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## ***Spatial Management of Wildlife Disease\****

**Richard Horan, Christopher A. Wolf, Eli P. Fenichel,  
and Kenneth H. Mathews, Jr.**

The spread of wildlife diseases is a major threat to livestock, human health, resource-based recreation, and biodiversity conservation (Cleaveland, Laurenson, and Taylor). The development of economically sound wildlife disease-management strategies requires an understanding of the links between ecological functions (e.g., disease transmission and wildlife dispersal) and economic choices, and the associated tradeoffs. Spatial linkages are particularly relevant. Yet while ecologists have long-argued that space is important (Hudson et al.), prior economic work has largely ignored spatial issues.

For instance, Horan and Wolf analyzed a case study of bovine tuberculosis (bTB) in Michigan deer, a problem where the disease appears to be confined to a single, spatially confined, wildlife population—an island. But wildlife disease matters generally are not spatially confined. Barlow, in analyzing bTB in possums in New Zealand, accounted for immigration of susceptible possums into a disease reservoir. However, he modeled immigration as fixed and unaffected by management. Bicknell, Wilen, and Howitt, also focusing on possums in New Zealand, developed a model that incorporates simple density-dependent net migration. This allowed the authors to account for endogenous immigration when deriving optimal culling strategies. However,

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they did not consider the role of management outside of the disease reservoir or disease spread.

In this article, we develop a simple spatial model to illustrate how harvest strategies, inside and outside a disease reservoir, could affect disease prevalence rates within the reservoir, dispersion into new areas, and the associated economic tradeoffs.

## A Spatial Model

We adopt a metapopulation model to analyze spatial wildlife interactions and disease transmission (Hudson et al.). These models split wildlife into spatially-distinct populations (each residing in a “patch” of habitat) that have infrequent contact relative to contact within a population. For simplicity, we consider a two-patch model, although the model could easily be extended to incorporate additional patches.

The two patches are denoted by  $m$  and  $n$ , and indexed by  $j$ . The aggregate wildlife population level in patch  $j$  is  $X_j$ . Each patch contains two sub-populations,  $X_{jS}$  and  $X_{jI}$  ( $j = m, n$ ), where the subscripts  $S$  and  $I$  denote susceptible and infected, respectively. Net changes in population levels depend on density-dependent growth, disease-related mortality (only for infected animals), migration, disease transmission, and harvests. Density-dependent growth for population  $X_{ji}$  ( $j = m, n; i = S, I$ ) is  $rX_{ji}g_j(X_j)$ , where  $r$  is the intrinsic growth rate and  $g_j(X_j)$  is a density-dependent function with  $g_j(0) = 1$  and  $g_j(K_j) = 0$ , where  $K_j$  is the carrying capacity of patch  $j$  (e.g., for logistic growth,  $g_j = [1 - X_j/K_j]$ ). Disease-related mortality is  $\alpha_j X_{jI}$ , where  $\alpha_j$  is the disease mortality rate for patch  $j$ .

Migration is modeled using Bulte and van Kooten’s generalization of the spatial model proposed by Sanchirico and Wilen. Specifically, migration of animals of health status  $i$  into patch  $j$  from patch  $l \neq j$  is  $\eta_{l,j}(K_j - X_j)X_{li}$ , where  $\eta_{l,j}$  is a parameter. This specification “subsumes most common biological interconnections as described in the ecological literature” (Bulte and van Kooten, p. 295), such as sink-source migration (i.e., one-way migration), fully-integrated migration, and, if the model involved more than two patches, it would also capture limited-distance migration (Bulte and van Kooten). It also distinguishes between migration from patch  $m$  to  $n$ , and *vice versa* (as opposed to simply net migration), which is necessary for capturing cross-patch disease movement.

Disease transmission involves contact between infected and susceptible individuals residing in the same patch at time  $t$ , given the continuous nature of the model. Denote transmission in patch  $j$  by  $\beta_j(X_j, X_{jI}, X_{jS})$  (see McCallum, Barlow, and Hone).

