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The Role of Sexual Dimorphism in the Economics of Wildlife Disease Management

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Abstract

Infected wildlife cannot be selectively harvested for most diseases, complicating disease control. Targeting harvests by sex improves efficiency because disease transmission and prevalence usually vary by sex. We present a bioeconomic model of optimal deer and disease management that incorporates a two-sex wildlife model and sex-specific disease transmission and prevalence.

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The Role of Sexual Dimorphism in the Economics of Wildlife Disease Management

The spread of disease within and between wildlife populations is a major threat to agricultural livestock operations, human health, natural resource-based recreation industries, and conservation of biodiversity worldwide. However, relatively little research has focused on the economics of wildlife disease control and associated trade-offs. Most work has estimated the private costs to farmers and consumers under alternative control strategies, and these works have excluded costs and benefits directly associated with wildlife (e.g. Mahul and Gohin 1999; McInerney; Ebel, Hornbaker, and Nelson 1996; Dietrich, Amosson, and Crawford 1987; Liu 1979).

Important exceptions are the few studies focusing on bovine tuberculosis (TB) spread by Australian brushtailed possums to dairy herds and the incentives for farmers to engage in disease control on and off the farm (Bicknell, Wilen, and Howitt 1999; Barlow 1991). But there are two facets of this model that make it difficult to abstract to other wildlife disease problems. First, these analyses focus mostly on private incentives, but wildlife resources are typically managed by state or national agencies that address broader social goals. Second, the brushtailed possum does not yield significant *in situ* or *ex situ* benefits (perhaps a low-valued pelt) and is in fact an exotic pest; therefore it only has costs associated with its harvest (Barlow 1991). In contrast, healthy members of many infected wildlife populations (e.g., deer, lions, and migratory birds) may be highly-valued, which could imply large economic costs when healthy animals are lost to disease or disease control measures (e.g., a cull). Managers have traditionally favored harvest-based strategies for controlling or eliminating wildlife disease since wildlife vaccination strategies are often infeasible. Harvest strategies cannot selectively target infected

wildlife for most diseases because it is not possible to identify infected individuals until after they have been killed and examined (Williams et. al 2002). This results in a number of healthy and valuable animals being killed. Policies that encourage depopulation may undervalue healthy wildlife, and therefore undermine economic efficiency.

In a recent paper, we investigated optimal management of bovine TB in Michigan white-tailed deer using a linear control model that had two state variables. Non-selective harvesting and supplemental feeding, the two controls, were chosen because harvest lowers population densities, and disease transmission may be related to population density. Existing supplemental feeding programs create economic benefits by enhancing *in situ* deer productivity but have also been linked to increased disease transmission. The optimum in that model was an interior cycle in which the disease continues to persist in the wildlife population. The first part of each cycle involved a double singular solution (defined by a nonlinear feedback law along a two-dimensional singular arc) in which it was optimal to invest in deer productivity via feeding, although at the expense of greater disease prevalence. The second part of the cycle involved a conditionally singular solution where feeding was constrained to zero (and the singular solution for the wildlife stock is conditional on this constrained feeding choice), allowing disease prevalence to fall before it became too high. But the opportunity cost of waiting until prevalence went to zero exceeded the costs associated with a residual amount of disease, so at some point it became optimal to start the cycle again.

In that prior model, we assumed a homogeneous deer population and so harvesting was truly non-selective. However, wildlife populations are not homogeneous, and so an important question is whether disease transmission and susceptibility are

affected by an observable feature that could aid in targeting wildlife that are more likely to carry and/or spread the disease. If so, this could reduce the costs of disease management, making it optimal to eradicate the disease.

Sex is the most basic and often observable difference arising in wildlife populations. Sexual dimorphism can be used to manage wildlife disease because physical, physiological, genetic, and behavioral 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 might play a greater role in transmission (O'Brien et al. 2002). This is emphasized by the current estimates of bovine TB prevalence in white-tailed deer. In males, prevalence is estimated at about 8% and in females about 2% (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 becomes selective with respect to sex, an important risk factor. This enhanced ability to selectively target a risk factor improves the manager's ability to manage the disease.

