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LIVESTOCK DISEASE MANAGEMENT:
ON-FARM BIOSECURITY ADOPTION,
ASYMMETRIC INFORMATION IN POLICY DESIGN, AND
DECENTRALIZED BIOECONOMIC DYNAMICS

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Benjamin M. Gramig

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By

Benjamin M. Gramig

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ABSTRACT

ESSAYS ON THE ECONOMICS OF LIVESTOCK DISEASE MANAGEMENT:
ON-FARM BIOSECURITY ADOPTION,
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Livestock disease management involves both private and public resources and takes place in an environment of uncertainty. An econometric procedure to estimate disease control functions that will inform herd-level decision making is proposed and demonstrated to shed light on the determinants of health management practice adoption. An incentive compatible, government provided indemnity for private livestock assets culled in response to an outbreak of contagious disease when the government is constrained by hidden action and hidden information is characterized and compared with status quo indemnities. A bioeconomic model with feedbacks between disease and behavioral strategies is constructed to evaluate the nature of strategic effects between private decision makers in a decentralized setting when government policies are a source of externalities.

This dissertation is dedicated to my wife Melodi, without whose love, patience, and understanding it would not have been possible.

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Introduction

The outbreak of disease in domestic livestock herds is an economic and potential human health risk that involves both the government and individual livestock producers. Because livestock diseases impose significant costs on society (Bennett 1992, 2003; Bennett, Christiansen and Clifton-Hadley 1999; Bennett and Ijpelaar 2005; Buhr, et al. 1993; Chi, et al. 2002; National Research Council 2005), there is a need to understand management aspects of these problems. It is useful to understand both optimal public (centralized) management in response to a disease outbreak (Kobayashi, et al. 2007a, b; Mahul and Durand 2000; Mahul and Gohin 1999), and the decentralized behavioral and disease responses to common policy initiatives (Hennessy 2005, 2007; Hennessy, Roosen and Jensen 2005).

Economic justifications for public intervention in disease control include externalities, public good aspects of disease problems, coordination failures, information failures, and income distribution considerations (Ramsay, Philip and Riethmuller). The potential of a disease outbreak to pose a large economic cost depends on many factors including trade laws and the nature of the disease (i.e., infectiousness). For infectious diseases of public concern, policies range from bounties for infected livestock to required herd depopulation and farm decontamination; for diseases without international trade implications disease management may be a purely private endeavor.

The United States Department of Agriculture's Animal and Plant Health Inspection Service (APHIS) provides inspection and quarantine services to prevent the introduction of disease across national borders and also coordinates response to disease outbreaks that originate from within the country. Border measures that protect against

incursion of disease are provided to protect the safety of the American food production system and to prevent infection of the domestic livestock industry. Diseases not endemic to or currently present in the U.S. are typically not accounted for in everyday farm-level biosecurity measures because their risk of occurrence is viewed as exogenous to individual farm-level decision making. However, it should be noted that private health management measures taken with production diseases (as distinct from those of international concern) in mind may provide social benefits in the event of outbreak of an exotic or highly contagious disease by lowering disease transmission rates or preventing spread altogether, as well as keeping the farm prevalence rate of tolerable endemic diseases low overall. For these reasons, the government reasonably takes an interest in aggregate private investments in biosecurity in a more general sense.

Livestock disease management is an inherently dynamic process that involves feedbacks between disease ecology and private behavioral decisions. Throughout this dissertation I focus on two common decisions that livestock managers must make: a livestock manager must sequentially decide whether or not to invest in *biosecurity* as a preventive measure and whether or not to *report* infection to government authorities in the event that a disease outbreak occurs. The decision to report infection may or may not arise depending on the uncertain disease outcome, but at some point everyone faces the biosecurity decision. Because it is unclear in advance whether or not livestock will be exposed to infection, private investment in biosecurity represents a definite investment of resources in exchange for an uncertain future benefit (own herd protection).

Biosecurity may not fully protect one's own herd, and, moreover, losses due to increased regulatory stringency may also arise as a result of a neighbor's herd becoming

infected – even in the absence of infection in one's own herd. This is because all herds within infected regions may be affected by costly regulatory actions taken by animal health authorities to eradicate infection. Such regulatory-induced externalities are a common feature of livestock disease problems. For instance, all farms in the bovine tuberculosis (bTB) infected region in Michigan's Lower Peninsula — regardless of infection status — incur private costs as a result of dealing with government testing, movement restrictions and stringent testing rules for trade in live animals that go uncompensated by the government.

It is important to note that from the government's perspective individuals have private information about the preventive biosecurity measures in place on their farms—hidden actions—prior to outbreak (*ex ante*), and following an outbreak (*ex post*) they possess private information—hidden information—about the disease status of their herd. Once uncertainty about the occurrence of an outbreak is resolved, regardless of the *ex ante* actions taken by producers, disclosure of disease status (as opposed to discovery by another party through transmission, slaughter, or testing) is required for timely government response to limit the spread of infectious disease and eradicate it. The length of time between outbreak and discovery is very important in determining the duration and severity of epidemic (UN-FAO). For these reasons, creating an incentive structure that results in reporting of disease status is of great interest for a social planner.

This dissertation consists of three essays which each address particular elements of livestock health management behavior and disease risks in order to fill distinct gaps in the scientific literature and provide insights for policy design. The essays progress from a static farm-level analysis of a single private decision, to the design of government

policy in the presence of asymmetric information when private decisions are two-dimensional, to a jointly-determined dynamic system with externalities and two-dimensional private decision making.

The first essay considers the farm-level focus on production diseases and the decision to adopt individual biosecurity practices on the basis of private benefits and costs (even though social benefits may be provided as discussed above). An empirical procedure is proposed to inform private herd health decision making and analyze the determinants of biosecurity adoption behavior by accounting for preventive spillovers (positive input externalities) in the management of production diseases. The second essay is concerned with the optimal design of government provided indemnities when the regulator cannot observe preventive behavior or the infection status of individuals in the population. The centerpiece of this essay is the use of a single policy instrument to structure incentives so that private biosecurity and reporting actions are compatible with policy objectives when the disease of concern is responded to by mandatory culling of private herds. The final essay is the most complex of the three and accounts for dynamic feedbacks between behavioral choices and disease risks. A bioeconomic model of decentralized private decision making is constructed to examine the nature of strategic effects associated with biosecurity and reporting behavior when government disease eradication programs result in cross-farm externalities.

Essay One

ECONOMIC ANALYSIS OF LIVESTOCK HEALTH MANAGEMENT DECISIONS

Introduction

Livestock disease health management decisions are made weighing the costs of illness, prevention and control. These decisions are informed by the research of animal scientists, veterinarians and epidemiologists that quantifies the production effects of disease, identifies the individual management practices that are associated with the occurrence of specific diseases, and provides recommendations for prevention of disease. Private economic decisions about adoption of disease management practices utilize this information in tandem with associated market prices and the impact of the management practices on infection level.

We examine adoption of disease prevention and management actions taken by livestock managers for endemic production diseases.[1] Our research objectives are twofold: (1) estimate disease prevalence as a function of farm practices and characteristics and (2) analyze the determinants of health management practice adoption.[2] To address our first objective we adopt the fractional logit model as a statistical method

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[1] We define production diseases as those endemic to the US national herd which pose no potential for international trade sanctions because they are neither highly contagious nor threatening to human health. We assume that exotic or foreign animal diseases are not taken into account in daily decision making by farm managers because the government provides border controls to prevent entry of exotic diseases and pays indemnities in the event that outbreak of such diseases occurs and animals are culled from private herds to control the spread of infection and eradicate the disease.

[2] The former topic is best targeted at the veterinary health sciences, while the latter is very economic in nature. The two objectives are tackled simultaneously in a single essay, though it is felt that the exposition may be improved by addressing them on an individual basis.

that directly estimates herd prevalence. We find that a fractional response model provides more useful information for disease management decision making than standard logistic regression performed at the herd-level, which is common in veterinary epidemiological studies. Estimated herd prevalence allows for the comparison of costs and benefits of undertaking disease management practices when information about the production effect of disease is known.

In the second stage, we use the results from the first stage, which provide an estimate of the marginal effect of adopting a management practice on the herd prevalence of disease, to create a new variable that is the estimated cost of damages from disease suffered as a result of *not* adopting the management practice. This new variable becomes an explanatory variable in an adoption equation which is estimated using the familiar binary outcome logit model. An adoption equation is estimated in this way for each practice found to have a negative, statistically significant association with herd prevalence in the first stage regression. The empirical method is demonstrated utilizing the U.S. Department of Agriculture's (USDA) National Animal Health Monitoring System (NAHMS) survey of dairy farms in 1996. Specifically, we examine the adoption of management practices related to bovine leukosis virus (BLV).

After addressing the motivation for our two research objectives in detail and the methods proposed to address each in the next two sections, an empirical application of the proposed methods using herd-level data on a single disease and farm management practices on dairy farms in the United States is presented. The article concludes with a discussion of empirical results and possible extensions to this analysis using the full scope of data that may be available to the researcher.

Considerations for Herd-level Disease Management

Information about the association between disease and particular management practices often comes from veterinary epidemiological studies which refer to those practices found to be positively associated with disease as "risk factors" for infection. Two factors are of critical importance in evaluating the usefulness of statistical estimates from veterinary epidemiology for economic decision making: the level at which disease data are collected and how the disease outcome is represented. First, consider that studies in the animal health literature present estimates based on either individual animal data or herd-level data, which can affect the usefulness of estimates from the study for economic analysis. Second, the disease outcome that is studied determines the form that the dependent variable takes in the statistical model adopted. The two types of dependent variables that may represent the disease outcome of interest are binary and fractional. A binary disease outcome variable is the standard in the veterinary epidemiology literature and takes the form $y \in \{0,1\}$ where $y = 1$ denotes that the animal or herd is infected. A fractional disease outcome variable, $y \in [0,1]$, falls in the unit interval, is interpreted as the within herd prevalence of disease, and is considered only for the case of herd-level data. Because the binary response model is the "workhorse" in the epidemiology literature we discuss its usefulness for economic analysis first and then compare it to the fractional response model using herd-level data.

Statistical analysis of veterinary epidemiological data to identify risk factors for infection almost uniformly uses logistic regression and reports odds-ratios for individual explanatory variables (management practices and demographics), which may be either binary indicators or continuous variables (see most any general or veterinary

epidemiology text, such as Kahn and Sempos (1989) or Petrie and Watson (1999)). Odds-ratios from binary response models indicate the odds of infection (a binary outcome) when the covariate is present (if represented by a dummy variable) or when the level of the covariate is increased (if represented by a continuous variable) relative to when it is not. This interpretation is helpful for small probability events like the occurrence of a rare disease and explains in part why management practices found to lead to greater odds of infection are referred to by epidemiologists as "risk factors". In a statistical sense, when the probability of infection is very low (less than around 0.1) the difference between relative risk and the interpretation of the odds-ratio is negligible (Gould 2000). Odds-ratios from logistic regression are commonly reported by epidemiologists because of their usefulness in describing associations for a variety of sampling designs (Martin, Meek and Willeberg 1987; Selvin 1991) and because odds-ratios for individual parameters are insensitive to the levels of other explanatory variables (Gould 2000).

Applied econometricians tend to find marginal effects—interpreted as the estimated increase in the probability of infection associated with a marginal change in the explanatory variable—more intuitively palatable than odds-ratios. This is why partial effects are normally reported when logistically distributed errors are assumed in micro-econometric binary response models instead of the exponentiated parameter estimates reported by epidemiologists. As we find below in our empirical example, the odds-ratio (OR) reported by itself may suggest that undertaking a particular practice relative to some alternative cuts in half, as given by OR=0.5, the likelihood of herd infection in a binary response model, but the corresponding marginal effect (ME) on the probability of

infection represented by that odds-ratio may only be a several percentage point reduction, as given by ME=-0.05. For economic purposes, the interpretation of the odds-ratio alone is not unlike considering the statistical significance of a parameter estimate in isolation of the practical or economic significance of the estimate. Viewed in isolation, a highly significant odds-ratio may suggest either a large relative reduction or increase in likelihood of infection while the associated estimate of the change in probability may be quite small. Because of the potential for divergence between the interpretation of ORs and MEs, odds-ratios are not particularly helpful in evaluating the economic tradeoffs between adopting competing disease management practices unless the disease in question cannot be tolerated in the herd at any level (e.g., foot and mouth or bovine spongiform encephalopathy). Because individual herd disease management decisions revolve around production diseases rather than mandatory eradication programs, relying solely on odds-ratios to evaluate resource allocation is akin to minimizing the losses from infection, irrespective of costs.

Thus far we have only considered how results from binary response models are reported, in general, but it is just as important to consider the nature of the dependent variable because this affects the economic usefulness of the estimated coefficients. Binary response models may be estimated for individual or herd-level data. For the individual animal case, the dependent variable takes the form $y_i \in \{0,1\}$ where i indexes individual animals, $y_i=1$ for an infected animal and 0 otherwise. The binary response model takes the familiar form

$$(1) \qquad E(y_i \mid \mathbf{x}) = P(y_i = 1 \mid \mathbf{x}) = G(\mathbf{x}\boldsymbol{\beta}),$$

where $\mathbf{x}$ is a vector of explanatory variables inclusive of disease management practices, $\boldsymbol{\beta}$ is a vector of coefficients, and $0 < G(z) < 1$. The function $G(z)$ is usually a CDF and maps the index $\mathbf{x}\boldsymbol{\beta}$ into the response probability (Wooldridge 2002). In most epidemiological applications $G(z)$ is the CDF for the standard logistic distribution, where $G(z) = \exp(z)/[1+\exp(z)]$. From individual animal data the estimated probability $\hat{y}_i$ for a given $\mathbf{x}$ can be multiplied by the number of animals in the herd to get an estimated within herd disease prevalence. A comparison of the estimated prevalence when a practice is undertaken relative to when it is not can be used to evaluate the expected profit and decide whether it is worthwhile to adopt the management practice.

For the case of herd-level disease data the binary dependent variable takes the form $y_h \in \{0,1\}$ and is identical to (1) except for the nature of the dependent variable. The binary response model then becomes

$$(2) \qquad E(y_h \mid \mathbf{x}) = P(y_h = 1 \mid \mathbf{x}) = G(\mathbf{x}\boldsymbol{\beta}),$$

where h indexes the herd or farm such that y_h is the herd infection status and indicates that one or more animals on the farm is infected when $y_h = 1$. Because $\hat{y}_h$ is the predicted probability that a herd is infected, this estimate cannot be used to arrive at a predicted within herd prevalence and there is no information provided by the herd-level binary response model that allows economic decision makers to evaluate the relative cost of different management practices.

As an alternative to the familiar binary response model already discussed, consider that herd-level disease can be expressed as a fractional value of the form $y_h^f \in [0,1]$, where h again indexes herds. The super-script f distinguishes this fractional

response variable from the herd-level binary response variable in (2), and the fractional response model takes the form,

$$(3) \qquad E(y_h^f \mid \mathbf{x}) = G(\mathbf{x}\boldsymbol{\beta}).$$

Fractional response variables[3] are of particular interest for herd-level livestock disease management because it is costly to draw and test blood samples from every animal on a farm (as is required for individual animal disease data discussed in the context of binary response models above), and for food animals this typically only occurs in a research setting. Most practical on-farm disease diagnostic programs rely on serological tests from a subset of the herd chosen based on the sensitivity and specificity (Kahn and Sempos, 1989) of a particular diagnostic test in order to achieve a desired level of statistical confidence about the estimated within herd prevalence rate. Surveys like those conducted for the USDA's NAHMS are designed to achieve national disease prevalence estimates and rely on this type of sampling within private herds. Serological surveys of wild animals are conducted based on similar principles where sampling may occur at a higher level of aggregation than the "herd", such as a wildlife management unit. Because we are interested in estimating the conditional expected within herd prevalence for

[3] Fractional response variables are frequently encountered by agricultural economists. Examples of fractional or proportion values of interest to agricultural economists include the share of land devoted to the production of a particular crop, the proportion of cultivated land where a particular management practice is in use (e.g., reduced tillage, split nitrogen application, integrated pest management), the proportion of land in a county, township, or other jurisdiction devoted to a particular use (e.g., agricultural, commercial, residential, protected natural area), and the disease prevalence rate in a livestock herd or flock. A researcher studying such topics is generally interested in inference that allows for the identification of the determinants of crop selection, management practice or technology adoption rates, land use patterns, or rate of infection, respectively. To this end, statistical methods that allow the researcher to estimate the conditional expected fractional outcome of interest are desirable for empirical analysis of public policy and individual decision making.

alternative disease management practices in order to be able to aid economic decision making, a statistical method like fractional logit regression (Papke and Wooldridge 1996; Wooldridge 2002) that allows the researcher to directly estimate the conditional fractional outcome of interest with a relatively limited number of assumptions is indispensable.[4]

Fractional logit regression (Papke and Wooldridge 1996) is a quasi-maximum likelihood method (QML) based on the work of Gourieroux , Montfort and Trognon (1984) and McCullagh and Nelder (1989). Fractional logit utilizes the Bernoulli log-likelihood function

$$(4) \qquad L(\boldsymbol{\beta}) = \sum_{h=1}^{H} \left\{ y_h^f \ln[G(\mathbf{x}\boldsymbol{\beta})] + (1 - y_h^f) \ln[1 - G(\mathbf{x}\boldsymbol{\beta})] \right\}$$

for sample size H, which is well defined for $0 < G(z) < 1$. This log-likelihood function is identical to that used in standard ML estimation of binary response index models except that y_h^f is continuous over the unit interval. Because equation (4) is a member of the linear exponential family of distributions, the QML estimator (QMLE) $\hat{\boldsymbol{\beta}}$ is consistent for $\boldsymbol{\beta}$ when (3) holds regardless of the true conditional distribution of y_h^f (Gourieroux, Monfort and Trognon 1984). This is a desirable characteristic of QMLEs in general because y_h^f could be discrete, continuous, or some combination of the two over its range (Papke and Wooldridge 1996).

[4] Papke and Wooldridge (1996) and Mullahy (1998 and 2005) discuss in detail competing models for fractional dependent variables and we do not consider this material in any detail here except to note some of the general strengths of fractional logit over competing models after introducing the model briefly.

Fractional logit regression improves upon alternative econometric methods for fractional response variables by ensuring that the conditional expected outcome lies in the unit interval, handling observations at the boundaries of the unit interval without "arbitrary adjustment" (which is required for methods like OLS using the log-odds transformation as discussed in Wooldridge (2002, p.662)), and directly estimating the desired fractional response.

