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

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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 hurt people in Kenya and elsewhere in Africa and Asia (Nyhu 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. These types of human-wildlife conflicts are occurring more frequently, and the costs 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; Nyhu et.al. 2003,).

An economic literature has emerged to examine public and private incentives to mediate human-wildlife conflicts. Most studies examine the socially efficient management of wildlife stocks when they cause external damages, such as crop damages (Zivin et.al. 2000; Rondeau 2001; Horan and Bulte 2004), auto accidents (Rondeau and Conrad 2003), and disease transmission to livestock (Bicknell et al. 1999; Horan and Wolf 2005; Fenichel and Horan 2007a,b). In particular, Fenichel and Horan (2007a,b) find that managing wildlife to control disease problems may be expensive. This is because it is often difficult to target management actions at diseased animals, as an animal’s disease status is often unobservable prior to post-mortem testing (Lanfranchi et al. 2003). Rather, Horan et al. (forthcoming) find that directing controls at the agricultural sector can be a better targeted approach to reducing damages.

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1 Zivin et al. (1999) and Bicknell et al. (1999) examine private incentives when the externality impacts a single farmer. In this case the public and private incentives would be equivalent.
Few studies have examined private incentives for managing human-wildlife conflicts (e.g., Horan and Bulte 2004; Rondeau and Bulte 2007). These studies investigate private responses to various (sub-optimal) policy mechanisms, such as trade bans on wildlife products and compensation schemes for wildlife damages, and show how such policies can reduce welfare and hurt wildlife populations. In particular, Rondeau and Bulte (2007) showed that compensation can in some cases even trigger wildlife extinction.

Herein we investigate private responses to policies that have been proposed to confront a human-wildlife conflict that likely emerged as a result of a management regime designed to address an earlier human-wildlife conflict. Specifically, Wyoming elk had been eating cattle forage during winter, causing damage to farmers. Instead of fencing off the land, the farmers instead were able to get the public sector to set up elk feed grounds across the state. This has done two things. First, it has reduced elk predation on cattle forage. Second, it has 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.

After losing brucellosis-free status in 2004, the cattle sector was required to adopt a costly vaccination policy (Koontz and Loomis 2005) and infected herds were to be destroyed (depopulated). In addition, there is pressure to either reduce or eliminate supplemental feeding of elk, and to reduce elk densities (Kreeger 2002). Formal modeling and analysis of this problem has been limited. Dobson and Meagher (1996) propose a simple SIR (susceptible-infected-resistant) epidemiological model to describe the population and disease dynamics of brucellosis among bison and elk in the Greater
Yellowstone Area (GYA). They found that the population threshold for brucellosis establishment – the population below which the disease will begin to vanish – is quite low. This makes it very hard to eradicate brucellosis from wild elk populations. However, their model only considered the population dynamics without human management choices and without interactions between wildlife and livestock.

Does it make sense to adopt stringent controls in one sector without consideration of what is going on in the other sector? 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 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 feed ground 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 in such a way that risks of infection are a function of livestock disease management choices, and economic choices are, in turn, a function of the risk of infection.

We examine population and disease dynamics under several different 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. We also examine livestock management when there is little-to-no consideration given to the risk posed by elk. In practice, the policies and proposals to address elk have been considered separately of the behavioral responses of farmers, with
many livestock advocacy groups are pushing for an elimination of the feeding grounds (Smith 2001). Our findings suggest that the feedbacks between jointly determined disease dynamics and decentralized economic behavior matters when choosing among various policy approaches.

**Background**

Brucellosis is a bacterial disease that causes cattle, elk, and bison to abort their calves. It also reduces birth rates and limits the milk-making capacity in livestock. Brucellosis is transmitted through sexual contact and direct contact with infected birthing materials. It is one of the most infectious bacterial agents in cattle, with the potential to cause large-scale outbreaks of abortion (Wyoming Brucellosis Coordination Team [WBCT] 2005).

Brucellosis has caused devastating losses to farmers in the United States over the last century. The USDA and animal industry embarked on a plan to eradicate brucellosis in the United States in the 1930’s. This effort has required 70 years and an estimated $3.5 billion in state, federal, and private funds (WBCT 2005). The only known focus of Brucella abortus infection left in the nation is in bison and elk in the GYA.

Wyoming is at special risk due to the large wildlife reservoir of brucellosis. Brucellosis was identified in Wyoming cattle in the late 1800’s, and in elk as early as 1917. It is now endemic in the elk and wild bison. Elk on winter feed grounds 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 feed grounds. In addition, the National Elk Refuge is managed by the U.S Fish and Wildlife Services. These feed grounds are largely concentrated in the western
part of Wyoming, and they are considered a significant risk factor to Wyoming’s cattle herd [WBCT 2005].

