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Wildlife Conservation Payments to Address Habitat Fragmentation and Disease Risks

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Abstract

We build a stylized model to gain insights into the application of conservation payments to protect endangered species in the face of wildlife-livestock disease risks and habitat fragmentation. Greater connectivity of habitat creates an endogenous trade-off. More connectedness ups the chance that populations of endangered species will grow more rapidly; however, greater connectivity also increases the likelihood that diseases will spread more quickly. We analyze subsidies for both habitat connectedness and livestock vaccination. We find the cost-effective policy is to initially subsidize habitat connectivity rather than vaccinations; this increases habitat contiguousness, which eventually also increases disease risks. Once habitat is sufficiently connected, disease risks increase to such a degree to make a vaccination subsidy worthwhile. Highly connected habitat requires nearly all the government budget be devoted to vaccination subsidies. The result of the conservation payments is significantly increased species abundance, for a wide range of initial levels of habitat connectedness.

Introduction

Diseases are one of the major threats to wildlife conservation in low income countries. Disease risks increase substantially with greater habitat fragmentation or other contributing factors of species decline (Simonetti 1995; McCallum and Dobson 1995). These risks are pervasive in low income countries where high rates of habitat loss and fragmentation have led to significant rates

of species losses (Daszak et al. 2000; Ceballos et al. 2005). Managing disease problems involving endangered species on fragmented habitats requires an understanding of both ecology and economics because both economic and ecological tradeoffs arise in response to the various ecological investment options that could improve the situation. The ecological tradeoffs have been elucidated in prior work (e.g., Hess 1996; Gog et al. 2002; McCallum and Dobson 2002), but the role of economics has been seriously under-explored.

The ecological literature on the joint problem of habitat fragmentation and disease is fairly limited, but it does illustrate that solutions may be more complicated than simply reducing the degree of fragmentation (e.g., Hess 1996; Gog et al. 2002; McCallum and Dobson 2002). The basic problem is as follows. Many national parks and wildlife reserves in low income countries are not large enough to support thriving populations of large mammals (Simonetti 1995). As these areas are not generally enclosed, this means that many individual animals wander outside of the reserve, where they may face increased risk of poaching and lack of habitat due to fragmentation among private landholdings. Such risks imply that conservation efforts outside the reserve also matter for success within the reserve—public and private lands are complementary (Povilitis 1998; Simonetti 1995).

Other than permanently increasing reserve sizes, one potential solution to increase species' protection and reduce the degree of habitat fragmentation involves paying private landowners to provide habitat and protect wildlife on their land. While habitat provision can improve a species' chances for survival, provision on or near livestock grazing lands can bring about its own set of risks. This is because livestock can serve as a reservoir for many diseases that can be harmful to wildlife (and vice versa; see footnote 3) – particularly species with small and already-stressed populations (Simonetti 1995; Gaydos and Gildardi 2004; Peterson 1991;

McCallum and Dobson 1995, 2002).¹

A *disease reservoir* can greatly amplify disease risks. In a single-host species-pathogen system (i.e., no disease reservoir), the ability of a disease to establish in a host population generally depends on the density of the population. Infectious contacts are more likely in a denser population. If a species is threatened or endangered, theory predicts its low numbers may actually protect the species from disease because there will be too few infectious contacts for the disease to establish.² But in a multi-host species-pathogen system involving a reservoir host, the disease can more easily establish within the endangered population because there is a sustained risk of exposure due to the reservoir (McCallum and Dobson 2002; Dobson 2004). Moreover, disease mortality can be very high among the non-reservoir species (McCallum and Dobson 1995, 2002; Dobson 2004), greatly increasing extinction risks.

Greater habitat area or reduced habitat fragmentation would seem to increase disease risks for endangered species, to the extent that there is a concomitant increase in infectious contacts between and within endangered species and a reservoir host (Simonetti 1995; Hess 1996).³ But others disagree (e.g., Gog et al. 2002). McCallum and Dobson (2002), for instance,

¹ McCallum and Dobson (2002, p.2042) define a host reservoir as a species “in which the pathogen is maintained with a less detrimental impact than that on the “target” endangered host species. Examples involving livestock disease reservoirs include the Rinderpest virus which has spread from Zebu cattle to wildebeest and cape buffalos in the Serengeti (McCallum and Dobson 1995; Plowright 1982), the tapeworm *Cysticercus tenuicollis* which infects Andean deer in Chile. See Hess (1986) and Gog et al. (2002) for more examples. Examples of wildlife disease reservoir problems include bovine tuberculosis among white-tailed deer in Michigan, USA (Schmitt et al. 2002), badgers in the UK (Smith et al. 2001), and Australian brushtailed possums in New Zealand (Barlow 1991), and also foot and mouth disease in Andean deer in southern Chile (Simonetti 1995) and feral pigs in Australia (Dexter 2001).

² There is some debate over whether it might be density (animals/unit area) or actual numbers that matter (McCallum and Dobson 2002; McCallum et al. 2001). If density matters, theory predicts an endangered population on a very small habitat area might be at most risk of extinction (McCallum and Dobson 2002).

³ A similar set of tradeoffs may impact farmers capable of habitat provision. Farmers appreciate the income generated from conservation payments. This conservation, however, may put their own livestock at risk, as it is also possible that wildlife could be a reservoir for other diseases that could adversely affect livestock. The presence of or the potential for disease reservoirs in wildlife could make it harder to encourage farmers and other landowners to set aside lands for species protection. Apart from disease, habitat provision may also lead to greater wildlife conflicts such as predation on livestock or crop damage.

show an opposing effect exists to counteract increased infectious contacts, namely increased re-colonization of extinct patches by healthy animals. They find that reducing habitat fragmentation (or increasing habitat connectedness) can, under certain circumstances, simultaneously improve a species' chances for survival and increase the proportion of healthy animals within the population.

The parameters of McCallum and Dobson's (2002) model define the circumstances under which increased connectedness is beneficial. They investigate various combinations of fixed parameter values and illustrate that the following three parameters are particularly important: (i) the colonization rate of endangered species relative to reservoir hosts (livestock in our model), (ii) the rate of infectious contacts between livestock and wildlife, and (iii) the rate of recovery from infection (to an immune or recovered state) within the livestock population. But these and other parameters are not fixed in reality – investments can be made to affect many parameter values, thereby altering the benefits of increased habitat connectedness. This holds with particular force when domestic animals are part of the ecological system. For instance, management of livestock movement affects the relative rate of re-colonization, biosecurity efforts reduce infectious contacts, and livestock health management prevents infection and improves recovery rates. Farmers are unlikely to make any of these investments at socially efficient levels because doing so is costly and they do not capture the full social benefits. Effective conservation may therefore require the public provision of incentives for these investments.⁴ And as provisioning payments to farmers requires re-allocating public (and sometimes private, in the case of international conservation aid) funds away from other public

good uses (e.g., public health, education), efficient conservation requires these incentive payments be allocated in accordance with the economic and ecological tradeoffs that they imply.

This paper investigates how conservation payments for various risk-reducing ecological investments can be used to affect wildlife conservation and disease risks. We highlight the joint determination of economic and wildlife disease systems (see also Horan and Wolf 2005), as disease risks – as with other ecological risks – are to some extent an endogenous function of human economic choices (Shogren and Crocker 1991; Crocker and Tschirhart 1992). The analysis also expands recent work on invasive species management and emphasizes the benefits of allocating some conservation payments for preventing new infections in lieu of investing it in *in situ* disease control or in habitat quality as a means of increasing species productivity (Leung et al. 2002; Finnoff et al. 2005; Horan and Lupi 2005a,b). We believe it is useful to begin our analysis by providing background on the problem that motivates how we construct our bioeconomic model—endangered Andean deer in Chile.

