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Essays on the Economics of Disease, with Particular Reference to Livestock

Tong Wang
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Essays on the economics of disease, with particular reference to livestock

by

Tong Wang

A dissertation submitted to the graduate faculty

in partial fulfillment of the requirements for the degree of

DOCTOR OF PHILOSOPHY

Major: Economics

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Table of Contents

List of Tables	iv
List of Figures	v
Acknowledgement	vii
Abstract.....	ix
Chapter 1. General Introduction	1
Chapter 2. Livestock Disease Indemnity Design under Common Uncertainty: A Multi-agent Problem.....	3
Abstract.....	3
Introduction.....	3
Model Setup.....	6
Second-best Situation.....	7
First-best Situation	16
An Example	19
Solution and Discussion.....	22
Conclusion	26
References.....	27
Appendix A.....	31
Appendix B.....	35
Chapter 3: Where are the Food Animal Veterinarian Shortage Areas Anyway?	46
Abstract.....	46
Introduction.....	46
Materials and Methods.....	48
Data Collection and Management.....	48

Models.....	51
Results.....	54
Discussion.....	59
Conclusion	60
References.....	61
Chapter 4: Modeling Interdependent Participation Incentives: Dynamics of a Voluntary Livestock Disease Control Program	
Abstract.....	69
Introduction.....	69
Model Scheme	78
Simulation.....	97
Conclusion	105
References.....	106
Appendix	112
Chapter 5: Role of Strategic Interactions in Disease Prevention and Cure Effort Choices	
Abstract.....	131
Introduction.....	131
Model	133
An example.....	144
Concluding Remarks.....	148
References.....	149
Appendix A.....	150
Appendix B.....	152

List of Tables

Table 2.1: Joint Probability Distribution Computation Formula	40
Table 2.2: Bounds on ρ	40
Table 2.3: Discrete Joint Probability Distribution for (q_1, q_2) when $\rho = 0$	40
Table 2.4: JC Indemnity Payments Varying with Correlations	40
Table 2.5: JC Indemnity Payments Varying with Biosecurity Effectiveness	41
Table 3.1: County descriptive statistics ^a	63
Table 3.2: Count model parameter estimates	63
Table 3.3: Designation model parameter estimates	64
Table 3.4: Designation model parameter estimates, Count model residual included	64
Table 3.5: Comparison of state-level shortage situations with national average	65
Table 3.6: Shortage situations in the ten states omitted from the 2010 VMLRP	66

List of Figures

Figure 2.1: Uniqueness of Solution	42
Figure 2.2: Utility Variability under Different Correlation Levels.....	43
Figure 2.3: Utility Variability under Different Biosecurity Effectiveness Levels.....	44
Figure 2.4: Utility Variability under Different Reservation Utilities	45
Figure 3.1: Veterinary medicine schools and colleges, and VMLRP participating states in Lower 48	67
Figure 3.2: Comparative severity of Food Animal Veterinarian shortage situations by state (2010 VMLRP participants)	68
Figure 4.1. A flow chart of the model scheme.....	115
Figure 4.2. Equilibria under different cost structures—voluntary program.	115
Figure 4.3 (a). Cobweb Effect---Non-Participation.....	116
Figure 4.3 (b). Cobweb Effect---Full Participation	116
Figure 4.3 (c). Cobweb Effect---Partial Participation.....	117
Figure 4.4. Participant Types under Different Costs---Mandatory Program	117
Figure 4.5. Participation rate and ratio of motivated Type A producers to all producers. ...	118
Figure 4.6 (a). Participation rates under different values of α	118
Figure 4.6 (b). Participation rates under different values of \underline{r}	119
Figure 4.6 (c). Participation rates under different values of V	119
Figure 4.7 (a). Subsidy and tipping— $\alpha = 0$	120
Figure 4.7 (b). Subsidy and tipping— $\alpha = -0.4$	120
Figure 4.8 (a). Cost heterogeneity— $\alpha = -0.5$	121
Figure 4.8 (b). Cost heterogeneity— $\alpha = 0.3$	121
Figure 4.9. Effective program vs. baseline program.....	122

Figure 4.SM1. Equilibria under Different Cost Structures—Voluntary Program, Coarse	
Grading.	130
Figure 4.SM2. Participant Types under Different Costs—Mandatory Program, Coarse	
Grading.	130

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Abstract

This dissertation analyzes and evaluates government policies regarding private sector livestock disease control. The first essay takes common uncertainty into account when designing the optimal livestock indemnity scheme. Relative performance evaluation method is suggested and justified in some special cases. The second essay provides an empirical analysis on the comparative shortage of food animal veterinarians in the United States. Based on this result we also evaluate the veterinary medicine loan repayment program implemented in 2010. The third essay studies the dynamic interactions among farmers' participation decisions to a voluntary livestock disease control program. Through a simulation with Johnes' disease the role of government subsidies on achieving tipping is illustrated. The fourth essay focuses on a susceptible-infected-susceptible type of disease and explores the interactions between farmers' biosecurity and eradication efforts. Our analysis provides evidence in favor of public prevention programs over public cure programs.

Chapter 1. General Introduction

Optimal design of government policies in private sector livestock disease control is of great importance due to increasing social cost of the disease. This dissertation analyzes and evaluates relevant policy issues with a series of four essays, each emphasizing a different facet of the problem.

While common uncertainty is not uncommon when it comes to livestock industry, it has not been incorporated in the optimal livestock indemnity design. Using a principal multi-agent framework, my first paper suggests that relative performance evaluation (RPE) method is most justified either when high correlation exists between farmers' disease prevalence rate or when the effectiveness of biosecurity effort proves to be low.

One aim of my second paper is to provide an objective evaluation of comparative shortage of food animal veterinarians in the United States. The other target of it is to evaluate the Veterinary Medicine Loan Repayment Program (VMLRP), implemented in 2010 to address the perceived regional shortages in veterinary occupations. On the whole the program appears to perform quite well. However, comparative shortage is generally more severe in states that have no VMLRP designated private practice shortage counties than in states that do.

When it comes to voluntary livestock disease control program, neither the magnitude nor the time length of government subsidy required in motivating participation is clear. The third paper demonstrates a strategic complementarity among participation decisions, where after a slow start momentum can build such that the market premium for participation and the participation rate increase sequentially. Guided by this finding, we identify plausible conditions under which temporary government subsidies to the least cost effective producers

could cause tipping toward full participation to occur.

On the farm level, an infectious livestock disease could be eradicated. However, permanent immunity from all livestock diseases is not possible. Moreover, once one farm contracts the disease, all the neighboring farms could be immediately affected. In this context, the fourth paper provides a succinct two-agent model to explore the interactions between farmers' biosecurity and eradication efforts. Intra-farm temporal interactions and inter-farm contemporaneous interactions coexist. Our model suggests a preference to the subsidy on biosecurity effort rather than on eradication efforts.

Chapter 2. Livestock Disease Indemnity Design under Common Uncertainty: A Multi-agent Problem

ABSTRACT

This article develops a principal multi-agent model to study the optimal livestock indemnity design. We show that both within farm disease prevalence and average regional disease prevalence are key factors in the determination of optimal second-best indemnity scheme. The properties of first-best indemnity payment scheme are studied. Special second-best cases exist where the perfect risk sharing property of the first-best still applies. A numerical example is provided. Results suggest that relative performance evaluation is most useful if there is high correlation of farmers' disease prevalence rates or if the effectiveness of biosecurity effort proves to be low.

INTRODUCTION

Government indemnity programs are based on the Fifth Amendment of the US Constitution, which specifies that compensation must be provided for private property taken for public use. In the face of highly contagious or zoonotic diseases, mass destruction is often necessary to return a country to the disease free status. Therefore, indemnities will be paid to the livestock owners in compensation for the destruction of their animals. The Animal Health Protection Act (AHPA) provides guidance on indemnity payments. According to Ott (2006), two guiding principles established by AHPA for compensation are “fair market value and reduction in payment for any other compensation received.”

Historically indemnity has been playing an important role in garnering farmer's support for disease eradication (Kuchler and Hamm 2000, Olmstead and Rhode 2004). However, it may also give farmers a reason to cut back the biosecurity measure (Muhammad and Jones 2008). Therefore in designing an optimal indemnity payment scheme it is important for the

government to take farmers' ex-ante biosecurity choice into account (Jin and McCarl 2006). Since biosecurity efforts are typically hard to observe, it is important to give incentive for farmers to comply voluntarily (Gramig, Horan, and Wolf 2005, 2009). However, the current government indemnification level, based on the animal's fair market value, fails to satisfy such an objective (Gramig, Horan and Wolf 2009).

With this ex-ante moral hazard problem in mind, Gramig, Horan and Wolf (2009) use a principal-agent model to address the incentive compatibility issue. A limitation of their paper is that the common uncertainties associated with the disease are assumed away. Under such simplification, all farms are treated as facing independent risks. This is hardly the case in reality. For example, when highly contagious exotic animal diseases such as Foot and Mouth Disease (FMD) break out in a region, typically a large number of farms become infected by this disease, even if adequate biosecurity measures are taken (Ekboir 1999).

Common uncertainties exist not only on the production side, but also on the demand side. In both cases farmers face interdependent risks. For example, even when very few animals are affected by Bovine Spongiform Encephalopathy (BSE), a much less contagious disease than FMD, major losses can occur through price impacts. Demand for all products will fall as consumers typically find it difficult to distinguish the contaminated food from the normal one.

In the face of common uncertainty, relative performance evaluation (RPE) is introduced in agency literature to improve contract efficiency (Hölmstrom 1979, Mookherjee 1984 and Luporini 2006). Under the RPE, the agents are evaluated on their performance relative to a comparison group, rather than an absolute standard. RPE is commonly used in organizations, e.g., corporate CEOs are often paid on their performance relative to their competitors (Kren 2002). Under a linear compensation model where the random variables affecting their outputs are normally distributed and negatively correlated, Hölmstrom and Milgrom (1990)

found that the optimal compensation to one agent is positively related to the other agents' performance. In a general setup and using First Order Approach (FOA), Luporini (2006) proved that affiliation of the random variables affecting the agents' outputs is both necessary and sufficient for one agent's compensation to be non-increasing in the other agent's output.

In this paper we will present a principal multi-agent model to address the moral hazard issue as well as the interactions among the farmers. Under this setup incentive compatibility (IC) is presented in a game context, which is an extension to the single farmer's IC specified by Gramig, Horan, and Wolf (2005, 2009) and Hennessy (2007). In this regard our IC conditions resemble those in Kobayashi and Melkonyan (Forthcoming) where the focus is on strategic interactions between biosecurity measures. The optimal government indemnity scheme will be presented in a RPE form. That is, the compensation to a farmer is not only contingent on his own disease status, but also on the other farmers' disease prevalence rate as well. We will show the advantage of such scheme over the one proposed by Gramig, Horan, and Wolf (2005, 2009). Feasibility for indemnity implementation will also be discussed.

Our paper will also extend the RPE literature on the following issues. First we will prove the result of Luporini (2006) without the assumptions of FOA. This proof renders FOA specific assumptions, such as convexity of the distribution function (CDF), unnecessary. In addition, we will suggest a condition equivalent to the affiliation condition suggested by Luporini, to avoid the need to go back and forth between the commonly used setup in agency literature and the original setup.¹ Lastly, our numerical solutions of optimal indemnity schemes will indicate possible scenarios where use of RPE may be justified to improve contract efficiency.

¹ Following Mirrlees (1974), in the agency literature it is common practice to use the joint distribution function of the outputs parameterized by the agents' efforts. Result of Luporini (2006), however, is presented in the notation of the original setup.

This paper will proceed as follows. After presenting the model setup, we will analyze the properties of optimal indemnity scheme under both second-best (SB) and first-best (FB) situations. We will also identify some special SB situations where the FB indemnity scheme is optimal. Following the theoretical results, we will use a numerical example to illustrate the method to solve for the optimal indemnity payments. Situations are identified where RPE will be most effective.

Model

Model Setup

To model interdependent disease risks, we will present a one-principal, two-agent model which resembles the model in Mookherjee (1984). Here the principal stands for the government, and the agents stand for two farmers whose livestock face a positive probability of contracting a certain contagious disease.

Let $B = \{b_i \mid b_i \geq 0, i = 1, 2\}$ denote the possible biosecurity practices for both agents.² The unit cost of each agent's biosecurity measure is w . A random variable $\theta_i \in [0,1]$ stands for an environmental risk factor that is beyond farmers' control. The disease is more prevalent in the area when θ_i takes a high value. The joint probability density function for θ_1 and θ_2 is $g(\theta_1, \theta_2)$.

Assume that all the farms are identical in scale and that the output produced by farmer i is $q_i(b_i, \theta_i) \in [\underline{q}, \bar{q}]$. Note that we use output here as an indicator of the disease, where low farm output is regarded as a result of a high within-herd disease prevalence rate. For

² Later on we assume in addition that the rational agent will not choose $b_i \geq \hat{b}_i$, where \hat{b}_i is the optimal biosecurity action specified by the government. Besides threatening the agricultural export market, a disease outbreak can have an adverse impact on other sectors. The social benefit from biosecurity investment exceeds the farmers' private benefit. Thus farmers' optimal level of biosecurity investment will not exceed the social optimal level specified by the government.

example, the lowest possible output \underline{q} stands for the output where all the livestock are diseased, while \bar{q} denotes the output where no livestock is diseased. The output of farmer i depends both on his own biosecurity effort and on the ambient disease prevalence rate such that $\partial q_i(b_i, \theta_i) / \partial b_i \geq 0$ and $\partial q_i(b_i, \theta_i) / \partial \theta_i \leq 0$. Without loss of generality, assume the output price is 1.

Indemnity to agent i is denoted by $I_i(q_i, q_j)$. Here (q_i, q_j) stands for a combination of outputs by farmers i and j , where $i, j = 1, 2$ and $i \neq j$. Denote farmer i 's utility function by $V(\cdot)$, where $V'(\cdot) \geq 0$ and $V''(\cdot) \leq 0$. Therefore farmer i 's utility is $V(q_i + I_i(q_i, q_j))$ when the combination of outputs by farmers i and j is (q_i, q_j) . Denote the agents' reservation utility as \underline{U} . Here \underline{U} could be understood as the maximum value a farmer can obtain without participating in the eradication program. For example, a farmer may obtain a discounted sales value by selling the diseased livestock to some illegal traders instead.

Following Mirrlees (1974), we will set up the optimal contracting problem by suppressing θ_i and consider output levels as random variables parameterized by the biosecurity input b_i . The joint probability density that the output level (q_1, q_2) is realized given the biosecurity input level (b_1, b_2) is $f(q_1, q_2; b_1, b_2)$, where $f(\cdot)$ is continuous w.r.t. q_1, q_2 . The distribution function corresponding to $f(\cdot)$ is $F(\cdot)$. Here $F(q_1, q_2; b_1, b_2) = \text{Prob}(q_1(b_1, \theta_1) \leq q_1; q_2(b_2, \theta_2) \leq q_2)$.

Second-best Situation

The second best (SB) situation stands for the case where farmers' biosecurity inputs are their own private information and could not be observed by the government. Take Bovine TB as an example. Some relevant biosecurity practices are readily observable, such as having livestock tested for Bovine TB and buying animals from an accredited TB-free herd. Many

other practices are not observable. For example, restricting contact with other herd and restricting on-farm visitors from contact with the herd (Coble 2010). As government monitoring is likely to be either impossible or expensive, we need incentive compatibility conditions so that farmers have no incentive to deviate from the optimal biosecurity inputs.

We assume that the government's objective is to find the most efficient indemnity scheme, while ensuring farmers have incentives to enroll in the eradication program and to take the optimal biosecurity measures. In addition assume that the optimal biosecurity level (\hat{b}_1, \hat{b}_2) is determined exogenously, e.g., by the most recent scientific breakthroughs and epidemiological evidences. The optimal contracting problem (OCP) can be written as:

$$\min_{I_1, I_2} \int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} I_1(q_1, q_2) + I_2(q_1, q_2) f(q_1, q_2; \hat{b}_1, \hat{b}_2) dq_1 dq_2$$

s.t.

$$\begin{aligned} \int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} V(q_1 + I_1(q_1, q_2)) f(q_1, q_2; \hat{b}_1, \hat{b}_2) dq_1 dq_2 - \hat{b}_1 w &\geq \underline{U} \\ \int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} V(q_2 + I_2(q_1, q_2)) f(q_1, q_2; \hat{b}_1, \hat{b}_2) dq_1 dq_2 - \hat{b}_2 w &\geq \underline{U} \end{aligned} \quad (1)$$

$$\begin{aligned} &\int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} V(q_1 + I_1(q_1, q_2)) f(q_1, q_2; \hat{b}_1, \hat{b}_2) dq_1 dq_2 - \hat{b}_1 w \\ &\geq \int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} V(q_1 + I_1(q_1, q_2)) f(q_1, q_2; b_1, \hat{b}_2) dq_1 dq_2 - b_1 w; \quad \forall b_1 \in B, b_1 < \hat{b}_1; \\ &\int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} V(q_2 + I_2(q_1, q_2)) f(q_1, q_2; \hat{b}_1, \hat{b}_2) dq_1 dq_2 - \hat{b}_2 w \\ &\geq \int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} V(q_2 + I_2(q_1, q_2)) f(q_1, q_2; \hat{b}_1, b_2) dq_1 dq_2 - b_2 w. \quad \forall b_2 \in B, b_2 < \hat{b}_2. \end{aligned} \quad (2)$$

Government's objective in OCP is to choose an indemnity payment level to minimize its expected indemnity payment, subject to each agent choosing the optimal biosecurity input level (\hat{b}_1, \hat{b}_2) . Equation set (1) stands for the participation constraints (PC). The government provides indemnity as a financial incentive for farmers to identify and destroy their infected

animals, thus accelerating the eradication efforts. Equation set (2) stands for the Nash incentive compatibility constraints (NIC) for agent 1 and 2, respectively. Note that under NIC no biosecurity effort less than the optimal one will be taken.

Under optimal indemnity scheme $\mathbf{I}_1, \mathbf{I}_2$, where $\mathbf{I}_1 = \{I_1(q_1, q_2), \forall q_1, q_2\}$ and $\mathbf{I}_2 = \{I_2(q_1, q_2), \forall q_1, q_2\}$. NIC ensures that the optimal strategy pair (\hat{b}_1, \hat{b}_2) constitutes a Nash equilibrium (NE). However, NIC does not exclude the possibility of multiple pure strategy equilibria, as noted in Ma (1988). By adding some trivial strategies for each agent and transforming the static game into a dynamic one, Ma (1988) refines the unwanted equilibrium in Mookherjee (1984) under the perfect Bayesian equilibrium. For the rest of this paper we will assume that a unique NE exists.

Following the analysis in Grossman and Hart (1983) and Mookherjee (1984), it is convenient to transform the constraints into a linear form with regard to the control variables. Upon defining $V(q_i + I_i(q_i, q_j)) \equiv v_i(q_i, q_j)$, it follows that $I_i(q_i, q_j) = h(v_i(q_i, q_j)) - q_i$, where $h(\cdot) = V^{-1}(\cdot)$. Next we can obtain the transformed optimal contracting problem (TOCP):

$$\begin{aligned} \min_{v_1, v_2} \int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} [h(v_1(q_1, q_2)) + h(v_2(q_1, q_2)) - q_1 - q_2] f(q_1, q_2; \hat{b}_1, \hat{b}_2) dq_1 dq_2 \\ \int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} v_1(q_1, q_2) f(q_1, q_2; \hat{b}_1, \hat{b}_2) dq_1 dq_2 - \hat{b}_1 w \geq \underline{U}; \\ \int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} v_2(q_1, q_2) f(q_1, q_2; \hat{b}_1, \hat{b}_2) dq_1 dq_2 - \hat{b}_2 w \geq \underline{U}; \end{aligned} \quad (3)$$

$$\begin{aligned} \int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} v_1(q_1, q_2) f(q_1, q_2; \hat{b}_1, \hat{b}_2) dq_1 dq_2 - \hat{b}_1 w \\ \geq \int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} v_1(q_1, q_2) f(q_1, q_2; b_1, \hat{b}_2) dq_1 dq_2 - b_1 w; \forall b_1 \in B, b_1 < \hat{b}_1; \\ \int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} v_2(q_1, q_2) f(q_1, q_2; \hat{b}_1, \hat{b}_2) dq_1 dq_2 - \hat{b}_2 w \\ \geq \int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} v_2(q_1, q_2) f(q_1, q_2; \hat{b}_1, b_2) dq_1 dq_2 - b_2 w. \forall b_2 \in B, b_2 < \hat{b}_2. \end{aligned} \quad (4)$$

The Lagrangian for the TOCP can be written as:

$$\begin{aligned}
L = & \int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} [h(v_1(q_1, q_2)) + h(v_2(q_1, q_2)) - q_1 - q_2] f(q_1, q_2; \hat{b}_1, \hat{b}_2) dq_1 dq_2 \\
& + \lambda_1 \left[\underline{U} - \int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} v_1(q_1, q_2) f(q_1, q_2; \hat{b}_1, \hat{b}_2) dq_1 dq_2 - \hat{b}_1 w \right] \\
& + \lambda_2 \left[\underline{U} - \int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} v_2(q_1, q_2) f(q_1, q_2; \hat{b}_1, \hat{b}_2) dq_1 dq_2 - \hat{b}_2 w \right] \\
& + \sum_{b_1 \in B} \gamma_1(b_1) \left[\int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} v_1(q_1, q_2) [f(q_1, q_2; b_1, \hat{b}_2) - f(q_1, q_2; \hat{b}_1, \hat{b}_2)] dq_1 dq_2 - b_1 w + \hat{b}_1 w \right] \\
& + \sum_{b_2 \in B} \gamma_2(b_2) \left[\int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} v_2(q_1, q_2) [f(q_1, q_2; \hat{b}_1, b_2) - f(q_1, q_2; \hat{b}_1, \hat{b}_2)] dq_1 dq_2 - b_2 w + \hat{b}_2 w \right]
\end{aligned}$$

Following Hölmstrom (1979), we can take point-wise optimization with regard to

$v_i(q_i, q_j)$ and $\gamma_i(b_i)$ respectively for any $b_i \in B$ and $q_i, q_j \in [\underline{q}, \bar{q}]$:

$$\begin{aligned}
(h'(v_1(q_1, q_2)) - \lambda_1) f(q_1, q_2; \hat{b}_1, \hat{b}_2) - \sum_{b_1 \in B} \gamma_1(b_1) [f(q_1, q_2; \hat{b}_1, \hat{b}_2) - f(q_1, q_2; b_1, \hat{b}_2)] &= 0; \\
(h'(v_2(q_1, q_2)) - \lambda_2) f(q_1, q_2; \hat{b}_1, \hat{b}_2) - \sum_{b_2 \in B} \gamma_2(b_2) [f(q_1, q_2; \hat{b}_1, \hat{b}_2) - f(q_1, q_2; \hat{b}_1, b_2)] &= 0;
\end{aligned} \tag{5}$$

$$\begin{aligned}
\int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} v_1(q_1, q_2) [f(q_1, q_2; b_1, \hat{b}_2) - f(q_1, q_2; \hat{b}_1, \hat{b}_2)] dq_1 dq_2 + (\hat{b}_1 - b_1) w &\leq 0; \\
\text{if } \gamma_1(b_1) > 0, \text{ then " = 0 " } \forall b_1 \in B; & \\
\int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} v_2(q_1, q_2) [f(q_1, q_2; \hat{b}_1, b_2) - f(q_1, q_2; \hat{b}_1, \hat{b}_2)] dq_1 dq_2 + (\hat{b}_2 - b_2) w &\leq 0; \\
\text{if } \gamma_2(b_2) > 0, \text{ then " = 0 " } \forall b_2 \in B. &
\end{aligned} \tag{6}$$

From (5) we further obtain:

$$\begin{aligned}
h'(v_1(q_1, q_2)) &= \lambda_1 + \sum_{b_1 \in B} \gamma_1(b_1) \left(1 - \frac{f(q_1, q_2; b_1, \hat{b}_2)}{f(q_1, q_2; \hat{b}_1, \hat{b}_2)} \right) \\
h'(v_2(q_1, q_2)) &= \lambda_2 + \sum_{b_2 \in B} \gamma_2(b_2) \left(1 - \frac{f(q_1, q_2; \hat{b}_1, b_2)}{f(q_1, q_2; \hat{b}_1, \hat{b}_2)} \right)
\end{aligned} \tag{7}$$

Equation set (7) is a standard result in principal agent literature (See e.g. Hölmstrom 1979)

and Mookherjee 1984).³ In this paper we will utilize this result to infer the possible structure of the government indemnity program. First, we will present Lemma 1 to explain the LHS of equation (7).

Lemma 1: If $h'(v_i(q_i, q_j))$ increases in q_i for $i, j = 1, 2; i \neq j$, then gross return $q_i + I_i(q_i, q_j)$ increases in output q_i , and vice versa.