Estimation of Disease Management Adoption Decision

Our second research objective is to shed some light on the decision to adopt or not adopt disease reducing practices identified by the kinds of analyses discussed in the previous section. In this way, the estimation of the marginal effect on herd prevalence from adopting an individual practice is the first of two stages in an econometric procedure to investigate what influences private herd manager decisions to adopt particular practices. Because an adoption decision is inherently binary, we consider the choice of each practice within a binary response modeling framework where the binary dependent variable of interest is $x_{kh} \in \{0,1\}$ where practice k is identified as having a significant negative association with herd prevalence in the first stage and h indexes private herds. The binary response model then is given by

$$(5) \qquad E(x_{kh} \mid \mathbf{z}) = P(x_{kh} = 1 \mid \mathbf{z}) = G(\mathbf{z}\boldsymbol{\beta}),$$

where $\mathbf{z}$ is a vector of demographic variables that control for heterogeneity across herds and, consistent with the conceptual framework of McInerney, Howe and Schepers (1992), includes variables for the cost of output losses from avoidable infection and the cost of undertaking practice k for the herd. The cost of output losses is a function of the

estimated marginal effect on within herd prevalence of adopting practice x_{kh} from the first stage, and the control expenditure is the estimated cost of adopting practice x_{kh}. More specifically, we hypothesize that when the cost of production losses from infection associated with the marginal change in prevalence attributed to the adoption of practice k are considered as an explanatory variable, the estimated coefficient will be positive in sign while a negative relationship is expected between adoption and the control cost variable in the second stage estimation.

Livestock managers may very well adopt biosecurity and disease management practices that are not found to have a significant association with any individual disease in a cross-sectional data set. Thus far we have only considered adoption of those practices identified as having such an association in the first stage estimation because without this information we cannot estimate the cost of infection associated with an individual practice. While this information is required in order to control for the cost of output losses avoided when investigating the determinants of adoption for individual practices, herd demographic variables and the cost of control expenditures are still logical explanatory variables that may be available to the researcher. We now provide an application of the proposed two-stage econometric procedure using a cross-sectional dataset with herd-level observations to compare the use of the fractional logit method and standard logistic regression in the first stage and examine the findings with respect to adoption behavior in the second stage.

Implementing the Two-Stage Estimation Procedure

Data for our empirical application come from the 1996 National Animal Health Monitoring System (NAHMS) survey of dairy cattle conducted by the U.S. Department of Agriculture's National Agricultural Statistics Service and Animal and Plant Health Inspection Service. Among the objectives that motivate the NAHMS survey is the estimation of prevalence for a given disease and species at the level of the national herd. Survey data include extensive farm-level behavioral information such as animal inventory and operational characteristics, health management and biosecurity practices, feeding and manure management practices, and livestock morbidity, mortality and culling details. The details of the NAHMS survey design are enumerated elsewhere (Ott, Johnson and Wells 2003). Statistical analysis must take the survey's complex random stratified sampling procedure into account for correct statistical inference when working with NAHMS data (Dargatz and Hill 1996).[5] We account for survey design effects throughout the statistical analysis that follows.

To demonstrate our proposed two-stage estimation procedure we focus on the production disease bovine leukosis virus, also referred to as bovine leukemia or enzootic bovine leucosis, which has previously been studied both in veterinary epidemiology (DiGiacomo, Darlington and Evermann 1985; DiGiacomo et al. 1986; Heald et al. 1992; Rhodes et al. 2003) and economic decision making studies (Chi et al. 2002a; Chi et al. 2002b; Ott, Johnson and Wells 2003; Pelzer 1997; Rhodes, Pelzer and Johnson 2003). Bovine leukosis virus (BLV) is a retrovirus that primarily affects lymphoid tissue of beef and dairy cattle and causes malignant lymphoma and lymphosarcoma (LS), although

[5] References on the statistical issues associated with complex survey designs are Lee and Forthofer (2006) and Lohr (1999).

leukemia is not a common finding, occurring in only 2-5% of BLV infected cows (Kirk 2000). BLV is horizontally transferred within blood lymphocytes, but it is uncertain whether or not it is transmitted vertically *in utero*. Economic losses to dairy farmers associated with BLV result from reduced milk production, increased replacement costs, and increased veterinary costs (Pelzer 1997). Because it is transmissible, the only way to eliminate losses from a herd is to cull all infected animals and routinely test new animals introduced to the herd to ensure the farm remains BLV free. Pelzer (1997) and Rhodes, et al. (2003) have pointed out the important difference between the economic effect of clinical LS and subclinical level infection (BLV seropositive status). Our data examine BLV seropositive animals (those found to have antibodies to BLV in their blood), for which one estimate found that "a basic BLV control program may be economically beneficial in herds in which the prevalence of BLV infection is (greater than or equal to) 12.5%" (Rhodes, Pelzer and Johnson 2003).

We proceed by demonstrating how our two-stage econometric estimation procedure can be used to estimate the effect of individual management practices on livestock disease prevalence levels for a herd and how this information can be used with the estimated cost of illness from production losses to evaluate how the economic damages from infection associated with individual practices influence practice adoption behavior. We utilized the Stata® statistical package (Stata Version 8.0 2003) to implement the first stage using standard logistic regression and fractional logistic regression. The fractional logit method (Papke and Wooldridge 1996) was implemented using the generalized linear model command which allows the analyst to directly predict herd prevalence and account for survey design when calculating standard errors.

First Stage: Disease Control Function Estimation

The first stage in the estimation procedure identified those management practice variables that have a statistically significant effect on the herd-level dependent variable. The 1996 NAHMS dairy dataset includes many more variables than those discussed in this analysis. Because we were interested in a comparison of the fractional response model and the standard binary response model used in epidemiological studies to identify risk factors for infection, we followed a backwards stepwise procedure that is common in the veterinary literature (e.g., Heald, et al., 1992; Johnson-Ifearulundu and Kaneene, 1998) to eliminate those management variables from our first stage regression that were insignificant before the final estimation of the impact of individual risk factors for infection on the herd-level disease outcome. Only the management variables that survived stepwise elimination and regional dummies intended to capture spatial heterogeneity were included in the first stage estimation results reported here. The second stage estimation of the adoption equations include a cost variable which is described in detail below and uses the results from the first stage estimation along with farm demographic variables in the NAHMS data which are taken to be measures of productivity. Continuous and binary variables in the 1996 NAHMS dairy survey that are used in our two-stage estimation are reported with summary statistics in Table 1.1. The summary statistics indicate that mean individual within herd prevalence was 40% and that 88% (not reported in Table 1.1) of all dairy herds had at least one seropositive cow. There was a strong statistically significant difference between positive and negative herds

only for the variables herd size, herd prevalence, and both "safe" and "unsafe" dehorning methods (also not reported).

First stage estimation results from both the fractional response and the binary response models are reported in Table 1.2 for comparison of the information provided by the two models. Recall that the key difference between the two models is the nature of the dependent variable. The fractional logit (flogit) model uses the herd prevalence $y_h^f \in [0,1]$ and directly estimates the fractional response associated with each explanatory variable. The binary dependent variable $y_h \in \{0,1\}$ in the standard logit model uses a latent variable approach where $y_h = 1[y_h^* = \mathbf{x}\boldsymbol{\beta} + \varepsilon > 0]$ and $1[\cdot]$ is an indicator function that equals one when its argument is true; the dependent variable takes on a value of 1 for any herd prevalence greater than zero, $y_h = 1[y_h^* > 0] = 1$, and 0 otherwise, $y_h = 1[y_h^* = 0] = 0$. We discuss the two models in turn because they are fundamentally different and to contrast the nature of the information provided by the models to decision makers.

The only regional dummy found to be significant in the flogit model was the southeast indicator and it was associated with 16.8% higher BLV prevalence relative to the Midwest (the control region). The herd size variable indicates that an additional hundred animals in the herd increases the expected BLV prevalence by 0.6%, while an additional thousand pounds of milk produced per animal per year on average is associated with a 1% reduction in expected prevalence. Binary management practices

that survived backward stepwise elimination prior to the first stage estimation were generally found to be significant in the flogit model.

Dehorning practices were combined into three categories based on practices suggested to minimize the spread of infection ("safe"), practices most likely to contribute to the spread of infection ("unsafe"), and not dehorning in order to better facilitate the comparison of recommended and discouraged practices in subsequent regression analysis. The use of caustic paste (to prevent the growth of horns before development) or electric dehorning was significant and found to be associated with an 8.6% reduction in predicted BLV prevalence compared to the discouraged practices of either saw or gouge dehorning. Not dehorning dairy cows was not found to affect herd BLV prevalence in a significantly different way than the use of unsafe dehorning methods. This result is consistent with veterinary studies that have identified electric dehorning as reducing the likelihood of BLV infection (DiGiacomo, Darlington and Evermann 1985; DiGiacomo, et al. 1986). Saw and gouge dehorning, though not recommended practices, were observed on 426 farms in our sample compared with 524 farms adopting a safe dehorning method.

We identified farms using "clean injection methods" as those that either use a new needle for every animal (single-use) or sterilize needles after each use in the NAHMS data. For the same reasons that "safe" dehorning practices are recommended, using such injection practices is generally recommended and may be of particular importance for BLV which is transmitted via blood lymphocytes. The flogit model finds a strong significant relationship between clean injection practices and BLV prevalence for both heifers (less than 24 months, never bred) and cows, but with negative and positive signs, respectively. One possible explanation of the difference in sign is the fact that dairy

cattle normally receive vaccinations as a preventive health measure at a young age (when designated as heifers in the survey), but injections administered later in their life (when designated a cow in the survey) are less common and may be administered because of other illness or because the cow was introduced to the herd from an off-farm source. This may be a plausible explanation for the finding that clean injection practices used on cows were associated with 16.4% higher within herd prevalence and the adoption of clean injection practices for heifers was associated with a 13% lower estimated prevalence than on farms where these practices were not adopted.

The predicted prevalence from the flogit model when all variables were evaluated at their means was 39.8% (compared to the observed mean prevalence of 40% in Table 1.1) and when both BLV reducing practices were undertaken the predicted mean herd prevalence was 23.9% (all other variables evaluated at their means) compared with 44.7% when BLV reducing practices were not undertaken.

The first stage binary response model, while modeling a related outcome, is fundamentally different from the flogit model because of the nature of the dependent variable already discussed. This said, the comparison of estimation results from the two models (Table 1.2) in isolation of our underlying objective of informing economic decision making does not make sense because these are not "competing models" in the usual sense encountered in applied econometric research. Rather, we compare the two models in order to point out the difference in the information provided to decision makers.

The southeast regional dummy is found to be significantly associated with 8.5% higher probability that one or more animals in the herd are infected in the herd-level

binary response model. The odds-ratio associated with this marginal effect indicates that herds from the southeast are nearly seven times more likely to be BLV seropositive than midwest herds. This finding demonstrates one point made above in the discussion about odds-ratios for binary herd-level disease data. Viewing the odds-ratio alone finds that herds in the southeast are multiple times more likely to be infected than those in the midwest, but the associated marginal effect on binary herd infection status indicates that this only translates into an eight point difference in the predicted probability that a herd is seropositive.

As in the flogit model, herd size and average annual milk production are found to be significant in the logit model. One hundred additional cows or an additional thousand pounds of average annual milk production are predicted to be associated with 2.4% higher and 0.6% lower probability that a herd is infected, respectively.

The biggest difference between the information provided to decision makers by the two models comes from examining the variables for dehorning and injection practices. The binary response model found only one of the four practice dummy variables to be significant, while three were highly significant in the fractional response model. Even though the magnitude of the estimated effect on herd prevalence of injection practice variables found to be significant in the flogit model exceeds that of dehorning practice dummies, these practices are not even significant in the logit model. The odds-ratio for safe dehorning suggests that farms that adopt the practice are half as likely to be infected relative to those who adopt unsafe methods. The binary response model predicted that the probability a herd was infected when all variables were evaluated at their means was 90.6% (compared to 89.1% of sampled herds being infected,

mentioned above). When both BLV reducing practices are undertaken (all other variables evaluated at their means) the logit model predicts a probability of infection equal to 90.3% compared with 92.6% when BLV reducing practices are not undertaken. This difference in the estimated probability that one or more animals are infected conditional on adoption does not allow a farm manager to calculate the expected change in the cost of infection associated with the practice unless the baseline probability of infection for the farm is known; this is information that is considered to be rarely available to farm managers in practice.

Information of this kind provided to a decision maker is very different than the estimated change in prevalence indicated by the flogit model (-20.8% mentioned above). When the difference in disease prevalence indicated by the flogit model has profitability implications, it is helpful information to have, even in the absence of information about the baseline level of infection, in deciding which management practices to undertake. Conversely, the information about livestock management practices provided by standard logistic regression (adopting the practices identified in the first stage reduces the probability that one or more animals in the herd are infected by 2.3%) provides no such information to guide managerial decisions because the difference in probability that the herd is infected cannot be monetized into an expected cost or benefit of undertaking the practices for comparison with cost of undertaking the practices.

Another issue to consider is that if we have information about within herd prevalence and not just binary herd infection status, adopting a binary response model—even if marginal effects are reported with odds-ratios—ignores some of the information available to the researcher which is valuable for economic decision making purposes.

Because of this we only use the first stage estimation results from the fractional response model in estimating the adoption equations in stage two.

Second Stage: Estimation of Adoption Equations

In the second stage we incorporated prevalence information from the estimated flogit model into an analysis of management practice adoption. We used an empirical estimate from the veterinary literature based on the 1996 NAHMS dairy survey data to construct variables that represent the cost of infection avoided (the dollar value of output losses, not taking into account the cost of adopting the practice) by adopting BLV reducing management practices identified in the first stage fractional logit regression. Ott, Johnson and Wells (2003) estimated that a 1% increase in BLV prevalence costs $1.28 per cow/year in terms of the impact on the average value of production that results from reduced milk output, lost calves, and net replacement costs. A separate study based on experimental data from a research herd appearing in a different journal and using a different approach arrived at an identical estimate of the cost of subclinical BLV infection (Rhodes, Pelzer and Johnson, 2003). Using separate estimates of the marginal effect on herd BLV prevalence of adopting a safe dehorning method and using clean injection practices for heifers, we constructed a variable that represented the estimated cost of BLV avoided by adopting each individual practice for every farm in the sample. To create a cost variable for each BLV reducing practice, the herd-specific marginal effect from the first stage was calculated for binary management practice k according to (Wooldridge 2002, p.458-459)

$$ME_{k,h} =$$

$$(6)\quad G(\beta_0 + x_1\beta_1 + x_2\beta_2 + \ldots + x_k + \ldots + x_{K-1}\beta_{K-1} + x_K\beta_K + \varepsilon_h) -$$
$$G(\beta_0 + x_1\beta_1 + x_2\beta_2 + \ldots + x_{k-1}\beta_{k-1} + x_{k+1}\beta_{k+1} \ldots + x_{K-1}\beta_{K-1} + x_K\beta_K + \varepsilon_h)$$

and multiplied by the product of the total number of cattle on the operation and the $1.28 per cow/year/percentage point BLV prevalence estimate from the veterinary literature for each practice. Two adoption equations could then be estimated in the second stage, one for each BLV reducing practice, relating adoption of a practice to the costs avoided.

Adoption equations were estimated for each practice using standard logistic regression because of the binary nature of practice adoption (Table 1.3). The binary response adoption model takes the form $P(x_{kh} = 1 \mid \mathbf{z}) = P(x^*_{kh} > 0) = G(\mathbf{z\beta})$ and the associated latent variable and indicator function are given by $x_{kh} = 1[x^*_{kh} = \mathbf{z\beta} + u > 0]$ where $\mathbf{z}$ is a vector of explanatory variables for practice k in herd h.[6] Explanatory variables included the estimated economic damages from BLV that were avoided by adopting the practice indicated by the dependent variable, farm demographic variables for region, average annual milk production, production cost per hundred-weight, and a constant. The variable for economic damages avoided was marginally significant in the safe dehorning method adoption equation (p=0.074) and suggests that an additional hundred dollars of damage avoided increases the probability of adoption by 9.8%. This indicates that the cost of damages avoided appears to have an economically significant

[6] In addressing the adoption of biosecurity and health management practices it is possible that there are econometric problems that arise as result of the two stage estimation procedure. There is an estimated regressor in the adoption equation which is known to be a potential problem, but was unavoidable because of a lack of available information about private costs in the data. Second, there may be simultaneity between adoption and the conditional prevalence of disease we estimate due to correlation in the error structure of the two equations we estimate in sequence.

impact on practice adoption in the 1996 NAHMS dairy sample. This result is consistent with estimates from the veterinary literature that suggest mean losses from subclinical BLV infection for a 100 cow herd with 50% prevalence is in the neighborhood of $6,400 compared to the predicted mean annual cost of a basic BLV control program of $1,765 (Rhodes, Pelzer and Johnson, 2003). The insignificance of damages in the clean injection equation, however, goes against economic logic; especially when you consider that the marginal effect on within herd prevalence in the disease control function is 4.4% greater for the injection practice than for the safe dehorning method.

There are at least two potential reasons for our finding that damages do not have a statistically significant influence on adoption of a "clean" injection practice for heifers despite the incongruence with economic intuition. The first is that dairy farmers may not have had information about costs of control relative to economic losses from subclinical BLV infection. This implicitly assumes that if farmers had this information it would have influenced their actions in an economically significant way, which is intuitively appealing but remains an empirical question. The second is that, given farmers did have information about the potential losses from BLV when making an adoption decision, opportunity costs of management, labor, and capital outweighed the disease costs. This includes other practices that are perceived to yield preventive spillovers to multiple diseases.

Among the set of regional dummy variables, only the west and northeast regions were found to be more than marginally significant and are positively associated with the adoption of clean injection practices for heifers and "safe" dehorning methods,

respectively.[7] The only other variable that significantly contributed to the adoption of either BLV reducing practice was average annual milk production.

Goodness of fit measures for the two adoption equations favor the dehorning equation over the clean injection practice equation. The "safe" dehorning equation correctly predicted adoption for 62% of the sample overall compared to the best naive prediction possible (that all farms adopt) which only resulted in 53% correctly predicted overall. The heifer clean injection equation had a higher percentage of observations correctly predicted overall but does no better at predicting adoption than a researcher who naively guesses that all operations will choose not to adopt. While there was nothing particularly remarkable about the predictive power of the latter equation, the former seemed to do a pretty good job on the basis of this simple metric.

Conclusions

This paper presented a method for analysis (1) of herd-level livestock health data that provides managers with information to improve resource allocation across individual management practices and (2) the determinants of disease management practice adoption.

