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**Public Policy and Private
Incentives for
Livestock Disease Control**

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The widely recognized externalities associated with livestock disease control have prompted countries throughout the world to invest in centralized 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 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 as to the potential behavioural responses of producers who raise livestock primarily for economic profit (Dietrich, et al., 1987; Ebel, et al., 1992; Liu, 1979). This paper utilizes recent advances in the dynamic bioeconomic literature to develop a behavioral 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 optimizing behaviour into one, unifying optimal control model.

General results from the theoretical model suggest that individual profit maximizing producers are not likely to eradicate disease from their herds due to the sharply increasing marginal cost of control as prevalence and vector density decline. Policies which encourage higher levels of disease control may be necessary, therefore, to achieve prevalence levels which meet stringent human health and international trade standards. Estimation and calibration of the theoretical model using bioeconomic data collected from a tuberculosis endemic region in New Zealand allows us to examine the potential impact of several proposed policy changes within the context of a specific empirical application. Numerical results, validated with actual whole herd testing histories, suggest that compensation payments for test positive cattle are inflating the

implicit value of infected stock and reducing private incentive to control disease. When annual testing is enforced by regulation, the introduction of a 'user pays' system for diagnostic testing simply transfers the cost of control to those who use the services. It should be noted, however, that subsidized testing provides producers with a powerful incentive to comply with program objectives. The success of policies which focus on controlling the wildlife reservoir for disease depends critically on whether the pest population recovers after control operations, as well as the probability of disease transmission between and among species.

1. Empirical Problem

This paper 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 in New Zealand. Although centralised control efforts have dramatically reduced the apparent prevalence of Tb in New Zealand over the past fifty 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 the 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.

Currently used control measures for bovine Tb in New Zealand include diagnostic testing and subsequent slaughter of test positive cattle, the controlled movement of cattle from infected herds, slaughterhouse surveillance, and possum control operations. In order to encourage participation in the test and slaughter program, policies such as subsidized testing and compensation for reactor cattle have been introduced. Animals are tested each year and positive reactors are slaughtered. Producers receive a subsidized price for test positive animals, which has recently been reduced from 85 to 65% of fair market value. An additional check for tuberculous cattle is conducted at the slaughterhouse. Lesioned carcasses are traced back to the farm of origin, where producers receive a salvage price determined by the market for cattle by-products. Subsidies have also been received 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 which will introduce a more market-oriented approach to disease control. Discussion has focused on reducing compensation payments and introducing a ‘user pays’ system for testing. Funding for possum control operations will be sought from those who contribute to the possum problem, as well as those who benefit from possum control. Government monies will therefore continue to be used to fund operations on Crown land.

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 which 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 disease state, 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 relatively densely populated uncontrolled habitat after control operations have reduced possum numbers near the cattle herd. In the absence of control, the change in the possum populations is determined by the biological growth rate and the migratory flux between controlled and uncontrolled habitats. Possums in the vicinity of a cattle operation can be further reduced by harvesting.

2. Model Structure

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 wildlife reservoir 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 maximizes discounted net revenue from the sale of cattle.

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

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 centralized disease control scheme. The control variable for marketing is expressed in percentage terms, so the gross revenue from marketing healthy cattle is $(p-l)M_t S_t$.

Because producers cannot distinguish between healthy and infectious animals by casual observation, the marketing activity reduces both categories of cattle indiscriminately. All infectious stock sent to market will, however, be detected through routine slaughterhouse 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 hypothesized 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:

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

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

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, who may become infected by either ingesting or inhaling live bacteria (Blood and Radostits, 1989). Following the methodology suggested by Anderson and May (Anderson and May, 1979a, Anderson and May, 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 comprised of two components: the probability of close contact between an infectious and a susceptible individual, and the probability that transmission will occur as a result of the contact (Nokes and Andersen, 1988). To reflect 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; Barlow, 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. The model implicitly specifies a maximum number of one whole-herd test per year. Although the overall sensitivity and specificity of a testing program can be improved by testing more frequently throughout the year, multiple testing is extremely uncommon in endemic areas. 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 (Clout and Barlow, 1982), the biological growth of the possum populations is described by a simple logistic equation.

