DISEASE AND BEHAVIOR DYNAMICS FOR BRUCELLOSIS IN ELK AND CATTLE IN THE GREATER YELLOWSTONE AREA

By

Fang Xie

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ABSTRACT

DISEASE AND BEHAVIOR DYNAMICS FOR BRUCELLOSIS IN ELK AND
CATLLE IN THE GREATER YELLOWSTONE AREA
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This paper investigates 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. The artificial elk feeding which is intended to conserve wildlife and reduce elk predation on cattle forage, now led to the emergence of brucellosis in elk and has allowed it to become endemic, in turn imposing great risk to the 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 infection between livestock and the Jackson elk herd in Wyoming. 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. Our findings suggest that the feedbacks between jointly determined disease dynamics and decentralized economic behavior matter when choosing among various policy approaches.

ACKNOWLEDGEMENTS

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

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).[1] 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 through 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.[2] 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

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

$$
\begin{aligned}
\dot{X}_S &= [(a-M)X_S + a\eta X_I(1-\zeta)) + aX_R](1-\frac{X}{K}) \\
&\quad - B_{ee}X_S X_I - B_{ec}X_S In + \sigma X_R - h\frac{X_S}{X}
\end{aligned}
$$
(1)

---

[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.

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, ζ is the proportion of infected females that produce infected offspring, and η 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_{ec} being the rate of infectious transmission between cattle and elk. The expression In 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 σ.

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

$$(2) \quad \dot{X}_I = [a\eta\zeta X_I - MX_I](1 - \frac{X}{K}) + B_{ee}X_S X_I + B_{ec}X_S nI - AX_I - \delta X_I - h\frac{X_I}{X}$$

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 δ 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

$$(3) \quad \dot{X}_R = \delta X_I - MX_R(1 - \frac{X}{K}) - \sigma X_R - h\frac{X_R}{X}$$

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 α 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 β_{ee} is the natural transmission rate

and μ_{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 $B_{ec}(f) = \beta_{ec}(1 - \mu_{ec}f)$, where, β_{ec} is the natural

cross-species transmission rate and μ_{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

(4) $\quad \dot{s} = \varepsilon e - \nu s - \beta_{cc}i([1 - \nu]s/n)^{\gamma} - (\beta_{ce}/\psi)X_{I}([1 - \nu]s/n)^{\gamma} + (\chi + \tau)r$

The first RHS term represents the number of newly-repopulated farms, where

repopulation occurs at the rate ε. The second RHS term is the number of farms that

become resistant due to vaccination, where ν 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 $\beta_{cc}i([1-v]s/n)^{\gamma}$, where β_{cc} is the disease transmission parameter. The expression $([1-v]s/n)^{\gamma}$ is the susceptibility function (Barlow 1995), where $[1\text{-}v]s$ represents the number of susceptible cattle after vaccination, and γ 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 γ should be set at a high level such as $\gamma =$ 10. An analogous expression is used to model elk-to-cattle transmission, though the elk-to-cattle transmission parameter β_{ce} must be divided by the average number of cattle in each farm, ψ, since we are measuring the number of farms (not animals) becoming infected due to contact with elk, which are measured in animal units. The last term represents the number of farms losing resistance. This occurs naturally at the rate χ, 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), τ.

The change in the number of infected farms over time is

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

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

(6) $\dot{r} = vs - (\chi + \tau)r$

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

(7) $\quad \dot{e} = qi - \varepsilon e$

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

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

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

(10) $\quad \dot{R} = vS - (\chi+\tau)R$

(11) $\quad \dot{E} = qI - \varepsilon E$.

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 be 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 π, with profits being zero during the empty state. Susceptible farms that vaccinate will also expect to incur vaccination costs of c/κ, where c is the cost of vaccinating an average herd at one time and κ 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 $Y_{zS}^{v_z}$ to be the expected lifetime income of the zth 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 zth 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

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[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.

on into the future, thereby taking P_{zjk} as fixed. Assuming a discount rate of ρ, the fundamental asset equations for susceptible, infected, resistant and empty farms are:

$$(12) \quad \rho Y_{zS}^{v_z} = \pi - [c/\kappa]v_z + P_{zSI}[Y_{zI}^{v_z} - Y_{zS}^{v_z}] + P_{zSR}^{v_z}[Y_{zR}^{v_z} - Y_{zS}^{v_z}]$$

$$(13) \quad \rho Y_{zI}^{v_z} = \pi - b + P_{zIE}[Y_{zE}^{v_z} - Y_{zI}^{v_z}]$$

$$(14) \quad \rho Y_{zR}^{v_z} = \pi + P_{zRS}[Y_{zS}^{v_z} - Y_{zR}^{v_z}]$$

$$(15) \quad \rho Y_{zE}^{v_z} = -d + P_{zES}[Y_{zS}^{v_z} - Y_{zE}^{v_z}]$$

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, $P_{zSI}[Y_{zI} - Y_{zS}] + P_{zSR}[Y_{zR} - Y_{zS}]$. 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 $Y_{zS}^{v_z}, Y_{zI}, Y_{zR}$ and Y_{zE} as functions of the behavioral strategies, the states of the world, and economic and epidemiological parameters.

We now introduce the *replicator dynamics* (Fudenberg and Levine 1998; Rice 2004; Weibull 1995) to analyze 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 it is when the vaccination decision is made. We assume a symmetric strategy, so that $v_z = v$ and $Y_{zS}^{v} = Y_S^{v}$ for all individual farms 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, $\overline{Y}_S = vY_S^{v=1} + (1-v)Y_S^{v=0}$:

(16) $\quad \dfrac{\dot{v}}{v} = \alpha[Y_S^{v=1} - \overline{Y}_S] \Rightarrow \dot{v} = \alpha v(1-v)[Y_S^{v=1} - Y_S^{v=0}]$,

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. In a steady state, the expected lifetime income is the same

whether farmer chooses to vaccinate or not, i.e. $Y_S^{v=1} = Y_S^{v=0}$ so that farmers are indifferent

about vaccination. So if disease risks increase, farmers will vaccinate more until the

vaccination cost is equal to the costs of infection.

The probability that a farm transits from the susceptible state to the infected state

can be obtained from the epidemiological model as

$P_{SI} = \beta_{cc}I[(1-v)S]^{\gamma-1} + (\beta_{ce}/n\psi)X_I[(1-v)S]^{\gamma-1}$. The probability of transitioning from

the susceptible state to the resistant state is simply the vaccination strategy: $P_{zSR} = v$. The

probability of transitioning from the resistant state to the susceptible state is $P_{zRS} = \chi + \tau$.

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 $P_{zES} = \varepsilon$.

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 population estimated at 12,904 for 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.

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 BTNF, Upper Gros

Ventre, Big Cow Creek) that overlap spatially with designated elk parturition ranges (WGFD, 2007). The four cattle allotments are located in Teton and Sublette County. In 2002, there were 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 3 in the appendix.