Proof. See Appendix C. \square

Note that gross return $q_i + I_i(q_i, q_j)$ increases in output q_i . Otherwise the farmers would have the incentive to destroy some of their output and obtain a greater utility afterwards. On the RHS of equation (7), λ_i ($i = 1, 2$) is the shadow cost of the participation constraint where it is clear that $\lambda_i > 0$. Were $\lambda_i = 0$, then the government can lower the indemnity payment for all possible levels of output with PC and NIC still holding.⁴ The shadow cost of the i -th incentive constraint is $\gamma_i(b_i)$. If $\gamma_i(b_i) = 0, \forall b_i \in B$, then NIC poses no cost to the government. In that case the OCP in SB case will be simplified to that in FB case, which will be discussed in section 2.3.

Given that farmer j takes the optimal biosecurity practice \hat{b}_j , the government will update its prior on b_i after the observation of q_i and q_j . A smaller density ratio on the RHS of equation will signal that \hat{b}_i is more likely. Therefore equation set (7) implies that farmer's utility increases when the optimal biosecurity measure is more likely to be taken. Next we

³ It's easy to see that (7) can be extended to the multi-agent case. Suppose there are n agents, then agent k 's optimality condition will be $h'(v^k(q_k, q_{-k})) = \lambda_k + \sum_{b_i \in B} \gamma_k(b_k)[1 -$

$f(q_k, q_{-k}; b_k, \hat{b}_{-k}) / f(q_k, q_{-k}; \hat{b}_k, \hat{b}_{-k})]$, where $q_{-k} = q_1, \dots, q_{k-1}, q_{k+1}, \dots, q_n$ and $\hat{b}_{-k} = \hat{b}_1, \dots, \hat{b}_{k-1}, \hat{b}_{k+1}, \dots, \hat{b}_n$.

⁴ See Mas-Colell (1995), pp. 485, footnote 8.

will demonstrate in Lemma 2 the conditions that render RPE unnecessary.

Lemma 2: The following two conditions are equivalent: 1) $f(q_1, q_2; b_1, b_2)$ can be factorized into $m(q_1; b_1)n(q_2; b_2)$; 2) $g(\theta_1, \theta_2) = g_1(\theta_1)g_2(\theta_2)$.

Proof. See Appendix A. \square

Mookherjee (1984) proved that Lemma 2 conditions are generically necessary and sufficient for a contract without considering RPE to be optimal. Lemma 2 conditions imply that the probabilities that the two farms contract diseases should be independent of each other. The current concepts on animal disease management through zoning or regionalization can help us understand the idea of farm level risk independence. As outlined in part 92 of 9 CFR (2008), zone recognition in the United States requires information such as veterinary infrastructure, disease status, vaccination status and degree of separation from adjacent regions etc. As an example, in the control of FMD a disease free zone is separated from the rest of the country by a buffer zone, or physical or geographical barriers (Fujita 2004). In this case, we can regard the farms located in the disease free zone as risk independent from the farms outside the zone. To the contrary, if several farms are located in the same zone, then the indemnity payment for one farm should be based on other farm's disease status as well. Hölmstrom (1979) demonstrated that RPE ensures less indemnity payment than that of the independent contract, as the output information of the other farmer is an informative signal about the environmental risk factor. However, no such information is utilized in the independent contract.

Assuming that the Lemma 2 does not hold, we will check the properties of the optimal indemnity scheme. First we introduce assumption 1 to describe the relationship between one farmer's output and its own biosecurity level.⁵

⁵ This assumption is originally brought up by Luporini (2006) as one of two conditions for FOA to be applicable in the principal multi-agent setup.

Assumption 1: Partial monotone likelihood ratio property (PMLRP):

$f(q_i, q_j; b_i^-, b_j) / f(q_i, q_j; b_i^+, b_j)$ is non-increasing in q_i for $b_i^- < b_i^+$, $i, j = 1, 2, i \neq j$.

Given farmer j 's output and biosecurity input, assumption 1 means that a higher output from farmer i implies that higher biosecurity measures are more likely to be taken. This assumption is reasonable as evidence does suggest that biosecurity measures lead to lower disease prevalence rate. For example, changes to swine systems such as ventilation systems and hygiene management have reduced the impact of some important health problems. As a result, once widespread problems such as sarcoptic mange and swine dysentery are very uncommon in modern systems (Davies and Leman 2008). Based on assumption 1, next we will show that the optimal indemnity payment to one farm should be contingent on its own disease prevalence rate.

Proposition 1: Under assumption 1, Farmer i 's gross return $q_i + I_i(q_i, q_j)$ is a non-decreasing function of farm i 's output level q_i .

Proof. See Appendix A. \square

As the output price is assumed to be 1, here q_i could be understood as the salvage value of the animals. A lower value of q_i implies greater within herd disease prevalence rate, thereby farmer i would obtain a decreased salvage value. The term $q_i + I_i(q_i, q_j)$ stands for the total value received by farmer i , which is made up of the salvage value and indemnity payment. Proposition 1 shows that the total value received by farmer i should be a non-increasing function of farm i 's within herd disease prevalence rate. This result also coincides with the conclusion of Gramig, Horan and Wolf (2009) who studied a principal single-agent model. AHPA, Subtitle E of the Farm Security and Rural Investment Act of 2002, instead

specifies that the total value any farmer receives should equal to the fair market value of depopulated animals. Such a specification does not take the prevalence level into consideration, thus is not optimal in providing biosecurity incentives.

Next we will relax Luporini (2006)'s FOA assumptions and study how RPE could be utilized in optimal indemnity payment. In addition, we will present an equivalent condition to the affiliation condition used in Luporini (2006) in Lemma 3.

Lemma 3: The following two conditions are equivalent: 1) $\frac{g(\theta_1^+, \theta_2^-)}{g(\theta_1^-, \theta_2^-)} - \frac{g(\theta_1^+, \theta_2^+)}{g(\theta_1^-, \theta_2^+)} \leq 0$;

2) $\frac{f(q_i, q_j; b_i^-, b_j)}{f(q_i, q_j; b_i^+, b_j)}$ is non-decreasing in q_j for $b_i^- < b_i^+$, $i, j = 1, 2$ and $i \neq j$.

Proof. See Appendix A. \square

Condition 1 in Lemma 3 is first proposed by Luporini (2006) to capture the affiliation relationship between the environmental shocks received by two farmers. The concept of affiliation was introduced by Milgrom and Weber (1982) in auction theory. The idea is that one bidder will expect other bidders' estimated values of a good to increase if his own estimated value increases. In our context it means that when the environmental shock turns out to be favorable for one farmer, then we can also expect a favorable condition for the other. Although affiliation is a stronger assumption than positive correlation, the former is true in many cases where the latter holds (de Castro 2007).

Note that condition 2 in Lemma 3 resembles that in Assumption 1. Additionally it is in line with the common practice after Mirrlees (1974), which uses the joint distribution function of the outcomes parameterized by the actions. The condition means that ceteris paribus a higher output q_j signals a lower level of b_i . Intuitively the equivalence of the two conditions in Lemma 3 is due to the following reason. Ceteris paribus a higher output q_j

signals a higher realization of θ_j . By condition 1 we know that the environmental factor for farmer i is likely favorable too. Thus the same level of q_i indicates a lower level of b_i .

Similar to Luporini (2006), Proposition 2 will demonstrate how the optimal indemnity received by farmer i is affected by farmer j 's output. Luporini applied the FOA under the additional CDF assumption, which is not required in the general approach shown in our proof.

Proposition 2: Under condition 2 of lemma 3, Farm i 's indemnity $I_i(q_i, q_j)$ is a non-increasing function of farm j 's output level q_j .

Proof. Similar to the proof of Proposition 1, this result follows readily from a combination of equation (7), Condition 2 of Lemma 3 and Lemma 1. \square

Proposition 2 shows that in the optimal RPE scheme, one farmer's indemnity payment is a non-increasing function of the other farmer's output, or a non-decreasing function of the other farm's disease prevalence rate. By taking the disease status of other farms into consideration, the government could curtail its indemnity payments to farmers based on the improvement of risk sharing (Hölmstrom 1979). This information is not reflected in the AHPA specification and is not considered by Gramig, Horan, and Wolf (2009) as common uncertainties are assumed away.

When there are more than two farms in the region, for convenience we could regard the other farms as a unit and use the average regional disease prevalence rate. The idea of RPE is already incorporated in the indemnity or insurance practices of other industries. For example, Federal Crop Insurance Corporation (FCIC) offers the Group Risk Plan (GRP) to producers whose farm yields positively correlate with the average county yield (FCIC 2010). GRP explicitly mentions that a farmer with a low yield may not even receive a payment when the

average county yield is high. Such statement follows directly from a combination of the results in Propositions 1 and 2.

First-best Situation

The first-best (FB) situation refers to the case where the moral hazard problem does not exist. That is, the government could observe the true level of biosecurity inputs by the farmers. The model under FB situation resembles the model we studied under SB situation except that the NIC conditions now become unnecessary. Therefore in the FB case a counterpart for equation (7), which implicitly determines the indemnity scheme becomes:

$$\begin{aligned} h'(v_1(q_1, q_2)) &= \lambda_1 \\ h'(v_2(q_1, q_2)) &= \lambda_2 \end{aligned} \quad (8)$$

Equation set (8) indicates that $v_i(q_1, q_2)$ is a constant whatever the realized pair of (q_1, q_2) is. This means that in the FB situation perfect risk sharing is obtained and the farmers will have the same utilities across all states. By (3) we can solve the optimal indemnity level as $I_i^*(q_1, q_2) = h(\hat{b}_i w + \underline{U}) - q_i$ when the optimal biosecurity measure \hat{b}_i is taken. Otherwise the indemnity payment will be arbitrarily small to ensure that farmers will not deviate from the optimal actions. Therefore in the FB case the indemnity payment to farmer i will increase when the disease prevalence rate on farm i increases. It does not depend on the prevalence level of the other farms, so RPE is no longer needed.

Current government indemnification practice implies that the indemnity payment plus any salvage value received by farmers should equal the fair market value. However, all the consequential losses such as loss from business downtime and loss of consumers and markets is not likely to be compensated (Umber, Miller, and Hueston 2010). As these losses could be substantial (Grannis and Bruch 2006), thus the current indemnity practice does not guarantee perfect risk sharing either.

Given the optimal biosecurity level next we will compare the scale of indemnity payment in SB with that in FB.

Proposition 3: To implement the same biosecurity levels, SB expected indemnity payment will be no less than that in the FB.

Proof. See Appendix A. \square

Proposition 3 is a standard result in the principal agent literature.⁶ In the SB situation, there is a conflict between incentive constraints and the farmer's risk-aversion. Thus SB expected indemnity payment will be no less than that in FB where incentive constraints do not exist.

Special SB cases where FB indemnity Scheme applies

This section will demonstrate several special SB cases where FB perfect risk sharing rule is supported under SB.

The first case involves diseases where current biosecurity investments prove to be least effective. Such diseases may include exotic or novel diseases. For example, biosecurity investments made in swine health management usually lack effectiveness for the viral diseases such as PRRS and influenza. Thus there is an emergence of novel animal diseases characterized by high virulence in individual species despite vast biosecurity investments (Davies and Leman 2008).

To describe the relationship between biosecurity investments and disease prevalence level under our model setup, we have $f(q_i, q_j; b_i, \hat{b}_j) = f(q_i, q_j; \hat{b}_i, \hat{b}_j), \forall b_i \in B$. In this case the minimum biosecurity level should be taken as it is not effective. By equation (5), we can see that $h'(v_i(q_i, q_j)) = \lambda_i$. Intuitively, this means that farmers will receive the same payment

⁶ See Mas-Colell (1995), pp. 486.

whatever the disease prevalence levels are. Thus a perfect risk sharing indemnification rule is warranted.

Secondly, it may be optimal to apply a perfect risk sharing scheme on only a subset of farms when farms can be categorized according to their risks. Here a high risk farm stands for a farm with a high potential for disease outbreak. Grabkowsky et al. (2006), for example, divided poultry farms into different risk classes according to factors such as distance to wetlands and distance to adjacent farms. Suppose that there are two farms in an adjacent region where disease typically originates from farm 2. Here we can imagine that farm 2 is frequented by wild bird carrying virus. Or perhaps farm 2 has an open production system that trades with a region where the disease is endemic. Farm 1 maintains a closed system instead. According to Grabkowsky et al. (2006), we could categorize farm 1 as a low risk farm and farm 2 as a high risk farm.

Assume that farm 1 will not suffer a loss from disease if farm 2 maintains a disease free status. To characterize farm 1's biosecurity effort on its disease prevalence rate, we have $f(q_1, q_2; b_1, \hat{b}_2) = f(q_1, q_2; \hat{b}_1, \hat{b}_2), \forall b_1 \in B$ in terms of density function. By equation (7), it follows that $h'(v_1(q_1, q_2)) = \lambda_1$. By lemma 1, we know the gross returns for farmer 1 stay the same for all possible q_1, q_2 . For farm 2, however, assumption 1 applies. According to Proposition 1, the unit indemnity payment should decrease when its disease prevalence level increases.

Potential benefit from risk classification of livestock farms has been address by Niemi et al. (2009). If the high-risk farms could be identified, then the livestock indemnification payments for them will be different. This result suggests a tiered indemnification scheme according to farmers' relative risk, with full indemnity for low risk farms and indemnity contingent on disease prevalence for high risk farms.

Thirdly, FB situation can be resumed when the biosecurity measure is almost always effective. For example, suppose for a certain disease vaccination is optimal and all the vaccinated animals are immune to the disease during the outbreaks. Thus the animals once vaccinated will not contract the disease. In this case, probability mass function is

$$P(q_i, q_j; \hat{b}_i, \hat{b}_j) = 1 \text{ when } q_i = \bar{q} \text{ and } P(q_i, q_j; \hat{b}_i, \hat{b}_j) = 0, \forall q_i \neq \bar{q}. \text{ If } b_i \neq \hat{b}_i, \text{ then}$$

$$P(q_i, q_j; b_i, \hat{b}_j) \neq 0 \text{ on the possible range of } q_i.$$

Under this situation the government can infer with certainty that the optimal biosecurity measure is not taken whenever the herd contracts the disease. This is equivalent to the FB situation where the government can observe the true biosecurity inputs by the agents. Therefore, farmers who fail to take the optimal biosecurity level will receive no indemnity.

Lastly, FB situation can be resumed if the government could audit the biosecurity practice efforts at a cost. Kunreuther, McNulty, and Kang (2002) suggested the use of third party auditing together with insurance instruments to address hidden action issues. The National Poultry Improvement Plan (NPIP) is one such example, where a veterinarian will conduct an audit each year to verify that the plan participants only purchase birds from NPIP stock. In this way, deviations from the optimal practice will be found at a cost. If the audit cost is less than the social welfare difference between FB and SB, then it will be beneficial to resume the FB situation. A possible implementation method is that farmers pay an audit fee in proportion to the quantity of their livestock. During the disease outbreak, all farmers acting in accordance with the optimal biosecurity requirements could obtain full indemnity as specified in the FB case.

AN EXAMPLE

In this section we will provide a simple example where there are only two possible output levels, q^L and q^H , and two possible biosecurity practices, b^L and b^H . The numerical results we obtain here will shed some insights on the structures of indemnity scheme under different

scenarios.

In the example to be analyzed, we assume that the unit biosecurity investment cost is $w = 1$ and that the two options available are $b_i^H = 1$ and $b_i^L = 0.3$. There are two possible output levels $q_i^H = 10$ and $q_i^L = 0$. Farmers can obtain a reservation utility $\underline{U} = 1$ when choosing not to participate in the eradication program.

We use a constant relative risk aversion (CRRA) function as the utility function, where $v(a) = \ln(a)$ and its inverse function is $h(v) = e^v$. Given the biosecurity action b , let $p(b)$ denote the probability that the output level is q^H and $1 - p(b)$ stand for the probability that output level is q^L . Assume $p(b^H) = 3/4$ and $p(b^L) = 1/2$. Finally, variable $\rho \geq 0$ denotes the correlation between the two farmers' output levels.

Note that when assigning the above parameter values, we do not have a specific disease or region in mind. Our main purpose is to illustrate in general how the indemnity payments to different farmers could be solved from the model we specified. Our model and methods will serve as a framework where real life data for a specific disease could be applied when they become available.

Problem Setup

(i) Separate Contract (SC) in SB

SC refers to the contract between the government and one farmer. The model setup is similar to TOCP specified in the “Second-best Situation” section, except that only one participation constraint and incentive constraint are required.

To render the notations simple, we will use (x_H, x_L) to denote $(v(q^H), v(q^L))$.

Suppose the optimal biosecurity level is $\hat{b} = b^H$. The optimal SC can be written as:

$$\min_{x_H, x_L} \frac{3}{4}(e^{x_H} - q^H) + \frac{1}{4}(e^{x_L} - q^L)$$

s. t.

$$\begin{aligned} \frac{3}{4}x_H + \frac{1}{4}x_L - 1 &\geq 1 \\ \frac{3}{4}x_H + \frac{1}{4}x_L - 1 &\geq \frac{1}{2}x_H + \frac{1}{2}x_L - 0.3 \end{aligned}$$

(ii) Joint Contract (JC) in SB

JC refers to the contract between the government and two farmers. Before we proceed to set up the optimal JC problem, we will derive the probabilities that (q^H, q^H) , (q^H, q^L) , (q^L, q^H) and (q^L, q^L) occur in Table 1. Note that Dasgupta and Maskin (1987) also presented a similar result, which we found contained an error. The detailed derivation of Table 1 and an explanation of the error in Dasgupta and Maskin (1987) can be found in Appendix B.

Based on our assumption that $p(b^H) = 3/4$ and $p(b^L) = 1/2$, we can calculate the bounds on ρ in Table 2 for different combinations of biosecurity inputs. The detailed derivation is shown in Appendix B. Based on Table 2 we will specify the possible values of ρ , which takes the values between 0 and 0.5 at 0.1 increments. Here we will set up the optimal JC when $\rho = 0$. Suppose the optimal biosecurity levels are $(\hat{b}_1, \hat{b}_2) = (b_1^H, b_2^H)$. The optimal JC when the parameters take different values could be formulized similarly, so we will not cover them here.

From Table 1 we could calculate the discrete joint distribution function for different (q_1, q_2) when $\rho = 0$, as listed in Table 3. To simplify the notation, let:

$$\begin{aligned} (v_1(q_1^H, q_2^H), v_1(q_1^H, q_2^L), v_1(q_1^L, q_2^H), v_1(q_1^L, q_2^L)) &= (x_1, x_2, x_3, x_4) \\ (v_2(q_1^H, q_2^H), v_2(q_1^H, q_2^L), v_2(q_1^L, q_2^H), v_2(q_1^L, q_2^L)) &= (y_1, y_2, y_3, y_4) \end{aligned}$$

Under this specification the optimal JC could written according to TOCP in section 3:

$$\begin{aligned} \min_{x,y} \quad & \frac{9}{16}(e^{x_1} + e^{y_1} - q_1^H - q_2^H) + \frac{1}{16}(e^{x_2} + e^{y_2} - q_1^L - q_2^L) \\ & + \frac{3}{16}(e^{x_3} + e^{y_3} - q_1^H - q_2^L) + \frac{3}{16}(e^{x_4} + e^{y_4} - q_1^L - q_2^H) \end{aligned}$$

s. t.

$$\begin{aligned} \frac{9}{16}x_1 + \frac{1}{16}x_2 + \frac{3}{16}x_3 + \frac{3}{16}x_4 - 1 &\geq 1 \\ \frac{9}{16}y_1 + \frac{1}{16}y_2 + \frac{3}{16}y_3 + \frac{3}{16}y_4 - 1 &\geq 1 \\ \frac{9}{16}x_1 + \frac{1}{16}x_2 + \frac{3}{16}x_3 + \frac{3}{16}x_4 - 1 &\geq \frac{3}{8}x_1 + \frac{1}{8}x_2 + \frac{1}{8}x_3 + \frac{3}{8}x_4 - 0.3 \\ \frac{9}{16}y_1 + \frac{1}{16}y_2 + \frac{3}{16}y_3 + \frac{3}{16}y_4 - 1 &\geq \frac{3}{8}y_1 + \frac{1}{8}y_2 + \frac{3}{8}y_3 + \frac{1}{8}y_4 - 0.3 \end{aligned}$$

(iii) Contract in FB

The FB contract resembles that in part (ii), except that there is no incentive constraint.

Note that in FB case, there is no need to distinguish between SC and JC. As perfect risk sharing is already obtained in FB case, there is no room for Pareto improvement even by JC.

Solution and Discussion

Under the optimal contracting problems set up above, we have convex objective functions and concave inequality constraints. Thus we can apply the SAS Nonlinear Programming (NLP), which is now a convex program with a unique minimum solution.⁷ Or we can transform the problem into a concave programming problem. Instead of minimizing the convex objective function, we can maximize the negative of the objective function, which is concave. For the concave programming problem, the first order conditions alone can lead to the optimal solution of the problem. In a two variables setup, figure 1 below displays x^* as the unique maximal point in a concave programming program.

Here we use the SAS/IML (SAS 9.2) nonlinear optimization subroutine NLPNRR, which

⁷ SAS/OR 9.22 User's Guide: Mathematical Programming 2010, pp. 868.

implements a ridge-stabilized Newton-Raphson method and computes Gradient and Hessian using analytic formulas. Values of $v_i(\cdot)$ could be obtained directly by solving the optimization problem, then the indemnity payments could be calculated using our previous definition $I_i(\cdot) = h(v_i(\cdot)) - q_i$. Here negative solutions could be understood as a tax imposed by government. Due to our symmetric problem setup here we only list government indemnity payment to farmer 1 in Table 4, the solution for farmer 2 will be the same.

Table 4 displays government indemnity payments for different realized disease status under the joint contract (JC). As the other farm's disease information will not be utilized in the separate contract (SC), correlation between two farms' output level has no effect on the optimal indemnity payment under the SC. Therefore the expected indemnity payment is fixed at 3.89 in SC, which is equal to the expected indemnity payment in JC when the shocks are independent.

When there is a stronger correlation between shocks in the JC problem, the government is expected to pay less indemnity. We can see that the indemnity payment in JC decreases from 3.89 to 2.15 when the correlation increases from 0 to 0.5. The reason underlying this finding is that government can now better utilize the information conveyed by the other farmer's output (Hölmstrom 1979). Therefore it is cost minimizing for the government to design an optimal JC when the two farms' disease status are correlated with each other.

Table 4 also shows that the government pays the lowest indemnity value in FB case, where both SC and JC will incur the same payment as correlation size does not matter. The difference in indemnity payment between FB and SB is $2.15 - (-0.11) = 2.26$ if $\rho = 0.5$ and $3.89 - (-0.11) = 4$ if $\rho = 0$. Thus to resume FB situation by periodical auditing will be more justified when the farms' disease prevalence rates are less correlated.

Given the optimal indemnity scheme in Table 4, farmer 1's utilities at different

realizations of disease status are demonstrated in Figure 2. Farmer 2's utilities will be depicted similarly given our symmetry assumption. We can see that under the JC, farmer 1's utility is a non-decreasing function of his own output (Proposition 1) and a non-increasing function of farmer 2's output (Proposition 2). Moreover, at the point where $\rho = 0$ farmer 1's optimal indemnity payment does not depend on that of farmer 2's disease status. Thus SC is still optimal (Mookherjee 1984).

Effectiveness of the optimal biosecurity measure

We will refer the optimal biosecurity measure as more effective if it contributes to a lower within farm disease prevalence rate. In our two-output setup, a decreased disease prevalence rate is equivalent to increased probability for the high output. Our objective is to check how the indemnity payments and utilities received by farmers will change when the effectiveness of the optimal biosecurity level varies.

First we will fix $\rho = 0.3$ and choose different effectiveness indicators for optimal biosecurity measure. Here b^H is assumed to be the optimal biosecurity measure, the effectiveness of which can be adjusted by increasing $p(b^H)$ from 0.6 to 0.90 at increments of 0.05. Note that value 0.95 cannot be chosen since in that case one of the joint probabilities will be negative. However, $p(b^H) = 1$ can always be chosen whatever ρ is. The detailed reasoning is in Appendix B. If the effectiveness is equal to 1 ($p(b^H) = 1$), then the FB risk sharing rule is optimal, as we discussed in section 2.4.

The optimal indemnity scheme under the specified biosecurity effectiveness levels can be summarized in table 5. Table 5 indicates that a larger indemnity payment will be incurred if the optimal biosecurity measure becomes less effective. This result is quite intuitive in that farmers need a larger incentive, or larger utility variability, to adopt less effective biosecurity

measures (Figure 3). There will be an increased conflict between incentive constraints and farmers' risk aversion. As a result greater indemnity payment will be incurred. From Table 5 we also observe that for $q_1 = q^H, q^L$, difference $I_1(q_1, q^L) - I_1(q_1, q^H)$ decreases when the optimal biosecurity measure becomes more effective. FB will be resumed when $p(b^H) = 1$, in that case SC is optimal as we discussed in section 2.3, which means that farmer 1's indemnity is independent of the farmer 2's biosecurity effort.

