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Public policy and private incentives for livestock disease control†

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This article presents a dynamic bioeconomic model of livestock disease control that is unique in its integration of disease dynamics, inter-species interaction, control-induced migration, and individual optimising behaviour. Examination of the first-order conditions highlights why profit-maximising producers cannot be expected to eradicate disease. Results from an empirical application of the model confirm that the current mix of policies to control bovine tuberculosis in New Zealand is achieving lower levels of prevalence than would prevail in the absence of a national strategy. These policies do, however, appear to remove some of the individual incentive to control disease.

1. Introduction

Externalities associated with livestock disease control have prompted countries throughout the world to invest in centralised control schemes designed to lower disease prevalence. As disease levels drop and fiscal deficits climb, however, many governments are beginning to reconsider the design and delivery of their animal health services (Umali *et al.* 1994). The Animal Health Board in New Zealand, for example, is concerned that the regulatory policies implemented to encourage participation in the national bovine tuberculosis control scheme have distorted market signals and removed some of the private incentive to control disease. Consistent with the ongoing shift to a more market-oriented economy, the Animal Health Board is attempting to

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identify and implement policies which encourage producer participation, yet convey more accurately the cost of disease (AHB 1995).

The success of the Animal Health Board's efforts to motivate cost-effective disease control depends critically on whether their new policies generate consistent rather than opposing incentives for individual livestock producers to control disease. Much of the previous literature on animal health economics, however, consists of *ex post* evaluations of national control schemes which offer limited insight into the potential behavioural responses of producers who raise livestock primarily for economic profit (Dietrich *et al.* 1987; Ebel *et al.* 1992; Liu 1979). This article utilises recent advances in the dynamic bioeconomic literature to develop a behavioural model of livestock disease control. The model is estimated and solved for a region in New Zealand where efforts to control bovine tuberculosis have been complicated by the existence of an effective wildlife reservoir for disease. The model is unique in its integration of disease dynamics, inter-species interaction, control-induced migration, and individual optimising behaviour into one, unifying optimal control model.

2. Empirical problem

This article was motivated by the need to develop a more thorough understanding of the complex biological and economic dynamics which influence the spread and maintenance of bovine tuberculosis (Tb) in New Zealand. Although centralised control efforts have dramatically reduced the apparent prevalence of Tb in New Zealand over the past 50 years, recent control efforts have been compromised by the existence of a persistent wildlife reservoir of disease. Epidemiological research suggests that the Australian brushtailed possum is serving as a primary non-cattle source of infection. Effective disease control therefore requires the combined efforts of ecologists as well as veterinarians, epidemiologists and economists. The model developed below seeks to integrate the biological dynamics of pest and disease control into an economic framework which captures the behavioural responses of individual producers making economic decisions in a constrained environment.

Control measures for bovine Tb in New Zealand currently include compulsory diagnostic testing and slaughter of test-positive cattle, the controlled movement of cattle from infected herds, slaughterhouse surveillance, and possum control operations. Testing requirements vary throughout New Zealand, depending on the risk of infection from non-cattle sources and the composition of the herd. In order to encourage participation in the compulsory test and slaughter program, policies such as subsidised testing and compensation for reactor cattle have been introduced. Subsidies have

also been applied in the form of possum control operations, which are funded from a combination of government, industry and individual landowner sources.

The Animal Health Board is concerned that the current mix of policies is distorting market signals and sheltering producers from the true cost of disease. As a consequence, the Board is considering a number of policy changes that will introduce a more market-oriented approach to disease control. The bioeconomic model presented in the next section was developed to gain insight into the economic trade-offs associated with various policy options, and to determine whether proposed changes are consistent with individual producer behaviour.

The dynamics of the cattle herd and the possum populations that constrain the optimal control model are depicted in figure 1. Susceptible cattle become infected with tuberculosis following effective contact with infected herdmates or possums. Disease incidence therefore depends primarily upon the number of cattle in each state (infected or susceptible), the size of the wildlife reservoir, a set of epidemiological rate parameters, and the level of testing and marketing activity in each period. Because intra-uterine transmission of bovine tuberculosis is extremely uncommon, both classes of cattle are assumed to contribute to the biological growth of the susceptible herd. The model also incorporates migratory pressure from a neighbouring possum population after control operations have reduced possum numbers near the cattle herd. In the absence of control, the biological growth rate and the migratory flux between controlled and uncontrolled habitats determine the change in the possum populations. Harvesting can further reduce possums in the vicinity of a cattle herd.

3. Model structure

In its most general form, the optimal control model contains four state and three control variables. The two state variables for cattle comprise susceptible (S_t) and infectious (I_t) stock, and the state variables for the possum populations include an 'in contact' population that may transmit disease to cattle ($P1_t$) and a neighbouring population in an uncontrolled habitat ($P2_t$). According to equation 1, the cattle producer's objective is to select a marketing (M_t) and testing (T_t) strategy for cattle, and a harvest scheme for the wildlife population (H_t) that maximises discounted net revenue from the sale of cattle.

$$\max_{T_t, M_t, H_t} \pi = \int_{t=0}^{T} e^{-\delta t}\{(p-l)(M_t(S_t+\mu I_t) + T_t(1-M_t)(\gamma_1\tau_1 S_t + \gamma_2\tau_2 I_t)) - v_1(S_t+I_t) - T_t v_2(1-M_t)(S_t+I_t) - PC(H_t, P1_t)\}dt \quad (1)$$

