# Jointly-Determined Livestock Disease Dynamics and Decentralized Economic Behavior

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Endemic livestock diseases impose significant costs on society (Bennett 1992, 2003; Bennett, Christiansen and Clifton-Hadley 1999; Bennett and Ijpelaar 2005; Buhr, et al. 1993; Chi, et al. 2002; National Research Council 2005), prompting a need to understand management aspects of these problems. It is useful to understand both optimal public (centralized) management in response to a disease outbreak (Kobayashi, et al. 2007a, b; Mahul and Durand 2000; Mahul and Gohin 1999; Rich and Winter-Nelson 2007), and the decentralized behavioral and disease responses to common policy initiatives (Hennessy 2005, 2007; Hennessy, Roosen and Jensen 2005).

Our focus in this paper is on decentralized outcomes. The majority of prior economic research in this area focuses on behavioral outcomes, holding disease risks fixed. Such risks have been considered a function of human choices but are not reflective of the underlying epidemiological dynamics (e.g., Hennessy 2005, 2007).[1] This work also focuses on long-run equilibrium outcomes and does not address the effect of public management actions on the approach path or the time required to disease eradication. In contrast, the majority of prior veterinary and epidemiological research focuses on how disease dynamics are affected by government intervention, such as herd depopulation, but without considering producer behavioral responses (e.g., Barlow et al. 1998). In this paper, we consider the joint-determination of disease and behavioral dynamics in a decentralized setting.

Livestock disease management is an inherently dynamic process that involves feedbacks

[1] Hennessy (2005) analyzes how disease externalities across farms in different spatial arrangements influence biosecurity decisions. Hennessy (2007) specifies a relationship between disease risk and biosecurity investment within a production region such that disease risks are endogenous (a function of biosecurity choice) and derives a number of general implications about the long-run equilibrium. In both cases, the disease risks are not based on an epidemiological model and therefore the relation between disease risks and biosecurity is fixed. Also, group-level impacts of government regulations, described below, are not modeled explicitly.

between disease ecology and private behavioral decisions. We consider the impact of these feedbacks while focusing on individual livestock managers' decisions of whether or not to invest in biosecurity as a preventive measure. A farmer's incentives to invest in biosecurity to protect his or her own herd are diminished by smaller infection risks and by lower effectiveness of biosecurity in reducing those risks. Indeed, there is only a chance that a farmer's livestock will be exposed to infection, and biosecurity may not fully protect one's own herd when exposure does occur. A farmer's investment incentives are also influenced by government responses to disease risks. In particular, the incentives for biosecurity are reduced if infection on a neighbor's farm triggers the imposition of costly regulations on all farms – even in the absence of infection in one's own herd. Animal health authorities commonly impose costly regulatory requirements on all herds within infected regions to eradicate infection. For instance, all farms in the bovine tuberculosis (bTB)-infected region in Michigan's Lower Peninsula — regardless of infection status — incur private costs as a result of dealing with government testing, movement restrictions and stringent testing rules for trade in live animals that go uncompensated by the government (Wolf 2006). These sorts of non-targeted requirements can alter the incentives to make private biosecurity investments, but prior economic and disease ecology work has not explored this issue.This article advances prior research in two principal ways. First, we show the importance of accounting for the jointly-determined nature of disease and decentralized economic outcomes. Disease risks are endogenous functions of human choices, while infection levels influence private incentives to invest in biosecurity. These feedbacks can influence how well various disease control policies perform. Second, we consider the economic and ecological impacts of government policies chosen to reduce disease risks. Specifically, we consider how the government's ability (or inability) to target policies towards biosecurity investments affects

producer incentives for these investments. The specific government policies, and the model in general, are based on the case of bTB in New Zealand cattle. Specifically, we analyze the regulatory choice of how frequently herds must be tested for bTB. Herd testing requirements are common for a variety of disease eradication programs in developed (and in some developing) nations (e.g., National Research Council 1994), though they are not as well targeted as policies that could be more effective in reducing disease risks.

We proceed with an analytical model of disease dynamics and then integrate economic behavior into this model. We then develop an application of our analytical model to examine the case of bovine Tuberculosis eradication in New Zealand. We present simulation results to illustrate the tradeoffs arising in a joint system, and we contrast these results with those arising from a non-joint system. We conclude by discussing some general implications of this research.

**Livestock Disease Dynamics**

Our model of cross-farm livestock disease dynamics is based on Barlow et al. (1998), who develop a model of bTB dynamics in New Zealand cattle. In their framework, which is essentially a metapopulation disease model (Levins 1969), individual farms (and not the individual animals on each farm) are the primary unit of interest. This herd-level focus is consistent with existing programs, as the herd is the most common unit for disease reporting and policy purposes.

Define $N$ to be the fixed number of homogeneous farms in a region. This region may be thought of as a disease surveillance zone established by a government authority to control the spread of infection. All farms within the zone may be subject to inspections and possible quarantine, depending on the disease in question. Of the $N$ farms, $S$ farms are susceptible, $I$ farms are infected but not yet identified as such (i.e., they are indistinguishable from susceptible

farms in the absence of an accurate test), and *M* farms being identified as infected and placed on movement control (for which transport of animals to or from the *M* farms is restricted).

The number of susceptible farms changes over time according to

(1) $\dot{S} = \varepsilon M - [\beta_0(1-b) + \beta_1 b]SI$ .