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 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 cattle farms also change in response to these fluctuations. The whole system reaches a steady state around 25 years, with infection rates of around 1.6 percent and vaccination of around 2.3 percent. (Figure 1).

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 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, because of the lack of data, 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 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 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 Figure 2 and Table 1. 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 (4,351 elk) and also the disease prevalence rate (8.2%) 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 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 vaccination converges to 4.0 percent after approximately 12 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 scenarios (c) and (d). Finally, the net benefit to the cattle sector is tied with scenario (b), and is greater than scenario (c) and (d). It is perhaps ironic that the net benefits are greater than when

there is feeding provided. (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 1; 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 30.4 percent, which is realistic. With similar numbers of infected elk, the results in the cattle sector are very tied to that of scenario (a).

Scenario (c): Elk feeding controls only

Now how about closing all the elk feedgrounds, but not applying any population controls? 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.

It turns out that the number of infected elk is the largest in this scenario relative to the others. Although infectious contacts among elk would be reduced with no feeding, there are more susceptible elk to become infected, and latter effect dominates. Why there are more susceptible elk? It is because the no-feeding policy increases mortality among all sub-populations, reducing the resistant stock in particular (as fewer infected animals live to become resistant) and also the density-dependent effects of resource competition. Indeed, in the steady state, each year 483 infected elk live to become resistant in scenario (a), while only 252 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) as, in the steady state, 3700 susceptible elk are born in each year in scenario (c) while only 1668 susceptible elk are born each year in scenario (a). This contradicts the idea of feeding as the cause of high brucellosis prevalence in the Greater Yellow Stone Area. In contrast, in scenario (a) with feeding, more elk survive to become resistant. This limits disease spread in scenario (a) relative to scenario (c). Instead of causing disease spread, elk feeding

actually decreases elk infection levels by allowing more infected elk to survive to become resistant.

The large number of infected elk, 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 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. Finally, farm net benefits are less than scenario (a), although the difference is trivial. 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

For our last scenario, we have both population and feeding controls. The simulation results (Table 1; Figure 5) show that the number of infected elk is very similar to scenarios (a) and (b).[4] This suggests that the proposed levels of population controls may not be useful in eradicating wildlife disease, even when the feed grounds are closed. Again, this is consistent with Dobson and Meagher (1996)'s finding that the threshold population for brucellosis establishment is low, and it is very hard 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

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[4] The infection rate (30.5%) is highest among all the scenarios, this is probably because of the smallest elk population in scenario (d).

scenarios (a) and (b), but still much less than scenario (c). 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 in scenarios (a) and (b) (but lower than in (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 benefit and the cost of higher vaccination rates caused by greater infection risks are essentially offsetting. Again we find that the existence of feeding grounds does not actually generate economic harm the livestock sector.

Years
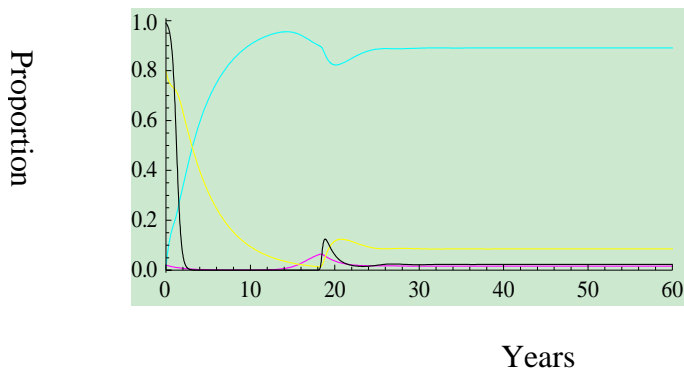
Figure 1. Cattle Only Model Simulation Results

"Note: susceptible: **blue**, infected: **Maroon**, resistant: **Yellow**, vaccination: Black "
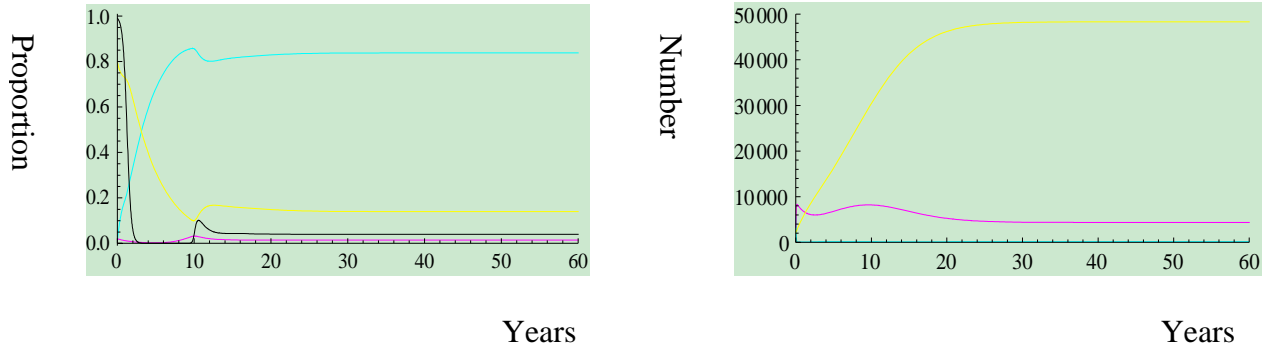
Figure 2. Joint Cattle-Elk Model, Scenario (a): No Elk Disease Management

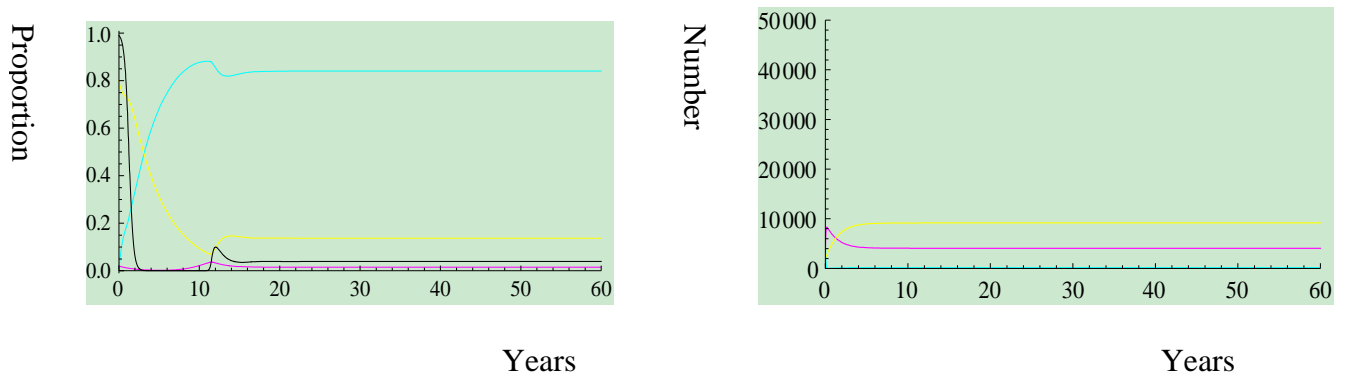Left: Cattle behavior and disease dynamics, Right: Elk disease dynamics (same for