Brucellosis infection of cattle has serious economic consequences for the cattle industry. Not only does it cause losses from abortion, but it also creates marketing limitations and farmers must pay to test cattle to reassure trading partners that the product is clean. When infection occurs, the disease can quickly spread though the herd, causing significant productivity losses. Upon detection of brucellosis, depopulation of the entire herd is often required. According to USDA, 112 brucellosis-infected cattle herds were depopulated in the U.S. at a cost (loss) of $3.06 million in FY 1996.

Wyoming lost its brucellosis-free status in February 2004 because of a brucellosis outbreak that was likely due to transmission from elk wintering on a feed ground. This loss of brucellosis-free status has great impacts on the Wyoming livestock industry. Under the rules of the brucellosis eradication program, all test-eligible cattle must be tested and demonstrated to be free of brucellosis within 30 days prior to interstate movement or change of ownership. This costs $3-10 per head, which is a significant added cost to producers. Producers in Wyoming, Idaho, and Montana will continue to have to vaccinate their cattle and participate in surveillance programs, because of the reservoir of brucellosis in elk and bison of the GYA. These activities are expensive for producers, and are not necessary in states where there is no brucellosis.

There are two vaccines available for cattle, Strain 19 and RB-51. Most herds are RB-51 vaccinated because cattle vaccinated with Strain 19 have a higher occurrence of testing false positive for brucellosis. However, neither vaccine is 100% effective. Vaccination alone is only 65-70% effective in protecting animals from the disease.
Vaccination of elk on State and Federal winter elk feed grounds is being done by the Wyoming Game and Fish Department using ballistic “bio-bullets”. Yet the vaccine is not preventing infection; it only protects about 30% (Dean and et al. 2004) of the elk from abortion.

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 feed grounds on this transmission. Politically, there is a lack of public or social awareness of the complications associated with developing solutions, and there are different perspectives about what should be done with elk feed grounds. Artificial feeding is a two-edged sword. On the one hand, it increases the probability that the elk and bison congregate, and therefore increases the transmission rate. When the wildlife move out of the feeding grounds, they are more likely to transmit brucellosis to the cattle. On the other hand, the feed grounds increase winter survivals of the wildlife and, to some extent, prevent the wildlife co-mingling with livestock by gathering them together. In addition, hunting groups want to keep elk populations as high as possible to provide the best available hunting, and many local economies rely on elk-related hunting and tourism revenues.

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

2 Although brucellosis in bison is a focus in the Yellowstone Area, there are relatively few bison congregating in the elk feeding grounds, 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.
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 that they incorporate human choices. Farmer choices are endogenously-determined, as 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 population model

The elk population \(X\) consists of three sub-populations: susceptible, \(X_s\), infected, \(X_i\), and resistant, \(X_r\). Changes in these population levels are based on Dobson and Meagher’s (1996) SIR model, adjusted for harvesting and feeding. Specifically, the change in the susceptible stock of elk is

\[
\dot{X}_s = [(a - M)X_s + a\eta X_i (1 - \zeta) + aX_r](1 - \frac{X}{K})
- B_{cc}X_sX_i - B_{ce}X_sIn + \alpha X_r - h \frac{X_s}{X}
\]

(1)

The first term of the right-hand-side (RHS) represents the net natural growth of susceptible elk, assuming logistic growth. The expression in brackets represents births less mortality prior to the impacts of density-dependent competition. The birth rate is \(a\),
\[ \zeta \] is the proportion of infected females that produce infected offspring, and \( \eta \) is the reduction of fecundity in infected animals. Natural mortality is given by \( M \). The second and third terms represent the number of elk being infected by elk and livestock, respectively. Following Dobson and Meagher (1996), elk-to-elk transmission is modeled as density-dependent with \( B_{ee} \) representing the rate of infectious transmission among elk. Cattle-to-elk transmission is also density-dependent, with \( B_{ej} \) being the rate of infectious transmission between cattle and elk. The expression \( I_n \) represents total infected cattle, where \( I \) is the proportion of infected farms and \( n \) is the number of cattle farms. The fourth 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 \( \sigma \).

The final term represents the decrease in susceptible elk due to hunting. Hunting is non-selective with respect to health status, since it is often not possible to identify infected animals until we capture or kill them (Lanfranchi et al 1993; Williams et al. 2003). We therefore assume only the total harvest, \( h \), is chosen, and that the harvest from each stock depends on the proportion of animal in that stock relative to the aggregate population. That is \( h_j = hX_j / X \), where \( h_j \) denotes the harvest from subpopulation \( j \) \((j=I,S,R)\).