Finally, harvests within a patch are nonselective with respect to health status, as it is often not possible to identify infected animals prior to the kill (Williams et al.). In this setting, a manager can only choose the aggregate harvest from a patch,  $h_j$ , with the harvest from each health class depending on the proportion of animals in that stock relative to the aggregate population  $X_j$ . That is, harvests of healthy animals from patch  $j$  are  $h_{jS} = h_j X_{jS}/X_j$ , and harvests of infected animals from patch  $j$  are  $h_{jI} = h_j X_{jI}/X_j$ .

Net growth of healthy and infected populations in patch  $m$  is:

$$(1) \quad \dot{X}_{mS} = r X_{mS} g_m(X_m) - \eta_{m,n}(K_n - X_n) X_{mS} + \eta_{n,m}(K_m - X_m) X_{nS} - \beta_m(\bullet) - h_{mS}$$

$$(2) \quad \dot{X}_{mI} = r X_{mI} g_m(X_m) - \alpha_m X_{mI} - \eta_{m,n}(K_n - X_n) X_{mI} + \eta_{n,m}(K_m - X_m) X_{nI} + \beta_m(\bullet) - h_{mI}.$$

It is more intuitive and convenient to work with the variables  $X_j$  and  $\theta_j = X_{jI}/X_j$ , where  $\theta_j$  is the disease prevalence rate in patch  $j$ . After making the appropriate transformations and defining  $G_j = r X_j g_j(X_j)$ , the model becomes (with analogous equations for patch  $n$ ):

$$(3) \quad \dot{X}_m = G_m(X_m) - \alpha_m \theta_m X_m - \eta_{m,n}(K_n - X_n) X_m + \eta_{n,m}(K_m - X_m) X_n - h_m$$

$$(4) \quad \dot{\theta}_m = \beta_m(X_m, X_{mI}, X_{mS})/X_m - \alpha_m(1 - \theta_m)\theta_m + \eta_{n,m}(K_m - X_m)(\theta_n - \theta_m)X_n/X_m.$$

From equation (4), intertemporal changes in  $\theta_m$  depend on transmission, disease-related mortality, and immigration. Harvests only indirectly influence prevalence via changes in population levels. If harvests could be made selectively, then  $h_{mI}$  would directly reduce prevalence. But prevalence cannot be targeted directly when harvests are nonselective.

### Impacts of Harvesting and Space on Prevalence

To understand the role of harvesting in a spatial setting, we first develop a point of comparison by assuming there is no migration. We also specify  $\beta_j(\bullet)$  by adopting the following transmission function, based on one proposed by McCallum, Barlow, and Hone:

$$(5) \quad (1 - \epsilon_j + \epsilon_j X_j)\beta_j X_{jS} X_{jI}/X_j = (1 - \epsilon_j + \epsilon_j X_j)\beta_j \theta_j (1 - \theta_j) X_j,$$

where  $\beta_j$  is now a parameter defining the contact rate per infectious animal, and  $\epsilon_j \in [0, 1]$  is a parameter that determines the degree of density dependence in transmission ( $\epsilon_{ij} = 1$  implies density-dependence, while  $\epsilon_{ij} = 0$  implies density-independence).

Without migration, equation (4) becomes:

$$(6) \quad \dot{\theta}_j = ([1 - \epsilon_j + \epsilon_j X_j]\beta_j - \alpha_j)(1 - \theta_j)\theta_j.$$

In the absence of the spatial terms and assuming  $\beta_j > \alpha_j$  and  $\epsilon_j > 0$  (so that the disease can be persistent), there exists a population threshold,  $X_j^* = (\alpha_j - [1 - \epsilon_j]\beta_j)/(\beta_j \epsilon_j)$ , so that  $\theta_j \rightarrow 0$  when  $X_j < X_j^*$  and  $\theta_j \rightarrow 1$  when  $X_j > X_j^*$ . The assumed existence of a threshold is the primary reason wildlife disease programs have focused on reducing wildlife density. Empirical evidence for many species suggests that  $\epsilon_j < 1$  (McCallum, Barlow, and Hone). This has important implications for harvest-based strategies, because  $\partial X_j^*/\partial \epsilon_j < 0$ ,

implying larger control costs for smaller values of  $\epsilon_j$ . At some value of  $\epsilon_j$ ,  $X_j^* = 0$  and extermination of the population is the only way to control the disease.