Wildlife managers traditionally establish differential hunting regulations based on sex, but their goals have focused mainly on sustainable wildlife management and not disease control. Important economic tradeoffs emerge from a sex-based management approach when disease control becomes an additional objective. Males and females influence demographic change differently. Differentially harvesting males and females affects levels of disease prevalence, and the make up of both the current stock and future harvests (Jensen 2000) – and, apart from disease control is also important because males and females of many species are valued differently.

A model of infectious disease transmission

Consider a closed deer population, N , evolving 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 TB, divides the deer population into healthy (but susceptible) animals, s , and infected animals, z . TB 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 (Barlow 1991). The second dimension, sex, is indexed by i and divides the deer into males ($i=M$) and females ($i=F$). Denote the total male and female populations by $N_M = s_M + z_M$ and $N_F = s_F + z_F$, respectively.

Four processes affect the growth of each sub-population: (i) recruitment via births, (ii) natural mortality, (iii) harvests, and (iv) new infections. Sick 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 (e.g., 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.¹ Fawns produced by healthy females will all be healthy, with a proportion being male, ϕ . Fawns produced by infected females may or may not be infected. Denote ν to be the proportion of fawns that are infected either *in utero* or shortly after birth through contact.² Given this specification, total births of healthy females is $s_F b(1-\phi) + z_F b(1-\nu)(1-\phi)$, total births of infected females is $z_F b\nu(1-\phi)$, total births of healthy males is $s_F b\phi + z_F b(1-\nu)\phi$, and total births of infected males is $z_F b\nu\phi$. Natural mortality is allowed to differ by sex, with the rate being defined by d_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 $(s_F b(1-\phi) + z_F b(1-\nu)(1-\phi) - \delta_F s_F)(1-N/k)$, and the net growth of healthy males is defined analogously by $(s_F b\phi + z_F b(1-\nu)\phi - \delta_M s_M)(1-N/k)$. However, we make one final modification to the density-dependent term to reflect the fact that environmental conditions might not be natural. Specifically, hunt club-sponsored supplemental feeding programs have been used intensively to artificially raise the carrying capacity in the

¹ 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. It is assumed that even if harvesting all males is optimal due to the instantaneous nature of the model that there are still sufficient males (via new births) to avoid detriment to female fecundity. This is analogous to a time sequence where males mate, are then harvested, and females give birth to new males.

² 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 known to be transmitted pseudo-vertically.

infected core area.³ Denote the effective carrying capacity by $k/(1-tf)$, where f is supplemental feed and t is a parameter. As $f \rightarrow 1/\tau$, the carrying capacity is effectively eliminated so that deer grow at their maximum rates. Given this modification, the net growth of healthy females becomes $(s_F b f + z_F b(1-v)f - d_F s_F)(1 - (N/k)(1-tf))$. 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. This is because it is often difficult to identify which individuals are infected prior to the kill; outward signs of an illness often take a long period to manifest (MDA 2002; Williams et al. 2002). Harvesting will therefore include both healthy and infected individuals, which could be costly for species that are highly valued for recreational purposes (such as deer) or that are endangered.⁴

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 $N_i = s_i + z_i$. 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 $h_{iz} = h_i z_i / N_i$.

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

³ Carrying capacity is a complex concept that includes much more than just food. However because food is an important component of carrying capacity, altering food availability can alter carrying capacity.

⁴ Non-selectivity is not unique to the current situation. For instance, hunters and fishermen cannot

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 for most of the year, except for the rut (mating season) and yarding (congregation to keep warm during severe winters) (Kie and Bowyer 1999; Sitar 1996; O'Brien et.al 2002).