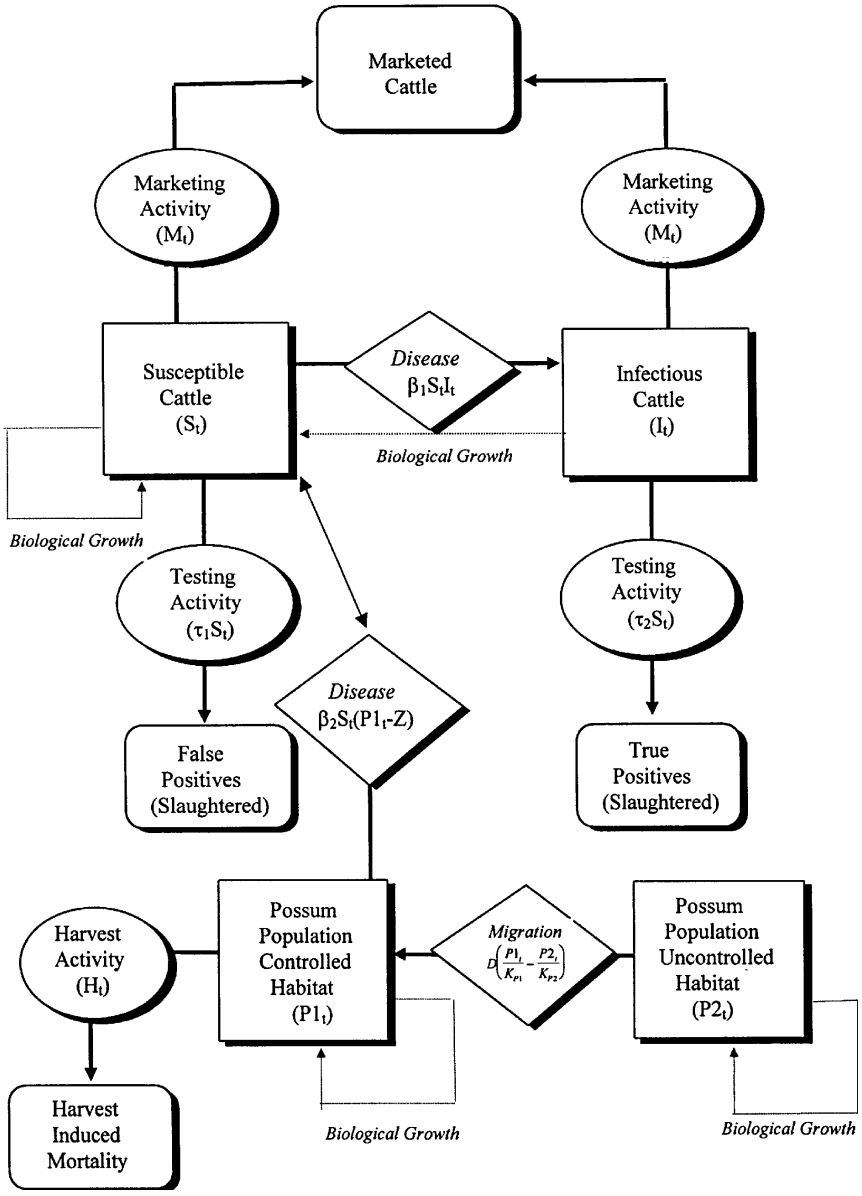

Figure 1 Schematic diagram of bioeconomic system

The model was specified with testing as a control variable to accommodate general policy analysis. Indeed, one of the major objectives of the research is to identify more market-oriented policies that could ultimately lessen the Animal Health Board's reliance on the current 'command and control'

approach. Tuberculosis testing is currently highly regulated in New Zealand, however, with annual testing required where wildlife vectors such as possums have been confirmed. To reflect this constraint on producer behaviour, T_t was treated as a parameter (equal to 1) for the numerical work presented in this article.

Cattle producers are assumed to be operating in a perfectly competitive environment, facing price p for non-infected cattle. The parameter l represents a levy on all cattle slaughtered, which provides funding for the centralised disease control scheme. The control variable for marketing is expressed in percentage terms, so the gross revenue from marketing healthy cattle is $(p - l)M_tS_t$.

Because producers cannot distinguish between healthy and infectious animals by casual observation, the marketing activity reduces both categories of cattle indiscriminately. An implicit assumption of the model is that all infectious stock sent to market will be detected through routine slaughter-house surveillance. Producers may receive a small percentage of market value (μ) for infected animals if all or part of the carcass can be salvaged for manufacturing purposes. One of the direct costs associated with disease, therefore, is the reduction in carcass value of infected cattle.

Testing is hypothesised to occur after the marketing activity has taken place, leaving a population of $(1 - M_t)(S_t + I_t)$ individuals to test. Cattle that test positive are removed from the herd and slaughtered. Compensation is paid for all test-positive animals as a percentage of market price, which is represented in the model by the parameters γ_i. The average annual cost per head of producing cattle (v_1) is assumed to be independent of disease status. The average cost of testing cattle is represented by the parameter v_2.

The cost of harvesting possums which serve as vectors for disease is represented in equation 1 by $PC(H_t, P1_t)$. Harvest costs can be expected to increase with harvest activity (H_t) and decrease with possum density ($P1_t$). The cost function for the empirical application was estimated with data from twenty ground control operations which had been performed in a variety of habitat sites throughout New Zealand.

Equations 2 and 3 describe the net rate of change in the population densities of healthy and infectious cattle through time:

$$\dot{S} = a(S_t + I_t)\left(1 - \frac{(S_t + I_t)}{K_c}\right) - \beta_1 S_t I_t - \beta_2 S_t(P1_t - Z) - M_t S_t - T_t\tau_1(1 - M_t)S_t - bS_t \tag{2}$$

$$\dot{I} = \beta_1 S_t I_t + \beta_2 S_t(P1_t - Z) - M_t I_t - T_t\tau_2(1 - M_t)I_t - bI_t. \tag{3}$$

Because supplementary feeding is uncommon in New Zealand, the beef cattle production process is heavily reliant upon the capacity of the pasture to

support stock. A modified logistic equation was therefore used to model the biological growth of the cattle herd, where a reflects the fecundity of the herd and K_c represents the carrying capacity of the pasture for cattle.