The above findings indicate that a RPE indemnity scheme will be less justified when effectiveness of biosecurity measure is high. This is because farmer's performance will be more dependent on his own biosecurity investment rather than on the common environmental factors. This result is complementary to the finding that farmer 2's output plays a minor role in farmer 1's optimal indemnity payment if the correlation in shocks is small (Hölmstrom 1979, Luporini 2006 and Proposition 2).

Therefore both increased effectiveness and decreased correlation suggest decreased information value of the other farmers' output. If the biosecurity measure is highly effective, RPE may not improve the contract efficiency even when the correlation in shocks is high.

Reservation Utility Changes

Now we will analyze how reservation utility will affect the indemnity payments and utilities received by farmers at different realizations of the disease prevalence rate.

We will increase \underline{U} from 0.5 to 1.2 at increments of 0.1, while fixing the other parameters as $\rho = 0.3$, $p(b^H) = 0.75$ and $p(b^L) = 0.5$. The solutions for optimal gross utilities for farmer 1 are shown in figure 4. We can see from figure 4 that the gross utilities for different outputs will increase by the same proportion when the reservation utility increases. This is because only PCs are altered by the change in reservation utility, which

result in level changes on the gross utilities agent receive. No change will occur in utility variability while NICs remain the same.

For example, if farmers could still obtain high profit without disclosing their true disease status to some illegal traders, then the government needs to pay more to give farmers participation incentives to report the truth and join the eradication program. However, utility variability remains the same as the farmers need the same incentive to take the optimal biosecurity level.

CONCLUSION

This article has studied the optimal design of a government indemnity program taking both moral hazard and common uncertainty into account. Our results suggest that besides the within-farm disease prevalence, the average disease prevalence rate in the region should be incorporated into designing the optimal indemnity payment. The RPE is most justified under the following two scenarios. One is the case where the disease prevalence rates among farms are highly correlated, the other being that the optimal biosecurity investment is not very effective in curtailing the disease. The design of indemnity payment in reality should take many other factors into account as well. Such factors may include nature of disease (endemic, exotic or novel), farmer's risk category, biosecurity effectiveness and auditing cost, etc.

An important policy implication of the use of RPE is that the government could reduce its expenditure in indemnity payment without compromising the farmers' biosecurity incentives. Beyond government indemnity program, our payment scheme could shed insights on potential livestock insurance designs. As noted by Green, Driscoll, and Bruch (2006), the adequacy of data is essential in determining the optimal indemnity payment. Our conceptual model suggests that development in data collection could be made in areas regarding disease prevalence correlation and biosecurity effectiveness in disease prevention. Experiences from

other industries such as the Group Risk Plan offered by FCIC could also be useful when designing and implementing the optimal indemnity scheme in practice.

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APPENDIX A

Proof of Lemma 1. To prove Lemma 1 is equivalent to show that $\partial(q_i + I_i(q_1, q_2)) / \partial q_i \geq 0$ is

the necessary and sufficient condition for $\partial h'(v_i(q_1, q_2)) / \partial q_i \geq 0$.

$$\begin{aligned} & \frac{\partial(q_i + I_i(q_i, q_j))}{\partial q_i} \geq 0 \\ \Leftrightarrow & V'(q_i + I_i(q_i, q_j)) \frac{\partial(q_i + I_i(q_1, q_2))}{\partial q_i} \geq 0 \quad (\text{By } V'(\cdot) \geq 0) \\ \Leftrightarrow & \frac{\partial V(q_i + I_i(q_i, q_j))}{\partial q_i} \geq 0 \\ \Leftrightarrow & h''(V(q_i + I_i(q_i, q_j))) \frac{\partial(V(q_i + I_i(q_i, q_j)))}{\partial q_i} \geq 0 \quad (\text{By } h''(\cdot) \geq 0) \\ \Leftrightarrow & \frac{\partial h'(V(q_i + I_i(q_i, q_j)))}{\partial q_i} \geq 0 \\ \Leftrightarrow & \frac{\partial h'(v_i(q_i, q_j))}{\partial q_i} \geq 0 \end{aligned}$$

Note: $h(\cdot) = V^{-1}(\cdot)$ is convex since $V(\cdot)$ is concave, therefore $h''(\cdot) \geq 0$. \square

Proof of Lemma 2. First we have:

$$\begin{aligned} F(q_1, q_2; b_1, b_2) &= F(q_1(b_1, \theta_1) \leq q_1, q_2(b_2, \theta_2) \leq q_2) \\ &= F(\theta_1 \leq q_1^{-1}(q_1, b_1), \theta_2 \leq q_2^{-1}(q_2, b_2)) \\ &= G(q_1^{-1}(q_1, b_1), q_2^{-1}(q_2, b_2)) \end{aligned} \tag{A-1}$$

Differentiating (A-1) with regard to q_1 and q_2 generates:

$$f(q_1, q_2; b_1, b_2) = g(q_1^{-1}(q_1, b_1), q_2^{-1}(q_2, b_2)) \frac{\partial q_1^{-1}(q_1, b_1)}{\partial q_1} \frac{\partial q_2^{-1}(q_2, b_2)}{\partial q_2} \quad (\text{A-2})$$

It follows immediately from (A-2) that:

$$f(q_1, q_2; \hat{b}_1, \hat{b}_2) = g(q_1^{-1}(q_1, \hat{b}_1), q_2^{-1}(q_2, \hat{b}_2)) \frac{\partial q_1^{-1}(q_1, \hat{b}_1)}{\partial q_1} \frac{\partial q_2^{-1}(q_2, \hat{b}_2)}{\partial q_2}$$

Similar to (A-2), we can write:

$$m(q_1; \hat{b}_1) = g_1(q_1^{-1}(q_1, \hat{b}_1)) \frac{\partial q_1^{-1}(q_1, \hat{b}_1)}{\partial q_1}; \quad n(q_2; \hat{b}_2) = g_2(q_2^{-1}(q_2, \hat{b}_2)) \frac{\partial q_2^{-1}(q_2, \hat{b}_2)}{\partial q_2}$$

Next we will prove the equivalence of the two conditions as follows.

$$\begin{aligned} f(q_1, q_2; \hat{b}_1, \hat{b}_2) &= m(q_1; \hat{b}_1) n(q_2; \hat{b}_2) \\ \Leftrightarrow g(q_1^{-1}(q_1, \hat{b}_1), q_2^{-1}(q_2, \hat{b}_2)) \frac{\partial q_1^{-1}(q_1, \hat{b}_1)}{\partial q_1} \frac{\partial q_2^{-1}(q_2, \hat{b}_2)}{\partial q_2} \\ &= g_1(q_1^{-1}(q_1, \hat{b}_1)) \frac{\partial q_1^{-1}(q_1, \hat{b}_1)}{\partial q_1} g_2(q_2^{-1}(q_2, \hat{b}_2)) \frac{\partial q_2^{-1}(q_2, \hat{b}_2)}{\partial q_2} \\ \Leftrightarrow g(q_1^{-1}(q_1, \hat{b}_1), q_2^{-1}(q_2, \hat{b}_2)) &= g_1(q_1^{-1}(q_1, \hat{b}_1)) g_2(q_2^{-1}(q_2, \hat{b}_2)) \\ \Leftrightarrow g(\theta_1, \theta_2) &= g_1(\theta_1) g_2(\theta_2) \end{aligned}$$

Note that $\theta_1 = q_1^{-1}(q_1, \hat{b}_1)$ and $\theta_2 = q_2^{-1}(q_2, \hat{b}_2)$. \square

Proof of Proposition 1. When q_i increases, $f(q_i, q_j; b_i^-, b_j) / f(q_i, q_j; b_i^+, b_j)$ is non-increasing by assumption 1. Thus the RHS of equation (7) is non-decreasing. So we can infer that $h'(v_1(q_1, q_2))$ is non-decreasing. By lemma 1, we know that $q_i + I_i(q_i, q_j)$ is a non-decreasing. \square

Proof of Lemma 3.

$$\frac{f(q_i, q_j^-; b_i^-, b_j)}{f(q_i, q_j^-; b_i^+, b_j)} - \frac{f(q_i, q_j^+; b_i^-, b_j)}{f(q_i, q_j^+; b_i^+, b_j)} \leq 0$$

$$\begin{aligned}
& \frac{g(q_i^{-1}(q_i, b_i^-), q_j^{-1}(q_j^-, b_j)) \frac{\partial q_i^{-1}(q_i, b_i^-)}{\partial q_i} \frac{\partial q_j^{-1}(q_j^-, b_j)}{\partial q_j}}{g(q_i^{-1}(q_i, b_i^+), q_j^{-1}(q_j^-, b_j)) \frac{\partial q_i^{-1}(q_i, b_i^+)}{\partial q_i} \frac{\partial q_j^{-1}(q_j^-, b_j)}{\partial q_j}} \\
& - \frac{g(q_i^{-1}(q_i, b_i^-), q_j^{-1}(q_j^+, b_j)) \frac{\partial q_i^{-1}(q_i, b_i^-)}{\partial q_i} \frac{\partial q_j^{-1}(q_j^+, b_j)}{\partial q_j}}{g(q_i^{-1}(q_i, b_i^+), q_j^{-1}(q_j^+, b_j)) \frac{\partial q_i^{-1}(q_i, b_i^+)}{\partial q_i} \frac{\partial q_j^{-1}(q_j^+, b_j)}{\partial q_j}} \leq 0 \quad (\text{By (A-2)})
\end{aligned}$$

$$\begin{aligned}
& \frac{\frac{\partial q_i^{-1}(q_i, b_i^-)}{\partial q_i}}{\frac{\partial q_i^{-1}(q_i, b_i^+)}{\partial q_i}} \left(\frac{g(q_i^{-1}(q_i, b_i^-), q_j^{-1}(q_j^-, b_j))}{g(q_i^{-1}(q_i, b_i^+), q_j^{-1}(q_j^-, b_j))} - \frac{g(q_i^{-1}(q_i, b_i^-), q_j^{-1}(q_j^+, b_j))}{g(q_i^{-1}(q_i, b_i^+), q_j^{-1}(q_j^+, b_j))} \right) \leq 0 \quad (\text{A-3})
\end{aligned}$$

$$\begin{aligned}
& \frac{\frac{\partial q_i^{-1}(q_i, b_i^-)}{\partial q_i}}{\frac{\partial q_i^{-1}(q_i, b_i^+)}{\partial q_i}} \left(\frac{g(\theta_i^+, \theta_j^-)}{g(\theta_i^-, \theta_j^-)} - \frac{g(\theta_i^+, \theta_j^+)}{g(\theta_i^-, \theta_j^+)} \right) \leq 0 \quad (\text{A-4})
\end{aligned}$$

$$\begin{aligned}
& \frac{g(\theta_i^+, \theta_j^-)}{g(\theta_i^-, \theta_j^-)} - \frac{g(\theta_i^+, \theta_j^+)}{g(\theta_i^-, \theta_j^+)} \leq 0 \quad (\text{A-5})
\end{aligned}$$

Note that in proving the equivalence between (A-3) and (A-4) we have assumed:

$$\begin{aligned}
q_i^{-1}(q_i, b_i^+) &\equiv \theta_i^- \leq \theta_i^+ \equiv q_i^{-1}(q_i, b_i^-); \\
q_j^{-1}(q_j^-, b_j) &\equiv \theta_j^- \leq \theta_j^+ \equiv q_j^{-1}(q_j^+, b_j).
\end{aligned} \quad (\text{A-6})$$

Assumption (A-6) is valid since:

$$\frac{\partial q_i^{-1}(q_i, b_i)}{\partial b_i} \leq 0; \quad (\text{A-7})$$

$$\frac{\partial q_i^{-1}(q_i, b_i)}{\partial q_i} \geq 0. \quad (\text{A-8})$$

In addition, note that the equivalence between (A-4) and (A-5) are established by (A-7).

The proofs for (A-7) and (A-8) is as follows:

First we have $q_i = q_i(b_i, q_i^{-1}(q_i, b_i))$, as $\theta_i = q_i^{-1}(q_i, b_i)$ from $q_i = q_i(b_i, \theta_i)$ ⁸. Take derivatives w.r.t. b_i and q_i on both sides:

$$0 = \frac{\partial q_i}{\partial b_i} + \frac{\partial q_i}{\partial \theta_i} \frac{\partial q_i^{-1}(q_i, b_i)}{\partial b_i}; \quad 1 = \frac{\partial q_i}{\partial \theta_i} \frac{\partial q_i^{-1}(q_i, b_i)}{\partial q_i}.$$

It follows that:

$$\frac{\partial q_i^{-1}(q_i, b_i)}{\partial b_i} = -\frac{\partial q_i}{\partial b_i} / \frac{\partial q_i}{\partial \theta_i} \leq 0; \quad \frac{\partial q_i^{-1}(q_i, b_i)}{\partial q_i} = 1 / \frac{\partial q_i}{\partial \theta_i} \geq 0. \quad \square$$

Proof of Proposition 3.

Denote FB indemnity payment to farmer 1 as $I_1(q_1, q_2)$ and SB indemnity payment to farmer 1 as $I_1^1(q_1, q_2)$.

From the above analysis, to achieve biosecurity input \hat{b}_1 in FB case, we have:

$$V(q_1 + I_1^*(q_1, q_2)) - \hat{b}_1 w = \underline{U} \quad (\text{A-9})$$

In the SB case, we have:

$$\int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} V(q_1 + I_1(q_1, q_2)) f(q_1, q_2; \hat{b}_1, \hat{b}_2) dq_1 dq_2 - \hat{b}_1 w = \underline{U} \quad (\text{A-10})$$

Combining (A-9) and (A-10) we can obtain:

$$\int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} V(q_1 + I_1(q_1, q_2)) f(q_1, q_2; \hat{b}_1, \hat{b}_2) dq_1 dq_2 = V(q_1 + I_1^*(q_1, q_2)) \quad (\text{A-11})$$

As the inverse function of the concave function $V(\cdot)$ is still concave, we have by Jensen's inequality:

⁸ Note q_i on the LHS is a single variable, while q_i on the RHS is a function w.r.t. b_i and θ_i .

$$\begin{aligned} & \int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} V^{-1}[V(q_1 + I_1(q_1, q_2))]f(q_1, q_2; \hat{b}_1, \hat{b}_2) dq_1 dq_2 \\ & \geq V^{-1} \left[\int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} V(q_1 + I_1(q_1, q_2))f(q_1, q_2; \hat{b}_1, \hat{b}_2) dq_1 dq_2 \right] \end{aligned} \quad (\text{A-12})$$

From (A-11) and (A-12), we have:

$$\begin{aligned} & \int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} V^{-1}[V(q_1 + I_1(q_1, q_2))]f(q_1, q_2; \hat{b}_1, \hat{b}_2) dq_1 dq_2 \geq V^{-1}[V(q_1 + I_1^*(q_1, q_2))] \\ \Leftrightarrow & \int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} [q_1 + I_1(q_1, q_2)]f(q_1, q_2; \hat{b}_1, \hat{b}_2) dq_1 dq_2 \geq q_1 + I_1^*(q_1, q_2) \\ \Leftrightarrow & \int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} [q_1 + I_1(q_1, q_2)]f(q_1, q_2; \hat{b}_1, \hat{b}_2) dq_1 dq_2 \geq \int_{\underline{q}}^{\bar{q}} \int_{\underline{q}}^{\bar{q}} [q_1 + I_1^*(q_1, q_2)]f(q_1, q_2; \hat{b}_1, \hat{b}_2) dq_1 dq_2 \\ \Leftrightarrow & E[I_1(q_1, q_2)] \geq E[I_1^*(q_1, q_2)] \end{aligned}$$

APPENDIX B

Derivation of Table 1

There are only two possible outcomes (S, F) . For $i = 1, 2$, use $X_i(\cdot)$ to denote the payoff function of the outcomes, where $X_i(S) = m$ and $X_i(F) = n$. For simplicity, denote $Prob(S | a_i) = p(a_i)$ and $Prob(F | a_i) = 1 - p(a_i)$. When agent i takes action a_i , Table B1 describes the joint probability distribution when the correlation between X_1 and X_2 is ρ .

Based on Table B1, we know:

$$\begin{aligned} \beta &= p(a_1) - \alpha \\ \gamma &= p(a_2) - \alpha \\ \delta &= 1 - p(a_2) - \beta = 1 - p(a_2) - p(a_1) + \alpha \end{aligned} \quad (\text{B-1})$$

Thus to obtain Table B1 the only value we need to solve is α . For $i = 1, 2$:

$$\begin{aligned} E(X_i) &= mp(a_i) + n(1 - p(a_i)); \\ E(X_i^2) &= m^2 p(a_i) + n^2 (1 - p(a_i)); \\ E(X_1 X_2) &= m^2 \alpha + n^2 \delta + mn(\beta + \gamma); \end{aligned} \quad (\text{B-2})$$

$$\begin{aligned}
\text{Var}(X_i) &= E(X_i^2) - [E(X_i)]^2 \\
&= m^2 p(a_i) + n^2 (1 - p(a_i)) - [mp(a_i) + n(1 - p(a_i))]^2 \\
&= m^2 p(a_i) + n^2 (1 - p(a_i)) - m^2 [p(a_i)]^2 - n^2 (1 - p(a_i))^2 - 2mnp(a_i)(1 - p(a_i)) \\
&= m^2 [p(a_i) - p(a_i)^2] + n^2 [(1 - p(a_i)) - (1 - p(a_i))^2] - 2mnp(a_i)(1 - p(a_i)) \\
&= (m^2 + n^2) p(a_i)(1 - p(a_i)) - 2mnp(a_i)(1 - p(a_i)) \\
&= (m - n)^2 p(a_i)(1 - p(a_i));
\end{aligned} \tag{B-3}$$

And finally:

$$\begin{aligned}
\text{Cov}(X_1 X_2) &= E(X_1 X_2) - E(X_1)E(X_2) \\
&= m^2 \alpha + n^2 \delta + mn(\beta + \gamma) - [mp(a_1) + n(1 - p(a_1))][mp(a_2) + n(1 - p(a_2))] \\
&= m^2 \alpha + n^2 \delta + mn(\beta + \gamma) - m^2 p(a_1)p(a_2) - n^2 (1 - p(a_1))(1 - p(a_2)) \\
&\quad - mn[p(a_1)(1 - p(a_2)) + p(a_2)(1 - p(a_1))] \\
&= m^2 (\alpha - p(a_1)p(a_2)) + n^2 [\delta - (1 - p(a_1))(1 - p(a_2))] \\
&\quad + mn[\beta + \gamma - p(a_1)(1 - p(a_2)) - p(a_2)(1 - p(a_1))].
\end{aligned} \tag{B-4}$$

From (B-1), we could solve (B-4) by part:

$$\begin{aligned}
&\delta - (1 - p(a_1))(1 - p(a_2)) \\
&= 1 - p(a_2) - p(a_1) + \alpha - (1 - p(a_1))(1 - p(a_2)) \\
&= \alpha - p(a_1)p(a_2)
\end{aligned} \tag{B-5}$$

$$\begin{aligned}
&\beta + \gamma - p(a_1)(1 - p(a_2)) - p(a_2)(1 - p(a_1)) \\
&= p(a_1) - \alpha + p(a_2) - \alpha - p(a_1) + p(a_1)p(a_2) - p(a_2) + p(a_1)p(a_2) \\
&= -2(\alpha - p(a_1)p(a_2))
\end{aligned} \tag{B-6}$$

By substituting results of (B-5) and (B-6) into (B-4), we can get:

$$\text{Cov}(X_1 X_2) = (m^2 + n^2 - 2mn)(\alpha - p(a_1)p(a_2)) = (m - n)^2 (\alpha - p(a_1)p(a_2)) \tag{B-7}$$

It follows from (B-3) and (B-7) that:

$$\begin{aligned}
\text{corr}(X_1 X_2) &= \frac{\text{Cov}(X_1 X_2)}{\sqrt{\text{var}(X_1)}\sqrt{\text{var}(X_2)}} \\
&= \frac{(m - n)^2 (\alpha - p(a_1)p(a_2))}{\sqrt{(m - n)^2 p(a_1)(1 - p(a_1))}\sqrt{(m - n)^2 p(a_2)(1 - p(a_2))}} \\
&= \frac{\alpha - p(a_1)p(a_2)}{\sqrt{p(a_1)(1 - p(a_1))}\sqrt{p(a_2)(1 - p(a_2))}} = \rho
\end{aligned} \tag{B-8}$$

From (B-8), we can solve α as:

$$\alpha = p(a_1)p(a_2) + \rho\sqrt{p(a_1)(1-p(a_1))}\sqrt{p(a_2)(1-p(a_2))} \quad (\text{B-9})$$

The other variables of Table 1 could be easily solved from (B-1), based on the value of α .

An Error of Dasgupta and Maskin (1987)

$$\begin{aligned} \alpha &= p(a_1)p(a_2) + (1-a_1+a_2)\sqrt{p(a_1)(1-p(a_1))}\sqrt{p(a_2)(1-p(a_2))} \\ &= (a_1-a_2)p(a_1)p(a_2) + (1-a_1+a_2)p(a_1)p(a_2) + (1-a_1+a_2)\sqrt{p(a_1)(1-p(a_1))}\sqrt{p(a_2)(1-p(a_2))} \\ &= (a_1-a_2)p(a_1)p(a_2) + (1-a_1+a_2)[p(a_1)p(a_2) + \sqrt{p(a_1)(1-p(a_2))p(a_2)(1-p(a_1))}] \\ &\leq (a_1-a_2)p(a_1)p(a_2) + (1-a_1+a_2)[p(a_1)p(a_2) + \frac{p(a_1)(1-p(a_2)) + p(a_2)(1-p(a_1))}{2}] \\ &= (a_1-a_2)p(a_1)p(a_2) + (1-a_1+a_2)\frac{p(a_1) + p(a_2)}{2} \\ &= \text{Prob}(S,S) \quad (\text{Dasgupta and Maskin (1987)}) \end{aligned}$$

The equality in the third to last step holds if and only if $p(a_1)(1-p(a_2)) = p(a_2)(1-p(a_1))$, which is equivalent to $p(a_1) = p(a_2)$. Thus we can conclude in table 1 of Dasgupta and Maskin (1987), probability of outcome (S,S) is generally overestimated, similarly is (F, F). However, probability of outcomes (S,F) and (F,S) are generally underestimated.

Derivation of Table 2 (Bounds of ρ)

Given the marginal distributions $p(a_1)$, $p(a_2)$ and the correlation coefficient ρ , we should be able to work out the joint distribution in table 1 from equations (B-1) and (B-9). However, in order to have the joint distribution well defined, i.e. $\alpha, \beta, \delta, \gamma \in [0,1]$, generally the range of ρ is no longer $[-1,1]$. Therefore it is useful to define a tighter bound on ρ as

functions of $p(a_1)$ and $p(a_2)$.

Given $p(a_1)$ and $p(a_2)$, the joint distribution is well defined if and only if:

$$\max\{0, p(a_1) + p(a_2) - 1\} \leq \alpha \leq \min\{p(a_1), p(a_2)\} \quad (\text{B-10})$$

Inequality (B-10) is known as Fréchet-Hoeffding bounds inequality, in recognition of the pioneering work in this field by Hoeffding (1940, 1941) and Fréchet (1951). In our case, it is obtained by (B-1) and $0 \leq \alpha, \beta, \delta, \gamma \leq 1$. Again by (B-9), (B-10) holds if and only if the following inequalities are satisfied:

$$\begin{aligned} p(a_1)p(a_2) + \rho\sqrt{p(a_1)(1-p(a_1))}\sqrt{p(a_2)(1-p(a_2))} &\leq \min\{p(a_1), p(a_2)\} \\ p(a_1)p(a_2) + \rho\sqrt{p(a_1)(1-p(a_1))}\sqrt{p(a_2)(1-p(a_2))} &\geq \max\{0, p(a_1) + p(a_2) - 1\} \end{aligned} \quad (\text{B-11})$$

It follows that the joint probabilities are well defined if and only if ρ is chosen in a such way that inequality set (B-11) are satisfied. Thus (B-11) implicitly defines the upper and lower bounds of ρ . Note that when $p(a_i) = 0$ or $p(a_i) = 1$, inequality set (B-11) are satisfied for any values of $\rho \in [0,1]$. If $p(a_i) \neq 0$ and $p(a_i) \neq 1$, then we will discuss the bounds of ρ as follows.