For the within-sex and cross-sex cases we adopt the following transmission function which is based on the one proposed by McCallum et al. (2001)

$$(1) \quad (1 - \mathbf{e}_{ij} + \mathbf{e}_{ij}N_l)(1 + \omega f) \mathbf{b}_{ij} x_i z_j / N_l \quad i, j \in (M, F)$$

where \mathbf{b}_{ij} is the contact rate per infectious deer, \mathbf{e}_{ij} and ω are parameters (with $i=j$ for within-sex transmission and $i \neq j$ for cross-sex transmission), and $N_l = N_i$ for within-sex transmission and $N_l = N$ for cross-sex transmission. Suppose for the moment that there is no supplemental feeding, i.e., $f=0$. If $\mathbf{e}_{ij}=1$, then (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 $\mathbf{e}_{ij}=0$, then (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 \mathbf{e}_{ij} within the unit interval imply something in between density-dependence and independence. Disease transmission has traditionally been modeled with the density-dependent model, but McCallum et al. (2001) note this model often does not hold up empirically. Frequency dependent transmission, on the other hand, often fits the data better for diseases such as

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.

cowpox in bank voles and wood mice (Begon et al. 1998, 1999) and brucellosis in Yellowstone bison (Dobson and Meagher 1996). Reality probably lies somewhere in between for most cases. 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.⁵

Due to sexual segregation, the density-dependent assumption would probably only hold for within-sex transmission.⁶ In contrast, frequency dependence is more likely for cross-sex transmission. 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, Barlow, and Hone 2001; Caley and Ramsey 2001). Rather, the number of sexual partners per animal is fixed (McCallum, Barlow, and Hone 2001), so that sexually transmitted diseases depend on the proportion of infected individuals (McCallum 2000).

Supplemental feeding can have an impact on disease transmission by attracting more animals into a smaller area, effectively increasing the density of the herd. In the

⁵ This is particularly important because for many diseases, such as bovine TB in wild deer, there are currently no effective vaccines (MDA 2002).

⁶ Males form herds and intermix regularly making this assumption valid. Females actually form family groups and have little interaction with individuals outside their family group, making true density-dependent transmission among females unlikely, but this is the best available way to model within female

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 as opposed to density.

In the case of cross-sex transmission, supplemental feeding programs can cause the social restriction of sexual segregation to break down (Garner 2001; Winterstein pers. com.). With the breakdown of this social regime, the transmission function shifts from frequency-dependence towards density-dependence.

The final component of population growth is mortality due to the disease, which obviously only affects infected sub-populations. Denote this mortality rate by \mathbf{a}_i . Supplemental feeding may decrease the effective mortality rate. Total mortality due to the disease is therefore specified as $\mathbf{a}_i(1-\mathbf{c}f)z_i$, where \mathbf{c} 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

$$(2) \quad \begin{aligned} \dot{z}_M = & (z_F b v \phi - \delta_M s_M)(1 - (N/k)(1 - \tau f)) - \alpha_m (1 - \chi f) z_m + \\ & (1 - \varepsilon_{MM} + \varepsilon_{MM} N_M)(1 + \omega f) \beta_{MM} s_M z_M / N_M + \\ & (1 - \varepsilon_{MF} + \varepsilon_{MF} N)(1 + \omega f) \beta_{MF} s_M z_F / N - h_M z_M / N_M \end{aligned}$$

$$(3) \quad \begin{aligned} \dot{z}_F = & (z_F (1 - \phi) v b - \delta_F z_F)(1 - (N/k)(1 - \tau f)) - \alpha_f (1 - \chi f) z_f + \\ & (1 - \varepsilon_{FF} + \varepsilon_{FF} N_F)(1 - \omega f) \beta_{FF} s_F z_F / N_F + \\ & (1 - \varepsilon_{MF} + \varepsilon_{MF} N)(1 - \omega f) \beta_{MF} s_F z_M / N - h_F z_F / N_F \end{aligned}$$

transmission. Models that have look to integrated population dynamics and disease transmission have made similar generalizations (Haydon, Laurenson, and Sillero-Zubiri 2002).