Figures 2-5)

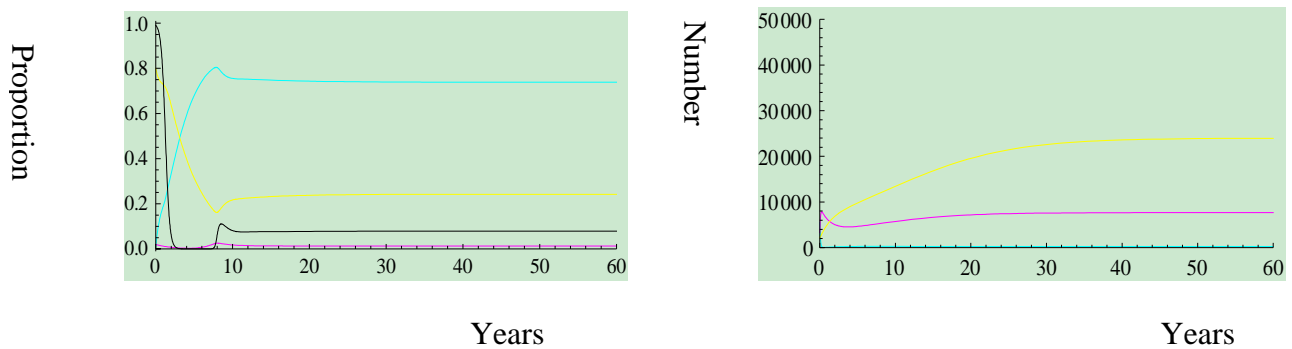**Figure 3. Joint Cattle-Elk Model, Scenario (b): Elk Population Controls Only**

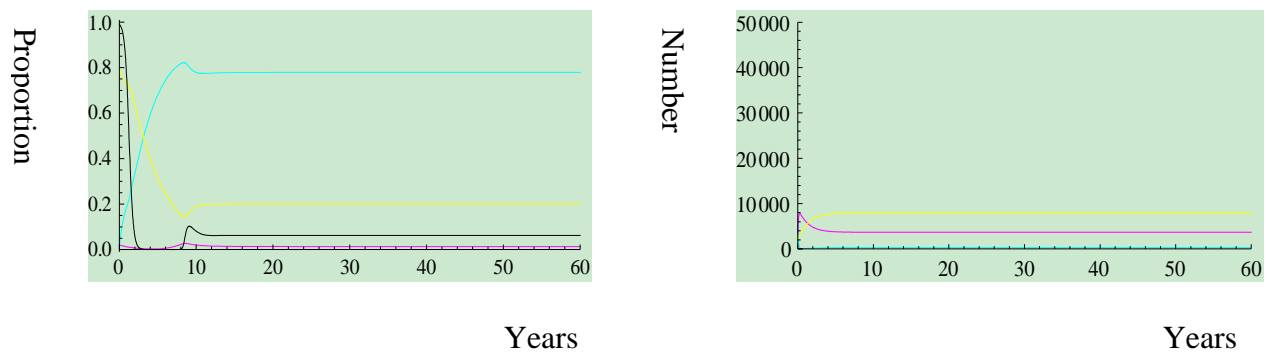**Figure 4: Joint Cattle-Elk Model, Scenario (c): Elk Feeding Controls only**

Figure 5. Joint Cattle-Elk Model, Scenario (d): Elk Population and Feeding Control

Table 1. Comparison of Steady State Dynamic Outcomes and Farmers' Net Benefits for Different Policy Scenarios

Steady State	(a) No elk disease management	(b) Elk population controls only	(c) Elk feeding controls only	(d) Elk feeding and population controls
Vaccination $(v)^*$	4.00	3.91	7.87	6.20
Susceptible Cattle $(S)^*$	83.8	84.1	73.9	77.9
Infected Cattle $(I)^*$	1.49	1.49	1.26	1.35
Resistant Cattle $(R)^*$	14.0	13.7	24.2	20.1
Susceptible Elk (X_S)	140	139	253	268
Infected Elk (X_I)	4,351	3,352	7,669	3,420
Resistant Elk (X_R)	48,335	7,537	23,879	7,340
Per Farm Net Benefits (Present Value, Millions USD)	40.550	40.552	40.548	40.549

*Expressed as a percentage of farm

Sensitivity Analysis

Sensitivity analyses are usually used to examine how changes in one or more parameters influence the results. There are many parameters in this model, and we have performed sensitivity analyses for most of them (simulation results are illustrated in the appendix).

Table 2 reports the results for those parameters that have a significant effect on the results. In all cases, we use scenario (a) as the baseline scenario, from which to evaluate parameter changes, that is, each sensitivity analysis is performed by holding all parameters and policy variable at the same level as scenario (a), and then changing a single parameter of interested as indicated. We also did the sensitivity analysis in other policy scenarios, and the results are similar to results the scenario (a).

Table 2 indicates the results when we decrease the recovery rate of elk (δ) by 50% (See Table 2). In the steady state, the number of infected elk increases significantly from 4,351 to 8,071, while both susceptible and resistant elk decrease. This makes sense because fewer infected elk will become resistant at lower recovery rate, and in turn infect more susceptible elk. Accordingly, more farms vaccinate (increases by 22.8 %) due to the greater risk imposed by the infected elk. Because of the high vaccination rate, the steady number of infected cattle decreases, while the number of resistant cattle increases (Table 2). Although the steady state values change, the disease and behavior dynamic process do not change much. Farm net benefits are still very similar to the situation when the recovery rate of elk is higher. This is because, as earlier, the benefits and costs of higher vaccination rates caused by greater infection risk essentially offset each other.

The instantaneous income flow variable (π) that we used might be high, according to Sarchet (2005). We therefore examine what happens when we decrease the value by

50%. There is no significant change in the disease and behavioral dynamic processes of the cattle model. In the steady state, fewer farms vaccinate than the baseline (decreases by 28.5%). This is because the marginal benefit of adopting vaccination decreases when profits decrease. In turn, the number of infected and susceptible cattle goes up, while the number of resistant cattle goes down (See Table 2). The farm net benefits decrease significantly, around 50%, and this is expected since the instantaneous profit has been decreased by 50%. The elk disease dynamics almost do not change at all when π changes.

