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# Disease and Behavioral Dynamics for Brucellosis Control in Elk and Cattle in the Greater Yellowstone Area

**Fang Xie and Richard D. Horan**

This paper investigates private responses and ecological impacts of policies proposed to confront the problem of brucellosis being spread from elk to cattle in Wyoming. The policies consist of combinations of changes in elk feeding and population levels. Farmers' responses to these dynamics are modeled along with the associated impacts to livestock population dynamics. Our findings suggest that feedbacks between jointly determined disease dynamics and decentralized economic behavior matter, and the elk feedgrounds do not actually generate economic harm to the individual farmers.

*Key words:* bioeconomics, brucellosis, disease ecology, epidemiology, replicator dynamics, susceptible-infected-recovered (SIR) model, wildlife disease, wildlife feeding

## Introduction

As human populations expand and natural habitats shrink, conflicts have arisen between people and wildlife. For instance, baboons in Namibia have attacked young cattle (Butler, 2000); elephants have destroyed crops and injured people in Kenya and elsewhere in Africa and Asia (Nyhus et al., 2003); bears, wolves, and other predators have killed livestock around the world, and are becoming a particular problem in developed areas such as Europe (Treves and Karanth, 2003); and deer, elk, and other species have spread diseases to livestock in all parts of the world. The costs of human-wildlife conflicts are sometimes substantial. Wildlife, many of which are already threatened or endangered, are often killed to solve current conflicts and to prevent future ones (Butler, 2000; Nyhus et al., 2003).

An economic literature examines public and private incentives to mediate some human-wildlife conflicts. Most studies examine the socially efficient management of wildlife stocks that cause external damages, such as crop damages (Zivin, Hueth, and Zilberman, 2000; Rondeau, 2001; Horan and Bulte, 2004), auto accidents (Rondeau and Conrad, 2003), and disease transmission to livestock (Bicknell, Wilen and Howitt, 1999;

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Horan and Wolf, 2005; Fenichel and Horan, 2007a,b).[1] The few studies that have examined private incentives for managing human-wildlife conflicts investigate private responses to various (suboptimal) policy mechanisms, such as trade bans on wildlife products (Horan and Bulte, 2004) and compensation schemes for wildlife damages (Rondeau and Bulte, 2007), and show how such policies can reduce welfare and hurt wildlife populations.

One type of human-wildlife conflict that is growing in importance and has received only limited attention from both the ecology and economic literatures is wildlife-transmitted diseases affecting livestock. Indeed, risks posed by infectious diseases are significant and escalating (World Resources Institute, 2005), and pathogen introductions may achieve a status similar to invasive species, the second most important cause of extinction (Daszak, Cunningham, and Hyatt, 2000). Moreover, wildlife populations increasingly serve as disease reservoirs for encroaching human or livestock populations (Daszak Cunningham, and Hyatt; McCallum and Dobson, 2002; Simonetti, 1995).

Where this problem has been studied, the focus in disease ecology has been on disease dynamics in the absence of human impacts (e.g., McCallum and Dobson, 2002), while the focus in economics has been on optimal disease control (e.g., Horan et al., 2008). There has been some limited research on the private incentives for management when the wildlife disease reservoir affects a single farmer (Bicknell, Wilen, and Howitt, 1999), in which case the farmer is the sole recipient of the externality and has the same incentives as a social planner. But no one has considered farmers' private incentives to respond to disease risks, and the associated ecological feedbacks in a bioeconomic-epidemiological framework, when a wildlife disease reservoir affects a number of farms over the landscape.

Herein we develop a bioeconomic model to investigate private responses to and the ecological impacts of policies proposed to confront the problem of brucellosis (*Brucella abortus*) being spread from elk to cattle in Wyoming. This human-wildlife conflict likely emerged from management directed at an earlier human-wildlife conflict (Dean et al., 2004). Wyoming elk had been eating cattle forage during winter, causing damage to farmers. Instead of farmers fencing off their land, the public sector set up elk feedgrounds across the state. This has done two things. First, it reduced elk predation on cattle forage. Second, it increased elk densities, which has led to the emergence of brucellosis in elk and has allowed it to become endemic. The elk have since spread the disease back to cattle.

Formal modeling and analysis of the bucellosis problem has been limited. Dobson and Meagher (1996) propose a simple susceptible-infected-resistant (SIR) epidemiological model to describe the population and disease dynamics of brucellosis among bison and elk in the Greater Yellowstone Area (GYA). They found the host-density threshold for brucellosis establishment—the population density below which disease prevalence will decline—is quite low. This makes it difficult to eradicate brucellosis from wild elk populations. However, their model did not consider the role of human management choices, nor did it examine interactions between wildlife and livestock.

We propose a joint model of wildlife and livestock population and disease dynamics, and behavioral dynamics, to gain insight into the challenges of managing brucellosis

[1] Zivin, Hueth, and Zilberman (2000) and Bicknell, Wilen, and Howitt (1999) examine private incentives when the externality impacts a single farmer. In this case the public and private incentives would be equivalent.

infection between livestock and the Jackson elk herd in Wyoming. The Jackson herd is one of the largest in Wyoming, and its range includes the largest feedground in the state—the National Elk Refuge (Dean et al., 2004). Unlike much prior literature which generally either treats behavioral variables as fixed parameters or has focused on economic choices and treated disease parameters as fixed, we integrate disease dynamics with economic choices so that infection risks depend on livestock disease management choices, and economic choices, in turn, depend on infection risks.

We assess population and disease dynamics under several management options for the Jackson elk herd, where each option involves a combination of changes in elk feeding and population levels. Farmer responses to these dynamics, when vaccination is not required, are modeled along with the associated impacts to livestock dynamics.[2] We also examine livestock management when there is little to no consideration given to the risk posed by elk. In practice, policies and proposals to address elk have been considered separately from farmer responses, with many livestock advocacy groups simply pushing to eliminate the feeding grounds (Smith, 2001).

Our findings suggest the feedbacks between jointly determined disease dynamics and decentralized economic behavior matter when choosing among various policy approaches. In particular, closing elk feedgrounds, which has been advocated by farm groups as a way to reduce livestock infection risks, may only marginally reduce cattle infections. This is because closing the feedgrounds reduces infectious contacts among elk, impacting both on new infections and the number of elk that have gained resistance to the disease. The net effect is a decrease in resistant elk and an increase in susceptible elk, which actually results in more new infectious contacts and more infectious elk. This increases livestock risks. Farmers respond with greater vaccination rates, with the net effect being only a small reduction in the number of infected farms. Hence, elk management policies chosen to reduce disease risks to livestock may lead to both ecological and farmer responses that largely offset these changes in risk.

## Background

Brucellosis is a bacterial disease that causes cattle and elk to abort their calves. It is transmitted through sexual contact and direct contact with infected birthing materials, and it is one of the most infectious bacterial agents in cattle [Wyoming Brucellosis Coordination Team (WBCT), 2005]. Brucellosis has caused devastating losses to U.S. farmers over the last century. The USDA and animal industry embarked on a plan to eradicate brucellosis in the United States in the 1930s, and this effort required 70 years and an estimated $3.5 billion in state, federal, and private funds (WBCT). The only known focus of *Brucella abortus* infection remaining in the nation is in the GYA.

Wyoming is at special risk due to the large reservoir of brucellosis in elk. Elk on winter feedgrounds in the GYA have an average serological (blood serum) prevalence of exposure of 30% (WBCT, 2005). Currently, the Game and Fish Department in Wyoming manages 22 state-operated elk feedgrounds. In addition, the National Elk

[2] Though Wyoming has recently regained brucellosis-free status, vaccination is still required. We ignore this regulation and instead model vaccination as a choice. This allows us to illustrate how bioeconomic models can be used to model private responses to disease risks and the associated epidemiological feedbacks to gain insights that can prove useful for management—not just in Wyoming but for the growing disease threats worldwide.

Refuge is managed by the U.S. Fish and Wildlife Services. These feedgrounds are largely concentrated in the western part of Wyoming, and they are considered a significant risk factor to Wyoming's cattle herds (WBCT).

