating more realistic natural resource management problem, especially since imperfect controls are common in natural resource management (Reed 1980; Clark 1990).

The results in both Horan and Wolf (2005) and Chapter 2 indicate it may not be optimal to eradicate the disease due to high costs related to control and the opportunities that must be forgone (e.g., lost deer productivity) to eradicate the disease. These high costs are in part due to the non-selective nature of the control instruments. In Chapter 3, we investigate the possibility of improving the targeting of controls by identifying an observable risk factor for the disease that can be targeted. Targeting a risk factor could help to indirectly target diseased animals, reducing the nonselectivity of controls and also control costs. To explore this possibility sex was identified a targetable risk factor. The model developed in Chapter 2 was expanded into a sex-specific model that allowed disease transmission to vary between male and female subpopulations. Furthermore, it was assumed that hunters could discriminate between males and females, so that harvesting effort could be preferentially targeted.

The key result in Chapter 3 was that increased targetablity created a larger opportunity set for managers, leading to greater control over the host-density threshold and ultimately the potential for lower control costs and eradication as an optimal outcome. This result emerged from the combination of incorporating more biological realism and an increased ability to target the same control methods. This allows managers to recognize a more complex array of tradeoffs that arise from economic and ecological relationships not captured by simpler models.

4.2 Caveats and Directions for Future Research

Care should be taken in constructing policy recommendations based on the results of this thesis, for two reasons. First, it should be re-emphasized that, while the numerical analysis utilized the best available data for the Michigan bTB case, research on this system is still evolving at a fairly early stage and so knowledge of many parameters is somewhat limited. The analysis is therefore best viewed as a numerical example rather than a prescription for optimal management of the Michigan bTB situation.

Second, the analysis presented here is based on a social planner's model, and so the results provide insights into economic and ecological tradeoffs in an efficient or first-best setting. But the real world is decentralized and not managed efficiently, and so it would be inappropriate to simply apply individual results, such as the result that supplemental feeding should be maintained at positive levels, in a piecemeal fashion to existing problems where some economic distortions are likely to persist. Indeed, unintended consequences would be more than likely to emerge in such instances.

The next logical step would be to use the results of this thesis to aid in the construction of a second-best model of management. Such a model would incorporate the responses of individual hunters, farmers, and other relevant actors to incentives in a decentralized economy in the presence of many economic distortions (i.e., inefficiencies). Indeed, human choices are based on a large number of incentives, and not all of these are managed optimally. For example, from the above analysis it is clear feeding should be regulated, but perhaps enforcement is likely to be imperfect. In this more realistic case, a feeding ban may be preferable.

Yet the social planner perspective is valuable because it reminds us that there are real costs to acting suboptimally (even when a second-best solution is pursued). Moreover, the social planner's problems helps us identify costs and lost opportunities that we must accept so that policy makers can see that these are distributed in a fair or equitable way.

Another important caveat to the analyses presented in this thesis is that the damage functions in Chapters 2 and 3 are taken to be exogenous. A truly optimal plan would also consider on-farm biosecurity choices that could reduce the likelihood of infection and hence damages to the livestock sector. Horan et al. (2004) explore this problem and argue that the pressure to eradicate the disease may be lessened if risk associated within the livestock sector may be directly targeted through biosecurity. This results in the only remaining damages being incurred by deer hunters, who may be only minimally harmed when disease prevalence is low. Future integration of these two models, which will involve a large number of state and control variables, would allow managers to incorporate the targetablity of jointly-determined systems and endogenous risk.

It is also important to note that this model is not spatial and cannot account for nonrandom movements or clumped distributions, to do so would require a spatially explicit model. Moreover, the models presented here treat the wildlife population as "closed" and it is assumed that disease is not spreading. This assumption may be acceptable for the current Michigan bTB problem, but in other cases the spread of wildlife disease is of great concern (Fulford et al. 2002). Horan et al. (2005) have made a preliminary investigation of spatially optimal management of wildlife disease. Along

with spatial relationships, landownership likely plays an important role in the actual decisions agents are making. Moreover, developing effective social policies for private lands with regard to wildlife disease may be one of the largest barriers to controlling disease persistence and spread. The integration of these issues with the work in this thesis would be a major advance. Finally, the models presented here are deterministic, yet there is much uncertainty in human behavior and in disease dynamics. Stochastic modeling efforts could be undertaken in order to understand how uncertainty may change optimal management strategies, and to help analyze how much should be invested in disease monitoring and surveillance programs.

The basic nature of these models has another value; one of the key contributions of modeling can be to help identify new directions for empirical research. Some relationships may be important for understanding management opportunities, but may be less important for developing ecological predictive models. Such potentially important relationships (such as pseudo-vertical transmission) were identified in this thesis. Empirical exploration of these relationships is important to improve managers' ability to manage wildlife disease efficiently. For instance, it may be worth investing in a better understanding of the pseudo-vertical transmission process since pseudo-vertical transmission is shown to affect management outcomes. Indeed, pseudo-vertical transmission is often vaguely-defined and it may be a more complex process than presented here as a number of mechanisms of inter-generational transmission may be possible (Blanchong 2003). Further investigation into these processes is merited as well as the collection of data to allow model calibration.