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[7] One explanation for the magnitude and direction associated with being from the northeast region on adoption of a safe dehorning method is the presence in New York of the only voluntary state BLV eradication program that the authors are aware of in the United States. There are 268 observations in the sample from the northeastern region states that rank among the top 20 dairy producing states (NY, VT, and PA) and 149 of these 268 observations are from New York, the only state with a publicly funded BLV eradication program. While we have no information to indicate if any of the sampled farms participated in the eradication program, at the time of the survey this program provided limited funds for serological testing, technical assistance in the form of disease eradication and control plan development for participating operations, and among the four baseline practices that all eradication program participants are expected to undertake are electric dehorning and single-use sterile, disposable needles (Brunner, et al. 1997).

We proposed a two-stage estimation procedure designed to achieve both objectives in sequence and demonstrated the proposed procedure in an empirical application.

When data are collected at the herd-level and the focus of analysis is a non-epidemic disease, the information provided to livestock managers by the fractional logit method was found to be superior to standard logistic regression commonly used in the analysis of herd-level livestock epidemiological data. Using herd-level data on bovine leukosis virus (BLV) from a cross-sectional survey of the top 20 dairy producing states, it was demonstrated that the management practices identified by the two models as having a significant negative effect on the dependent variable differ considerably and that these differences are relevant for economic decision making. The fractional logit model provides decision makers with information about the marginal effect of individual health management and biosecurity practices on the estimated prevalence of disease in their herd, while standard logistic regression provides estimates of the marginal change in the probability that one or more animals in a herd is infected when a particular practice in undertaken. There appears to be potentially valuable information contained in the fractional dependent variable herd prevalence that is ignored if such data are treated as a binary outcome in statistical analysis.

In the first stage of our estimation we consider that livestock managers must allocate resources across individual practices associated with production and find that the kind of information provided by the fractional logit model is particularly helpful. Standard logistic regression remains indispensable for analysis of highly contagious diseases, diseases with human health implications, and others that are the focus of international trade sanctions and cannot be tolerated at any level in a national herd. The

information content of standard logistic regression results for management of production diseases, however, is not viewed as being particularly helpful to economic decision makers. The use of an econometric method capable of directly estimating the fractional response of interest provides managers with information that can be used to weigh the benefits and costs of individual practices that is not provided by binary response models commonly used to analyze herd-level data in veterinary epidemiology.

In a second stage regression we use the information from the fractional logit model to estimate adoption equations for practices found to be statistically and practically significant in the first stage regression. The estimated cost of damages from BLV infection avoided by adopting the practices identified in the first stage was calculated for each farm in the sample, and was found to be insignificant in explaining heifer clean injection practice adoption and marginally significant in explaining safe dehorning practice adoption. The estimated economic damages from BLV associated with not adopting a particular management practice might be expected to be significant from the standpoint of economic intuition and we find this to be the case for one of the two BLV reducing practices identified in the first stage. This estimated effect is also economically significant for the adoption of a safe dehorning method, contributing to the probability of practice adoption in a meaningful way.

We cannot make any statements about the second of our hypotheses about the insignificance of economic damages avoided because we only consider a single disease in our empirical application. Of particular interest is the role that preventive spillovers to other diseases may play in the adoption of individual management practices. The NAHMS data (in certain years and for certain species) include serological tests for

multiple diseases and work to quantify preventive spillovers and the role of those spillovers in disease management and biosecurity practice adoption is underway (Gramig and Wolf 2007).

The fractional logit method has not previously been used to analyze herd-level livestock disease data and seems to be a promising analytical tool when compared to standard logistic regression, which has been used almost exclusively in empirical veterinary studies of livestock disease management. This econometric technique is likely to be useful to agricultural economists dealing with a variety of other fractional dependent variables.

Appendix A

Table 1.1 Weighted 1996 NAHMS Dairy Sample Statistics for Bovine Leukosis Virus (BLV)

Continuous Variable	**Mean**	**Std. Error**
Within herd BLV prevalence	0.400	0.011
Herd size (cows)	200.819	5.889
Average annual milk production (pounds/cow/year)	17488.360	137.172
Binary Variable	**Mean**[a]	**Std. Error**
Midwest region	0.612	0.004
Southeast region	0.043	0.002
Northeast region	0.261	0.004
West region	0.083	0.002
No dehorning on farm	0.012	0.003
Safe dehorning method	0.449	0.020
Clean injection of heifers(< 24 months old)	0.067	0.009
Clean injection of cows (≥ 24 months old)	0.065	0.009

Notes: The n=980 herds in the sample were drawn from the top 20 dairy producing states and represent 76,037 herds. Sample statistics calculated in Stata® fully account for complex survey design.

[a] Reported mean for binary variables is interpreted as the weighted proportion of operations in the sample

Table 1.2. Stage I Estimation Results from Fractional (flogit) and Binary (logit) Response Models

Variable	**flogit:** $E(y\|\mathbf{x}), y\in[0,1]$ Coefficient [Marginal Effect[a]]	**logit:** $E(y\|\mathbf{x}), y\in\{0,1\}$ Odds-Ratio [Marginal Effect[a]]
West	0.0405 [0.009]	0.53* [-0.067]
Southeast	0.6814** [0.168**]	6.99* [0.085***]
Northeast	0.1029 [0.024]	0.95 [-0.003]
Herd size (hundreds of cows)	0.0275*** [0.006***]	1.32*** [0.024***]
Average Annual Milk Production (thousands of pounds)	-0.042*** [-0.010***]	0.92* [-0.006*]
Safe dehorning method (caustic paste or electric dehorner)	-0.3647*** [-0.086***]	0.52** [-0.056**]
No dehorning	-0.1111 [-0.026]	0.50 [0.076]
Clean injection methods- Heifers	-0.5829*** [-0.130***]	1.42 [0.026]
Clean injection methods- Cows	0.6668*** [0.164***]	1.12 [0.009]
Intercept coefficient	0.373	3.237***
R-squared	0.0708	0.0526

Notes: n=980; Standard errors calculated using the delta method, account for complex survey design

Asterisk denotes level of statistical significance: *10%, **5%, ***≤1%

[a] Marginal effects evaluated at mean for continuous variables and 0 to 1 change for dummies

Table 1.3. Stage II Estimation of Determinants of BLV Reducing Management Practice Adoption

Variable	Safe Dehorning Method Coefficient [Marginal Effect[a]]	Clean Injection of Heifers Coefficient [Marginal Effect[a]]
Total damages from BLV Avoided if adopt practice ($100)	0.0400* [0.098*,[b]]	-0.0660 [-0.004]
West region	0.3746* [0.093*]	1.1346** [0.100**]
Southeast region	-0.2452 [-0.059]	1.14 [0.095]
Northeast region	0.5346*** [0.132***]	0.5092 [0.033]
Avg Annual Milk Production (thousands of pounds)	0.1674*** [0.041***]	0.0613 [0.004]
Cost per CWT ($ per hundred pounds milk)	0.0069 [0.001]	0.0379 [0.002]
Intercept	-3.4481***	-4.3191***
F-statistic for overall significance	10.27***	3.09***
% Yes correctly predicted	61.6	0
% No correctly predicted	62.3	100
% Overall correctly predicted	61.9	92.1

Notes: n=980; Standard errors calculated using the delta method, account for complex survey design

Asterisk denotes level of statistical significance: *10%, **5%, ***<1%

[a] Marginal effects evaluated at mean for continuous variables and 0 to 1 change for dummies

[b] p-value = 0.074

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Essay Two

LIVESTOCK DISEASE INDEMNITY DESIGN WHEN MORAL HAZARD IS FOLLOWED BY ADVERSE SELECTION

Introduction

The outbreak of disease in domestic livestock herds is an economic and potential human health risk that involves both the government and individual livestock producers. Economic justifications for public intervention in disease control include externalities, public good aspects, coordination failures, information failures, and income distribution considerations (Ramsay, Philip and Riethmuller). The potential to pose a large economic cost depends on many factors including trade laws and level of infectiousness. Public policies range from bounties for infected livestock to required herd depopulation and farm decontamination. The United States Department of Agriculture's Animal and Plant Health Inspection Service (APHIS) provides inspection and quarantine services to prevent the introduction of disease across national borders and also coordinates response to disease outbreaks from within the country. Border measures that protect against incursion of disease are provided to protect the safety of the American food production system and to prevent infection of the domestic livestock industry.

Diseases that are highly contagious or have human health implications are often the target of government eradication programs. When livestock is taken by the government for public health or economic reasons, the Fifth Amendment of the US Constitution specifies that private property taken for public use must receive just compensation. This compensation takes the form of indemnity payments. The current federal compensation level is defined by the Animal Health Protection Act, Subtitle E of

the Farm Security and Rural Investment Act of 2002 which states that compensation shall be based on the fair market value, as determined by the Secretary of Agriculture, adjusted for any other compensation received for that event (i.e., disaster payments or perhaps even private market insurance). States may also offer compensation in the form of indemnities.

This paper focuses on the structure of indemnity payments, which are currently the primary form of compensation by the government, as the key mechanism to provide incentives to farmers in order to achieve policy objectives. Existing indemnity payments represent an implicit insurance policy for all livestock producers with respect to the diseases where they are applicable, but are really more akin to ad hoc disaster payments commonly made to farmers because of the lack of risk classification and underwriting involved. Indeed, these payments do not have the desirable risk pooling properties associated with insurance; there are no premiums based on the risk represented by an insured as part of a portfolio of policies and all taxpayers fund the indemnities. Government-provided indemnities should be designed with careful attention to the private incentives they create and the public objectives of livestock disease risk management. It is clear that providing adequate incentives to biosecure *and* report has been an issue of concern within public agencies responsible for livestock disease outbreak response (Ott 2006). It is our intent to address this problem in a rigorous, systematic fashion to ensure incentive compatibility between the government and private decision makers.

Previous economic research dealing with livestock disease (e.g., Kuchler and Hamm; Mahul and Gohin; Bicknell, Wilen and Howitt; Horan and Wolf; Hennessy) has

also ignored incentive compatibility, at least in the presence of asymmetric information.[8] The current paper uses a principal-agent model to examine incentive compatibility in the presence of information asymmetry between the government and individual farmers.[9] The nature of the information asymmetry is depicted in Figure 2.1. Individuals have private information about preventive biosecurity measures they adopt on their farms prior to outbreak (*ex ante*), and following outbreak (*ex post*) they possess private information about the disease status of their herd. We investigate the role of incentives in individual producer behavior that influences the occurrence, duration and magnitude of a disease epidemic. Our focus is on farm level biosecurity choices and reporting of disease status as the two primary areas of concern for the government. The government can set indemnity rules in an effort to influence private incentives in order to achieve the stated government objective that all farms invest in biosecurity and all infected farms report infection (Ott 2006).

After setting up our model of farmer decision-making in the following two sections, we first address the *ex post* problem because once uncertainty about the occurrence of an outbreak is resolved, regardless of the *ex ante* actions taken by producers, reporting of disease status (as opposed to discovery by another party through transmission, slaughter, or testing) is required for timely government response to limit the

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[8] Prior economic research in this area has examined producer response to prices in conjunction with a government bounty program for scrapies in the U.S. (Kuchler and Hamm), optimal actions to contain Foot and Mouth Disease outbreak in France (Mahul and Gohin), the effect of government programs to eradicate disease on prevalence level and private control efforts in New Zealand (Bicknell, Wilen and Howitt), the dynamics of optimally controlling infection from a disease which is transmitted between wildlife and livestock (Horan and Wolf), and behavioral incentives when there is endemic disease in a decentralized setting (Hennessy 2007).

[9] A less formal discussion of these issues may be found in Gramig et al. (2006).

spread of infectious disease and eradicate it. The length of time between outbreak and discovery is very important in determining the cost, duration, and severity of epidemic (UN-FAO). For these reasons, an incentive structure that results in reporting of any infection is of great interest for a social planner. Second, we investigate the design of *ex ante* incentives for biosecurity investment together with *ex post* truthful disclosure. The characteristics of an incentive compatibile indemnity rule are derived for the case of a risk averse agent (the farmer) and a risk neutral principal (government agency). A comparison of the relative size of optimal indemnities and constrained efficient ones that are second best under information asymmetry follows. Implications of the theoretical model results for public policy and market insurance design are considered and conclusions are offered.

A Dynamic Model of On-Farm Decision Making

We develop a dynamic capital valuation model of the livestock enterprise fashioned after that of Hennessy (2007), who adapts the efficiency wage model of Shapiro and Stiglitz (1984) to the problem of livestock disease management. Our farmer decision model departs from Hennessy (2007) by (i) introducing risk aversion on the part of a single farmer (only briefly addressed by Shapiro and Stiglitz), and (ii) considering biosecurity *and* disease reporting decisions. A diagram of the decision-making process described below is provided in Figure 2.2. The farmer is risk averse with an instantaneous utility function $U(\omega)$, where $U'>0$, $U''<0$. Wealth, ω, is contingent on the disease state and farmer choices in our model.

The farmer will be in one of two disease states at any given point in time: susceptible (non-infected) or infected. In the susceptible state ($\theta=0$, where $\theta \in [0,1]$ is a random variable denoting the within-herd disease prevalence rate) farmers must choose their biosecurity effort investment level, b. Biosecurity reduces the probability of transitioning to the infected state, $P_{SI}(b)$, such that $\partial P_{SI}(b)/\partial b \leq 0$. Biosecurity also reduces the expected magnitude of a disease outbreak, should one occur. The conditional probability density function of θ is denoted $g(\theta|b)$, such that $G(\theta|b)$ is the twice continuously differentiable conditional cumulative distribution function with $\partial G(\theta|b)/\partial b \geq 0 \ \forall \ b$. The conditions imposed on the distribution of prevalence mean that G satisfies first-order stochastic dominance in the sense that the cumulative density for a given level of infection is non-decreasing (the desirable outcome) in biosecurity.[10]

The farmer has a baseline profit flow when disease-free, gross of any biosecurity investment, denoted by π_0. An investment in biosecurity involves both initial capital investments and the variable cost of ongoing management. In our model such investments are, for the sake of tractability, treated as having a single variable cost w per unit time and are incurred only in the susceptible state because once infected there is no incentive to invest in biosecurity. The utility of wealth in the susceptible state can therefore be expressed as

(1) $$U_S = U(\pi_0 - bw).$$

[10] Because disease is a "bad", higher outcomes of the random variable are *less* desirable and so what we normally refer to as the "dominated" distribution is relatively *more* attractive for our application. For $b_0 < b_1$, $G(\theta|b_1) \geq G(\theta|b_0)$ for all b, where the inequality is strict for at least one value of b.

In the infected state, the farmer must decide whether or not to report infection. Disease reporting is modeled as a mixed strategy, denoted *r*. Reporting results in government testing, verification of infection, and culling of infected animals to eradicate the disease. The farmer is compensated for any culled animals with a government transfer denoted by $\tau(\theta)$. Culling results in two types of losses for the farmer—a loss of asset value $\lambda(\theta)$ associated with the livestock itself and consequential losses from business interruption $\chi(\theta)$. Business interruption losses may vary widely depending on the characteristics of the individual operation affected and possibly disease characteristics. For instance, the presence or absence of breeding stock or having high fixed costs associated with a specific capital asset (e.g., a dairy or egg laying operation) could contribute to the magnitude of business interruption losses. Reporting disease ensures that infected farms return to the susceptible state such that the transition probability from infected to susceptible when you report is equal to one. The farmer's instantaneous utility when he/she reports is given by $U_I^R(\pi_0 - \lambda(\theta) - \chi(\theta) + \tau(\theta))$.

Not reporting disease means that the farmer's instantaneous expected utility is determined by two distinct probabilistic outcomes. Government disease surveillance activities detect non-reported infection with exogenous probability *q* and fail to detect non-reported infection with probability (1-*q*).[11] Detection leads to government culling of infected animals, compensation by government transfer $\tau(\theta)$ (as occurs under reporting), and the farmer is fined an amount *f* for not reporting. In contrast, private culling of infecteds when non-reported disease is not detected by the government means that

[11] Government disease surveillance is modeled as being exogenous to reflect the fact that ongoing surveillance activities prior to reporting or discovery of outbreak are conducted based on prior budgetary commitments independent of a given disease outbreak

government compensation is not forthcoming and culled animals are assumed to be sold at salvage value $\sigma(\theta)$. Whether infection is discovered by the government or not, all farms that do not report incur asset value losses and associated consequential losses (as occurs under reporting).

Government culling is followed by a certain transition to the susceptible state, as is the case when farms report disease. However, private culling may not be as effective and only results in transition to the susceptible state with probability $h<1$. This means that the expected instantaneous utility from not reporting is given by $qU_I^C(\pi_0 - \lambda(\theta) - \chi(\theta) + \tau(\theta) - f) + (1-q)U_I^{NC}(\pi_0 - \lambda(\theta) - \chi(\theta) + \sigma(\theta))$. The overall expected utility of wealth in the infected state, conditional on the current level of infection, can therefore be expressed as

$$U_I = rU_I^R(\pi_0 - \lambda(\theta) - \chi(\theta) + \tau(\theta)) + (1-r)\begin{pmatrix} qU_I^C(\pi_0 - \lambda(\theta) - \chi(\theta) + \tau(\theta) - f) + \\ (1-q)U_I^{NC}(\pi_0 - \lambda(\theta) - \chi(\theta) + \sigma(\theta)) \end{pmatrix} \cdot \quad (2)$$

Equations (1) and (2) are individual components of a farm's inter-temporal decision-making process. In the next section we incorporate state transition probabilities to derive the system of equations that represents the full scope of the farmer's dynamic problem.

Fundamental Asset Equations

Define V_S to be the expected lifetime utility of the decision maker in the susceptible state. Using the continuous time discount rate ρ, we can define ρV_S to be the "time value" of the livestock asset when susceptible (Hennessy, p.702). Similarly, let V_I be the expected

lifetime utility in the infected state. This notation gives rise to the "fundamental asset equations" (Shapiro and Stiglitz, p.436) [12,13]

$$(3) \qquad \rho V_S = U_S(b) + P_{SI}(b)(E_\theta[V_I] - V_S),$$

$$(4) \qquad \rho V_I = U_I(r, \tau(\theta), f, \theta) + P_{IS}(r)(V_S - V_I),$$

where

$U_I(r, \tau(\theta), f, \theta) = rU_I^R(\lambda, \chi, \tau(\theta), \theta) + (1-r)[qU_I^C(\lambda, \chi, \tau(\theta), f, \theta) + (1-q)U_I^{NC}(\lambda, \chi, \sigma(\theta), \theta)]$ and $P_{IS}(r) = r + (1-r)[q + h(1-q)]$. The time value of the susceptible livestock asset in (3) equals the sum of the instantaneous utility in the susceptible state, $U_S(b)$, and the expected capital loss if the disease state changes from susceptible to infected, $P_{SI}(b)(E_\theta[V_I] - V_S)$. Because the post-transition level of infection is unknown to farm managers in the susceptible state, the expected capital loss associated with this state transition is a function of the expectation of the lifetime stream of utility in the infected state with respect to the level of disease prevalence, θ.