Infected animals are the principal source of tuberculosis for susceptible cattle, that may become infected by either ingesting or inhaling live bacteria (Blood and Radostits 1989). Following the methodology suggested by Anderson and May (1979a; 1979b), the rate at which cattle become infected is proportional to the number of encounters between susceptible cattle and either infectious cattle or the wildlife reservoir. The constants of proportionality (β_i) are therefore rate parameters that reflect the likelihood of close contact between an infectious and a susceptible individual, and the likelihood that transmission will occur as a result of the contact (Nokes and Andersen 1988).

Consistent with the strong empirical relationship between possum abundance and disease prevalence in cattle (Livingstone 1991), the transmission of tuberculosis between species is assumed to be proportional to the density of the controlled possum population. Prior epidemiological modelling of Tb in possum populations suggests that eradication of the disease in possums may be possible if possum density is maintained below a particular threshold level (Barlow 1991a; 1991b). The threshold concept is characteristic of this class of models, which require that the generation of secondary cases must be sufficient to maintain infection within the population of interest. The disease threshold for the possum population is incorporated into the current model by making transmission proportional to $(P1_t - Z)$, where Z represents the critical density of possums below which the disease will not persist in the population. It is therefore possible, although it may not be economically optimal, to remove possums as a source of disease by holding them at their disease threshold.

While it is assumed that producers cannot distinguish between healthy and infectious animals by casual observation, disease status can be estimated with the aid of a diagnostic test. The parameter τ_1 defines the proportion of healthy cattle that are removed from the herd because of test-positive status. The number of false positive reactors varies inversely with the level of the test specificity (specificity $= 1 - \tau_1$), which reflects the ability of the test to correctly detect non-diseased animals. Similarly, τ_2 determines the proportion of infected animals that are removed from the herd during testing. This proportion depends directly on the test sensitivity, which reflects the ability of the test to detect infected animals. Finally, a low rate of natural mortality (b) slows the growth of both cattle populations.

Equations 4 and 5 represent the equations of motion for possums in the controlled and uncontrolled habitats, respectively. Following Clout and Barlow (1982), the biological growth of the possum populations is described by a simple logistic equation.

$$\dot{P}1 = r_{P1}P1_t\left(1 - \frac{P1_t}{K_{P1}}\right) - D\left(\frac{P1_t}{K_{P1}} - \frac{P2_t}{K_{P2}}\right) - H_t \tag{4}$$

$$\dot{P}2 = r_{P2}P2_t\left(1 - \frac{P1_t}{K_{P1}}\right) + D\left(\frac{P1_t}{K_{P1}} - \frac{P2_t}{K_{P2}}\right) \tag{5}$$

In spatially complex ecological systems, harvesting efforts may be compromised by immigration from neighbouring habitats (Hickling 1993). This migratory pressure is captured by a flux term which dictates the rate of movement between controlled and uncontrolled habitats. The flux term is based upon the ecological modelling work of Hestbeck (1988) and Stenseth (1988), who have observed that animals will emigrate from relatively densely populated habitats if the opportunity arises. Migration is therefore hypothesised to be density-dependent, and will increase the speed at which managed populations recover from control activities. The parameter D represents the maximum number of possums that will recolonise a neighbouring area that has been completely cleared of possums. A similar specification has been applied to the optimal control of beavers in North America (Huffaker *et al.* 1992) and the management of possum populations in New Zealand (Barlow 1993).

The producer's economic problem is therefore to maximise equation 1, subject to equations 2–5, which represent the biological equations of motion for this bioeconomic model. Equations 6 and 7 place additional restrictions on the state and control space, respectively, and equation 8 specifies the initial conditions for the state variables.

$$S_t, I_t, P1_t, P2_t, \geq 0 \tag{6}$$

$$0 \leq M_t \leq 1; 0 \leq T_t \leq 1; 0 \leq H_t \leq 0.85P1_t \tag{7}$$

$$S(0) = S_0, I(0) = I_0, P1(0) = P1_0, P2(0) = P2_0 \tag{8}$$

4. General implications of optimising behaviour

Eradication is the objective of many food animal disease control programs throughout the world. Necessary conditions derived from the model presented above are used in this section to describe in very general terms why it is not optimal for individual profit-maximising producers to eradicate disease from their herds. This section also highlights some of the policy instruments disease control authorities may use to encourage compliant behaviour as prevalence declines. A more detailed policy analysis is presented in the sections that follow.

The current valued Hamiltonian for this problem is defined as follows:

$$
\begin{aligned}
H_{CV} = {} & \{(p-l)(M_t(S_t+\mu I_t)+T_t(1-M_t)(\gamma_1\tau_1 S_t+\gamma_2\tau_2 I_t)) \\
& -v_1(S_t+I_t)-T_t v_2(1-M_t)(S_t+I_t)-PC(P1_t,H_t)\} \\
& +m_1\left\{a(S_t+I_t)\left(1-\frac{(S_t+I_t)}{K_c}\right)-\beta_1 S_t I_t-\beta_2 S_t(P1_t-Z)\right. \\
& \left.-M_t S_t-T_t\tau_1(1-M_t)S_t-bS_t\right\} \\
& +m_2\{\beta_1 S_t I_t-\beta_2 S_t(P1_t-Z)+M_t I_t-T_t\tau_2(1-M_t)I_t-bI_t\} \\
& +m_3\left\{r_{P1}P1_t\left(1-\frac{P1_t}{K_{P1}}\right)-D\left(\frac{P1_t}{K_{P1}}-\frac{P2_t}{K_{P2}}\right)-H_t\right\} \\
& +m_4\left\{r_{P2}P2_t\left(1-\frac{P1_t}{K_{P1}}\right)+D\left(\frac{P1_t}{K_{P1}}-\frac{P2_t}{K_{P2}}\right)\right\}
\end{aligned}
\tag{9}
$$

The variables m_i $(i = 1, 2, 3, 4)$ are costate variables and can be interpreted as the implicit value of the stock with which they are associated (Chiang 1992).