The number of susceptible farms grows when farms are taken off of movement controls, at the rate $\varepsilon$ (the first right-hand-side (RHS) term in (1)), and allowed to freely resume trade. *S* is reduced by transitions to the infected state, as represented by the second term. Disease transmission occurs based on movement of animals between farms. The rate of animal movement is captured by the disease transmission parameters $\beta_i$ ($i$=0,1) (see Barlow et al. 1998 for details). New infections occur at different rates based on whether or not farms invest in biosecurity: $\beta_0$ is the transmission rate when there is no biosecurity, while $\beta_1 < \beta_0$ is the transmission rate when there is biosecurity. In the present context, we take biosecurity to mean the farm quarantines and tests each animal brought onto the farm. The effect of this investment is to reduce the transmission rate, though it will not be reduced to zero as testing is imperfect (Barlow et al. 1998). The proportion of susceptible farms that biosecure at time *t* is denoted by *b*, so that the average transmission rate is $\beta_0(1\text{-}b) + \beta_1 b$. The expected number of new infections is given by $[\beta_0(1\text{-}b) + \beta_1 b]SI$. The specification in (1) is identical to Barlow et al. (1998), except that they model a constant average transmission rate. Our modified specification accounts for the fact that risk is endogenous (Shogren and Crocker 1999), as farmers can take actions to control their level of risk exposure.

The change in the number of infected farms over time is

(2) $\dot{I} = [\beta_0(1-b) + \beta_1 b]SI - qI$ .

The first term denotes newly infected farms, as in (1). With $\beta_1 < \beta_0$, this term indicates new

infections are declining in biosecurity. The last term represents the transition of infected farms onto movement control, which occurs at the rate $q$. Following Barlow et al. (1998), the rate $q$ is a function of slaughterhouse and government disease surveillance activities. In our numerical simulations below, we consider two alternate specifications for $q$.

The first specification for $q$ is $q = q_0+(\sigma/\tau)$, where $q_0$ is the fixed rate at which infected animals are detected at slaughter and traced back to the infected farm, and $(\sigma/\tau)$ is the rate at which government diagnostic testing (applied at the interval $\tau$, and with test sensitivity $\sigma$) identifies infected farms. The rate $q$ is an endogenous function of the testing interval, $\tau$, with more frequent testing resulting in a smaller interval and hence a greater detection rate. Barlow et al. (1998) examine how changes in this interval affect disease dynamics.

The second specification for $q$ is $q = q_0+(\sigma/\tau)M$. Here, the government testing interval is $\tau/M$, so that testing becomes more frequent when more herds have been identified as infected. This is consistent with Barlow et al.'s observation that testing is more frequent in areas of New Zealand exhibiting greater disease prevalence. It is also consistent with the fact that regulatory authorities, like individual farmers, are generally responsive to changing conditions. Since regulatory authorities are often focused on disease eradication, it makes sense that the government would respond to an increase in the number of detected herds by devoting more resources to on-farm testing. The result is to increase the rate at which infected farms are detected and placed on movement control when the number of farms on movement control increases ($\partial q/\partial M>0$). Finally, all transitions between disease states in (1)-(2) are balanced by changes in the number of farms on movement control, given by

(3) $$\dot{M} = qI - \varepsilon M .$$

Typical disease ecology models treat $b$ in (1)-(3) as an exogenous parameter and typically

compare steady state dynamics over a range of initial values for the disease state variables and other parameters. In contrast, we take $b$ to be an endogenous behavioral choice on the part of farmers. In the next section we develop the behavioral dynamics governing the selection of the economic strategy $b$, which is made in response to current disease risks. The economic choice, in turn, endogenously affects the infection dynamics in our joint model. In this way, we account for dynamic feedbacks between the economic and disease systems.

**A Dynamic Model of Farmer Behavioral Choices**

We assume the individual farms are identical except possibly for their current disease status (indexed by $j = S, I, M$) and their biosecurity strategy, which is chosen in response to their current and expected future disease risks. Specifically, farmers make biosecurity choices taking into account how these choices affect the possibility that their farm will transition to a new disease state at some time in the future. Denote the biosecurity strategy of an individual farmer by $z$. The strategy is a discrete choice: $z = 1$ implies biosecurity investment, $z = 0$ implies no biosecurity investment. The proportion of farms adopting biosecurity at any point in time is given by $b$, as defined above. As there is no reason to biosecure when farms are on movement controls, the biosecurity investment choice will only be made when farms *believe* they are non-infected. This means the farm could be in state $j=S$ or state $j=I$, as infection has not yet been detected in the infected state $j=I$. However, farmers in this state will generally believe they are in state $j=S$.

A farm in a given disease state receives an expected flow of income associated with its current disease state. Denote a farm's baseline profit in each period by $\pi(M) = \pi_0 + \pi_1 R[N - M]$, where $\pi_0$ represents profits when the farm is placed on movement controls and $\pi_1 R[N - M]$ represents variable profits attributable to animal movement. Here $R$ is the rate of animal

movement between farms and $R[N - M]$ represents the level of exchange activity per farm (Barlow et al. 1998).[2] When a farm is in the susceptible state ($j=S$) or the (undetected) infected state ($j = I$), then profits are gross of any biosecurity investment costs, $w(M, z) = \varpi R[N - M]z$, where $\varpi$ is the cost of biosecurity per unit trade. Regulatory costs, denoted $G$, may also be imposed when farms are in the susceptible or infected states ($j=S,I$). We describe the nature of these costs, and the incentives they generate, below after our description of the farmer decision model. Farms in the infected state ($j=I$) may also incur private losses from infection, $\delta\pi$, where $\delta$ is the rate at which infection reduces baseline profits. Finally, farms in the movement control state ($j=M$) will incur losses due to movement restrictions over and above infection losses, earning only $\pi_0(1-\delta) < \pi(M)$.

Following Shapiro and Stiglitz (see also Hennessy, 2007), denote $V_j^z$ to be the expected lifetime income of a farmer who is currently in state $j=S,I,M$ and has adopted the strategy of choosing action $z$. A farm's biosecurity strategy, as well as current infection levels, influence the likelihood the farm transitions from one state to another. Specifically, the individual's probability of transitioning from state $S$ to state $I$, given the strategy $z$, is $P_{SI}^z$. This value can be obtained from the epidemiological model as $P_{SI}^{z=1} = \beta_1 I$, $P_{SI}^{z=0} = \beta_0 I$, which changes over time as infection risks change. The individual's probability of transitioning from the infected state to the movement control state is $P_{IM} = q$. Finally, the individual's probability of transitioning from the empty state to the susceptible state is $P_{MS} = \varepsilon$.