Upper bounds of ρ

(i) If $p(a_1) \leq p(a_2)$, then (B-11) becomes:

$$p(a_1)p(a_2) + \rho\sqrt{p(a_1)(1-p(a_1))}\sqrt{p(a_2)(1-p(a_2))} \leq p(a_1) \quad (\text{B-12})$$

We can further solve the bounds of ρ from (B-12) as:

$$\begin{aligned} \rho &\leq \frac{p(a_1) - p(a_1)p(a_2)}{\sqrt{p(a_1)(1-p(a_1))}\sqrt{p(a_2)(1-p(a_2))}} \\ &= \frac{p(a_1)(1-p(a_2))}{\sqrt{p(a_1)(1-p(a_1))}\sqrt{p(a_2)(1-p(a_2))}} \\ &= \left[\frac{p(a_1)(1-p(a_2))}{p(a_2)(1-p(a_1))} \right]^{\frac{1}{2}} \end{aligned} \quad (\text{B-13})$$

(ii) If $p(a_1) \geq p(a_2)$, we can derive the results similarly. The only change is to exchange the role of $p(a_1)$ and $p(a_2)$.

$$\rho \leq \left[\frac{p(a_2)(1-p(a_1))}{p(a_1)(1-p(a_2))} \right]^{\frac{1}{2}} \quad (\text{B-14})$$

Lower bounds of ρ

(i) If $p(a_1) + p(a_2) - 1 \geq 0$, then the second inequality of (B-11) becomes:

$$p(a_1)p(a_2) + \rho\sqrt{p(a_1)(1-p(a_1))}\sqrt{p(a_2)(1-p(a_2))} \geq p(a_1) + p(a_2) - 1 \quad (\text{B-15})$$

We can further solve (B-15) as:

$$\begin{aligned} \rho &\geq \frac{p(a_1) + p(a_2) - 1 - p(a_1)p(a_2)}{\sqrt{p(a_1)(1-p(a_1))}\sqrt{p(a_2)(1-p(a_2))}} \\ &= -\frac{(1-p(a_1))(1-p(a_2))}{\sqrt{p(a_1)(1-p(a_1))}\sqrt{p(a_2)(1-p(a_2))}} \\ &= -\left[\frac{(1-p(a_1))(1-p(a_2))}{p(a_1)p(a_2)} \right]^{\frac{1}{2}} \end{aligned} \quad (\text{B-16})$$

(ii) If $p(a_1) + p(a_2) - 1 \leq 0$, then the second inequality of (B-11) becomes:

$$p(a_1)p(a_2) + \rho\sqrt{p(a_1)(1-p(a_1))}\sqrt{p(a_2)(1-p(a_2))} \geq 0 \quad (\text{B-17})$$

We can further solve (B-17) as:

$$\begin{aligned} \rho &\geq -\frac{p(a_1)p(a_2)}{\sqrt{p(a_1)(1-p(a_1))}\sqrt{p(a_2)(1-p(a_2))}} \\ &= -\left[\frac{p(a_1)p(a_2)}{(1-p(a_1))(1-p(a_2))} \right]^{\frac{1}{2}} \end{aligned} \quad (\text{B-18})$$

Table 2.1: Joint Probability Distribution Computation Formula

	q^H	q^L
q^H	α	$p(b_1) - \alpha$
q^L	$p(b_2) - \alpha$	$1 - p(b_2) - p(b_1) + \alpha$

Note: $\alpha = p(b_1)p(b_2) + \rho\sqrt{p(b_1)(1-p(b_1))}\sqrt{p(b_2)(1-p(b_2))}$.

Table 2.2: Bounds on ρ

	b^H	b^L
b^H	$[-1/3, 1]$	$[-\sqrt{1/3}, \sqrt{1/3}]$
b^L	$[-\sqrt{1/3}, \sqrt{1/3}]$	$[-1, 1]$

Table 2.3: Discrete Joint Probability Distribution for (q_1, q_2) when $\rho = 0$.

	(q^H, q^H)	(q^H, q^L)	(q^L, q^H)	(q^L, q^L)
(b^H, b^H)	9/16	3/16	3/16	1/16
(b^H, b^L)	3/8	3/8	1/8	1/8
(b^L, b^H)	3/8	1/8	3/8	1/8
(b^L, b^L)	1/4	1/4	1/4	1/4

Table 2.4: JC Indemnity Payments Varying with Correlations

correlation	$I_1(q^H, q^H)$	$I_1(q^H, q^L)$	$I_1(q^L, q^H)$	$I_1(q^L, q^L)$	Mean
0	4.88	4.88	0.9	0.9	3.89
0.1	4.3	4.98	0.55	3.36	3.71
0.2	3.49	4.81	0.43	4.99	3.38
0.3	2.65	4.54	0.36	6.03	3
0.4	1.77	4.16	0.3	6.94	2.59
0.5	1	3.84	0.26	7.15	2.15
FB	-2.61	-2.61	7.39	7.39	-0.11

Table 2.5: JC Indemnity Payments Varying with Biosecurity Effectiveness

$p(b^H)$	$I_1(q^H, q^H)$	$I_1(q^H, q^L)$	$I_1(q^L, q^H)$	$I_1(q^L, q^L)$	Mean
0.6	48.47	61.93	0	20.49	36.1
0.65	15.27	20.37	0.03	9.66	12.58
0.7	6.46	9.34	0.15	6.97	6.03
0.75	2.65	4.54	0.36	6.03	3
0.8	0.58	1.89	0.62	5.74	1.18
0.85	-0.7	0.22	0.89	5.76	-0.09
0.9	-1.56	-0.93	1.17	5.98	-1.07

Table 2.B1: Joint Probability Distribution

$X_1 \backslash X_2$	S	F	
S	α	β	$p(a_1)$
F	γ	δ	$1 - p(a_1)$
	$p(a_2)$	$1 - p(a_2)$	

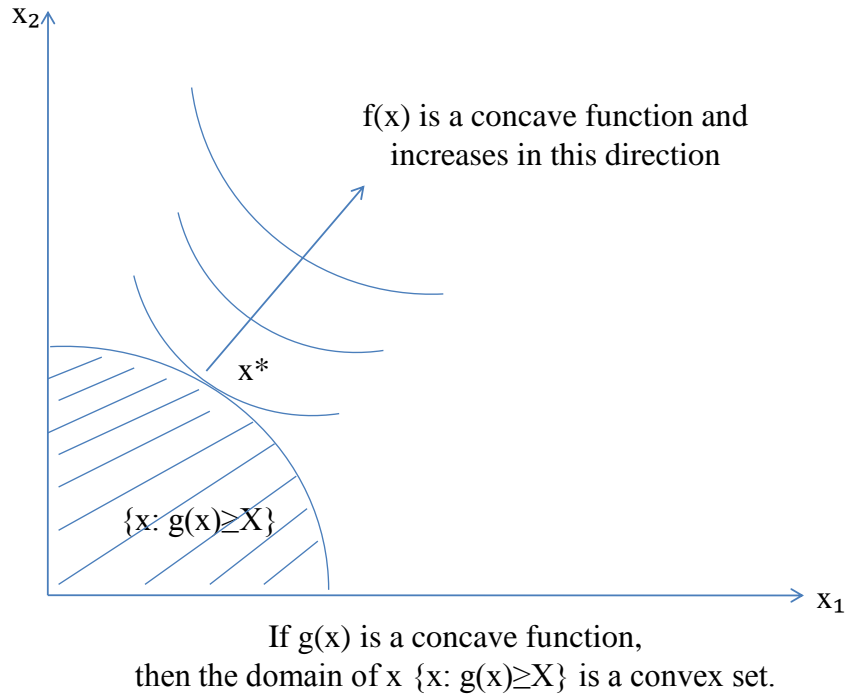


Figure 2.1: Uniqueness of Solution

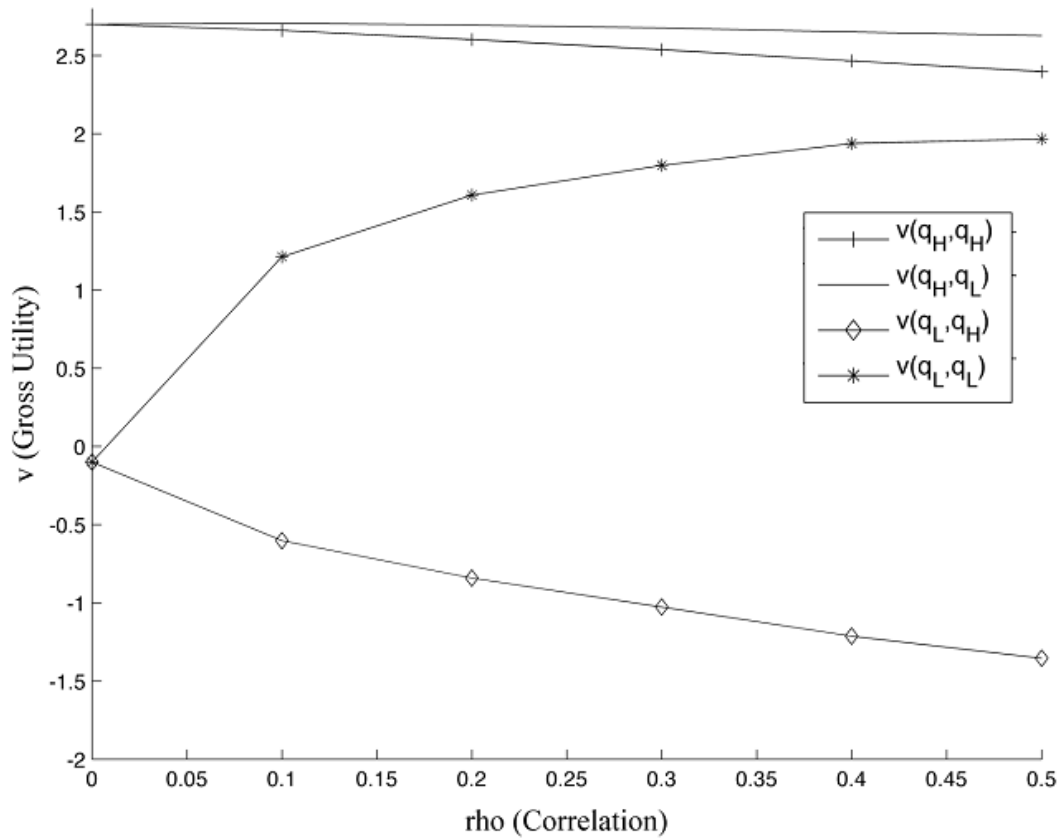


Figure 2.2: Utility Variability under Different Correlation Levels

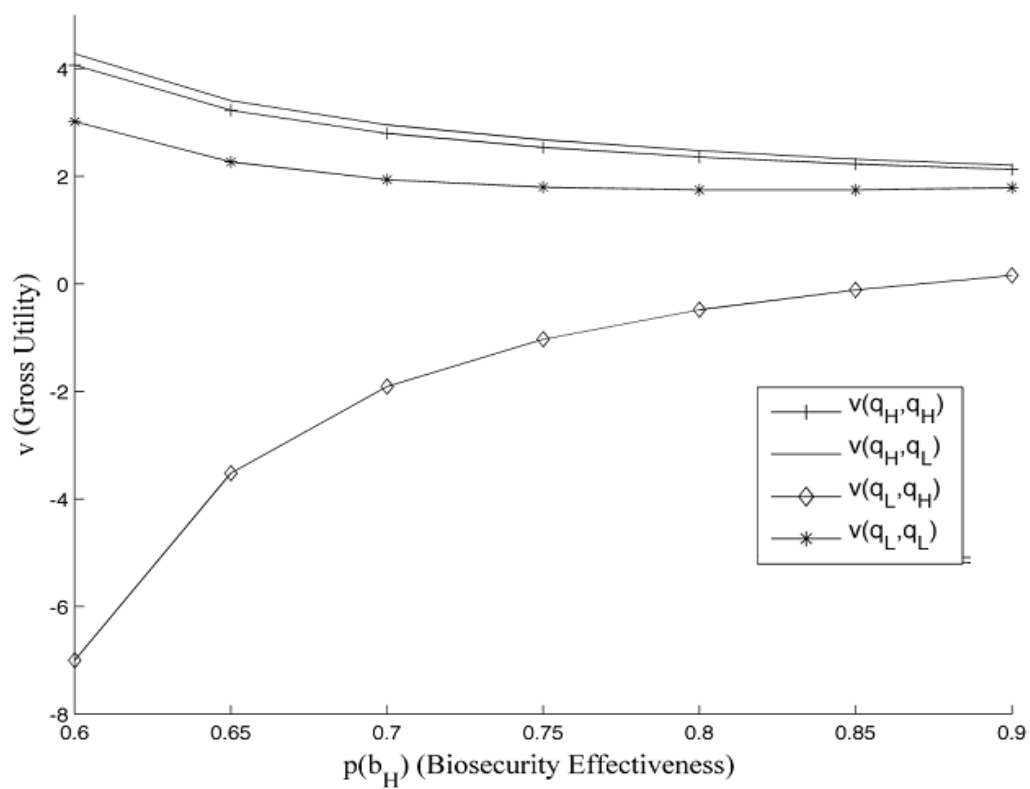


Figure 2.3: Utility Variability under Different Biosecurity Effectiveness Levels

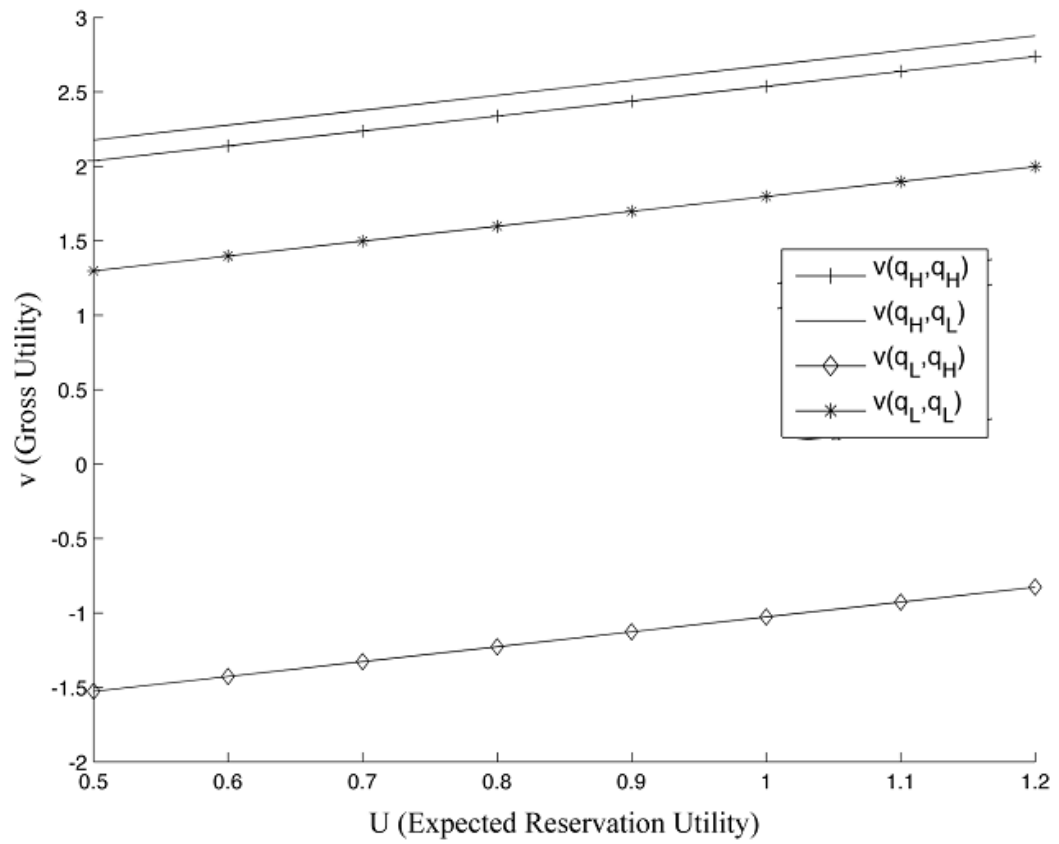


Figure 2.4: Utility Variability under Different Reservation Utilities

Chapter 3: Where are the Food Animal Veterinarian Shortage Areas Anyway?

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ABSTRACT

In 2010 the United States implemented the Veterinary Medicine Loan Repayment Program (VMLRP) to address perceived regional shortages in certain veterinary occupations, including food animal practice. With county level as the unit of analysis, this paper describes a pair of models to evaluate factors associated with being designated a private practice shortage area in 2010. One model is used to explain food animal veterinarian location choices so as to provide an objective evaluation of comparative shortage. The other model seeks to explain the counties chosen as shortage areas. Model results are then used to evaluate the program. On the whole the program appears to perform quite well. For several states, however, VMLRP shortage designations are inconsistent with the food animal veterinarian location model. Comparative shortage is generally more severe in states that have no VMLRP designated private practice shortage counties than in states that do.

INTRODUCTION

As of 2011, American Veterinary Medical Association (AVMA) state legislative resources at http://www.avma.org/advocacy/state/loan_repayment_programs/default.asp report twenty U.S. state veterinary loan programs. All but one of these programs specifically target farm animal, large animal or rural practices.

Pursuant to the National Veterinary Medical Service Act of 2003, the Veterinary Medicine Loan Repayment Program (VMLRP) became law as U.S. Code Title 7, Section 3151a. The program was implemented at the federal level in 2010. For qualified veterinarians

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who agree to serve in certain high-priority veterinary shortage areas for a period of three years, the U.S. Department of Agriculture's (USDA) National Institute of Food and Agriculture (NIFA) will repay a maximum of \$25,000 of student loans per year.

As amended under Section 7105 of the 2008 Farm Bill, when implementing the VMLRP the Secretary for Agriculture may consider “(1) geographical areas that the Secretary determines have a shortage of veterinarians”; and “(2) areas of veterinary practice that the Secretary determines have a shortage of veterinarians, such as food animal medicine, public health, epidemiology, and food safety” when identifying ‘veterinarian shortage situations.’ In addition, “In administering the program, the Secretary shall give priority to agreements with veterinarians for the practice of food animal medicine in veterinarian shortage situations.”

Under the VMLRP, private veterinary shortage areas are determined according to the following steps: 1) “NIFA will release a Federal Register (FR) notice soliciting nominations for veterinary shortage situations from all State Animal Health Officials”; 2) “State Animal Health Officers will prepare nominations corresponding to the highest priority veterinary shortage situations within their entities and then submit completed nomination forms by email to NIFA”; 3) “A review panel composed of Federal and State animal health experts will be convened by NIFA to evaluate the submitted nomination packages.” Then “Final decisions regarding recommendation status will be made by the NIFA Program Manager, on behalf of the Secretary of Agriculture. Designated shortage situations will be made accessible to the public in list and/or map form.” See

http://www.nifa.usda.gov/nea/animals/in_focus/vmlrp_11/vmlrp_shortage_situation_usmap.html (last visited 8/18/2011).

The issue of whether the United States has a food animal veterinarian (FAV) shortage has been addressed previously, with diverging conclusions. Some argue that there is an excess supply (Getz, 1997; Brown and Silverman, 1999). Others suggest that a shortage exists and

will persist through the near future (Prince *et al.*, 2006; Narver, 2007). Our inquiry does not seek to establish whether the United States has an absolute shortage in the sector, but rather where comparative shortages are and how effective the 2010 VMLRP implementation was in targeting them.

NIFA does publish evaluation criteria and a scoring algorithm for the VMLRP, see http://www.nifa.usda.gov/nea/animals/in_focus/an_health_if_vmlrp_nomination_and_designation_of_veterinary_shortage_situations.html#review (visited 8/18/2011). But the case made for each nominated area will inevitably be unique and no specific quantifiable data were required of all nominations. An obvious question then is: What basis did NIFA use in the process? The primary objective of our study is to assess the extent of agreement between the NIFA nominated areas and other measures of FAV coverage.

Our approach to meeting this objective is to test the main working hypothesis that there is an association between regions that have a comparatively low FAV presence given the number of animals present and regions designated as shortage areas under the VMLRP. We do so by building two models where one seeks to identify factors that determine FAV presence in a county and the other seeks to explain designation status under the VMLRP in 2010. We will then assess whether our estimate of shortage, upon accounting for FAV determining factors, is consistent with VMLRP designation.

MATERIALS AND METHODS

Data collection and management

For the analysis the unit of concern was the county in the United States' 48 contiguous states. Alaska, Hawaii and unincorporated territories were excluded in large part because their extreme geographic features would distort findings. For all 3,120 counties or equivalents the following data were collected.

Data on whether a county was designated a private veterinary practice shortage situation

were obtained at NIFA Web site

http://www.nifa.usda.gov/nea/animals/in_focus/vmlrp/vmlrp_shortage_situation_usmap.html

(visited 8/18/2011). Ten of the 48 states do not have any designated shortage counties, either because “no shortage situation nominations were submitted,” or “nominations were submitted, but the external review panel did not recommend them for official designation.” These are Alabama, Connecticut, Georgia, Massachusetts, Mississippi, Nevada, New Jersey, Tennessee, Washington and Wyoming (Figure 1). NIFA staff have communicated to us (July 10, 2010) that 181 of the 249 nominations received were recommended. Further details on the rejected nominations were considered confidential, and were not available.

The number of FAVs in a county was obtained from the AVMA website <http://www.avma.org/fsvm/maps/default.asp> (visited 8/18/2011), which uses data extracted from the AVMA membership database. These data are compiled by the AVMA from member and non-member information. An e-mail received from the AVMA (3/11/2011) clarifies that veterinary students are provided with a year’s membership upon graduation, and employment classification data are collected at that time. Thereafter, members can update information on their employment classification while those who do not renew membership are assumed to continue their prior employment. For the year 2010, AVMA records identify 95,430 positions held by U.S. veterinarians. Of these about 18% have unknown employment while about 8% are in food animal predominant or exclusive private clinical practice.

For each county, livestock species numbers were obtained from the 2007 USDA Census of Agriculture. Data for livestock were all cattle (labeled cattle), all hogs (hogs), all sheep (sheep) and all horses (horses). Horses were included as FAVs may care for them, especially in shortage counties where specialist equine veterinarians are unlikely to locate.

Consistent and reliable data were not available on pet populations at the county level of analysis. Therefore demographic variables were used to proxy for possible demands on

veterinarian time from serving pets in the county. These variables were the county's human population in 2000 and average per capita income in 1999, where census forms ask about income in the preceding year. These data were extracted from U.S. Census Bureau, decennial census 2000. Population and income should be important factors in determining the number of pets in a county and the population's willingness to pay for veterinary services.

A rurality index was also obtained for each county. The rurality index used was designed by the Purdue University's Center for Regional Development and Indiana University's Indiana Business Research Center (<http://www.ibrc.indiana.edu/innovation/maps.html>, visited 1/6/10). The index is based on four dimensions: population, population density, extent of urbanized (built-up) area and distance to the nearest metro area (Waldolf, 2006). A rurality index was included in the model to accommodate several possible issues. These include *i*) a preference for a rural lifestyle, which may be associated with ownership of livestock for reasons other than food production; *ii*) the effect of rurality on spousal career constraints; *iii*) the relationship between rurality and the business costs of serving food animal premises. Because of these multiple, and likely opposing, effects and because true shortages are likely to be correlated with rurality, we will interpret inferences concerning the variable with caution.

County land area was obtained from the U.S. Census Bureau, census 2000. The rationale for including area as an explanatory variable was that travel time matters in food animal practice. Given fixed food animal stocks, a larger county area would require more travel to provide the same service. The FAV (directly) and/or client (through service charges) would have to absorb travel costs. If FAVs absorb these costs then net income falls and alternative business locations may be sought. If clients absorb these costs then clients have stronger incentives to reduce FAV use. So, while time required to serve a given animal stock increases with county area, the effect on FAV presence is less clear.

Distance to veterinary college was obtained for each county. Figure 1 presents veterinary college locations. Distance is calculated by CDXZipStream software, see <http://www.cdxtech.com/CDXZipStream/Overview.aspx> (visited 6/7/2010). This software calculated the distance between each county and each of the 27 veterinary colleges using zip codes information, and chose the minimum distance. A county generally has a large number of zip codes. For simplicity we use the county's first zip code as listed by CDXZipStream software. All zip codes in the same county generate very similar minimum distances to a veterinarian school. This minimum distance variable was included in the analysis because practitioners may wish to avail of college services. Consequently, counties around schools may have more self-identifying FAVs than market forces might otherwise predict. In addition, the graduate or spouse may have established personal or professional roots while attending college.

We also sought to capture any distinctive effects associated with counties in which a veterinary college is located. We did so by use of a veterinary college dummy (college), assigned value 1 if a veterinary college is located in the county and value 0 otherwise. The effects at issue may include the aforementioned inertia and career opportunities that can arise for FAVs from being near a major teaching and clinical facility. Table 1 provides descriptive statistics for all the county level variables.

Models

The approach used to identify FAV location and shortage areas was to model the county level FAV count based on livestock and human populations, distance to a veterinary college, income, area and rurality. The modeling approach adapted Getz's (1997) ordinary least squares analysis on early 1990s state-level data by using discrete count regression methods on county-level data. The outcome for the model was the count of FAVs in a county.

The starting point for our FAV *Count* model is

$$\ln(\lambda_i) = \beta_0 + \beta_1 \ln(\text{cattle}_i) + \beta_2 \ln(\text{sheep}_i) + \beta_3 \ln(\text{hogs}_i) + \beta_4 \ln(\text{horses}_i) + \beta_5 \ln(\text{population}_i) + \beta_6 \text{distance}_i + \beta_7 \text{college}_i + \beta_8 \ln(\text{income}_i) + \beta_9 \text{area}_i + \beta_{10} \text{rurality}_i + \varepsilon_i, \quad (1)$$

where λ_i is the estimated mean FAV count and ε_i is a random error term. Subscript i denotes the county identifier. A nominal small number, 0.0001, was added to each of the variables to be logged to avoid the undefined value $\ln(0)$. Equation (1), through specifying $\ln(\lambda_i)$ rather than λ_i , ensures that the estimated mean FAV count is always nonnegative.