The discount rate (ρ) is always a big concern in economic models, so we examine its impact by increasing it by 100% to $\rho=0.1$. Because future profits are valued less when the discount rate increases, fewer farms vaccinate (an 11% reduction). This in turn causes higher cattle infection. The number of susceptible cattle increases while the number of resistant cattle decreases (Table 2). The net benefits decrease significantly, almost 50%, because future profits are valued less. Again, the change of discount rate has little impact on elk disease dynamics and population.

We also examine the effect of transmission exponent parameter (γ) by decreasing it by 25%. The disease and behavioral dynamics processes of the cattle model do not change significantly. However, in the steady state, significantly more farms vaccinate than in the baseline (increases more than 44%), because cattle are now more likely to get infected. The high vaccination rate causes significant changes in the livestock disease status. There is a shift from susceptible farms to resistant farms, and the proportion of infected farms also decreases (Table 2). The transmission exponent has little influence on elk disease dynamics.

The results of sensitivity analyses for a number of other parameters

(χ, B_{cc}, B_{ec}, B_{ce}, σ, ζ, η, α, ω, ψ) are illustrated in the Appendix. The results were not

very sensitive in these cases, and so we do not discuss them here.

Table 2. Sensitivity Analysis[5]

Policy Scenario Steady State	1.Recovery Rate of Infected Elk (δ)— Decrease by 50%	2. Instantaneous Income Flow (π)— Decrease by 50%	3. Discount rate (ρ)— Increase by 100%	4. Transmission exponent parameter (γ) decrease by 25%
Vaccination (ν)[*]	4.91	2.86	3.54	5.77
Susceptible Cattle (S)[*]	81.2	85.4	84.6	79.0
Infected Cattle (I)[*]	1.43	2.91	1.95	1.38
Resistant Cattle (R)[*]	16.6	10.2	12.5	19.0
Susceptible Elk (X_S)	69	140	140	140
Infected Elk (X_I)	8,071	4,351	4,351	4,351
Resistant Elk (X_R)	44,814	48,335	48,335	48,335
Per Farm Net Benefits (Present Value, Millions USD)	40.549	20.132	21.131	40.549

[*]Expressed as a percentage of farms.

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[5] The sensitivity analysis results shown in this table is under the no elk disease management (Scenario a).

Conclusion

This paper investigates 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 conflict. It expands the literature by integrating disease dynamics with economic choices in such a way that risks of infection are a function of livestock disease management choices, and then economic choices are, in turn, a function of disease states. Our findings suggest that the feedbacks between jointly determined disease dynamics and decentralized economic behavior matter when choosing among various policy approaches. In particular, the farm disease and behavior dynamics reach the steady state much faster after considering the interactions with the elk sector.

By examining the population and disease dynamics under several different management options for the Jackson elk herd in Wyoming, we illustrate that farms' choices vary among different combinations of elk feeding and hunting policies. However, farms' net benefits under different combination of elk feeding and hunting policies are very similar to each other, as the cost and benefit of higher vaccination rates in response to greater infection risk essentially offset each other. The results suggest that the existence of feeding grounds does not actually generate economic harm to the livestock sector. This is surely not something expected by the livestock advocacy groups who are pushing for an elimination of the feeding grounds.

Because of the unavailability of willingness to pay data on elk hunting, we did not calculate the net benefits of elk hunting under the different policy scenarios. However, the social net benefits could vary greatly under the different policy scenarios, since there is great impact of elk hunting on the local economy (Koontz and Loomis), and different

elk population and hunting policies might change elk hunters' willingness to pay for elk hunting. In addition, other visitors' behavioral intentions appear to plausibly follow changes in wildlife populations. For instance, there would be a 10% decrease in visitation if bison populations were reduced from 600 to 400 animals and elk populations were reduced in GTNP and the NER (Loomis and Caughlan). Our results suggest that the polices should be chosen based on the elk-related benefits, not based on perceived farm benefits, since the latter varies little under the different combinations of feeding and hunting policies.

There are other infectious diseases such as chronic wasting disease in the elk, and these have imposed great risks in livestock and human society. However the mode of transmission of CWD has not been identified (Dean et.al.), and these disease should not affect Brucellosis vaccination decisions. Therefore we only model brucellosis in the joint model of wildlife and livestock disease dynamics.

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

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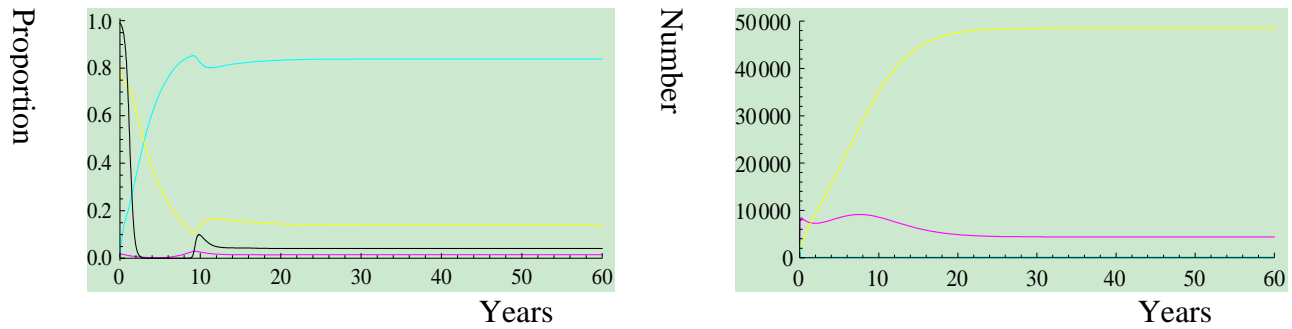
Appendix
Sensitivity Analysis[6]

Figure 6. Sensitivity Analysis: Rate of Loss of Resistance for Cattle (χ)—Increases by

100%

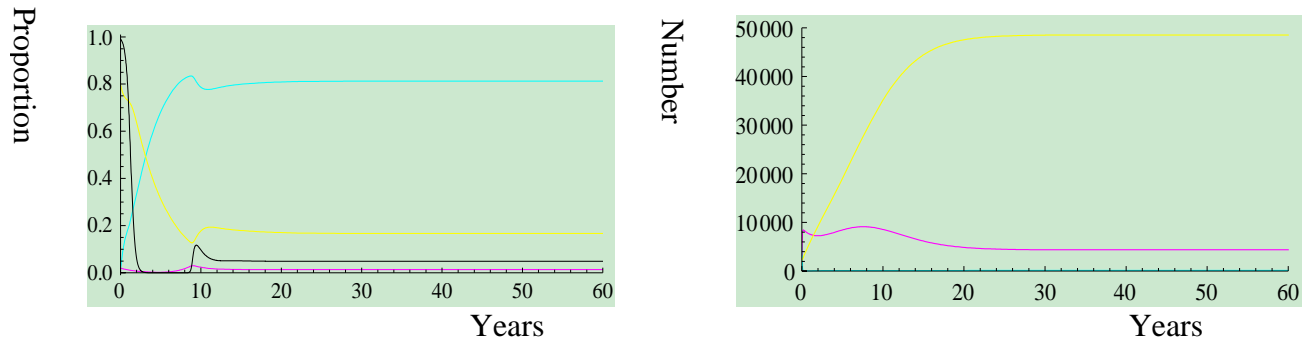Left: Cattle behavior and disease dynamics, Right: Elk disease dynamics (same for