$$(4) \quad \dot{P}_1 = r_{P1} P_{1,t} \left(1 - \frac{P_{1,t}}{K_{P1}}\right) - D \left(\frac{P_{1,t}}{K_{P1}} - \frac{P_{2,t}}{K_{P2}}\right) - H_t$$

$$(5) \quad \dot{P}_2 = r_{P2} P_{2,t} \left(1 - \frac{P_{1,t}}{K_{P1}}\right) + D \left(\frac{P_{1,t}}{K_{P1}} - \frac{P_{2,t}}{K_{P2}}\right)$$

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 Steneth (1988), who have observed that animals will emigrate from relatively densely populated habitats if the opportunity arises. Migration is therefore hypothesized to be density dependent, and will increase the speed at which managed populations recover from control activities. The parameter D essentially represents the maximum number of possums that will

recolonize 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 maximize Equation 1, subject to Equations 2 - 5, which represent the biological equations of motion for this bioeconomic model. Equations 6 and 7 place additional restriction on the state and control space, respectively, and Equation 8 specifies the initial conditions for the state variables.

$$(6) \quad S_t, I_t, P1_t, P2_t \geq 0$$

$$(7) \quad 0 \leq M_t \leq 1; 0 \leq T_t \leq 1; 0 \leq H_t \leq 0.85P1_t$$

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

3. General Implications of Optimizing Behaviour

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

$$(9) \quad H_{CV} = \left\{ (p-l) \left(M_t(S_t + \mu I_t) + T_t(1-M_t)(\gamma_1 \tau_1 S_t + \gamma_2 \tau_2 I_t) \right) - \right. \\ \left. v_1(S_t + I_t) - T_t v_2(1-M_t)(S_t + I_t) - PC(P1_t, H_t) \right\} + \\ m_2 \left\{ f_1(S_t, I_t) + f_2(S_t, P1_t) + M_t I_t - T_t \tau_2(1-M_t)I_t - bI_t \right\} + \\ m_3 \left\{ g_2(P1_t) - f_3(P1_t, P2_t) - H_t \right\} + m_4 \left\{ g_3(P2_t) - f_3(P1_t, P2_t) \right\}$$

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).

The Hamiltonian is linear in testing and marketing, and non-linear in wildlife harvest. The optimal solution will therefore consist of some combination of singular and bang-bang controls for T_t and M_t , and an interior solution for H_t (unless the marginal cost of harvest outweighs the discounted benefits). 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 as to what motivates profit maximizing producers control disease.

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

$$(10) \quad \frac{\partial \mathcal{H}_{cv}}{\partial T_t} = \sigma_T = (p-l)(1-M_t)(\gamma_1 \tau_1 S_t + \gamma_2 \tau_2 I_t) - \nu_2(1-M_t)(S_t + I_t) - m_1(1-M_t)\tau_1 S_t - m_2(1-M_t)\tau_2 I_t.$$

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

$$(11) \quad T_t = \begin{cases} 0, & \sigma_T < 0 \\ T^*, & \sigma_T = 0, \\ 1, & \sigma_T > 0 \end{cases}$$

which 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:

$$(12) \quad \sigma_T = (p-l)(1-M_t)(\gamma_1 \tau_1 S_t + \gamma_2 \tau_2 I_t) - \nu_2(1-M_t)(S_t + I_t) - m_1(1-M_t)\tau_1 S_t - m_2(1-M_t)\tau_2 I_t < 0$$

implies that:

$$(13) \quad m_2 > (p-l)\gamma_2 + \frac{((p-l)\gamma_1 - m_1)\tau_1 S_t}{\tau_2 I_t} - \frac{\nu_2(S_t + I_t)}{\tau_2 I_t}.$$

The LHS of Equation 13 represents the value of an infectious animal as a productive asset of (healthy) calves. The first term on the RHS represents the gross revenue obtained for each

correctly identified diseased animal. This is followed by a term representing the gross value of healthy animals slaughtered due to false positive status after a Tb test (also expressed per diseased animal identified). The gross value of false positive animals slaughtered is their salvage value minus their shadow value as a producing unit. 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 subsidizing testing costs (v_2), regulators encourage testing. On the other hand, the levy (l) reduces the implicit value of a slaughtered beast, and therefore reduces the incentive to test. Similarly, subsidized 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 also illustrates clearly why individual producers cannot generally be expected to voluntarily follow a testing program which completely eradicates disease. As the ratio of healthy to infected animals rises, the costs of identifying the last diseased animals rise, making it uneconomic to test and remove animals from the herd.