Farmers are forward looking because their choices have intertemporal consequences. However, farmers do not have rational expectations with respect to transition probabilities.

[2] More specifically, Barlow et al. (1998) define $R$ as the rate of movement of animal *groups*, where an average group consists of seven animals.

Rather, farmers know the current disease risks and assume these continue on into the future, thereby taking the transition probabilities as fixed when decisions are made within a given period. As we describe below, the probabilities are updated at each decision node, so that farmers exhibit adaptive expectations.[3]

Assuming a discount rate of ρ, the fundamental asset equations for susceptible, infected, resistant and empty farms are[4]

(4) $$\rho V_S^z = \pi(M) - w(M,z) - G + P_{SI}^z[V_I^z - V_S^z]$$

(5) $$\rho V_I^z = \pi(M)(1-\delta) - w(M,z) - G + P_{IM}[V_M^z - V_I^z]$$

(6) $$\rho V_M^z = \pi_0(1-\delta) + P_{MS}[V_S^z - V_M^z]$$

Equation (4) is 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, $\pi$-*wz*-*G*, and the "expected capital loss that would arise were the state to change" (Hennessy 2007, p.702) from susceptible to infected, $P_{SI}^z[V_I^z - V_S^z]$. Equations (5) and (6) have analogous interpretations.

Equations (4)-(6) can be solved simultaneously for $V_S^z, V_I^z$, and $V_M^z$ as functions of the behavioral strategy, the states of the world, and model parameters. In particular, we focus on the susceptible state, for the biosecurity choice is made while the farm believes itself to be in the

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[3] The assumption of rational expectations seems too strong, as this would involve a differential game between *N* farms. Each farm would have to perfectly predict the actions of every other farm to accurately predict changes in risks. It seems unlikely that individual farmers, with limited information about their neighbors, would be able to do this (though see footnote 5 for more on the comparison between the two approaches). At the other extreme would be a completely myopic farmer who does not take any future impacts into account and instead maximizes static profits. We also view this as unrealistic, as farmers are accustomed to making long-run decisions about their asset holdings (i.e., their cattle stocks).

[4] The asset equations are derived following Shapiro and Stiglitz (1984), who assume an infinite time horizon. Focusing on the case of *j*=*S* as an example, we take $V_S^z$ and $V_I^z$ as given and examine expected lifetime utility over a small time interval [0, *t*]:

(4a) $$V_S^z = [\pi(M) - w(M,z) - G]t + (1-\rho t)[P_{SI}^z t V_I^z + (1 - P_{SI}^z t)V_S^z].$$

Note that $(1-\rho t) \approx e^{-\rho t}$. Equation (4) is obtained by solving (4a) for $V_S^z$ and evaluating it as $t \to 0$.

susceptible state. The solution for $V_S^z$ is

$$(7) \qquad V_S^z = \frac{[\pi(M) - w(M,z) - G][\Lambda + \Gamma_z] - \pi(M)\delta\Gamma_z + (1-\delta)\pi_0\beta_z Iq}{\rho[\Lambda + \Gamma_z + \beta_z Iq]} \quad z = 0, 1,$$

where $\Lambda = \rho[\rho + q + \varepsilon] + \varepsilon q > 0$ and $\Gamma_z = \beta_z I[\rho + \varepsilon] > 0$. The term $\Lambda/(\rho[\Lambda + \Gamma_z + \beta_z Iq])$ is the risk-adjusted discount factor associated with the susceptible state, assuming the farmer is initially susceptible. With no risk of becoming infected, the discount factor would simply be $1/\rho$. This factor is adjusted by $\Lambda/[\Lambda + \Gamma_z + \beta_z Iq]$ to account for the proportion of time the farmer will actually spend in the susceptible state. Similarly, the term $\Gamma_z/(\rho[\Lambda + \Gamma_z + \beta_z Iq])$ is a risk-adjusted discount factor associated with the infected state, assuming the farmer is initially susceptible. If the risk of becoming infected and then staying infected were one, the discount factor would be $1/[\rho(1+\rho)]$: the farmer would earn the annuity value $[\pi(M)(1-\delta) - w(M,z) - G]/\rho$ after becoming infected, but this value must be discounted by $1/[1+\rho]$ since infection occurs after the first period. Finally, and analogously, the term $\beta_z Iq/(\rho[\Lambda + \Gamma_z + \beta_z Iq])$ is a risk-adjusted discount factor associated with the movement control state, assuming the farmer is initially susceptible. In equation (7), the risk-adjusted discount factors weight the expected net benefits accruing in each state to determine the expected present value of net benefits when the farmer is initially susceptible, $V_S$.

The expected benefits of biosecurity depend on the current state of the world (via the transition probabilities). As the state of the world changes, the farmer updates the transition probabilities to reflect the current state of the world (hence exhibiting adaptive expectations) in order to re-evaluate the incentives to biosecure. In the long run, the system will equilibrate at a point of indifference, i.e,. $V_s^{z=1} = V_s^{z=0}$ (if such a point exists), so that no farmer has an incentive

to change his or her biosecurity strategy. We use replicator dynamics to model adjustment to such an equilibrium. The basic idea behind replicator dynamics is that the adoption of a particular strategy will increase in frequency within the overall farm population when the net benefits from that choice outweigh average net benefits associated with the current frequency of adoption (Rice, 2004). Specifically, frequency of adoption increases when expected lifetime income from adopting biosecurity exceeds the average expected lifetime income associated with the current distribution of biosecurity strategies, $\overline{V_S} = bV_S^{z=1} + (1-b)V_S^{z=0}$:

$$(8) \qquad \frac{\dot{b}}{b} = \alpha[V_S^{z=1} - \overline{V_S}] \Rightarrow \dot{b} = \alpha b(1-b)[V_S^{z=1} - V_S^{z=0}],$$

where $\alpha>0$ is a speed of adjustment parameter.[5] Equation of motion (8) indicates that frequency of biosecurity adoption is increasing (decreasing) when the expected profit from always investing in biosecurity exceeds (is less than) the expected profit from never investing in biosecurity. Hence the incentives for biosecurity adoption are given by $V_S^{z=1} - V_S^{z=0}$. As described above, farmers are indifferent about altering their biosecurity strategy in the steady state, i.e., $V_S^{z=1} = V_S^{z=0}$: biosecurity investment occurs until the biosecurity cost equals the opportunity costs of infection. Though expression (7) indicates that $V_S^{z=1} - V_S^{z=0}$ is not a function of *b*, it is a function of the state variables. A steady state for *b* (i.e., $V_S^{z=1} = V_S^{z=0}$) will arise if the values of the state variables *S*, *I*, and *M* attain an equilibrium in which there are no incentives to

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

further adjust *b*. Such outcomes arise in each of our simulations.