When diagnostic testing is a control variable, the first-order conditions for this model consist of three algebraic expressions and eight differential equations that must be solved simultaneously for the optimal trajectories of the state, control and costate variables. Consequently, the complexity of this empirical problem precludes a complete analytical solution. The first-order conditions for testing and harvesting do, however, offer some insight into what motivates profit-maximising producers to control disease.

4.1 The private decision to use diagnostic testing

The switching function for testing is derived by differentiating the current valued Hamiltonian with respect to the testing variable:

$$
\begin{aligned}
\frac{\partial H_{CV}}{\partial T_t} = \sigma_T = {} & (p-l)(1-M_t)(\gamma_1\tau_1 S_t+\gamma_2\tau_2 I_t)-v_2(1-M_t)(S_t+I_t) \\
& -m_1(1-M_t)\tau_1 S_t-m_2(1-M_t)\tau_2 I_t.
\end{aligned}
\tag{10}
$$

The resulting expression is used to synthesise the following optimal testing sequence:

$$
T_t = \begin{cases} 0, \sigma_t < 0 \\ T^*, \sigma_T = 0, \\ 1, \sigma_T > 0 \end{cases}
\tag{11}
$$

where T^* represents the optimal interior solution. Equation 11 requires that the testing activity be set at one of its extreme values when the switching

function is nonzero. For example, testing will cease when the implicit value of an infected individual is greater than the net revenue from testing:

$$\begin{aligned}\sigma_T = (p-l)(1-M_t)(\gamma_1\tau_1 S_t + \gamma_2\tau_2 I_t) - v_2(1-M_t)(S_t+I_t) \\ - m_1(1-M_t)\tau_1 S_t - m_2(1-M_t)\tau_2 I_t < 0\end{aligned} \tag{12}$$

implies that:

$$m_2 > (p-l)\gamma_2 + \frac{((p-l)\gamma_1 - m_1)\tau_1 S_t}{\tau_2 I_t} - \frac{v_2(S_t+I_t)}{\tau_2 I_t}. \tag{13}$$

The left-hand side of equation 13 represents the implicit value of an infectious animal, which reflects its potential to produce healthy calves as well as its salvage value. The first term on the right-hand side represents the gross revenue obtained for each correctly identified diseased animal. This is followed by a term representing the net return for healthy animals slaughtered due to false positive status after a testing event (also expressed per diseased animal identified). The value of false positive animals slaughtered is their salvage value minus their implicit value as a productive asset. As the true level of prevalence in a herd declines, the proportion of reactors that are false positives will increase. Because producers only receive a fraction of fair market value for these animals, false positives become an important cost of testing at low levels of disease. The final term on the RHS represents the direct cost of testing.

Note that various components of the disease control program affect this private decision function in different ways. For example, by subsidising testing costs (v_2), regulators encourage testing. Similarly, subsidised compensation for test-positive animals (γ_1, γ_2) increases the incentive to test, as would adopting a testing procedure with a higher test sensitivity (τ_2) or specificity ($1-\tau_1$). Equation 13 illustrates clearly why individual producers cannot be expected to voluntarily follow a testing program which eradicates disease. As the ratio of healthy to infected animals increases, the cost of identifying diseased animals also increases, making it uneconomic to test and remove animals from the herd.

4.2 The private decision to control wildlife vectors

In addition to testing and removing infected cattle, producers have a private incentive to reduce the disease reservoir, or local possum population, in this example. Assuming an interior control, profits from raising cattle will be maximised when the harvest rate for the wildlife vector population is adjusted each period so that marginal harvest costs are just balanced by the discounted marginal benefits of removing the potential source of disease.

According to equation 14, the producer reduces the possum population in a manner which balances the marginal removal costs with the (negative) marginal benefit of the last unit left in the disease reservoir:

$$\frac{\partial H_{CV}}{\partial H_t} = -\frac{\partial PC(P1_t, H_t)}{\partial H_t} - m_3 = 0 \tag{14}$$

Estimation of a cost function for vector control in New Zealand suggests that control costs escalate as pest densities decline, implying that the marginal cost of harvest will increase as successful control efforts lower pest densities.

The above analysis confirms conclusions drawn earlier (Morris and Blood 1969; Rubenstein 1977; Stoneham and Johnston 1986), that profit-maximising producers will control disease in the absence of regulation provided that the benefits of control outweigh the costs. Private benefits and costs can be influenced in important ways, however, by the mix and level of incentives associated with the centralised disease control program. The complete elimination of disease is unlikely to be optimal for any particular individual due to the sharply increasing marginal costs associated with controlling disease and the wildlife reservoir at low levels of prevalence and density, respectively. A collective approach may therefore be required to achieve eradication objectives that are more stringent than those that would be attained exclusively under private incentives.

5. Results from the empirical model

The control model was calibrated with parameter values estimated and/or derived to approximate current conditions in the Clarence/Waiau Tb endemic region in the South Island of New Zealand (see table 1 for variable definitions and calibrated values). Following Standiford and Howitt (1992), the model was specified as a non-linear programming problem and solved numerically on GAMS. Results from the base run of the empirical model are displayed in the first column of table 2. Assuming an enforced annual testing regime is in place ($T_t = 1$ for all t), a steady state cattle herd of 231 is reached within the first five time periods. The marketing activity follows a singular path of 36 per cent, or 83 head of cattle sold each time period. Annual revenue is maintained at \$27.44/ha, or \$44 672.32 for the cattle enterprise. When the initial values of the parameters are set substantially above or below this level, the steady state is reached as quickly as possible by setting the marketing variable at one of its extreme values. This 'most rapid approach' path is characteristic of linear control problems.