Vector control. In addition to testing and removing infected animals, 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 maximized 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:

$$(14) \quad \frac{\partial \mathcal{H}_{cv}}{\partial H_t} = - \frac{\partial PC(P1_t, H_t)}{\partial H_t} - m_3 = 0$$

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 maximizing 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 Animal Health Board's 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.

4. Results from the Empirical Model

The control model was calibrated with parameter values estimated and/or derived to approximate current conditions in the Clarence/Waiiau 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, a steady state cattle herd of 231 is reached within the first five time periods. The marketing activity follows a singular path of 36%, 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% of the opening herd. Net revenue per hectare for the cattle enterprise was \$27.01.

Numerical results suggest that disease will persist in the herd at a real prevalence of 1.11%, and that seventy-five percent of the reactor cattle will be false positives. In an effort to validate the model, this output was compared to actual herd histories for a small number of beef breeding herds in the Clarence/Waiiau endemic region that have been experiencing trouble

with persistent levels of Tb. While the reactor rate predicted by the model (1.7%) is within the observed range of the averages calculated from the sample data (0.66% -- 1.8%), the model over estimated the number of false positive reactors.

Post mortem analysis of reactor carcasses from the Clarence/Waiiau 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. The shadow value for testing 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 maximizing 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 early in the first few years, 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 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% 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 precludes a more rigorous level of control.

5. General Policy Implications

The Animal Health Board is concerned that national tuberculosis control policies such as reactor compensation and subsidized 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.

5.1 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 (second column of 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 \$0.11 per hectare, or 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. Numerical work with the empirical model 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).

5.2 User Pays for Tuberculosis Testing

In an effort to make the costs of tuberculosis control more transparent, the Board considered several proposals which 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. 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 (Policy 3 in Table 2). The very slight reduction in prevalence results from the maintenance of a smaller steady-state herd. Producers who fatten store stock for slaughter would clearly be the major beneficiaries of a policy which increase the variable cost of testing as they maintain proportionately fewer breeding animals which require regular testing.

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 maximizes profit.

5.3 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 subsections 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.

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 possum 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 in 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.

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 5 years.

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%. Reducing the dispersion parameter slows the rate of recolonization, 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.

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 subsidized 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 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. 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.

Subsidizing 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 which exogenously reduce the possum population. A subsidy on the cost of possum control was simulated by reducing the cost per day of harvesting possums. 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 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.

6. Conclusions

This paper 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 paper embeds models of disease transmission and species interaction into a dynamic optimization framework, incorporating non-linear relationships between the four state and three control variables. First order conditions from the theoretical model confirm results from earlier studies: individual profit maximizing 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 while 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 strategy, they 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 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 subsidized 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 paper 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.