***The effect of the level of infection on biosecurity incentives***

Our model of adaptive expectations differs from some prior economic work in which it is assumed that *S*, *I*, and *M* are fixed (e.g., Hennessy, 2007). Indeed, *I*, *S*, and *M* are not fixed in the joint dynamic system. This means the probability of becoming infected is not fixed, and therefore assuming the risks are fixed when performing policy analysis may result in misleading policy recommendations. It is important to consider the interconnectedness of disease and behavioral dynamics. We focus in particular on changes in *I* (and not in *S* or *M*, as *S* is usually decreased and *M* increased as *I* is increased), as this directly affects the likelihood of infection and, in turn, the incentives to biosecure.

Consider the marginal effect of an increase in infection on asset values under investment strategy *z*

$$\frac{\partial V_S^z}{\partial I} = -\Lambda\beta_z \frac{[\pi(M) - w(M,z) - G - \pi_0(1-\delta)]q + \pi(M)\delta[\rho+\varepsilon]}{\rho[\Lambda + \Gamma_z + \beta_z Iq]^2}, \tag{9}$$

which is negative provided $\pi(M) - w(M,z) - G - \pi_0(1-\delta) > 0$ – which must be the case or else farms would all want to go on to movement controls. Analytically, expression (9) is sufficiently complex that generally it is not possible to sign $\partial(V_S^1 - V_S^0)/\partial I$. However, heuristically we can determine the sign (which is also verified numerically). The marginal value $\partial V_S^1/\partial I$ approaches zero as $\beta_1$ becomes very small (i.e., biosecurity becomes extremely effective at reducing risks so that increases in *I* do not really matter). This is the case in our numerical simulations where $\beta_1$ is 80 percent smaller than $\beta_0$. Hence, the sign of $\partial(V_S^1 - V_S^0)/\partial I$ is of the same sign as

$-\partial V_S^0 / \partial I > 0$: increases in infection increase the incentives for biosecurity investments. This result is intuitive, and along with the impact of *b* on $\dot{I}$, it suggests that biosecurity and infection are *joint dynamic substitutes*: greater biosecurity reduces the number of new infections, while a reduction in the number of new infections reduces the incentives to biosecure in subsequent periods. Such relations often result in interior equilibria (e.g., Rice 2004).[6]

In the numerical example below, we consider behavioral and disease dynamics jointly by incorporating the replicator dynamics model with the disease dynamics model. We then compare the joint model with a disease-only model to highlight the importance of incorporating economic feedbacks into disease models.

***The effect of government policies on biosecurity incentives***

The government implements disease control policies to promote disease eradication (or to otherwise reduce disease risks). These policies may reduce disease risks directly, but they will also generate costs (or potentially benefits, in the case of a subsidy) to the farm, *G*, as described above. Both the level of risk reduction and the regulatory costs *G* will influence private incentives to biosecure. The specific risk reductions and the form of *G* will depend on the specific policy choices. In this section we describe several alternative specifications and the incentive impacts.

In our model, farms control infection risks via their biosecurity choices. Ideally,

[6] Note that, in contrast to Hennessy's (2007) model, there are no cross-farm strategic interactions in our model. That is, the biosecurity strategy of one farm at time *t* does not influence the biosecurity strategy of others at time *t*. The reason is that disease risks are dynamic, and so one farm's actions at one point in time will not generally affect disease risks to others until the next instant. These intertemporal effects are captured, however, by the notion of joint dynamic substitutes between *b* and *I*. Since *I* depends on past choices, biosecurity investments by others in the previous period will generally reduce *I* (or at least the rate of increase of *I*), reducing the incentives for others to biosecure in the next period. The joint dynamic system is therefore characterized by intertemporal behavioral effects that are analogous to Hennessy's assumption of biosecurity investments being strategic substitutes across farms.

government policy would therefore target these choices directly so as to encourage investment. Suppose $G$ is introduced as an incentive policy to promote biosecurity, so that the government is not directly mandating a risk reduction. All incentive effects in this case arise via the specification of $G$, which would be decreasing in $z$. A linear relation would be of the form $G = -sz$, where $s$ is a subsidy rate and in which case expression (4) becomes[7]

$$\rho V_S^z = \pi - [w(M, z) - sz] + P_{SI}^z [V_I^z - V_S^z] \quad (10)$$

Clearly, a sufficiently large value of $s$ will offset private biosecurity costs enough to ensure the private benefits of adoption (i.e., the reduction in private risks) offset the post-subsidy adoption costs. The problem with basing a subsidy on private biosecurity effort is that biosecurity effort levels are generally difficult to observe (Gramig, Horan and Wolf, in press). While private testing could be performed and certified for all newly acquired animals, it would be very difficult to monitor whether a farm adequately quarantined its newly purchased animals. There is a moral hazard problem that limits the feasibility of biosecurity-based policies.