Similarly, the time value of the infected livestock asset in (4) equals the sum of the expected instantaneous utility in the infected state, $U_I(r, \tau(\theta), f, \theta)$, and the expected capital gain from transitioning to the susceptible state $P_{IS}(r)(V_S - V_I)$. The expectations

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[12] Equations (3) and (4) below are provided in explicit form in Appendix B.1 for the interested reader.

[13] Equations (3) and (4) are derived following Shapiro and Stiglitz (1984, p.436). Focusing on V_S, we examine expected lifetime utility when decisions are made over small intervals of size [0,t]: (3a) $V_S = U_S(b)t + (1-\rho t)[P_{SI}(b)tE_\theta[V_I] + (1 - P_{SI}(b)t)V_S]$. Note that $(1-\rho t) \approx e^{-\rho t}$. Equation (3) is obtained by solving (3a) for V_S and evaluating it as $t \to 0$. Equation (4) is derived similarly. An implicit assumption in this formulation is that farm businesses are "infinitely lived entities", as is assumed in Hennessy (2007, p.702) and Shapiro and Stiglitz (1984, p.435).

operator is not needed in (4) because the infected farmer is assumed to know his/her current level of infection.

Equations (3) and (4) may be solved as a system to get

$$(5) \qquad V_S(b, E_\theta[V_I]) = \frac{U_S(b) + P_{SI}(b)E_\theta[V_I]}{\rho + P_{SI}(b)}, \text{ and}$$

$$(6) \qquad V_I(b, r, \tau(\theta), f, \theta, E_\theta[V_I]) = \frac{U_I(r, \tau(\theta), f, \theta) + P_{IS}(r)\left[\frac{U_S(b) + P_{SI}(b)E_\theta[V_I]}{\rho + P_{SI}(b)}\right]}{\rho + P_{IS}(r)}.$$

Equation (5) shows the lifetime expected utility from being in the susceptible state to be an annuity value. If there was no chance of transitioning to the infected state (i.e., $P_{SI} = 0$), then lifetime utility when susceptible equals the annuity value U_S/ρ (i.e., U_S is received into perpetuity). When there is a chance of becoming infected (i.e., $P_{SI} > 0$), the annuity value changes in two ways: (i) a risk premium, P_{SI}, is added to the risk-free rate ρ to yield a risk-adjusted discount rate that has the effect of reducing the annuity value associated with the susceptible state to $U_S/(\rho+P_{SI})$; (ii) we must account for the expected annuity value that accrues in the infected state, $E_\theta[V_I]/(\rho+P_{SI})$, weighted by the probability of transitioning to that state.

Equation (6) illustrates a similar valuation of the expected flow from the capital asset, though conditioned on starting in the infected state and accounting for the probability of transitioning to the susceptible state. Note that the term in brackets in equation (6) represents $V_S(b, E_\theta[V_I])$, as derived in equation (5). If there were no chance

of transitioning to the susceptible state (i.e., $P_{IS} = 0$), then lifetime utility equals the annuity value U_I/ρ if U_I were received in perpetuity. As with equation (5), because there is a chance of returning to the susceptible state (i.e., $P_{IS} > 0$), the discounted stream of benefits takes this into account via the risk-adjusted discount rate ($\rho+P_{IS}$) and the transition probability-weighted annuity value that accrues in the susceptible state, $P_{IS}V_S(b,E_\theta[V_I])$.

Expressions (5) and (6) are not in reduced form since they both have expected lifetime utility from infection, $E_\theta[V_I]$, on the right hand side (RHS). To eliminate $E_\theta[V_I]$ from the RHS, take the expectation of both sides of (6) with respect to θ, isolate $E_\theta[V_I]$ and substitute the expression back into (5) and (6). The resulting implicit functions are

$$(7) \qquad V_S(b,r,f) = \frac{U_S(b)(\rho + P_{IS}(r)) + P_{SI}(b)E_\theta[U_I]}{\rho(\rho + P_{IS}(r) + P_{SI}(b))} \text{ and}$$

$$(8) \qquad V_I(b,r,\tau(\theta),f,\theta) = \frac{U_I(r,\tau(\theta),f,\theta) + P_{IS}(r)\left[\frac{U_S(b)(\rho + P_{IS}(r)) + P_{SI}(b)E_\theta[U_I]}{\rho(\rho + P_{IS}(r) + P_{SI}(b))}\right]}{\rho + P_{IS}(r)}.$$

Note that the expression in square brackets in (8) is equal to (7).

The general form of these equations is similar to the comparable equations derived in Hennessy (2007, p.702-703). However, the specific formulations are more complicated than in Hennessy (2007, p.702). The level of complexity is greater because

(i) his model includes a single binary action while we account for two separate continuous choices selected by the farmer, and (ii) we focus on the asymmetric information problem and the design of indemnification policy which involves business interruption and consequential losses along with government disease surveillance not treated in the earlier work.

The Indemnity Design Problem

We use the asset value equations to solve the overarching problem of indemnity design that achieves public objectives for private biosecurity investment and disease reporting behavior. In practice, we normally think of indemnification in connection with private insurance or government-subsidized risk management programs like crop and flood insurance. Even though indemnities are required when takings occur in response to a livestock disease outbreak, indemnities are also intended to serve a risk management function by providing incentives to report infectious disease and invest in biosecurity that prevents outbreak and may limit disease spread (Ott 2007). For these reasons, we concern ourselves with the design of an indemnification scheme that achieves government risk management objectives and address whether or not required compensation for takings are in conflict with stated risk management objectives in the presence of information asymmetry. Recall that our proposed payment structure pays one amount, $\tau(\theta)$, to an infected farmer who reports, and a lesser amount, $\tau(\theta) - f$ (which may be negative), to an infected farmer who does not report and is caught. As we describe in detail below, our proposed indemnity structure uses government transfers $\tau(\theta)$

to address *ex ante* moral hazard (biosecurity actions) and fines f to address *ex post* adverse selection (disease reporting)

The reporting problem is addressed by imposing a fine in response to a given reporting strategy, r: $f=0$ when $\theta=0$ or when $\theta>0$ and the farmer's strategy is $r=1$; $f>0$ otherwise when the farmer is caught. This structure, which involves one value of f for each action– reporting or not, given $\theta>0$ – is intended to address the adverse selection problem. This is analogous to how adverse selection problems, commonly encountered in the literature (Rothschild and Stiglitz, 1976) and arising in practice, have been addressed. We use fines to mimic a "menu of contracts" which induces the agent to reveal private information. In the next section, we propose a method for setting the fine so that it achieves the desired reporting behavior.

The moral hazard problem is addressed by setting the transfer, $\tau(\theta)$. Here the government's transfer to the farmer is based on θ, which is observed (verified as a result of testing) after infection is discovered or reported. The transfer influences the farmer's incentives to take biosecurity actions because the likelihood of becoming infected is influenced by b. We might therefore expect a lower marginal payment for larger infection rates. This would mimic the risk sharing property of deductibles or co-pays commonly used to address moral hazard problems in the principal-agent literature (Laffont and Martimort, 2002), the crop insurance literature (Chambers, 1989), and the broader insurance literature (Arrow, 1963; Raviv, 1979).

In the presence of both adverse selection and moral hazard, some combination of the instruments used to address both types of information problems individually can be expected to induce the desired biosecurity investment and reporting behaviors from the

agent in the current application. However, to simplify the exposition, we discuss the different components of the indemnity rule ($\tau(\theta)$ and f) individually. We proceed by addressing each of the two parts of the information problem in reverse chronological order. That is, we propose a way to solve for the indemnification scheme that will (i) lead the farmer to report any infected animals to the government, thereby revealing his/her hidden information, and (ii) create incentives for positive investment in biosecurity, thereby solving the hidden action problem.

The Adverse Selection Problem—Reporting

Reporting is assumed to be socially desirable (all other things equal) because early detection of infection limits the duration of a disease outbreak event and has been found to be the most important factor in minimizing total economic damages from a livestock disease epidemic (UN-FAO). The government's objective is to set fines in such a way that reporting of suspected disease always occurs. The farmer is the agent in our Principal-Agent framework and we assume he/she chooses a reporting strategy, $r \in [0,1]$, to maximize his/her discounted utility stream in the infected state, given by (8). This means that the marginal incentive to report (positive, negative or zero) is given by the sign of $\partial V_I(b,r,\tau(\theta),f,\theta)/\partial r$ and the Kuhn-Tucker conditions imply the optimal private reporting strategy.

The principal wants to set the fine so that the agent always finds reporting to be privately optimal. The difficulty is that the marginal incentives to report will differ depending on both b and θ, each of which are unobservable to the principal. The principal must therefore set a single fine such that the agent finds it optimal to report regardless of the values of b and θ. Specifically, the fine is set so that reporting occurs

even when the marginal incentives to report are at their minimum value, given by the expression

$$(9) \qquad \Lambda(f) = \min_{\forall b,\theta}\left[\lim_{r\to 1}\left(\frac{\partial V_I(b,r,\tau(\theta),f,\theta)}{\partial r}\right)\right].$$

Choosing f such that $\Lambda(f)\geq 0$ for all values of b and θ ensures that the farmer always has a non-negative marginal incentive to report. That is, as long as the agent, when operating at the margin, is indifferent between increasing and decreasing his/her reporting strategy, then the farmer will have a positive incentive to report for any value of $\theta>0$. This concept is depicted graphically in Figure 2.3.[14] Here b is taken as given and the dashed curve indicates that the farmer will not have a positive incentive to report disease for all levels of θ when $f=0$. We denote by f^* the fixed fine that achieves $\Lambda(f^*)=0$ and ensures a non-negative marginal incentive to report over the range of θ.

We have introduced fines because prompt reporting yields social benefits and is outside the scope of constitutionally required compensation for takings. We now turn to the design of government transfers to indemnify farmers whose animals are culled by the government, in order to provide incentives to private decision makers to make investments in biosecurity.

The Moral Hazard Problem—Biosecurity Investment

Given that f^* ensures reporting, we now turn to the government's problem of designing transfer payments, $\tau(\theta)$, that provide incentives for biosecurity investment. As

[14] Figure 2.3 does not reflect the shape of any particular functional form for V_I, rather it is provided to shore up the intuition associated with the proposed method for setting fines so that reporting occurs.

indicated above in relation to deductibles or co-pays used to address moral hazard problems, some amount of risk sharing between the government and the farmer will be necessary to solve the hidden action problem. This can be seen very easily by a quick comparison of the instantaneous utility of wealth in the susceptible state and in the infected state (when $r = 1$) for the special case in which the farmer is fully indemnified against all losses: $\tau(\theta) = \lambda(\theta) + \chi(\theta)$. In this case, utility when infected reduces to $U(\pi_0)$ which is not dependent on biosecurity and is strictly greater than utility when susceptible if there is *any* positive investment in biosecurity $U(\pi_0 - bw)$. In this situation, it is not clear why anyone would biosecure and it suggests that farmers will need to bear some share of the risk of disease related losses in order for there to be an incentive to invest in biosecurity. The question we turn to now is how the government should structure indemnities to facilitate risk sharing in a constrained-efficient manner.

Assume the government takes into account the private net benefits of livestock production, $V_S(b,1,f^*)$, and the expected social cost of government transfers and disease surveillance, $\kappa\left(\int_0^1 \tau(\theta)g(\theta \mid b)d\theta + m(q)\right)$, where $m(q)$ represents disease surveillance costs and $\kappa>0$ represents the constant marginal cost of diverting funds to this program, which may include transactions costs (Alston and Hurd 1990),[15] The government's objective function in setting transfers to induce biosecurity effort may be written as

[15] Because ours is a model of a single agent we do not account for market or social benefits associated with private livestock production, but this model could be extended to a multi-agent setting which would realistically consider such broader social benefits or costs.

$$(9)\qquad \max_{\tau(\theta),b} \; V_S\left(b,1,f^*\right)-\kappa\left(\int_0^1 \tau(\theta)g(\theta\mid b)d\theta+m(q)\right) \text{ s.t. } b\in \arg\max_{\hat{b}} V_S\left(\hat{b},1,f^*\right),$$

where the use of fine f^* drives r to 1, the agent's unobservable choice of biosecurity effort constrains the principal, and "argmax" denotes the set of arguments that maximize the objective function that follows.

Denote $\alpha(b)=(\rho+P_{IS})/(\rho(\rho+P_{IS}+P_{SI}))$ and

$\beta(b)=P_{SI}/(\rho(\rho+P_{IS}+P_{SI}))$, which are the risk-adjusted discount factors associated with the outcomes U_S and U_I^R. Then we can write equation (7), evaluated at r=1 and f^*, as

$$(10)\qquad V_S\left(b,1,f^*\right)=U_S(b)\alpha(b)+\beta(b)\int_0^1 U_I^R(1,\tau(\theta),\theta)g(\theta\mid b)d\theta.$$

Equation (10) is the focus of farmer decision making in the susceptible state where the agent optimizes to select a biosecurity investment $\hat{b}$ which is constrained to be b, the government's desired level of investment, without loss of generality by the revelation principle (Myerson 1979; Dasgupta, Hammond and Maskin 1979). The regulator chooses $\tau(\theta)$ to maximize farmer utility while taking into account the cost of indemnities, monitoring, and response to outbreak required to implement the chosen disease risk management policy. The agent's first-order condition (FOC) with respect to b implies the optimal private choice and we substitute the agent's FOC for the constraint in equation (9), using the notation introduced in equation (10), so that (9) can be re-written as

$$\max_{\tau(\theta),b} \; U_S(b)\alpha(b)+\beta(b)\int_0^1 U_I^R(1,\tau(\theta),\theta)g(\theta\mid b)d\theta-\kappa\left(\int_0^1 \tau(\theta)g(\theta\mid b)d\theta+m(q)\right)$$

(11) s.t. $U_S'(b)\alpha(b)+U_S(b)\alpha'(b)+$

$$\int_0^1\left[\beta'(b)U_I^R(1,\tau(\theta),\theta)+\beta(b)U_I^R(1,\tau(\theta),\theta)\frac{g_b(\theta\mid b)}{g(\theta\mid b)}\right]g(\theta\mid b)d\theta=0$$

The approach of substituting the farmer's FOC in for the constraint is called the first-order approach (FOA), e.g., Spence and Zeckhauser (1971), Ross (1973), Harris and Raviv (1979), Holmstrom (1979), Mirrlees (1975), Rogerson (1985). The FOA is valid as a general solution method for (9) when the convexity of the distribution function condition (CDFC) and the monotone likelihood ratio condition (MLRC) are satisfied (Mirrlees 1975, Rogerson 1985). We assume both of these conditions are satisfied in what follows. The CDFC is satisfied by $\partial^2 G(\theta\mid b)/\partial b^2 \geq 0$. The conditional pdf of disease satisfies the MLRC if $g_b(\theta\mid b)/g(\theta\mid b)$ is non-increasing in θ (Milgrom 1981).[16] Whitt (1980) has proven that the MLRC implies FOSD, and is therefore a slightly stronger condition than FOSD.

Because it may be hard to garner intuition from $g_b(\theta\mid b)/g(\theta\mid b)$, Milgrom (1981) provides an alternative explanation of the relevance of the MLRC in terms of the government's ability to infer the agent's hidden actions from the observation of θ. He describes the MLRC in terms of a principal who has a prior over the agent's choice of b, observes the level of disease realized, and then updates her prior to calculate a posterior on the biosecurity effort choice. Denote the posterior probability distribution of b given

[16] Milgrom (1981) finds the term should be non-decreasing in θ, but in his model the action has the opposite effect on the distribution as our action b. Accordingly, the sign is reversed here.

observed outcome θ by $F(b|\theta)$. Using Milgrom's (1981) results, the nature of disease is such that the MLRC is equivalent to $\theta_0 \le \theta_1 \Rightarrow F(b \mid \theta_1) \ge F(b \mid \theta_0)\ \forall b$, where θ_0 is a more favorable signal than θ_1 that the agent exerted the desired level of biosecurity effort.[17] The importance of the assumptions about the nature of the MLRC will become clearer when the conditions for divergence from the first-best indemnity and the implications of the model for indemnity design are considered below.

Using the FOA, the Lagrangian for the government's problem is

$$\begin{aligned}\mathcal{L} = {} & U_S(b)\alpha(b) + \beta(b)\int_0^1 U_I^R(1,\tau(\theta),\theta)g(\theta \mid b)d\theta - \kappa\left(\int_0^1 \tau(\theta)g(\theta \mid b)d\theta + m(q)\right) \\ & + \mu\left(\begin{array}{l} U_S'(b)\alpha(b) + U_S(b)\alpha'(b) + \\ \int_0^1 \left[\beta'(b)U_I^R(1,\tau(\theta),\theta) + \beta(b)U_I^R(1,\tau(\theta),\theta)\frac{g_b(\theta \mid b)}{g(\theta \mid b)}\right]g(\theta \mid b)d\theta \end{array}\right)\end{aligned} \tag{12}$$

where μ is the shadow value of the constraint. The existence of the constraint, due to the farmer's freedom to make their own biosecurity decision, renders this a second-best problem. In Appendix B.2 we illustrate that μ>0 because the government would like the farmer to increase his investment in biosecurity given the optimal indemnity payment. That is, the optimal indemnity here is only second-best due to the information problem; a first-best indemnity could be used to attain greater welfare in the absence of information asymmetry (in which case μ would optimally vanish). Holmstrom (1979) also finds such a result.

[17] It should be noted that the interpretation of the MLRC in the current context of a malady is different than the examples most often found in the literature because lower realized values of the random variable prevalence represent the desirable outcome, whereas in the typical wage contract example higher values of the random variable output are desirable and signal greater effort.

Following Holmstrom (1979), pointwise optimization with respect to $\tau(\theta)$ yields the following necessary condition, which must hold for all θ

$$(13)\quad \beta(b)\frac{\partial U_I^R(1,\tau(\theta),\theta)}{\partial \tau(\theta)} - \kappa = -\mu \frac{\partial U_I^R(1,\tau(\theta),\theta)}{\partial \tau(\theta)}\left[\beta'(b) + \beta(b)\frac{g_b(\theta \mid b)}{g(\theta \mid b)}\right].$$

Condition (13) implicitly defines $\tau^{SB}(\theta)$, the second-best indemnity as a function of θ.

