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# Jointly determined livestock disease dynamics and decentralised economic behaviour

Benjamin M. Gramig and Richard D. Horan<sup>†</sup>

A dynamic model of livestock disease and decentralised economic behaviour is constructed as a jointly determined system. By accounting for feedbacks between behavioural choices and disease outcomes, the model captures the endogenous nature of infection risks. Government mandated testing of livestock herds and how private biosecurity incentives are affected by the structure of disease eradication policies are considered. How well disease control policies are targeted affects their effectiveness and may result in farmers substituting government testing and disease surveillance for private biosecurity. Numerical simulation results demonstrate that failing to account for feedbacks between the disease ecology and economic systems may overestimate the effectiveness of government disease control policies.

**Key words:** bioeconomics, biosecurity, disease eradication, epidemiology, replicator dynamics.

## 1. Introduction

Livestock diseases impose significant costs on society (Bennett 2003; Bennett and Ijpelaar 2005; National Research Council 2005), prompting a need to understand management aspects of these problems. It is useful to understand both optimal public (centralised) management in response to a disease outbreak (Mahul and Gohin 1999; Mahul and Durand 2000; Kobayashi *et al.* 2007a,b; Rich and Winter-Nelson 2007), and the decentralised behavioural and disease responses to common policy initiatives (Hennessy 2005, 2007; Hennessy *et al.* 2005).

Our focus in this paper is on decentralised outcomes, taking into account dynamic feedbacks between private behavioural decisions and the underlying epidemiology of the disease system. We consider the impact of these feedbacks whilst focussing on individual livestock managers' decisions of whether or not to invest in biosecurity as a preventive measure. A farmer's incentives for biosecurity are diminished by smaller infection risks and by lower effectiveness of biosecurity in reducing those risks. Indeed, infection risks to any one herd are often small, and biosecurity investment may not eliminate these risks. A farmer's investment incentives are also influenced by government responses to disease risks. In particular, one's biosecurity incentives are

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<sup>†</sup> Benjamin M. Gramig (email: bgramig@purdue.edu) is Assistant Professor, Department of Agricultural Economics, Purdue University, West Lafayette, Indiana, USA. Richard D. Horan, is Professor, Department of Agricultural, Food and Resource Economics, Michigan State University, East Lansing, Michigan, USA.

reduced if infection on a neighbour'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 regions in Michigan's Lower Peninsula (Wolf 2006) and in New Zealand (Animal Health Board 2009a,b) – 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. These sorts of regulatory requirements can alter the incentives to make private biosecurity investments, but prior economic and disease ecology work has not explored this issue.

The majority of prior economic research in this area focusses on behavioural outcomes, when disease risks only depend on human choices and are not reflective of the underlying epidemiological dynamics. Hennessy (2005) analyses 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 an endogenous function of biosecurity choices, and he 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 are not modelled explicitly. This prior work focusses 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 focusses on how disease dynamics are affected by government intervention, such as herd depopulation, but without considering behavioural responses of producers (e.g. Barlow *et al.* 1998). In this paper, we consider the joint-determination of disease and behavioural dynamics in a decentralised setting.

This article advances prior research in two principal ways. First, we show the importance of accounting for the jointly determined nature of disease and decentralised economic outcomes. Disease risks are endogenous functions of human choices, whilst 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 analyse the regulatory choice of how frequently herds must be tested for bTB. Herd testing requirements are common for a variety of disease eradication programs (e.g. National Research Council 1994).

We proceed with an analytical model of disease dynamics and then integrate economic behaviour into this model. We then develop an application of our analytical model to examine the case of bTB 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.

## 2. Livestock disease dynamics

Our model of cross-farm livestock disease dynamics is based on Barlow *et al.*'s (1998) 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 within a disease surveillance zone established by a government authority to control the spread of infection. All farms within the zone are subject to inspections and possible quarantine. Of the  $N$  farms,  $S$  are susceptible,  $I$  are infected but not yet identified as such (i.e. they are indistinguishable from susceptible farms in the absence of an accurate test), and  $M$  are identified as infected and placed on movement control. Following Barlow *et al.* (1998), farms on movement control do not engage in transactions involving the movement of animals across farms, although some animals on the  $M$  farms may be sold for slaughter.