Brucellosis has serious economic consequences for the cattle industry. After losing brucellosis-free status in 2004, the cattle sector was required to adopt costly brucellosis testing and vaccination practices (Koontz and Loomis, 2005) and infected herds were to be destroyed (depopulated).[3] Still, vaccination alone is only 65–70% effective in protecting animals from the disease. Also, cattle must be tested and demonstrated to be free of brucellosis within 30 days prior to interstate movement or change of ownership. Producers in Wyoming, Idaho, and Montana must also vaccinate their cattle and participate in surveillance programs, due to the reservoir of brucellosis in elk and bison of the GYA. The response to these costly requirements has been significant pressure to either reduce or eliminate supplemental feeding of elk, and to reduce elk densities (Kreeger et al., 2002). Wyoming regained brucellosis-free status in 2008, yet disease risks to the cattle sector still remain as the wildlife reservoir of brucellosis remains a threat.

Wyoming's brucellosis situation is complicated by both scientific uncertainty and political issues. Scientific uncertainty arises about transmission rates between the different species as well as within species, and also the impact of the feedgrounds on this transmission. Politically, there is a lack of public or social awareness of the implications associated with developing solutions, and there are different perspectives about what should be done with elk feedgrounds. Artificial feeding is a double-edged sword. On the one hand, it increases the probability that the elk and bison congregate, and therefore increases the transmission rate.[4] When the wildlife move out of the feeding grounds, they are more likely to transmit brucellosis to the cattle. On the other hand, the feedgrounds increase winter survival of wildlife and, to some extent, draw wildlife away from livestock areas so as to prevent commingling between wildlife and livestock. In addition, hunting groups want large elk populations, and many local economies rely on elk-related hunting and tourism revenues (Loomis and Caughlan, 2004).

## Epidemiological Model

We begin with an epidemiological model of population and disease dynamics within and across elk and cattle. The model is a "hybrid" of two commonly used forms of the SIR model, each of which reflects a different degree of aggregation. Disease transmission in elk is modeled using individual elk as the primary unit of analysis. Transmission in cattle is modeled using a metapopulation model defined at the herd level, which is the most common unit of analysis for disease reporting and policy purposes. Interaction between these entities is modeled by appropriately scaling cross-species transmission parameters. The models are also modified relative to traditional epidemiological models in the way they incorporate human choices. Farmer vaccination choices are endogenously determined, as farmers are no longer required to vaccinate but are still at risk of

[3] There are two vaccines available for cattle, Strain 19 and RB-51. Most herds are RB-51 vaccinated because Strain 19 results in a higher occurrence of false positive tests (Dean et al., 2004).

[4] Although brucellosis in bison is a focus in the Greater Yellowstone Area, there are relatively few bison congregating in the elk feedgrounds, and their effects on the nearby farms are smaller than those of elk. For simplicity, we only model the disease transmission between elk and livestock.

infection. Disease prevalence in the elk population and in cattle herds will affect vaccination decisions. The elk component of the model includes management choices involving feeding and hunting, which have not been modeled in previous analyses (e.g., Dobson and Meagher, 1996).

*Elk*

The elk population ($X$) consists of three subpopulations: susceptible ($X_S$), infected ($X_I$), and resistant ($X_R$). Population dynamics are based on Dobson and Meagher's (1996) SIR model, adjusted for harvests and feeding. Specifically, the change in $X_S$ is:

$$\dot{X}_S = \left[X_S + \eta X_I(1-\zeta) + X_R\right][a - \varphi X] - MX_S - B_{ee}X_S X_I - B_{ec}X_S In + \sigma X_R - h\frac{X_S}{X}, \tag{1}$$

where the first term on the right-hand side (RHS) represents the natural reproduction of susceptible elk, accounting for the impact of density-dependent competition; the birth rate is given by $a$; $\zeta$ is the proportion of infected females that produce infected offspring; $\eta$ is the reduction of fecundity in infected animals; and $\varphi$ represents the magnitude of the density-dependent competition effect. The second term represents natural mortality, with the natural mortality rate given by $M$. The third and fourth terms represent the number of elk infected by elk and livestock, respectively. Following Dobson and Meagher (1996), elk-to-elk transmission is of the form $B_{ee}X_S X_I$, with $B_{ee}$ representing the rate of infectious transmission among elk. Cattle-to-elk transmission is similarly defined, with $B_{ec}$ denoting the rate of transmission between cattle and elk.[5] The expression $In$ represents total infected cattle, where $I$ is the proportion of infected farms and $n$ is the number of cattle farms. The fifth term represents the number of newly susceptible elk that were previously recovered and immune, but which have lost their resistance to brucellosis. The rate of the lost resistance is expressed by $\sigma$.

The final term in (1) represents hunting. Hunting is nonselective with respect to health status, since it is not possible to identify infected animals until they are harvested (Lanfranchi et al., 2003; Williams et al., 2002). Only the total harvest ($h$) is chosen, and the harvest from each stock then depends on the proportion of animals in that stock relative to the total population, $X$—i.e., $h_j = hX_j/X$, where $h_j$ denotes the harvest from subpopulation $j$ ($j = I, S, R$).

The change in the infected stock of the elk is specified as:

$$\dot{X}_I = \eta\zeta X_I(a - \varphi X) - MX_I + B_{ee}X_S X_I + B_{ec}X_S nI - AX_I - \delta X_I - h\frac{X_I}{X}, \tag{2}$$

[5] Using Barlow's (1995) notation, disease transmission takes the general form $\beta I \times f(S/N) \times g(N)$, where $\beta$ is the transmission rate, $I$ is the relevant infected population, $S$ is the relevant susceptible population, $N$ is the total number of hosts associated with the susceptible population, $f$ is the susceptibility function, and $g$ is the density function. The susceptibility function specifies "the relationship between the effective or local proportion susceptible (per infectious) and overall proportion susceptible" (Barlow, p. 235). Dobson and Meagher (1996) set $f = S/N$ for elk, which implies uniform mixing of susceptible elk. The density function indicates how the contact rate, $\beta g$, varies with host density. Though $g = 1$ is often used for sexually transmitted and indirectly transmitted diseases (Barlow), Dobson and Meagher suggest $g = N$ is appropriate for elk, so that contacts are proportional to density. We adopt their specifications for $f$ and $g$.

where the first RHS term of equation (2) represents the reproduction of infected elk; the second term is natural mortality; the third and fourth terms represent the respective number of elk being infected by elk and livestock; the fifth term reflects disease-related mortality, where $A$ is virulence (disease mortality rate); the sixth term is the number of infected elk that recover from brucellosis, where $\delta$ is the recovery rate; and the final term represents the reduction in $X_I$ due to hunting.

The change in the resistant stock of elk is given by:

$$\dot{X}_R = \delta X_I - MX_R - \sigma X_R - h\frac{X_R}{X}, \tag{3}$$

where the first RHS term represents the number of elk that recover from infection, the second and third RHS terms reflect the decrease in the number of resistant elk due to mortality and loss of resistance, and the final term is the harvest of recovered elk.

Unlike Dobson and Meagher's (1996) model, where all the ecological parameters are exogenous to human choices, we assume some parameters are endogenous functions of the supplemental feeding choice, $f$. Specifically, natural mortality is assumed to be declining in $f$. We model this as $M(f) = m(1 - \omega_M f)$, where $m$ is the natural mortality rate and $\omega_M$ is a parameter representing the effect of feeding on reducing mortality. Virulence is also declining in feeding and is modeled by $A(f) = \alpha(1 - \omega_A f)$, where $\alpha$ is the natural virulence rate and $\omega_A$ is the feeding effect parameter. Elk-to-elk transmission is increasing in $f$, as the feeding activity causes animals to congregate in large densities. We model the transmission rate as $B_{ee}(f) = \beta_{ee}(1 + \mu_{ee} f)$, where $\beta_{ee}$ is the natural transmission rate and $\mu_{ee}$ is a parameter reflecting how feeding increases this rate.[6] Finally, cattle-to-elk transmission decreases in $f$, as the feeding activity causes fewer cross-species contacts. The transmission rate is expressed as $B_{ec}(f) = \beta_{ec}(1 - \mu_{ec} f)$, where $\beta_{ec}$ is the natural cross-species transmission rate and $\mu_{ec}$ is a parameter reflecting how feeding decreases this rate.