Motivating Example—Endangered Andean Deer in Chile

The Andean deer (*Hippocamelus bisulcus*), known commonly as huemul in South America, is a cultural symbol of Chile, appearing on its national coat-of-arms alongside the threatened Andean condor (*Vultur gryphus*). There is widespread interest in preserving and restoring the species in Chile. Predation, habitat degradation, low reproductive rates, a fragmented population, and the integrity of the population (viability requires some level of interchange of individuals between groups of animals over time, including re-colonization of unoccupied sites) are cited as reasons for the decline in the Chilean population (Povilitis 1998). Huemul is found in greatest

⁴ While farmers may consider investing in recovery, we assume disease impacts to reservoir populations are comparatively mild. But even if they were significant, there are still external social benefits from this investment (as

abundance (about 1500 individuals) in Argentinian Patagonia (Povilitis 1983; Smith-Fleuck and Fleuck 1995). The northern part of the species' historic range has been reduced to a confined area of Central Chile called Nevados de Chillán and was estimated to have a population here of about 60 individuals in 1997 (Povilitis 1998).

Connectivity of habitats and disease risks are both areas of concern for Andean deer conservation. Huemul (and deer in general) are susceptible to *Cysticercus tenuicollis*, a bladderworm that is the larval stage of the canine and feline tapeworm *Taenia hydatigena*. *C. tenuicollis* is a common parasite of deer and other ruminant species (Pybus 1990; Michigan DNR), and has been identified as a source of mortality for huemul (Texera 1974; Simonetti 1995; Povilitis 1998). *C. tenuicollis*, along with habitat fragmentation, is viewed as a major obstacle to population recovery (Simonetti 1995; McCallum and Dobson 2002; Povilitis 1998). Cross-species transmission of *C. tenuicollis* to huemul from livestock (or other infected wildlife) is of serious concern. Free-ranging domestic cattle and goats are known carriers of *C. tenuicollis* as well as a number of other pathogens that are transmitted to huemul (Povilitis 1998).

Earlier analysis of management actions that could be taken to conserve the huemul and work toward recovery of the species throughout its historic range has highlighted four main areas: protecting core habitat areas as *strict reserves*, establishing habitat connectivity, implementing conservation management plans on private lands, and taking measures to augment the population. Because huemul range beyond strict reserves where poaching, livestock-related disease and habitat degradation are entirely eliminated, private actions taken on livestock grazing lands are also necessary for species conservation (Simonetti 1995; Povilitis 1998). Livestock health measures, the mobility of livestock, and habitat investments on grazing lands appear to be

with the other investments) that the farmer would not fully capture in the market price.

particularly important factors for Chilean officials or international donor agencies funding a conservation initiative to consider in allocating financial and management resources.

A bioeconomic model of livestock health management and wildlife mobility can be a helpful tool to examine resource allocation when the presence of endangered wildlife has social benefits and conservation funds are in short supply. Our bioeconomic model herein focuses on habitat fragmentation and disease causes of population decline in the species. The biological component of our model utilizes a metapopulation model to analyze the issue of habitat fragmentation, like that present in the Nevados de Chillán area, that is a cause of concern for many endangered species. Meta-population models are increasingly used to analyze the ecological implications of increasing habitat connectivity (i.e., mobility as a model parameter), particularly in disease settings (e.g., Hess 1996; Gog et al., 2002; McCallum and Dobson 2002). We expand the set of management options to include vaccination for cases in which livestock health management is a mortality risk factor for an endangered species. Research suggests that vaccination is an effective preventive measure against *C. tenuicollis* in livestock (Wikerhauser et al. 1971; Babiker and Eldin 1987), and to the extent that huemul come into contact with livestock grazing areas where the pathogen can be transmitted, vaccination of livestock against the helminth may be an important management option to consider.

Earlier meta-population models (Hess 1996; McCallum and Dobson 2002) have indicated under a variety of different conditions that when management of habitat connectivity is considered alone, species extinction in individual patches or the entire meta-population may be imminent. By incorporating livestock vaccination as a management option and taking into account costs and benefits of different conservation management activities our bioeconomic model suggests that a more broadly distributed population of the endangered species is possible.

Bringing such an analysis to bear on the specific problem of huemul or a number of other endangered species facing livestock disease mortality risks in developing countries may provide useful insights to decision makers and funding agencies in a particular locale.

The ecological model

The model we use to investigate the disease-habitat fragmentation problem is motivated by the problem of *C. tenuicollis* in Andean deer. We adopt a slight variation of the metapopulation model used by McCallum and Dobson (2002).⁵ Our model provides a qualitative representation of the situation, designed to produce insights into the problem. McCallum and Dobson take a similar approach – their model is also motivated by this problem and they use a hypothetical simulation to address the relevant issues. Assume there are N available patches, which are identical in terms of species' carrying capacities and distances from each other.⁶ Each patch may be in one of seven states at time t

- (i) Empty (no wildlife or livestock present). The number of patches in this state is e .
- (ii) Occupied only by susceptible livestock; the number of patches is s_L .
- (iii) Occupied only by susceptible wildlife; the number of patches is s_W .
- (iv) Occupied only by susceptible livestock and wildlife; the number of patches is

⁵ Their model extends Hess (1996) and Gog et al. (2002) and is ultimately rooted in the metapopulation modeling approach developed by Levins (1969). These sorts of metapopulation models depict species occupation of various patches of land, without modeling the within-patch dynamics. Simplifying assumptions are also made with respect to homogeneity of patches. Specifically, all patches have identical characteristics, within-patch dynamics (not modeled), and environmental conditions.

⁶ Hess (1996) begins his analysis with a simple metapopulation model that models only between patch dynamics involving uniform patches (an island model), but then moves on to consider a more complicated model that considers dynamics both within and between patches, as well as different spatial configurations. The problem of Andean deer in Chile is more like the necklace configuration examined by Hess. But, at least when modeling a single host, Hess's results for the island and necklace models are qualitatively similar.

S_{LW} .

- (v) Occupied only by resistant or immune livestock; the number of patches is r .
- (vi) Occupied only by resistant livestock and susceptible wildlife; the number of patches is r_W .
- (vii) Occupied only by infected livestock (assume any infected wildlife within a patch immediately go extinct); the number of patches is i .

Note there are no patches containing resistant wildlife. This is because wildlife never recover from infection to become resistant. We also assume vaccination of wildlife is either unavailable or infeasible, which is common for many wildlife disease problems (e.g., Michigan Department of Agriculture 2002).

First consider factors influencing the status of livestock patches — susceptible (s), resistant (r), or infected (i). Following McCallum and Dobson (2002) and Hess (1996), the rates of extinction of patches for the various classes of livestock are denoted x_s, x_r , and x_i , with all rates being non-negative and $x_i > x_s, x_r$. The term “extinction” when made in reference to a livestock patch does not refer to a local biological extinction in the same sense as an endangered species; rather, it means the removal of livestock from one patch without relocation into another patch within the region. This would occur when a herd is either culled or sold. Migration of livestock across patches occurs at a rate m_L . If susceptible livestock enter an empty patch, that patch switches to susceptible livestock status. If infected livestock enter a patch of susceptible livestock, that patch has a probability δ of becoming infected. Resistant livestock entering a patch do not affect the patch status and neither do infected livestock entering a resistant patch. Infected patches recover to a resistant state at a rate γ . Finally, susceptible livestock become

resistant (e.g., due to vaccination) at a rate ψ , and resistant patches revert back to susceptible status (as resistance is lost) at a decay rate λ .