$$(4) \quad \begin{aligned} \dot{s}_M &= (s_F b \phi + z_F b (1 - \nu) \phi - \delta_M s_M) (1 - (N/k)(1 - \tau f)) - \\ & (1 - \varepsilon_{MM} + \varepsilon_{MM} N_M) (1 + \omega f) \beta_{MM} s_M z_M / N_M - \\ & (1 - \varepsilon_{MF} + \varepsilon_{MF} N) (1 + \omega f) \beta_{MF} s_M z_F / N - h_M s_M / N_M \end{aligned}$$

$$(5) \quad \begin{aligned} \dot{s}_F &= (s_F b (1 - \phi) + z_F (1 - \phi) (1 - \nu) b - \delta_F z_F) (1 - (N/k)(1 - \tau f)) - \\ & (1 - \varepsilon_{FF} + \varepsilon_{FF} N_F) (1 - \omega f) \beta_{FF} s_F z_F / N_F - \\ & (1 - \varepsilon_{MF} + \varepsilon_{MF} N) (1 - \omega f) \beta_{MF} s_F z_M / N - h_F s_F / N_F \end{aligned}$$

It is more intuitive and mathematically convenient to work in terms of the variables N_i and q_i instead of s_i and z_i , where q_i is the infected proportion of sub-population of i . Substituting the relations $z_i = q_i N_i$ and $s_i = N_i - z_i$ for s_i and z_i into the equations of motion, we can instead focus on the following equations of motion

$$(6) \quad \begin{aligned} \dot{\theta}_M &= (\theta_F \nu - \theta_M) \phi b (N_F / N_M) (1 - (N/k)(1 - \tau f)) - \alpha_M (1 - \chi f) \theta_M (1 - \theta_M) + \\ & (1 - \varepsilon_{MM} + \varepsilon_{MM} N_M) (1 + \omega f) \beta_{MM} (1 - \theta_M) \theta_M + \\ & (1 - \varepsilon_{MF} + \varepsilon_{MF} N) (1 + \omega f) \beta_{MF} (1 - \theta_M) \theta_F N_F / N \end{aligned}$$

$$(7) \quad \begin{aligned} \dot{\theta}_F &= (1 - \phi) b \theta_F (\nu - 1) (1 - (N/k)(1 - \tau f)) - \alpha_F (1 - \chi f) \theta_F (1 - \theta_F) + \\ & (1 - \varepsilon_{FF} + \varepsilon_{FF} N_F) (1 + \omega f) \beta_{FF} (1 - \theta_F) \theta_F + \\ & (1 - \varepsilon_{MF} + \varepsilon_{MF} N) (1 + \omega f) \beta_{MF} (1 - \theta_F) \theta_M N_M / N \end{aligned}$$

$$(8) \quad \dot{N}_M = (N_F \phi b - \delta_M N_M) (1 - (N/k)(1 - \tau f)) - \alpha_M (1 - \chi f) \theta_M N_M - h_M$$

$$(9) \quad \dot{N}_F = (N_F (1 - \phi) b - \delta_F N_F) (1 - (N/k)(1 - \tau f)) - \alpha_F (1 - \chi f) \theta_F N_F - h_F$$

Economic specification

The economic specification is similar to that of our previous work in this area, with a few important changes. Hunters gain utility from the actual process of shooting wildlife and/or consuming meat and other wildlife products, such as trophies. All animals are not equal in regard to their value. Hunters value male white-tailed deer more highly than female white-tailed deer (Wenders 1991; Loomis, Updike, and Unkel 1987). 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_z , i.e., $p_i \geq p_z$. For simplicity, and without loss, we set $p_z = 0$ so that harvests of infected animals yield zero benefits.