Finally, note that these assumptions about the role of feeding result in an endogenous carrying capacity for elk. For instance, the carrying capacity when there are no infected animals is given by $K(f) = (a - M(f))/\varphi$. The expression for carrying capacity is significantly more complex, but still endogenous (due to the endogeneity of $M$, $A$, and the disease transmission rates), in the presence of the disease.

### *Cattle*

A metapopulation disease model (Levins, 1969) is used to model livestock disease dynamics. Disease transmission occurs based on contact among $n$ homogeneous farms (e.g., when animals are on public grazing areas) and between farms and wildlife (e.g., on farmlands or public grazing areas). Each farm is in one of the four disease states at any point in time. Specifically, $s$ farms are susceptible, $i$ farms are infected, $r$ farms are resistant, and $e$ farms are empty.

The change in the number of susceptible farms over time is denoted by:

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[6] Alternatively, the transmission rate could be viewed as a constant and instead feeding could be viewed as acting on the density function $g$ [using Barlow's (1995) terminology, as described in footnote 5] so that $g = N(1 + \mu_{ee} f)$. The two perspectives are analytically equivalent, since the overall impact in each case is to increase the contact rate $\beta g$.

$$\dot{s} = \varepsilon e - \upsilon s - \beta_{cc} i\left([1-\upsilon]s/n\right)^{\gamma} - (\beta_{ce}/\psi)[1-\mu_{ce} f]X_I\left([1-\upsilon]s/n\right)^{\gamma} + (\chi+\tau)r, \tag{4}$$

where the first RHS term represents the number of newly repopulated farms, where repopulation occurs at the rate $\varepsilon$; the second RHS term is the number of farms that become resistant due to vaccination, where $\upsilon$ is the proportion of farms that vaccinate at time $t$; the third and fourth terms are the number of new infections due to contacts with infected cattle and elk, respectively. Cattle-to-cattle transmission is given by $\beta_{cc} i([1-\upsilon]s/n)^{\gamma}$, where $\beta_{cc}$ is the disease transmission parameter. The expression $([1-\upsilon]s/n)^{\gamma}$ is the susceptibility function (Barlow, 1995), where $[1-\upsilon]s$ represents the number of susceptible cattle after vaccination, and $\gamma$ is a spatial heterogeneity parameter.[7] This specification allows the population to mix homogeneously locally but heterogeneously globally, which is what would be expected when dealing with a larger number of individual herds. Accounting for this heterogeneity yields more realistic predictions for diseases that do not result in high prevalence, which is the case for brucellosis at the herd level. Barlow indicates this specification is good at approximating heterogeneous mixing behavior when $\gamma$ is large, such as $\gamma = 10$, with larger values of $\gamma$ indicating greater heterogeneity in herd mixing across the broader landscape.

An analogous expression is used to model elk-to-cattle transmission, though the elk-to-cattle transmission parameter $\beta_{ce}$ must be divided by the average number of cattle per farm, $\psi$, since we are measuring the number of farms (not animals) becoming infected due to contact with elk, which are measured in animal units. The parameter $\mu_{ce}$ reflects how feeding decreases the rate of elk-cattle contacts. The last term in (4) represents the number of farms losing resistance. This occurs naturally at the rate $\chi$, and it is enhanced by the rate at which new (nonresistant) animals are purchased from outside the region (i.e., an animal turnover rate), $\tau$.

The change in the number of infected farms over time is written as:

$$\dot{i} = \beta_{cc} i\left([1-\upsilon]s/n\right)^{\gamma} + (\beta_{ce}/\psi)X_I\left([1-\upsilon]s/n\right)^{\gamma} - qi, \tag{5}$$

where the first two terms denote newly infected farms, as described above, and the last term represents depopulation of infected farms, which occurs at the rate $q$.

The change in the number of resistant farms is designated by:

$$\dot{r} = \upsilon s - (\chi+\tau)r. \tag{6}$$

The resistant stock is increased due to vaccination and is decreased as resistance is lost. Finally, all transitions between disease states in (4)–(6) are balanced by changes in the number of empty (depopulated) farms, given by:

$$\dot{e} = qi - \varepsilon e. \tag{7}$$

We rewrite the dynamic system in terms of proportions of farms in each state (e.g., Hess, 1991; McCallum and Dobson, 2002), as public decision makers are often concerned with prevalence rates (i.e., the proportion of farms infected). Specifically, define $S = s/n$ as the

[7] Using the notation of footnote 5, $f(S/N) = ([1-\upsilon]s/n)^{\gamma}$ and $g(N) = 1$. Dobson and Meagher (1996) investigate both $g = 1$ and $g = N$ for the case of bison and find that $g = 1$ produces more reasonable results. These results are also in line with the view that $g = 1$ is often realistic for sexually transmitted and indirectly transmitted diseases (Barlow, 1995). Because cattle are behaviorally similar to bison, i.e., both are herd species, we adopt $g = 1$.

proportion of susceptible farms, $I = i/n$ as the proportion of infected farms, $R = r/n$ as the proportion of resistant farms, and $E = e/n$ as the proportion of empty farms. Upon making this transformation, equations (4)–(7) become:

$$\dot{S} = \varepsilon E - \upsilon S - \beta_{cc} I(1-\upsilon)^{\gamma} S^{\gamma} - (\beta_{ce}/n\psi) X_I (1-\upsilon)^{\gamma} S^{\gamma} + (\chi + \tau) R, \tag{8}$$

$$\dot{I} = \beta_{cc} I(1-\upsilon)^{\gamma} S^{\gamma} + (\beta_{ce}/n\psi) X_I (1-\upsilon)^{\gamma} S^{\gamma} - qI, \tag{9}$$

$$\dot{R} = \upsilon S - (\chi + \tau) R, \tag{10}$$

and

$$\dot{E} = qI - \varepsilon E. \tag{11}$$

Metapopulation models of disease transmission generally treat vaccination ($\upsilon$) as an exogenous behavioral parameter. In contrast, we take vaccination to be endogenous. Next we develop the behavioral dynamics that govern the vaccination choices, which are made in response to economic factors and current disease risks. In turn, the vaccination choices endogenously affect disease dynamics in our joint model. In this way, we account for dynamic feedbacks between the economic and disease systems.

## A Dynamic Model of Farmer Behavioral Choices

We assume the individual farms are identical except possibly for their current disease status (indexed by $j = S, I, R, E$) and their vaccination strategy, which is chosen in response to their current and expected future disease risks. Specifically, farmers make vaccination choices taking into account how these choices affect the possibility that the farm will transition to a new disease state at some time in the future. Denote the vaccination strategy of an individual farmer by $z$. The strategy is a discrete choice: $z = 1$ implies whole-herd vaccination, $z = 0$ implies no vaccination. The proportion of farms adopting vaccination at any point in time is given by $\upsilon$, as defined above.

A farm in a given disease state receives an expected flow of income associated with its current disease state. Denote a farm's baseline profit in each period in which the farm operates (i.e., $j \neq E$) by $\pi$, with profits being zero during the empty state. Susceptible farms that vaccinate will also expect to incur vaccination costs of $c/\kappa$ where $c$ is the cost of vaccinating an average herd at one time and $\kappa$ is the effectiveness of the vaccination. Infected farms will incur private losses from infection, $b$. Empty farms earn no profits.[8]

Following Shapiro and Stiglitz (1984) (see also Hennessy, 2007), denote $Y_j^z$ to be the expected lifetime income of a farmer who is currently in state $j = S, I, R, E$ and has adopted the strategy of choosing action $z$. A farm's vaccination strategy, as well as current infection levels, influences the likelihood the farm transitions from one state to another. Specifically, the individual's probability of transitioning from state $S$ to state $I$, given the strategy $z$, is $P_{SI}^z$. This value can be obtained from the epidemiological model as $P_{SI}^{z=1} = 0$, $P_{SI}^{z=0} = \beta_{cc} I[(1-\upsilon)S]^{\gamma-1} + (\beta_{ce}/n\psi) X_I [(1-\upsilon)S]^{\gamma-1}$ (see appendix), which changes

[8] Farmers are usually paid the market value of the animal, after which time the farm must repopulate the herd. Generally, not all costs are reimbursed and the farmer earns a net loss in this situation (Gramig et al., 2006). We assume the loss is sufficiently small so as not to worry about it.

over time as infection risks change. The individual's probability of transitioning from the susceptible state to the resistant state is simply the vaccination strategy: $P_{SR}^{z} = z$. The individual's probability of transitioning from the resistant state to the susceptible state is $P_{RS} = \chi + \tau$. The individual's probability of transitioning from the infected to the empty state is $P_{IE} = q$. Finally, the individual's probability of transitioning from the empty state to the susceptible state is $P_{ES} = \varepsilon$.