Figures 6-17)

Figure 7. Sensitivity Analysis: Disease Transmission Parameter Between The Livestock

(B_{cc})—Increase by 100%

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[6] The sensitivity analysis are done under the no elk disease management (Scenario a), the result is very
similar is other Scenarios.
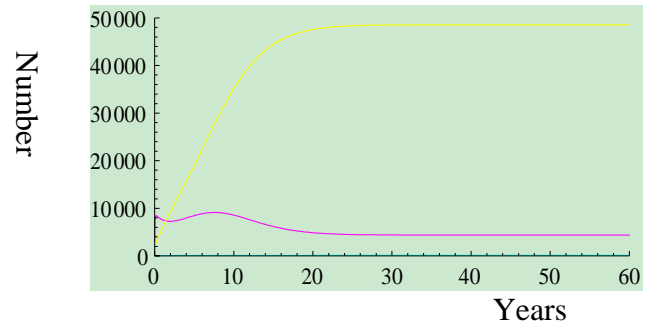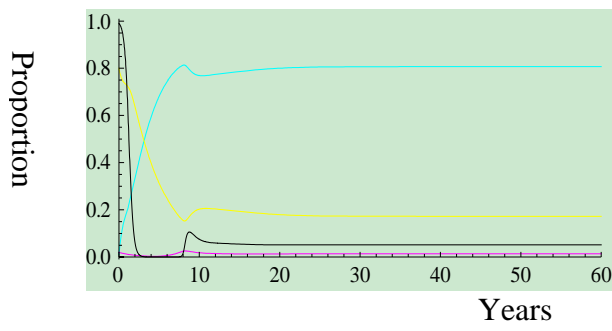
Figure 8. Sensitivity Analysis: Disease Transmission Parameter from Elk to Cattle (B_{ec})—

Increase by 100%
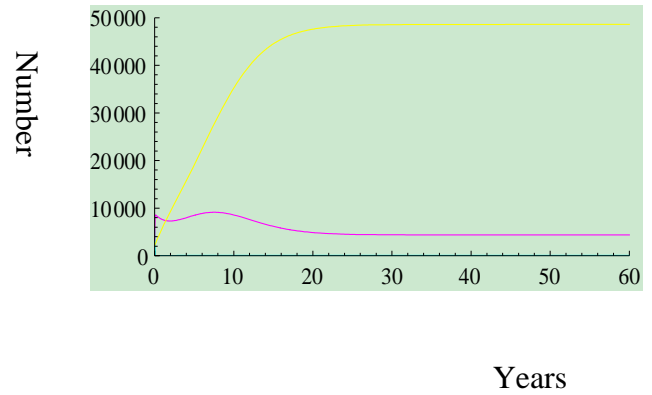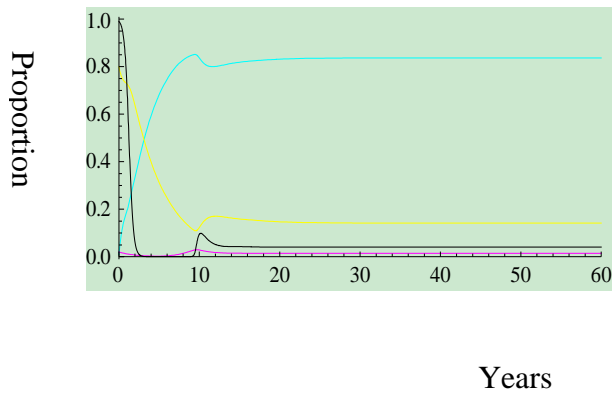
Figure 9. Sensitivity Analysis: Disease Transmission Parameter Between Elk (B_{ee}) —

Increase by 50%
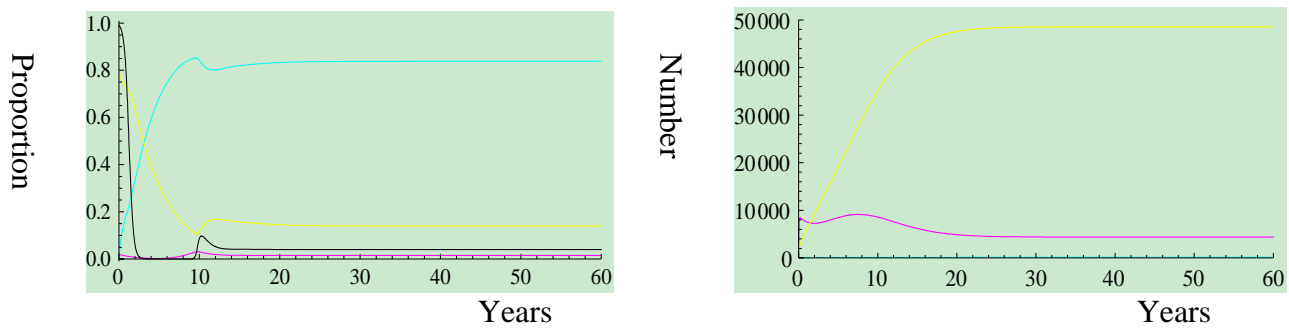
Figure 10. Sensitivity Analysis: Disease Transmission Parameter from Cattle to Elk (B_{ce})—