The alternative policy that we consider, and which is often used in infected areas, is regular testing of each farm's herd. This policy is clearly less desirable from a pure disease control standpoint because the spread of infection may be well-underway (particularly for a highly infectious pathogen) before infected herds are identified whenever the testing interval is sufficiently long. Such a policy is also less desirable because of the incentive effects it may have on the biosecurity investment decision. Testing requirements are a mandate that will directly affect risks, and which will also impose direct costs of $G$ (which is no longer viewed as an incentive policy but rather the costs of the regulation imposed on the farm). The impact of a decrease in $\tau$ (i.e., a more frequent testing interval) on the incentives to biosecure is given by

[7] Analogous incentive effects would emerge from $G = s(1-z)$, where $s$ is now a tax applied to those who do not adopt biosecurity.

$$(11) \qquad \frac{d(V_S^1 - V_S^0)}{d(-\tau)} = \frac{\partial(V_S^1 - V_S^0)}{\partial q}\frac{\partial q}{\partial(-\tau)} + \frac{\partial(V_S^1 - V_S^0)}{\partial G}\frac{\partial G}{\partial(-\tau)}$$

There are two effects. The first term on the right hand side of (11) represents the risk effect, while the second term represents the regulatory cost effect.

Consider the risk effect. This term does not reflect a reduction in disease risks. Rather, it reflects an increase in the risk that a farm will eventually be placed on movement controls, as $\partial q / \partial(-\tau) > 0$. To analyze the term $\partial(V_S^1 - V_S^0)/dq$, we begin by focusing on the impact of *q* on the asset value arising under a particular biosecurity choice *z*

$$(12)\ \frac{\partial V_S^z}{\partial q} = \frac{\beta_z I}{\rho[\Lambda + \Gamma_z + \beta_z I q]^2} \times \begin{bmatrix} -[\pi(M)(1-\delta) - w(M,z) - G - \pi_0(1-\delta)][\rho(\rho+\varepsilon) + \Gamma_z] \\ +\pi(M)\delta[\varepsilon(\rho+\varepsilon) + \Gamma_z] \end{bmatrix}$$

The sign of expression (12) is ambiguous. The first term in brackets is negative if farms prefer staying in the infected state to being detected and put on movement controls, and positive otherwise. Assuming the term is negative, increased detection is costly for farms in the short run as they are placed on movement controls. We refer to this as the movement control risk effect (or simply, the risk effect). The second term in brackets is positive and reflects the gain from eventually transitioning from the infected state back to the susceptible state, as increased detection speeds up this transition. We refer to this as the disease control effect.

The complexity of expression (12) prevents us from analytically signing $\partial(V_S^1 - V_S^0)/dq$. However, as above, we note that the marginal value $\partial V_S^1 / dq$ will be small if biosecurity is highly effective, in which case the sign of $\partial(V_S^1 - V_S^0)/dq$ is the same as the sign of $-\partial V_S^0 / dq$. If $-\partial V_S^0 / dq > 0$, then the risk effect dominates the disease control effect. A reduction in $\tau$ therefore provides incentives for farms to biosecure to reduce the risk of eventually transitioning to the movement control state. If $-\partial V_S^0 / dq < 0$, then the disease control effect dominates the

risk effect. A reduction in $\tau$ therefore reduces incentives for farms to biosecure because they will spend less time earning infection-related losses if they ever do become infected.

Now consider the regulatory cost effect in expression (11). Again, this will depend on the form of *G*. Previously we indicated two types of herd-level testing policies: (i) testing at a constant interval of $\tau$, and (ii) testing at a non-constant interval $\tau/M$, which depends on the current identified infection level. In each case, the policies are implemented regardless of a farm's biosecurity choice, and they are applied uniformly across farms.[8] The testing-based policy implies a regulatory cost function of the form $G(\tau,M)$, with $G_\tau < 0$ (since a larger $\tau$ implies less-frequent testing) and $G_M \geq 0$. For instance, if *v* is the per unit cost of testing the herd, then $G = x = v/\tau$ in the case of a constant testing interval, and $G = xM$ in the case of a non-constant testing interval.

Since *G* does not depend on *z*, the incentive effects are now more complex to untangle as *G* enters into the expressions for $V_j^1$ and $V_j^0$, for *j*=*S*,*I*. It is possible (e.g., using Wolfram Mathematica 6.0.3) to derive the following analytic expression

$$(13) \qquad \frac{\partial(V_S^1 - V_S^0)}{\partial G} = \frac{-Iq(\beta_0 - \beta_1)\Lambda}{\rho[\Lambda + \Gamma_0 + \beta_0 Iq][\Lambda + \Gamma_1 + \beta_1 Iq]} < 0.$$

A larger *G* reduces the private incentives to invest in biosecurity. The reason is that *G* is not at all targeted towards the biosecurity investment decision. Instead, within a particular time period, farm operators view *G* as a fixed, lump-sum tax. The result of this tax is to reduce the value of the farmer's assets, and so the farmer has fewer incentives to invest in biosecurity to protect those assets from a loss of value due to disease risks. This result is not unique to policies based

[8] In principle, the testing interval could vary across farms. Perhaps larger farms that are at greater risk would be tested more frequently (farms are homogenous in our model). This generalization would not change our qualitative results.

on diagnostic testing intervals. The same results would arise for any policy not based on the activity of concern (biosecurity in this case). For instance, Barlow et al. (1998) also consider partial trade restrictions for farms not placed under movement controls, as this would reduce disease transmission rates (and also profits). Our results here indicate that such approaches would also reduce the incentives for biosecurity investments.