The following adjoint equation is also necessary

$$\frac{\partial \mathcal{L}}{\partial b} = U_S'(b)\alpha(b) + U_S(b)\alpha'(b) + -\kappa \int_0^1 \tau(\theta) g_b(\theta \mid b) d\theta$$

$$(14)\quad \int_0^1 \left[\beta'(b) U_I^R(1,\tau(\theta),\theta) + \beta(b) U_I^R(1,\tau(\theta),\theta)\frac{g_b(\theta \mid b)}{g(\theta \mid b)}\right] g(\theta \mid b) d\theta + \mu \left\{ \begin{array}{l} U_S''(b)\alpha(b) + 2U_S'(b)\alpha'(b) + U_S(b)\alpha''(b) + \\ \int_0^1 \left(U_I^R(1,\tau(\theta),\theta)[\beta''(b)g(\theta \mid b) + 2\beta'(b)g_b(\theta \mid b) + \beta(b)g_{bb}(\theta \mid b)] \right) d\theta \end{array} \right\} = 0$$

Using the agent's FOC and recognizing that the final term in (14) can be written as $\mu\left(\partial^2 V_S\left(b,1,f^*\right)/\partial b^2\right)$, condition (14) reduces to

$$(15)\quad \kappa \int_0^1 \tau(\theta) g_b(\theta \mid b) d\theta = \mu\left(\partial^2 V_S\left(b,1,f^*\right)/\partial b^2\right).$$

Condition (15) determines μ while the constraint in (11) determines b.

How do second-best indemnity payments compare to first-best payments? First-best payments arise when there are no constraints on the government's problem – that is, the regulator is neither constrained by the farmer's first order condition nor is truthful disclosure an issue, so that there is neither an *ex post* adverse selection nor an *ex ante* moral hazard problem. In this case, condition (13) reduces to

$$(16) \qquad \beta(b)\frac{\partial U_I^R(1,\tau(\theta),\theta)}{\partial \tau(\theta)} - \kappa = 0,$$

implicitly defining the first-best indemnity payment $\overset{*}{\tau}(\theta)$. Comparing condition (16) with condition (13), we find that the following must hold

$$(17) \qquad \begin{aligned} (i): \tau^{SB}(\theta) < \tau^*(\theta) \quad &\Leftrightarrow \quad \frac{g_b(\theta \mid b)}{g(\theta \mid b)} + \frac{\beta'(b)}{\beta(b)} < 0 \\ (ii): \tau^{SB}(\theta) > \tau^*(\theta) \quad &\Leftrightarrow \quad \frac{g_b(\theta \mid b)}{g(\theta \mid b)} + \frac{\beta'(b)}{\beta(b)} > 0. \end{aligned}$$

Condition (17) indicates that farmers receive information rents, relative to the first-best case, under the conditions defined by (17*ii*), while the government reduces payments below the first-best level under the conditions defined by (17*i*) (the conditions are described in detail below). The resulting payment level depends on the realized value of θ, which the government views as evidence of the farmer's unobservable biosecurity effort. Holmstrom (p.79) points out that the term g_b / g is simply the derivative of the maximum likelihood function ln[$g(\theta|b)$], when b is taken as an unknown variable, and suggests that g_b / g measures how strongly one is inclined to infer from θ that the agent did not undertake the assumed action. The second-best solution is dependent on the distribution of θ and its relationship to b. The deviation from perfect risk sharing implies that the farmer (agent) must carry extra responsibility for the disease outcome, as was discussed in terms of instantaneous utility of wealth above. We present the mathematical conditions that facilitate the result summarized in (17) along with a description of the intuition behind the relative magnitude of first- and second-best indemnities under the different circumstances.

It follows from the definition of $\beta(b)$ that $\beta'(b)/\beta(b) < 0$. The overall sign of $g_b(\theta \mid b)/g(\theta \mid b) + \beta'(b)/\beta(b)$ is therefore determined by both the sign and magnitude of the term g_b / g. Define the single level of infection at which $\tau^{SB}(\theta) = \tau^{*}(\theta)$ as θ^{*}, such that $g_b\left(\theta^{*} \mid b\right)/g\left(\theta^{*} \mid b\right) + \beta'(b)/\beta(b) = 0$. This means that $\tau^{SB}(\theta) < \tau^{*}(\theta)$ for $\theta > \theta^{*}$, and $\tau^{SB}(\theta) > \tau^{*}(\theta)$ for $\theta < \theta^{*}$. How small is θ^{*}? It should be small enough to infer that a sufficient investment in biosecurity has been made. In particular, θ^{*} is smaller than the value of θ, denoted $\tilde{\theta}$, at which $g_b(\tilde{\theta}) = 0$. This point in the distribution is of interest because for $\theta < \tilde{\theta}$ the marginal benefit of biosecurity, in terms of reducing the cumulative density of prevalence, is increasing.[18] A smaller θ yields even larger marginal benefits of biosecurity.

By structuring indemnities according to (17), the government provides very strong incentives for a farmer to make a significant investment in biosecurity. If the government observes $\theta > \tilde{\theta}$ it will pay farmers for culled animals at a lower rate than it would if it could observe biosecurity actions directly; this is because a relatively high level of infection suggests a small likelihood of private biosecurity effort. Even for observed levels of infection $\theta^{*} < \theta < \tilde{\theta}$, the level of effort inferred by the government is still sufficiently low that the farmer cannot extract any information rents from the government. Only if the level of observed infection falls below the critical value θ^{*} will

[18] A point which might correspond with $\tilde{\theta}$ on a hypothetical second-best indemnity schedule is depicted in Figure 4, which we discuss below.

the government be convinced that the agent has invested in biosecurity at a high enough level to give up information rents to the farmer.

The relationship between the prevalence level and size of indemnities is also of great interest. To evaluate the slope of $\tau^{SB}(\theta)$, differentiate condition (13) with respect to θ to get

$$(18) \quad \tau'(\theta) = \lambda'(\theta) + \chi'(\theta) + \left(\frac{U'}{-U''}\right) \frac{\mu \frac{\partial (g_b(\theta \mid b)/g(\theta \mid b))}{\partial \theta}}{\left(1 + \mu\left[\frac{g_b(\theta \mid b)}{g(\theta \mid b)} + \frac{\beta'(b)}{\beta(b)}\right]\right)}.$$

The first two RHS terms are positive and equal the slope of the first-best indemnity $\tau^{*}(\theta)$ (since $\mu=0$ in the first-best case). The third term arises in the second-best case, and the sign of this term depends on the value of θ. In the third RHS term, $\left(\frac{U'}{-U''}\right)>0$ for a risk averse agent, the numerator is negative by the MLRC, and the sign of the denominator is determined by the sign of $\gamma = \left[\frac{g_b(\theta \mid b)}{g(\theta \mid b)} + \frac{\beta'(b)}{\beta(b)}\right]$ and the relative magnitude of terms, which for a given b depends on θ.

Several different possibilities for the shape of $\tau^{SB}(\theta)$ relative to that of $\tau^{*}(\theta)$ are depicted in Figure 2.4. For very low levels of θ, $\tau^{SB}(\theta)>\tau^{*}(\theta)$. The second-best indemnity may be initially increasing or decreasing based on the sign of equation (18). This is because $\gamma>0$ for low values of θ, making the third RHS term in (18) negative and the sign of $\tau'(\theta)$ ambiguous. The negative third term means the slope of $\tau^{SB}(\theta)$ is initially less than the slope of $\tau^{*}(\theta)$, so that these curves eventually intersect and become

equal at θ^*. When $\theta > \theta^*$, then $\gamma < 0$ and the denominator of the third RHS term is of ambiguous sign. Denote $\hat{\theta}$ (with $\hat{\theta} > \theta^*$) to be the value of θ such that the denominator of the third RHS term vanishes; the slope of the second-best indemnity goes to $-\infty$ and there is a discontinuity in the graph of $\tau^{SB}(\theta)$. This means the slope of $\tau^{SB}(\theta)$ becomes negative prior to $\hat{\theta}$. If $\hat{\theta} < 1$, this suggests that for any observed prevalence $\theta^* < \theta < \hat{\theta}$, the second-best indemnity should be paid at a level below the first-best level to maintain the appropriate incentives to invest in b.[19]

While it is mathematically possible for the slope of the second best indemnity function to be positive and/or negative over the range of θ, an indemnity schedule that is monotonically decreasing in the prevalence level like $\tau_1^{SB}(\theta)$ would provide the strongest incentives for biosecurity.[20] In such cases the information rents paid to the

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[19] It should be noted that for $\theta > \hat{\theta}$ the slope of the second-best indemnity is expected to be $+\infty$ as you approach $\hat{\theta}$ from the right. This suggests that it is possible that the first- and second-best indemnities cross again at another point $\theta > \hat{\theta}$, but it does not seem likely that an information reward would be optimal at high levels of θ. Moreover, whether or not $\hat{\theta}$ falls within the unit interval is unknown.

[20] For $\theta < \hat{\theta}$, the mathematical condition required for a monotonically decreasing second-best indemnity schedule like the one depicted by $\tau_1^{SB}(\theta)$ in Figure 4 is $\lambda'(\theta) + \chi'(\theta) < \left| \left(\frac{U'}{-U''} \right) \frac{\mu \frac{\partial (g_b(\theta \mid b)/g(\theta \mid b))}{\partial \theta}}{(1+\mu\gamma)} \right|$. This condition may be reasonable in practice because $\lambda'(\theta) + \chi'(\theta)$ is likely a very small positive quantity at lower values of θ. Alternatively, the condition that gives rise to the increasing segments of τ_2^{SB} and τ_3^{SB}

farmer would be strictly decreasing with prevalence up until $\theta = \theta^*$. Beyond this point, the reduction in payments relative to the first-best case would be increasing. Biosecurity incentives would seem to be weaker under the curves labeled τ_2^{SB} and τ_3^{SB}, which seem contrary to the government's objective.

The extent to which observed prevalence is a good signal of actual preventive bio-security effort is clearly important for implementing the indemnity schedule, $\tau^{SB}(\theta)$. It is conceivable that for diseases that are extremely contagious (i.e., Foot and Mouth Disease, Highly Pathogenic Avian Influenza, Exotic Newcastle's Disease, or Classical Swine Fever) an individual's herd could become infected regardless of the extent of biosecurity measures taken *ex ante*. For this reason, a "one size fits all" policy that only pays indemnities on the basis of observed prevalence levels is likely to be problematic in practice.

This does not preclude a disease-specific indemnification rule which would pay farmers $\tau^{SB}(\theta)$ for all but the most infectious diseases and pay them according to $\tau^*(\theta)$ during outbreaks of highly contagious diseases (where observed prevalence is not a good signal of effort). Indemnifying farmers in this disease-specific manner does not diminish reporting incentives because of the use of f^*; also, if low prevalence levels are verified when responding to an outbreak involving such pernicious diseases, this could be treated

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over the relevant range of θ is $\lambda'(\theta)+\chi'(\theta) > \left|\left(\frac{U'}{-U''}\right)\frac{\mu\frac{\partial(g_b(\theta\mid b)/g(\theta\mid b))}{\partial\theta}}{(1+\mu\gamma)}\right|$ for that range of θ.

as an even stronger signal that the farmer made a significant investment in biosecurity and such behavior should be rewarded by paying $\tau^{SB}(\theta) > \tau^{*}(\theta)$. The World Organization for Animal Health (Office International des Épizooties or OIE) maintains a list of diseases that must be reported to the international community. The OIE's list of reportable diseases (formerly called "List A" diseases) could serve to determine which diseases should be associated with such a disease-specific indemnification rule—that is, diseases on the list are compensated on the basis of $\tau^{*}(\theta)$ (with information rents if low prevalence is verified) and all other disease losses are indemnified according to $\tau^{SB}(\theta)$. An indemnification rule similar to that implemented in Belgium and the Netherlands seems to match the suggested indemnity from our model (Horst, deVos, Tomassen, and Stelwagen). This indemnification rule is briefly discussed below.

Implications for Public Policy and Market Insurance Design

Our model uses two distinct mechanisms to provide incentives for biosecurity and truthful disclosure: (i) indemnities to achieve desired levels of biosecurity, and (ii) optimal fines that induce disclosure of disease status (alternatively, these mechanisms can be viewed as a differential indemnity schedule based on whether an infected farmer reports or not). By using two distinct policy instruments, individually designed with each information problem in mind, it is possible to create clear incentives for farmers to behave in a manner that is consistent with government risk management objectives.

Status quo indemnification for livestock disease losses by USDA has paid producers on the basis of "compensation value" equal to "fair market value assuming disease-free status" (Ott 2006, p.72). This amount is necessarily greater than the true

market value of diseased animals culled by the government and is intended to create incentives for reporting. The government has also recognized that unless farmers face some uncompensated losses as a result of outbreak they cannot be expected to take preventive biosecurity measures and thus does not compensate farmers for consequential losses when issuing indemnities. Animal health authorities have relied on a single mechanism—indemnities—to facilitate both *ex ante* biosecurity effort and *ex post* reporting. By using a single mechanism to induce biosecurity and reporting simultaneously, the incentives for each individual private action are not clear.

Direct comparison of the relative magnitude of *status quo* indemnities and the second-best indemnities implied by our analysis is not possible, but the major difference is the shape of the indemnity schedules implied by the different policies. *Status quo* policy suggests the indemnity schedule is strictly increasing in θ (just like the first-best indemnity, $\tau^{*}(\theta)$, depicted in Figure 2.4), while the second-best indemnity implied by condition (17) is strictly declining over a range of θ. It is not at all clear how the incentives created by the *status quo* policy facilitate the government's joint objectives. An upward sloping indemnity schedule, in the absence of any penalty for not reporting, may actually create incentives *for* infection when you consider that the *status quo* has sought to use indemnities based on the disease-free fair market value of livestock as a means of resolving the *ex post* adverse selection problem.

In an effort to induce early reporting, Belgium and the Netherlands no longer compensate producers for dead animals and only partially compensate them for diseased stock (Horst, deVos, Tomassen, and Stelwagen). This approach shares some important elements with our second-best indemnities. First, while there is not an explicit fine for

not reporting, there is a penalty to waiting to report since dead animals fetch no payment. This feature can help to achieve incentive compatibility with reduced or eliminated monitoring costs. Second, the partial compensation for only diseased animals shifts some of the risk to farmers, as do our payments. An indemnity plan that does not shift risk in this fashion may actually create incentives for infection, which could be one problem associated with *status quo* U.S. policy.

The discussion of incentive compatibility applies not only to public policy but also to the development of private insurance for livestock disease protection. If private coverage is available to farmers, the incentives provided by livestock insurance contracts could potentially be in competition with the objectives of policy while satisfying the individual objectives of producers (i.e., income smoothing as risk management). Careful consideration in the design of private market coverage for livestock disease losses is required in order to ensure that public policy and private risk management products are jointly incentive compatible. Also, design of public policy should take into account the role that private coverage could play in achieving public policy objectives and how government decisions may hinder or bolster private markets for insurance. If this is not the case then the constrained efficient result analyzed here will not be achievable.

Appendix B

Appendix B.1

Based on equations (1) and (2) and incorporating transition probabilities with the expected lifetime utility notation introduced in the text, equations (3) and (4), respectively, are written in explicit form as

(3') $\rho V_S = U_S(\pi_0 - bw) + P_{SI}(b)[E_\theta[V_I] - V_S]$ and

(4')
$$\rho V_I = r\left(U_I^R(\pi_0 - \lambda(\theta) - \chi(\theta) + \tau(\theta)) + (V_S - V_I)\right) + (1-r)\begin{pmatrix} q\left[U_I^C(\pi_0 - \lambda(\theta) - \chi(\theta) + \tau(\theta) - f) + (V_S - V_I)\right] + \\ (1-q)\left[U_I^{NC}(\pi_0 - \lambda(\theta) - \chi(\theta) + \sigma(\theta)) + h(V_S - V_I)\right] \end{pmatrix}.$$

Appendix B.2

The first order condition associated with the farmer's problem is given by the constraint to problem (10), or

(B.2-1)
$$\frac{\partial V_S}{\partial b} = U_S'(b)\alpha(b) + U_S(b)\alpha'(b) + \int_0^1 \left[\beta'(b)U_I^R(1,\tau(\theta),\theta) + \beta(b)U_I^R(1,\tau(\theta),\theta)\frac{g_b(\theta \mid b)}{g(\theta \mid b)}\right] g(\theta \mid b)\,d\theta = 0$$

This is an optimal response to the indemnity payment, $\tau(\theta)$; however, the indemnity payment is only second-best because the regulator is constrained by the farmer's response. If the regulator were not constrained, then for a first-best outcome it would have the farmer choose additional biosecurity, such that $\frac{\partial V_S}{\partial b} < 0$. Generalize condition (B.2-1) to be

(B.2-2) $\frac{\partial V_S}{\partial b} < c$,

where c is a constant. $c=0$ for the farmer's problem, while $c=c^* <0$ for the first-best outcome. Using this notation, the Lagrangian can be re-written as

$$\mathcal{L} = U_S(b)\alpha(b) + \beta(b)\int_0^1 U_I^R(1,\tau(\theta),\theta)g(\theta \mid b)d\theta - \kappa\left(\int_0^1 \tau(\theta)g(\theta \mid b)d\theta + m(q)\right)$$

(B.2-3)
$$+\mu\left(\begin{array}{l} U_S'(b)\alpha(b) + U_S(b)\alpha'(b) + \\ \int_0^1 \left[\beta'(b)U_I^R(1,\tau(\theta),\theta) + \beta(b)U_I^R(1,\tau(\theta),\theta)\frac{g_b(\theta \mid b)}{g(\theta \mid b)}\right]g(\theta \mid b)d\theta - c \end{array}\right)$$

Clearly, $\left.\frac{\partial \mathcal{L}}{\partial c}\right|_{c=0} = -\mu < 0$ since an increase in c when $c=0$ implies a decrease in welfare as the solution moves farther away from the first-best outcome $c^* <0$. Hence, $\mu>0$.