Now consider wildlife. Endangered species become extinct on any patch at a rate x_W . They immediately become extinct on a patch if the livestock on their patch becomes infected or if an infected immigrant arrives. The migration rate of endangered species is m_W . McCallum and Dobson (2002) refer to m_W as a measure of *wildlife habitat connectedness*. For example, m_W is large for large contiguous reserves and small for fragmented patches across private lands, e.g., villages, public grazing lands.

The metapopulation dynamics of patches of susceptible livestock is given by the equation of motion

$$(1) \quad \frac{ds_L}{dt} = m_L(s_L + s_{LW} + r + r_W) \frac{e}{N} - x_s s_L - m_L \delta i \frac{s_L}{N} - \psi s_L + \lambda r - m_W(s_W + s_{LW} + r_W) \frac{s_L}{N} + x_W s_{LW}$$

The first right hand side (RHS) term represents the colonization of empty patches by livestock migrants of other susceptible patches (i.e., it equals the migration rate of livestock, times the number of patches containing susceptible and resistant livestock, times the probability that these animals colonize empty patches). The second RHS term reflects extinction of susceptible livestock patches (e.g., due to sales). The third term is the number of susceptible livestock patches that become infected. The fourth term is the number of susceptible patches that become resistant due to vaccination, while the fifth term reflects the loss of resistance. The sixth term is the number of susceptible livestock patches that become susceptible patches containing both species (i.e., it equals the migration rate of wildlife, times the number of patches containing wildlife, times the probability that these wildlife move onto patches of type L). Finally, the

seventh term reflects switching of LW -type patches to L -type patches due to local extinctions of wildlife within those patches.

Following McCallum and Dobson (2002), we scale the model to focus on the proportions of each patch type (as opposed to absolute numbers). We do this by dividing all state variables by N . We also redefine time in units of x_s , and all rate parameters (except the infection rate δ) in units of x_s . All scaled variables and parameters are denoted by upper-case symbols (except time, t , which is denoted by $\tau = x_s t$). Given these modifications, equation (1) is re-written as

$$(2) \quad \frac{dS_L}{d\tau} = M_L(S_L + S_{LW} + R + R_W)E - S_L - M_L\delta IS_L - \Psi S_L + \Lambda R \\ - M_W(S_W + S_{LW} + R_W)S_L + X_W S_{LW}$$

where $E = 1 - (S_L + S_W + S_{LW} + R + R_W + I)$.

The equations of motion for the other state variables can be defined in a similar manner:

$$(3) \quad \frac{dS_W}{d\tau} = M_W(S_W + S_{LW} + R_W)E - X_W S_W - M_W\delta IS_W \\ - M_L(S_L + S_{LW} + R + R_W)S_W + S_{LW} + X_r R_W$$

$$(4) \quad \frac{dS_{LW}}{d\tau} = M_L(S_L + S_{LW} + R + R_W)S_W + M_W(S_W + S_{LW} + R_W)S_L \\ - M_L\delta IS_{LW} - \Psi S_{LW} + \Lambda R_W - (1 + X_W)S_{LW}$$

$$(5) \quad \frac{dR}{d\tau} = -R(X_r + \Lambda) + \Gamma I + X_W R_W - M_W(S_W + S_{LW} + R_W)R + \Psi S_L$$

$$(6) \quad \frac{dR_W}{d\tau} = M_W(S_W + S_{LW} + R_W)R - (X_r + X_W + \Lambda)R_W + \Psi S_{LW}$$

$$(7) \quad \frac{dI}{d\tau} = I(M_L E - X_i + \delta[M_W S_W + M_L(S_L + S_W + S_{LW})]) - \Gamma$$

Incorporating economic choices

The model so far is similar to McCallum and Dobson (2002), except we allow for resistance to form within susceptible livestock as a result of preventative veterinary medicine (vaccination) administered by ranchers.⁷ Denote the level of vaccination within a type k patch ($k=L, LW$) by Ψ_k . This is strictly a preventative measure. It does not directly affect the *in situ* productivity of the endangered species population, but it does provide indirect benefits by reducing disease propagule pressure.

We further depart from McCallum and Dobson's model by allowing wildlife habitat connectedness, M_w (again a measure of outward migration of wildlife from a patch), to be endogenously affected by habitat management choices. Denote the change in habitat connectedness by Z_k ($k=W, LW, RW$), which is patch-specific and which could be negative if disinvestment in connectivity is warranted. After the investments are made, the ecological parameters also become patch-specific: $M_{w,k} = M_{w0} + Z_k$, where M_{w0} is the pre-investment level of migration. Habitat connectedness has direct effects on endangered species conservation – greater connectivity increases the growth of endangered species populations, although it might also allow for greater disease spread. Our model allows us to capture this critical endogenous tradeoff.

Landowners generally make investments in habitat connectivity and ranchers make investments in livestock vaccination, although these two groups could overlap. For ease of

⁷ Veterinary medicine can also increase the rate of livestock recovery from infection, Γ , but prevention of disease occurrence is the only way to avoid costs associated with the loss of endangered species. Biosecurity, under some situations, is also a preventative measure that ranchers could invest in to reduce the rate of infectious contact between wild and domestic species. This usually involves separating wildlife from livestock by a physical barrier (fences) or some other means. Since livestock in close proximity to Chilean parks tend to be free-ranging (Povilitis 1998), physical separation would not be straightforward unless wholesale cultural and production system changes are made – changes that would probably be untenable at least in the short run. We therefore take these systems as given and do not consider biosecurity as a choice variable.

notation and without loss for the present stylized model, we assume all landowners are ranchers. Assume the existence of a representative rancher in each patch, and that ranchers are homogeneous across patches (at least, in the absence of any patch-specific subsidies that might be applied to them). Each rancher's vaccination and habitat management costs at time τ are given by the separable cost function, $C = c(\Psi) + g(Z)$. The function c is increasing and convex, with $c(0)=0$. Habitat management costs also vanish when there is neither investment nor disinvestment in habitat connectivity, i.e., $g(0)=0$. Both investment and disinvestment are costly, with $g', g'' > 0$ for $Z > 0$ and $g' < 0, g'' > 0$ for $Z < 0$. Finally, assume ranchers invest nothing in vaccination or habitat management unless publicly-provided incentives exist because they do not otherwise internalize any benefits from these investments (i.e., $c'(0) = g'(0) = 0$).⁸

A bioeconomic model

Suppose an agency (governmental or non-governmental) wants to provide monetary support for the protection of the wildlife population. The value that the agency attributes to the population in any given period is $U(S_W + S_{LW})$ (with $U' > 0, U'' < 0$), which could be thought of as a combination of existence and eco-tourism values. To promote this value on private lands, the agency subsidizes habitat connectivity and livestock vaccination. The subsidy rate for the j th investment ($j=\Psi, Z$) in patch k is denoted $\sigma_{j,k}$, so that post-subsidy costs are

$$(8) \quad c(\Psi_k) + g(Z_{M,k}) - \sigma_{\Psi,k} \Psi_k - \sigma_{Z,k} Z_k$$

Minimization of expression (8) leads to the following first order conditions and response