When we allow for spatial interactions, then equation (6) becomes (for  $j = m$ )

$$(7) \quad \dot{\theta}_m = ([1 - \epsilon_m + \epsilon_m X_m] \beta_m - \alpha_m)(1 - \theta_m) \theta_m + \eta_{n,m}(K_m - X_m)(\theta_n - \theta_m) X_n / X_m.$$

Assuming that  $K_m \geq X_m$ , the effect of immigration on  $\dot{\theta}_m$  depends on whether  $\theta_m > \theta_n$  or  $\theta_m < \theta_n$ . If  $\theta_m < \theta_n$ , then immigration increases  $\dot{\theta}_m$ . Hence, reduced immigration, through increases in  $X_m$  (by reducing  $h_m$ ) and reductions in  $X_n$  (by increasing  $h_n$ ), reduces prevalence. These effects are in contrast to, and possibly larger than, the effects that a larger population  $X_m$  has on transmission. Indeed, the threshold  $X_m^*$  may cease to exist when  $\theta_n > \theta_m$  and  $\theta_m$  is small, rendering harvest strategies in patch  $m$  ineffective. To see this, assume  $\theta_n > \theta_m$  and note that the first two terms in (7) approach zero as  $\theta_m \rightarrow 0$ , while the final term is positive so that  $\dot{\theta}_m > 0 \forall X_m < K_m$  (indeed, the final term carries more weight, the smaller is  $X_m$ ). The ability to control the disease in patch  $m$  in this case will depend critically on the management choices in patch  $n$ .

Alternatively, immigration will diminish  $\dot{\theta}_m$  if  $\theta_m > \theta_n$ . In this case, increased immigration, through reductions in  $X_m$  and increases in  $X_n$ , reduces prevalence. Immigration in this case increases  $X_m^*$  (possibly to  $K_m$  when  $\theta_m$  is small). This occurs, for instance, under one-way migration (i.e.,  $\eta_{m,n} = 0$ ; the case considered by Barlow) as healthy animals immigrate into patch  $m$  and help crowd out infected ones. Sufficient levels of sustained immigration could push  $\theta_m \rightarrow 0$ .

Finally, note that the immigration term in patch  $m$  is positive when the immigration term in patch  $n$  is negative. This linkage means that strategies to reduce prevalence in one patch may have the opposite effect in the other patch. Moreover, the relative magnitudes of  $\theta_m$  and  $\theta_n$  may change over time, so that cross-patch tradeoffs are nonstationary. An economic objective can help us to sort out intertemporal and spatial tradeoffs, in order to develop socially desirable strategies.

### Economically Optimal Management

Now consider the economic tradeoffs associated with disease control. Assume a harvest price of  $p$  and that only healthy wildlife are valued, so that patch  $j$  harvests have a value of  $p(1 - \theta_j)h_j$ . Harvest costs in patch  $j$  are  $c_j(X_j)h_j$ . Finally, the external damage costs (e.g., livestock sector impacts) associated with infected wildlife are an increasing function of the infected wildlife in each patch,  $D(X_n\theta_n, X_m\theta_m)$ .

Given this specification, the social planner's problem is

$$(8) \quad \max_{h_n, h_m} \int_0^\infty \left\{ \sum_{j=m,n} [p(1 - \theta_j)h_j - c_j(X_j)h_j] - D(X_n\theta_n, X_m\theta_m) \right\} e^{-\rho t} dt$$

s.t. (3), (4), and analogous conditions for patch  $n$

where  $\rho$  is the discount rate. The Hamiltonian for this problem is

$$(9) \quad H = \sum_{j=m,n} [p(1 - \theta_j)h_j - c_j(X_j)h_j] - D(X_n\theta_n, X_m\theta_m) + \sum_{j=m,n} \lambda_j \dot{X}_j + \sum_{j=m,n} \phi_j \dot{\theta}_j.$$