The above values compare vary favourably to statistics available from the New Zealand Sheep and Beef Farm Survey. At the beginning of the

1992–93 financial year, Class II farms reported an average herd size of 233 beef animals. Throughout the year 85 animals were marketed, which represents 36.5 per cent of the opening herd. Net revenue per hectare for the cattle enterprise was $27.01.

In an effort to further validate the model, numerical output on disease status was compared to actual herd histories for a small number of beef breeding herds in the Clarence/Waiau endemic region that have been experiencing trouble with persistent levels of Tb. Model results (not reported in table 2) suggest that 1.7 per cent of the cattle tested will return a positive result, but that 75 per cent of these reactor cattle will be false positives. While the reactor rate predicted by the model (1.7 per cent) is within the observed range of the averages calculated from the sample data (0.66–1.8 per cent), the model appears to have over-estimated the number of false positive reactors.

Post-mortem analysis of reactor carcasses from the Clarence/Waiau endemic region reveals that approximately half of the cattle destroyed for test-positive status in 1993–94 exhibited visible tuberculous lesions at slaughter, suggesting that at least half of the reactor cattle were actually tuberculous. The proportion of false positive reactors is a function of the true prevalence of disease in the herd as well as the specificity and sensitivity of the tuberculosis test. In particular, small increases in the specificity of the test (decreases in τ_1) will result in relatively large decreases in the absolute number of false positives. Subsequent discussions with animal health officials suggest that MAF veterinarians are achieving higher levels of both sensitivity and specificity than those suggested by the field trial upon which the model parameter estimates were based, which could account for the discrepancy between model output and the empirical data.

The marginal values on a number of the constraints provide interesting economic information on this complex system, as they represent shadow values for their respective constraints. The shadow value for testing, for example, is small in magnitude, but negative in all time periods. The implication is that enforced testing on an annual basis imposes an economic cost on the system despite the fact that the veterinary charges are not borne directly by the producer. Under the assumed values of the parameters, therefore, profit-maximising producers cannot be expected to test in every period. Increasing the initial number of infected animals results in a positive marginal value for the testing constraint in the beginning time periods, suggesting that the optimal strategy is to test early in the time horizon.

The marginal values on the equations of motion represent the costate variables for each respective state variable. Of particular interest is the costate variable for infected cattle. Although infected cattle have a smaller implicit value than healthy cattle, the fact that the costate variable is positive

Table 1 Variable definitions and parameter values

Variable	Definition	Base Value
S_t	Density of susceptible cattle (State variable, $S_0 = 0.14$)	hd/ha
I_t	Density of infected cattle (State variable, $I_0 = 0.002$)	hd/ha
$P1_t$	Density of farm possums (State variable, $P1_0 = 3$)	hd/ha
$P2_t$	Density of forest possums (State variable, $P2_0 = 2$)	hd/ha
M_t	Marketing activity (Control variable)	%
T_t	Tuberculosis testing effort (Control variable)	%
H_t	Harvest rate for possums (Control variable)	hd/ha
δ	Annual discount rate [a]	7.05%
p	Average price of cattle ($/hd) [b]	$570
l_t	Slaughter levy ($/hd) [c]	$8.70
γ_1	Compensation for non-lesioned test positive cattle [c]	85%
γ_2	Compensation for lesioned test positive cattle [c]	85%
μ	Market value salvaged from infected cattle [d]	35%
v_1	Variable costs of maintaining herd ($/hd) [e]	$15
v_2	Cost of testing cattle ($/hd) [f]	$1.50
$PC(H_t, P1_t)$	Cost function for possum harvest [g]	$\frac{w}{6.45}\left(\frac{H_t}{P1_t}\right)^2$
w	Cost of time spent possum hunting ($/day) [h]	$136
a	Maximum rate of growth for cattle herd (year^{-1}) [i]	0.67
K_c	Carrying capacity of pasture – cattle (hd/ha) [i]	0.35
b	Mortality for cattle (year^{-1}) [i]	0.02
r_{P1}	Intrinsic growth rate for farm possums (year^{-1}) [j]	0.3
K_{P1}	Carrying capacity for farm possums (hd/ha) [j]	3
r_{P2}	Intrinsic growth rate for forest possums (year^{-1}) [j]	0.2
K_{P2}	Carrying capacity for forest possums (hd/ha) [j]	2
D	Dispersion parameter (hd/ha/yr) [k]	1
Z	Possum disease threshold (hd/ha) [l]	1.4
β_1	Cattle-cattle disease transmission coefficient (year^{-1}) [m]	3
β_2	Possum-cattle disease transmission coefficient (year^{-1}) [n]	0.003
τ_1	1 - Specificity of the tuberculosis test	0.02
τ_2	Sensitivity of the tuberculosis test	0.66

Notes:

[a] Seasonal loan rate for farm-related business expenditure, secured with farm property and/or stock (Burt and Fleming 1994).

[b] Weighted average of farm-gate prices for all cattle sold from Class II farms (NZMWB various issues).

[c] *National Tb Strategy* (AHB 1995).

[d] Scott and Forbes (1988) estimated that producers receive 35 per cent of full market value for infected animals.

[e] Average direct expenditure per head taken from the Financial Budget Manual (Burt and Fleming 1994).

[f] Average mustering and handling costs for typical North Island Hill Country sheep and beef farm stocking 340 head of cattle (Nimmo–Bell 1994). This should be considered 'lower bound'. This does not include veterinary charges, which are currently covered by the National Tb Control Scheme.

[g] The cost function for possum control was derived by solving the following economic problem: $\min_{Et} TC(E) = wE$, subject to $H_t = qE_t^{\alpha_1} P1_t^{\alpha_2}$. The harvest function was estimated econometrically using data collected from twenty ground control operations throughout New Zealand. Parameter values of $q = 2.54$, $\alpha_1 = 0.56$ and $\alpha_2 = 0.96$ were all significant at the 10 per cent level or above. A test of the joint hypothesis that $\alpha_1 = 0.5$ and $\alpha_2 = 1.0$ could not be rejected at a 0.5 per cent level of significance, yielding the parameter values listed above.