⁸ Vaccination may provide some private benefits in terms of improved livestock productivity. But if these are limited (as is the case given the assumption made in footnote 4 about disease impacts on reservoir populations), vaccination may not yield any net private gains. Accordingly, we make the simplifying assumption that no investments are made without a subsidy.

functions

$$(9) \quad c'(\Psi_k) - \sigma_{\Psi,k} = 0 \quad \forall k \Rightarrow \Psi_k(\sigma_{\Psi,k}), \quad k = L, LW$$

$$(10) \quad g'(Z_k) - \sigma_{Z,k} = 0 \quad \forall k \Rightarrow Z_k(\sigma_{Z,k}), \quad k = W, LW, RW$$

Assume the agency is concerned with the intertemporally efficient management of the wildlife resource and of disease risks. Given a discount rate of ϕ , the agency chooses subsidy rates to solve

$$(11) \quad \begin{aligned} \text{Max}_{\sigma_{\Psi,k}, \sigma_{Z,k}} \quad & \int_0^\infty \left[U(S_W + S_{LW}) - \sum_k [c(\Psi_k(\sigma_{\Psi,k})) + g(Z_k(\sigma_{Z,k}))] \right. \\ & \left. - \beta \sum_k [\sigma_{\Psi,k} \Psi_k(\sigma_{\Psi,k}) + \sigma_{Z,k} Z_k(\sigma_{Z,k})] \right] e^{-\phi\tau} d\tau \end{aligned}$$

subject to the equations of motion (2)-(7) and the rancher's response functions defined by (9) and (10). The term $\beta \sum_k [\sigma_{\Psi,k} \Psi_k(\sigma_{\Psi,k}) + \sigma_{Z,k} Z_k(\sigma_{Z,k})]$ represents the social welfare impacts of the subsidy payments, as allocating more subsidies to this conservation problem implies less money is now available for other conservation or public goods investments. The parameter β represents the (constant) marginal cost of diverting funds to this conservation problem, which may include transactions costs (Alston and Hurd 1990).

The Hamiltonian associated with this problem is

$$(12) \quad \begin{aligned} H = & U(S_W + S_{LW}) - \sum_k [c(\Psi_k(\sigma_{\Psi,k})) + g(Z_k(\sigma_{Z,k}))] \\ & - \beta \sum_k [\sigma_{\Psi,k} \Psi_k(\sigma_{\Psi,k}) + \sigma_{Z,k} Z_k(\sigma_{Z,k})] + \mu_{S_L} \dot{S}_L \\ & + \mu_{S_W} \dot{S}_W + \mu_{S_{LW}} \dot{S}_{LW} + \mu_R \dot{R} + \mu_{R_W} \dot{R}_W + \mu_I \dot{I} \end{aligned}$$

where μ_j represents the co-state variable associated with the j th state variable. The first order conditions associated with an interior solution are

$$(13) \quad \frac{\partial H}{\partial \sigma_{\Psi,k}} = 0 \quad k = L, LW$$

$$(14) \quad \frac{\partial H}{\partial \sigma_{Z,k}} = 0 \quad k = W, LW, RW$$

In addition to these first order conditions, the following adjoint conditions are also necessary for an optimum

$$(15) \quad \dot{\mu}_j = \phi \mu_j - \frac{\partial H}{\partial j} \quad j = S_L, S_W, S_{LW}, R, R_W, I$$

These conditions in (15) ensure no intertemporal arbitrage opportunities arise (see Clark 1990).

Optimal subsidies

We derive expressions for the optimal subsidy rates from the first order conditions (9), (10), (13) and (14). For instance, consider condition (13) for $k=L$

$$(16) \quad \frac{\partial H}{\partial \sigma_{\Psi,L}} = -c' \Psi'_L - \beta [\Psi_L + \sigma_{\Psi,L} \Psi'_L] - \mu_{S_L} \Psi'_L S_L + \mu_R \Psi'_L S_L = 0$$

Using (9), we manipulate condition (16) to derive an expression for the optimal subsidy rate

$$(17) \quad \sigma_{\Psi,L} = \frac{[\mu_R - \mu_{S_L}] S_L}{[1 + \beta(1 + 1/\epsilon_{\Psi})]}$$

where ϵ_{Ψ} is the elasticity of supply of vaccination. The optimal subsidy rate (which varies over time) is a relative price equaling the ratio of marginal external benefits of vaccination relative to the marginal external costs. The numerator is the net marginal value of an increase in vaccination in type- L patches, as vaccination converts these susceptible patches into resistant patches. Although the creation of resistant patches has no direct effects on wildlife conservation, altering patch dynamics in this manner still has value as a means of protecting wildlife from

disease. The *net price* of this service is the difference in shadow values, $[\mu_R - \mu_{S_L}]$, which should be positive since resistant patches provide greater protection than susceptible patches.

The optimal subsidy would equal the marginal benefits of disease protection if there was no opportunity cost of subsidization (i.e., if $\beta=0$ so that the denominator would equal unity). Since the opportunity costs are positive, $\beta>0$, this means a larger subsidy reduces the funds available for other conservation activities. The denominator of (17) reflects these additional costs at the margin, capturing how the agency is a monopsonist in the market for vaccination (with $1/\epsilon_\psi$ representing the degree of monopsony power) and so its demand for vaccination affects the subsidy price that must be paid to producers.

The optimal subsidy for vaccinations in type- LW patches can be derived in a similar manner

$$(18) \quad \sigma_{\psi,LW} = \frac{[\mu_{R_W} - \mu_{S_{LW}}]S_{LW}}{[1 + \beta(1 + 1/\epsilon_\psi)]}$$

The denominators of (17) and (18) are essentially the same (although the elasticities may differ somewhat in the optimal solution if these are non-constant), and so the primary differences in the subsidy rates lie in the numerators – the marginal external benefits of the subsidy. Two factors influence the differences in marginal benefits. First, we have a difference in the net prices of conversion (i.e., $[\mu_{R_W} - \mu_{S_{LW}}]$ versus $[\mu_R - \mu_{S_L}]$): other things equal, patches having a larger net price of conversion will optimally command larger vaccination subsidies because the marginal benefits of conversion are larger. Second, we have a difference in the proportion of patches of type L or LW : other things equal, vaccination subsidy rates will be larger for more abundant patch types. This is because there are more of these patches at risk of becoming infected, resulting in greater marginal benefits of protection. The net effect of these two factors (i.e.,

differences in the net price of conversion and in the proportion of patch types) on the relative subsidy rates is uncertain *a priori*.

The habitat connectivity subsidies can be derived in a similar manner. For instance, using condition (14) along with condition (10), we have

$$(19) \quad \sigma_{Z,W} = \frac{\{\mu_{S_W} S_W E + [\mu_{S_{LW}} - \mu_{S_L}] S_W S_L + [\mu_{R_W} - \mu_R] S_W R\} + [\mu_I - \mu_{S_W}] \delta I S_W}{[1 + \beta + \beta / \varepsilon_Z]}$$

where ε_Z is the elasticity of supply of habitat connectedness. This subsidy has a similar interpretation as the vaccination subsidies: it is the net marginal external benefit of increased connectivity divided by the marginal cost of subsidization. The terms in brackets $\{\}$ in the numerator represent the marginal benefits of adding wildlife to non-infected patches (a productivity effect). For each type of possible conversion of a non-wildlife patch to one that includes wildlife, the marginal benefits are calculated as the net price of conversion, times the proportion of wildlife-only patches, times the proportion of the non-wildlife patches. Note that the net price of conversion should be positive since patches with wildlife should be worth more to society than patches without wildlife. The marginal benefits are small if the net price of conversion is small, or if there are few healthy patches that wildlife can colonize, or if there are few wildlife-only patches from which colonization can occur. It may seem surprising that the marginal benefits are small with few healthy patches for wildlife to colonize, as the marginal benefits of colonizing such patches should be high. But an increase in Z_W only ensures greater migration out of W -type patches – it does not ensure that wildlife migrate to healthy patches. Most wildlife will migrate to infected patches if there are few healthy patches to be found, and this is undesirable. The last term in the numerator of (19) is negative. This term represents the marginal cost of adding wildlife to infected patches, as this hurts conservation efforts.