We focus on the singular solution, for which the necessary conditions are (for  $j = m$ ; analogous conditions arise for  $j = n$ ):

$$(10) \quad \lambda_m = p(1 - \theta_m) - c_m(X_m)$$

$$(11) \quad \dot{\lambda}_m = \rho \lambda_m + c'_m(X_m)h_m + \frac{\partial D}{\partial(X_m\theta_m)}\theta_m - \lambda_m \frac{\partial \dot{X}_m}{\partial X_m} - \lambda_n \frac{\partial \dot{X}_n}{\partial X_m} - \phi_m \frac{\partial \dot{\theta}_m}{\partial X_m} - \phi_n \frac{\partial \dot{\theta}_n}{\partial X_m}$$

$$(12) \quad \dot{\phi}_m = \rho \phi_m + ph_m + \frac{\partial D}{\partial(X_m\theta_m)}X_m - \lambda_m \frac{\partial \dot{X}_m}{\partial \theta_m} - \lambda_n \frac{\partial \dot{X}_n}{\partial \theta_m} - \phi_m \frac{\partial \dot{\theta}_m}{\partial \theta_m} - \phi_n \frac{\partial \dot{\theta}_n}{\partial \theta_m},$$

where condition (10) comes from  $\partial H/\partial h_m = 0$ . The time derivative of (10) yields

$$(13) \quad \dot{\lambda}_m = -p\dot{\theta}_m - c'_m(X_m)\dot{X}_m.$$

Substitute  $\lambda_m$  and  $\dot{\lambda}_m$  from (10) and (13) into (11) to obtain the implicit form condition:

$$(14) \quad \Psi_m(X_m, X_n, \theta_m, \theta_n, \phi_m, \phi_n) = 0.$$

Unlike standard linear control models, condition (14) is a function of co-state variables. Equation (14) and its analog for  $j = n$  can be solved for  $\phi_j(X_m, X_n, \theta_m, \theta_n)$ ,  $j = m, n$ , and these relations can then be plugged back into (11) and (12). Next, take the time derivatives of  $\phi_m(X_m, X_n, \theta_m, \theta_n)$  and set them equal to condition (12) to obtain  $\Phi_m(X_m, X_n, \theta_m, \theta_n, h_m, h_n) = 0$ . This expression, along with an analogous one derived for  $j = n$ , can be solved for nonlinear feedback rules for harvests in each patch,  $h_j(X_m, X_n, \theta_m, \theta_n)$  ( $j = m, n$ ).

The harvest rules can be substituted into the equations of motion (3) and (4) to create a system of differential equations that can be solved numerically given the starting values  $X_j^0, \theta_j^0$  ( $j = m, n$ ). The solution, therefore, depends on the values of the initial state variables. This means the singular solution can possibly (but not necessarily, in the case of multiple solutions) be pursued from the initial time period, without the need for bang-bang controls to move to the singular solution. Moreover, different initial states imply different optimal paths (see also Horan and Wolf).

In traditional linear control models, bang-bang controls are used to move to a singular path that is independent of the initial states (Conrad and Clark). Our state-dependent solution arises because we are using a single control (nonselective harvest) to affect multiple state variables (population and prevalence). The result is that the control is imperfect (second-best), as decisions

to control one state variable create unintended consequences related to control of the other, and *vice versa*. In a sense, the imperfect control creates adjustment costs, and these costs lead to the optimality of nonlinear adjustment—similar to a model in which convex adjustment costs are explicit.

### Numerical Example

A numerical example can provide additional insight into the role of space in wildlife disease management. Assume  $\rho = 0.05$  and the economic parameters are identical in each patch, with  $p = 5$ ,  $c(X) = 50/X$ , and constant marginal damages equal to 5. Initially, patch  $m$  is the diseased patch and patch  $n$  is disease-free. Population growth in each patch is logistic and identical with  $r = 0.2$  and  $K = 100$ . Disease mortalities are  $\alpha_m = 0.1$  and  $\alpha_n = 0.05$ , and transmission parameters are  $\beta_m = 0.005$ ,  $\beta_n = 0.0005$ , and  $\varepsilon_j = 0.7$  ( $j = m, n$ ). Without migration, this specification yields  $X_m^* = 28.1$ , so that the disease can be eradicated in patch  $m$  if the stock is held below 28.1% of  $K_m$ , and  $X_n^* = 142 > K_n$ , so that the disease cannot even establish in patch  $n$ .