The total value of harvests are therefore $p_F h_F (1 - q_F) + p_M h_M (1 - q_M)$.

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 regardless 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 catchability coefficient. Supplemental feed is taken to have a constant per unit cost, w . Finally, the costs of the disease to farmers and related agribusiness must also be considered.⁷ Denote the economic damages caused by infected deer by $D(\theta_F N_F + \theta_M N_M)$ where $D(0)=0, D' > 0, D'' \leq 0$.

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

$$(10) \underset{h_M, h_F, f}{Max} SNB = \int_0^{\infty} [p_M (1 - \theta_M) h_M + p_F (1 - \theta_F) h_F - \frac{c h_M}{q_M N_M} - \frac{c h_F}{q_F N_F} - w f - D(\cdot)] e^{-\rho t} dt$$

subject to the equations of motion (6)-(9).⁸ The current value Hamiltonian is

⁷ The imposition of trade restrictions in response to the disease may result in a significant lump sum damage component, which if large enough could affect the optimal plan. Deer are also important causes of automobile accidents and damage to agricultural crops (Rondeau 2001; Rondeau and Conrad 2003). We ignore these other damages in order to focus on the impacts of disease, but we note that these other damages could be important.

⁸ It is implicitly assumed that $f \leq \min(1/c, 1/t, 1/w)$. A value of $f > 1/c$ would result in a negative mortality

$$(11) \quad H = p_M(1-\theta_M)h_M + p_F(1-\theta_F)h_F - ch_M/(q_M N_M) - ch_F/(q_F N_F) - wf - D(\theta_F N_F + \theta_M N_M) + \lambda_M \dot{N}_M + \lambda_F \dot{N}_F + \mu_M \dot{\theta}_M + \mu_F \dot{\theta}_F$$

where \mathbf{l}_i and \mathbf{m}_i are co-state variables associated with N_i and \mathbf{q}_i respectively.

The marginal impacts of harvest of males on the Hamiltonian is

$$(12) \quad \partial H / \partial h_M = p_M(1-\theta_M) - c/q_M N_M - \lambda_M$$

If this expression is positive so that marginal rents exceed marginal user cost, then harvests 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.

The following arbitrage condition for the male population is also a necessary condition that must be satisfied in each period

$$(13) \quad \dot{\lambda}_M = \rho \lambda_M - \frac{ch_M}{q_M N_M^2} - \lambda_M \frac{\partial \dot{N}_M}{\partial N_M} - \lambda_F \frac{\partial \dot{N}_F}{\partial N_M} - \mu_M \frac{\partial \dot{\theta}_M}{\partial N_M} - \mu_F \frac{\partial \dot{\theta}_F}{\partial N_M}$$

We know that \mathbf{m}_M and \mathbf{m}_F must both be negative as a larger disease prevalence is never beneficial. We also know from (6)-(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 they do create a larger pool for disease transmission). Assuming that $\lambda_M > 0$ and that $ch_M/q_M N_M^2$ is sufficiently small for the relevant range of values for the male population – which is the case in our numerical simulations below, then the right hand side (RHS) of (13) 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 (12) male harvests would eventually cease and the

rate due to the disease, which is not possible. A value of $f > 1/t$ or $f > 1/w$ would result in negative density dependence factors, which also does not seem realistic. In our numerical example these assumptions are

male population would grow with the effect of reduce reproduction and increasing disease transmission. If λ_M were to asymptotically approach a maximum value, then $\dot{\lambda}_M$ would approach zero. However, condition (13) 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 prevalence is not too large, so that the marginal rents of deer harvesting are positive, then condition (12) implies that h_M should be set at its maximum rate.