Species numbers have been log transformed so that proportional changes in species numbers match proportional changes in FAV count. In addition, the log transformation allows us to interpret parameters $\beta_i, i \in \{1, \dots, 5\}$, as percent of veterinary time allocated to the respective species. That is, with derivative $\partial(\text{mean FAV}) / \partial \text{cattle}$ as the FAV time required by one bovine then $\beta_1 = \text{cattle} \times (\partial \text{mean FAV} / \partial \text{cattle}) / [\text{mean FAV}]$. When building the model all the explanatory variables were first included, and removed sequentially whenever a variable's p value exceeded 0.1. We also assessed the model's explanatory power and fit with and without the rural index as accounting for it may possibly account for any FAV shortage.

In the *Count* model, and also the *Designation* model to follow, all available explanatory variables were included and then removed sequentially whenever a variable's Wald test p value was greater than 0.1. To measure the fraction of total variability that can be explained by our *Count* model, we computed the pseudo- R^2 statistic, $R_\alpha^2 = (\alpha_0 - \alpha) / \alpha_0$, see, e.g., Han *et al.* (2009) for another application. Here α_0 is the overdispersion parameter under the null (intercept-only) model and α is the overdispersion parameter under our selected model. The R_α^2 statistic would have value 1 were the explanatory variables perfectly specified, as then $\alpha = 0$. On the other hand, the statistic

would have value 0 were the included variables to have no explanatory power and $\alpha = \alpha_0$.

We then used the model residuals to develop an alternative definition of a shortage county. Residuals were calculated as $\text{resid}_i = v_i - \hat{v}_i$, where v_i is the i th county observed number of FAVs and \hat{v}_i is the corresponding predicted value, according to (1). A negative residual for the i th county means that fewer veterinarians are observed in the county than the model predicts. This can be interpreted in two ways. The model may be severely misspecified or there are fewer FAVs in the county than should be the case given information on FAV determinants. Conditional on acceptable specification, when a state has a larger share of counties with negative residuals than the national average then there is evidence that the state's shortage situation is more severe than the national average. Our hypothesis is that when a state's percentage of negative residual counties increases then that state faces a more severe FAV shortage problem. We provide a residual analysis by state in order to shed light on where FAV coverage is lightest.

We refer to the second model as the *Designation* choice model. It is a logistic regression model. The response variable takes only two values; 1 whenever the county was shortage designated under the 2010 VMLRP assignments and 0 otherwise. Our tentative *Designation* choice model is

$$\ln \left(\frac{s_i}{1 - s_i} \right) = \gamma_0 + \gamma_1 \text{FAV}_i + \gamma_2 \text{cattle}_i + \gamma_3 \text{sheep}_i + \gamma_4 \text{hogs}_i + \gamma_5 \text{horses}_i + \gamma_6 \text{population}_i + \gamma_7 \text{distance}_i + \gamma_8 \text{college}_i + \gamma_9 \text{income}_i + \gamma_{10} \text{area}_i + \gamma_{11} \text{rurality}_i + \eta_i, \quad (2)$$

where η_i represents the random error term. Here s_i is the estimated probability that the i th county is designated as short private veterinarians. We use the Hosmer-Lemeshow goodness-of-fit test (p. 360 in Dohoo *et al.*, 2003) to test the fit of our finalized designation model. We also calculated the area under ROC curve (AUC) as an indicator of a logistic model's

discriminatory power (p. 362 in Dohoo *et al.*, 2003). The AUC statistic is bounded between 0 and 1 with 0.5 being no better than random and 1 representing perfect discriminatory power.

Finally we tested whether the 2010 VMLRP designated shortage areas conform with our alternative definition of veterinary shortage areas. We do this by inquiring into how the residual from the *Count* model, which we take as indicating comparative shortage counties, was associated with VMLRP county designation. The modifications to the *Designation* model take the following two forms:

$$\ln\left(\frac{s_i}{1-s_i}\right) = \tau_0 + \tau_1 \text{resid}_i, \quad (3A)$$

$$\ln\left(\frac{s_i}{1-s_i}\right) = \psi_0 + \psi_1 \text{resid}_i + \psi_2 \text{cattle}_i + \psi_3 \text{sheep}_i + \psi_4 \text{hogs}_i + \psi_5 \text{horses}_i + \psi_6 \text{population}_i + \psi_7 \text{distance}_i + \psi_8 \text{college}_i + \psi_9 \text{income}_i + \psi_{10} \text{area}_i + \psi_{11} \text{rurality}_i + \eta_i, \quad (3B)$$

Here resid_i in (3B) replaces FAV_i in (2) as the residual was obtained through a modeling of FAV. Regression (3A) seeks to establish whether resid_i is effective by itself in explaining county shortage designation. Regression (3B) asks whether other variables carry additional explanatory power, and so seeks to identify finer detail on what drove designation decisions.

RESULTS

Model validity and selection

Count model

We initially used a Poisson regression model but detected over-dispersion and so adopted the negative binomial regression model to accommodate over-dispersion. The likelihood ratio statistic to check for over-dispersion (p. 400 in Dohoo *et al.*, 2003) gave LR statistic = $-2(\text{LL}(\text{Poisson}) - \text{LL}(\text{negative binomial})) = 576.88$. Here $\text{LL}(\text{model})$ is the model's log likelihood statistic. The null hypothesis was rejected at significance level 0.001, and so we

chose the negative binomial model over the Poisson model.

For the *Count* model the area variable was excluded as it was not statistically significant at the 10% level. The final model was:

$$\ln(\lambda_i) = \beta_0 + \beta_1 \ln(\text{cattle}_i) + \beta_2 \ln(\text{sheep}_i) + \beta_3 \ln(\text{hogs}_i) + \beta_4 \ln(\text{horses}_i) + \beta_5 \ln(\text{population}_i) + \beta_6 \text{distance}_i + \beta_7 \text{college}_i + \beta_8 \ln(\text{income}_i) + \beta_9 \text{rurality}_i + \varepsilon_i. \quad (1')$$

As previously discussed, rurality can have multiple effects on the number of FAVs in a county. Given the intent of the loan program, including it may explain away the shortage. For this reason we also consider the *Count* model without rurality and refer to it as (1''). For *Count* model (1') we have $R_a^2 = 0.754$. Residuals from estimation (1') will be used to assess the appropriateness of 2010 VMLRP designation choices.

Designation model

Six variables were removed from the model: sheep, horses, population, college, income and area. The finalized *Designation* model was:

$$\ln\left(\frac{s_i}{1-s_i}\right) = \gamma_0 + \gamma_1 \text{FAV}_i + \gamma_2 \text{cattle}_i + \gamma_3 \text{hogs}_i + \gamma_4 \text{distance}_i + \gamma_5 \text{rurality}_i + \eta_i. \quad (2')$$

Estimation (2') will be used to understand the designation choices. The result of the Hosmer-Lemeshow goodness-of-fit was not significant (Chi-square=12.03, DF=8, p-value=0.15). The model's AUC equals 0.71.

Count residual as explanation of Designation

Using the Wald test, we found that the variable "resid" should be included in the test model (3A) since it is significant at the 0.1 level. However, the Hosmer-Lemeshow goodness-of-fit test (Chi-square=17.5, DF=8, p-value=0.03) indicated poor model fit. We applied the Wald test as the model selection criterion for (3B) and removed seven variables

from the model, namely sheep, hogs, horses, population, college, income and area. The finalized model (3B) was:

$$\ln\left(\frac{s_i}{1-s_i}\right) = \psi_0 + \psi_1 \text{resid}_i + \psi_2 \text{cattle}_i + \psi_3 \text{distance}_i + \psi_4 \text{rurality}_i + \eta_i, \quad (3B')$$

The Hosmer-Lemeshow goodness-of-fit test result was not significant (Chi-square=6.17, DF=8, p-value=0.63) with AUC=0.705.

Model estimates and interpretation

The estimated *Count* models, with and without the rurality regressor, are provided in table 2. Estimates are robust to removing rurality. The only coefficient that changes significantly is that for population. This is not surprising as the rurality index includes a population component.

The coefficient estimates are 0.394 for cattle, 0.09 for sheep, 0.047 for hogs and 0.055 for horses. So, upon holding other model variables constant, a one percent increase in cattle population would increase expected veterinarian count by about 0.39%. That is, about 40% of FAV time is allocated to bovines. While the sheep coefficient is surprisingly large, the others appear to be reasonable and all are significant at the 10% level.

The sum of animal species coefficients equals about 0.59 while the human population coefficient is approximately 0.3. If time commitment to companion animals scales with human population then about 30% of a FAV's time is allocated to companion animals. Thus about 90% of FAV time has been accounted for. So the estimates are reasonable under this criterion.

The ln(income) coefficient was just above 1 so FAV time allocation scales in rough proportion to income. Very little of this income response is likely to be allocated to food animals. According to <http://www.ers.usda.gov/Data/RuralAtlas/atlas.htm#map> (visited

7/30/2011), four of 3,141 counties recorded had $\geq 50\%$, 34 had $\geq 33.3\%$ and 112 had $\geq 25\%$ employment in agriculture over 2005-2009. This suggests that income responses are likely to be concentrated in a practice's companion animal component (including horses). Concerning other factors, rurality and having a veterinary college were positively associated with a county's FAV count. Increased distance to a veterinary college was associated with a decrease in the predicted number of veterinarians.

The estimated logistic regression coefficients in the county designation model are given in table 3. From the Estimate column, a unit increase in FAV count decreases the log odds of being designated as a shortage county by 0.063. Interpretation is easier when the coefficients are exponentiated, as shown in the point estimate column. A unit increase in FAV count decreases the odds ratio of being listed as a shortage county by factor 0.939, or $e^{-0.063}$. This means that, *ceteris paribus*, when the FAV count increase from 0 to 2 then the odds of being shortage designated decreases by factor $(0.939)^2 \approx 0.882$. When cattle count increases by one unit (i.e., 10,000 cattle), then the odds ratio for being listed increases by factor 1.055.

As distance from the nearest veterinary college increased by one mile, the odds ratio for being a designated county increased to 1.005. So, *ceteris paribus*, when the distance to the closest veterinarian school increase by 100 miles, the odds ratio to be listed increased by factor $(1.005)^{100} \approx 1.647$. When rurality increases by one unit (i.e., from most urban to most rural), the odds ratio for being designated increases by the factor 5.836. This suggests that rurality has played a key role in deciding whether a county is listed as a veterinarian shortage county.

The estimated logistic regression coefficients in the residual only *Designation* model, (3A), and residual added *Designation* model, (3B), are given in table 4. In each case the sign on the *Count* model residual is as expected, i.e., whenever a county is less likely to be a

shortage area according to our *Count* model then the odds that it is designated as a shortage county by the VMLRP decreases. This result confirms that in general the 2010 VMLRP performs well according to our *Count* model criterion.

Comparing state-level shortage situations with national average

To obtain a sense of which states may have a more ‘dire’ veterinarian shortage situation when compared to the national average, we provide in table 5 the percentage of counties with negative residuals arising from the *Count* model. The average across all 38 states is 57.9% and the standard deviation is 15.9%. Five states (Idaho, Iowa, Maine, New Hampshire, Wisconsin) have no more than 42% of counties with negative residuals, i.e., one standard deviation below the national average. The states are mapped in figure 2. Of these, Maine and New Hampshire do not provide even an undergraduate program, but are small states proximate to colleges in Massachusetts and New York states.

Six states have at least 73.9% of counties with negative residuals, i.e., one standard deviation above the national average. These are mapped in figure 2. Two, North Carolina and Virginia, have veterinary colleges. The other four, Delaware, Rhode Island, South Carolina and West Virginia, do not provide an undergraduate program but are comparatively small states. If one were to discern a pattern here, it might be that the Northern tier of the Atlantic South is least well-served and the MidWest may be quite well-served. Kansas (56.2%), Montana (51.8%), Nebraska (48.4%), North Dakota (56.6%), Oregon (47.2%) and South Dakota (53%) have below average (57.9%) shortage levels, according to our definition.

Table 6 gives a sense of what the shortage situation might be for those states that don’t have a designated private practice shortage area. Our model suggests that one of the ten states that did not receive VMLRP funding in 2010 did not have a comparative shortage issue. This is Connecticut, where 25% of counties have *Count* model negative residuals. Alabama, New

Jersey and Tennessee clearly register as having significant comparative shortage situations. So our model suggests that any shortage problem is generally more severe for those states that do not have any VMLRP designated private practice shortage counties than for those that do.

DISCUSSION

Some notes on model selection

In the *Designation* model we find that six explanatory variables are not significant at the 10% level. These are sheep, horses, population, college, income and area. Sheep is a low valued livestock of limited presence in the United States while few view horses as food animals. Not surprisingly, the model infers that these species were not factor in county designation. Omission of college is not surprising as these 21 counties are likely to compete for funds under the separate VMLRP public practice category. We may think of income and population as control variables for the possibility that serving pets is a significant component of a practice. The veterinarians under scrutiny self-designated as serving primarily food animals, although the *Count* regression suggests that 30% of their time was allocated to companion animals. Given Section 7105 of the 2008 Farm Bill, insignificance of the income and population variables (as proxies for companion animals) was expected, and perhaps reassuring from the perspective of effective policy implementation.

Area is significant in neither *Count* nor *Designation* models. Based on our discussion in section 2.1, the area variable has two opposing effects on the number of FAVs. Other conditions equal, a large area means an increased gross demand for FAV service time, as travel time goes up. But the price for FAV service per animal is likely to increase in order to recoup travel costs, leading to decreased private demand for FAVs. The estimated *Count* model suggests that these effects offset. By contrast with FAV time available, actual services rendered are likely to decline with an increase in county area because more time is devoted to

travel. So it is somewhat surprising that area does not enter the *Designation* model.

Given the number of FAVs in a county, the significance of both cattle and hogs in the *Designation* regression is reassuring. Foot and Mouth disease is arguably the exotic disease of most concern to the United States (Monke, 2007). That both distance to a veterinary college and rurality also emerge as factors in determining county designation is also reassuring. They provide evidence that features motivating the VMLRP legislation did play a role during 2010 shortage designations.

The residual included versions of the *Designation* model, i.e., (3A) and (3B), infer that our measure of shortage works well as a factor in explaining designation. In the residual included *Designation* model other factors do retain statistical explanatory power beyond their role in residual determination. Cattle, distance and rurality measures increased likelihood of designation even having accounted for shortage, as we measure it. This suggests that the designation process has sought to distinguish between different types of shortages. Additional weight has been placed on factors, such as cattle numbers, along which many argue coverage vulnerabilities are greatest.

On the whole, this comparison between results of the *Count* and *Designation* models suggests to us that the process of designating shortage counties has performed reasonably well.

CONCLUSION

This paper has done three things. It has studied what determines the number of FAVs in a county and has pointed toward where shortages are likely to be. It has inquired into the factors that affected a county's designation as private practice shortage area under the VMLRP in 2010. And it has sought to establish whether counties we infer to be short veterinarians were indeed chosen under the program.

Factors determining FAV location include the populations of food animals and humans, proximity to veterinary college, average per capita income, and degree of rurality. Using shortfall relative to predicted FAV count as our measure of shortage, our analysis suggests that shortages are most severe in the South-Atlantic region, and more specifically in the Virginias and Carolinas. As to determinants of shortage designation, overall we found that a designated shortage area is typically a county that is characterized by few FAVs but many cattle and hogs, is rural and is far from a veterinary college. We also show that our measure of shortage has statistical power in explaining county designation under the 2010 VMLRP, providing support for the claim that VMLRP designation choices were consistent with declared program goals. This is true even after including such control variables as rurality and proximity to a Veterinary College, so the finding is quite strong.

An interesting wrinkle to the analysis is that three of ten VMLRP excluded states had what might be referred to as severe comparative shortage situations. Two are in the interior south. Perhaps infrastructural weaknesses in these states might lead to both comparative shortages and also limit their capacities to present their case for more public resources?

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Table 3.1: County descriptive statistics^a

Variable	Year	Mean	Std. Dev.	Min.-Max.
Shortage $\in \{0,1\}$	2010	0.210	0.408	0-1
Food animal veterinarians	2008	2.804	3.676	0-49
Cattle ($\times 10^4$)	2007	3.134	4.876	0-107.2
Sheep ($\times 10^4$)	2007	0.189	0.690	0-19.0
Hogs ($\times 10^4$)	2007	2.208	8.871	0-228.5
Horses ($\times 10^4$)	2007	0.131	0.150	0-3.113
(Human) Population ($\times 10^3$)	2000	89.5	292.5	0.07-9519
Distance (miles)	-----	140.95	94.34	0-601.9
Veterinary College $\in \{0,1\}$	2010	0.009	0.093	0-1
Income ($\times \$10^3$)	1999	17.13	3.916	4.96/44.30
Area (Square miles)	2000	967.22	1314.44	23.0-20053
Rurality $\in [0,1]$	2000	0.500	0.177	0-1

^a Data sources are explained in the text.

Table 3.2: Count model parameter estimates

Variable	With rurality		Without rurality	
	Estimate	p-value	Estimate	p-value
Intercept	-3.146	<0.0001	-2.544	<0.0001
ln(cattle)	0.394	<0.0001	0.392	<0.0001
ln(sheep)	0.090	<0.0001	0.091	<0.0001
ln(hogs)	0.047	<0.0001	0.047	<0.0001
ln(horses)	0.055	0.0342	0.068	0.0078
ln(population)	0.325	<0.0001	0.255	<0.0001
distance	-0.001	0.0004	-0.001	0.0006
college	0.485	0.0002	0.466	0.0001
ln(income)	1.041	<0.0001	1.025	<0.0001
rurality	0.585	0.01	-----	-----

Table 3.3: Designation model parameter estimates

Effect	Estimate	Pr>ChiSq	Point Estimate	95% Wald Confidence Limits
intercept	-3.247	<0.0001	–	–
FAV	-0.063	0.0008	0.939	0.905-0.974
cattle	0.054	<0.0001	1.055	1.034-1.078
hogs	0.009	0.0655	1.009	0.999-1.018
distance	0.005	<0.0001	1.005	1.004-1.006
rurality	1.764	<0.0001	5.836	3.193-10.673

Table 3.4: Designation model parameter estimates, Count model residual included

	Effect	Estimate	Pr>ChiSq	Point Estimate	95% Wald Confidence Limits
Model (3A), residual only	intercept	-1.308	<0.0001	–	–
	residual	-0.031	0.0551	0.969	0.939-1.001
Model (3B), residual added	intercept	-2.801	<0.0001	–	–
	residual	-0.050	0.0067	0.952	0.918-0.986
	cattle	0.036	<0.0001	1.036	1.018-1.054
	distance	0.005	<0.0001	1.005	1.004-1.006
	rurality	1.971	<0.0001	7.178	3.883-13.271

Table 3.5: Comparison of state-level shortage situations with national average

	# counties	Vet College	# Residuals ≤ 0	% residuals ≤ 0
Arizona	15	No	10	66.7
Arkansas	75	No	54	72
California	58	Yes	37	63.8
Colorado	64	Yes	31	48.4
Delaware	3	No	3	100
Florida	66	Yes	48	72.7
Idaho	44	No	16	36.4
Illinois	102	Yes	59	57.8
Indiana	92	Yes	56	60.9
Iowa	99	Yes	39	39.4
Kansas	105	Yes	59	56.2
Kentucky	120	No	80	66.7
Louisiana	64	Yes	36	56.3
Maine	16	No	5	31.3
Maryland	23	Yes	11	47.8
Michigan	83	Yes	61	73.5
Minnesota	87	Yes	43	49.4
Missouri	114	Yes	67	58.8
Montana	56	No	29	51.8
Nebraska	93	No	45	48.4
New Hamp.	10	No	2	20
New Mexico	32	No	17	53.1
New York	62	Yes	30	48.4
N. Carolina	100	Yes	74	74
N. Dakota	53	No	30	56.6
Ohio	88	Yes	47	53.4
Oklahoma	77	Yes	41	53.2
Oregon	36	Yes	17	47.2
Pennsylvania	67	Yes	38	56.7
Rhode Island	5	No	4	80
S. Carolina	46	No	37	80.4
S. Dakota	66	No	35	53.0
Texas	254	Yes	164	64.6
Utah	29	No	21	72.4
Vermont	14	No	6	42.9
Virginia	98	Yes	73	74.5
W. Virginia	55	No	44	80
Wisconsin	72	Yes	24	33.3
United States	2544	21 Yes, 17 No	1493	

Table 3.6: Shortage situations in the ten states omitted from the 2010 VMLRP

	# counties	Vet College	# Residuals ≤ 0	Percent that are ≤ 0
Alabama	67	Yes	56	83.6
Connecticut	8	No	2	25
Georgia	159	Yes	91	57.2
Massachusetts	14	Yes	8	57.1
Mississippi	82	Yes	46	56.1
Nevada	17	No	11	64.7
New Jersey	21	No	16	76.2
Tennessee	95	Yes	72	75.8
Washington	39	Yes	19	48.7
Wyoming	23	No	14	60.9

Chapter 4: Modeling Interdependent Participation Incentives: Dynamics of a Voluntary Livestock Disease Control Program

Modified from a paper submitted to *European Review of Agricultural Economics*

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ABSTRACT

This paper models producers' interdependent incentives to participate in a voluntary livestock disease control program. Under strategic complementarity among participation decisions, after a slow start momentum can build such that the market premium for participation and the participation rate increase sequentially. Nonparticipation, partial participation and full participation can all be Nash equilibria while participation cost heterogeneity will dispose the outcome toward incomplete participation. We find plausible conditions under which temporary government subsidies to the least cost effective producers could cause tipping toward full participation to occur. Applying parameters from the literature on Johnes' disease, we illustrate factors that may affect participation incentives. These include unit livestock value, perceived disease prevalence rate, consequences to human health, cost heterogeneity, as well as program effectiveness.

INTRODUCTION

Mandatory eradication programs have been a focus of the United States Department of Agriculture (USDA) for more than a century. For example, the campaign to eradicate the bovine tuberculosis (TB) between 1917 and 1940 was a huge success and prevented at least 25,000 human TB deaths annually (Olmstead and Rhode 2004). While mandatory programs can be effective in bringing highly contagious diseases under control, staunch resistance to such mandates are common (Olmstead and Rhode 2007; Anderson 2010). As a result, voluntary control and certification programs have been suggested for some diseases that are

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not serious enough to warrant eradication. Examples include the Voluntary Trichinae Certification Program, the Chronic Wasting Disease (CWD) Voluntary Herd Certification Program and the Voluntary Bovine Johne's Disease Control Program. Voluntary programs generally develop program standards as well as detail requirements for program certification and for certifying participants that meet program standards. The reward for being labeled as disease-free under a program is a market-determined premium. For Johne's disease, survey results show that premiums exist for producers who participate in voluntary certification programs (Kovich, Wells and Friendshuh 2006; Benjamin et al. 2009).

The success of a voluntary program hinges on producer participation (CDCJD, 2003). Therefore a natural question to ask is whether a voluntary livestock disease control program provides producers with sufficient incentive to participate. Such a question has been addressed extensively in the environmental literature (Khanna 2001). At the individual firm level, it is generally assumed that voluntary programs involve lower implementation costs (Segerson and Miceli 1998; Schmelzer 1999); and that government subsidies provide firms with incentives to participate (Stranlund 1995; Wu and Babcock 1999). Participation incentives at an industry level has also been studied (Segerson and Miceli 1998; Dawson and Segerson 2008; Millock and Salanie 2000). When multiple firms are involved and the government's aim is to reach a certain aggregated abatement level, some firms may have incentives to free ride as the required abatement level can be reached when the other firms participate. We can view the firms' abatement decisions as strategic substitutes according to Bulow, Geanakoplos and Klemperer (1985). There is also an emerging literature on voluntary food safety programs where the incentive to participate is only analyzed at the individual firm level. The producer's incentive to join the voluntary program could come from the looming threat of a mandatory program (Segerson 1999; Fares and Rouviere 2010) or from government subsidies (Cho and Hooker 2007).

Extant analyses of voluntary programs tend to omit the interrelated nature of participation incentives. Even among studies that do consider firm interactions, interdependence in participation incentives is only studied in a static framework. In reality, however, almost all voluntary programs span multiple years, with participation rates evolving from year to year. Therefore, to evaluate firm participation incentives it is important to consider dynamic interactions among participant choices. This paper provides a pilot work on the issue. We provide a dynamic model in the context of a voluntary livestock disease control program. However, as we assume that the motivation for improving and testing animal health comes from the concern over human health, our model can also apply to participation incentives in a voluntary food safety program.

Critical to model mechanics is the dynamic evolution of the price premium for proven disease-free product. Our goal is to analyze producers' incentives to participate in a disease control program that involves a disease status test, and subsequent incentives to release that information in order to acquire any available market premium at that time. As such our paper is closely connected with the quality disclosure literature. With two strict assumptions that *i*) that disclosure is costless and *ii*) producers have full information about their quality, earlier models in this literature found that every producer will disclose except the one with the lowest possible quality type (Grossman and Hart 1980; Grossman 1981; Milgrom 1981). The market solves the information problem through unraveling, in a manner that is exactly the reverse of that encountered in Akerlof's (1977) lemons problem (Viscusi 1978).