The change in the infected stock of the elk is

\[
\dot{X}_I = [a_\zeta X_I - MX_I] (1 - \frac{X}{K}) + B_{ee} X_S X_I + B_{ej} X_S nI - AX_I - \delta X_I - h \frac{X_I}{X}
\]

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

The change in the resistant stock of elk is

\[
\dot{X}_R = \delta X_i - MX_R (1 - \frac{X}{K}) - \sigma X_R - h \frac{X_R}{X}
\]

The first term of the right-hand-side (RHS) represents the number of infected elk that become recovered. The second and third RHS terms reflect the decrease in the number of resistant elk due to mortality and resistance loss. 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 $M(f) = m(1 - wf)$, where $m$ is the natural mortality rate and $w$ is a parameter that represents the effect that feeding has on reducing mortality. Virulence is also declining in feeding and is modeled by $A(f) = \alpha(1 - wf)$, where $\alpha$ is the natural virulence rate. The elk-to-elk transmission rate is increasing in $f$, as the feeding activity causes animals to congregate in large densities. We model this 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. Finally, the cattle-to-elk transmission rate is decreasing in $f$, as the feeding activity causes fewer cross-species contacts. This transmission rate is $B_{ce}(f) = \beta_{ce} (1 - \mu_{ce} f)$, where $\beta_{ce}$ is the natural
cross-species transmission rate and $\mu_{ec}$ is a parameter reflecting how feeding increases this rate.

**A metapopulation model for farms**

A metapopulation disease model (Levins 1969) is adopted to model livestock disease dynamics. We assume there are $n$ homogeneous farms are dispersed across the region, and disease transmission occurs based on indirect contact among farms (e.g., when animals are on public grazing areas) and between farms and wildlife (e.g., on farmlands or public grazing areas) according to a simple SIR model of disease. Each farm can be in one of four states at any point in time: susceptible (non-infected), infected, resistant, or empty. 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

$$
\dot{s} = \epsilon e - vs - \beta_{cc}i(1-\nu)s/N^i - \beta_{ce}X_{1}((1-\nu)s/N)^i + (\chi + \tau)r
$$

The first RHS term represents the number of newly-repopulated farms, where repopulation occurs at the rate $\epsilon$. The second RHS term is the number of farms that become resistant due to vaccination, where $\nu$ 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. We assume frequency dependent cattle-to-cattle and cattle-to-elk transmission, which is commonly applied in metapopulation models (Hess 1991; McCallum and Dobson 2002) and is commonly used to model transmission of sexually transmitted and indirectly-transmitted diseases (Dobson 2004). Cattle-to-cattle transmission is given by $\beta_{cc}i([1-\nu]s/n)^i$, where $\beta_{cc}$ is the disease transmission
parameter. The expression \( ([1 - \nu]s/n)^\gamma \) is the susceptibility function (Barlow 1995), where \([1-\nu]s\) represents the number of susceptible cattle after vaccination, and \(\gamma\) is a parameter. For diseases that do not result in high prevalence, which is the case for brucellosis at the herd level, Barlow (1995) says \(\gamma\) should be set at a high level such as \(\gamma = 10\). An analogous expression is used to model elk-to-cattle transmission. The last term represents the number of farms losing resistance. This occurs naturally at the rate \(\chi\), and it is enhanced by the rate at which new (non-resistant) 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

\[
\dot{i} = \beta_{cc} i ([1 - \nu]s/n)^\gamma + \beta_{cc} X_i ([1 - \nu]s/n)^\gamma - qi
\]

where the first two terms denote newly infected farms, as described above, and the last term represents depopulation of infected farms. Depopulation is assumed to occur at the rate \(q\).

The change in the number of resistant farms is

\[
\dot{r} = vs - (\chi + \tau)r
\]

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

\[
\dot{e} = qi - \varepsilon e
\]

It is customary in metapopulation models to re-write the dynamic system in terms of proportions of farms in each state (e.g., Hess 1991; McCallum and Dobson 2004). That is, define \(S = s/n\) as the 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) can be rewritten as

\begin{align}
\dot{S} &= \varepsilon E - vS - \beta_{ce} I (1 - v) S^\gamma - (\beta_{ce} / n) X_I (1 - v) S^\gamma + (\chi + \tau) R \\
\dot{I} &= \beta_{ce} I (1 - v) S^\gamma + (\beta_{ce} / n) X_I (1 - v) S^\gamma - qI \\
\dot{R} &= vS - (\chi + \tau) R \\
\dot{E} &= qI - \varepsilon E.
\end{align}