Analogous incentive effects have been described in the context of environmental taxation. For instance, Cabe and Herriges (1992) analyzed an ambient pollution tax (a group tax based on the combined outcomes of farmers' pollution abatement decisions) applied to farmers in a watershed who did not believe their individual actions would affect the value of their tax payment. Cabe and Herriges found the tax could have a binary effect: a small tax would not affect farmers' actions, while a large enough tax would cause them to shut down and exit the industry.[9]

The net effect of the risk effect, the disease control effect, and the regulatory cost effect in expression (11) are ambiguous. If the risk effect dominates the other two effects, we might expect to see a decrease in $\tau$ result in greater biosecurity investments relative to a disease-only model that does not consider behavioral effects. If the disease control and regulatory cost effects dominate, we might expect to see a decrease in $\tau$ result in fewer investments. Of course, these results are for a given $I$. As $I$ is reduced in response to the regulations, incentives to invest will be further diminished relative to the disease-only model, as indicated above on account of $b$ and $I$

[9] In the present model, regulatory costs of the form $G(\tau,M) > 0$ could be considered an intertemporal ambient tax for a farmer exhibiting rational expectations. This is because the actions of all farmers would influence future values of $M$ and the farmer would consider these future impacts when making current investment decisions. If individual farmers did not believe their actions had a significant impact on future values of $M$ relative to the choices of all other farmers, then the incentive effects of this policy would be akin to those described by Cabe and Herriges (1992) and also for the adaptive management case that we consider. Regulatory costs stemming from government sanctions to control the spread of disease often operate as a group penalty affecting all farms not already on movement control. For instance, all farms in the non-bTB free area in Michigan—regardless of infection status—incur private costs as a result of dealing with government testing, movement restrictions and stringent testing rules for trade in live animals that go uncompensated by the government.

being joint substitutes. Therefore, in order for the joint model to predict more biosecurity investment than the disease-only model, the risk effects in equation (11) would have to be of sufficient magnitude as to dominate the joint substitute effect, the disease control effect, and the regulatory cost effect.

**Numerical Simulations and Model Comparisons**

We now apply our jointly-determined model to the case of bovine tuberculosis (bTB) transmission between herds in the Waikato region of New Zealand's North Island. This region is largely free from wildlife reservoirs of the disease and allows us to focus on cattle herd disease dynamics that are closely linked to private herd management (biosecurity investment behavior) and government actions (more frequent mandatory testing).

The policy objective for the Waikato region is to achieve "disease-free status", which is defined by the World Animal Health Organization (OIE) as having less than 0.2% of herds on movement control (Barlow et al. 1998). The question is what combinations of public policies can help to achieve this goal, and at what cost? Barlow et al. (1998) address this question by developing a model of disease transmission in which farmer behaviors are fixed (a disease-only model). In this section, we use the joint model that incorporates feedbacks between behavioral choices and disease outcomes (equations (1)-(3) and (8)) to simulate the disease path and the overall rate of biosecurity investment under different policy options. We also compare the results from the joint model with the predictions that arise from a disease-only model, which is a special case of the joint model such that $db/dt = 0$.[10] The specific policy option that we consider

[10] We do not consider a behavior-only model in which disease dynamics (risks) are fixed. This is because, by holding disease variables (i.e., the number of infected farms or farms on movement controls) fixed, changes in private biosecurity or government disease control activities have no effect on disease levels. Our simulations are focused on the government objective of attaining disease-free status and a behavior-only modeling approach is not capable of considering these issues.

is increased stringency of testing requirements, modeled via reductions in the testing interval, $\tau$, which increases $q$.

Variable descriptions, parameter values and initial values for state variables used in the numerical simulations are listed in Table 1. Economic parameters in equation (8) of our behavioral model are drawn from Bicknell, Wilen and Howitt's (1999) economic analysis of bTB control in New Zealand.[11] Initial values for the state variables and parameters in equations (1)-(3) of our disease model are drawn from the no wildlife reservoir area ("Area 1") data in Barlow et al. (1998).[12] Following Barlow et al., the "calibrated" parameter values are derived under the assumption that the disease system begins in a steady state at the initial values, given the initial value of $b$ and the initial value of the policy variable $\tau$. A similar calibration is performed for the economic variables and policy response function (in the case when the government dynamically adjusts testing in response to changes in detected infections) to ensure we begin at a steady state – implicitly, Barlow et al. also assume economic and policy variables are in a steady state so as to start their disease model in a steady state. This initial steady state represents our baseline scenario from which all other outcomes are evaluated.

The initial, steady state percentage of herds on movement control is 0.547%, which is almost three times higher than the policy objective of 0.2% required to meet international animal health standards. A change in the policy variable $\tau$ is therefore required to achieve this goal. We first derive results for cases in which government testing requirements do not adjust dynamically in response to changes in known disease prevalence (i.e., changes in $M$), so that $q$ is fixed for a given choice of $\tau$. Later, we examine how the baseline results change when government testing

[11] Their analysis is for a single farm and does not deal with cross-farm externalities. Note that we have scaled Bicknell, Wilen, and Howitt's (1999) parameter values to be consistent with the units used by Barlow et al. (1998).
[12] The exception is the parameter $q_0$, which is drawn from area 2 as slaughterhouse detection is thought to be higher now given how long the disease has been a problem (spurring additional surveillance) and given advances in surveillance methods.

requirements, and hence $q$, depends on $M$ so that a reduction in $\tau$ results in an upward shift in the function $q(M)$.

***Results with no endogenous government response***

We begin our simulation analysis with the case of no endogenous government response, in which $q$ is fixed for a given choice of $\tau$. Simulation results for both increases and decreases in $\tau$ relative to the baseline value, denoted $\tau_0 = 36$ months, are reported in Table 2. All solutions are derived using Mathematica 6.0.3. We report the percentage of herds on movement controls in the steady state, the number of years required to attain the steady state (as opposed to months, to simplify the exposition), along with the number of years it takes to attain disease-free status, as this may happen before the steady state is achieved. We also present the percentage of biosecurity adopters in the steady state for each scenario. Note that steady state biosecurity levels do not vary in the disease-only model, whereas they adjust in the joint model.