Farmers are forward looking because their choices have intertemporal consequences. However, farmers do not have rational expectations with respect to transition probabilities. Rather, farmers know the current disease risks and assume these continue on into the future, thereby taking the transition probabilities as fixed when decisions are made within a given period. As we describe below, the probabilities are updated at each decision node, so that farmers exhibit adaptive expectations.[9]

Assuming a discount rate of $\rho$, the respective fundamental asset equations for susceptible, infected, resistant, and empty farms are:

$$\rho Y_S^z = \pi - [c/\kappa]z + P_{SI}^z\left[Y_I^z - Y_S^z\right] + P_{SR}^z\left[Y_R^z - Y_S^z\right], \tag{12}$$

$$\rho Y_I^z = \pi - b + P_{IE}\left[Y_E^z - Y_I^z\right], \tag{13}$$

$$\rho Y_R^z = \pi + P_{RS}\left[Y_S^z - Y_R^z\right], \tag{14}$$

and

$$\rho Y_E^z = -d + P_{ES}\left[Y_S^z - Y_E^z\right]. \tag{15}$$

Equation (12) represents the "time value of the asset" in the susceptible state, which equals the sum of the "instantaneous income per unit time" conditional on being susceptible, $\pi - [c/\kappa]z$, and the "expected capital loss that would arise were the state to change" (Hennessy, 2007) from susceptible to infected, $P_{SI}^z[Y_I^z - Y_S^z] + P_{SR}^z[Y_R^z - Y_S^z]$. Equations (13)–(15) have similar interpretations. In equation (15), $d$ is the cost to farms when they are depopulated. These equations can then be solved simultaneously for $Y_j^z$ $(j = S, I, R, E)$ as functions of the behavioral strategies, the states of the world, and economic and epidemiological parameters.

The vaccination choice is made while the farm is in the susceptible state, with the benefits of vaccination depending on the actions of all farmers (via the transition probability $P_{SI}^z$). At each point in time, the farmer makes the decision anew, updating the transition probabilities to reflect the current state of the world (hence exhibiting adaptive expectations). In the long run, the system will equilibrate at a point of indifference, i.e, $Y_S^{z=1} = Y_S^{z=0}$ (if such a point exists), so that no farmer has an incentive to change his or her vaccination strategy. We use *replicator dynamics* to model adjustment to such an equilibrium. The basic idea behind replicator dynamics is that the adoption of a particular strategy will increase in frequency when the net benefits from that choice

[9] The assumption of rational expectations seems too strong, as this would involve a differential game between $n$ farms ($n$ = 190 in our numerical example). Each farm would have to perfectly predict the actions of every other farm to accurately predict changes in risks. It appears unlikely that individual farmers, with limited information about their neighbors, would be able to accomplish this (though see footnote 10 for more on the comparison between the two approaches). At the other extreme would be a completely myopic farmer who does not take any future impacts into account and instead maximizes static profits. We also view this as unrealistic, as farmers are accustomed to making long-run decisions about their asset holdings (i.e., their cattle stocks).

outweigh average net benefits associated with the current frequency of adoption (Rice, 2004). Specifically, frequency of adoption increases when expected lifetime income from adopting vaccination exceeds the average expected lifetime income associated with the current distribution of vaccination strategies, $\overline{Y}_S = \upsilon Y_S^{z=1} + (1-\upsilon)Y_S^{z=0}$:

$$\frac{\dot{\upsilon}}{\upsilon} = \alpha\left[Y_S^{z=1} - \overline{Y}_S\right] \Rightarrow \dot{\upsilon} = \theta\upsilon(1-\upsilon)\left[Y_S^{z=1} - Y_S^{z=0}\right], \tag{16}$$

where $\theta > 0$ is a speed-of-adjustment parameter.[10] Equation of motion (16) indicates that frequency of vaccination adoption is increasing (decreasing) when the expected profit from always investing in vaccination exceeds (is less than) the expected profit from never investing in vaccination. As described above, farmers are indifferent about vaccination in the steady state, i.e., $Y_S^{z=1} = Y_S^{z=0}$. So if disease risks increase, farmers will vaccinate more until the vaccination cost is equal to the costs of infection.

Our model of adaptive expectations differs from some prior economic work in which it is assumed that $S$, $I$, and $R$ are fixed (e.g., Hennessy, 2007). Indeed, $S$, $I$, and $R$ are not fixed in the joint dynamic system, especially when we consider the effects of the elk and cattle populations together. This means the probability of becoming infected is non-stationary, and therefore assuming stationarity when performing policy analysis may result in misleading policy recommendations. In the example below, we consider behavioral and disease dynamics jointly by incorporating the replicator dynamics model with the disease dynamics model of the elk and cattle population.

## Numerical Example

The Jackson elk herd in northwestern Wyoming is one of the largest elk herds in the world, with a population estimated at 12,904 for the post-2006 hunting season (WGFD, 2007). The area it concentrates is called the Jackson Elk Herd Unit (EHU), which is located in the upper Snake River drainage and including all drainages of the Snake River downstream to and including the Gros Ventre River drainage and Flat Creek north of the town of Jackson. The total area of the Jackson EHU is approximately 2,350 mi$^2$.

There are three state-operated elk feedgrounds within the Jackson EHU: Alkali, Patrol Cabin, and Fish Creek. Elk also receive supplemental winter feeding on the National Elk Refuge (NER), which is managed by the WGFD and U.S. Fish and Wildlife Services. In 2006, there were 3,217 elk on feedgrounds in the Gros Ventre drainage, 6,730 elk on the NER, 331 elk being baited away from private feedlines, and 1,575 elk on native winter ranges (WGFD, 2007).

The Jackson elk herd has contact with bison and cattle. During the elk calving period from late May to mid-June, the risk of brucellosis transmission to cattle on overlapping ranges increases. Within the Jackson EHU, four allotments (Burro Hill, Pacific Creek-

[10] The assumption of replicator dynamics is consistent with our assumption of adaptive expectations. For instance, Berck and Perloff's (1984) model of adaptive expectations in the decision to enter or exit a fishery is essentially analogous to our replicator dynamics with $Y_{SI}^{z=0} = 0$, at least for interior outcomes. Moreover, their comparison of adaptive and rational expectations models results in identical steady states, with only the paths to these steady states differing. Likewise, our adoption of replicator dynamics does not affect the steady state, given the discrete nature of the vaccination choice. The primary way in which replicator dynamics might differ from alternative approaches is in the path to the steady state. Using Berck and Perloff's results as a guide, we would expect fewer oscillations en route to the steady state under rational expectations. This is because farmers would be making better interim predictions of changes in transition probabilities and would therefore be less likely to overshoot or undershoot the eventual outcome.

GTNP and BTNF, Upper Gros Ventre, Big Cow Creek) overlap spatially with designated elk parturition ranges (WGFD, 2007). The four cattle allotments are located in Teton and Sublette counties. In 2002, there were around 190 farms with 57,010 cattle and calves in the two counties [USDA/National Agricultural Statistics Service (NASS), 2004].

The Wyoming Game and Fish Commission's population objective for the Jackson elk herd is 11,029. There are also proposals to reduce feeding operations to lower disease risks. Some proposals involve reductions in feeding, while others involve closing a number of state feedgrounds as well as the NER (Smith, 2001). These proposals form the basis for our simulation experiments below.

## Simulation

We now turn to a simulation of the brucellosis problem because the dynamic system is too complex to examine analytically. Simulation results are solved using the software Mathematica 7.0 (Wolfram Research, Inc., 2008) to derive numerical solutions to the differential equations presented above, using the model parameters defined in table 1 (all of which are developed for the continuous-time algorithm). The model is best viewed as a numerical example rather than a rigorous case study, as many of the epidemiological parameters—particularly those involving cross-species transmission and the impacts of the feedgrounds on disease transmission—are not available. We have performed sensitivity analyses with respect to each parameter, and we present some results of those analyses in a later section.