When assumption *i*) is relaxed, models by Jovanovic (1982), and Levin, Peck and Ye (2005) have found that only high-quality types would engage in disclosure. Alternatively, Matthews and Postlewaite (1985), Farrell (1986) and Shavell (1994) relaxed assumption *ii*) and assumed instead that sellers originally do not have information on their products' quality. Sellers could incur a test cost to obtain information on product quality, where it is costless to

disclose such information once it is acquired. They show that whenever disclosure is voluntary, then the sellers of the low cost type would acquire such information and disclose it if it is favorable. A mandatory disclosure rule in this case would decrease the sellers' incentive to acquire the quality information in the first place.

Our model is based on Shavell (1994), where producers need to make two choices, whether to participate in the program to obtain quality information and possibly improve their quality, and then, if obtained, whether to disclose such information. Whereas Shavell assumed that participation is voluntary while disclosure could be either voluntary or mandatory, in this paper we use mandatory participation as a benchmark for comparison while disclosure is always voluntary. We extend Shavell's model to a dynamic setup, where we show that the participation premium hinges on the participation rate over time. This allows us to prove that producer decisions are strategic complements. Therefore even if only very few producers have the incentive to participate initially, they might in turn provide the remaining producers with sufficient incentive to participate. This phenomenon is referred to as tipping. The reasoning here resembles that in Dixit (2003), who shows how a small group of enthusiasts could initiate a process which will later induce everybody else to join a club. This observation is important in that it provides insights on how animal disease program managers can engineer more efficient equilibria through selective subsidies.

Originated by Schelling (1978), the idea of tipping is generalized by Gladwell (2000) to a wide range of problems. From a game theory perspective, tipping is extensively studied in a general interdependent risk setup called interdependent security (IDS) problems (Heal and Kunreuther 2005, 2007). Nyborg, Howarth and Brekke's (2006) work is another application of the idea of tipping in the market for green good consumption. Tipping will occur when there are two or more equilibria and the system displays sufficient increasing difference (Heal and Kunreuther 2006, 2010). In the present paper, increasing differences arise because

when more firms participate then the change in payoff to a firm upon participating will increase, i.e., the premium from participation will increase.

The premium increase arises from the declining health status of the non-disclosing herd, which is made up of *i*) participated producers who prefer not to reveal the disease status, and also *ii*) non-participating producers. Every period, the average disease-free rate among non-disclosing herds is Bayesian updated and it decreases when more producers participate. As a result, momentum will build where three events, namely the decrease in the average disease-free rate among non-disclosing herds, the increase in participation premium and the increase in participation rate, occur repeatedly over time. Note that our theory of disease control is based on market price premium dynamics due to rational producer responses to incentives. Besides tipping and strategic complementarity issues, our paper also addresses any roles for mandates and targeted subsidy policies as well as implications of cost heterogeneity.

The paper's layout is as follows. After presenting examples of voluntary and mandatory programs, we study how producers' participation decisions are related to each other in a voluntary program setup. To provide a comparison, we will further investigate the alternative mandatory program and define three possible equilibria. To illustrate our model findings, a simulation analysis on a voluntary Johne's disease herd status program (VJDHSP) will be carried out where the parameters are obtained from current Johne's literature.

EXAMPLES OF VOLUNTARY/MANDATORY PROGRAMS

In this section we will summarize several historical or current animal disease control programs, for which either a voluntary or mandatory participation is required at different points of the program implementation. Although not related to a particular animal disease, the national animal ID system (NAIS) is included to illustrate the dynamic cost and benefit analyses of livestock owners. Leafy greens marketing agreement (LGMA) is covered here since it intends to eliminate the food contamination issue on the leafy greens, which is similar

to the objective of a disease control program that targets the livestock.

Voluntary Johne's Disease Herd Status Program (VJDHSP)

Johne's disease (JD) has a long period of incubation and clinical signs are rarely seen before two years of age. It is highly prevalent in the United States. According to the National Animal Health Monitoring System (NAHMS) dairy survey study of 2007, 68.1% of U.S. dairy herds were infected with the causative bacterium, *Mycobacterium paratuberculosis* (Mptb) (USDA 2008). Although the weight of evidence presently suggests no direct link between JD and Crohn's disease in humans, there is less agreement about what role it may play (Friswell, Campbell and Rhodes 2010). Due to agricultural production losses and zoonotic concerns, Johne's disease is has been prioritized for control in the United States (CDCJD, 2003). VJDHSP was developed in the United States in an effort to certify herds that are free of paratuberculosis. Three key components of the program are i) education, ii) management, and iii) herd testing and classification. The objective of herd testing and classification is to recognize producers in the program publicly if they so desire.

Many states have established, or are currently establishing, programs similar to the national VJDHSP (USDA 2010a). Minnesota's program (MNJDGP) is one of the most successful state JD control programs. Starting from less than 0.9% in 1999, the dairy herds participation rate in the MNJDGP had increased to 30.8% by the end of 2006. Meanwhile the Minnesota beef herd participation rate increased from less than 0.1% to 2.1%. There has been steady growth in program participation, especially after federal funding was initiated in 2003. Larger herds were more likely to participate than smaller herds. During 2005-'06, 52.9% of Minnesota dairy herds with ≥ 500 cows participated, in contrast with a 9.9% participation rate among herds with < 50 cows (Wells, Hartmann and Anderson 2008).

Benefits from participation include increased productivity, premiums from replacement

cattle and increased marketing opportunities (Kovich, Wells, and Friendshuh 2006; Benjamin et al. 2009). Concern over consumer health is another key factor to consider when producers make participation decisions. As one of the program purposes is to provide a source of low-infection risk replacement cows (Kovich, Wells, and Friendshuh 2006), the industry would be well-prepared in the event that the proposed link between Johne's disease in cattle and Crohn's disease were confirmed.

Bovine Tuberculosis (BTB) Eradication Program

In 1917, U.S. Congress passed a \$1 million appropriations bill initiating the State-Federal Cooperative Bovine Tuberculosis Eradication Program (Meyers 1940). This voluntary program authorized payment of indemnities. The program generally started from counties where a majority of dairymen consented to participate. Later on, states often made testing compulsory and required the slaughter of reactors (Olmstead and Rhode 2007). The reactor rate was about 4.9 percent at the start of this program. By 1940, the reactor rate had dropped to 0.46 percent and all jurisdictions in the United States were actively seeking to eradicate BTB (Meyers 1940). Despite eventual widespread cooperation, initial eradication efforts encountered opposition at every step. Although financial loss was likely the primary concern, stated points of opposition included questioning the underlying science, the program's implementation and incidence of cost. Indeed during the 'Cow War' of 1931 Iowa's Governor felt compelled to call up the State National Guard to maintain order along country roads and to protect veterinarians engaging in program activities (Sage 1983). Perceived economic interests of the participants were likely the first and foremost reason for the protesters.

Texas fever

Texas tick fever, caused by blood parasites *Babesia bovis* and *Babesia bigemina*, was a

major threat to the U.S. cattle industry from the Antebellum until the end of World War I. Efforts to eradicate tick carriers started as early as 1898, where initially participation had been voluntary. However, researchers soon realized that the eradication efforts would not be successful unless all cattle in a given area were treated and so mandatory participation was implemented. Active resistance emerged and most resistance to the programs came from small-scale farmers.

Resistance was intense among small-scale operators where compliance costs were greatest, leading to at least one 1922 murder in Arkansas (Hope 2005, pp. 10-12). Strom (2000) noted, however, that small farmer's "violent opposition to this program was founded in sound economic reasoning." When larger ranchers began to see the benefit of eradication as re-infection became less common, prospects for controlling residual infected areas increased. More areas were removed from federal quarantine and the return on treated animals increased. That is, a virtuous cycle of events led to a better equilibrium for those who could bear eradication costs. Eventually the eradication programs prevailed and by 1933 Texas fever was no longer a major problem for the cattle industry (USDA 1933).

The National Animal ID System (NAIS)

NAIS is a U.S. government initiative to establish a nationwide farm-level animal ID system in the event of a disease outbreak to trace an animal disease to its source. The U.S. Animal Identification Plan (USAIP) was initially intended to be mandatory. In 2006, however, NAIS participation was made voluntary in the face of stiff opposition to compulsion. Only approximately 35% of U.S. livestock premises had been registered. Participation rates in the premises registration step have been very high for poultry (95%), sheep (95%), high for swine (80%), but only 18% for cattle (Schnepf 2009).

Heterogeneity in participation costs could explain the dramatic differences in

participation rate. According to NAIS (2009), the average per animal cost was \$1.39 per sheep, \$0.059 per swine, \$0.0007 per broiler, \$0.002 per turkey, and \$0.0195 per layer. In contrast, the cost was \$5.97 per bovine. The swine and poultry industries have a much lower cost because animal tracing requirements for these species involve no individual identification devices. Typically unit participation costs decrease with herd size; see, e.g., tables 2 and 3 of NAIS (2009).

The benefit from NAIS implementation increases as participation levels increase. According to simulation results in (NAIS Benefit-Cost Research Team 2009), “producer monetary losses for an animal identification and tracking program with a 90 percent participation rate would be \$4.5 billion less than a program with a 30 percent participation rate.” For bovines, this program was largely unsuccessful, due partly to failure by the U.S. Dept. of Agriculture to communicate program benefits to livestock producers (Anderson 2010).

Leafy Greens Marketing Agreement (LGMA)

In 2007, LGMA was established in California in response to the September 2006 E. coli outbreak attributed to contaminated spinach. The agreement would require producers and handlers to implement the best industry practices.

The LGMA is a success example of the voluntary programs as it achieved almost full participation only two years after the enactment of this industry initiative. As reported by the LGMA website (www.caleafygreens.ca.gov), “Over 100 handlers, representing approximately 99% of the volume of California leafy greens, are LGMA members”.

According to California Department of Food Safety and Agriculture (CDFA) website (www.cdfa.ca.gov/agvision/docs/Food_Safety.pdf), the benefit of LGMA participation includes expanded market access, reduced likelihood of food safety incidents and regaining

consumers' confidence in the event of a food safety incident. On the cost side, large fixed costs lead to significant economies of scale (Hardesty and Kusunose 2009).

VOLUNTARY PROGRAM

In a voluntary program, a producer makes his own decision on whether to participate based on the benefits and costs of program participation. These benefits and costs evolve during the course of the program, and it is necessary to explicitly model the evolution of incentives.

Model Scheme

Similar to Shavell (1994), we commence with a model of information acquisition and disclosure. In our context, information is referred to as knowledge on a herd's disease-free rate, which is defined as the number of disease-free animals divided by the total number of animals in the herd. In this paper we will analyze a situation where neither producers nor buyers know the precise quality of their goods (Shavell 1994; Matthews and Postlewaite 1985). A third party provides tests as a part of the voluntary program to reveal the disease status to producers.

A herd's disease-free rate is denoted by a variable $r \in [\underline{r}, 1]$ with probability distribution function $F(r)$. While $F(r)$ is common knowledge among all producers, a herd's particular disease-free rate remains unknown prior to program participation.¹³ Participation cost is denoted by $c \in [\underline{c}, \bar{c}]$ with probability distribution function $G(c)$. Each producer is only aware of his own participation cost. In Assumption 1, we will define the relationship between the participation cost and disease-free rate.

Assumption 1: Assume that the participation cost is independent of the herd's disease-free

¹³ The current literature on animal disease generally assumes an asymmetric information structure, i.e., only producers have full information about their herd's disease status (Gramig, Horan, and Wolf 2009; Sheriff and Osgood 2010). This assumption might be valid for some acute animal diseases. However, producer knowledge about some chronic diseases can be very limited due to its long incubation period.

rate.

Assumption 1 is supported by observations in Pillars et al. (2009), who conducted a 5-year longitudinal study of six Michigan dairy herds infected with Johne's disease.¹⁴ The data collected in their paper shows no pattern of correlation between disease prevalence rate and participation cost.

The model scheme is as follows. In period t , producers decide whether to participate based on the expected price premium realized in the previous period, I_{t-1} . Assume that producers participate if their expected price premium I_{t-1} is no less than the cost. Proportion $\eta_t = G(I_{t-1})$ of producers will participate. After obtaining the test result, a participant will disclose whenever it exceeds the average disease-free rate among silent producers in the previous period, r_{t-1}^S . The silent producers are comprised of two groups: producers 1) who choose not to participate in the program; and 2) who participate in the program but prefer not to disclose any information. Here we assume that buyers cannot distinguish between the two groups. We also assume that there are many producers in the market and a single producer's participation decision cannot affect the participation rate in the market.

Based on the participation and disclosure rates, a new average disease status among silent producers in period t will be determined as r_t^S . In return, a new price premium from participation will be solved as I_t . We will elaborate on the determination of I_t and r_t^S later. Note that except for the specified probability distributions on disease-free rate and participation cost, all other variables in the model scheme are endogenous. The process

¹⁴ For example, from their table 3 we can see that herd 4 has the highest average implementation cost across the 5-year period, \$65.02, while table 2 shows that herd 4 has a relatively low disease prevalence rate, ranging from 4% to 12% with average 8%. Herd 2, to the contrary, has a much lower average implementation cost during the same period, \$31.73, while its disease rate ranges from 5% to 44% with an average of 19.6%.

continues indefinitely through time. A scheme of the model to be studied is displayed in Figure 1. The price premium from participation will also depend on the information conveyed by the test results. We assume that the test reveals the exact disease-free rate to the participating producers.¹⁵

Determination of Participation Premium

In period t testing reveals the exact disease-free rate, r_t , to the producer. The expected unit animal value for the herd with disease-free rate r_t is $r_t V + (1 - r_t)\alpha V$. Here V denotes the value of a healthy animal and αV stands for the value of a diseased animal, where $\alpha \leq 1$ could vary by disease and is an indicator of the perceived consequences caused by the disease. If buyers perceive no harm in the disease, then $\alpha = 1$. However, α would take negative values whenever the disease causes serious human health problems. This means that the producer may suffer large losses from a small percentage of diseased animals, either from compensation to consumers or from a trade embargo. The average disease-free rate among silent producers is r_t^S . How equilibrium r_t^S is determined will be discussed shortly.

As non-participants belong to the group of silent producers, the unit livestock price of a non-participating herd is $p_t^1 = r_t^S V + (1 - r_t^S)\alpha V$. A participant will reveal r_t whenever it is greater than r_t^S and will remain silent otherwise. Thus the unit realized price of a participating herd takes the following form:

$$p_t^2 = \begin{cases} r_t^S V + (1 - r_t^S)\alpha V, & \text{whenever } r_t \leq r_t^S; \\ r_t V + (1 - r_t)\alpha V, & \text{whenever } r_t > r_t^S. \end{cases} \quad (1)$$

¹⁵ A second scenario, referred to as the coarse grading case, is studied in supplemental materials. It seeks to better replicate the threshold rates classifications in actual programs. For example, in VJDHSP, cattle herds in the initial year of testing will be placed in classification levels 1 to 3. After the second, third and fourth year of testing with no positive test results, cattle herds can be upgraded into level 4, level 5 and level 6 respectively (USDA 2010a).

The realized premium from participation is calculated as:

$$p_t^2 - p_t^1 = \begin{cases} 0, & \text{whenever } r_t \leq r_t^S; \\ (1 - \alpha)V(r_t - r_t^S), & \text{whenever } r_t > r_t^S. \end{cases} \quad (2)$$

The ex-ante expected price premium from participating in the program is:

$$I_t(r_t^S, \alpha, V) = (1 - \alpha)V \int_{r_t^S}^1 (r_t - r_t^S) dF(r). \quad (3)$$

Proposition 1: The expected premium from participation will increase whenever one of the following holds: *i)* Society becomes more aware of the disease, $\partial I_t / \partial \alpha \leq 0$; *ii)* the value of an animal increases, $\partial I_t / \partial V \geq 0$, or *iii)* the average disease-free rate among silent producers decreases, $\partial I_t / \partial r^S \leq 0$.

Proof: The inferences follow from $\partial I_t(r_t^S, \alpha, V) / \partial \alpha = V \int_{r_t^S}^1 (r_t^S - r_t) dF(r) \leq 0$,

$$\partial I_t(r_t^S, \alpha, V) / \partial V = (1 - \alpha) \int_{r_t^S}^1 (r_t - r_t^S) dF(r) \geq 0 \text{ and } \partial I_t(r_t^S, \alpha, V) / \partial r_t^S =$$

$$(\alpha - 1)V[1 - F(r_t^S)] \leq 0. \quad \square$$

Item *i)* in Proposition 1 makes the intuitive assertion that whenever the disease is perceived to be of greater harm, then participating producers can expect a higher price premium. From item *ii)* we learn that producers will have stronger incentives to participate whenever the unit livestock value increases. Therefore compared to dairy cow producers, beef cow producers have weaker incentives to participate, as illustrated by the different participation rates of dairy and beef herds in the MNJDPC. Finally, item *iii)* conveys that the premium from participation will increase whenever the average disease-free rate among non-disclosing herds decreases. Intuitively, as the perceived mean quality in the unknown pool declines, buyers are willing to pay more to obtain livestock with a confirmed high disease-

free rate.

Market Equilibrium

The program starts in period 0. Initially $\eta_0 = 0$ as no producer is part of the program. The average disease status of silent producers is the unconditional mean of r , i.e.,

$r_0^S = E(r) = \int_{\underline{r}}^1 r dF(r)$. By eqn. (3), the price premium in period 0 is:

$$I_0 = I(r_0^S) = \gamma \int_{r_0^S}^1 (r - r_0^S) dF(r), \quad (4)$$

where $\gamma \equiv (1 - \alpha)V$. To render more concise expressions we will use γ for the rest of the text. According to the model scheme in Figure 1, in period 1 producers with participation costs no more than I_0 will participate. Thus the overall participation rate is

$\eta_1 = G(I_0) = G(I(r_0^S))$. Among participants, those who obtain a disease-free rate less than r_0

will not disclose any information on their disease status. This group has mass measure

$\eta_1 F(r_0^S)$ with the average disease-free rate $E(r | r \leq r_0^S)$. In addition, all non-participants

cannot disclose their disease-free rates without being tested. They have mass measure $1 - \eta_1$

with the average disease-free rate $E(r)$. Now the average disease-free rate among silent

producers is Bayesian updated in period 1 as:

$$r_1^S = \begin{cases} \pi(\eta_1)E(r | r \leq r_0^S) + (1 - \pi(\eta_1))E(r) & \text{whenever } \eta_1 < 1; \\ \underline{r}, & \text{whenever } \eta_1 = 1; \end{cases} \quad (5)$$

$$\pi(\eta_1) = \frac{\eta_1 F(r_0^S)}{\eta_1 F(r_0^S) + 1 - \eta_1}.$$

The denominator in the expression for $\pi(\eta_1)$ represents the proportion of all silent producers, while the numerator represents the proportion of participants who choose not to

disclose the result. After solving for r_1^S by way of (5), we can calculate the expected premium in period 1 as $I(r_1^S)$ by (4). Note that $r_1^S = \underline{r}$ when $\eta_1 = 1$. This is because after all the producers obtain their disease status information, producers with a disease-free rate higher than the average will choose to disclose. The average disease-free rate among non-disclosers will continue to decrease until it reaches \underline{r} . Eventually all participants will disclose (Grossman and Hart 1980).

The process repeats in each period t , $t \geq 1$. Equilibrium will be reached at time t whenever $\eta_{t+1} = \eta_t = \eta^*$ and $r_{t+1}^S = r_t^S = r^*$. Similar to (5), the equilibrium condition is characterized by:

$$r^* = \begin{cases} \pi(\eta^*)E(r | r \leq r^*) + (1 - \pi(\eta^*))E(r) & \text{whenever } \eta^* < 1; \\ \underline{r} & \text{whenever } \eta^* = 1; \end{cases} \quad (6)$$

$$\pi(\eta^*) = \frac{\eta^* F(r^*)}{\eta^* F(r^*) + 1 - \eta^*}; \quad \eta^* = G(I(r^*)).$$

When $\eta^* = 1$, then of course $r^* = \underline{r}$. In this case the full-participation (FP) equilibrium is reached and the pool of silent producers will disappear. If $\eta^* = 0$, then $\phi(r^*) = 0$ and $r^* = E(r)$. This characterizes the non-participation (NP) equilibrium. Lastly a partial-participation (PP) equilibrium is defined whenever $\eta^* \in (0, 1)$, which generates $\phi(r^*) \in (0, 1)$ and $r^* \in (\underline{r}, E(r))$.

The equilibrium condition in (6) was previously defined in Shavell (1994). However, Shavell did not recognize the need to redefine r^* at $\eta^* = 1$. In addition, our paper demonstrates dynamically how the equilibrium could be reached, a feature not captured in Shavell (1994). An understanding of the underlying dynamics is critical because, as we will show, the complementary nature of participation decisions allows for multiple equilibria where tweaking the decision environment through policy interventions can tip equilibrium

participation from very low to very high.

Presently, for the sake of illustrating the nature of equilibrium and the potential for policy interventions we focus on static Nash equilibrium solutions. In Figure 2, the horizontal and vertical axes stand for possible values of \underline{c} and \bar{c} , which denote the producers' lowest and the highest participation costs respectively. As $\bar{c} \geq \underline{c}$, the regions below the 45 degree line in Figure 2 are not feasible. The area above the diagonal line is divided into six regions. Two thresholds used in this division are $I(E(r))$ and $I(\underline{r})$, which stand for the premium at NP and FP equilibria. Note that $I_{\min} = I(E(r))$ and $I_{\max} = I(\underline{r})$, as implied by equation (5) and Proposition 1.

According to producer participation incentives, we will introduce these six regions in two categories. The first category consists of Regions R2, R3, R5 and R6, where without subsidy all the producers will inevitably make the same participation decisions. For R2, R3 and R5 we have $I_{\min} \leq \underline{c} \leq \bar{c}$, thus NP is an equilibrium. In contrast, region R6 with $\underline{c} \leq \bar{c} < I_{\min}$ only displays the FP equilibrium. The second category covers only R1 and R4, where with no subsidy participation decisions are not clear.

Of the six regions, we are particularly interested in region R4 and R5, where we have respectively $\underline{c} < I_{\min} \leq \bar{c} < I_{\max}$ and region R5 with $I_{\min} \leq \underline{c} \leq \bar{c} < I_{\max}$. In these two regions if government entices (perhaps by cheap talk) a sufficiently large subset of producers to participate first, then FP could be maintained without any subsidy. Moving from the former to the latter equilibrium, whether through some market event or through economic engineering, is referred to as tipping. Here we assume that the NP and PP equilibria are viewed by the government as undesirable equilibria when compared to the FP equilibrium.

A last note on Figure 2 is that it also illustrates the effect of cost heterogeneity on equilibrium participation status. All points on the 45 degree line represent the case where

minimum cost equals to the maximum cost. In other words, on this line participation costs are homogenous among producers. In this case the equilibrium could be either FP or NP. By contrast, points further away from the 45 degree line denote cases where participation costs are more heterogeneous. Among the six regions, R1 has the most heterogeneous cost structure. In this region a proportion of lowest cost producers will always participate. However, FP cannot be reached unless the government consistently provides subsidies to the highest cost producers.

Take the cattle sector in the NAIS program for example. Due to its high average participation cost and cost heterogeneity (NAIS 2009), its cost structure is mostly likely located in either R1 or R2. Therefore FP is unlikely to be reached without a government subsidy. In contrast, poultry and sheep sectors in NAIS program have much lower participation costs together with nearly full participation rates, so we are almost certain that cost structures for those two sectors are located in R6.

As participation cost scale economies exists, cost heterogeneity is most likely to exist in industries where large and small firms co-exist. In this case large producers tend to join the program first, while small producers will most likely find it unprofitable to join the program without government subsidies. For example, more than half of Minnesota dairy herds with ≥ 500 cows participated in MNJDCP, in contrast with a participation rate of less than 10% among herds with less than 50 cows (Wells, Hartmann and Anderson 2008).

Next, in Proposition 2 we will show that at least two equilibria can exist in R5. For region R4 a similar proof can be readily developed.

Proposition 2: Under a voluntary program, when $I(E(r)) \leq \underline{c} \leq \bar{c} < I(\underline{r})$ then at least two equilibria exist. These are NP and FP.

Proof: If no producer participates, then $r = E(r)$. The expected premium from participation is $I(E(r))$. As $I(E(r)) \leq \underline{c}$, nobody participates. Therefore $(r^*, \eta^*) = (E(r), 0)$ is one

equilibrium. If all producers participate, then $r = \underline{r}$. The expected premium from participation is $I(\underline{r})$. As $\bar{c} < I(\underline{r})$, all producers will participate. Thus $(r^*, \eta^*) = (\underline{r}, 1)$ is another equilibrium. \square

Note that Proposition 2 only shows the two guaranteed equilibria. However, whenever FP and NP are two guaranteed equilibrium, a PP equilibrium may also be possible depending on the exact cost structure of all producers. This point will be further illustrated in Example 1 below. Example 1 provides a numerical example showing an instance where tipping occurs in region R5 after the government motivates a subset of producers to participate.