We begin with the case of no migration, assuming  $\theta_m^0 = 0.02$ . In this case, we find an economic threshold of  $X_m^{**} = 29$ . If  $X_m^0 < X_m^{**}$ , then it is optimal to follow the singular path that begins at  $X_m^0$  and remain on this path until the disease is eradicated; if  $X_m^0 > X_m^{**}$ , then it is optimal to initially jump to  $X_m^{**}$  and then follow the singular path emerging from this point until the disease is eradicated. The economic threshold  $X_m^{**}$  does vary inversely with  $\theta_m^0$ , however. For instance,  $X_m^{**} = 19$  when  $\theta_m^0 = 0.05$ .

Now suppose, there is one-way migration from the healthy patch  $n$  to the diseased patch  $m$ , with  $\eta_{n,m} = 0.002$  and  $\eta_{m,n} = 0$ . In this case, immigration can increase the ecological threshold  $X_m^*$ , although this depends on the management strategy in patch  $n$ . For instance, if we set  $h_n = 0$  until the disease was eradicated so that there was maximum immigration into patch  $m$ , then  $X_m^* = 54$  (note that  $X_m^*$  does not depend on  $X_n^0$  or  $\theta_m^0$ , although the speed at which the disease vanishes does depend on  $X_n^0$  and  $\theta_m^0$ ). But fixing  $h_n = 0$  is a costly and sub-optimal approach to management in patch  $n$ , particularly if the disease is slow to die out. It would be possible to set  $h_n > 0$  and still have  $X_m^* > 28.1$ , potentially resulting in a shorter disease eradication time than when there is no migration (but a longer eradication time than if  $h_n = 0$ ).

But what of the economic threshold  $X_m^{**}$  for the case of one-way migration? Is  $X_m^{**} > X_m^*$ , as in the no-migration case? The answer is no, although the optimal rule is the same. Specifically, we find that  $X_m^{**} = 0$  (regardless of the values of  $X_m^0$ ,  $X_n^0$ , and  $\theta_m^0$ ), with the optimal rule being: if  $X_m^0 > X_m^{**}$  (which is always the case), then an initial jump to  $X_m^{**}$  is optimal. After wildlife, and hence the disease, are exterminated in patch  $m$ , immigration from patch  $n$  repopulates patch  $m$ . Repopulation obviously cannot occur in the no-migration model; hence it was optimal to slowly wait out the disease in that case. But with migration, the opportunity to naturally repopulate patch  $m$  makes immediate extermination an optimal plan. The disease then is eradicated quickly, so as to avert damages. Moreover, management in each patch immediately reverts to a first-best strategy, as harvests no longer must control both population and disease.

Finally, consider the case of two-way migration, with  $\eta_{n,m} = \eta_{m,n} = 0.002$  (although we tried many parameter values and the results were unaffected). In

this case, equation (7) indicates that  $\theta_m > \theta_n$  helps to increase the threshold  $X_m^*$  (as verified above), but the opposite occurs in patch  $n$  as  $X_n^*$  is reduced. This is a concern because, while  $X_m^*$  can be viewed as the threshold for disease eradication (as  $X_m$  is reduced below this value) when  $\theta_m^0 > 0$ ,  $X_n^*$  is the threshold for disease establishment (as  $X_n$  is increased above this value) when  $\theta_n^0 = 0$  and there is immigration of diseased animals. Indeed, we find that immigration enables the disease to establish in patch  $n$ , given our parameter values. This provides even greater incentives for immediately exterminating  $X_m$  in our model, and maintaining  $X_m = 0$  until any disease in patch  $n$  vanishes on its own (since  $X_m = 0$  implies no migration from patch  $m$  to patch  $n$ , so that  $X_n^* > K_n$  and the disease cannot persist in patch  $n$ ).