Our simulation is based on the current state of the world in which Wyoming has just been given brucellosis-free status after a period of intense regulation, and it is assumed that all regulations have been lifted. The initial level of vaccination is $v(0) = 0.99$ due to the intensive vaccination requirements previously in effect. We also assume the initial level of infection is small but not zero, as perhaps a few infected herds do remain or there was some new introduction of disease via the elk herd. Specifically, let $I(0) = 0.02$. We then investigate the decentralized farm vaccination decisions, cattle disease dynamics, and elk dynamics under different types of cattle-elk interactions, and under alternative elk management strategies—i.e., we focus on what might happen if the risks due to the infected elk lead to increased infections within cattle, assuming vaccination mandates were not reinstated. In this situation, farmers only vaccinate if it is cost-effective for them to do so.

### *The Cattle Sector Only: No Infection Risk by Elk*

We first analyze the cattle sector without infection risks from the elk sector. This serves as a benchmark for understanding the importance of modeling wildlife disease risks, which is relevant because most livestock disease problems are modeled without consideration of these external risks. The results are illustrated in figure 1. Given our starting values, the infection rate initially goes down because initial vaccination rates are high. Farmers respond to the lower infection rate by gradually decreasing their investment in vaccination. But then infection levels increase when vaccination becomes very low, creating incentives for some farmers to vaccinate again. The result is an oscillation of vaccination and infection rates. The proportions of susceptible and resistant

**Table 1. Parameters for Simulation**

| Parameter | Value | Source (if applicable) |
|---|---|---|
| $\rho$ (discount rate) | 0.05 | Assumption |
| $b$ (infection cost) | \$600 | Derived from Bittner (2004) |
| $d$ (depopulation cost) | \$15,150 | Assume 5% of the total cattle value |
| $c$ (vaccination cost) | \$300 | Based on online prices for the RB51 vaccine and average herd size |
| $\pi$ (income flow) | \$11,420 | Derived from USDA/NASS (2004) |
| $n$ (number of farms in Jackson EHU) | 190 | Derived from USDA/NASS (2004) |
| $\chi$ (rate of lost resistance for cattle) | 0.01 | Assumed same as for elk |
| $\varepsilon$ (transition rate from empty to susceptible) | 1 | Assumption |
| $\kappa$ (vaccination effectiveness) | 0.7 | Bittner (2004) |
| $\beta_{cc}$ (cattle-cattle infectious contact rate) | 2 | Derived based on equation in Dobson and Meagher (1996) |
| $\beta_{ce}$ (elk-cattle infectious contact rate) | 2 | Derived based on equation in Dobson and Meagher (1996) |
| $\psi$ (average number of cattle per farm) | 300 | USDA/NASS (2004) |
| $q$ (depopulation rate) | 0.5 | Assumption |
| $\theta$ (adjustment parameter) | $1 \times 10^{-5}$ | Assumption |
| $\tau$ (cattle "turnover rate") | 0.22 | Assumption |
| $\sigma$ (rate of lost resistance for elk) | 0.01 | Dobson and Meagher (1996) |
| $a$ (elk birth rate) | 0.327 | Derived from Lubow and Smith (2004) |
| $m$ (elk natural mortality rate) | 0.15 | Dobson and Meagher (1996) |
| $K$ (elk carrying capacity when feeding occurs) | 59,000 | Lubow and Smith (2004) |
| $\zeta$ (proportion of infected female elk that produce infected offspring) | 0.9 | Dobson and Meagher (1996) |
| $\eta$ (reduction of fecundity in infected elk) | 0.5 | Dobson and Meagher (1996) |
| $\beta_{ee}$ (elk-elk infectious contact rate) | 0.002 | Derived based on equation in Dobson and Meagher (1996) |
| $\beta_{ec}$ (cattle-elk infectious contact rate) | 0.002 | Derived based on equation in Dobson and Meagher (1996) |
| $\mu_{ij}$ (feeding effect on transmission from species $j$ to species $i$) | 0.001 | Assumption |
| $\gamma$ (spatial heterogeneity in herd mixing) | 10 | Barlow (1995) |
| $\varphi$ (density-dependent competition effect) | $5 \times 10^{-6}$ | Derived from $\varphi = (a - M)/K$ |
| $\delta$ (elk recovery rate) | 0.5 | Dobson and Meagher (1996) |
| $\alpha$ (elk virulence rate) | 0.005 | Dobson and Meagher (1996) |
| $\omega_M$ (feeding effect on mortality rate) | 0.001 | Calibrated given $m = 0.15$ and $M = 0.037$ (Kreeger et al., 2002) |
| $\omega_A$ (feeding effect on virulence) | 0.001 | Assumption |
| $X(0)$ (initial elk stock) | 12,904 | Wyoming Game and Fish Dept. (2007) |
| $X_R(0)/X(0)$ (initial proportion of resistant elk) | 0.2 | Kreeger et al. (2002) |
| $X_I(0)/X(0)$ (initial proportion of infected elk) | 0.3 | Kreeger et al. (2002) |

( continued . . . )

**Table 1. Continued**

| Parameter | Value | Source (if applicable) |
|---|---|---|
| $R(0)/n$ (initial proportion of resistant farms) | 0.8 | Assumption |
| $I(0)/n$ (initial proportion of infected farms) | 0.02 | Assumption |
| $S(0)/n$ (initial proportion of susceptible farms) | 0.01 | Assumption |
| $v(0)$ (initial vaccination rate) | 0.99 | Assumption |

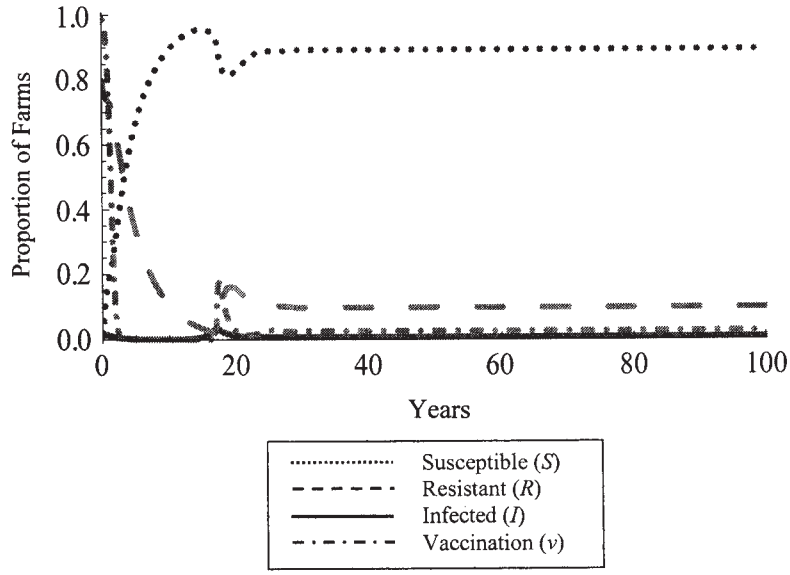


**Figure 1. Cattle-only model simulation results**

cattle farms also change in response to these fluctuations. The whole system reaches a steady state at around 25 years, with infection rates of approximately 0.68% and vaccination of approximately 2.5%.

*The Joint Model*

The brucellosis problem in Wyoming, compared to the problem in the cattle-sector-only model, is more complex. Elk in the feedgrounds pose a constant risk of brucellosis, and should be considered in any simulation model. Brucellosis in the elk population can be reduced by reducing infectious contacts, and this can occur in two ways in our model: (*a*) reduce the elk population, and (*b*) reduce feeding. Dobson and Meagher (1996) suggest that population controls alone may be ineffective, as the threshold population level at which the disease begins to dissipate naturally is quite low (around 250 animals). There is less research on the transmission impacts of the feedgrounds, but feeding is important in other disease systems such as bovine tuberculosis among Michigan white-tailed deer (e.g., Hickling, 2002). In Wyoming, 80% of the elk winter in the feedgrounds (Dean et al., 2004), and it is believed that feedground management plays an important role in the disease dynamics (Dean et al.; Smith, 2001).