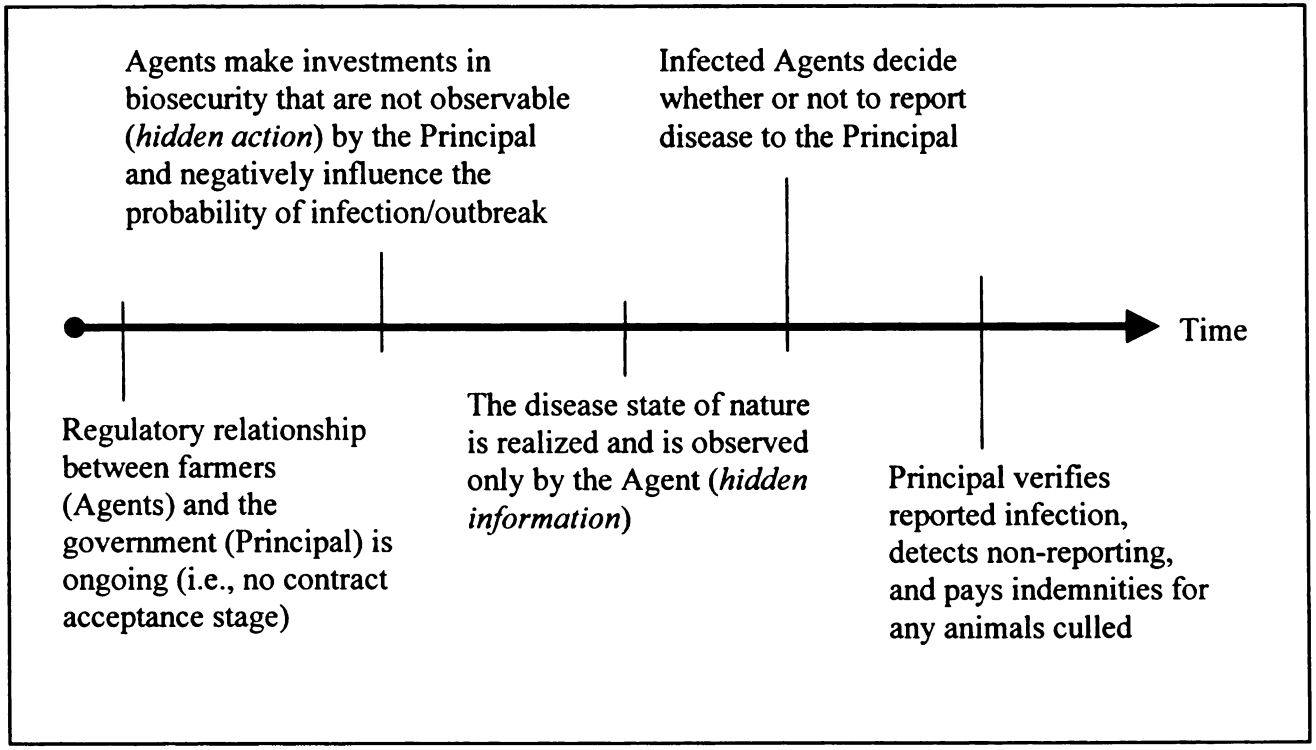

Figure 2.1. Timing and information setting of interaction between the government and farmers

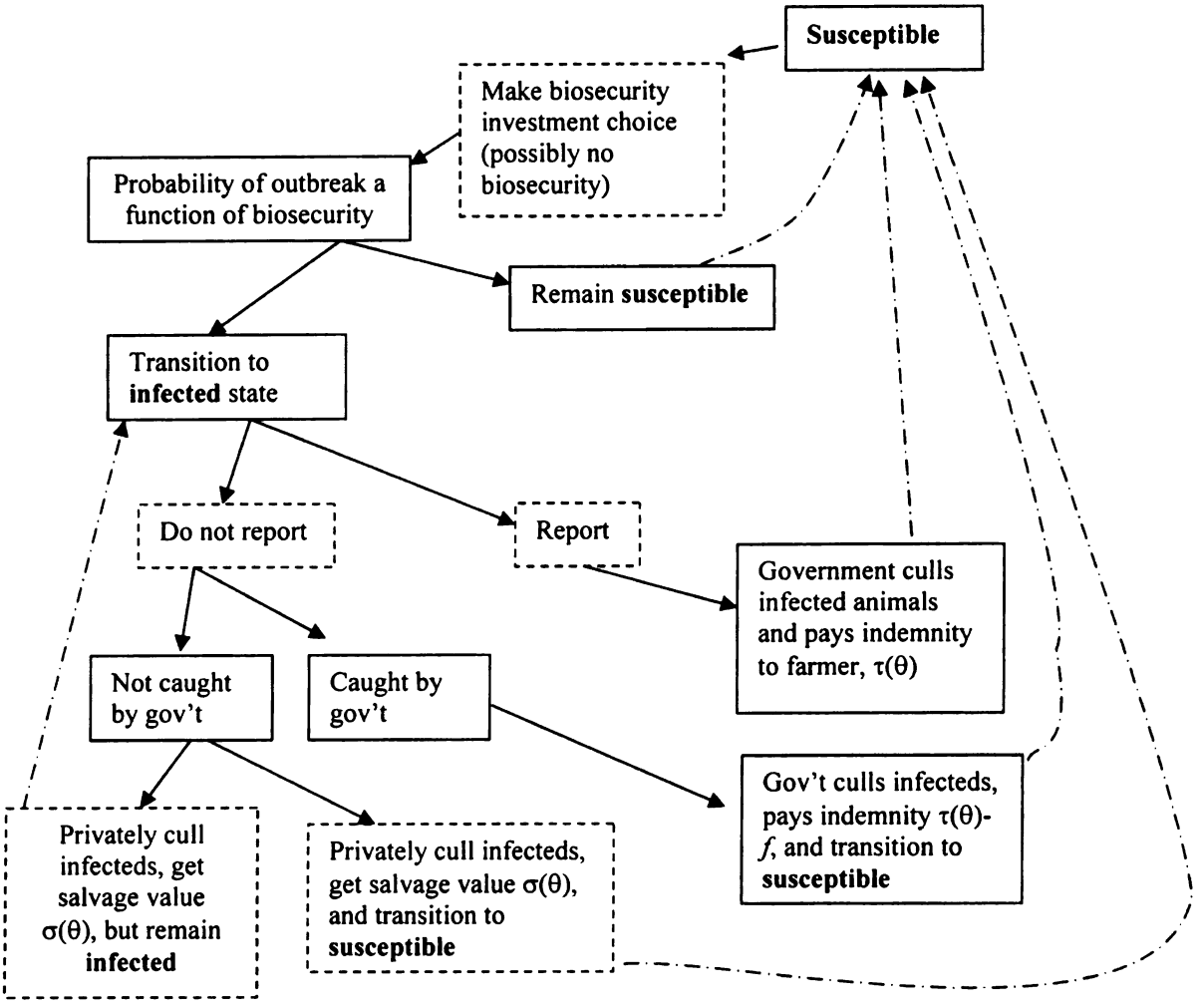

Figure 2.2. Individual farmer decision tree: disease states in bold and farmer choices in dashed boxes

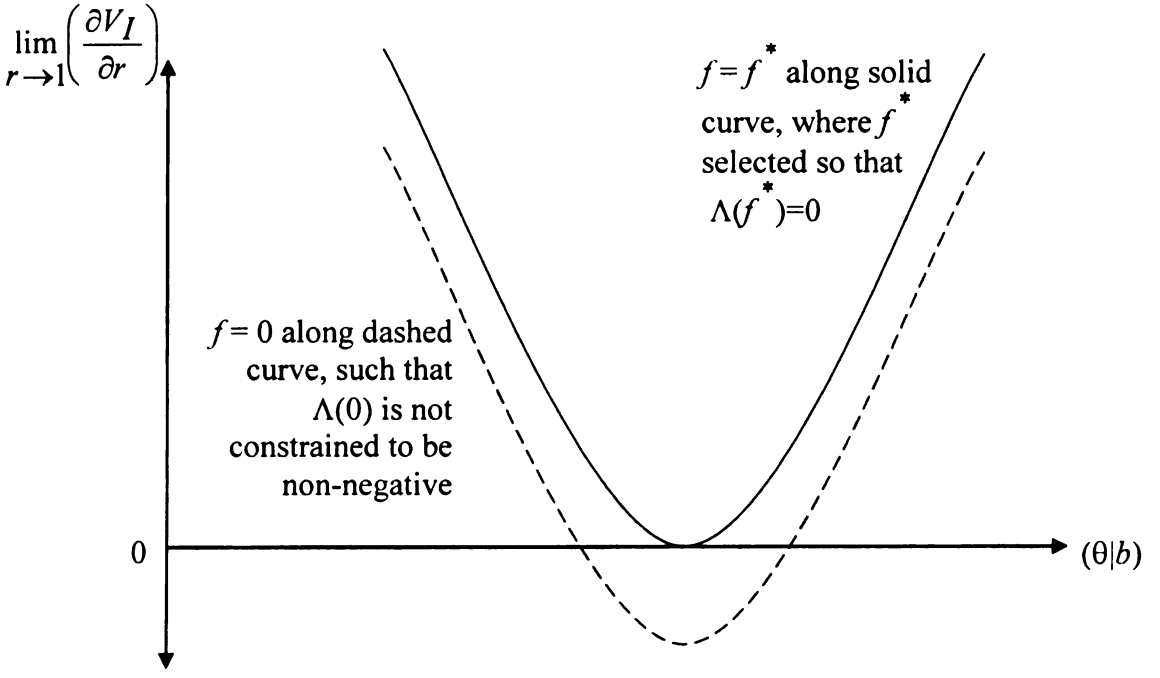

Figure 2.3. Fines f are set to ensure positive marginal incentives to report for all prevalence levels θ.

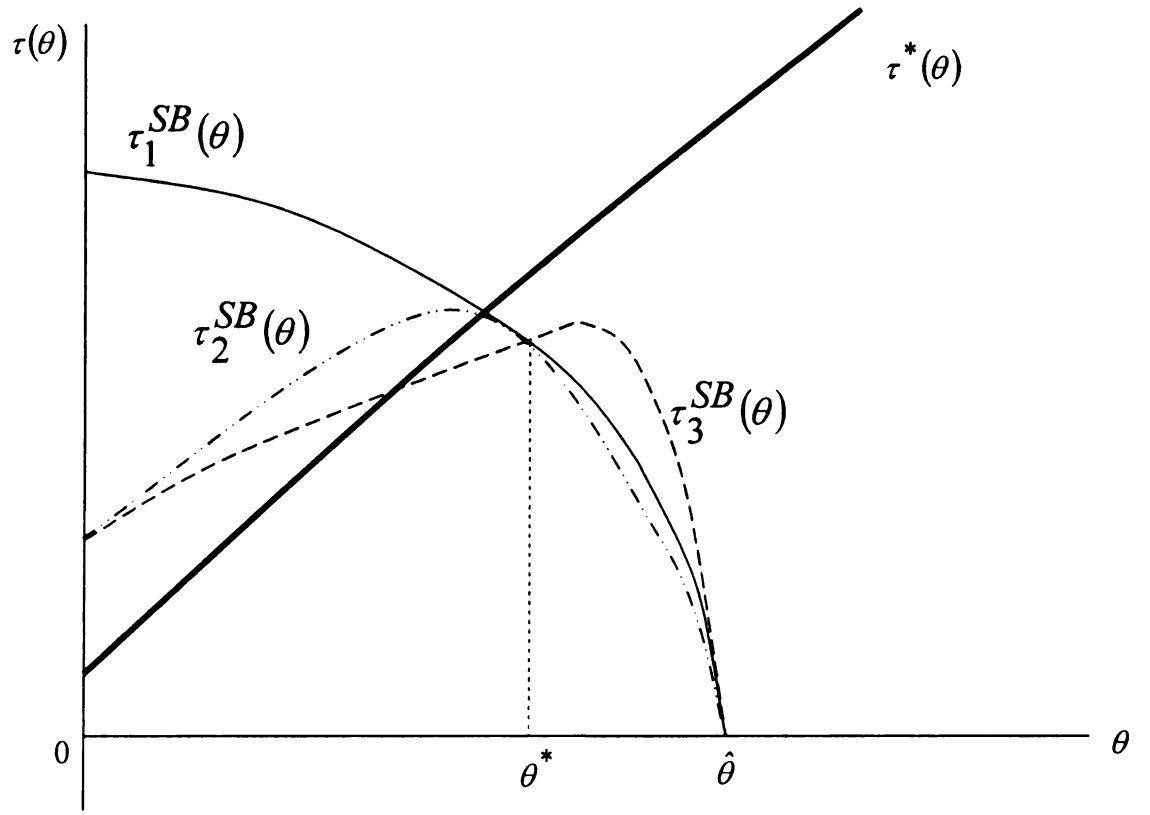

Figure 2.4. Possible shapes for first- and second-best (SB) indemnity payment schedules with a discontinuity at $\hat{\theta}$.

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Essay Three

JOINTLY-DETERMINED LIVESTOCK DISEASE DYNAMICS AND DECENTRALIZED ECONOMIC BEHAVIOR

Introduction

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

Our focus in this paper is on decentralized outcomes. The majority of prior economic research in this area focuses on behavioral outcomes, holding disease risks stationary (i.e., possibly a function of human choices but not reflective of the underlying epidemiological dynamics; Hennessy 2005, 2007). Prior economic work has also generally failed to account for cross-farm externalities, though Hennessy (2005, 2007) are exceptions. Analogously, the majority of prior veterinary and epidemiological research focuses on how disease dynamics are affected by government intervention, such as herd depopulation, without considering producer behavioral responses. In this paper, we consider the joint-determination of disease and behavioral dynamics across farms, in a decentralized setting.

Livestock disease management is an inherently dynamic process that involves

feedbacks between disease ecology and private behavioral decisions. We consider the impact of these feedbacks while focusing on two common decisions that livestock managers must make: a livestock manager must sequentially decide whether or not to invest in *biosecurity* as a preventive measure and whether or not to *report* infection to government authorities in the event that a disease outbreak occurs. The decision to report infection may or may not arise depending on the uncertain disease outcome, but at some point everyone faces the biosecurity decision. Because it is unclear in advance whether or not livestock will be exposed to infection, investment in biosecurity represents a definite investment of resources in exchange for an uncertain future benefit (own herd protection). But biosecurity may not fully protect one's own herd, and, moreover, losses due to increased regulatory stringency may also arise as a result of a neighbor's herd becoming infected – even in the absence of infection in one's own herd. This is because all herds within infected regions may be affected by costly regulatory actions taken by animal health authorities to eradicate infection. Such regulatory-induced externalities are a common feature of livestock disease problems. For instance, all farms in the bovine tuberculosis (bTB) infected region in Michigan's Lower Peninsula — regardless of infection status — incur private costs as a result of dealing with government testing, movement restrictions and stringent testing rules for trade in live animals that go uncompensated by the government.

Our paper builds in particular on the work of Hennessy (2005, 2007), who also accounts for cross-farm spillovers. Hennessy (2005) constructs a topological model to analyze how disease externalities across farms in different spatial arrangements influence biosecurity decisions. In that model, biosecurity investments exhibit strategic

complementarities with respect to *entry* of a disease and strategic substitution with respect to *spread* of an already-introduced infection. Hennessy (2007) specifies a relationship between disease risk and biosecurity investment within a production region such that disease risks are endogenous and biosecurity is a strategic substitute, and derives a number of general implications about the long-run equilibrium. In both cases, the disease risks are not based on an epidemiological model and therefore the relation between disease risks and biosecurity is stationary. Also, group-level impacts of government regulations are not modeled explicitly. Our model extends this previous work in two principal ways. First, we account for the jointly determined nature of disease and economic outcomes in a fully dynamic bioeconomic model, with endogenous and non-stationary disease risks. Second, we characterize distinct static and inter-temporal strategic effects that arise when government response to infection gives rise to externalities.

We proceed with an analytical model of disease dynamics and then integrate economic behavior into this model. We then present simulation results to illustrate the tradeoffs arising in a joint system, and we contrast these results with those arising from a non-joint system. We conclude with a discussion of the general implications of this research, identifying additional research needs in this area.

Livestock Disease Dynamics

A metapopulation disease model (Levins 1969) is adopted to model cross-farm livestock disease dynamics. In this framework, individual farms (and not the individual animals on each farm) are the primary unit of interest. The farms are dispersed across space, and

disease transmission occurs according to a simple susceptible-infected (*S-I*) model of disease (Mangel (2006) provides a gentle introduction to disease models from theoretical biology). Each farm can be in one of three states at any point in time, indexed by j: susceptible (non-infected, $j=S$), infected ($j=I$), or empty ($j=E$). As we detail below, the rate at which farms transition between disease states depends on farmer decisions to invest in biosecurity (in the S state) or report infection to animal health authorities (in the I state), government disease surveillance effort (to discover non-reporters), and the rate at which the government allows repopulation of empty farms.

Define n to be the fixed number of homogeneous farms in a region, with s farms being susceptible, i farms being infected, and e farms being empty.[1] The number of susceptible farms changes over time according to

$$\dot{s} = \varepsilon e - b\beta_0(s/n)i - (1-b)\beta_1(s/n)i. \quad (1)$$

The number of susceptible farms grows when empty farms are repopulated at the rate ε (the first right-hand-side (RHS) term in (1)), and s is reduced by transitions to the infected state as represented by the remaining terms. New infections occur at different rates based on whether or not farms invest in biosecurity. The proportion of farms that biosecure at time t is denoted by b. For these farms, the disease transmission parameter is β_0, and $\beta_0 s/n$ represents the expected number of new infections generated by each infected individual.[2] For farms that do not biosecure, the disease transmission parameter

[1] Strictly speaking, our "production region" considers the area encompassed by a *disease surveillance zone* established by a government authority to control the spread of infection and within which all farms will be subject to inspections and possible quarantine, depending on the disease in question.

[2] This type of transmission is known as *frequency-dependent* because the expected number of new infections depends on the proportion or frequency of susceptible farms

is $\beta_1 > \beta_0$: farms that biosecure face a lower risk of infection than farms that do not.

The change in the number of infected farms over time is

(2) $$\dot{i} = b\beta_0(s/n)i + (1-b)\beta_1(s/n)i - ri - (1-r)qi\,.$$

The first two terms denote newly infected farms, as in (1). The last two terms represent depopulation of infected farms. Depopulation occurs when a farm reports infection, which is the strategy adopted by a proportion of farms r, or when the government discovers the infection, at a rate q, on the (1-r) proportion of infected farms that did not initially report it.[3] Finally, all transitions between disease states in (1)-(2) are balanced by changes in the number of empty (depopulated) farms given by

(3) $$\dot{e} = ri + (1-r)qi - \varepsilon e\,.$$

The dynamic system can be rewritten in terms of proportions of farms in each state (Hess 1990; McCallum and Dobson 2004). Define $S=s/n$ as the proportion of susceptible farms, $I=i/n$ as the proportion of infected farms, and $E=e/n$ as the proportion of empty farms. Upon making this transformation, equations (1)-(3) can be rewritten as

(1a) $$\dot{S} = \varepsilon E - b\beta_0 SI - (1-b)\beta_1 SI\,,$$

(McCallum, Barlow, and Hone 2001). The most common alternative to frequency-dependent transmission is *density-dependent* transmission or what is referred to as the "mass action model" in the disease ecology literature, where the likelihood of contact between *all* farms is the same. Because it seems reasonable that all herds do not have an equal probability of coming into contact with one another and that the probability of contact is increasing (decreasing) in the proportion of farms that are infected (susceptible), we model disease transmission as being a frequency-dependent process. McCallum, Barlow and Hone (2001) and Begon, et al. (2002) discuss different ways that pathogen transmission can be modeled and how to judge which method is appropriate for a given application.

[3] A disease model that tracks within-patch dynamics (that is, where the animal is the primary unit) would be needed to consider a policy response that culls only individual infected animals ("test-and-slaughter" policy) and not entire herds.