The number of susceptible farms changes over time according to:

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

The number of susceptible farms grows when farms are taken off movement controls, at the rate  $\varepsilon$  (the first right-hand-side (RHS) term in (Eqn 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 parameter when there is no biosecurity, whilst  $\beta_1 < \beta_0$  is the transmission parameter when there is biosecurity. Transmission parameters are the rate at which herds in state  $S$  transition to state  $I$ , per  $I$  herd per unit time, so that  $\beta_i SI$  is a number of herds (Barlow *et al.* 1998). Biosecurity in our model involves quarantining and testing each animal brought onto the farm. The effect of this investment is to reduce the transmission rate, although it will not be reduced to zero as testing is imperfect (Barlow *et al.* 1998). The proportion of susceptible farms that invest in biosecurity at time  $t$  is denoted by  $b$ , so that the average transmission

rate is  $\beta_0(1 - b) + \beta_1 b$  and the expected number of new infections is  $[\beta_0(1 - b) + \beta_1 b]SI$ . The specification in (Eqn 1) is identical to Barlow *et al.* (1998), except that they model a constant average transmission rate. Our modification 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:

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

The first term denotes newly infected farms, from (Eqn 1). With  $\beta_1 < \beta_0$ , these 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 mentioned elsewhere, 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  $\tau$  with test sensitivity  $\sigma$ ) identifies infected farms. The rate  $q$  is an endogenous function of the testing interval, 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 (i.e.  $\partial q/\partial M > 0$ ). This is consistent with the fact that regulatory authorities, like individual farmers, are generally responsive to changing conditions. Because regulatory authorities are often focussed 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. Indeed, Barlow *et al.* observe that testing is more frequent in areas of New Zealand exhibiting greater disease prevalence, and this is the continuing practice in New Zealand (Animal Health Board 2009a,b). Finally, all transitions between disease states in Equations (1) and (2) are balanced by changes in the number of farms on movement control, given by:

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

Typical disease ecology models treat  $b$  in (Eqns 1–3) as an exogenous parameter. In contrast, we take  $b$  to be an endogenous behavioural choice on the part of farmers, so that economic behaviour affects infection dynamics. In the next section, we develop the behavioural dynamics governing the selection of the economic strategy  $b$ , which is made in response to current disease risks. In this way, we account for dynamic feedbacks between the economic and disease systems.

### 3. A dynamic model of farmer behavioural 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 earlier. As there is no reason to invest in biosecurity when farms are on movement controls, the 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). 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. We describe the specific regulatory interventions, including the nature of the regulatory costs and the incentives generated, later after our description of the farmer decision model. Farms in the infected state 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 will incur losses because of movement restrictions over and above infection losses, earning only  $\pi_0(1 - \delta) < \pi(M)$ .

Following 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, government testing, and current infection levels all 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 movement control 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. 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 later, the probabilities are updated at each decision node, so that farmers exhibit adaptive expectations consistent with Berck and Perloff (1984).

Assuming a discount rate of  $\rho$ , the fundamental asset equations for susceptible, infected, and movement controlled farms are (see Appendix for a derivation):

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

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

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

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,  $\pi - w - G$ , and the ‘expected capital loss that would arise was the state to change’ (Hennessy 2007, p. 702, adapted from Shapiro and Stiglitz 1984) 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 behavioural strategy, the states of the world, and model parameters. We focus on the susceptible state because the biosecurity choice is made whilst the farm believes itself to be in the susceptible state. The solution for  $V_S^z$  is:

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

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 was 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

$1/[1 + \rho]$  because 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,  $V_S^z$ .