Typical metapopulation models of disease transmission treat vaccination $v$ as an exogenous behavioral parameter. In contrast, we take vaccination to be endogenous. Next we develop the behavioral dynamics that govern the economic strategies $v$ which are made in response to current disease risks. In turn, the economic choices $v$ 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**

The farm decision model follows that of Hennessy (2007), which is based on Shapiro and Stiglitz (1984). Individual farmer $z$ ($z=1\ldots,n$) chooses his or her own vaccination investment strategy, denoted as $v_z$. The strategy is a discrete choice for the farmer: $v_z=1$ implies vaccination, $v_z=0$ implies no vaccination. The individual strategies $v_z$ are distinct from $v$, which as previously been defined is the proportion of farms adopting vaccination at any point in time.

A farm in a given disease state receives an expected flow of income associated with its current disease state, taking into account the possibility that it will transition to a new disease state at some time in the future. Denote farm $z$’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.\(^3\) A farm’s vaccination strategy, as well as current infection levels, will influence the likelihood the farm transitions from one state to another.

Following Shapiro and Stiglitz (1984), denote $Y_{zS}^{vz}$ to be the expected lifetime income of the $z$th farmer who is currently in the susceptible state and choosing action $v_z$, and denote $Y_{zj}$ to be the expected lifetime income of the $z$th farmer who is currently in state $j=I,R,E$. The probability of transitioning from state $j$ to state $k$ is $P_{zjk}$. As we show below, this value will change over time as infection risks change. We assume farmers are forward looking, but that they 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 $P_{zjk}$ as fixed. Assuming a discount rate of $\rho$, the fundamental asset equations for susceptible, infected, resistant and empty farms are:

\begin{align*}
(12) \quad \rho Y_{zS}^{vz} &= \pi - [c / \kappa] v_z + P_{zSI} [Y_{zI} - Y_{zS}] + P_{zSR} [Y_{zR} - Y_{zS}] \\
(13) \quad \rho Y_{zI} &= \pi - b + P_{zIE} [Y_{zE} - Y_{zI}] \\
(14) \quad \rho Y_{zR} &= \pi + P_{zRS} [Y_{zS} - Y_{zR}] \\
(15) \quad \rho Y_{zE} &= P_{zES} [Y_{zS} - Y_{zE}] 
\end{align*}

\(^3\) 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 are assuming the loss is sufficiently small so as not to worry about it.
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, \( \pi - [c / \kappa]v_z \), and the “expected capital loss that would arise were the state to change” (Hennessy 2007) from susceptible to infected, \( P_{zs}[Y_{zl} - Y_{zs}] + P_{zs}[Y_{zr} - Y_{zs}] \).

Equations (13)-(15) have similar interpretations. These equations can then be solved simultaneously for \( Y_{zs}^0, Y_{zl}^0, Y_{zr}^0 \) and \( Y_{ze}^0 \) as functions of the behavioral strategies, the states of the world, and economic and epidemiological parameters.

The probability that producer \( z \) transitions from the susceptible state to the infected state can be obtained from the epidemiological model as

\[
P_{zs} = \beta q(1 - v) S(I - (1 - v) S^{l-1}).
\]

The probability of transitioning from the susceptible state to the resistant state is simply the vaccination strategy: \( P_{zsr} = v_z \). The probability of transitioning from the resistant state to the susceptible state is \( 1 - (1 - \frac{\beta}{n})X(I - (1 - v) S^{l-1}) \).

The probability of transitioning from the infected to the empty state is \( P_{zIE} = q \). Finally, the probability of transitioning from the empty state to the susceptible state is \( P_{zES} = \varepsilon \).

The marginal return to vaccination for a representative farm at an individual point in time is given by \( \frac{\partial Y_{zs}}{\partial v_z} \) and the optimal vaccination strategy \( v_z^*(v, I, S, R) \) is the solution to \( \frac{\partial Y_{zs}}{\partial v_z} = 0 \). Vaccination investment dynamics are derived from taking the time derivative of \( v_z^*(v, I, S, R) \). However, there is no closed form solution to \( v_z^*(v, I, S, R) \). Numerical analysis suggests that \( Y_{zs}(v_z, v, I, S, R) \) is monotonic and approximately linear in \( v_z \) for a wide range of feasible values of \( v, I, S, R \) and model parameters, therefore we develop a linear approximation of \( Y_{zs} \) using a linear
combination of the known endpoints \( Y_{zS}^V(v, I, S) = Y_{zS} \mid_{v_z=1} = Y_{zS} (1, v, I, S) \) and 
\[
Y_{zS}^{NV} (v, I, S) = Y_{zS} \mid_{v_z=0} = Y_{zS} (0, v, I, S)
\]
to approximate \( Y_{zS} \) at each point in time according to 
\[
\bar{Y}_{zS} = v_z Y_{zS}^V + (1 - v_z) Y_{zS}^{NV} .
\]
We then use replicator dynamics to model inter-temporal changes in behavior.