Increase by 100%
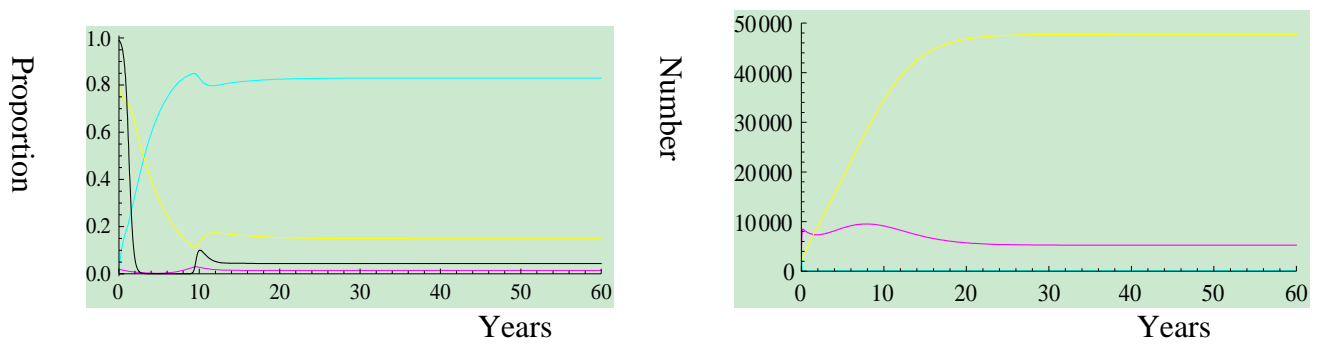
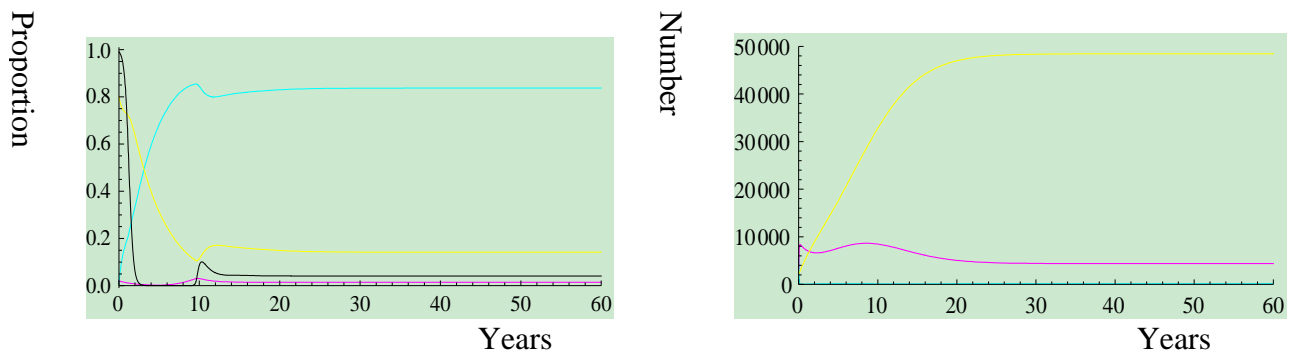**Figure 11. Sensitivity Analysis: Rate of Loss of Resistance for Elk (σ)—Increase by 100%**

Figure 12. Sensitivity Analysis: Proportion of Infected Female Elk Which Produce Infected Offspring (ζ)—Decrease by 50%
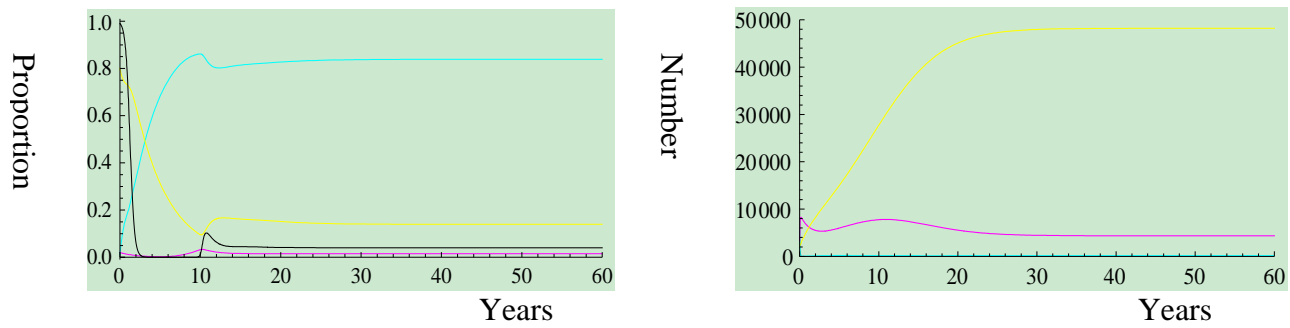
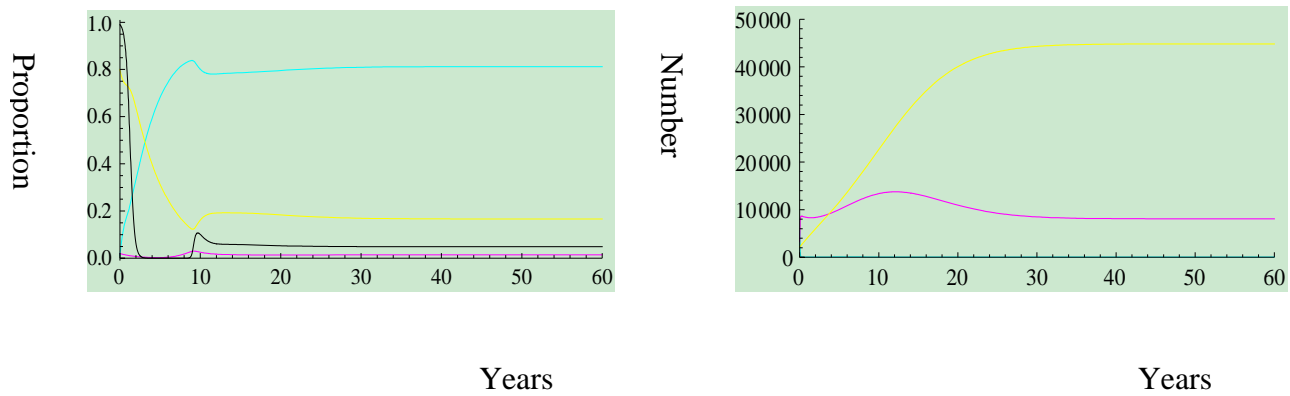**Figure 13. Sensitivity Analysis: Reduction of Fecundity in Infected Elk (η)—Decrease by 50%**

Years

Years

Figure 14. Sensitivity Analysis: Recovery Rate of Infected Elk (δ)—Decrease by 50%
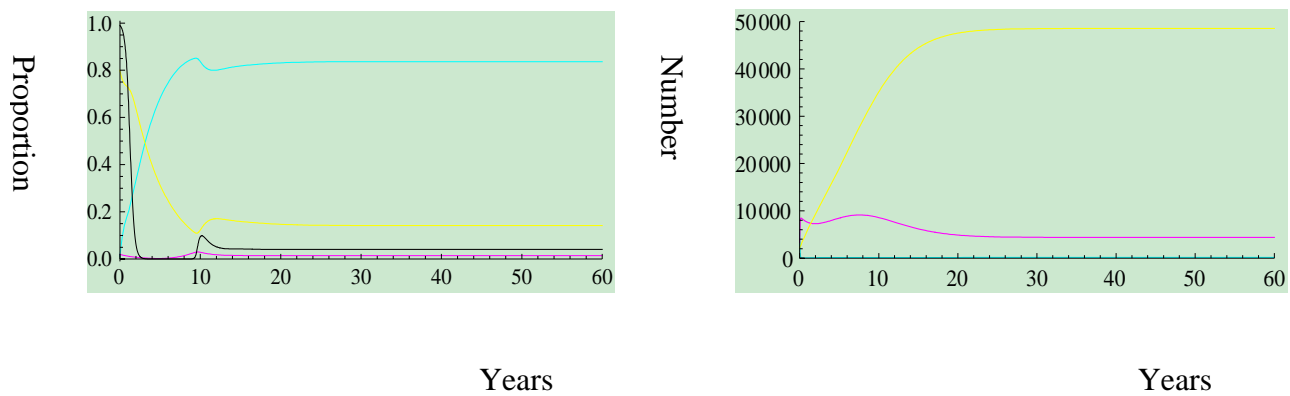