The expected net 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) to re-evaluate the incentives to invest in biosecurity. We use replicator dynamics to model adjustment of the biosecurity strategy. The basic idea behind replicator dynamics is that the frequency of biosecurity adoption within the overall farm population will increase when the net benefits from that choice,  $V_S^{z=1}$ , outweigh average net benefits associated with the current frequency of adoption,  $\bar{V}_S = bV_S^{z=1} + (1 - b)V_S^{z=0}$  (Rice 2004):

$$\frac{\dot{b}}{b} = \alpha[V_S^{z=1} - \bar{V}_S] \Rightarrow \dot{b} = \alpha b(1 - b)[V_S^{z=1} - V_S^{z=0}], \quad (8)$$

where  $\alpha > 0$  is a speed of adjustment parameter. The use of replicator dynamics to model biosecurity adoption in the population is consistent with the finding that profitability is an important driver of diffusion of agricultural innovations (Pannell *et al.* 2006). 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  $V_S^{z=1} - V_S^{z=0}$ . Farmers are indifferent about altering their biosecurity strategy, i.e.  $V_S^{z=1} = V_S^{z=0}$ , in an interior steady state; biosecurity investment occurs until the biosecurity cost equals the opportunity costs of infection. Although 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. An interior 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 further adjust  $b$ . Such outcomes arise in each of our simulations.

### 3.1. The effect of the level of infection on biosecurity incentives

The probability of becoming infected depends on the current values of  $b$ ,  $I$ ,  $S$ , and  $M$  in the joint dynamic system. Accordingly, misleading policy recommendations may result from assuming, as does some prior economic work that does not model the epidemiological dynamics, that  $S$ ,  $I$ , and  $M$  are fixed – and hence that infection risks are fixed functions of  $b$ . The importance of considering the interconnectedness of disease and behavioural dynamics can be seen by focussing on changes in  $I$ , as this directly

affects the likelihood of infection and, in turn, the incentives to invest in biosecurity.

Consider how a susceptible herd's asset value under investment strategy  $z$ ,  $V_S^z$ , responds to a marginal increase in  $I$ . This effect is given by:

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

Equation (9) 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. Expression (9) can be used to derive the marginal impact of  $I$  on the incentives to invest in biosecurity,  $\partial(V_S^1 - V_S^0)/\partial I$ . Although the resulting expression is too complex to sign, we can determine the sign heuristically (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 per cent 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, whilst a reduction in the number of new infections reduces the incentives to invest in biosecurity in subsequent periods. This joint dynamic system is characterised by intertemporal behavioural effects that are somewhat analogous to the assumption of biosecurity investments being strategic substitutes across farms in Hennessy (2007). Such relations often result in interior equilibria (e.g. Rice 2004).

### 3.2. The effect of government policies on biosecurity incentives

The government implements disease control policies to promote disease eradication. These policies may reduce disease risks directly, but they will also generate costs (or benefits, in the case of a subsidy) to the farm,  $G$ , as described earlier. Both the level of risk reduction and the regulatory costs  $G$  will influence private incentives to invest in biosecurity. 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.

Farms control infection risks via their biosecurity choices, but individual farmers will generally underinvest in biosecurity choices because they will not have incentives to consider how their choices affect risks to others. Ideally, government policy would 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:

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

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 *et al.* 2009). Whilst 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.

The alternative policy that we consider, and which is often used in infected areas, is regular testing of each herd. This policy is clearly less desirable from a pure disease control standpoint because whenever the testing interval is sufficiently long, the spread of infection may be well underway before infected herds are identified. 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 invest in biosecurity is:

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

There are two effects. The first term on the right-hand-side of (Eqn 11) represents the risk effect, whilst 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 analyse the term  $\partial(V_S^1 - V_S^0) / \partial q$ , we begin by focussing on the marginal impact of  $q$  on the asset value arising under a particular biosecurity choice  $z$ :