Example 1 (Tipping): There are four types of producers that differ by their participation costs $(c^1, c^2, c^3, c^4) = (5.5, 5.7, 6.7, 8)$.¹⁶ Assume that N producers exist for each cost type. Prior to participation, the disease-free rate of any herd satisfies a uniform distribution on $[0.90, 1]$. The unit livestock value is $V = 500$, and $1 - \alpha = 0.8$.

First we will check the values of $I_{\min} = I(E(r))$ and $I_{\max} = I(\underline{r})$:

$$I_{\min} = 500 * 0.8 \int_{0.95}^1 \frac{r - 0.95}{1 - 0.90} dr = 5; \quad I_{\max} = 500 * 0.8 \int_{0.90}^1 \frac{r - 0.90}{1 - 0.90} dr = 20.$$

Thus $I_{\min} \leq \underline{c} \leq \bar{c} \leq I_{\max}$. By Proposition 2, both NP and FP are equilibria. Next we will illustrate how the NP equilibrium can be tipped to the FP equilibrium.

Period 0: We know that $r_0^S = E(r) = 0.95$ so that the NP equilibrium will be reached without government intervention.

Period 1: Suppose now that in period 1 the government provides subsidy $s = 0.5$ to producers of all cost types contingent on their participation. For type 1 producers the new cost will be reduced to 5. Thus 25 percent (as $N / 4N = 0.25$) of producers will participate in

¹⁶Note that superscript denotes the cost type, while subscript denotes the time period.

period 1. Among participants, fraction $(0.95 - 0.90) / (1 - 0.90) = 0.5$ of producers do not disclose. Therefore $\pi(\eta_1) = (0.25 * 0.5)(0.25 * 0.5 + 0.75) = 0.1429$. In period 1, the average disease status of silent producers is:

$$r_1^S = \pi(\eta_1)E(r | r \leq r_0^S) + (1 - \pi(\eta_1))E(r) = 0.1429 * E(r | r \leq 0.95)$$

$+ (1 - 0.1429) * 0.95 = 0.9464$ and the expected premium in period 1 is determined as:

$$I_1 \equiv I(r_1^S) = 400 \int_{r_1^S}^1 \frac{r - r_1^S}{1 - 0.90} dr = 5.75.$$

Period 2: In this period producers of cost type 1 and 2 will participate without subsidy because $\max[c^1, c^2] < I_1$. Among participants, share $(0.9464 - 0.90) / (1 - 0.90) = 0.464$ will choose not to disclose their disease status. Thus $\pi(\eta_2) = 0.5 * 0.464 / (0.5 * 0.464 + 0.5) = 0.3169$. In period 2, the average disease status among silent producers is $r_2^S =$

$\pi(\eta_2)E(r | r \leq r_1^S) + (1 - \pi(\eta_2))E(r) = 0.3169 * E(r | r \leq 0.9464) + (1 - 0.3169) * 0.95 = 0.9415$, and the expected premium in period 2 is determined as:

$$I_2 \equiv I(r_2^S) = 400 \int_{r_2^S}^1 \frac{r - r_2^S}{1 - 0.90} dr = 6.84.$$

Period 3: Given I_2 , cost types 1, 2 and 3 producers will participate without subsidy.

Among participants, 41.5% will choose not to disclose their disease status. Thus

$\pi(\eta_3) = 0.75 * 0.415 / (0.75 * 0.415 + 0.25) = 0.5546$. In period 3, the average disease status among silent producers is

$$r_3^S = \pi(\eta_3)E(r | r \leq r_2^S) + (1 - \pi(\eta_3))E(r) = 0.5546 * E(r | r \leq 0.9415) +$$

$(1 - 0.5546) * 0.95 = 0.9338$ and the expected premium in period 3 is determined as:

$$I_3 \equiv I(r_3^S) = 400 \int_{r_3^S}^1 \frac{r - r_3^S}{1 - 0.90} dr = 8.76.$$

Period 4: Given the value of I_3 all producers will participate without subsidy. In turn all

producers will disclose, so $r_4^S = \underline{r} = 0.90$ and $I(r_4^S) = 20$. Therefore in period 5 and thereafter, all producers will participate. The FP equilibrium $(\eta^*, r^*) = (1, 0.90)$ is reached.

As an additional note on Proposition 2's conclusion, suppose instead that the type 2 cost producers exceeds 5.75 in the example. Then, as $I_1 = 5.75$, the tipping process will stall and only PP will be reached unless the government also provides a sufficient subsidy to types other than type 1. Therefore the PP equilibrium is also possible for region R5.

Following Definition 2 in Heal and Kunreuther (2007), type 1 producers in Example 1 form a critical coalition. This is because if producers of cost type 1 participate, then the NP equilibrium will switch to the FP equilibrium. In our example, by taking advantage of this critical coalition, government only provides a total subsidy payment of $0.5N$. Without recognizing the existence of this group, and so providing subsidies to all to bring net costs down to 5, the government would pay a total subsidy of $0.5N + 0.7N + 1.7N + 3N = 5.9N$. An even more costly case is where all producers are provided the marginal subsidy required to elicit participation by type 4. Then cost is $12N$, a 24-fold increase over the tipping solution.

To find out how producers welfare changes over time, we can compute $\partial p_t^1 / \partial r_t^S$ and $\partial E(p_t^2) / \partial r_t^S$ as

$$\begin{aligned} \frac{\partial p_t^1}{\partial r_t^S} &= (1 - \alpha)V \geq 0; \\ \frac{\partial E(p_t^2)}{\partial r_t^S} &= \frac{\partial p_t^1}{\partial r_t^S} + \frac{\partial I(r_t^S)}{\partial r_t^S} = (1 - \alpha)V + (\alpha - 1)V[1 - F(r^S)] = (1 - \alpha)VF(r^S) \geq 0. \end{aligned} \tag{7}$$

Therefore if the disease-free rate for a non-disclosing herd decreases, i.e., when the participation rate increases, both non-participating and participating herds will experience a decrease in expected price. This suggests that as more producers participate, the advantage to

participation will erode; while non-participants will be in a comparatively more disadvantageous position.

When nobody participates then the price received by a non-participating herd is $p_0^1(E(r))$, which is greater than that at the equilibrium, $p_t^1(r^*)$. At the equilibrium the expected price for a participating herd is $E(p_t^2(r^*))$. If the herd has a participation cost $c < I_t(r^*)$ then the herd will participate in equilibrium. However, we could have $E(p_t^2(r^*)) - c < p_0^1(E(r))$, see Example 2 below. This shows that the firm actually becomes worse off when participating in the program, when compared to its welfare where the program does not exist.

Example 2: Assume that all the parameters given in Example 1 apply. We could have

$E(p_t^2) - c < p_0^1$, where $t \geq 4$. Initially the non-participating herd has a unit value at $p_0^1 = \gamma E(r) + \alpha V = 480$ and $E(p_0^2) = I_0(E(r)) + p_0^1 = 485$ while at the FP equilibrium ($t \geq 4$), $p_t^1 = \gamma \underline{r} + \alpha V = 360$ and $E(p_t^2) = I_t(\underline{r}) + p_t^1 = 480$. Clearly $E(p_t^2) - c < p_0^1$ for any $c > 0$.¹⁷

As shown in Example 1, in each period producers make decisions by comparing the current premium with their participation costs. In the long run they could be trapped into making a decision they do not like, as illustrated in Example 2. In this case we encounter the phenomenon studied by Dixit (2003), in which some (and perhaps most) voluntary participants are eventually “trapped” in a program that they do not like.

¹⁷ It should be noted that, more generally, $p_t^1(r^e) = E(p_t^2(\underline{r}))$. To ascertain this, on the one hand we know that $p_t^1(r^e) = r^e V + (1 - r^e) \alpha V$ while on the other hand

$$E(p_t^2(\underline{r})) = p_t^1(\underline{r}) + I_t(\underline{r}) = \underline{r}V + (1 - \underline{r})\alpha V + \gamma \int_{\underline{r}}^1 (r - \underline{r})dF(r) = r^e V + (1 - r^e)\alpha V .$$

The underlying reason for tipping in Example 1 is the strategic complementarity property of the producers' participation decisions, or "increasing difference" (Heal and Kunreuther 2006, 2010). Following the definition in Bulow, Geanakoplos and Klemperer (1985), we define the producers' participation decisions as strategic complements if one producer's marginal payoff from participating increases whenever the participation rate increases. In a game theory context, this means that the marginal returns to participating rise when more producers participate. Such a game is referred to as a supermodular game (Milgrom and Roberts 1990).

Here the expected marginal payoff from participating at time t is I_t . Therefore, the strategic complementarity property holds if whenever $0 \leq \eta'_t \leq \eta''_t \leq 1$, then $I_t(r_t^S(\eta'_t)) \leq I_t(r_t^S(\eta''_t))$. Next we will investigate whether this property is satisfied in the general model setup. This leads to our paper's main result; the strategic complementarity among producer decisions. In order to arrive there we will prove a lemma to show that the participation rate will increase whenever the previous period's premium increases.

Lemma 1: Suppose that the expected price premium in period $t+1$ is greater than that in period t . Then the participation rate in period $t+2$ will be greater than that in period $t+1$. That is, if $I''_t \geq I'_t$, then $\eta_{t+1}(I''_t) \geq \eta_{t+1}(I'_t)$.

Proof: This result follows immediately from relation $\eta_t = G(I_{t-1})$, where cost distribution function $G(\cdot)$ is non-decreasing. \square

Next we will establish a proof for strategic complementarity, i.e., that the premium will increase when the participation rate in the same period increases.

Proposition 3: Producer participation decisions at any time period t are strategic complements. That is, whenever $0 \leq \eta'_t \leq \eta''_t \leq 1$ then $r_t^S(\eta'_t) \geq r_t^S(\eta''_t)$ and

$$I_t(r_t^S(\eta_t')) \leq I_t(r_t^S(\eta_t'')) .$$

Proof. By equations (5) and (6) we have $r_t^S(\eta_t) \equiv \pi(\eta_t)E(r | r \leq r_{t-1}^S) + (1 - \pi(\eta_t))E(r)$ where $\pi(\eta_t) = \eta_t F(r_{t-1}^S) / [\eta_t F(r_{t-1}^S) + 1 - F(r_{t-1}^S)]$ whenever $\eta_t \neq 1$. If $\eta_t = 1$, then $r_t^S(1) = \underline{r}$. Given r_{t-1}^S fixed, we can easily show that $r_t^S(\eta_t)$ is decreasing in η_t . Thus $\eta_t' \leq \eta_t''$ implies $r_t^S(\eta_t') \geq r_t^S(\eta_t'')$. By Proposition 1 it follows that $I_t(r_t^S(\eta_t')) \leq I_t(r_t^S(\eta_t''))$. \square

It is worth noting that strategic complementarity alone cannot guarantee a high participation rate. According to a simulation result in NAIS (2009), the benefit from NAIS would increase if participation rates increase. However as producers are generally unaware of potential program benefits (Anderson 2010) the program is likely to be unattractive at the outset, even among the most cost efficient producers. As a result only a small fraction of producers are likely to participate, confirming the belief that participation generates little benefit. Such a vicious cycle repeats so that the program stalls at a low participation rate equilibrium.

We will refer to the participation rates when plotted against time as the participation curve. Next we will provide a plausible condition under which the participation rate will increase over time without any intervention. This means the participation curve could not contain any downward sloping segment.

Assumption 2: Assume that $J(r^S) = \phi(r^S)(E(r) - E(r | r \leq r^S))$ is decreasing in

$r^S \in [\underline{r}, E(r)]$, where $\phi(r^S) = g(r^S)F(r^S) / [g(r^S)F(r^S) + 1 - g(r^S)]$ denotes the proportion of participants among all silent producers. Here $g(r^S) \equiv G(I(r^S))$.

Proposition 4, to follow, shows that the purpose of Assumption 2 is to guarantee that

$r_0^S \geq r_1^S \geq \dots \geq r_k^S$ holds without any exogenous forces. This means that market interactions would ensure that the average disease-free rate among silent producers decreases monotonically over time. From Example 1, we can see that average disease-free rate among the silent producers will strictly decrease conditional on types 1 having a participation cost of below 5. This will generate momentum whereby the premium and participation rate both increase over time.

The outcome $r_0^S \geq r_1^S \geq \dots \geq r_k^S$ will generally apply when the easily computable uniform distribution is assumed on cost and disease rate distributions and value to be protected is not small. In Appendix A we provide a demonstration. Therefore we could be assured that Assumption 2 is a plausible condition.

Proposition 4: Under Assumption 2, the following three inferences apply: *i)* Average disease-free rate of silent producers will be non-increasing over time, i.e., $r_0^S \geq r_1^S \geq \dots \geq r_k^S$; *ii)* Premium from participation will be non-decreasing over time, i.e., $I_0 \leq I_1 \leq \dots \leq I_k$; *iii)* Participation rate will be non-decreasing over time, i.e., $\eta_0 \leq \eta_1 \leq \dots \leq \eta_k$.

Proof. See Appendix A. \square

Proposition 4 is similar to the Momentum Theorem of Milgrom, Qian and Roberts (1991). This asserts that the underlying force for momentum in the development of what they refer to as a manufacturing system is knowledge accumulation. Once a system starts along a path of growth in core variables¹⁸, this process will continue indefinitely until some exogenous forces disturb the system. Consistent with result *ii)* in Proposition 4, a steady increase in participation rates has occurred over the years for MNJDCP (Wells, Hartmann and Anderson 2008), and also for the tick eradication program to control Texas fever.

¹⁸ Here core variables refer to the variables that are mutually complementary.

Effect of Program Effectiveness on Premium

To this point we have assumed that the program does not improve the herd's health status of the herd. We will refer to a program that only generates information on r_t in period t as the baseline program. In a technologically effective program, the act of participation will improve an initial disease-free rate r_t to $\lambda(r_t) \in [0,1]$, where $\lambda(r_t) \geq r_t, \forall r_t \in [0,1]$ and $\exists r_t \in [0,1]$ for which $\lambda(r_t) > r_t$. Define the premium under the technologically effective program in period t as $\tilde{I}_t \equiv \tilde{I}(\tilde{r}_t^S)$, and the premium under the baseline program in period t as $I_t \equiv I(r_t^S)$. Next in Lemma 2 we will compare those two premiums assuming that $\tilde{r}_t^S \leq r_t^S$ always hold.

Lemma 2: If $\tilde{r}_t^S \leq r_t^S$, then $\tilde{I}_t > I_t$.

Proof: By Proposition 1 we have $\tilde{I}_t \equiv \tilde{I}(\tilde{r}_t^S) \geq \tilde{I}(r_t^S)$ whenever $\tilde{r}_t^S \leq r_t^S$. Under the effective program at least producers with $r_t \geq r_t^S$ can get the price premium as $\lambda(r_t) \geq r_t, \forall r_t \in [0,1]$ and $\exists r_t \in [0,1]$ for which $\lambda(r_t) > r_t$. Therefore:

$$\begin{aligned} \tilde{I}_t &\geq \tilde{I}(r_t^S) > \gamma \int_{r_t^S}^1 (\lambda(r_t) - r_t^S) dF(r_t) \\ &\geq \gamma \int_{r_t^S}^1 (r_t - r_t^S) dF(r_t) = I_t(r_t^S) \equiv I_t. \end{aligned}$$

□

Under Assumption 2 we can show that the price premium of the effective program will be no less than that of the baseline program in every period.

Proposition 5: Compared to the baseline program, the effective program always generates a greater expected price premium if Assumption 2 holds. That is $\forall t \geq 1, \tilde{I}_t > I_t$.

Proof: As $\tilde{r}_0^S = r_0^S$, by Lemma 2 we know $\tilde{I}_0 > I_0$. By Lemma 1, it implies that $\tilde{\eta}_1 \geq \eta_1$, which

further leads to $\tilde{r}_1^S \leq r_1^S$ by Proposition 3.

By Proposition 4, part *i*), we know that $\forall t \geq 1, \tilde{r}_t^S \leq r_t^S$ whenever assumption 2 holds. By applying Lemma 2 again, we have $\forall t \geq 1, \tilde{I}_t > I_t$. \square

A Cobweb Version

We could present our model in a manner tantamount to cobweb model, which shows dynamically how price and quantity converge to the equilibrium. Here we will show how the participation rate and disease free rate of the non-disclosing producers converge to the equilibrium. At period t the participation rate η_t is determined by the disease free rate of the non-disclosing producers at the previous period, r_{t-1}^S . That is $\eta_t = g(r_{t-1}^S), \forall t \geq 1$. Here $g(r_{t-1}^S) \equiv G(I(r_{t-1}^S))$, meaning that $g(\cdot)$ is contingent on producers' participation cost distribution. By Proposition 1, it follows immediately that $g(\cdot)$ is decreasing in r_{t-1}^S .

Now given participation rate η_t , r_t^S is determined at period t as $r_t^S = h(\eta_t)$
 $\equiv \pi(\eta_t)E(r | r \leq r_{t-1}^S) + (1 - \pi(\eta_t))E(r)$ where $\pi(\eta_t) = \eta_t F(r_{t-1}^S) / [\eta_t F(r_{t-1}^S) + 1 - F(r_{t-1}^S)]$
 whenever $\eta_t \neq 1$. If $\eta_t = 1$, then $r_t^S = h(1) = \underline{r}$. Here $r_{t-1}^S = g^{-1}(\eta_t)$. We can easily see that function $h(\cdot)$ is decreasing in η_t whenever Assumption 2 is true. Here $h(\cdot)$ is contingent on producers' disease free rate distribution.

Now we have two functions in the system, namely $\eta = g(r^S)$ that is decreasing in r^S and $r^S = h(\eta)$ that is decreasing in η . These two functions are represented by two downward sloping lines. The relative slopes of those two lines could not be easily compared. However, we could easily check the intercepts of these two functions on the two axes. The original point of the coordinate plane is $(r^S, \eta) = (\underline{r}, 0)$. The intercepts of $g(\cdot)$ and $h(\cdot)$ are $g(\underline{r})$ and

$h^{-1}(\underline{r})$ on the r^S -axis, and $g^{-1}(0)$ and $h(0)$ on the η -axis. Those intercepts satisfy the following two properties: 1) $g(\underline{r}) \equiv G(I(\underline{r})) \leq 1 = h^{-1}(\underline{r})$; and 2) $g^{-1}(0) \geq h(0)$, implied by $g(h(0)) = g(r_0^S) \geq 0$ and that the inverse function of $g(\cdot)$ is decreasing.

Based on these two properties, we could have three different cases: i) $g^{-1}(0) = h(0)$; ii) $g^{-1}(0) > h(0)$ and $g(\underline{r}) = h^{-1}(\underline{r}) = 1$; iii) $g^{-1}(0) > h(0)$ and $g(\underline{r}) < h^{-1}(\underline{r}) = 1$. Using linear approximations of those two functions, we could obtain three figures for those three cases. In the case of i) the NP equilibrium will be immediately reached, as illustrated in Figure 3 (a). This is because $g^{-1}(0) = h(0)$ implies $g(h(0)) = g(r_0^S) = 0$, therefore the participation rate will be zero in the first period and will continue to be so without any exogenous change. For the case of ii) the FP equilibrium will be reached, as displayed by Figure 3 (b). Since a percentage of $g(r_0^S) > 0$ producers will participate in the first period, it is clear from equation (3) that $r_1^S < r_0^S$. As a result $\eta_1 > \eta_2$ since $g(\cdot)$ is decreasing. This process will continue until $\eta^* = g(\underline{r}) = 1$. Case iii) is shown in Figure 3 (c) where a PP equilibrium is reached. The same process of case ii) will repeat here except that $\eta^* = g(r^*) \leq g(\underline{r}) < 1$.

Suppose the initial average disease free rate $r_0^S = h(0)$ decreases, then we can expect case i) to be transformed into either ii) or iii). This means more producers will participate if the disease is more serious in the system. On the other hand, the cost structure could determine the intercepts of $g(\cdot)$. When the lowest cost \underline{c} decreases, we can expect participation rate at period 1, $g(r_0^S)$ to increase. This means the intercept on the r^S -axis, $g^{-1}(0)$, will increase. When the highest cost \bar{c} increases, we can expect the intercept on the η -axis, $g(\underline{r})$, to decrease. Therefore, when the cost structure becomes more heterogeneous, we can expect the intercept of $g(\cdot)$ increases on the r^S -axis, while decreases on the η -axis. Therefore Figure

3(c) is more likely to represent such a case, where PP is more likely to be the outcome. This is consistent with our conclusion based on Figure 2 in the previous section.

MANDATORY PROGRAM

A mandatory program requires all the producers to participate. However, the incentive to voluntarily participate in mandatory programs is often overlooked. This is a pity because mandates are likely to be most unpopular and likely ineffective when the post-mandate equilibrium involves a large fraction of involuntary participants. In this section we will show that the premium reaches a maximum when a program is mandatory. Therefore a mandate creates the greatest incentive for producers to remain in the program, given the strategic complementarity of participation decisions.

Assume that there is no non-participant in the mandatory program. Again by Grossman and Hart (1980), the average disease-free rate among silent producers will be \underline{r} . This means that even though disclosure is voluntary, it comes naturally as a by-product of the mandatory program participation requirement. By (4) the expected participation premium is:

$$I(\underline{r}) = \gamma \int_{\underline{r}}^1 (r - \underline{r}) dF(r) \quad (8)$$

By Proposition 1, we know that $I(\underline{r}) = \max_{r^e} I(r^e)$, where $r^e \in [\underline{r}, E(r)]$ is defined by the equilibrium condition provided in equation (6). Therefore, the premium under a mandate will be no less than that in a voluntary program.

There could be two types of participants in a mandatory program. Motivated type A participants incur a lower cost than the premium under full participation, so they have incentives to participate. The remainder belong to unmotivated type B; they participate only when the government spends effort auditing and imposes a fine for non-participants. According to this definition, we will present three possible cases of equilibrium as shown in Figure 3. These are when:

1. $\underline{c} \leq I_{\max} \leq \bar{c}$. This cost structure is represented by area M1, where there exist a mixture of motivated type A and unmotivated type B participants in the market. Participants with $c \in [\underline{c}, I_{\max})$ are motivated types while those with $c \in (I_{\max}, \bar{c}]$ are unmotivated types;
2. $\underline{c} \geq I_{\max}$. This is represented by area M2, where the market is comprised solely of type B participants;
3. $\bar{c} \leq I_{\max}$. This is represented by area M3, where the market is comprised solely of motivated types.

Note that region M3 in Figure 4 contains exactly regions R4, R5 and R6 in Figure 2. This means that only type A participants exist in the alternative mandatory program whenever FP is an equilibrium in the voluntary program. This is because a mandatory program generates maximum incentives for producers to join. According to our calculations in Example 1, we know that this example will be located in region M3 in a mandatory program. Therefore the market is comprised solely of type A participants. No participant will deviate from participation even if the government spends no effort on auditing.

When the cost structure lies in region M1 in Figure 3, which is most likely when cost structures vary widely (as in the U.S. cow-calf production sector) in reality, then opposition from some producers is inevitable. For example, the BTB eradication program encountered continuous resistance during its implementation (Olmstead and Rhode 2007). Opposition was also intense to the Texas fever tick eradication program (Strom 2000) and more recently to the NAIS (Anderson 2010).

SIMULATION

In this section we will use Johne's disease as an example for simulation purposes. One objective of this section is to understand whether the current participation rate will increase in the long run or stagnate, an important factor to judge whether a voluntary program could

be successful or not. We will also calculate the percentage of motivated type A participants under the alternative mandatory program. Sensitivity analyses will be carried out to see how certain parameter changes might affect our results.

Similar to Example 1, we will use uniform distributions to capture a producers' cost structure and herd disease-free rate. The values of the model parameters are based on the current literature on Johne's disease. This renders the theoretically defined equilibria under voluntary and mandatory programs calculable. We assume throughout the simulations that the average disease-free rate is uniformly distributed as $r \sim U[\underline{r}, 1]$ and participation cost is uniformly distributed as $c \sim U[\underline{c}, \bar{c}]$.

Voluntary program

By (4), in the original program the expected premium in period t is calculated as:

$$I_t = I(r_t^S) = \gamma \int_{r_t^S}^1 (r - r_t^S) dF(r) = \gamma \int_{r_t^S}^1 \frac{r - r_t^S}{1 - \underline{r}} dr = \frac{\gamma(1 - r_t^S)^2}{2(1 - \underline{r})}. \quad (9)$$

For simplicity, we will choose a linear technology that satisfies the definition of a technologically effective program as $\lambda(r) = 0.5(1 + r)$. This technology is appealing because it reflects decreasing marginal returns in that the increase in disease-free rate is $0.5(1 + r) - r = 0.5(1 - r)$, which is decreasing in the value of r .