Years

Years

Figure 15. Sensitivity Analysis: Virulence Rate of Elk (α)—Increase by 100%
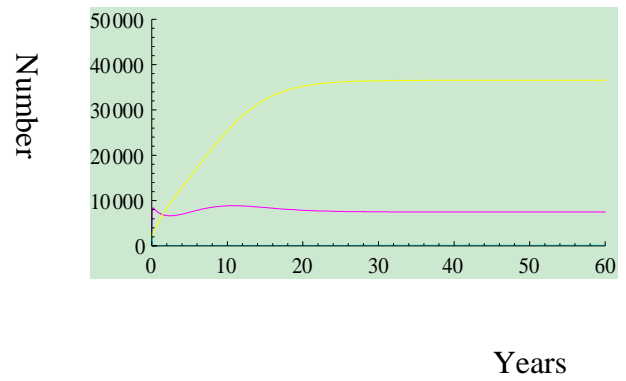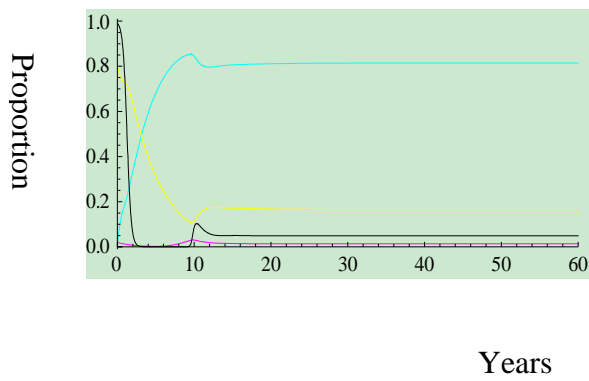
- 41 -

Figure 16. Sensitivity Analysis: Feeding Effect on Mortality Rate (ω)—Decrease by 50%
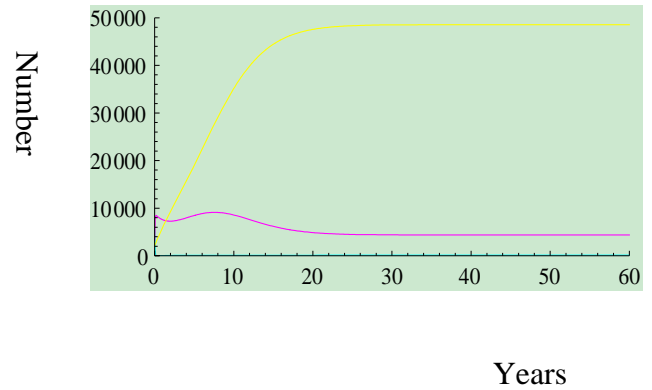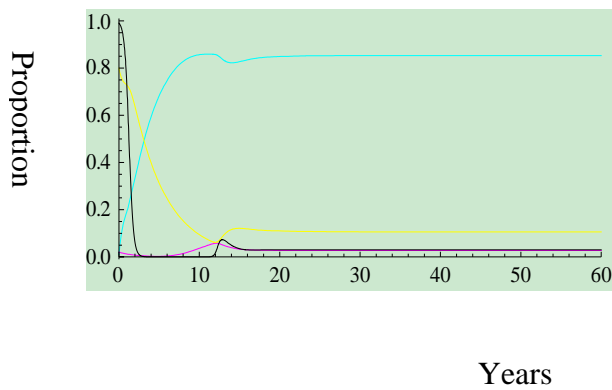

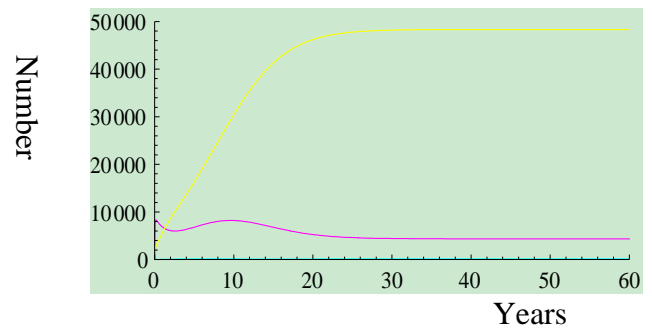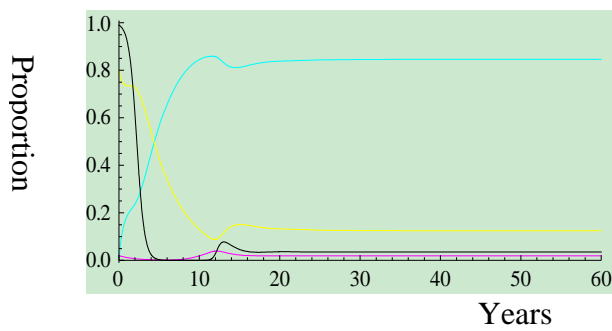**Figure 17. Sensitivity Analysis: Instantaneous Income Flow (π)—Decrease by 50%**

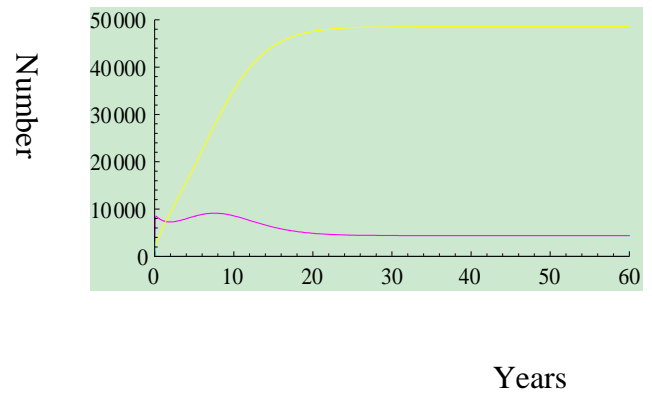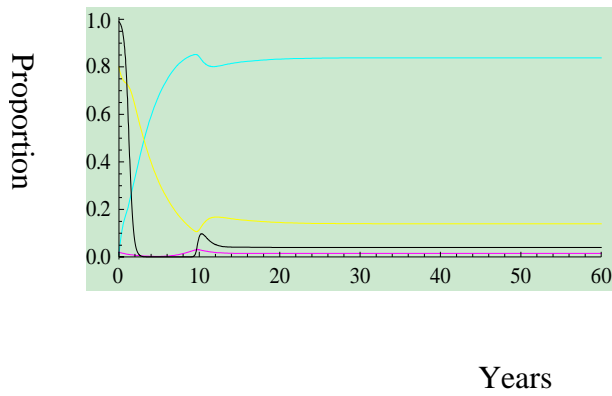**Figure 18. Sensitivity Analysis: Discount Rate (ρ)—Increase by 100%**