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

The sign of Equation (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. 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 Equation (12) prevents us from analytically signing  $\partial(V_S^1 - V_S^0)/\partial q$ . However, as mentioned earlier, we note that the marginal value  $\partial V_S^1/\partial q$  will be small if biosecurity is highly effective, in which case the sign of  $\partial(V_S^1 - V_S^0)/\partial q$  is the same as the sign of  $-\partial V_S^0/\partial q$ . If  $-\partial V_S^0/\partial q > 0$ , then the risk effect dominates the disease control effect. A reduction in  $\tau$  therefore provides incentives for farms to invest in biosecurity that reduces the risk of eventually transitioning to the movement control state. If  $-\partial V_S^0/\partial q < 0$ , then the disease control effect dominates the risk effect. A reduction in  $\tau$  therefore reduces incentives for farms to invest in biosecurity because they will spend less time earning infection-related losses if they ever do become infected.

Now consider the regulatory cost effect in Equation (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. The testing-based policy implies a regulatory cost function of the form  $G(\tau, M)$ , with  $G_\tau < 0$  (because 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.

Because  $G$  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 using Mathematica (2008) to derive the following analytic expression:

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

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 because of disease risks. Because investments in biosecurity are represented by both fixed costs (e.g. facilities investments to facilitate quarantine of introduced stock) and variable costs associated with management effort, the lump-sum tax  $G$  might be expected to reduce both fixed and variable expenditures on private biosecurity. This result

is not unique to policies based 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.

The net effect of the risk effect, the disease control effect, and the regulatory cost effect in Equation (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 behavioural 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  $I$  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 to dominate the joint substitute effect, the disease control effect and the regulatory cost effect.

#### 4. Numerical simulations and model comparisons

We now apply our model to the case of bTB transmission between herds in the Waikato region of New Zealand's North Island. Dairy and beef cattle are an important part of the rural economy of New Zealand, with dairy goods and services comprising one-sixth of total exports; 80 per cent of all New Zealand dairy cows are located on the North Island, principally in Waikato and Taranaki (Easton 2009). Because wildlife disease reservoirs are not a major concern in this region, the focus on cattle herd disease dynamics that are closely linked to private herd management (biosecurity investment) and government actions (more frequent mandatory testing) is appropriate.

The original (Barlow *et al.* 1998) and continuing (Animal Health Board 2009a) policy objective for the Waikato region is to achieve 'disease-free status', which is defined by the World Animal Health Organisation (OIE) as having <0.2 per cent of herds on movement control. Recent observed herd prevalence is lower than that projected in 2001 by the National Pest Management Strategy, but the number of infected herds must still be cut in half and maintained for 3 years to attain disease-free status (Animal Health Board 2009b). 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 using a disease-only model in which farmer behaviours are fixed. Here, we use the joint model that incorporates feedbacks between behavioural choices and disease outcomes (Eqns 1–3 and 8) to simulate disease and biosecurity dynamics under different reductions in the testing interval,  $\tau$ . We compare the

results with the predictions arising from a disease-only model, which is a special case of the joint model such that  $db/dt = 0$ .

Variable descriptions, parameter values and initial values for state variables used in the simulations are listed in the Appendix. Economic parameters in Equation (8) of our behavioural model are drawn from Bicknell *et al.*'s (1999) single-farm economic analysis of bTB control in New Zealand. Bicknell *et al.*'s (1999) parameter values were scaled to be consistent with the units used by Barlow *et al.* (1998). Initial values for the state variables and parameters in Eqns (1–3) of our disease model are drawn from the no wildlife reservoir area ('Area 1') data in Barlow *et al.* (1998). The exception is the parameter  $q_0$ , which is drawn from area 2 because slaughterhouse detection is thought to be higher now given how long the disease has been a problem and given advances in surveillance methods. Following Barlow *et al.* (1998), 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 baseline value of the policy variable  $\tau = \tau_0$ . A similar calibration is performed for the economic variables and policy response function (in the case when the testing interval depends on detected infections) to ensure we begin at a steady state. Implicitly, Barlow *et al.* (1998) 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 percentage of herds on movement control in our simulation is 0.547 per cent, which is higher than the recent estimated prevalence cited earlier and the policy objective of 0.2 per cent required to meet international animal health standards, but this level facilitates comparison with Barlow *et al.*'s (1998) disease-only model. A change in the policy variable  $\tau$  is therefore required to achieve the goal. We derive results for two cases: (i)  $q = q_0 + (\sigma/\tau)$  (no endogenous government response) and (ii)  $q(M) = q_0 + (\sigma/\tau)M$  (government feedback response). All solutions are derived using Mathematica (2008).