[h] This represents the average cost per day for contract hunters for 9 ground-based operations on blocks of less than 1 000 ha (Warburton and Cullen 1993). This does not include the cost of planning the

operations or their subsequent monitoring.
[i] Estimated from data in the New Zealand Sheep and Beef Farm Survey (NZMWB various issues).
[j] Estimates of r for possums range from 0.2 to 0.59, while values for K range from fewer than 1 to over 25 (Batcheler and Cowan 1988). Values chosen for r_{P1} and K_{P1} appear to be most representative of farmland/scrub habitat. Possum populations in forest habitats, by contrast, grow at a slower intrinsic rate to a smaller carrying capacity, suggesting lower parameter values for the uncontrolled parcel.
[k] Data for the estimation of this parameter were taken from a number of ecological studies (Clout and Efford 1984; Green and Coleman (reported in Batcheler and Cowan 1988), Barlow 1993) which imply that under a variety of habitat conditions, when the population of possums in a controlled area has been substantially reduced, possums from surrounding undisturbed habitats will immigrate at the rate of 1 hd/ha.
[l] Following current control practices, which are based on Barlow's modelling work (1991a; 1991b), Z is set at a number representing 40 per cent of the possum carrying capacity.
[m] Based on past attempts to estimate a coefficient for the transmission of tuberculosis between infectious and susceptible cattle in New Zealand, which suggest a range for β_i of 2.77 to 6.12 (Kean 1993).
[n] Data limitations and cost constraints have precluded precise estimates of the probability of transmission between possums and cattle.
[o] A field trial of the tuberculosis test under New Zealand field conditions suggests a specificity and sensitivity of 98 per cent and 66 per cent, respectively (Ryan *et al.* 1991).

Table 2 Summary of steady-state values for key variables: policy analysis

	Policy [a]						
Variable	1	2	3	4	5 [b]	6	7
Prevalence	1.11%	1.02%	1.10%	0%	0.57%	1.11%	1.0%
Herd size (hd)	231	229	228	233	232	231	231
Sales (hd)	83	83	83	85	84	83	84
Harvest Rate	2.16%	3.61%	2.16%	–	2.54%	2.16%	4.24%
Density $P1$ (hd/ha)	2.84	2.73	2.84	–	2.1	2.84	2.68
Density $P2$ (hd/ha)	1.92	1.87	1.92	–	1.54	1.92	1.85
Costate for S	\$562	\$566	\$569	\$565	\$563	\$562	\$563
Costate for I	\$202	−\$52	\$205	\$187	\$194	\$202	\$200
Costate for $P1$	−\$0.32	−\$0.55	−\$0.32	\$0.00	−\$0.51	−\$0.32	−\$0.32
Costate for $P2$	−\$0.21	−\$0.37	−\$0.21	\$0.00	−\$0.38	−\$0.21	−\$0.22
Revenue (\$/ha)	\$27.44	\$27.33	\$27.23	\$27.76	\$27.59	\$27.44	\$27.46

Notes:
[a] (1) Base values for the parameters and enforced testing of all animals ($T_i = 1$ for all t). (2) No compensation. $\gamma_1 = 0.9, \gamma_2 = 0.35, l = \$7.70$. (3) User pays for testing. $v_2 = \$5.00, l = \$5.50$. (4) Elimination of disease reservoir. $\beta_2 = 0$. (5) Periodic possum control. Exogenous harvest every 5 years. (6) One exogenous possum control operation. (7) 50 per cent subsidy on cost of harvest;
[b] Results reported in terms of averages for $t = 10 - 40$.

implies that infected cattle are a productive economic asset despite their contribution to the disease process. Policy analysis in the following sections indicates that reactor compensation has a profound effect on the implicit value of infectious cattle.

The model predicts that possum harvesting activities will be maintained at a low level of approximately 2 per cent of the farm possum population, which encourages a small amount of migration from the adjacent habitat.

Although the costate variable for possums indicates that they have negative economic value, the costs of harvesting under the assumed wage preclude a more rigorous level of control.

6. Policy implications

The Animal Health Board is concerned that national tuberculosis control policies such as reactor compensation and subsidised possum control have distorted market signals and deterred individual producers from more active participation in the disease control process. As a consequence, the Board is seeking to identify policies which achieve lower levels of disease by encouraging individual producers to take more responsibility for the level of disease within their herds (AHB 1995). Numerical simulation with the model estimated above provides a means by which the relationship between potential policies and the resulting level of disease can be explored before costly policies are instituted.

6.1 Eliminating compensation payments

One of the policies under investigation by the Animal Health Board is payment for reactor cattle. Compensation payments will be reduced under the latest National Tb Strategy, and closely reviewed in the years that follow. For simulation purposes, a 'no compensation' policy was approximated by increasing γ_1 and decreasing γ_2 (policy 2, table 2). Results suggest that producers would achieve a slightly lower prevalence without compensation by increasing the possum harvest rate and maintaining a smaller herd to reduce both between and within species transmission. A similar level of sales is obtained by marketing a larger percentage of a smaller herd. Annual revenue falls by approximately \$179.08.

One variable of particular interest is the value of the costate variable on infected cattle. In the *ceteris paribus* absence of compensation, the implicit value of an infected animal becomes negative. While it is still not optimal to test every period (the marginal value on the testing constraint becomes negative after the fifth period), the elimination of compensation implies a very different value for infected stock. As a consequence, producers take a more active role in controlling the spread of disease within their herds.