We assume all males are harvested in each period, only to be replenished by newly recruited males (see Clark (1990) for a similar analysis of pulse harvesting). Specifically, from equation (8) we have the male population in period $t+\zeta$ equaling period t male births

$$(14) \quad N_M(t+\zeta) = N_F(t)\phi b(1 - (N(t)/k)(1 - \tau f(t)))$$

As $\zeta \rightarrow 0$, we can solve for the instantaneous replenishment of males

$$(15) \quad N_M = \frac{\phi b N_F (1 - (N_F / k)(1 - \tau f))}{1 + (\phi b N_F / k)(1 - \tau f)}$$

Using a similar approach, we can solve for the prevalence rate of males in each period as a function of female prevalence

$$(16) \quad \theta_M = \nu \theta_F$$

Given (15) and (16), we can re-write the problem in terms of only two state variables: female population and female prevalence. Specifically, the social planner's problem would be to choose female harvests and supplemental feeding to maximize SNB

explicit.

subject to (7), (9), (15), and (16). Setting $h_M = N_M$ and using (15) and (16), the

Hamiltonian for this problem is

$$(17) \quad H = p_M(1 - v\mathbf{q}_F) \frac{\mathbf{f}bN_F(1 - (N_F/k)(1 - \mathbf{t}f))}{1 + (\mathbf{f}bN_F/k)(1 - \mathbf{t}f)} + p_F(1 - \mathbf{q}_F)h_F - c/(q_M) - \\ ch_F/(q_F N_F) - wf - D(\mathbf{q}_F N_F + v\mathbf{q}_F \frac{\mathbf{f}bN_F(1 - (N_F/k)(1 - \mathbf{t}f))}{1 + (\mathbf{f}bN_F/k)(1 - \mathbf{t}f)}) + \mathbf{l}_F \dot{N}_F + \mathbf{m}_F \dot{\mathbf{q}}_F$$

Note that the Hamiltonian is nonlinear in supplemental feeding, whereas the problem defined by (10) is linear in supplemental feeding and all other control variables. The reason is that the singular solution to (10) involves a nonlinear feedback rule for all control variables (see Bryson and Ho 1975 for more on nonlinear feedback rules in the context of singular solutions), as was also the case in Horan and Wolf (2003). The nonlinearity in supplemental feeding in (17) ensures a nonlinear solution for feeding will be optimal. As we show below, the singular solution for female harvests in (17) is also a nonlinear feedback rule.

The marginal impact of female harvests on the Hamiltonian is given by

$$(18) \quad \frac{\partial H}{\partial h_F} = p_F(1 - \theta_F) - \lambda_F$$

If this value is positive, then female harvests should be set at their maximum level. If the value is negative, then female harvests should be set at zero. If the value is zero, then the singular solution should be pursued.

The Kuhn-Tucker condition with respect to feeding is of the form:

$$(19) \quad \frac{\partial H}{\partial f} = \Gamma(N_F, \theta_F, f, \lambda_F, \mu_F) \leq 0; \quad \frac{\partial H}{\partial f} f = 0$$

In addition, the following arbitrage conditions are necessary

$$(20) \quad \dot{\lambda}_F = \rho\lambda_F - \partial H / \partial N_F$$

$$(21) \quad \dot{\mu}_F = \rho\mu_F - \partial H / \partial \theta_F$$

Assuming a singular solution, equations (19) and (20) can be solved for λ_F and μ_F . By taking the time derivative of (19) and substituting for $\dot{\mu}_F$, we get an expression that can be written in implicit form

$$(22) \quad \Gamma(N_F, \theta_F, h_F, f) = 0$$

Similarly, taking the time derivative of equation (18) and setting it equal to (20), we get the following implicit expression

$$(23) \quad \Phi(N_F, \theta_F, f) = 0$$

Equation (23) can be solved for the nonlinear feedback rule $f(N_F, \theta_F)$. Plugging this rule back into equation (22), we can solve for a feedback rule for female harvests, $h_F(N_F, \theta_F)$. These rules can then be plugged into the equations of motion (7) and (9) and used along with the initial state values to solve for the optimal path along the singular arc. Because the singular arc is two-dimensional, the entire (N_F, θ_F) plane – or at least a subset of it – satisfies the necessary conditions for the singular solution. Horan and Wolf (2003) find similar results for a sexless model of disease management in deer.