#### 4.1. Results with no endogenous government response

Suppose there is no endogenous government response, so that  $q$  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 1. We report the percentage of herds on movement controls in the steady state, the number of years required to attain the steady state, and the number of years it takes to attain disease-free status, as this status may be achieved prior to the steady state. We also present the percentage of biosecurity adopters in the steady state for each scenario. Note that steady state biosecurity levels adjust in the joint model but not in the disease-only model.

First consider the disease-only model. Here, reductions in  $\tau$  are the only way to reduce  $M$ . A disease-free steady state ( $M < 0.2$  per cent of farms) is

**Table 1** Simulation results for baseline parameterisation of the disease-only and joint models

Testing interval (months) = 1/ $\tau$	Disease-only model				Joint model			
	% MC in SS	No. years to SS	$b$ in SS	Time to disease-free (years)	% MC in SS	No. years to SS	$b$ in SS	Time to disease-free (years)
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

  

Normalised testing interval (months) = 1/ $\tau$ when $M = 1$	Disease-only with government feedbacks				Joint model with government feedbacks			
	% MC in SS	No. years to SS	$b$ in SS	Time to disease-free (years)	% MC in SS	No. years to SS	$b$ in SS	Time to disease-free (years)
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  $\leq 0.2\%$  herds on MC.

achieved for each of the smaller values of  $\tau$  examined in Table 1, although 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.

The same general trend is observed in the joint model, although the testing interval must be shortened considerably more – to 12 months – before a disease-free outcome is possible, both in the short run (5 years) and as a long-run steady state. Hence, attaining disease-free status is more difficult when behaviour 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 are evident in Table 1 as private biosecurity is scaled back in response to more frequent government testing. The private costs of government testing requirements reduce each farm's asset value, thereby reducing the incentives for biosecurity investments to protect this value from disease losses. 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 two models begin to converge.

#### 4.2. Results with a government feedback response

Now consider a government feedback response to changes in disease levels, using the testing rule  $\tau/M$ . As described earlier, this results in the endogenous detection rate  $q(M)$  and associated private costs  $G = \chi M$ .

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

In the case of the joint model, the results are analogous but even more dramatic as it becomes harder to eradicate the disease via reductions in  $\tau$ . Specifically, the disease-free status is only attainable for the 6 month normalised testing interval, and even then this takes 37 years. But we also see an interesting opposite result in the joint model with a government feedback response; the long-run steady state level of  $M$  falls when  $\tau$  is increased (i.e. when testing occurs less frequently for a given value of  $M$ ). This results because the reduction in government testing from the baseline stimulates private biosecurity

investments, even as  $M$  becomes small. Table 1 indicates that biosecurity adoption is also increasing in  $\tau$  for the joint model when there is no government feedback response, but the impact is much more pronounced in the presence of the feedback. Finally, in 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.

## 5. Discussion and conclusion

Two results of our analysis are worth highlighting. First is the need for disease ecology models to account for human behaviour 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 the 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 invest in biosecurity. 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 parameterise models of this kind to analyse 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 Gohin 1999; Mahul and Durand 2000). When data are available for a particular disease and geographical area, it may even be possible to integrate disease epidemiology with decentralised 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 behaviour necessary to parameterise 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.