Compensation was initiated in part to encourage compliance with Tb control measures (AHB 1995). There is a perception, therefore, that the elimination of compensation payments may prompt non-compliant behaviour. Additional numerical work with the empirical model (not reported in this analysis) indicates that when testing is a choice variable, the elimination of compensation leads to a decrease in testing activity, which

results in a higher level of prevalence on average. These results support the Board's concern, and provide a prime example of how the success of one policy (elimination of compensation) depends critically on the existence of another (enforced testing).

6.2 User pays for tuberculosis testing

In an effort to make the costs of tuberculosis control more transparent, the Board considered several proposals that would have required producers to pay directly for tuberculosis testing. The latest National Tb Strategy indicates that whole herd testing will continue to be funded from the slaughter levy, but that producers are expected to pay for ancillary testing and tests associated with movement control. As with reactor compensation, the Board will monitor the testing policy, and propose changes if they feel that the goals of the strategy are being compromised. A 'user pays' system for whole herd testing was simulated by increasing the cost of the test, but continuing to force the producer to test annually (policy 3, table 2). When annual Tb testing is enforced by regulation, increasing the cost of testing serves primarily to reduce annual revenue for producers of breeding and/or store stock in endemic regions by approximately \$0.21/ha (or \$342 for the cattle enterprise). The very slight reduction in prevalence results from the maintenance of a smaller steady-state herd.

Once again, it is important to note that the results of this analysis rely on the fact that annual tuberculosis testing is enforced by regulation. The variable cost of testing is an important component of the switching function for testing, and can be expected to influence the annual decision to test. Numerical analysis confirms this expectation, by revealing a strong (direct) relationship between the cost of testing and the resulting level of disease when producers choose the level of testing activity that maximises profit.

6.3 The wildlife reservoir

Given (1) the base values for the parameters; (2) the 'closed herd' assumption; and (3) an enforced testing regime, the key force driving disease dynamics in the system is reinfection from the wildlife reservoir. The following sub-sections examine the implications of an exogenous reduction in the population of possums that have direct access to cattle. The results of the analysis are shown to depend critically on (1) whether and how quickly the possum population manages to recover from control operations; and (2) whether producers are expected to fund continuing maintenance control directly. A subsidy on the direct cost of possum control is considered in the final sub-section.

6.4 Elimination of the wildlife reservoir

The successful elimination of the wildlife reservoir was simulated by setting β_2 equal to zero. Results indicate that if possums are eliminated as a source of tuberculosis, annual testing will eventually drive disease from the cattle population. Steady-state values for the state and control variables (policy 4, table 2) indicate that in the absence of tuberculosis, producers would market more animals from a larger herd, thereby increasing their annual revenue by over $520 per year.

6.5 Recovery of the possum population

While the results reported above imply that eliminating the wildlife reservoir will lead to the eventual eradication of disease in cattle, concern has been raised about the implications of population recovery through reproduction and immigration of possums from neighbouring (uncontrolled) habitats. Prior modelling work suggests that immigration may pose a particular threat to producers on small blocks of land when possum control is periodic (Hickling 1993). To develop an appreciation for the possible effects of population recovery, periodic possum control operations were simulated by exogenously reducing the possum population in the controlled habitat every five years (policy 5, table 2).

Under the assumed values of the parameters, the possum population recovers from control operations quite rapidly through reproduction and immigration from the uncontrolled habitat. Disease consequently persists in the cattle herd at an average level of approximately 0.56 per cent. Reducing the dispersion parameter slows the rate of recolonisation, but disease remains in the herd at low levels even in the absence of immigration. The clear implication of these simulation results is that possum control operations must be persistent and well monitored if they are to be effective as a disease eradication strategy.

6.6 Implications of a single exogenous reduction in the possum population

The AHB has signalled a commitment to a policy that would involve a one-time subsidised reduction of the possum population, provided landowners actively maintain these populations at low levels. The Board envisions that landowners will fund these operations either collectively, or by direct contribution (AHB 1995). While it may be tempting to assume that producers acting independently will maintain a control program initially undertaken on their behalf, economic theory tells us that such sunk costs are irrelevant for current (and future) decisions. Exogenously funded control

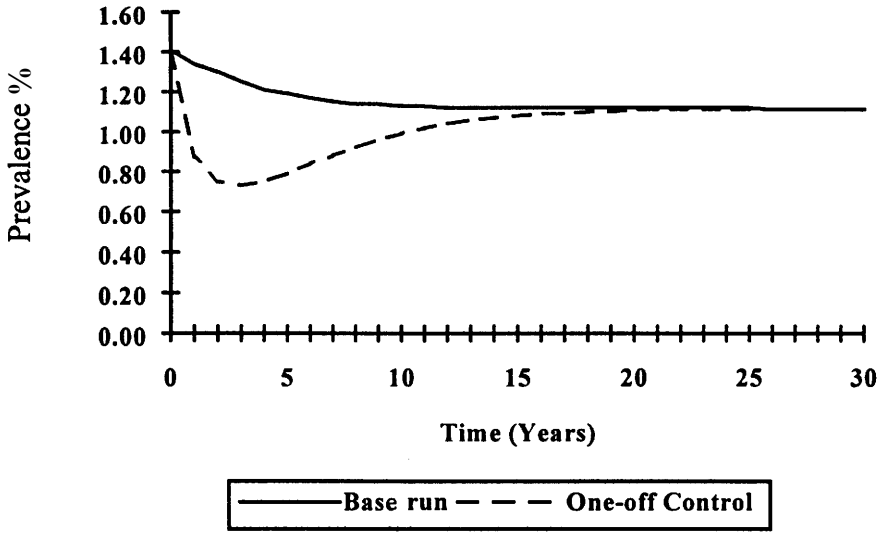

Figure 2 Trajectory for disease: one-off control versus base run

efforts do not change the basic incentive structure facing an individual producer. Paradoxically, reductions in the possum population serve only to make individually funded control more expensive, as control costs are inversely related to population density. Producers should therefore not be expected to maintain possum numbers at low levels if such control activities were not optimal prior to any activity undertaken by the AHB. This phenomenon is demonstrated in figure 2, which compares the timepath for prevalence following a single exogenously funded control operation to the disease level in the base run.