Numerical Example

We now examine the optimal solution numerically because the feedback rules and the differential equations that define the solution are too complex to analyze analytically. The data used to parameterize the model are described in the Appendix. While we have made every effort to calibrate the model realistically, research on the Michigan bovine TB problem is still evolving at a fairly early stage so knowledge of many parameters is somewhat limited. The following analysis is therefore best viewed as a numerical example rather than a true reflection of reality. Nonetheless, the results shed light on the

economics of wildlife disease management in general and specifically on TB in Michigan deer.

The numerical solution is presented in Figure 1 for the case of $\rho=0.1$. Given N_{F0} and θ_{F0} , represented by point a in Figure 1, the singular path 1 is followed. This path moves northwest, increasing θ_F while decreasing N_F . θ_F is increased because supplemental feeding is increasing along this path. Feeding represents an investment in deer productivity and generates near term gains that outweigh the costs of increased prevalence. The result that feeding should be initially encouraged runs contrary to Michigan's current policy approach of banning feeding.

Feeding and also prevalence rates continue to grow along the path 1 . Eventually a path that supports both feeding and harvesting is no longer optimal as the necessary conditions could no longer be satisfied. It turns out that feeding must optimally be set at a constrained value of $f=0$: although feeding is increasing along path 1 , μ_F would actually become positive if we were to set feeding equal to some maximum value. A jump in the female deer stock is required to move to the constrained $f=0$ solution (path 2), with the jump occurring at a point such as b . The singular solution for female harvests resumes at point c , and path 3 is followed. Nowhere along this path does it become optimal to move back to an unconstrained path. Rather, path 3 is followed until the disease finally dies out, with approximately 250 females remaining. Once the disease dies out, feeding is set at its maximum value and the system moves along a most rapid approach path to a steady state of $N_M = 1,611$ and $N_F = 31,700$.

Conclusion

The results of the two-sex model differ significantly from models that only consider an

aggregate (sexless) population. Qualitatively, each starts out with extensive feeding to boost productivity, and each eventually moves via a cull in the population to a path of no feeding and population recovery. At this point the qualitative results between the two types of models differ. The two-sex model leads to eradication of the disease. In contrast, the sexless model moves to a cycle of feeding (which increases the disease prevalence) and culling combined with no-feeding (which reduces prevalence), with the disease remaining endemic. The disease remains endemic because the opportunity cost of forgoing productivity investments via supplemental feeding becomes too great, so that it is too costly to wait for the disease to die out.

The results of both models are largely driven by the fact that wildlife managers cannot selectively harvest infectious deer. Any offtake of sick deer will be accompanied by healthy deer, which could be costly – particularly if prevalence is low. But in the two-sex model, managers have the ability to target an important observable risk factor – male deer, which tend to have higher disease transmission rates and prevalence rates than females. By targeting this risk factor, the cost of waiting for the disease to die out is sufficiently reduced so that eradication becomes optimal.

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Appendix

Parameters use to calibrate the model were obtained from a variety of sources. The initial number of deer in the core area (deer management unit [DMU] 452), N_0 , was estimate to be 13,298 in the spring of 2002 (after the previous winter morality and prior to births) (Hill 2002). A sex ratio of deer in Alpena, Montmorency, and Presque Isle Counties

(area in and just north of the core) was estimate over two years and averaged to 3.035 (Sitar 1996). This indicates 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 (2003) $k=14,049$ for the 1561 km² core area, $\tau=0.00008$. Estimates of disease prevalence by sex are 2% for females and 8% for males, and believed to have remained fairly constant over the last few years (O'Brien et. al 2002; McCarthy and Miller 1998).