In general, the overall sign of the habitat connectivity subsidy (19) is ambiguous and depends on the relative magnitude of the marginal cost and benefit terms. The marginal cost term could dominate if the marginal benefits of habitat connectivity are small, in which case the subsidy would be negative to discourage connectivity. For instance, if there are few healthy patches relative to infected patches, connectivity is socially costly. The agency wants to maintain the isolation of the healthy patches. In this case, the subsidy is negative but so is the level of investment, so that the subsidy outlay is still positive, i.e., ranchers are paid to dis-invest in connectivity. But the subsidy rate and the level of investment will be positive if there are sufficient healthy patches so that connectivity is socially beneficial.

The subsidy defined by (19) and other subsidies to influence habitat connectivity are optimally patch-specific. The relative magnitudes of these subsidies is analytically ambiguous, however, as are the relative magnitudes of habitat subsidies versus vaccination subsidies. Therefore, analytic insights into the targeting of subsidy payments across activities and space are limited. A numerical example is needed to gain more insight into the economic problem of designing conservation payments in a disease setting.

Numerical example

The ecological component of our numerical example is based on the example in McCallum and Dobson (2002), which allows us to compare directly the results of our bioeconomic model with those of their ecological model. Their numerical example is based on a series of equilibrium equations that are identical to steady state versions of our equations (2)-(7) except that (i)

$\Psi_k = Z_k = 0$ in their model, and (ii) they specify the following proportional relation between the

connectivity parameters M_L and M_W : $M_W = M_V M_L$, where M_ζ is a scaling parameter.⁹

We also adopt the use of a scaling parameter for connectivity in our model, but only for the initial value of wildlife connectivity: $M_{W0} = M_\zeta M_L$. In our model, investments in Z result in a decoupling of M_L and M_W from the proportionality constant, so that M_L and M_W are no longer proportional. This means wildlife take advantage of greater connectivity and ranchers do not, even though opportunities may exist for ranchers to do so. Subsidies for increased habitat connectivity can therefore be viewed as having a secondary impact of effectively reducing livestock connectivity, at least to some extent.

McCallum and Dobson (2002) investigate four different scenarios, each described by applying a distinct set of values for all model parameters except the parameter M_L and hence M_{W0} – which we focus on as our parameter of interest. We adopt an analogous approach, but with the key difference of allowing investment choices to be made endogenously in response to the particular scenario and value of M_{W0} . M_{W0} is allowed to vary in each scenario to illustrate the relation between habitat connectivity and model outcomes for that scenario.

We focus on McCallum and Dobson’s ecological scenario a (using their parameter values, with our equilibrium calculations for this scenario presented in Figure 1) as that scenario best illustrates a case in which an endangered species can survive with moderate connectivity, but otherwise faces a high risk of extinction.¹⁰ Figure 1 illustrates that no disease outbreak

⁹ Presumably, they use this scaling parameter because an increase in connectivity of habitat for one species would also increase habitat connectivity for the other. Moreover, the scaling parameter allows them to increase connectivity by altering a single parameter value, M_L , and to therefore illustrate changes in connectivity for both species on a single axis.

¹⁰ McCallum and Dobson’s (2002) graph is qualitatively the same as ours, but some values appear to differ slightly. This could be a result of the accuracy of the numerical methods being used (we have solved the model using Mathematica 5.0; Wolfram 2005). Also note that McCallum and Dobson’s model produces multiple equilibria (our estimation of their model indicates one interior and several corner solutions), but they only present results for the

occurs under conditions of low connectivity ($M_w = M_{w0}$) because there are too few infectious contacts with the disease reservoir to support spread of the disease. The endangered species goes extinct, however, because habitat fragmentation makes it hard for the species to re-colonize extinct patches. The endangered species also goes extinct under conditions of high connectivity. The reason is that high connectivity leads to more infectious contacts with the reservoir species, creating significant disease pressures on the endangered species. With the disease causing high mortality among the endangered species and without the endangered species being able to re-colonize extinct patches quickly relative to livestock (as M_w is smaller than M_L), the result is extinction of the endangered species. Only under intermediate/moderate levels of connectivity, when the proportion of infected patches is small, can the endangered species survive.

Now consider our bioeconomic model. The economic components of the model must be specified to conduct a numerical analysis. All cost functions take on constant elasticity forms: vaccination costs in patch k are defined as $c_k = \psi_k^\eta$, and habitat connectivity costs in patch k are defined as $g_k = z_k^\xi$, where η and ξ are elasticities. Dividing the choice variables by x_s , as in the ecological model, these cost functions become $c_k = \mathbf{a}_c \Psi_k^h$ and $g_k = \alpha_g Z_k^\xi$, where $\alpha_c = x_s^\eta$ and $\alpha_g = x_s^\xi$. Existence/tourism values also take a constant elasticity form, $U = (s_w + s_{LW})^\kappa = \alpha_u (S_w + S_{LW})^\kappa$, where $\alpha_u = N^\kappa$ and κ is an elasticity. Unlike the ecological model, the economic model is not dimensionless – the values of N and x_s matter. The numerical model is fully specified by assigning values to these parameters and the elasticities η , ξ , and κ , the marginal cost term β , and the discount rate, r (see Figure 2 for values). Then it is possible to

interior equilibrium, as do we. In contrast, our bioeconomic equilibrium appears to be unique, at least for a large

numerically explore steady state solutions for the necessary conditions (2)-(7) and (13)-(15).

The results are presented in Figures 2 – 4 and Table 1.

Comparison of the bioeconomic results of Figure 2 with the ecological-only results of Figure 1 illustrates the impacts of economic investments on ecological outcomes.¹¹ The most obvious result is that the endangered species is now prevalent at both very small and very large values of M_{w0} .

Now compare the use of our two subsidies—habitat connectivity and vaccination. At low values of M_{w0} , Figure 3 illustrates that subsidies are optimally targeted at increasing wildlife habitat connectivity, which enable wildlife to re-colonize extinct patches more quickly. The habitat connectivity subsidies are initially applied at the same rate for both W -type and LW -type patches, but then become larger on LW -type patches (and on RW -type patches, once disease outbreak occurs) as these become the most prevalent type of patch that the endangered species occupies. Table 1 indicates that the investments in wildlife habitat connectivity, Z_W and Z_{LW} , are substantial in relation to M_{w0} when M_{w0} is low. For instance, Z_W and Z_{LW} are 189 percent of M_{w0} when $M_{w0}=1$. These levels decline, however, for larger values of M_{w0} as the marginal benefits of connectivity decline. Initially this decline is because there are diminishing marginal returns to connectivity for larger values of M_{w0} . A second effect contributes to this decline after the disease establishes: as the number of infected patches increases, greater connectivity may lead to more movement into infected patches.

range of initial values being applied in the numerical approximation algorithm.