Another area of interest relates to horizontal disease dynamics, which are unlikely to be constant and which may be affected by various types of management intervention. Future ecological research that would improve management models includes understanding baseline values of a contact parameter in the transmission function and the functional relationship between management actions and this parameter.

APPENDICES

APPENDIX A: CALIBRATION FOR THE ECOLOGICAL ECONOMICS THRESHOLD MODEL IN CHAPTER 2

Only three parameters differ from those used by Horan and Wolf (2005); for all parameters not described here consult their work.[37] The three parameters that differ are the transmission coefficient (β), the disease induced mortality rate, (α), and the coefficient for the feed effect on α, (χ). These parameters differ because Horan and Wolf calibrate their model based on an assumption of frequency-dependant transmission, while some degree of density-dependence is used here. The procedure to compute the transmission coefficient is the same as that in Horan and Wolf (2005), except that the contact term must be included to account for some degree of density dependence. Assuming that $\varepsilon = 0.75$, the transmission coefficient is derived to be $\beta = 3.39 \times 10^{-5}$. The total transmission term was set equal to the total mortality under an assumption of zero feeding and solved for α (these are the terms in the square brackets of equation 2.7). This value was then multiplied by 1.05 (also following Horan and Wolf) so that disease would not persist under a sustained no-feeding regime, resulting in $\alpha = 0.3556$. Following Horan and Wolf (2005), the values α were set equal to 0.2 to calibrate $\chi = 0.5 \times 10^{-5}$.

[37] Also see the appendix for associated with the sex explicit model as some parameters are described in more detail there, since there were greater changes in parameter values between that work and Horan and Wolf (2005).

APPENDIX B: CALIBRATION FOR THE SEX EXPLICIT MODEL IN CHAPTER 3

Parameters use to calibrate the model in Chapter 3 were obtained from a variety of sources. Though most of the calibration is similar to that in Horan and Wolf (2005) and the calibration of the sexless model presented in Chapter 2, more details are provided in this appendix because a greater number of parameters had to be adjusted. The initial number of deer in the core area (deer management unit [DMU] 452), N_0, was estimated to be 13,298 in the spring of 2002 (after the previous winter morality and prior to births) (Hill 2002). The sex ratio of deer in Alpena, Montmorency, and Presque Isle Counties (the area in and just north of the core) was estimated over two years and averaged to 3.035 (Sitar 1996). Given this sex ratio we compute a male population of, $N_{M0} = 3296$, and a female population, $N_{F0} = 10{,}002$. Core carrying capacity and feeding parameter estimates follow Horan and Wolf (2005) $k = 14{,}049$ for the 1561 km2 core area, $\tau = 0.00008$ (based on data in O'Brien et al. 2002 and Miller et al. 2003). Estimates of disease prevalence by sex were 2% for females and 8% for males, and are believed to have remained fairly constant over the last few years (O'Brien et. al 2002; McCarthy and Miller 1998).

Following Horan and Wolf (2005) the constant marginal value of a harvested deer was $p = \$1270.80$. The relative values reported by Loomis et al. (1987) were then used to compute values for males and females. These were \$1,534 for males and \$936 for females.

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. Based on Miller and Corso (1999) we find that $\beta_M(1+\omega f) = 0.672$ and $\beta_F(1+\omega f) = 0.1855$. The calibration of the parameter ω is identical to that in Horan and Wolf (2005) and is taken from Miller et al. (2003). Assume that $\varepsilon_{ii} = 1$ and $\varepsilon_{ij} = 0$ so that within sex transmission is density-dependent but cross-sex transmission is frequency-dependent (this would result in $0 < \varepsilon < 1$ for an aggregated population). Following a similar method to Horan and Wolf (2005), after accounting for the addition of the contact term and that some transmission is cross-sex and some within-sex, we can solve for $\beta_{MM} = \beta_{FM} = 9.454\text{x}10^{-5}$ and $\beta_{FF} = \beta_{MF} = 3.229\text{x}10^{-5}$ (the difference in between cross-sex and within-sex transmission is accounted for with the contact parameter ε).

The birth rate per female was taken to be 1.22 based on an average of the yearly birth rates reported by Sitar (1996). The sex ratio at birth was assumed to be 0.5. Mortality parameters less disease and less harvest were computed using survival estimates (Sitar 1996) and mortality due to hunting (McCarthy and Miller 1998) resulting in $\delta_i = 0.3623$. We also require the additional mortality rate due to the disease (α_i). It was assumed that $\alpha_M = \alpha_F$. The total transmission for the female population was set equal to mortality under an assumption of zero feeding and solved for α_F. This value was then multiplied by 1.05 so that disease would not persist under a sustained no-feeding regime, resulting in $\alpha_F = 0.339$. Following Horan and Wolf (2005), the value of α modified by feeding was set equal to 0.2 to solve for $\chi = 0.5 \times 10^{-5}$.

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