## References

- Animal Health Board, TB Free New Zealand (2009a). *National Bovine Tuberculosis Pest Management Strategy, Amendment, Ministry of Agriculture, Wellington, New Zealand*. Available from URL: <http://tbfree.ahb.org.nz/LinkClick.aspx?fileticket=8Lnl%2fPb3D78%3d&tabid=313> [accessed 22 November 2010].
- Animal Health Board, TB Free New Zealand (2009b). *National Pest Management Strategy, Progress*. Available from URL: <http://tbfree.ahb.org.nz/Default.aspx?tabid=167> [accessed 22 November 2010].
- Barlow, N.D., Kean, J.M., Caldwell, N.P. and Ryan, T.J. (1998). Modelling the regional dynamics and management of bovine tuberculosis in New Zealand cattle herds, *Preventive Veterinary Medicine* 36, 25–38.
- Bennett, R. (2003). The 'Direct Costs' of livestock disease: the development of a system of models for the analysis of 30 endemic livestock diseases in Great Britain, *Journal of Agricultural Economics* 54, 55–71.
- Bennett, R. and Ijpelaar, J. (2005). Updated estimates of the costs associated with thirty four endemic livestock diseases in Great Britain: a note, *Journal of Agricultural Economics* 56, 135–144.
- Berck, P. and Perloff, J.M. (1984). An open-access fishery with rational expectations, *Econometrica* 52, 489–506.
- Bicknell, K.B., Wilen, J.E. and Howitt, R.E. (1999). Public policy and private incentives for livestock disease control, *The Australian Journal of Agricultural and Resource Economics* 43, 501–521.
- Easton, B. (2009). *Economy-Agricultural production, Te Ara: The Encyclopedia of New Zealand*. Available from URL: <http://www.TeAra.govt.nz/en/economy/2> [accessed 18 November 2010].
- Gramig, B.M., Horan, R.D. and Wolf, C.A. (2009). Livestock disease indemnity design when moral hazard is followed by adverse selection, *American Journal of Agricultural Economics* 91, 627–641.
- Hennessy, D.A. (2005). Biosecurity and infectious animal disease, Working paper, Center for Agricultural and Rural Development, Iowa State University.
- Hennessy, D.A. (2007). Behavioural incentives, equilibrium endemic disease, and health management policy for farmed animals, *American Journal of Agricultural Economics* 89, 698–711.
- Hennessy, D.A., Roosen, J. and Jensen, H.H. (2005). Infectious disease, productivity, and scale in open and closed animal production systems, *American Journal of Agricultural Economics* 87, 900–917.
- Kobayashi, M., Carpenter, T.E., Dickey, B.F. and Howitt, R.E. (2007a). A dynamic, optimal disease control model for foot-and-mouth disease: II. Model results and policy implications, *Preventive Veterinary Medicine* 79, 274–286.
- Kobayashi, M., Carpenter, T.E., Dickey, B.F. and Howitt, R.E. (2007b). A dynamic, optimal disease control model for foot-and-mouth disease: I. Model description, *Preventive Veterinary Medicine* 79, 257–273.
- Levins, R. (1969). Some demographic and genetic consequences of environmental heterogeneity for biological control, *Bulletin of the Entomological Society of America* 15, 237–240.
- Mahul, O. and Durand, B. (2000). Simulated economic consequences of foot-and-mouth disease epidemics and their public control in France, *Preventive Veterinary Medicine* 47, 23–38.
- Mahul, O. and Gohin, A. (1999). Irreversible decision making in contagious animal disease control under uncertainty: an illustration using FMD in Brittany, *European Review of Agricultural Economics* 26, 39–58.
- Mathematica (2008). *Software Version 6.0.3*. Wolfram Research, Inc., Champaign, IL.
- National Research Council (1994). *Livestock Disease Eradication: Evaluation of the Cooperative State-Federal Bovine Tuberculosis Eradication Program*. National Academies Press, Washington, DC.