¹¹ Although we do not present bioeconomic results for comparison with McCallum and Dobson's other scenarios b - d , the resulting comparisons are qualitatively similar to those of scenario a . Quantitative differences do arise, however, in that there are fewer incentives to subsidize conservation activities when endangered species are not really in danger (scenario b and a range of scenario c), and there are greater incentives to subsidize conservation when the endangered species is in even more danger (scenario d and part of scenario c).

Vaccination subsidies are introduced when M_{w0} becomes large enough for a disease outbreak to occur. These subsidies initially increase as M_{w0} increases, with a corresponding decrease in habitat connectivity subsidies. This substitution of vaccination subsidies for connectivity subsidies is illustrated in both Figures 3 and 4. Vaccination subsidies initially increase for larger values of M_{w0} because the marginal benefits of vaccination initially increase as more patches become infected. Vaccination increases the safety of endangered species even at high levels of connectivity by reducing the proportion of infected patches and increasing the proportion of resistant patches (compare Figures 1 and 2), thereby significantly reducing infectious contacts relative to the case of no vaccination. But even vaccination is not immune to diminishing marginal returns, and eventually the subsidy rates begin to taper off. Figure 3 illustrates that vaccination subsidies are targeted more heavily at L -type patches as their relative abundance puts these patches at the most risk for new infections from migrating infected herds.

The overall picture of conservation and disease management within our model is as follows: conservation payments are best targeted towards habitat connectivity when disease risks are initially low, and they are best targeted towards preventing infectious contacts through vaccination when risks are initially high. One reason this result arises is that disease control in this model is a *weakest-link public good* – every patch but one could be fully vaccinated, but it only takes that one susceptible patch to become infected and spread the disease to other wildlife patches (which are equally likely to become infected), where local extinction occurs immediately (Perrings et al. 2002). If these patches remain fragmented, any local extinction is likely to be permanent because re-colonization by healthy wildlife is not likely to occur. Therefore, when fragmentation is extensive, even an aggressive livestock vaccination program is unlikely to significantly reduce risks to endangered species. Rather, vaccination only becomes an effective

risk-reduction strategy once habitats are sufficiently connected and disease risks are significant.

An interesting issue emerges in this model regarding the role of habitat connectivity and vaccination as prevention and control strategies against disease. Traditionally, prevention of disease involves keeping the disease away (i.e., lower chance of illness); whereas control of disease involves minimizing the damage of the disease once it is realized (see for example Leung et al. 2002). This distinction fits into the self-protection and self-insurance framework created by Ehrlich and Becker (1972) three decades ago. Prevention is self-protection; control is self-insurance. But as pointed out by Ehrlich and Becker, sometimes this distinction is rather blurry. That is the case for our model with habitat connectivity and vaccination. Here prevention would imply keeping the disease out of any given patch; control would imply minimizing damages if the disease makes it to any given patch. But habitat connectivity has both effects. If the policy decides not to subsidize connectivity, there is no disease spread, which is a preventive strategy. But if they promote connectivity, the disease might spread but the damage can be mitigated because of the increased population that arises, which is control strategy. Similarly, with vaccination, livestock is immune to a disease (which may or may not be an issue), which again implies both prevention of its spread and control, in that the disease has no impact and dies away. For the situation considered herein, methods of prevention and control are intertwined, and one cannot make general claims about “preventive ounces” versus “pounds of cure”.

Distributional impacts

Table 1 presents the equilibrium welfare results. Conservation costs are smallest when the wildlife stock is at its healthiest level and the disease is just starting to take hold. Costs are also smaller when initial levels of connectivity are fairly sufficient and ranchers can focus more of

their efforts on vaccination. Ranchers are willing to bear these costs because the subsidies generate rents for them. These rents are double the costs incurred for the numerical example because the marginal cost function for each activity is linear and increasing in the level of the activity. The rents would be more than double if the marginal costs were increasing at an increasing rate, as might be expected.

Additionally, the subsidies lead to a slight increase in the total number of patches containing livestock (compare Figures 1 and 2, although the differences might be too small to discern visually). We do not explicitly model any benefits associated with more livestock patches, but it is reasonable to assume they would exist. Similarly, although we do not model any private benefits to ranchers from having a healthy stock of domestic animals, it is likely that at least some benefits would accrue in reality. If so, the private benefits of subsidized vaccination would be even greater.

Concluding remarks

Herein we build a stylized model to gain insights into the application of conservation payments to protect endangered species in the face of wildlife-livestock disease risks and habitat fragmentation. Greater connectivity of habitat creates an endogenous trade-off. More connectedness ups the chance that populations of endangered species will grow more rapidly; however, greater connectivity also increases the likelihood that diseases will spread more quickly. We derive a habitat connectedness subsidy based on the net marginal external benefit of increased connectivity divided by the marginal opportunity cost of subsidization, and we derive a similar expression for a vaccination subsidy. Our results suggest that the cost-effective policy is to initially subsidize habitat connectivity rather than vaccinations; this increases the

contiguosness of habitat, which eventually also increases disease risks. Once habitat is sufficiently connected, disease risks increase to such a degree to make a vaccination subsidy worthwhile. Highly connected habitat requires nearly all the government budget be devoted to vaccination subsidies. The result of the conservation payments is significantly increased species abundance, for a wide range of initial levels of habitat connectedness.

Our model has three obvious caveats, which do not easily allow for insights into specific issues. First, the “island” metapopulation model is a restriction both for ecological relations and for the ability to target management activities. For instance, in reality, one could develop corridors between specific patch types but not between others due to economic and physical constraints. This would change the basic model from an island model to something else (maybe a “necklace”). Second, metapopulation modeling based on the proportion of patches in various states is elegant for ecological modeling and for bioeconomic maximization because it is parsimonious in state variables, but it creates complications when trying to incorporate management because we cannot keep track of individual patches. We do not know where these patches are located in space, and we don’t know which patch is in which category at any particular point in time. Finally, ranchers have many other choices that could be included, at some loss in model parsimony. Future work should look to create powerful and tractable ways to relax these current restrictions on modeling disease risk between wildlife and livestock.

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Table 1. Equilibrium outcomes for welfare measures and rancher investment choices

Initial connectivity (M_{W0})	Net Social Welfare	Recreation/ tourism benefits	Vaccination and habitat management costs	Subsidy payments	Connectivity investment choices			Vaccination choices	
					Z_W	Z_{LW}	Z_{RW}	Ψ_L	Ψ_{LW}
0	0	0	0	0	0	0	0	0	0
1	0.69	1.76	1.43	0.72	1.89	1.89	0	0	0
2	1.58	2	0.55	0.27	0.52	1.57	0	0	0
3	2.02	2.15	0.17	0.09	0.14	0.91	0.01	0.09	0
4	1.67	1.89	0.3	0.15	-0.01	0.38	0.1	1.02	0.53
5	1.18	1.43	0.34	0.17	0	0.32	0.13	1.15	0.47
6	0.87	1.11	0.33	0.16	0.02	0.3	0.17	1.16	0.4
7	0.66	0.89	0.31	0.15	0.03	0.27	0.2	1.13	0.37
8	0.53	0.73	0.27	0.14	0.03	0.24	0.22	1.06	0.35