First consider the disease-only model with a constant level of biosecurity. Here, reductions in $\tau$ are required to reduce $M$. A disease-free steady state ($M < 0.2\%$ of farms) is achieved for each of the smaller values of $\tau$ examined in Table 1, though not within a reasonable time period for all values considered. Achieving disease-free status within 10 years requires that $\tau$ be cut in half to 18 months, and achieving disease-free status within 5 years requires cutting $\tau$ to 12 months. These results are consistent with those of Barlow et al.'s (1998) disease-only model.[13]

The same general trend is observed in the joint model, though the testing interval must be shortened considerably more—to 12 months—before a disease-free outcome is possible, both in

[13] Note: Barlow et al. did refer to steady state outcomes in their analysis, but they did not focus on a true steady state. Rather, they focused on cases where further changes in $M$ were sufficiently small, where "small" was chosen in an ad hoc manner.

the short run (5 years) and as a long-run steady state. Hence, attaining disease-free status is more difficult when behavior is considered. The reason, as described earlier in our analytic model, is that risk effect incentives provided by increased testing are insufficient to overcome the joint substitute effect, the disease control effect, and the regulatory cost effect embedded in farms' biosecurity incentives. In particular, the disease control and regulatory cost effects can be seen in Table 2 as private biosecurity is scaled back in response to more frequent government testing. Indeed, *b* falls to zero in the steady state for all joint model scenarios involving $\tau < 3$ years. The private costs of government testing requirements reduce the asset value of the farm, which has the effect of reducing the incentives to invest in biosecurity intended to protect a farm's asset value from being reduced by the disease. Moreover, more frequent testing reduces the losses that would be incurred anyway. Essentially, government testing substitutes for private biosecurity investments.

The disease-only model systematically estimates lower levels of infection than the joint model because it does not account for the substitution away from private biosecurity investments. As the testing interval becomes progressively shorter, results for the disease-only and joint models begin to converge. By convergence we mean that differences between the predicted values for both models become progressively smaller at all points in time, as do the times at which a steady state is attained in each model.

***Results with a government feedback response***

It makes sense to consider an endogenous government feedback response to changes in disease levels, particularly when eradication is the policy objective and given the length of time required

to attain this goal in the no-government-response case described above.[14] We therefore now investigate the use of a testing rule $\tau/M$, which results in the endogenous detection rate $q(M)$ described in our epidemiological model and associated private costs $G = xM$.

The impact of a government response in the disease-only case is that a much smaller value of $\tau$ is required to compensate for less-frequent testing as $M$ falls. For instance, in the disease-only model with the government feedback response, the testing interval must be reduced to 12 months before disease-free status is attainable, with eradication taking 15 years as compared to only 4 years without the government feedback response for the same testing interval.

The results are analogous but even more dramatic for the joint model, as it becomes even more difficult to eradicate the disease in the joint model via reductions in $\tau$. Specifically, the joint model with a government feedback response is only capable of achieving disease-free status for the 6 month testing interval, and this takes 37 years. But we also see an interesting opposite result in the joint model with a government feedback response: we find that the long-run steady state level of $M$ falls when $\tau$ is increased so that testing occurs less frequently for a given value of $M$. This result is driven by the fact that the reduction in government testing from the initial level $\tau_0$, even as $M$ becomes small, stimulates private biosecurity investments. Table 2 indicates that biosecurity adoption is also increasing in $\tau$ when there is no government feedback response, but the impact is much more pronounced in the presence of the feedback. Finally, note that, in

[14] There are many ways that a regulatory agency could respond. An economically optimal approach would minimize the discounted sum of damages and disease control costs, subject to disease dynamics and farmer responses to the policy choices. Intuitively, such an approach would likely involve greater (lower) control efforts when infections levels and hence damage costs are high (low). Though a truly optimal approach is unlikely to be implemented in practice, a politically feasible approach may still follow this basic rule of thumb. Indeed, as described earlier, Barlow et al. (1998) reports smaller testing intervals in New Zealand in regions where prevalence rates $M/N$ are larger. As $N$ is fixed in our model, the inverse relation between testing intervals and disease prevalence carries over to the absolute number of infected farms.

all cases, the magnitude of the substitution effect is diminished the larger is $\tau$. Accordingly, eliminating government testing altogether is not expected to lead to eradication.

**Discussion and Conclusion**

Two results of our analysis are worth highlighting. First is the need for disease-ecology models to account for human behavior when evaluating policies to control livestock disease outbreaks. Not doing so may lead to overestimates of policy effectiveness, as an individual farmer's incentives for biosecurity investment are contingent on policy choices – perhaps especially those policies that do not target biosecurity directly.

The second result that we highlight relates to this issue of targeting policies. The targeting of policies becomes an issue when hidden action problems prevent implementing policies based on biosecurity. Disease control policies not based on private biosecurity investments will operate like a lump sum cost that reduces asset values and thereby reduce the private incentives to invest in biosecurity to protect those assets from disease-related losses. Moreover, policies that eliminate the disease from the farm more quickly reduce any losses that would be incurred, also reducing farms' incentives to biosecure. When private biosecurity levels fall, more stringent government responses are required to compensate. Otherwise disease levels may remain at higher-than-desired levels. On the one hand, this result provides a rationale for the current approach of using stringent, area-wide policies like a quarantine to confront livestock disease problems. On the other hand, this result highlights the need to develop better-targeted policies, as they may ultimately be less-stringent and therefore less-costly.

The final implication of this research is the critical need for data necessary to parameterize models of this kind to analyze specific cases, as has been done in social planner oriented models, like those developed for Foot-and-Mouth disease in the US (Kobayashi, et al.

2007a,b) and France (Mahul and Durand 2000; Mahul and Gohin 1999). When data are available for a particular disease and geographical area, it may even be possible to integrate disease epidemiology with decentralized strategic interactions to better inform models of optimal allocation of public resources to respond to an epidemic. In developing our numerical simulations we were made aware of the general lack of empirical estimates of inter-herd disease transmission coefficients and longitudinal data on livestock disease prevalence trends or farmer behavior necessary to parameterize such a model. Without such data available, it will remain impossible to evaluate the performance of joint disease ecology-economic models which is necessary in order for such models to be of greatest use for policy making or economic decision making purposes.