In the simulated 'one-off' control scenario prevalence is initially driven below the base level, but it climbs to the steady-state level reached in the base run relatively rapidly (policy 6, table 2). This is hardly surprising, as none of the parameters which dictate the steady-state values for the state and control variables have changed. Removal of the dispersion flux terms does not change the qualitative results of the model, it simply takes longer for the controlled population to recover. In either case the producer-funded harvest rate starts at a lower level than without the one-off policy, and then increases smoothly to the original steady-state level.

6.7 Subsidising the possum harvest

Results from the analytical model indicate that when making possum control decisions, producers balance the (private) marginal cost of harvest against

the benefits of a reduction in the level of prevalence. Policies aimed at changing the marginal cost of harvest can therefore be expected to have a much more profound effect on producer behaviour than policies that exogenously reduce the possum population. Reducing the cost per day of harvesting possums simulated a subsidy on the cost of possum control (policy 7, table 2). Predictably, there is a direct relationship between the level of subsidy and the resulting amount of effort directed towards possum harvest. More detailed numerical work, however, indicates that the responsiveness of prevalence to a reduction in the cost of harvesting possums depends critically on the level of transmission between possums and cattle, as well as the range of subsidy under consideration.

As demonstrated in figure 3, the response of prevalence is much more dramatic when the probability of effective contact between possums and cattle is high. This makes intuitive sense since the implicit cost associated with not harvesting possums is much higher when the probability of disease transmission is high. This is reflected in the empirical model by a much more negative marginal value on both of the possum populations at relatively high values of β_2. Prevalence is also revealed to be relatively unresponsive to decreases in the cost of harvesting at low levels of subsidy, particularly when the probability of disease transmission between possums and cattle is low. In general, the reduction in the possum population that follows a subsidy on possum harvest allows the producer

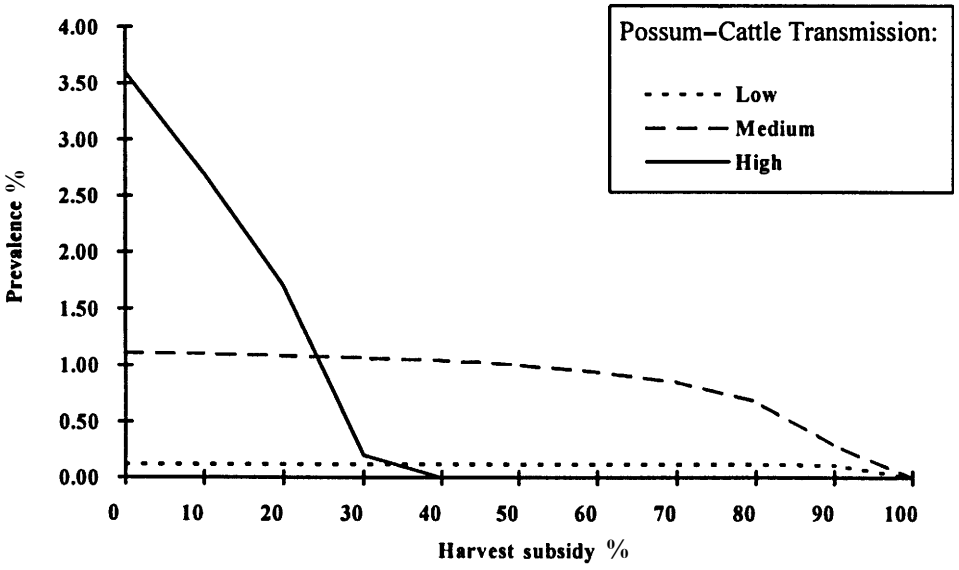

Figure 3 Disease response to harvest subsidy at various levels of possum–cattle transmission

to maintain a slightly larger herd, from which a greater number of cattle are marketed each year. As expected, net revenue from the cattle operation increases as prevalence declines.

7. Conclusion

This article was motivated by the ongoing debate regarding the role of the public sector in livestock disease control, and the empirical difficulties associated with controlling a disease for which an effective wildlife reservoir exists. The optimal control methodology developed in this article embeds models of disease transmission and species interaction into a dynamic optimisation framework, incorporating non-linear relationships between the state and control variables. First-order conditions from the theoretical model suggest that individual profit-maximising producers are not likely to eradicate disease from their herds due to the increasing marginal cost of control as prevalence declines.

Results from the empirical model confirm that bovine tuberculosis control policies in New Zealand are currently achieving lower levels of prevalence than would prevail in the absence of a national strategy. These policies also appear to be distorting market signals and removing some of the individual incentive to control disease. The elimination of compensation payments for reactor cattle provides a more accurate indication of the value of infected stock, and encourages producers to reduce prevalence by increasing their possum harvest activities and maintaining slightly smaller herds. In a regulated environment the introduction of a 'user pays' philosophy for testing simply transfers the cost of testing cattle to those who use the service most heavily. It should be noted, however, that compensation payments and subsidised testing both serve to encourage testing activity. The elimination of these policies may therefore increase the risk of non-compliance. Policies aimed directly at suppressing the wildlife reservoir appear to be the most effective at reducing prevalence in bovine Tb endemic areas. Given the tendency for possum populations to recover through a combination of migration and reproduction, however, control efforts must be well monitored and diligently maintained.

The theoretical framework and the empirical bioeconomic model developed in this article show considerable promise for the analysis of a wide range of disease control issues. While countries throughout the world are reconsidering the role of the public sector in the delivery of animal health services, externalities associated with the control of infectious diseases suggest that private solutions to disease control problems will not be socially optimal. This model provides a means by which important trade-offs among disease control inputs can be analysed, and the effects of proposed policies can be examined in a relatively inexpensive *ex ante* manner.

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