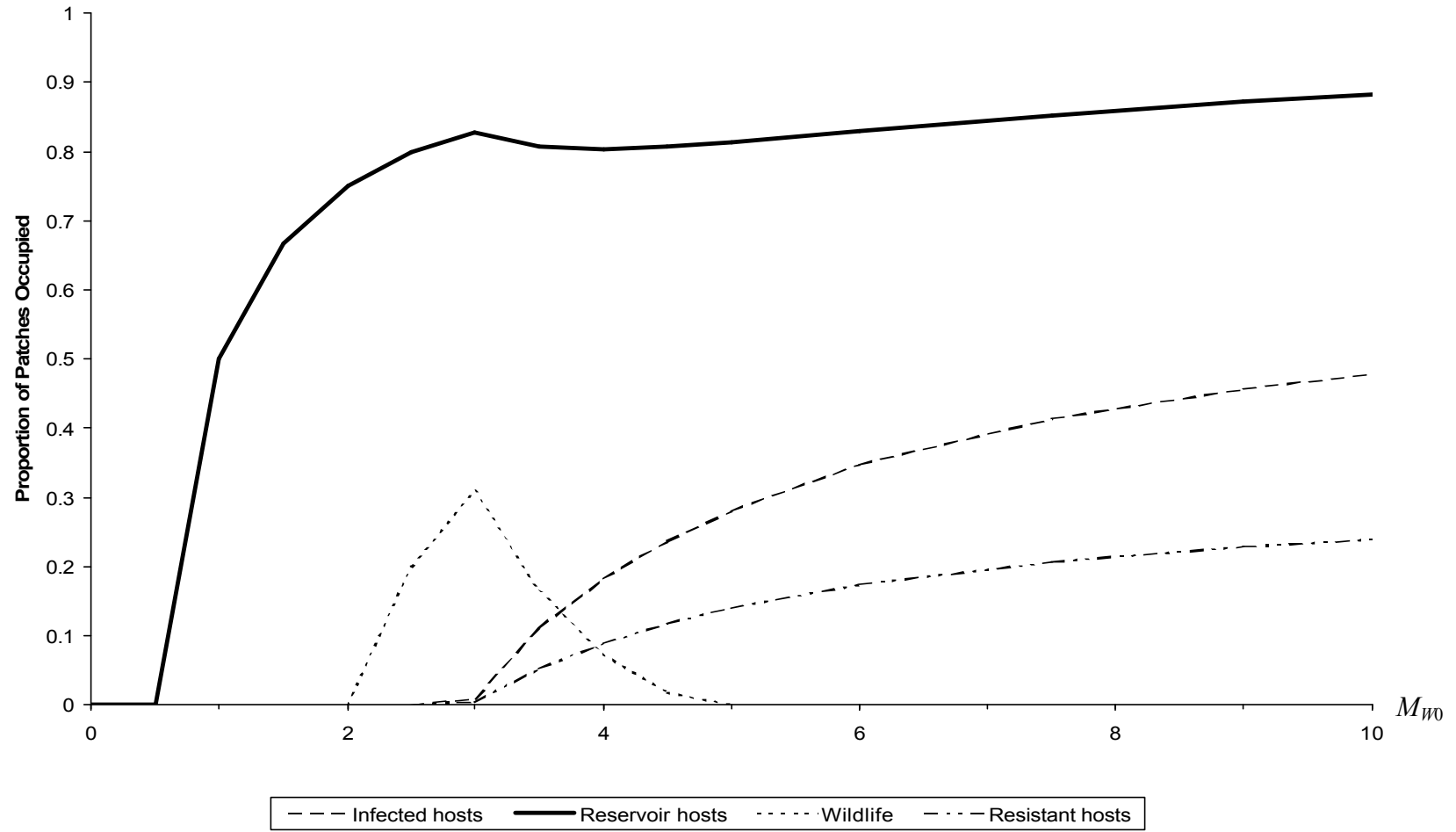


Figure 1. McCallum and Dobson's ecological scenario *a*. Parameter values: $\delta=0.8$, $X_i=4.0$, $X_r=4.0$, $X_W=4.0$, $M_\zeta=4.0$, $\Gamma=1.0$, $\Lambda=2.0$. Reservoir hosts are defined as all patches including livestock. Wildlife is defined as all patches including wildlife. Resistant hosts include all patches with resistant livestock.

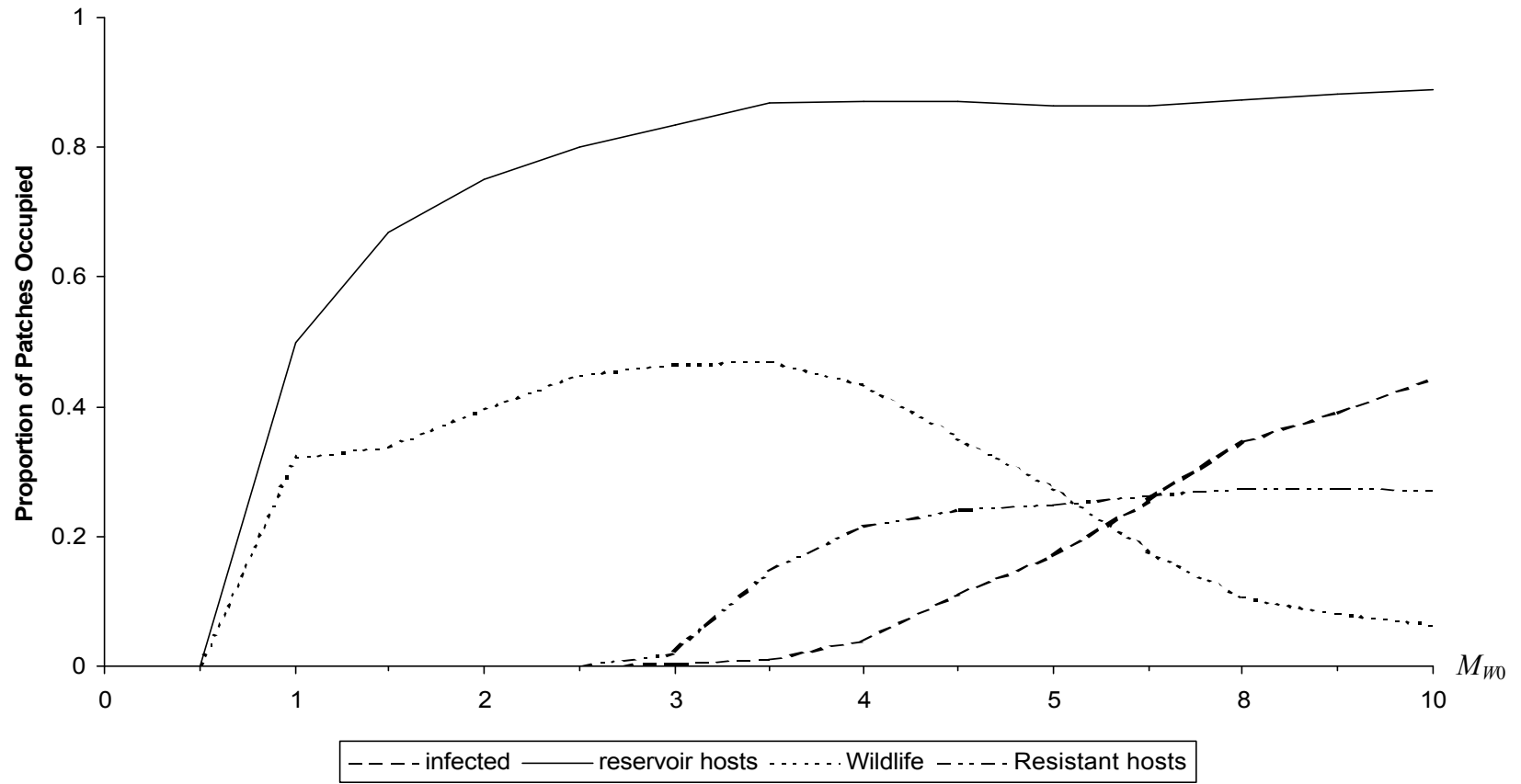


Figure 2. Bioeconomic results. Parameter values: $\eta=2$, $\xi=2$, $\kappa=0.5$, $\beta=0.25$, $x_s=0.1$, $N=10.0$, $\phi=0.05$, all other parameters as in Figure 1.