- National Research Council (2005). *Animal Health at the Crossroads: Preventing, Detecting, and Diagnosing Animal Diseases*. National Academies Press, Washington, DC.
- Pannell, D.J., Marshall, G.R., Barr, N., Curtis, A., Vanclay, F. and Wilkinson, R. (2006). Understanding and promoting adoption of conservation practices by rural landholders, *Australian Journal of Experimental Agriculture* 46, 1407–1424.
- Rice, S.H. (2004). *Evolutionary Theory: Mathematical and Conceptual Foundations*. Sinauer Associates, Inc., Sunderland, MA.
- Rich, K.M. and Winter-Nelson, A. (2007). An integrated epidemiological-economic analysis of foot and mouth disease: applications to the southern cone of South America, *American Journal of Agricultural Economics* 89, 682–697.
- Shapiro, C. and Stiglitz, J. (1984). Equilibrium unemployment as a worker discipline device, *American Economic Review* 74, 433–444.
- Shogren, J.F. and Crocker, T.D. (1999). Risk and its consequences, *Journal of Environmental Economics and Management* 37, 44–51.
- Wolf, C.A. (2006). Livestock disease eradication programmes and farm incentives: the case of bovine tuberculosis, in Koontz, S.R., Hoag, D.L., Thilmany, D.D., Green, J.W. and Granis, J.L. (eds.), *The Economics of Livestock Disease Insurance*. CABI, Oxfordshire, UK, pp. 181–192.

## Appendix

### Derivation of asset value equations

The asset Equations (4)–(6) are derived following Shapiro and Stiglitz (1984), who assume an infinite time horizon. Focussing 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  $[0, t]$ :

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

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

**Table A1** Baseline modelling parameters

Description	Parameter	Value or calibration method	Source
Slaughterhouse detection rate	$q_0$	0.01663	Barlow <i>et al.</i> (1998)
Transition rate from movement control to susceptible (month <sup>-1</sup> )	$\varepsilon$	1/9.6	Barlow <i>et al.</i> (1998)
Diagnostic test (based on Caudal fold test) sensitivity	$\sigma$	0.8	Barlow <i>et al.</i> (1998)
Government mandated testing interval (month <sup>-1</sup> ), initial value	$\tau(0)$	36 with no gov't feedback 36 $\times$ $M(0)$ with gov't feedback	Barlow <i>et al.</i> (1998)
Number of farms	$N$	7310	Barlow <i>et al.</i> (1998)
Number of farms initially on movement controls	$M(0)$	40	Barlow <i>et al.</i> (1998)
Number of farms initially infected (non-detected)	$I(0)$	$M(0) \times \varepsilon/q_0$	Barlow <i>et al.</i> (1998)†
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 <i>et al.</i> (1998)†
Transmission coefficient: with biosecurity	$\beta_1$	$(1 - \sigma)\beta_0$	Calculation
Transmission coefficient: no biosecurity	$\beta_0$	$\beta/[(1 - b(0)) + (1 - \sigma)b(0)]$ (this solves $\beta = b(0)\beta_1 + [1 - b(0)]\beta_0$ when $\beta_1 = (1 - \sigma)\beta_0$ )	Calculation
Movement rate of animal groups per month	$R$	$3.47 \times 10^{-5}$	Barlow <i>et al.</i> (1998)
Initial proportion of farms investing in biosecurity	$b(0)$	0.5	Assumption
Average number of animals/herd	$n$	164.08	Barlow <i>et al.</i> (1998)‡
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)§
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 $b _{t=0} = 0$	Ensures $b(0)$ is a steady state
Testing cost per head	$\omega$	\$1.50	BWH (1999)
Average number of animals/trade	$\zeta$	7	Barlow <i>et al.</i> (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

Notes: †The formula is a calibration based on Barlow *et al.* (1998). ‡Based on values in Barlow *et al.* (1998) for entire Waikato region. §BWH, Bicknell *et al.* (1999).