Figure 1: Schematic Diagram of Bioeconomic System

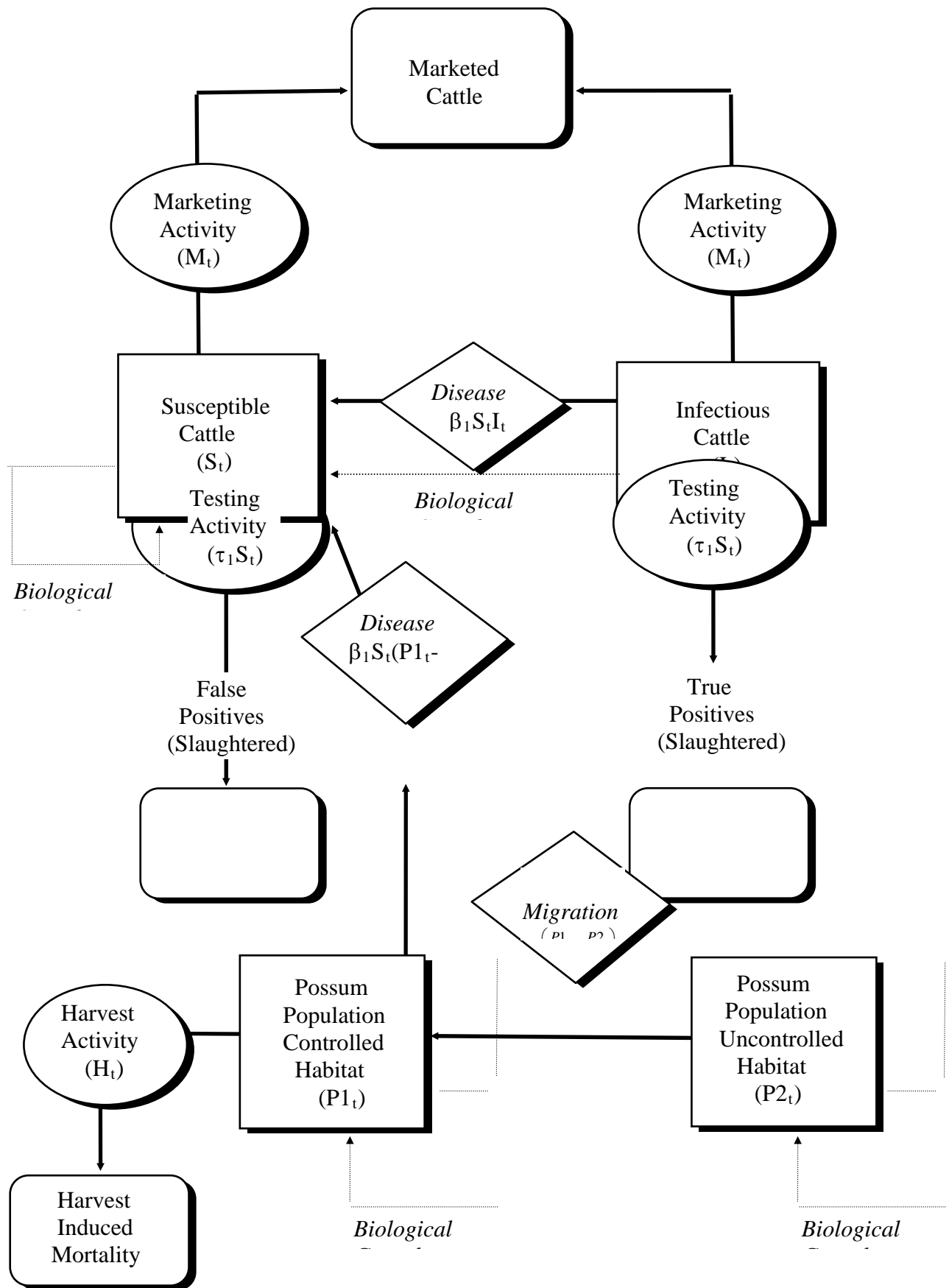


Figure 2: Trajectory for Disease: One-off Control versus Base Run

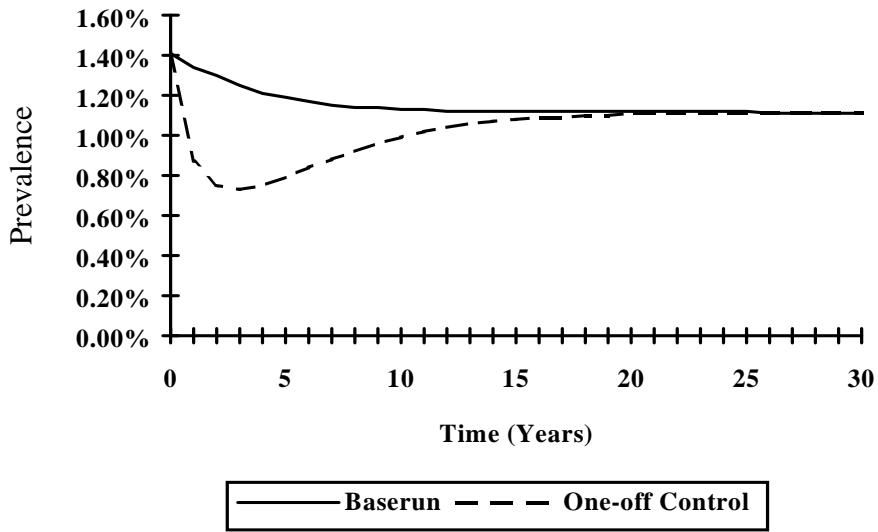


Figure 3: Disease Response to Harvest Subsidy at Various Levels of Possum-Cattle Transmission