(2a) $\dot{I} = b\beta_0 SI + (1-b)\beta_1 SI - rI - (1-r)qI$,

(3a) $\dot{E} = rI + (1-r)qI - \varepsilon E$.

Typical metapopulation models of disease transmission treat b and r in (1)-(3) as exogenous parameters and typically compare steady state dynamics over a range of initial values for the disease state variables and other parameters. In contrast, we take b and r to be endogenous. Next we develop the behavioral dynamics that govern the economic strategies b and r, which are made in response to current disease risks. In turn, the economic choices b and r endogenously affect infection dynamics in our joint model. In this way, we account for dynamic feedbacks between the economic and disease systems.

A Dynamic Model of Farmer Behavioral Choices

Farmers make decisions about whether to invest in biosecurity and whether to report infection based on their current disease state. Denote individual farmer z's ($z=1,\ldots,n$) biosecurity and reporting strategies as b_z and r_z, respectively. These are *mixed strategies* that may be interpreted as the proportion of the time that an individual chooses each of these binary actions. The distributions of mixed strategies in the population are denoted as b and r, which are analytically equivalent to the proportion of individuals in the population who choose biosecurity and reporting. The individual strategies b_z and r_z are treated as being distinct from the strategies b and r prior to equilibrium; in equilibrium, we will have $b_z=b$ and $r_z=r$ due to the assumption of homogeneous farms.

The farm decision model follows that of Hennessy (2007), which is based on Shapiro and Stiglitz (1984). In this framework, a farm in a given disease state receives an

expected flow of income associated with its current disease state, taking into account the possibility that it will transition to a new disease state at some point in the future. Denote farm z's baseline level of profit in each period in which the farm operates (i.e., $j \neq E$) by π, with baseline profits being zero during the empty state. These profits are gross of any expected biosecurity investment costs, wb_z (where w is the cost of biosecurity), private losses from infection, δ, and regulatory costs, G_{zj}. P_{zjk} represents the rate at which farm z transitions from state j to state k. Both G_{zj} and P_{zjk} may be influenced by farm z's and others' strategies. These terms are specified in greater detail below.

Denote V_{zj} to be the expected lifetime income (or utility) of the zth farmer who is currently in state j. Assuming a discount rate of ρ, the *fundamental asset equations* for susceptible, infected, and empty farms are, respectively[4]

(4) $$\rho V_{zS} = \pi - wb_z - G_{zS} + P_{zSI}[V_{zI} - V_{zS}]$$

(5) $$\rho V_{zI} = \pi - \delta - G_{zI} + P_{zIE}[V_{zE} - V_{zI}]$$

(6) $$\rho V_{zE} = -G_{zE} + P_{zES}[V_{zS} - V_{zE}]$$

Equation (4) is the "time value of the asset" in the susceptible state, which equals the sum of the "instantaneous income per unit time" conditional on being susceptible, π-wb_z-G_{zS}, and the "expected capital loss that would arise were the state to change" (Hennessy 2007,

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

(4a) $$V_{zS} = [\pi - wb_z - G_{zS}]t + (1-\rho t)[P_{zSI}tV_{zI} + (1 - P_{zSI}t)V_{zS}].$$

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

p.702) from susceptible to infected, $P_{zSI}[V_{zI}-V_{zS}]$. Equations (5) and (6) have analogous interpretations. Equations (4)-(6) can be solved simultaneously for V_{zS}, V_{zI}, and V_{zE} as functions of the behavioral strategies, the states of the world, and model parameters.

Now consider the P_{zjk} terms. P_{zSI} is the transition rate to the infected state, which can be obtained from equation (1) as $P_{zSI} = b_z\beta_0 I + (1-b_z)\beta_1 I$.[5] Similarly, the transition rate from the infected to the empty state is $P_{zIE} = r_z + (1-r_z)q$. Finally, the transition rate from the empty state to the susceptible state is $P_{zES} = \varepsilon$.

Three types of regulatory costs may arise in various states of the world. First is a per unit "penalty", x, based on the number of new infections in the region, $[b\beta_0 + (1-b)\beta_1]SI$, and imposed on all operating (non-empty) farms. Hence the total expected penalty for each farm is $[b\beta_0 + (1-b)\beta_1]SIx$. This group penalty can be thought of as the private economic cost of government sanctions that affect all operations. For instance, all farms in the non-bTB free area in Michigan—regardless of infection status—incur private costs as a result of dealing with government testing, movement restrictions and stringent testing rules for trade in live animals that go uncompensated by the government. The penalty intensifies progressively as more new farms become infected and is gradually reduced (to a level that is effectively zero) when the number of new infections is reduced, reflecting the fact that government responses tend to be commensurate with the nature of the disease event. For endemic diseases that regularly

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[5] Note that while farmers are forward-looking and P_{zSI} is state-dependent, we do not suppose that individual farms have perfect foresight about states that are beyond their own control. Instead, farmers (naively) assume the current state of the world will persist. This is a common assumption in decentralized models of resource use (Clark 1990).

occur with low frequency in the population, there may be almost no government intervention unless the number of new infections is increasing so rapidly as to indicate potential economic disruptions or a coming epidemic.

The second type of penalty is imposed only on infected farms that report. Farms that self-report infection are penalized at the rate f, where f is a net cost incurred after government-provided indemnification. Reported infection in our model results in herd depopulation, resulting in an asset loss of A. The government may provide some level of indemnification for these losses. In the United States, the *status quo* federal policy is to indemnify producers based on the disease-free fair market value of animals. Specifically, indemnities are paid to livestock owners whose herds are depopulated—regardless of whether reporting occurred or not—to compensate for the legal taking that has occurred under the U.S. Constitution and to encourage reporting (Ott 2006). Currently, all indemnification is "...equal to fair market value assuming disease-free status" and this is intended to reduce "claims by livestock owners...that they didn't receive full value for their animals" (Ott 2006, p.72). Suppose indemnities are paid as depopulation occurs in the infected state. If they are paid at the rate A, then f represents transaction costs associated with government intervention and business interruption.[6] If indemnities are paid at a rate less than A (or not at all), then f reflects these additional losses.

The third type of penalty is incurred by infected farms that do not report and are discovered, at rate q. These farms are penalized at the rate $g>f$. The transaction costs of dealing with animal health authorities are larger under the non-reporting case, and this is

[6] Technically, fixed costs associated with business interruption would arise in the empty state. Without loss, we economize on notation by accounting for the discounted value of these costs at the time of reporting in the infected state. We make similar assumptions with respect to the cost g, defined below.

reflected by the larger value of g. The larger penalty under the non-reporting case provides some incentives to report, but may not guarantee reporting if farm losses δ and the expected cost of being caught, $(1\text{-}r)qg$, are insufficiently large relative to f. Given these types of penalties, the state-dependent regulatory costs are written as $G_{zE} = 0$, and

(7) $$G_{zS} = [b\beta_0 + (1-b)\beta_1]SIx$$

(8) $$G_{zI} = [b\beta_0 + (1-b)\beta_1]SIx + r_z f + (1-r_z)qg .$$

Given the model specification, we can derive $V_{zS}(b_z, b, r_z, I, S)$, $V_{zI}(b_z, b, r_z, I, S)$, and $V_{zE}(b_z, b, r_z, I, S)$ and use these to determine optimal levels of biosecurity and reporting. As indicated above, a farm's biosecurity and reporting decisions are made at each point in time based on the farm's current disease state. The biosecurity decision is only made by susceptible farms because, once infected or emptied, biosecurity investment yields no private benefits. The decision of whether or not to report infection to animal health authorities is only made by infected farms, whereas susceptible and empty farms face no such decision.

Consider the biosecurity decision. The marginal return to biosecurity for a representative farm at an individual point in time is given by $\partial V_{zS} / \partial b_z$ and the optimal biosecurity strategy $b_z^*(b, r_z, I, S)$ is the solution to $\partial V_{zS} / \partial b_z = 0$. However, there is no closed-form solution.[7] Numerical evaluation of $V_{zS}(b_z, b, r_z, I, S)$ suggests that it is monotonic and approximately linear in b_z for a wide range of feasible values of b, r_z, I, S,

[7] If there was, then biosecurity investment dynamics would be derived from taking the time derivative of $b_z^*(b, r_z, I, S)$. Without a closed form solution, it is impossible to derive analytically the behavioral dynamics given by db_z/dt, and any dynamic analysis based on numerical methods is complicated significantly.

and model parameters. We therefore develop an approximation of V_{zS}. Using a linear combination of the known endpoints $V_{zS}^{B}(b,r_z,I,S) = V_{zS}\,|_{b_z=1} = V_{zS}(1,b,r_z,I,S)$ and $V_{zS}^{NB}(b,r_z,I,S) = V_{zS}\,|_{b_z=0} = V_{zS}(0,b,r_z,I,S)$, we approximate V_{zS} at each point in time according to $\bar{V}_{zS} = b_z V_{zS}^{B} + (1-b_z)V_{zS}^{NB}$. We then use replicator dynamics to model inter-temporal changes in behavior.

An analogous approach is used to model the reporting decision. As this decision is made in the infected state, we approximate V_{zI} by $\bar{V}_{zI} = r_z V_{zI}^{R} + (1-r_z)V_{zI}^{NR}$, where $V_{zI}^{R}(b_z,b,I,S) = V_{zI}\,|_{r_z=1} = V_{zI}(b_z,b,1,I,S)$ and $V_{zI}^{NR}(b_z,b,I,S) = V_{zI}\,|_{r_z=0} = V_{zI}(b_z,b,0,I,S)$.

Biosecurity Dynamics and Strategic Effects

We now focus on the biosecurity decision and introduce *replicator dynamics* (Fudenberg and Levine 1998; Rice 2004; Weibull 1995) to describe the change in the aggregate frequency of biosecurity adoption, b. In a symmetric equilibrium of $b_z = b$ (Rice 2004), b increases when the expected lifetime income from adopting the pure strategy b=1 exceeds the *average* expected lifetime income associated with the current distribution of biosecurity strategies

$$(9) \qquad \frac{\dot{b}}{b} = \alpha[V_S^B - \bar{V}_S] \Rightarrow \dot{b} = \alpha b(1-b)[V_S^B - V_S^{NB}],$$

where $\alpha>0$ is a speed of adjustment parameter. Equation of motion (9) indicates that frequency of biosecurity adoption is increasing (decreasing) when the expected lifetime

utility from always investing in biosecurity exceeds (is less than) the expected lifetime utility from never investing in biosecurity. From the definition of $\overline{V}_{zS}$, the term $V_S^B - V_S^{NB}$ equals the marginal value of an individual's biosecurity strategy, $\partial \overline{V}_{zS} / \partial b_z$, in a symmetric equilibrium. Accordingly, from (9) the sign of $\dot{b}$ is determined by the sign of this marginal value.

Consider the $\dot{b} = 0$ isocline, which implicitly defines the equilibrium proportion of farms that invest in biosecurity, conditional on the current values of other state variables. Three values of b potentially satisfy $\dot{b} = 0$: b=0, b=1, and $b^*(S,I,r) \ni V_S^B - V_S^{NB} = 0$. The equilibrium b^* is of particular importance, as movement away from b^* determines the sign of $V_S^B - V_S^{NB}$ and therefore the sign of $\dot{b}$ in this neighborhood and also the stability of the various equilibria. Specifically, we are interested in the sign of $\partial(V_S^B - V_S^{NB}) / \partial b$ in the neighborhood of b^*.

Given that the term $V_S^B - V_S^{NB}$ equals the marginal value of an individual's biosecurity strategy, the derivative $\partial(V_S^B - V_S^{NB}) / \partial b$ represents how an individual's marginal benefits from biosecurity, ... , are impacted by an increase in aggregate biosecurity adoption, b:

$$(10) \quad \frac{\partial^2 \overline{V}_{zS}}{\partial b_z \partial b} = \frac{\partial\left(\partial \overline{V}_{zS} / \partial b_z\right)}{\partial b} = \frac{\partial(V_{zS}^B - V_{zS}^{NB})}{\partial b} = \frac{(\rho+\varepsilon)(\rho+\eta)x\varepsilon^2\rho\eta SI^2(\beta_1-\beta_0)^2}{\chi(\beta_0)\chi(\beta_1)} > 0$$

where $\eta = q(1-r_z)+r_z > 0$ and $\chi(\beta_k) = -\varepsilon\left\{\eta\varepsilon I\beta_k + (\eta\rho+\rho^2)(\rho+\varepsilon+I\beta_k)\right\} < 0$ (for k =0,1). The positive derivative in (10) indicates that an individual has greater incentives to adopt biosecurity when it is being adopted more by others in the region – a *behavioral externality*. Specifically, biosecurity investments are *strategic complements* when $x>0$, and biosecurity is neither a strategic complement nor a strategic substitute when $x=0$. The strategic complement property means that $V_S^B - V_S^{NB} > 0$ for $b>b^*$ and $V_S^B - V_S^{NB} < 0$ for $b<b^*$.[8] The opposite would hold if b was a strategic substitute (though this case does not arise in our current specification). If b is neither a strategic complement nor substitute ($x=0$), then the term $V_S^B - V_S^{NB}$ does not depend on b and, other things equal, there are only two potential equilibria: $b=1$ or $b=0$.

The dynamics for the case of strategic complementarity are depicted in one dimension in Figure 3.1, holding I, S, and r fixed. Arrows denote the direction of behavioral dynamics for a given value of b relative to b^* (*ceterus paribus*). In Figure 3.1a, $b^* \in (0,1)$ is unstable, while $b=0$ and $b=1$ are both locally stable. While b^* represents a point of indifference between biosecuring and not (i.e., $V_S^B - V_S^{NB} = 0$), it is also a *critical point* at which the marginal incentives to biosecure change (all else equal). Starting at b^*, if one person were to adopt just a little more biosecurity, then this increases the marginal value of biosecurity for everyone else, triggering others to increase

[8] The case of strategic substitutes could arise if farms undertake biosecurity actions to prevent infections from leaving their farm. Farmers will not generally make such investments if the primary benefits accrue to others, at least in the absence of government programs to create incentives for this.

their investment in biosecurity. This process snowballs until everyone is an adopter. The opposite occurs if, starting at b^*, one farmer were to adopt just a little less biosecurity—the marginal value would decrease for everyone and aggregate investment would diminish until no one invests. In Figures 3.1b and 3.1c, b^* lies outside the unit interval and is therefore not a feasible steady state, but remains relevant for determining the stability properties of the other equilibria: b=1 is globally unstable and b=0 is globally stable in Figure 3.1b; b=0 is globally unstable and b=1 is globally stable in Figure 3.1c.

Analogous figures could be drawn to illustrate strategic substitution by drawing all arrows in opposite directions such that b^* in Figure 3.1a is stable—if one farmer were to adopt just a little less (more) biosecurity, the marginal value of biosecuring increases (decreases) for everyone and aggregate investment would increase (decrease) until the equilibrium is re-established. This is consistent with conjectures made about the nature of "out-of-equilibrium dynamic adjustment" in Hennessy (2007, Figure 1, p.704) where biosecurity actions are strategic substitutes. Finally, if b is neither a strategic complement nor substitute (x=0), then the sign of $V_S^B - V_S^{NB}$ is fixed for a given I, S, and r, and b will increase or decrease accordingly until b=1 or b=0.

So far in our analysis of biosecurity dynamics, as in previous analyses (Hennessy 2005, 2007), we have held I, S, and r fixed as we have discussed the stability properties. Hence the probability of becoming infected is a stationary function of b. We found that the system always moves to a corner for the case of strategic complementarities (which are present in our model when x>0), and also for the case of no behavioral externalities (x=0). The case of strategic complementarities is of particular interest because policy

makers seek to maintain incentives for adoption of biosecurity (and reporting). In the context of the myopic model with I and r (and thus state transition rates) held fixed, policies that shift b^* to a value less than zero, so that b=1 is globally stable (Figure 3.1c), are consistent with stated government objectives.

But I, S, and r are not fixed in the joint dynamic system. This means the probability of becoming infected is non-stationary, and so assuming stationarity to derive policy implications may lead to erroneous insights. It is important to consider the interconnectedness of disease and behavioral dynamics. We focus in particular on changes in I (and not in S, as S and I are generally inversely related), as this directly affects the likelihood of infection and, in turn, the marginal incentives to biosecure. This latter effect is given by

$$(11) \qquad \frac{\partial(\partial \bar{V}_{zS} / \partial b_z)}{\partial I} = \frac{\partial^2 \bar{V}_{zS}}{\partial b_z \partial I} = \frac{\partial(V_{zS}^{B} - V_{zS}^{NB})}{\partial I} \gtrless 0,$$

which is too complicated to report and analytically ambiguous in sign. As I changes the incentives to invest are altered and this affects the critical value b^* and thus the basins of attraction to the equilibria b=0 and b=1. Numerical evaluation of the sign of (11) suggests that $\partial^2 \bar{V}_{zS} /(\partial b_z \partial I) > 0$ for "low" values of I, so that an increase (decrease) in I results in a greater (reduced) incentive to invest in biosecurity. In particular, the incentives to invest are low when the risk of infection is low, and so it may be difficult to encourage sufficient, sustained investments to eradicate the disease. Numerical evaluation of (11) also suggests that for "high" values of I the effect of increased infection on the marginal return to biosecurity is $\partial^2 \bar{V}_S /(\partial b_z \partial I) < 0$, which means that

an increase (decrease) in I implies there is a reduced (greater) incentive to invest in biosecurity. The intuition behind this result is that once the prevalence of infection reaches a certain level, the cost of direct (i.e., the group penalty) and indirect (i.e., via transition probabilities to unfavorable economic states) behavioral externalities becomes such that the cost of further biosecurity investments exceeds the marginal benefits.

When we consider feedbacks between disease and behavioral dynamics the path of economic choices over time is found to exhibit much more complex behavior than suggested by analyses that treat I as fixed. While the usual static analysis of strategic effects given by (10) indicates that biosecurity investments are strategic complements, the joint dynamic system reveals that there are *inter-temporal behavioral effects* not previously identified in the literature. Because of the inter-temporal relationship between b and I and given that the marginal effect of disease prevalence on the marginal incentive to invest in biosecurity operates in different directions based on the level of I (which is changing over time), we find that aggregate investments in biosecurity in earlier periods are inter-temporal substitutes for investments in later periods when prevalence is "low", while prior period aggregate investments in biosecurity are inter-temporal complements to investments in later periods when prevalence is "high".[9] To avoid confusion, however, we use the term *behavioral externalities* to refer to the familiar strategic effects represented in equation (10), and we use the term *infection externalities* to refer to the

[9] Hennessy (2005, 2007) modeled biosecurity investments as being strategic substitutes for spreading diseases. The reason for this assumption was not clearly articulated, but it might stem from the stationary disease risks in his models – that is, the lack of infection dynamics. One important way (possibly the only way) a neighbor's biosecurity actions can substitute for one's own is if the neighbor's actions first prevent infection on his farm, which in turn prevents infection spreading to one's own farm. When infection dynamics are not modeled explicitly, then the inter-temporal nature of these two processes cannot be distinguished.

inter-temporal effects represented in equation (11).