We consider four different combinations of elk disease management policies: (*a*) no disease management (i.e., no hunting and feeding at current levels); (*b*) population controls only (with feeding at current levels); (*c*) feeding controls (and no population controls); and (*d*) feeding and hunting controls. Specifically, feeding controls involve closing the feedgrounds, while hunting controls involve maintaining a total elk population of 11,029 animals. We then study the effects of these controls on the elk disease dynamics and the disease and behavioral dynamics in the cattle sector. We also analyze economic impacts to the cattle sector.[11]

### Scenario (*a*): No Elk Disease Management

We start with the no management case, in which there is no hunting, and feeding occurs at current levels. We calculated the current average feeding in the NER and other three state-operated feedgrounds, which is approximately 766.6 kg/km$^2$. We restrict our attention to this feeding level, but note that our qualitative results show that different feeding levels have a similar effect on the disease and behavior dynamics. This scenario serves as a baseline for examining various population and feeding control policies.

The simulation results are presented in table 2 and in figures 2 and 3. Disease dynamics in the elk sector are only minimally affected by feedbacks from the cattle sector. Starting from current elk population levels, the combination of large supplemental feeding and no hunting allows the overall population to grow, as well as the number of infected animals. The steady-state number of infected animals (4,828 elk) is 41% larger than in scenarios (*b*)–(*d*), but 37% smaller than scenario (*c*), in which there are only feeding controls. This last result arises due to feeding. Feeding increases the disease transmission between elk, but with the process of becoming resistant, both the number and percentage of infected elk actually decrease. The majority of elk end up resistant, which is consistent with Dobson and Meagher's (1996) results.

The disease and behavior dynamics for the cattle sector are a little different from the cattle-only model. The constant force of infection from the elk herd causes the proportion of vaccination and infected states to converge to steady states much faster than in the cattle-only model (figure 2). The proportion of herds that vaccinate converges to 5.62% after approximately 12 years. This scenario is effectively tied with scenario (*b*) for having the smallest vaccination rate among all scenarios. The reason is that with so many resistant elk, infection risks from the elk sector are actually small. These risks are further reduced because the feedgrounds help to separate elk and cattle, resulting in fewer contacts.

Though the infection risks from elk are low, the low level of vaccination results in higher overall cattle infection levels compared to scenarios (*c*) and (*d*) (table 2). Still, the net benefits to the cattle sector are not sufficiently different from those of scenario (*c*), while being slightly less than those of scenarios (*b*) and (*d*) (table 2).

### Scenario (*b*): Elk Population Controls Only

The Wyoming Game and Fish Department's population objective for the Jackson elk herd is 11,029 elk, so this scenario actually best reflects the status quo. Hunting levels

[11] We do not analyze economic impacts to the elk sector due to a lack of data on elk hunting values. Hunters will generally benefit from a larger supply of healthy elk, and so feeding and elk conservation can be valuable for its non-disease impacts to the hunting sector. Visitors to the area also benefit from viewing elk (Loomis and Caughlan, 2004).

**Table 2. Comparison of Steady-State Dynamic Outcomes and Farmers' Net Benefits for Different Policy Scenarios**

| | Policy Scenario | | | |
|---|---|---|---|---|
| Description | (*a*) No Elk Disease Management | (*b*) Elk Population Controls Only | (*c*) Elk Feeding Controls Only | (*d*) Elk Feeding and Population Controls |
| Vaccination ($v$)[a] | 5.62 | 5.37 | 9.95 | 8.18 |
| Susceptible Cattle ($S$)[a] | 79.6 | 80.4 | 69.3 | 73.2 |
| Infected Cattle ($I$)[a] | 0.59 | 0.60 | 0.49 | 0.53 |
| Resistant Cattle ($R$)[a] | 19.5 | 18.7 | 30.0 | 26.0 |
| Susceptible Elk ($X_S$) | 148 | 140 | 289 | 282 |
| Infected Elk ($X_I$) | 4,828 | 3,416 | 7,539 | 3,436 |
| Resistant Elk ($X_R$) | 53,634 | 7,473 | 23,559 | 7,312 |
| Per Farm Net Benefits (present value, $US mil.) | 41.8919 | 41.8921 | 41.8919 | 41.8921 |

[a] Expressed as a percentage of farms.

are calculated as the total elk population minus the objective of 11,029. No hunting takes place in years when there are less than 11,029 elk.

The simulation results (table 2, figures 2–3) show that the number of infected elk decrease relative to scenario (*a*) when hunting occurs. Here the impacts of continued feeding play a much stronger role in maintaining infection levels than population controls do in reducing infection levels. That population controls have little impact on reducing the number of infected elk is consistent with Dobson and Meagher's (1996) finding that the threshold population for brucellosis establishment is low, and this makes it very hard to apply population controls to eradicate, or even reduce the incidence of, brucellosis from wild populations. The elk population controls are effective at reducing the number of infected elk by 29% relative to scenario (*a*), though prevalence levels have actually increased from 8.2% to 31%. The overall number of infected farms is essentially the same as in scenario (*a*), though the lower level of vaccination under scenario (*b*) results in slightly larger net benefits to the cattle sector relative to scenario (*a*).

### Scenario (*c*): Elk Feeding Controls Only

Now consider closing all the elk feedgrounds, but not applying any population controls (table 2, figures 2–3). Relative to scenario (*a*), the total elk population will decrease as less food is available for them to survive in the winter, and the elk which used to concentrate in feedgrounds will immigrate to other areas, such as public grazing land, to search for food. This will reduce infectious contacts among elk, but it may cause elk to come into greater contact with cattle. Indeed, the feedgrounds were originally developed to reduce elk predation on cattle forage.

The number of infected elk is the largest in this scenario relative to the others. Although total contacts among elk are reduced with no feeding, there are more susceptible elk to become infected—i.e., infected elk are more likely to contact susceptible elk