Figure 19. Sensitivity Analysis: Cost of Infection (b)—Decrease from $600 to $400
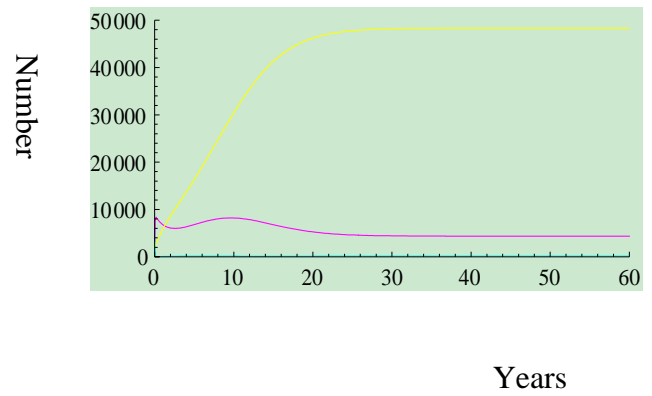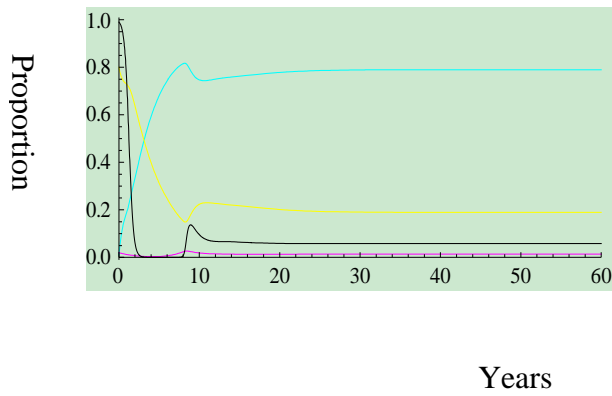

Figure 20. Sensitivity Analysis: Transmission Exponent Parameter (γ)—Decrease by 25%

Table 3. Description of parameters and data

Description	Parameter	Fixed Value for Parameter or Starting Value	Reference or Explanations
Discount rate	ρ	0.05	Assumption
Cost of infection	b	600	Infection cost is 200 per head, assuming 1% of a herd with an average size of 300 get infected (Wyoming Department of Administration and Information 2004)
Depopulation cost	d	15150	Assume 5% of the total cattle value loss to the farms when depopulated[7]
Cost of vaccination	c	300	Assume the cost of vaccination is $10 per head; the average size of farm is 300.
Instantaneous income flow at each point in time	π	11,420	The net cash income in average farm is $11906 in Sublette, and $10228 in Teton, so we use an weighted average number for approximation (NASS)[8]
Rate of loss of resistance for cattle	χ	0.01	The probability that a resistant cattle will loss resistance
Transition rate from empty (depopulated) to susceptible	ε	1	The probability that a depopulated farm will buy new susceptible cattle

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[7] When a farm is depopulated, farmer will receive an indemnity payment which covers most part of the cattle value, we also assume there are no fixed costs in the farm operation.

[8] There are 270 farms in Sublette County and 110 in Teton; we use these to calculate the average net income per farm in these two counties. The net cash income is an average value for all farms in the county, according to Sarchet, Brent, the total cash income per cow is $333.59 for a small farm with upon 325 cow head. So we think the average value is within a reasonable range.

Table 3. Description of parameters and data (continued)

Description	Parameter	Fixed Value for Parameter or Starting Value	Reference or Explanations
Effectiveness of the vaccination	κ	0.7	Wyoming Department of Administration and Information (2004)
Disease transmission parameter between the livestock,	β_{cc}	2	Dobson and Meagher (1996)
Disease transmission parameter from elk to cattle	β_{ec}	2	Assume frequency dependent transmission, $\beta = w^{-0.26}$, where w is the mass of host species (Dobson and Meagher 1996)
Average number of cattle in each farm	ψ	300	USDA (2002)
Rate of depopulation	q	0.5	Assumption
Speed of adjustment parameter in the vaccination strategy replicator dynamics	α	1×10^{-5}	Assumption
Cattle "turnover rate"	τ	0.23	Proportion of new susceptible cattle purchased every period
Rate of loss of resistance for elk	σ	0.01	Dobson and Meagher (1996)
Elk birth rate	a	0.25	Dobson and Meagher (1996)
Natural mortality rate of elk	m	0.15	Dobson and Meagher (1996)

Table 3. Description of parameters and data (continued)

Description	Parameter	Fixed Value for Parameter or Starting Value	Reference or Explanations
Proportion of infected female elk that produce infected offspring	ζ	0.9	Dobson and Meagher (1996)
Reduction of fecundity in infected elk	η	0.5	Dobson and Meagher (1996)
Rate of infectious transmission among elk	β_{ee}	0.002	Assume density dependent transmission, $\beta = 0.0247w^{0.44}$, where w is the mass of host species (Dobson and Meagher 1996)
Rate of infectious transmission from cattle and elk	β_{ce}	0.002	Assume density dependent transmission, $\beta = 0.0247w^{0.44}$, where w is the mass of host species (Dobson and Meagher 1996)
Transmission exponent parameter	γ	10	Assumption
Recovery rate of infected elk	δ	0.5	Dobson and Meagher (1996)
Virulence rate of elk	α	0.005	Dobson and Meagher (1996)
Feeding effect on mortality rate	ω	0.001	Assume $M = (1 - \omega f)m$, calibrated using natural mortality rate and an observed mortality rate 0.037 (Kreeger et.al. 2002).
Aggregated harvest	h	0	Dobson and Meagher (1996)
Number of elk in Jackson herd	$X(0)$	11853	WGFD, starting value

Table 3. Description of parameters and data (continued)

Description	Parameter	Fixed Value for Parameter or Starting Value	Reference or Explanations
Elk natural resistant rate	$X_R(0)/X(0)$	0.2	Kreeger et.al. (2002)
Initial elk infection level	$X_I(0)/X(0)$	0.3	Kreeger et.al. (2002)
Resistant proportion farms	$R(0)$	0.1	Assumption
Empty proportion farms	$E(0)=qP(0)$	0.25	Assumption
Infected proportion farms	$P(0)$	0.02	Assumption
Susceptible proportion farms	$S(0)=1-R(0)-E(0)-P(0)$	0.8	Assumption
Aggregate rate of vaccination in the population	$\nu(0)$	0.99	Assumption