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**Table 1.** Baseline Modeling Parameters

| *Description* | *Parameter* | *Value or Calibration Method* | *Source* |
|---|---|---|---|
| Slaughterhouse detection rate | $q_0$ | 0.01663 | Barlow et al. (1998) |
| Transition rate from movement control to susceptible (month$^{-1}$) | $\varepsilon$ | 1/9.6 | Barlow et al. (1998) |
| Diagnostic test (based on Caudal fold test) sensitivity | $\sigma$ | 0.8 | Barlow et al. (1998) |
| Government mandated testing interval (month$^{-1}$), initial value | $\tau(0)$ | 36 with no gov't feedback<br>$36 \times M(0)$ with gov't feedback | Barlow et al. (1998) |
| Number of farms | $N$ | 7310 | Barlow et al. (1998) |
| Number of farms initially on movement controls | $M(0)$ | 40 | Barlow et al. (1998) |
| Number of farms initially infected (non-detected) | $I(0)$ | $M(0) \times \varepsilon / q_0$ | Barlow et al. (1998)[A] |
| Number of farms initially susceptible | $S(0)$ | $N - M(0) - I(0)$ | Calibration |
| Weighted average of biosecurity-dependent transmission rates | $\beta$ | $q_0 / S(0)$ | Barlow et al. (1998)[A] |
| Transmission coefficient: with biosecurity | $\beta_1$ | $(1 - \sigma)\beta_0$ | Calculation |
| Transmission coefficient: no biosecurity | $\beta_0$ | $\beta / [(1 - b(0)) + (1 - \sigma)b(0)]$<br>(this solves $\beta = b(0)\beta_1 + [1-b(0)]\beta_0$ when $\beta_1=(1 - \sigma)\beta_0$) | Calculation |

**Table 1 Continued.** Baseline Modeling Parameters

| *Description* | *Parameter* | *Value or Calibration Method* | *Source* |
|---|---|---|---|
| Movement rate of animal groups per month | $R$ | $3.47 \times 10^{-5}$ | Barlow et al. (1998) |
| Initial proportion of farms investing in biosecurity | $b(0)$ | 0.5 | Assumption |
| Average number of animals/herd | $n$ | 164.08 | Barlow et al. (1998)[B] |
| Discount rate (monthly) | $\rho$ | 0.004 | Based on annual rate of 0.05 |
| Initial monthly profit per farm | $\pi(M(0))$ | \$2843.35 | BWH (1999)[C] |
| Profit when no movement occurs | $\pi_0$ | $\pi(M(0)) - \pi_1 \times R \times [N - M(0)]$ | |
| Variable profits per unit of animal movement | $\pi_1$ | Calibrated to solve $\dot{b}\|_{t=0} = 0$ | Ensures $b(0)$ is a steady state |
| Testing cost per head | $\omega$ | \$1.50 | BWH (1999) |
| Average number of animals/trade | $\zeta$ | 7 | Barlow et al. (1998) |
| Testing cost per trade | $\varpi$ | $\omega \times \zeta$ | Calculated |
| Herd-level testing cost | $v$ | $\omega \times n$ | Calculated |
| Proportional reduction in profits when infected | $\delta$ | 0.65 | BWH (1999) |
| Adjustment parameter for biosecurity investments | $\alpha$ | 0.00007 | Assumption |

[A] The formula is a calibration based on Barlow et al. (1998)
[B] Based on values in Barlow et al. (1998) for entire Waikato region
[C] BWH = Bicknell, Wilen, and Howitt (1999)

**Table 2.** Simulation Results for Baseline Parameterization of the Disease-only and Joint Models

| Testing Interval (months) | Disease-only model | | | | Joint model | | | |
|---|---|---|---|---|---|---|---|---|
| | % MC in SS | # Yrs to SS | b in SS | Time to Disease-free(yrs) | % MC in SS | # Yrs to SS | b in SS | Time to Disease-free(yrs) |
| 48 | 3.88 | 125 | 0.5 | N/A | 0.275 | 208 | 0.612 | N/A |
| 42 | 2.55 | 166 | 0.5 | N/A | 0.323 | 175 | 0.567 | N/A |
| Baseline=36 | 0.547 | 0 | 0.5 | N/A | 0.547 | 0 | 0.5 | N/A |
| 30 | 6.50E-05 | 100 | 0.5 | 23 | 10.1 | 66.7 | 0 | N/A |
| 24 | 2.39E-07 | 83.33 | 0.5 | 11 | 7.9 | 75 | 0 | N/A |
| 18 | 0.002 | 25 | 0.5 | 6.5 | 2.79 | 75 | 0 | N/A |
| 12 | 0 | 14.5 | 0.5 | 4 | 0.002 | 25 | 0 | 5 |
| 6 | 0 | 10.4 | 0.5 | 3 | 0 | 8.3 | 0 | 3 |
| Testing Interval (months) | Disease-only with Gov't Feedbacks | | | | Joint model with Gov't Feedbacks | | | |
| | % MC in SS | # Yrs to SS | b in SS | Time to Disease-free(yrs) | % MC in SS | # Yrs to SS | b in SS | Time to Disease-free(yrs) |
| 48 | 0.721 | 25 | 0.5 | N/A | 0.117 | 158.3 | 0.864 | 21 |
| 42 | 0.634 | 16.7 | 0.5 | N/A | 0.125 | 100.0 | 0.852 | 25 |
| Baseline=36 | 0.547 | 0 | 0.5 | N/A | 0.547 | 0 | 0.5 | N/A |
| 30 | 0.459 | 16.7 | 0.5 | N/A | 0.976 | 33.3 | 0.004 | N/A |
| 24 | 0.368 | 20.8 | 0.5 | N/A | 0.788 | 33.3 | 0.002 | N/A |
| 18 | 0.278 | 23 | 0.5 | N/A | 0.594 | 30.9 | 0.003 | N/A |
| 12 | 0.187 | 25 | 0.5 | 15 | 0.4 | 34.3 | 0 | N/A |
| 6 | 0.092 | 25 | 0.5 | 2.5 | 0.2 | 37.1 | 0 | 37 |

Notes: MC=movement control; SS=steady state; Disease-free when achieve ≤0.2% herds on MC; Cells that are enclosed in a double box within the table indicate scenarios that achieve a disease free steady state.