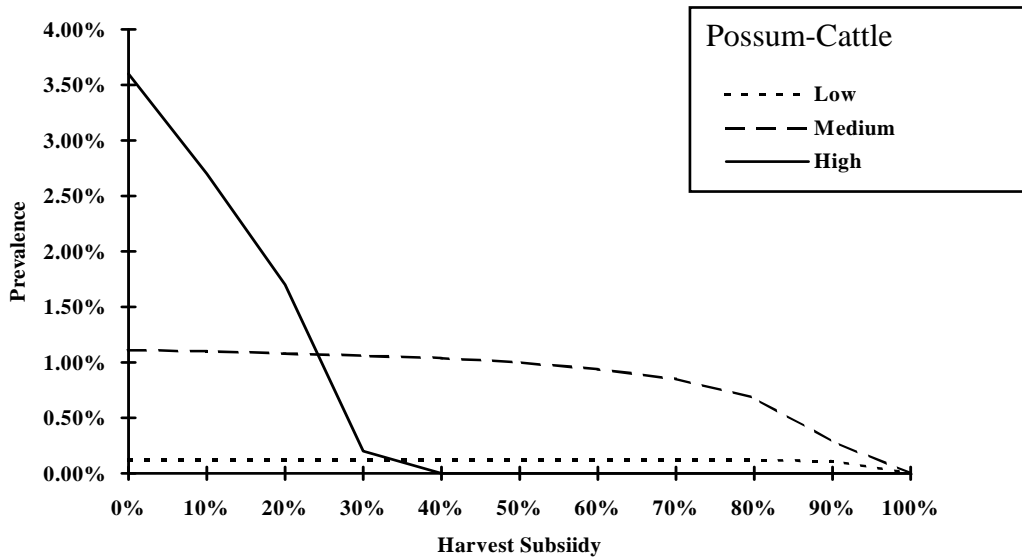


Table 1
Variable Definitions and Parameter Values

Variable	Definition	Base
S_t	Density of susceptible cattle (state variable)	hd/ha
I_t	Density of infected cattle (state variable)	hd/hd
$P1_t$	Density of farm possums (state variable)	hd/ha
$P2_t$	Density of forest possums (state variable)	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-leisioned test positive cattle ^c	85%
γ_2	Compensation for leisioned 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	$\frac{w}{645} \left(\frac{H_t}{P1_t} \right)^2$
w	Cost of time spent possum hunting (\$/day) ^h	\$136
a	Maximum rate of growth for cattle herd (-t) ⁱ	67%
K_c	Carrying capacity of pasture -- cattle (hd/ha) ⁱ	0.35

Table 1 (continued)

Variable	Definition	Base
b	Mortality for cattle (-t) ⁱ	2%
r _{P1}	Intrinsic growth rate for farm possums (-t) ^j	30%
K _{P1}	Carrying capacity for farm possums ^j	3
r _{P2}	Intrinsic growth rate for forest possums ^j	20%
K _{P2}	Carrying capacity for forest possums ^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 ^m	3
β ₂	Possum-cattle disease transmission coefficient ⁿ	0.003
τ ₁	(1 - specificity of the tuberculosis test) = % False ^o	2%
τ ₂	Sensitivity of the tuberculosis test = % True	66%

- ^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 thirty-five percent 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). Should be considered 'lower bound'. 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: $\min_{E_t} TC(E) = wE$, subject to $H_t = qE_t^{\alpha_1} P1_t^{\alpha_2}$. The harvest function was estimated econometrically using data collected from 20 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% 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% level of significance, yielding the parameter values listed above.

- ^h 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). 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_{PI} and K_{PI} 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 was taken from a number of ecological studies (Clout and Efford, 1984); Green and Coleman [reported in Batchelor 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 to a number representing 40% 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% and 66%, respectively (Ryan, et al., 1991).

Table 2
Summary of Steadystate Values for Key Variables: Policy Analysis

Variable	Policy ¹						
	1	2	3	4	5 ²	6	7
Prevalence	1.11%	1.02%	1.10%	0%	0.57%	1.11%	1.0%
Herds size	231	229	228	233	232	231	231
Sales	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

¹ 1) Base values for the parameters. 2) No compensation. $\gamma_1 = 0.9$, $\gamma_2 = 0.9$, $l = \$7.70$. 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% Subsidy on cost of harvest;

² Results reported in terms of averages for $t = 10 - 40$.

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