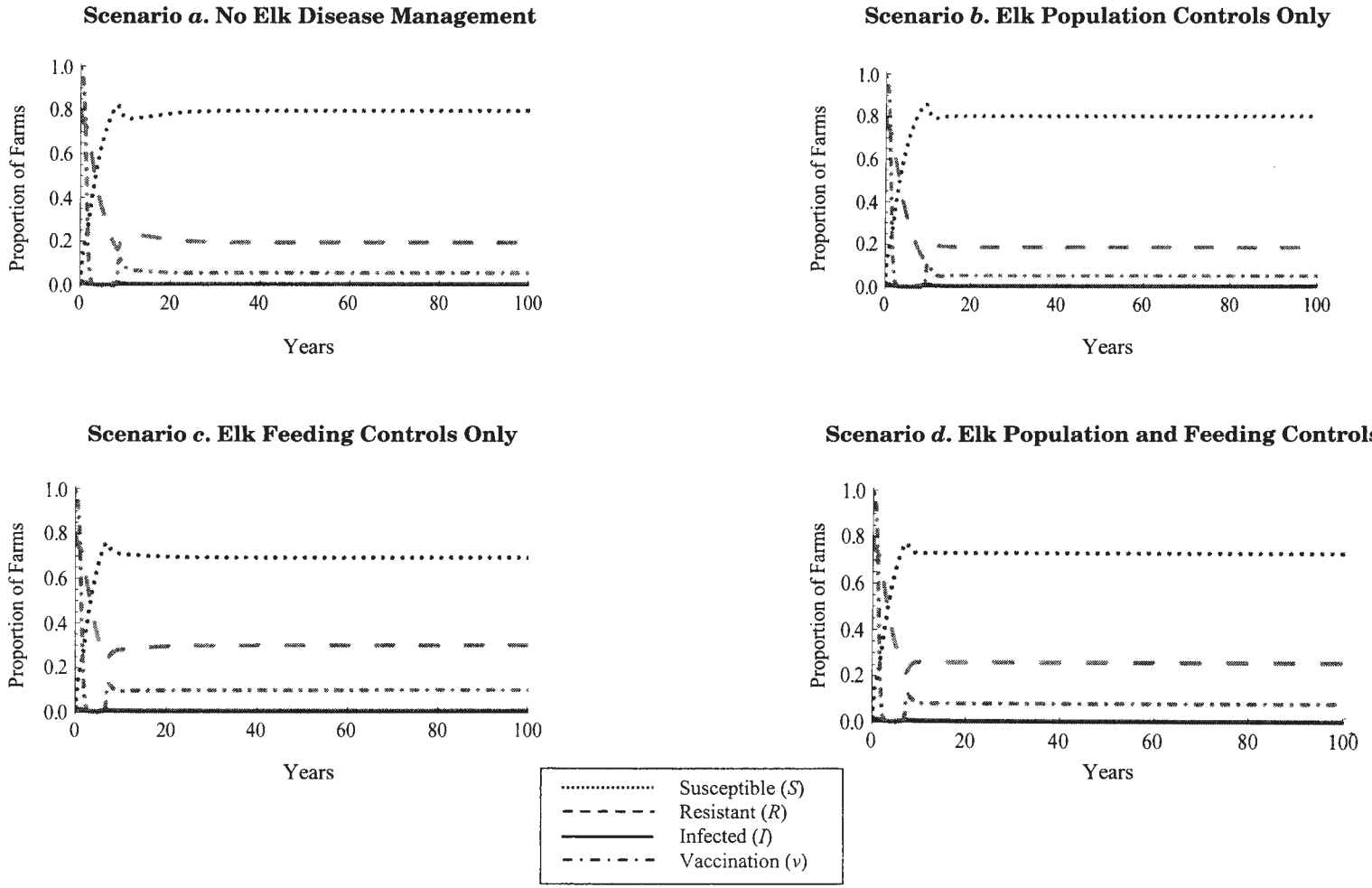


**Figure 2. Cattle results for the joint cattle-elk model**

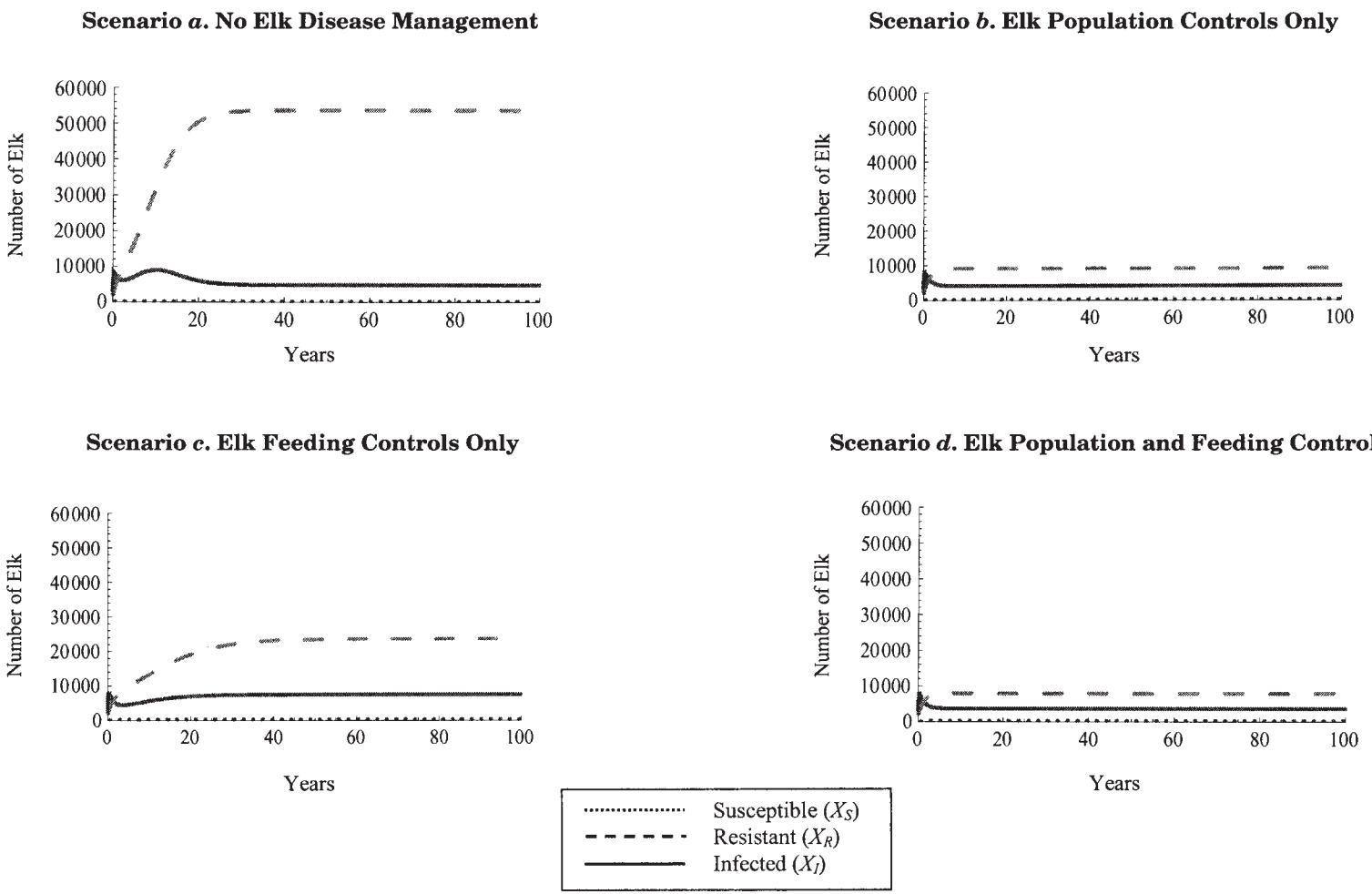


**Figure 3. Elk results for the joint cattle-elk model**

instead of resistant elk. The result is an increase in the number of infected elk. Why are there more susceptible elk? It is because the no-feeding policy increases mortality among all subpopulations, reducing the resistant stock in particular (as fewer infected animals live to become resistant) and also the density-dependent effects of resource competition. In fact, in the steady state, each year 536 infected elk live to become resistant in scenario (*a*), while only 236 infected elk survive each year in scenario (*c*). The result is there are more births of susceptible elk (as both susceptible and resistant elk usually give birth to susceptible elk, and half of births via infected mothers are susceptible), with 3,712 susceptible elk born in each year in the steady state in scenario (*c*) as compared to only 2,531 susceptible elk born each year in scenario (*a*). This contradicts the notion of feeding as the cause of high brucellosis prevalence in the Greater Yellowstone Area. In scenario (*a*) with feeding, more elk survive to become resistant. This limits disease spread in scenario (*a*) relative to scenario (*c*). Instead of spreading disease, elk feeding actually decreases elk infection levels by allowing more infected elk to survive to become resistant.

The large number of infected elk in scenario (*c*), along with immigration to cattle areas, creates more infectious contacts with cattle and thereby generates the greatest infection risks for the cattle sector. Farmers respond by increasing vaccinations to the largest level among all scenarios, though the number of infections is actually the smallest among all the scenarios. The combination of larger vaccination levels and lower infection levels suggests that vaccination efforts are responsive to risks from elk. Finally, farm net benefits are essentially the same as in scenario (*a*). This implies the costs of increased vaccination are offset by the benefits of reduced cattle infection relative to scenario (*a*).

### Scenario (*d*): Elk Population and Feeding Controls

Our last scenario involves both population and feeding controls. The simulation results (table 2, figures 2–3) show that the number of infected elk is similar to scenarios (*a*) and (*b*), suggesting the proposed levels of population controls may not be useful in eradicating wildlife disease, even when the feedgrounds are closed. Again, this is consistent with Dobson and Meagher's (1996) finding that the threshold population for brucellosis establishment is low, and it is very difficult to apply population controls to eradicate brucellosis from wild populations.

As with scenario (*b*), population controls reduce the number of infected elk. But, relative to scenario (*b*), closing the feeding grounds increases the number of infected elk. The net result is that the number of infected elk occurs at an intermediate level relative to scenarios (*a*) and (*c*), and is slightly larger than in scenario (*b*). The level of infected elk, along with increased migration of elk, results in higher infection risks to the cattle sector compared to scenarios (*a*) and (*b*). Therefore, the farmers invest in higher vaccination rates than under scenarios (*a*) and (*b*) [but lower than under (*c*)]. The net result is that the number of cattle infections is actually less than in scenarios (*a*) and (*b*).