**Behavioral dynamics**

We now introduce the *replicator dynamics* (Fudenberg and Levine 1998; Rice 2004; Weibull 1995) to analysis the change in the aggregate frequency of adoption of vaccination. 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 outweigh average net benefits associated with the current frequency of adoption. We focus on benefits during the susceptible state, since that is when the vaccination decision is made, and we assume a symmetric equilibrium so that \( Y_{zS}^v = Y_{zS}^v \). 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, 
\[
Y_{zS} = v Y_{zS}^{v=1} + (1 - v) Y_{zS}^{v=0} ;
\]

\[
(16) \quad \frac{\dot{v}}{v} = \alpha [Y_{zS}^{v=1} - Y_{zS}] \Rightarrow \dot{v} = \alpha v (1 - v)[Y_{zS}^{v=1} - Y_{zS}^{v=0}],
\]
where \( \alpha > 0 \) is a speed of adjustment parameter. 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.
So far in our analysis of vaccination dynamics, as in previous analyses (Hennessy 2007), we have held $I$, $S$, and $R$ fixed. But $I$, $S$, and $R$ are not fixed in the joint dynamic system, especially when we consider the effect of elk population together. This means the probability of becoming infected is non-stationary, therefore assuming stationarity may develop misleading policy recommendations. It is important to consider the interconnectedness of disease and behavioral dynamics. Thus, in the example below, we will consider the behavior 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 2004 winter population estimated at 13,500. 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$^2$.

Grand Teton National Park (GTNP) and the National Elk Refuge (NER) comprise most of the valley floor north of the town of Jackson. There are also three state-operated elk feedgrounds within the Jackson EHU: Alkali, Patrol Cabin, and Fish Creek. Elk also receive supplemental winter feed on the NER, which is managed by the U.S Fish and Wildlife Services, and the WGFD operated state-operated elk feedgrounds. 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 is a dynamic and migratory population that 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, there are four allotments (Burro Hill, Pacific Creek-GTNP and BTN, Upper Gros Ventre, Big Cow Creek) that overlap spatially with designated elk parturition ranges (WGFD, 2007). The four allotments are located in Teton and Sublette County. In 2002, there are around 190 farms with 57,010 cattle and calves in the two counties (National Agricultural Statistics Service).

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, 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 analyze analytically. 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 now the regulations are lifted. The initial level of vaccination is therefore \( v(0) = 0.99 \). 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. All parameters are defined in Table 1.

**The cattle sector only: no infection risk by elk**

We first analyze the cattle sector without effects from the elk sector. This represents a brucellosis problem where there is no infection risk imposed by wildlife, which is the way most livestock disease problems are modeled and which is probably realistic in many cases. Given our starting values, the proportion of vaccination and disease states fluctuate erratically following a path consistent with dynamic chaos over a bounded range of values for each individual state (Figure 2).\(^4\) When infection is very low, farmer’s incentive to vaccinate will decrease. But then infection levels increase when vaccination becomes very low, creating incentives for some farmers to vaccinate again. This pattern repeats over and over again, with infection rates rising to around 3.2 percent every ten years or so, followed by vaccination by around 29 percent of the herds.

**The joint model**

The brucellosis problem in Wyoming, compared to the problem in the cattle-sector-only model, is much more complicated. 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

\(^4\) The chaotic nature of the solution is better illustrated visually over a much longer time interval, such as 600 years. In that case it is more readily apparent that the fluctuations are not a form of periodic cyclical behavior.
ways in our model: (i) reduce the elk, and (ii) 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 impacts of the feedgrounds on transmission, but feeding is important in other disease systems such as bovine tuberculosis among Michigan white-tailed deer (e.g., Hickling 2002; O’Brien et al. 2002). In Wyoming, 80% of the elk winter in the feedgrounds (Dean and et al. 2004), and it is believed that feedground management plays an important role in the disease dynamics (Dean and et al. 2004, Smith 2001).

We will consider 4 cases of different combination of elk disease management policy, that is (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); (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 behavior dynamics in the cattle sector. We also analyze economic impacts to the cattle sector. We do not analyze economic impacts to the elk sector, though these would be important. Hunters will generally benefit from a larger supply of elk, and so feeding and elk conservation can be valuable for its non-disease impacts to the hunting sector.

*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 at a level of 740 kg/km². We restrict our attention to this feeding level, but note that our qualitative results show that different feeding levels have similar effect on the disease and behavior dynamics.