Following Horan and Wolf (2003) we use a price per harvested deer where $p=\$1270.80$, scaled harvesting costs, $c/q = \$231,192$, and a fixed feed price of $w= \$36.53$. The relative values reported by Loomis, Updike, and Unkel (1987) were then used to compute values for males and females. These were \$1,534 for males and \$936 for female. Finally, total damages are estimated to be \$12 million per year at current stocks and infection rates (Wolf and Ferris 2000). Using a linear damage function implies a damage coefficient of 5491.

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 $\beta_M(1+uf) = 0.672$ and $\beta_F(1+uf) = 0.1855$. Miller et al. (2003) results are used to calibrate u . Following the same procedure used in Horan and Wolf (2003), we can solve for $\beta_M = 0.6577$ and $\beta_F = 0.1816$. In our analysis, we set the maximum value of f equal to $f^{\max} = 10,000$. This choice is somewhat arbitrary but it has little bearing on our qualitative graphical results. The values calculate for β_m and β_f however are not the true $\beta_{MM}, \beta_{FF}, \beta_{MF}$, and β_{FM} . The above estimates are therefore taken to be the sums of with-in sex and between sex coefficients for sex i . To calculate the β_{MF}, β_{MM} multiply by the proportion of the year over which the rut takes place (taken to be 0.16667 or seven weeks) and then by the proportion of the population that is female. The resulting coefficient is 0.0735. A new β_{FF} was calculate by subtracting this number of β_F so that $\beta_{FF} = 0.1081$. A similar process was follow for males resulting in $\beta_{MM} = 0.6477$, and $\beta_{FM} = 0.0100$. We assume that infected females have significant close contact with their fawns and therefore assume a vertical transmission, v , rate of one.

The birth rate 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 taken to be 0.5. Mortality parameters less disease and less harvest were computed using Sitar's (1996) survival estimates and McCarthy and Miller's (1998) mortality due to hunting estimates and were estimated to be 0.3623.⁹ But we also require the additional mortality rate due to the disease (α). In our model we assume that $\alpha_m = \alpha_f$ while this assumption may not hold in nature it is a good starting point. Hill (2002) estimates that 1,340 deer out of an initial population of 20,418, or 6.56 percent, died from reasons other than legal hunting mortality in 2001. We need an estimate of natural mortality outside the core area to enable us to separate out the effects of natural and disease-based mortality. Hill's (2002) outside core estimates vary considerably depending on the amount of snowfall received by various areas. Medium snowfall areas outside the core imply a natural mortality rate of only 5.6 percent. If we take this value to be the natural mortality rate for healthy deer inside the core, then we would expect 1117 natural deaths among 19,948 healthy deer, leaving 223 deaths for the remaining 470 infected deer. Some of these deaths were likely due to illegal hunting and other reasons unrelated to the disease. We therefore adopt an effective mortality of $\alpha(1-\chi_f) = 0.2$, which would account for slightly less than half of these other deaths. This rate does not imply that 20 percent of all infected deer die as a direct result of the disease, as few deer actually die from tuberculosis. Rather, the deer are weakened by their infection and ultimately die from something else. One more piece of information is still required to calibrate α and χ . The unsustainable nature of the disease outside the core suggests that $\alpha > \beta_{FF}$. The disease does not have to be sustainable in both does and bucks for it to be sustained in the population. By assuming that it is only sustainable in a doe population we estimate the minimum possible value for α . We have chosen $\alpha/\beta = 1.05$ because this value produces reasonable results relative to historical changes in disease prevalence when recent deer populations, disease prevalence rates, harvests, and feeding choices are plugged into the model. There for $\alpha_m = \alpha_f = 0.1907$, and $\chi = 0.00025$.

⁹ Harvest mortality is implicitly assumed to be additive, this is only relevant if the optimal level of harvest is 100% compensatory and thus there is additional unaccounted for mortality.

Figure 1. Optimal Management Path