## Discussion

Economic and ecological systems are spatially connected and jointly determined. It is, therefore, not surprising that the efficiency of disease control is improved by developing policies around economic thresholds as opposed to ecological thresholds, and that these economic thresholds and the associated optimal management strategies can change significantly when dealing with spatially interacting systems.

Our numerical results generally support the types of tradeoffs that we identified in our discussion of the general ecological model, and they are consistent with economic intuition. Still, we cannot say how robust our numerical findings are, as many other permutations of the model could be considered. We did examine a number of other qualitatively similar parameter combinations and found similar results to those presented here, although space limitations prevent us from reporting these.

Some qualitatively different formulations could also be interesting to analyze, and we leave this for future work. For instance, the case where the disease parameters in patch  $n$  are such that  $X_n^* < K_n$  in the absence of migration would be interesting, as the disease could then persist in patch  $n$  even if  $X_m = 0$ . Expanding the number of patches to more than two is also of obvious interest. Also excluded from the model are nonmarket values related to health of wildlife stocks (existence values) or individual animals (humane values) (Horan and Shortle). Such values might be particularly relevant when diseases threaten large, charismatic mammals, or endangered species. For example, public outcry associated with implementation of the rapid, stamping-out policy to eradicate foot and mouth disease in 2003 in the United Kingdom resulted in policy modifications.

Other fruitful areas for future research include consideration of how spatially-distributed nonharvest activities, such as supplemental feeding of wildlife (see Horan and Wolf) and land-use decisions, influence animal movement and disease spread. These are issues of growing importance, and recognizing how they affect relevant ecological processes can only lead to improved disease management. A final important spatial issue is how increased human encroachment into wild lands has increased human–livestock–wildlife contacts, affecting the evolution of infectious diseases and the potential for greater cross-species transmission. Indeed, 61% of human diseases are zoonotic

in origin, and most emerging diseases in humans involve free-ranging wildlife (Cleaveland, Laurenson, and Taylor).

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## References

- Barlow, N. "Control of Endemic Bovine TB in New Zealand Possum Populations: Results from a Simple Model." *J. Appl. Ecol.* 28(1991):794–809.
- Bicknell, K.B., J.E. Wilen, and R.E. Howitt. "Public Policy on Private Incentives for Livestock Disease Control." *Aust. J. Agr. Resour. Ec.* 43(1999):501–21.
- Bulte, E., and G.C. van Kooten. "Metapopulation Dynamics and Stochastic Bioeconomic Modeling." *Ecol. Econom.* 30(1999):293–99.
- Cleaveland, S., M. Laurenson, and L. Taylor. "Disease of Humans and Their Domestic Mammals: Pathogen Characteristics, Host Range, and the Risk of Emergence." *Philos. T. Roy. Soc. B* 356(2001):991–99.
- Conrad, J.W., and C.W. Clark. *Natural Resource Economics: Notes and Problems*. New York: Cambridge University Press, 1987.
- Horan, R.D., and C.A. Wolf. "The Economics of Managing Infectious Wildlife Disease." *Am. J. Agr. Econ.*, forthcoming.
- Horan, R.D., and J.S. Shortle. "Optimal Management of Multiple Renewable Resource Stocks: An Application to Minke Whales." *Environ. Resour. Econ.* 13(1999):435–58.
- Hudson, P., A. Rizzoli, B. Grenfell, H. Heesterbeck, and A. Dobson, eds. *The Ecology of Wildlife Diseases*. New York: Oxford University Press, 2002.
- McCallum, H., N. Barlow, and J. Hone. "How Should Pathogen Transmission be Modelled?" *Trends Ecol. Evol.* 16(2001):295–300.
- Sanchirico, J., and J.E. Wilen. "Bioeconomics of Spatial Exploitation in a Patchy Environment." *J. Environ. Econom. Management* 37(1999):129–50.
- Williams, E.S., M.W. Miller, T.J. Kreeger, R.H. Kahn, and E.T. Thorne. "Chronic Wasting Disease of Deer and Elk: A Review with Recommendations for Management." *J. Wildlife Manage.* 66(2002):551–63.

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