The simulation results are presented in Figure 2 and Table 2. 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. But, considering the large elk population, the steady state number of infected animals (4,666 elk) and also the disease prevalence rate (8.9%) are actually quite small relative to scenarios (b)-(d) – particularly scenario (c), in which there are also no hunting controls. This result arises here 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.

For the cattle sector, the disease and behavior dynamics in this case are quite different from the cattle-only model. Instead of exhibiting dynamic chaos, the constant force of infection from the elk herd causes the proportion of vaccination and infected states converge to steady states after some fluctuation (Figure 2). The proportion of vaccination converges to 28.7 percent after less than 30 years. Of all the scenarios, this scenario is tied with scenario (b) for having the smallest vaccination rate. 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 existence of the feedgrounds helps 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 the other scenarios. Finally, it is perhaps ironic that net benefits to the cattle sector are greatest in this scenario (though not by much, as net benefits do not vary significantly among the scenarios analyzed).

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. We set up the number of hunting every year as the total population minus the objective of 11,029. The total elk population under this approach still exceeds 11,029 due to growth, which is assumed to occur after the harvest. No hunting takes place in years when there are less than 11,029 elk.

The simulation results (Table 2; Figure 3) show that the number of infected elk decrease when hunting occurs, yet the number of infected elk in the population does not significantly change relative to scenario (a). 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 (1996)’s 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. Indeed, prevalence levels have actually increased to 33.4 percent, which is
realistic. With similar numbers of infected elk, the results in the cattle sector are almost identical to that of scenario (a).

*Scenario (c): Elk feeding controls only*

Now how about closing all the elk feedgrounds? This reduces infectious contacts among elk and increases elk mortality, but it may increase infectious contacts with cattle. This is because, without feeding, elk will spend less time around the feedgrounds and more time around cattle areas where food may be more plentiful. Indeed, the feedgrounds were originally developed due to elk predating on cattle forage.

The number of infected elk is the largest in this scenario relative to the three other scenarios (though prevalence, at 24.1 percent, has been reduced somewhat relative to scenario (b), due to a smaller overall population; see Table 2; Figure 4). The rate of infectious contacts between elk is reduced due to a lack of feeding, but there are more susceptible elk to become infected and this latter effect dominates. The number of susceptible elk has increased because increased mortality among all sub-populations reduces the aggregate elk population and therefore eases the density-dependent effects of resource competition among elk. The result is increased births of susceptible elk.

The large number of infected elk, along with no feeding which creates more contacts with cattle, actually creates greatest infection risks for the cattle sector. Farmers therefore end up with largest vaccination rates, though the percent of infected herds is actually less than in scenarios (a) and (b). The combination of large vaccination levels and low infection levels suggests that vaccination is highly responsive to risks from elk. Moreover, comparison of farmers’ net benefits indicates that, relative to scenarios (a) and
(b), the increased vaccination costs are almost completely offset by decreased infection costs.

**Scenario (d): Elk population and feeding controls**

The last case involves the most stringent set of elk controls: both population and feeding controls. The simulation results (Table 2; Figure 5) show that the use of both controls results in the smallest number of infected elk among the four scenarios (though it has the highest prevalence rate, at 34.3 percent, because this scenario contains the lowest population overall).

Though there are few infected elk in this scenario, the lack of feeding leads to more contacts between elk and cattle. The result is sizable infection risks to the cattle sector. Farmers end up with significant vaccination rates, even though the percentage of infected herds is actually less than in scenarios (a) and (b). Finally, as with scenario (c), comparison of farmers’ net benefits indicates that, relative to scenarios (a) and (b), the increased vaccination costs are almost completely offset by decreased infection costs.

**Discussion**

We have examined the impacts of various proposed elk brucellosis management strategies on elk disease dynamics, and on farmers’ private vaccination behaviors and associated disease dynamics in the cattle sector. Overall, our results contradict the conventional wisdom that farmers would benefit from more stringent disease controls in the elk sector. We find that farmers do experience lower herd-level infection rates when more stringent elk controls are implemented. But this is primarily due to increased
vaccination by farmers in response to increased risks to cattle that result from elk spending more time on public grazing areas and farmlands as feedgrounds are closed. This is surely not something expected by the livestock advocacy groups who are currently pushing for an elimination of the feeding grounds. Moreover, farmers are really no better off economically under any particular scenario once the changes in disease risks and the farmer responses are accounted for. These results suggest that the most appropriate policy for disease control in elk may be that policy that provides the largest net benefits to the elk sector. More research on what those benefits might be is needed.
References


Fudenberg and Levine 1996, Theory of Learning in Games, MIT University Press.