Finally, consider the case of no strategic effects (x=0). Holding I, S, and r fixed, equation (9) suggests there is no interior steady state b^*. But when I, S, and r are allowed to change, then it is possible for these values to stabilize at levels such that $V_S^B - V_S^{NB} = 0$. If and when this occurs, $\dot{b} = 0$ and b will stabilize at whatever interior value achieves the steady state.

Reporting Dynamics

Now consider reporting dynamics, assuming a symmetric equilibrium with $r_z = r$. The replicator dynamics for reporting are defined analogously to those for biosecurity and produce an analogous result: frequency of reporting is increasing (decreasing) when the expected lifetime utility from reporting exceeds (is less than) the expected lifetime utility from never reporting,

$$(12) \quad \frac{\dot{r}}{r} = \gamma[V_I^R - \overline{V}_I] \Rightarrow \dot{r} = \gamma r(1-r)[V_I^R - V_I^{NR}],$$

where γ>0 is a speed of adjustment parameter. As with the biosecurity case, the term $V_I^R - V_I^{NR}$ equals the marginal value of an individual's reporting strategy, $\partial \overline{V}_{zI} / \partial r_z$, in a symmetric equilibrium. Hence, the sign of $\dot{r}$ is determined by the sign of this marginal value. But in contrast to the behavioral externalities influencing biosecurity dynamics, there are no *reporting externalities* because others' reporting actions r do not influence farm z's expected asset value (i.e., $\partial \overline{V}_{zI} / \partial r = 0$).

Because there are no reporting externalities, there is no interior equilibrium for r. Rather, when $r \in (0,1)$, reporting is either always increasing or decreasing over time

depending on the relative magnitude of asset values given by the sign of $\partial \overline{V}_I / \partial r_z$. That is, the direction of the reporting dynamics is determined solely by the condition

$$V_I^R(b,1,I,S) - V_I^{NR}(b,0,I,S) > (<)0 \Rightarrow \dot{r} > (<)0.$$

Numerical Simulations and Model Comparisons

We now develop a numerical example to simulate the model described above, which we refer to as the *joint model.* We also simulate two other models to illustrate how the joint model differs from prior work, and to highlight the importance of including feedbacks between the disease and behavioral models. We define an *S-I-only model* that treats economic choices as fixed parameters, and we define a *behavior-only model* that treats infection probabilities (P_{zjk}) as fixed parameters. In each model, the simulation begins at the same initial values for all state variables. In the joint model, all behavioral and disease variables are updated endogenously according to equations (1a)-(3a), (9), and (12). With this structure, there are feedbacks between economic choices b and r and disease states S, I, and E. In the *S-I*-only model, economic choices b and r are fixed parameters (i.e., $\dot{b} = \dot{r} = 0$) while disease states are governed by equations of motion (1a)-(3a). In the behavior-only model, S and I are fixed parameters (i.e., $\dot{I} = \dot{S} = \dot{E} = 0$) while the rate of change in biosecurity investment and reporting are governed by equations of motion (9) and (12), respectively.

Besides shedding light on the results of traditional approaches that treat either behavior or disease risks as fixed, comparisons across the three models for different values of x also helps to illustrate the role of the strategic effects. In the absence of

behavioral externalities (x=0), $\partial(V_S^B - V_S^{NB})/\partial b = 0$ so that biosecurity investments are neither strategic complements nor strategic substitutes. Also, there are no infection externalities in either the behavior-only model (in which I is fixed, so as not to affect behavior) or the *S-I*-only model (in which b is fixed, so as not to be influenced by changes in I). Infection externalities do arise in the joint model, as changes in I affect the marginal incentive to invest in biosecurity in each period.

Table 3.1 lists variable descriptions and the baseline parameterization used in all numerical simulations in order to facilitate comparison of results across models given the same starting values. The initial values for the behavioral variables in the baseline case were selected so that farms are equally likely to invest in biosecurity (report infection) and not invest (not report). The starting values for the disease states reflect a situation where infection in a region has risen to a level where government chooses to intervene to limit losses from the endemic disease.

The long run (steady state) results that emerge for each model are illustrated in Figure 3.2. State variables are listed on the vertical axis and the results for each (in terms of proportions of farms) are represented as bars. In the *S-I*-only and behavior-only models, solid bars denote variables that are endogenous and striped bars denote variables that are treated as fixed parameters.

In the *S-I*-only model, farms invest (do not invest) in biosecurity and report (do not report) infection with constant, equal probability. The steady state is reached very quickly, with endemic infection on about one-third of farms and less than half of farms being susceptible. High rates of infection in the long run are due to the lack of behavioral response to infection risks. Also note that about one-fourth of farms are depopulated in

the steady state as a result of the high level of infection that persists through the region. This seems like a rather unlikely outcome when depopulation is used to respond to discovered or reported infection for diseases that may become endemic to regions of the U.S.

In the behavior-only model, the steady state consists of maximum biosecurity investment and reporting rates ($b = r = 1$ in Figure 3.2), due to the constant high rate of infection that does not respond to behavioral choices. The steady state is reached even faster than in the *S-I*-only model, also via a direct approach path.

Results of the joint model differ notably from both the *S-I*-only and behavior-only models. The first difference arises in the steady state results because the joint model accounts for feedbacks, which allows disease and behavioral outcomes to respond over time. In the steady state, nearly all patches are non-infected (S=0.95), with very low endemic infection and an equally low proportion of empty farms (I=E=0.025). This contrasts markedly with the proportions of infected and empty farms in the behavior-only and *S-I*-only models. Biosecurity (b=0.72) is at an intermediate level in the steady state: it is less than in the behavior-only model because infection risks (a function of I) are reduced as a result of prior biosecurity investments, reducing later incentives to invest; biosecurity is greater than in the *S-I*-only model because in the joint model farmers can respond to disease risks by increasing their level of investment. Reporting strategies (r=1) are the same as in the behavior-only model, and greater than in the *S-I*-only model so as to reduce the expected fine from getting caught in a non-reporting, infected state.

The second difference between the joint model and the other two models, due to infection externalities in the joint model, lies in the approach path to the steady state.

Specifically, the approach path for each state variable in the joint model (i) exhibits *dampened oscillations* (Figures 3.3 and 3.4a), unlike the other two models, and (ii) takes a much longer time to reach the steady state relative to the other two models, which approach their equilibrium levels quickly without any overshooting (not pictured). The oscillations result from the inter-temporal feedbacks between disease states and economic behavior: the response of infection dynamics to changes in behavior and the infection externalities present in the behavioral dynamics (expression (11)). In particular, the sign of (11) is positive in our simulation due to sustained low levels of endemic disease. This indicates that the marginal incentives to invest (divest) in biosecurity are increasing (decreasing) when I is increasing (decreasing). Thus, when I rises (falls) to a certain level, b rises (falls) in the population (as in the blown-up section of Figure 3.3). For instance, suppose b was rising in response to increased infection. In turn, these greater biosecurity levels reduce the force of infection and there is a decline in $\dot{I}$. If the investments change the sign of $\dot{I}$, then the magnitude of $\dot{b}$ will also begin to fall and could eventually change signs. The result is a series of oscillations until a steady state is reached. The process of overshooting the steady state greatly prolongs the period of adjustment before the system settles down.

Now consider altering the baseline scenario by setting $x>0$. This group-penalty scenario allows us to evaluate the effect of behavioral externalities on system dynamics in the different models. Because behaviors are fixed in the *S-I*-only model, there is no change in this model relative to the baseline parameterization. We also find that introducing behavioral externalities has an unnoticeable effect in the behavior-only model (not pictured); b and r both move to one very quickly. The effect of introducing

behavioral externalities (for example, x=4 in Figure 3.4b) does however have significant effects on dynamic outcomes in the joint model. In particular, by introducing strategic complementarities within periods, the jointly-determined dynamic system no longer reaches a steady state: overshooting persists indefinitely. Strategic complementarities exacerbate the volatility of the system sufficiently that the system no longer settles down. In contrast to the dampened oscillations observed in the baseline parameterization with no externalities (Figure 3.4a), biosecurity investment and disease states fluctuate erratically following a path consistent with dynamic chaos over a bounded range of values for each individual state.

Further sensitivity analysis (increasing x to 10) indicates that (i) the range over which disease (not pictured) and biosecurity strategy states fluctuate chaotically is increasing in x (compare Figure 3.4c to Figure 3.4b), and (ii) the strategic complementarities are increasing in response to changes in I (i.e., $\partial\left(\partial(V_S^B - V_S^{NB})/\partial b\right)/\partial I > 0$), or equivalently that the infection externalities (or the inter-temporal complementarities) are increasing in response to changes in b. In particular, this second effect implies that the speeds of adjustment of both b and I are increased, and this can be seen by the increased frequency of oscillations over the same length of time in Figure 3.4c relative to Figure 3.4b. The overall effect on the joint system of increased group costs x associated with government response to infection is to increase the variance of disease and behavioral outcomes over time.

Discussion and Conclusion

There are four main implications of this research. First, when we account for feedbacks

in jointly-determined dynamic systems, it can yield insights not captured by myopic models of disease or economic behavior. Such insights include the distinction between behavioral and infection externalities and the expectation that it may be difficult to achieve an eradication objective without adjusting government actions over time. For instance, the need for a shift in policy to achieve eradication has been suggested for the bTB problem in New Zealand (Livingstone, et al. 2006), which has had a similar experience to that in Michigan. The US experience with scrapie eradication in sheep provides one example of a (bounty) program that had to adapt over time in order to eventually achieve eradication (Kuchler and Hamm 2000).

Second, by jointly modeling disease and behavioral dynamics the endogeneity of infection risk is captured. When we account for feedbacks in co-determined systems, the influence of economic choices on infection risks is taken into account and transition rates between economic states are non-stationary, affecting the inter-temporal tradeoffs that arise. The notion of endogenous risk may also be instructive in thinking about government response to widespread infection in the presence of externalities.

Third, if economic damages from disease are assumed to be convex, then the finding of greater variation in endemic disease levels from our simulations can be expected to result in greater sustained average costs associated with disease in the region affected. It is important to note that the origin of these costs in our model lies in the structure of government disease control and indemnification policies because movement restrictions impose costs on *all* farmers in a region but only *infected* farms that are depopulated to control disease are compensated for their losses. This is an element common to standing disease eradication programs as well as government response to

foreign animal disease outbreaks with the potential to have market-level impacts.

The final implication of this research is the critical need for data necessary to parameterize models of this kind to analyze specific cases, as has been done in social planner oriented models, like those developed for Foot-and-Mouth disease in the US (Kobayashi, et al. 2007a,b) and France (Mahul and Durand 2000; Mahul and Gohin 1999). When data are available for a particular disease and geographical area, it may even be possible to integrate disease epidemiology with decentralized strategic interactions to better inform models of optimal allocation of public resources to respond to an epidemic. In developing our numerical simulations we were made aware of the general lack of empirical estimates of inter-herd disease transmission coefficients and longitudinal data on livestock disease prevalence trends or farmer behavior necessary to parameterize such a model. Without such data available, it will remain impossible to evaluate the performance of joint disease ecology-economic models which is necessary in order for such models to be of greatest use for policy making or economic decision making purposes.

Appendix C

Table 3.1. Baseline Modeling Parameters for the *S-I*, Behavioral, and Joint Model Simulations

Description	*Parameter or State Variable*	*Value*
Underlying disease transmission coefficient applicable when invest in biosecurity	β_0	0.25
Disease transmission coefficient applicable when do not invest in biosecurity	β_1	3.1
Probability infection is discovered when don't report to animal health authorities	Q	0.5
Instantaneous income flow at each point in time	π	100
Cost of biosecurity	W	10
Cost of infection	δ	40
Per unit "penalty" imposed on all farms based on the change in overall infection	X	0
Cost of reporting infection	F	40
Cost of getting caught not having reported infection (with probability q)	G	80
Discount rate	ρ	0.05
Speed of adjustment parameter in the biosecurity strategy replicator dynamics	α	0.5
Speed of adjustment parameter in the reporting strategy replicator dynamics	γ	0.5
Reporting strategy on farm *z*, aggregate reporting rate in the population	$r(0)$	0.5
Biosecurity investment strategy on farm *z*, aggregate rate of investment	$b(0)$	0.5
Infected proportion farms	$I(0)$	0.4
Empty proportion farms; $E_0=r_0I_0+(1-r_0)I_0q$	$E(0)$	0.3
Susceptible proportion farms	S(0)	0.3
Transition rate from empty (depopulated) to susceptible	ε	1.0

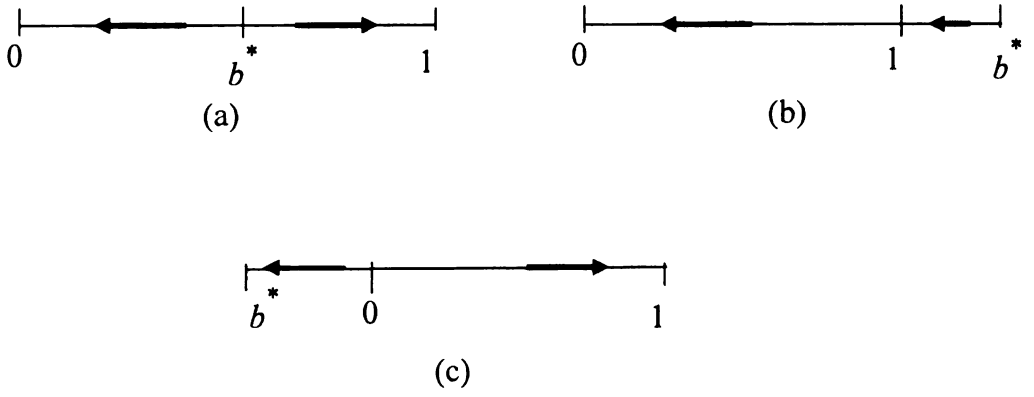

Figure 3.1. One-Dimensional Illustration of Strategic Complementarities in Biosecurity b, When S, I, and r Are Held Fixed: (a) b=0,1 are both stable equilibria; (b) b=0 is the only stable equilibrium; (c) b=1 is the only stable equilibrium.

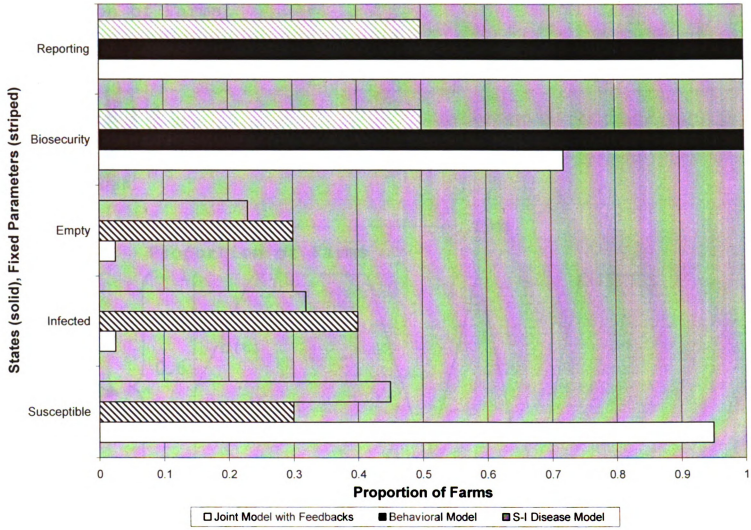

Figure 3.2. Comparison of Steady State Model Results for Baseline Parameterization of *S-I*, Behavioral and Joint Model

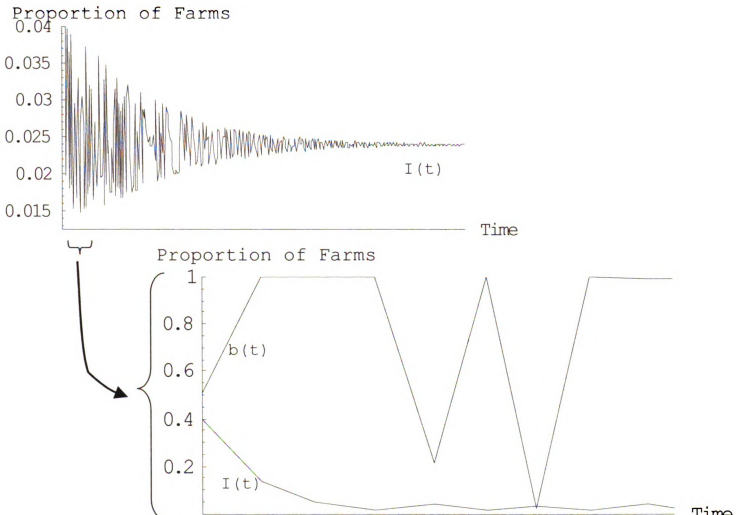

Figure 3.3 Approach Paths to Steady States Exhibit Dampened Oscillations in the Joint Model Due to Feedbacks

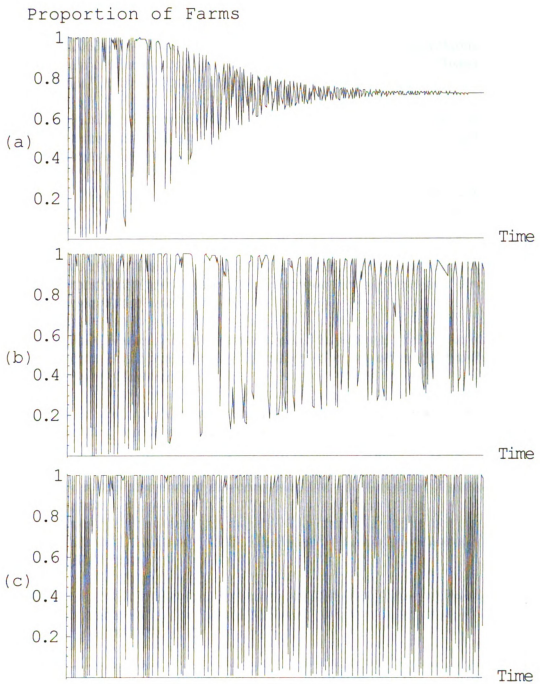

Figure 3.4. Time Paths for Biosecurity Strategy, $b(t)$, Under Joint Model: (a) No behavioral externalities (x=0); (b) "Small" behavioral externalities (x=4); (c) "Large" behavioral externalities (x=10)

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