Farm net benefits in this scenario are roughly the same as in the other scenarios, which implies the benefits and the costs of higher vaccination rates caused by greater infection risks are essentially offsetting. Again, the model predicts the feeding grounds do not actually generate economic harm to the livestock sector.

**Table 3. Sensitivity Analysis**

| Description | Scenario | | | | |
|---|---|---|---|---|---|
| | Reduce Feeding Effect on Elk-Cattle Infectious Contact Rate ($\mu_{ce}$) by 50% | Reduce Recovery Rate of Infected Elk ($\delta$) by 50% | Reduce Instantaneous Income Flow ($\pi$) by 50% | Increase Discount Rate ($\rho$) by 100% | Reduce Transmission Exponent Parameter ($\gamma$) by 25% |
| Vaccination ($v$)[a] | 7.66<br>(9.95) | 6.85<br>(10.9) | 5.16<br>(9.31) | 5.11<br>(9.23) | 8.04<br>(14.5) |
| Susceptible Cattle ($S$)[a] | 74.4<br>(80.5) | 76.4<br>(67.3) | 80.7<br>(70.5) | 80.9<br>(70.6) | 73.5<br>(60.9) |
| Infected Cattle ($I$)[a] | 0.54<br>(0.49) | 0.56<br>(0.47) | 0.76<br>(0.63) | 0.78<br>(0.65) | 5.89<br>(0.41) |
| Resistant Cattle ($R$)[a] | 24.8<br>(30.0) | 22.8<br>(32.0) | 18.1<br>(28.5) | 18.0<br>(28.4) | 20.5<br>(38.5) |
| Susceptible Elk ($X_S$) | 148<br>(289) | 77<br>(160) | 148<br>(289) | 148<br>(289) | 148<br>(289) |
| Infected Elk ($X_I$) | 4,828<br>(7,539) | 8,876<br>(10,872) | 4,828<br>(7,539) | 4,828<br>(7,539) | 4,828<br>(7,539) |
| Resistant Elk ($X_R$) | 53,634<br>(23,559) | 49,298<br>(16,986) | 53,634<br>(23,559) | 53,634<br>(23,559) | 53,634<br>(23,559) |
| Per Farm Net Benefits (present value, $US mil.) | 41.8922<br>(41.8919) | 41.8917<br>(41.8922) | 20.5345<br>(20.5342) | 20.6933<br>(20.6908) | 41.8926<br>(41.8935) |

Notes: All parameter values are the same as in table 2, except for the parameters indicated. The results are shown for the case of no elk disease management (scenario *a*) and elk feeding controls only (scenario *c*, shown here in parentheses).

[a] Expressed as a percentage of farms.

### *Sensitivity Analysis*

Sensitivity analyses are used to examine how changes in parameters of our model influence the results. We use scenario (*a*) as the baseline scenario from which to evaluate parameter changes—i.e., each sensitivity analysis is performed by holding all parameters and policy variables at the same level as scenario (*a*), and then changing a single parameter of interest as indicated. Model results were generally not very sensitive for most of the model parameters. The exceptions were the feeding effect on the elk-cattle infectious contact rate ($\mu_{ce}$), the recovery rate of elk ($\delta$), profit ($\pi$), the discount rate ($\rho$), and the spatial heterogeneity parameter ($\gamma$) (table 3). We also performed the sensitivity analyses for the other policy scenarios, and the results are qualitatively similar. The results of the sensitivity analysis for scenario (*c*) are presented in parentheses in table 3.

First consider the impacts of the parameter changes in terms of scenario (*a*) results. A reduction in $\mu_{ce}$ leads to more cross-species contacts. Farmers respond to this increased risk with a 36% increase in vaccination rates, resulting in a small reduction in the number of infected farms. Net benefits are largely unaffected.

A reduction in $\delta$ means more elk stay infected while fewer elk become resistant. The result is more infectious contacts, as the number of infected elk increases by 84%. Farmers respond to this increased risk with a 22% increase in vaccination rates, again leading to a small reduction in the number of infected farms. Net benefits are largely unaffected.

A reduction in $\pi$ or an increase in $\rho$ reduces the expected value of farm profits. With lower profits, farmers have fewer incentives to vaccinate. Accordingly, vaccination rates decline and the number of infected farms increases. Net benefits obviously fall due to the reduction in profits.

Finally, a reduction in the spatial heterogeneity parameter $\gamma$ (implying less heterogeneity) results in more infectious contacts, increasing the risks to farmers. Farmers respond to a 25% reduction in $\gamma$ by increasing vaccination rates by 43%. As a consequence, the number of infected farms is reduced slightly, though net benefits are essentially unchanged.

A comparison of the table 3 outcomes for scenario (*a*) and scenario (*c*) illustrates the impact of elk feeding is qualitatively unchanged relative to table 2, even under the alternative parameter values analyzed here. Specifically, closing the feedgrounds results in significantly more infected elk, farmer responses of increased vaccination, and slightly lower percentages of infected herds. This lends further support to our earlier finding that closing the elk feedgrounds will be of limited effectiveness.

## Conclusion

This paper expands the disease ecology literature by integrating disease dynamics with economic choices in such a way that risks of infection are a function of decentralized livestock disease management choices, and then economic choices are, in turn, a function of disease states. Our findings suggest these jointly determined feedbacks matter when choosing among various wildlife policy approaches.

By examining the population and disease dynamics under several different management options for the Jackson elk herd in Wyoming, we illustrate that elk disease dynamics and farmers' choices vary among different combinations of elk feeding and hunting policies. However, farmers' net benefits under different combinations of elk feeding and hunting policies are very similar to one another, as the costs and benefits of higher vaccination rates in response to greater infection risk essentially offset each other. In particular, closing elk feedgrounds reduces infectious contacts among elk but may increase infectious contacts between elk and cattle. The reduced contacts among elk actually lead to more infected elk, as the number of resistant elk decline. This results in increased risks to cattle, incentivizing farmers to respond with greater vaccination rates, with the net effect being only a small reduction in the number of infected farms. Hence, elk management policies chosen to reduce disease risks to livestock may lead to both ecological and farmer responses that largely offset these changes in risk. Our finding that the feeding grounds do not actually generate economic harm to individual farmers, and only marginally result in smaller herd infection levels (caused by farmer responses and not reduced risks from elk!), is surely not something expected by the livestock advocacy groups who are pushing for an elimination of the feeding grounds.

Finally, it may be hard to eradicate brucellosis from wild elk populations by population controls, even when feedgrounds are closed. This finding is consistent with the results of our prior ecological model which only considered the population dynamics without human management choices and without interactions between wildlife and livestock.

There is a growing need for analyses that combine disease ecology models with economic decision models, particularly as disease risks from wildlife reservoirs increase.

But an understanding of how human choices, such as habitat and livestock management decisions, influence within- and cross-species disease risks is surprisingly limited. More research in this area is needed. Without it, models such as the one presented here can only provide general insights—not detailed guidance on how to manage disease problems.

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## Appendix: Derivation of an Individual Farm's Transition Probabilities from State *S* to State *I*

Farmer $z$ must consider how his vaccination choice affects his transition probability to the infected state. Since his vaccination choice is a discrete one, we must define two probabilities: $P_{Sz}^{v=1}$ and $P_{Sz}^{v=0}$, such that

(A1)
$$P_{Sz}^{v=1} = 0,$$
$$P_{Sz}^{v=0} \in (0,1),$$
and
$$nS\left[vP_{Sz}^{v=1} + (1-v)P_{Sz}^{v=0}\right] = T_{cc}.$$

The last part of condition (A1) indicates that the expected number of cattle-to-cattle infections within the susceptible population equals the total number of cattle-to-cattle infections, $T_{cc}$. Note that the $z$ subscripts remain in condition (A1) only to indicate that these represent farm-level probabilities, not to indicate heterogeneities among the farmers, who are homogeneous. Using the first part of (A1), the final requirement can be written as:

(A2)
$$S(1-v)P_{Sz}^{v=0} = T_{cc}/n = \beta_{cc}\left([1-v]S\right)^{\gamma} I.$$

This can then be solved for

$$P_{Sz}^{v=0} = \beta_{cc}\left([1-v]S\right)^{\gamma-1} I.$$