Kreeger (Editor), 2002. Brucellosis in Elk and Bison in the Greater Yellowstone Area, Wyoming Game and Fish Department for the Greater Yellowstone Interagency Brucellosis Committee.


Smith, B. L. 2005. Disease and winter feeding of elk and bison: A review and recommendations pertinent to the Jackson bison and elk management plan and environmental impact statement Report prepared for the Greater Yellowstone Coalition.


Wyoming Brucellosis Coordination Team. Report & Recommendation, January 11th, 2005


Figure 1: Cattle Only Model Simulation Results

"Note: susceptible: blue, infected: Maroon, resistant: Yellow, vaccination: Black "

Figure 2: Joint Cattle-Elk Model, Scenario (a): No Elk Disease Management

Left: Cattle behavior and disease dynamics, Right: Elk disease dynamics (same for Figures 3-5)
Figure 3. Joint Cattle-Elk Model, Scenario (b): Elk Population Controls Only

Figure 4: Joint Cattle-Elk Model, Scenario (c): Elk Feeding Controls only

Figure 5. Joint Cattle-Elk Model, Scenario (d): Elk Population and Feeding Controls
Table 1. Description of parameters and data

<table>
<thead>
<tr>
<th>Description</th>
<th>Parameter</th>
<th>Fixed Value for Parameter or Starting Value</th>
<th>Reference or Explanations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Discount rate</td>
<td>$\rho$</td>
<td>0.05</td>
<td>Assumption</td>
</tr>
<tr>
<td>Cost of infection</td>
<td>$b$</td>
<td>600</td>
<td>Assumption</td>
</tr>
<tr>
<td></td>
<td>$c$</td>
<td>300</td>
<td>Assume the cost of vaccination is $10 per head; the average size of farm is 300.</td>
</tr>
<tr>
<td>Cost of vaccination</td>
<td>$\pi$</td>
<td>11,067</td>
<td>The net cash income in average farm is $11906 in Sublette, and $10228 in Teton, so we use an average number for approximation (NASS)</td>
</tr>
<tr>
<td>Instantaneous income flow at each point in time</td>
<td>$\chi$</td>
<td>0.01</td>
<td>The probability that a resistant cattle will loss resistance</td>
</tr>
<tr>
<td>Rate of loss of resistance for cattle</td>
<td>$\varepsilon$</td>
<td>1</td>
<td>The probability that a depopulated farm will buy new susceptible cattle</td>
</tr>
<tr>
<td>Transition rate from empty (depopulated) to susceptible</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Effectiveness of the vaccination</td>
<td>$\kappa$</td>
<td>0.7</td>
<td>Wyoming Department of Administration and Information (2004)</td>
</tr>
<tr>
<td>Disease transmission parameter between the livestock,</td>
<td>$\beta_0$</td>
<td>2</td>
<td>Dobson and Meagher (1996)</td>
</tr>
<tr>
<td></td>
<td>$\beta_1$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Disease transmission parameter from elk to cattle</td>
<td>$\beta$</td>
<td>$w^{-0.26}$, where $w$ is the mass of host species (Dobson and Meagher 1996)</td>
<td></td>
</tr>
<tr>
<td>Rate of depopulation</td>
<td>$q$</td>
<td>0.5</td>
<td>Assumption</td>
</tr>
<tr>
<td>Speed of adjustment parameter in the vaccination strategy replicator dynamics</td>
<td>$\alpha$</td>
<td>$1 \times 10^{-5}$</td>
<td>Assumption</td>
</tr>
<tr>
<td>Parameter</td>
<td>Symbol</td>
<td>Value</td>
<td>Description</td>
</tr>
<tr>
<td>--------------------------------------------------------------------------</td>
<td>--------</td>
<td>-------</td>
<td>-------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Cattle “turnover rate”</td>
<td>( \tau )</td>
<td>0.22</td>
<td>Proportion of new susceptible cattle purchased every period</td>
</tr>
<tr>
<td>Rate of loss of resistance for elk</td>
<td>( \sigma )</td>
<td>0.01</td>
<td>Dobson and Meagher (1996)</td>
</tr>
<tr>
<td>Elk birth rate</td>
<td>( a )</td>
<td>0.25</td>
<td>Dobson and Meagher (1996)</td>
</tr>
<tr>
<td>Natural mortality rate of elk</td>
<td>( m )</td>
<td>0.15</td>
<td>Dobson and Meagher (1996)</td>
</tr>