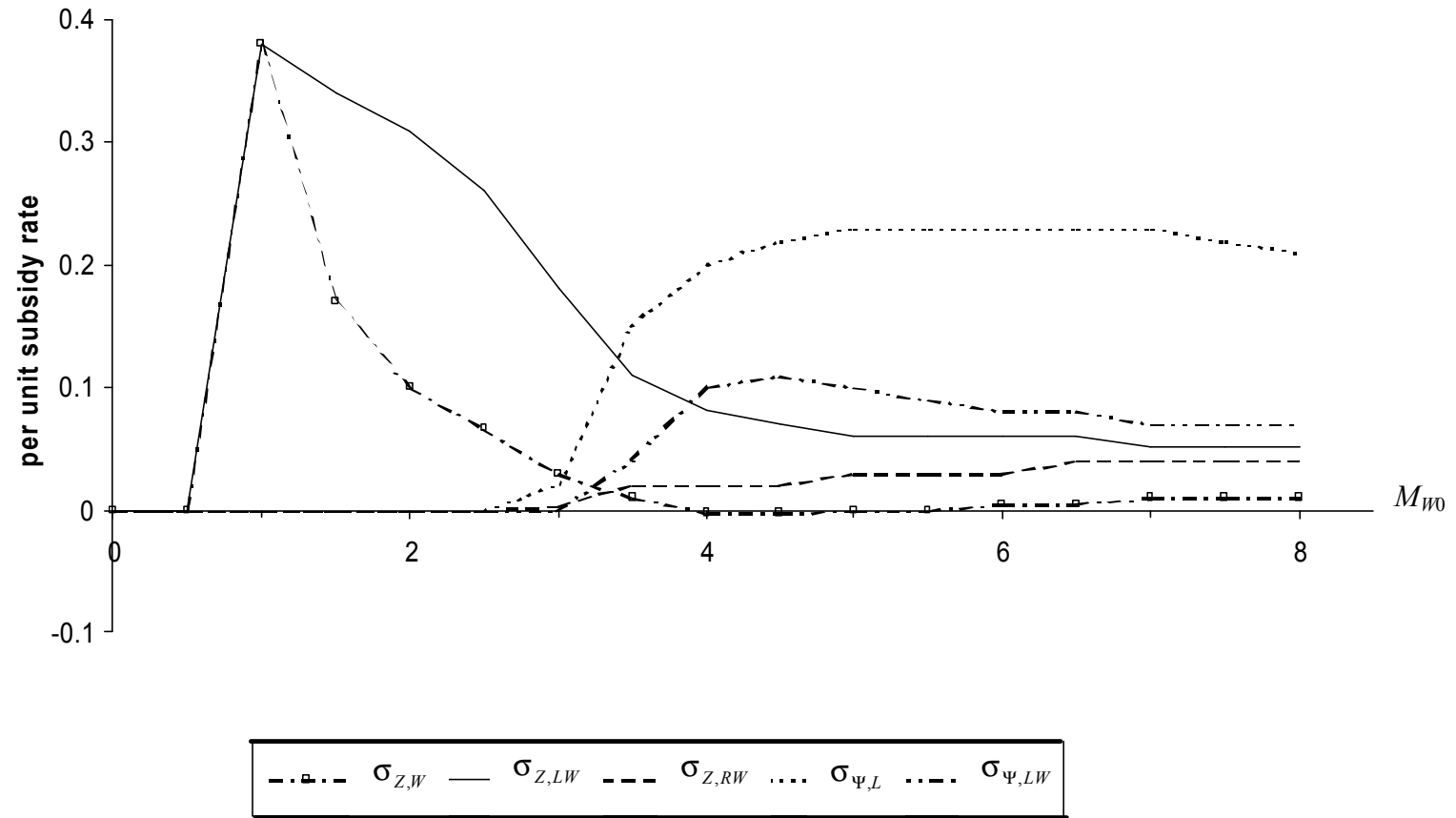


Figure 3. Optimal subsidies from bioeconomic model. Parameters the same as in Figures 1 and 2.

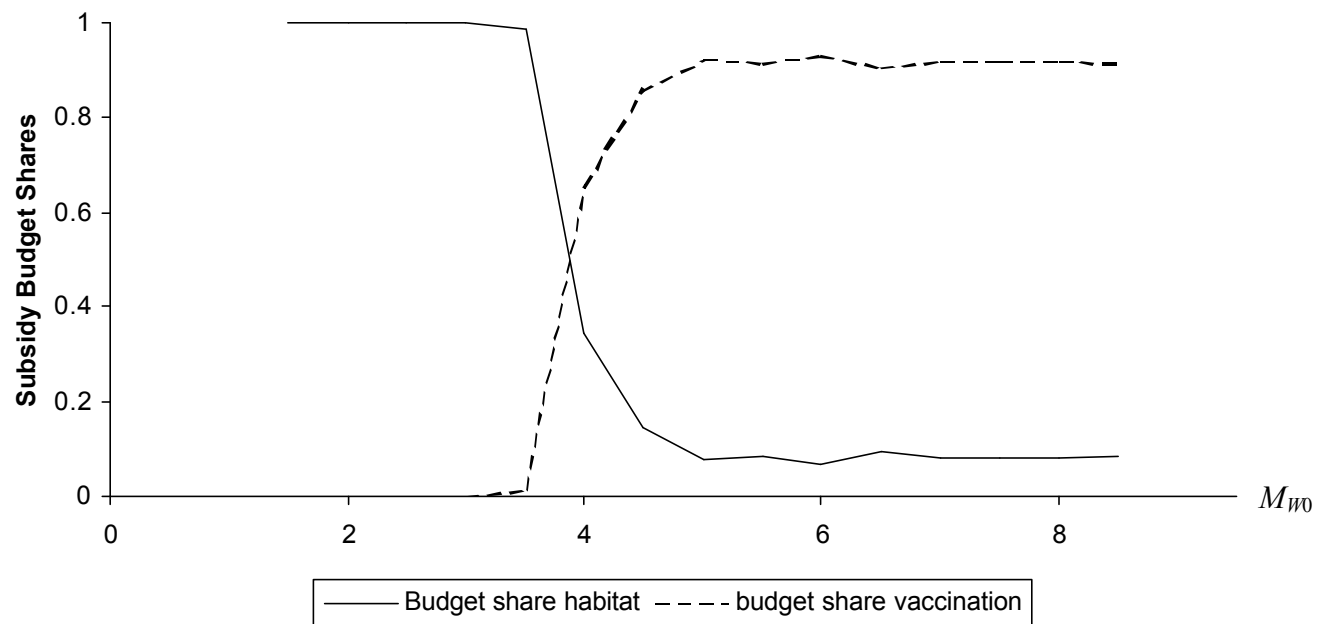


Figure 4. Optimal shares of subsidy payments. Parameters the same as in Figures 1 and 2.