<tr>
<td>Proportion of infected female elk that produce infected offspring</td>
<td>( \zeta )</td>
<td>0.9</td>
<td>Dobson and Meagher (1996)</td>
</tr>
<tr>
<td>Reduction of fecundity in infected elk</td>
<td>( \eta )</td>
<td>0.5</td>
<td>Dobson and Meagher (1996)</td>
</tr>
<tr>
<td>Rate of infectious transmission among elk</td>
<td>( \beta_2 )</td>
<td>0.002</td>
<td>Assume density dependent transmission, ( \beta = 0.0247w^{0.44} ), where ( w ) is the mass of host species (Dobson and Meagher 1996)</td>
</tr>
<tr>
<td>Rate of infectious transmission from cattle and elk</td>
<td>( \beta_3 )</td>
<td>0.002</td>
<td>Assume density dependent transmission, ( \beta = 0.0247w^{0.44} ), where ( w ) is the mass of host species (Dobson and Meagher 1996)</td>
</tr>
<tr>
<td>Recovery rate of infected elk</td>
<td>( \delta )</td>
<td>0.5</td>
<td>Dobson and Meagher (1996)</td>
</tr>
<tr>
<td>Virulence rate of elk</td>
<td>( \alpha )</td>
<td>0.005</td>
<td>Dobson and Meagher (1996)</td>
</tr>
<tr>
<td>Feeding effect on mortality rate</td>
<td>( \omega )</td>
<td>0.001</td>
<td>Assume ( M = (1 - \omega f)M ), calibrated using natural mortality rate and an observed mortality rate 0.037 (Kreeger et.al. 2002).</td>
</tr>
<tr>
<td>Aggregated harvest</td>
<td>( h )</td>
<td>0</td>
<td>Dobson and Meagher (1996)</td>
</tr>
<tr>
<td>Number of elk in Jackson herd</td>
<td>( X(0) )</td>
<td>11853</td>
<td>WGFD, starting value</td>
</tr>
<tr>
<td>Elk natural resistant rate</td>
<td>( X_{r}(0) / X(0) )</td>
<td>0.2</td>
<td>Kreeger et.al. (2002)</td>
</tr>
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<td>Initial elk infection level</td>
<td>( X_{i}(0) / X(0) )</td>
<td>0.3</td>
<td>Kreeger et.al. (2002)</td>
</tr>
<tr>
<td>Resistant proportion farms</td>
<td>( R(0) )</td>
<td>0.1</td>
<td>Assumption</td>
</tr>
<tr>
<td>Empty proportion farms</td>
<td>( E(0)=qP(0) )</td>
<td>0.25</td>
<td>Assumption</td>
</tr>
<tr>
<td>Infected proportion farms</td>
<td>( P(0) )</td>
<td>0.02</td>
<td>Assumption</td>
</tr>
<tr>
<td>Susceptible</td>
<td>( S(0)=1- R(0)- )</td>
<td>0.8</td>
<td>Assumption</td>
</tr>
<tr>
<td><strong>proportion farms</strong></td>
<td>( E(0) - P(0) )</td>
<td>( v(0) )</td>
<td>0.99</td>
</tr>
<tr>
<td>----------------------</td>
<td>------------------</td>
<td>-----------</td>
<td>------</td>
</tr>
<tr>
<td>Aggregate rate of vaccination in the population</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Steady State</td>
<td>Policy Scenario</td>
<td>(a) No elk disease management</td>
<td>(b) Elk population controls only</td>
</tr>
<tr>
<td>-------------------</td>
<td>---------------------------------------------</td>
<td>-------------------------------</td>
<td>---------------------------------</td>
</tr>
<tr>
<td>Vaccination (v)*</td>
<td>28.7</td>
<td>28.7</td>
<td>42.4</td>
</tr>
<tr>
<td>Susceptible Cattle (S)*</td>
<td>45.4</td>
<td>45.5</td>
<td>36.0</td>
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<tr>
<td>Infected Cattle (I)*</td>
<td>0.04</td>
<td>0.04</td>
<td>0.01</td>
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<tr>
<td>Resistant Cattle (R)*</td>
<td>54.4</td>
<td>54.4</td>
<td>63.9</td>
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<tr>
<td>Susceptible Elk (X_s)</td>
<td>134</td>
<td>145</td>
<td>223</td>
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<tr>
<td>Infected Elk (X_i)</td>
<td>4,666</td>
<td>4,633</td>
<td>8,508</td>
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<tr>
<td>Resistant Elk (X_r)</td>
<td>47,616</td>
<td>9,078</td>
<td>26,608</td>
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<tr>
<td>Farmers’ Net Benefits (Present Value, Millions USD)</td>
<td>39.06</td>
<td>39.05</td>
<td>39.04</td>
</tr>
</tbody>
</table>

*Expressed as a percentage of farms.