The expected premium in period t in the technologically effective program is:

$$\begin{aligned} \tilde{I}_t = \tilde{I}(\tilde{r}_t^S) &= \gamma \int_{\lambda^{-1}(\tilde{r}_t^S)}^1 [\lambda(r) - \tilde{r}_t^S] dF(r) = \frac{\gamma}{1 - \underline{r}} \int_{2\tilde{r}_t^S - 1}^1 (0.5r + 0.5 - \tilde{r}_t^S) dr \\ &= \frac{\gamma}{1 - \underline{r}} (1 - \tilde{r}_t^S)^2. \end{aligned} \quad (10)$$

By (5), the disease-free rate among silent sellers in period $t + 1$ is:

$$\begin{aligned}
r_{t+1}^S &= \phi(r_t^S)E(r | r \leq r_t^S) + (1 - \phi(r_t^S))E(r); \\
\phi(r_t^S) &= \frac{G(I_t)F(r_t^S)}{G(I_t)F(r_t^S) + 1 - G(I_t)}; & G(I_t) &= \frac{I_t - \underline{c}}{\bar{c} - \underline{c}}; & F(r_t^S) &= \frac{r_t^S - \underline{r}}{1 - \underline{r}}; \\
E(r | r \leq r_t^S) &= \int_{\underline{r}}^{r_t^S} \frac{r}{r_t^S - \underline{r}} dr = 0.5(r_t^S + \underline{r}); & E(r) &= \int_{\underline{r}}^1 \frac{r}{1 - \underline{r}} dr = 0.5(1 + \underline{r}).
\end{aligned} \tag{11}$$

From an initial value of $r_0 = E(r)$ we calculate premium I_0 by either (9), when considering the baseline program, or (10), when considering the technologically effective program. Next when r_0 and I_0 are known, r_1 will be solved by use of (11). For $t \geq 1$, I_t and r_{t+1} can be computed in turn. Lastly, participation rate $G(I_t)$ can be obtained in each period given I_t .

Mandatory program

By (8), the expected premium from participation is:

$$I(\underline{r}) = \gamma \int_{\underline{r}}^1 \frac{r - \underline{r}}{1 - \underline{r}} dr = 0.5\gamma(1 - \underline{r}). \tag{12}$$

Thus the fraction of motivated type A participants is:

$$G(0.5\gamma(1 - \underline{r})) = \frac{0.5\gamma(1 - \underline{r}) - \underline{c}}{\bar{c} - \underline{c}}. \tag{13}$$

Parameter Values

Average milk cow prices¹⁹ for replacement cows in 2006, 2007 and 2008 were \$1,730, \$1,830 and \$1,950, respectively (USDA 2010b). For the years 2009 and 2010, we will use the first quarter milk cow prices as provided by USDA: NASS, which are \$1,630 and \$1,340 respectively. Based on these statistics, we will estimate the value of a healthy dairy cow as the simple average of prices over the five most recent years, i.e., $V = \$1,696$. For cows

¹⁹ These are simple averages of quarterly prices, by States, weighted by the number of milk cows on farms on Jan. 1 of that year.

infected with Johne's disease, the estimated slaughter value is assumed to be in the range 0-30% of the original value (Groenendaal and Galligan 2003). So we assume that

$$\alpha = (0 + 0.3) / 2 = 0.15 .$$

It is estimated that the average within-herd prevalence in the United States is about 5.5% (USDA 2005). Therefore the average disease-free rate is $E(r) = 0.945$. As we assume that the herd disease-free rate satisfies a uniform distribution on $[\underline{r}, 1]$, it immediately follows that $\underline{r} = 0.89$. The cost of the JD control program ranged from \$5.79 to \$81.07 per cow per year (Pillars et al. 2009). Thus we assume that the cost satisfies a uniform distribution on $[\$5.79, \$81.07]$.

Results

According to the discussion on parameters in the last section, we have $r \sim U[0.89, 1]$, $c \sim U[\$5.79, \$81.07]$, $V = \$1,696$ and $\alpha = 0.15$. By (9) and (11) we can obtain the participation rates under the voluntary original program for the first 20 periods as displayed in Figure 5. Under the voluntary program, we can see that the participation rate increases slightly in the beginning and then stabilizes at an equilibrium participation rate of below 20%. This means that the current VJDHSP is unlikely to attract the majority of producers.

Under the alternative mandatory program which generates the greatest incentives, there will be a mix of both types of participants where 80% are content with the mandate and 20% are discontent, as obtained from (13). This means that the current voluntary program is located in region R1 of Figure 2 and that its mandatory alternative is located in region M1 of Figure 4. Therefore FP could not be an equilibrium in the voluntary program. A FP could only be achieved in the mandatory program if program implementation expenditures such as auditing cost should be incurred. However, the alternative mandatory program is likely to be

resisted by the remaining 20% of producers who do not have the incentive to participate.

Sensitivity analyses

Next we will carry out sensitivity analyses with regard to \underline{r} , α and V on the voluntary participation program. Other parameters fixed, we obtain the participation rates for the first 20 periods as shown in Figure 6(a) by choosing α among $\{-1, 0.6, 0, 0.3\}$. Similarly, we choose $\underline{r} \in \{0.8, 0.82, 0.85, 0.95\}$ and the participation rate dynamics are provided in Figure 6(b). Finally, we choose if $V \in \{\$1,300, \$1,500, \$1,700, \$1,900\}$ and simulations are displayed in Figure 6(c).

The results in Figure 6(a) follow from Proposition 1, where a lower α indicates a larger premium in the initial period and thus a greater participation rate during period 1. Next, by the strategic complementarity property proved in Proposition 3 we know that the equilibrium participation rate will increase as well. Similarly, Figures 6(b) and 6(c) could be explained by a combination of propositions 1 and 3.

From Figure 6(a) we obtain a sense of how the consequences of Johne's disease will affect participation rates in a voluntary program. For example, if the harm to human health is still unknown and the main loss comes from decreasing productivity, then we can assume that $\alpha = 0.3$. In this case the participation rate stabilizes at a rate lower than 20%. However, if Johne's disease is proved to be connected to Crohn's disease, then α is likely to be negative. In cases where $\alpha \in \{-1, -0.6\}$, FP equilibrium will be reached.

Figure 6(b) indicates that the equilibrium participation rate could be influenced by producer's beliefs on within-herd disease prevalence. While our simulations have less than 10% of producers participating when average prevalence level is 2.5%, FP equilibrium will be realized if the average prevalence rate reaches 10%. Increased unit cow value will also

enhance the equilibrium participation rate, but to a very limited extent because the value range is narrow. From Figure 6(c) we can see that, all else fixed, FP equilibrium will not be reached even if the unit value increase to \$2,500. The effect of unit animal value on participation rate can also explain why the participation rate for beef herds is much lower than that for dairy herds (Wells, Hartmann, and Anderson 2008).

A more general comment in this figure is that participation rates, and so eventually disease-free rates, increase as value to be protected increases. This value could take the form of non-stock production assets such as feeding, housing and manure management investments. This provides another form of complementarity; namely between a particular grower's asset value at risk and the participation decision rather than between grower participation decisions. But the two forms of complementarity will themselves interact in a complementary manner. That is, regions with a preponderance of competitive species X growers will have high asset values to protect and will participate. This will encourage others in the region to participate and high region-wide participation rate is likely to promote investment in the region's species X production sector. On the other hand, if a region has a marginal species X production sector then there will be low incentive to participate and this just confirms the region's low sector productivity.

Subsidy and Tipping

The effect of tipping will be illustrated in this section. Suppose that the government provides temporary subsidies to a proportion of producers. These subsidies may eventually motivate all the producers to participate in the voluntary program. Due to our momentum inference in Corollary 2, subsidies will no longer be necessary after a certain proportion of producers participate. We assume that $r \sim U[0.89,1]$, $c \sim U[5.79,81.07]$, $V = \$1,696$, and we compare $\alpha = 0$ with $\alpha = -0.4$.

Figure 7(a) displays the case where $\alpha = 0$. The equilibrium without subsidy will be reached in around the 5th period, where 29 percent of producers participate and the price premium is \$27 for participation. In the 6th period, suppose the government could provide subsidies to 30 percent of producers. Specifically, suppose the government could subsidize all producers whose cost lies in the upper 30% percentile of the cost distribution, i.e., $c \sim U[58.47, 81.07]$ so that their cost will fall below \$27. So long as low cost participants do not believe that they would become eligible for subsidies were they to procrastinate then the timing of the subsidies will not affect their participation decision.

In reality, as the smaller producers are more likely to incur higher participation costs, government could provide subsidies to 30% of the smallest producers once it is realized that the participation rate is leveling off. The uniform subsidy could be set as \$55 to ensure that even the highest cost producers incur a cost less than \$27 after the subsidy. Then the participation rate will climb again and the new full participation equilibrium will be reached after another 13 periods. The FP equilibrium will generate a price premium of \$85. Thus no producer in the FP equilibrium has the incentive to deviate from it even without a government subsidy. An alternative approach that could more quickly and more reliably secure an high participation rate would be to subsidize the participation costs of the smallest producers right from the outset.

Figure 7(b) displays the case where $\alpha = -0.4$. The equilibrium without subsidy will settle in around the 6th period, with a 54% participation rate and \$46 price premium. In the 7th period, suppose the government could subsidize all producers whose cost lies in the upper 10% percentile of the cost distribution, i.e., $c \sim U[73.54, 81.07]$ so that their cost will fall below \$46. This could be a uniform subsidy of \$36 to the smallest 10% of producers. Then after another seven periods the FP equilibrium will be reached, generating a price premium of \$130.

This result is consistent with the results in the sensitivity analyses section. That is, as α decreases only reduced subsidies to a smaller percentage of producers are needed to tip the PP equilibrium to a FP one. In the extreme, no subsidy will be needed when the value of α is sufficiently low. Similar results hold when either V increases or \underline{r} decreases.

Cost Heterogeneity

Next we will simulate the effect of participation cost heterogeneity. We will compare $c \sim U[\$5.79, \$81.07]$ with a distribution that is a mean-preserving decrease in dispersion, $c \sim U[\$15.79, \$71.07]$. We will also choose two different values for α , namely -0.5 and 0.3. The resulting participation rates are displayed in Figure 8(a) and 8(b), respectively. In the case of a less heterogeneous cost structure, the equilibrium participation rate is either close to 0% or 100%; while a more heterogeneous cost structure results in the PP equilibrium. When the participation premium increases relative to cost and costs are less heterogeneous then FP will result. This simulation result conforms with our observation based on Figure 2.

Program Effectiveness

From equations (10) and (11) we can obtain the participation rates under the technologically effective program for the first 20 periods. Compared to the baseline program, the effect of program effectiveness on program participation is displayed in Figure 9. When the program is effective in increasing the herd level disease-free rate, the participation rate will increase in every period. The equilibrium participation rate will also increase. In our case a FP is reached for the effective program. Intuitively, this is because if the program proves to be more effective then sellers could expect to obtain a larger price premium and this would strengthen participation incentives among sellers.

CONCLUSION

This paper contributes to the existing literature on voluntary programs in the following regards. Participation incentives are modeled at an industry level and in a dynamic setup. This allows to show that, due to strategic complementarity, momentum can build where both premium and participation rate increase iteratively in a mutually reinforcing manner and may in time support full participation even without government subsidies. This is in contrast with much of the current literature on infectious animal disease management programs, which implicitly assume that incentives for voluntary participation come either from direct productivity effects or government subsidies.

Secondly, we show that the participation premium is contingent on disease consequences to human health, disease prevalence rate and the unit livestock value. Therefore participation rates may differ when these characteristics change. For example, if consumers perceive a serious human health consequence or producers believe the disease to be highly prevalent, then a full participation equilibrium could be more readily attained.

We point out that due to the strategic complementarity property, the mandatory program maximizes the price premium and thus the incentive to participate. The private benefits to participation will be larger when calculated after the mandate is implemented. As our historical review attests, many initially hostile to a mandate may think differently afterwards. Theoretically, a sticks approach can dominate the carrots approach in regard to participation incentives. However enforcing the mandate might be difficult at the outset and when heterogeneity in implementation cost is large then many producers may never benefit from the mandate. A voluntary program with modest government subsidies might be welcome by all producers, perhaps in part because of the positive publicity generated (Lyon 2003). It would also place some of the costs on taxpayers/consumers who may be happy to pay for

greater confidence in the food supply chain. A voluntary approach may also afford producers with sufficient time to appreciate program benefits and initiate a virtuous cycle at low political cost.

Finally, as demonstrated in the simulation section, our theoretical model can also be tailored for simulation purposes. This allows us to predict under given situations how the program participation rate may evolve over time, a new feature that has not been explored in the existing literature. Take Johne's disease as an example. Our simulation results indicate that full participation is unlikely under the status quo. However, if the disease is were shown to cause serious human health consequences that becomes reflected in livestock values then the full participation rate is more likely to be attained. To the contrary, an underestimation of the disease prevalence rate could deter producers from participation. We also illustrate the process of tipping in the simulation, where the government could secure a FP equilibrium by subsidizing a small proportion of high cost producers to participate. Therefore the success and cost effectiveness of a voluntary Johne's control program, or of any livestock disease control program in general, hinges crucially on obtaining the right statistics and educating the producers and consumers accordingly.

A shortcoming of this paper is that animal health benefits are accounted for in a very reduced form manner. This feature might be improved upon, but at some loss in model tractability. Omission of animal health benefits in our simulations cause an underestimation of the program's true benefit. Future developments could strengthen these model features.

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APPENDIX

Effect of test sensitivity on premium

In reality tests may only identify a small group of diseased animal. For example, sensitivity of Enzyme-linked immunosorbent assay (ELISA) tests are assumed to be 25% and that of fecal cultures are assumed to be 40% (USDA, 2010a). Jubb et al. (2004) defines ELISA sensitivity measure as " $T_n = A / (A + B)$ where $n =$ Test round, $A =$ Number of reactors at T_n and $B =$ Number of animals that tested negative at T_n but tested positive at subsequent tests." Following Jubb et al. (2004), we will define the test sensitivity as the number of animals that tested positive divided by the number of true diseased ones.

Next we will show in Proposition A1 that as long as test sensitivity is common knowledge, it will not have an effect on the expected premium from participation.

Proposition A1: Test sensitivity will not affect the expected price premium from participation.

Proof. Assume D percent of the animals in the herd are diseased. And the sensitivity of the test is δ , which means that only δ percent of diseased animal can be correctly identified.

Suppose δ is common knowledge among all the parties involved. Now $D' = \delta D$ percent of the animal is identified as diseased. The buyer is willing to pay $(1 - D)V + D\alpha V$ for a herd with disease free rate D when the sensitivity is 1.

Now suppose that the sensitivity of the test is δ , and the producers claim that the tested disease free rate is $1 - D'$. Then the buyer can infer that the actual disease free rate is $1 - D' / \delta$. Thus the buyer is willing to pay $(1 - D' / \delta)V + (D' / \delta)\alpha V$. This is exactly the same as the price the buyer will pay when the sensitivity is 1. \square

Validity of Assumption 2

Assumption 2 always holds if we take on the computable function form and assume that both the average disease free rate and participation cost are uniformly distributed as $r \sim U[0,1]$ and $c \sim U[0,1]$, and that $\gamma \equiv (1-\alpha)V \geq 2$.

Proof. Obviously $E(r) - E(r | r \leq r^S)$ is decreasing in r^S . Therefore, assumption 2 will automatically hold if $\phi(r^S)$ is decreasing in r^S .

$$\begin{aligned} \phi'(r^S) &= \left(1 - \frac{1 - g(r^S)}{g(r^S)F(r^S) + 1 - g(r^S)} \right)' \\ &= - \frac{-g'(r^S)[g(r^S)F(r^S) + 1 - g(r^S)] - [1 - g(r^S)][g(r^S)F'(r^S) + g'(r^S)F(r^S) - g'(r^S)]}{[g(r^S)F(r^S) + 1 - g(r^S)]^2} \\ &= \frac{g(r^S)(1 - g(r^S))F'(r^S) + g'(r^S)F(r^S)}{[g(r^S)F(r^S) + 1 - g(r^S)]^2} \end{aligned}$$

Equivalently, this is to prove :

$$g'(r^S)F(r^S) \leq -g(r^S)(1 - g(r^S))F'(r^S) \quad (\text{A-1})$$

Assume that the average disease free rate is uniformly distributed as $r \sim U[0,1]$ and participation costs satisfy the uniform distribution $c \sim U[0,1]$. Then $F(r^S) = r^S$; $F'(r^S) = 1$;

$g(r^S) = I(r^S) = \gamma \int_{r^S}^1 (r - r^S) dr = 0.5\gamma(1 - r^S)^2$ and $g'(r^S) = -\gamma \bar{F}(r^S) \leq 0$. Under these

specification we can show that condition (A-1) always holds as below.

$$\begin{aligned} -\gamma r^S(1 - r^S) / \bar{c} &\leq -0.5\gamma(1 - r^S)^2(1 - 0.5\gamma(1 - r^S)^2 / \bar{c}) / \bar{c} \\ \Leftrightarrow 2r^S &\geq (1 - r^S)[1 - 0.5\gamma(1 - r^S)^2 / \bar{c}] \end{aligned} \quad (\text{A-2})$$

Assuming that $\gamma \geq 2\bar{c}$, condition (A-2) will hold if we could prove

$2r^S \geq (1 - r^S)[1 - (1 - r^S)^2]$, which immediately holds as $2 \geq (1 - r^S)(2 - r^S)$. \square

Proof of Corollary 2.

Similar to (3) and (4), we have:

$$\begin{aligned} r_{t+1}^S &= \phi(r_t^S)E(r | r \leq r_t^S) + (1 - \phi(r_t^S))E(r) = E(r) - \phi(r_t^S)(E(r) - E(r | r \leq r_t^S)) \\ r_t^S &= \phi(r_{t-1}^S)E(r | r \leq r_{t-1}^S) + (1 - \phi(r_{t-1}^S))E(r) = E(r) - \phi(r_{t-1}^S)(E(r) - E(r | r \leq r_{t-1}^S)) \end{aligned}$$

Suppose $r_t^S \leq r_{t-1}^S$. In order to have $r_{t+1}^S \leq r_t^S$, we need the following condition to hold:

$$\phi(r_t^S)(E(r) - E(r | r \leq r_t^S)) \geq \phi(r_{t-1}^S)(E(r) - E(r | r \leq r_{t-1}^S)) \quad (\text{A-3})$$

Condition (A-3) holds when Assumption 2 is true.

Next, inference ii) holds by inference i) and Proposition 1. Based on Lemma 1 and the assumption that $\eta_0 = 0$, inference iii) follows immediately. \square

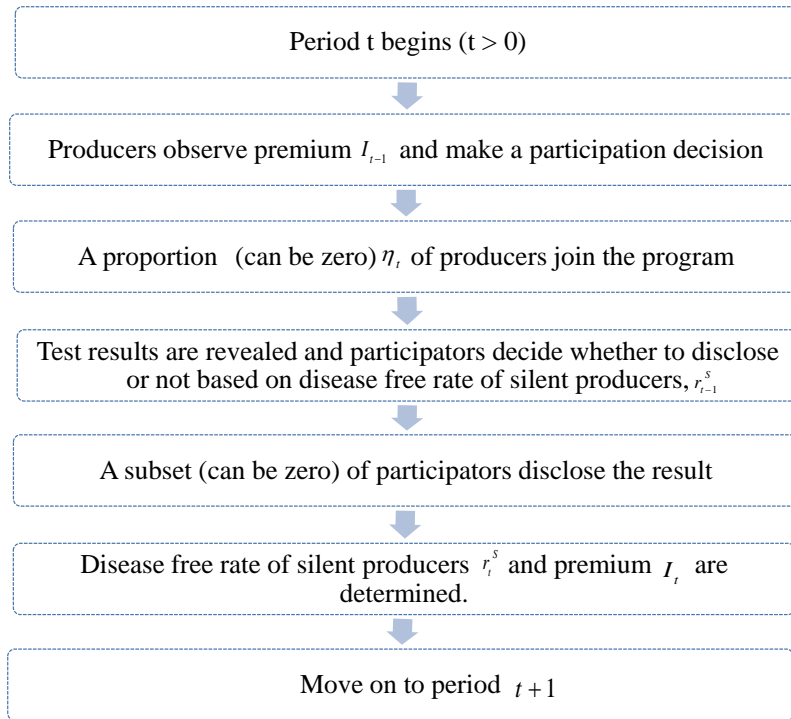


Figure 4.1. A flow chart of the model scheme.

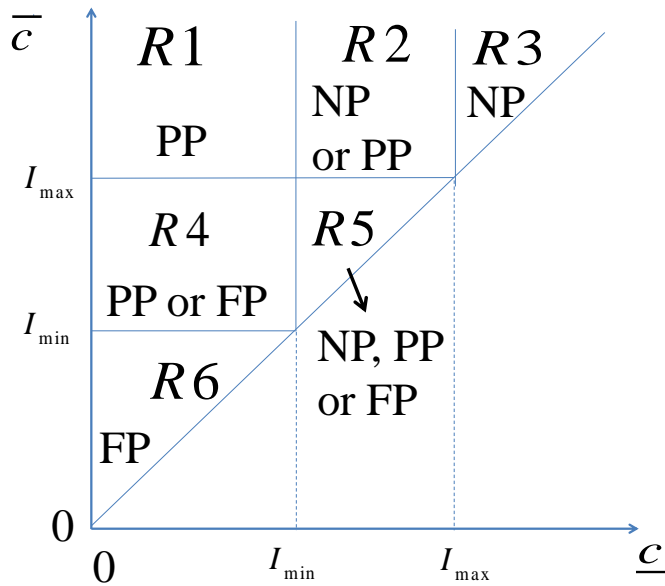


Figure 4.1. Equilibria under different cost structures—voluntary program.

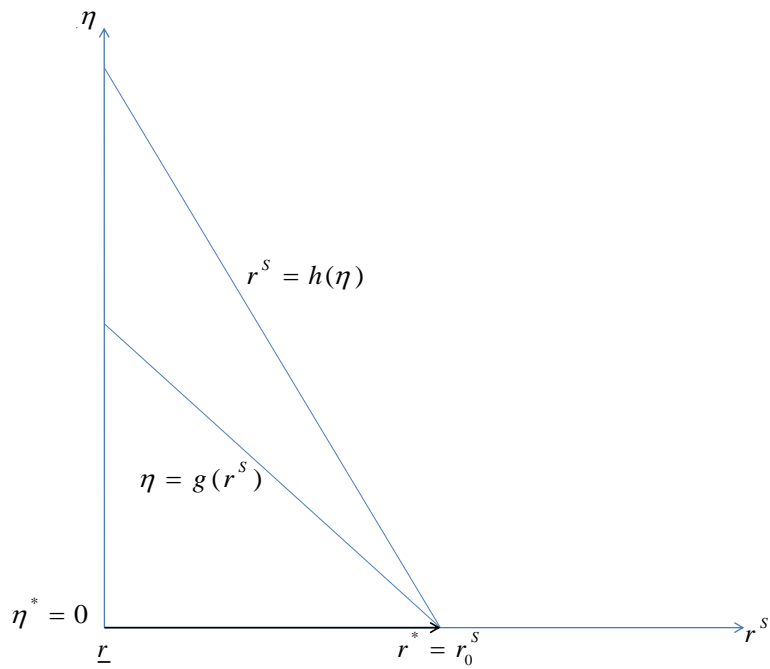


Figure 4.2 (a). Cobweb Effect---Non-Participation

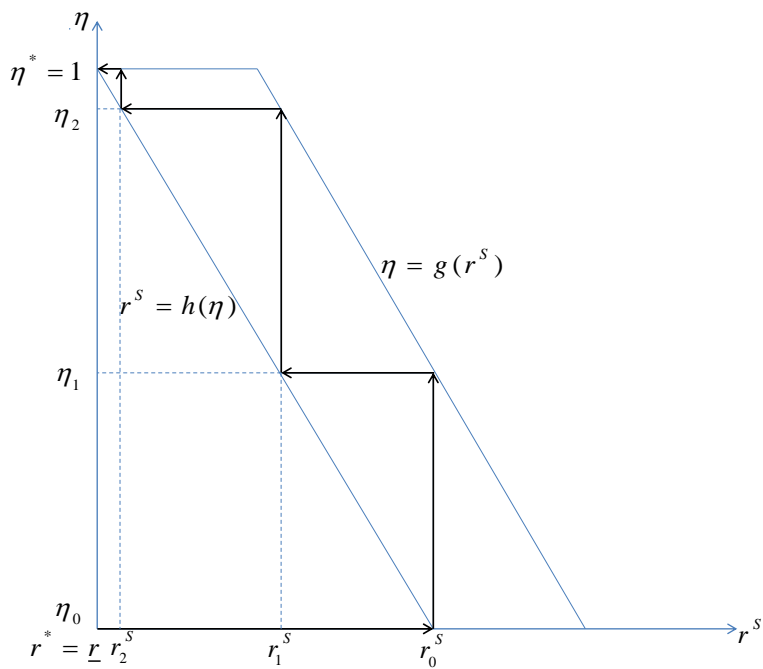


Figure 4.3(b). Cobweb Effect---Full Participation

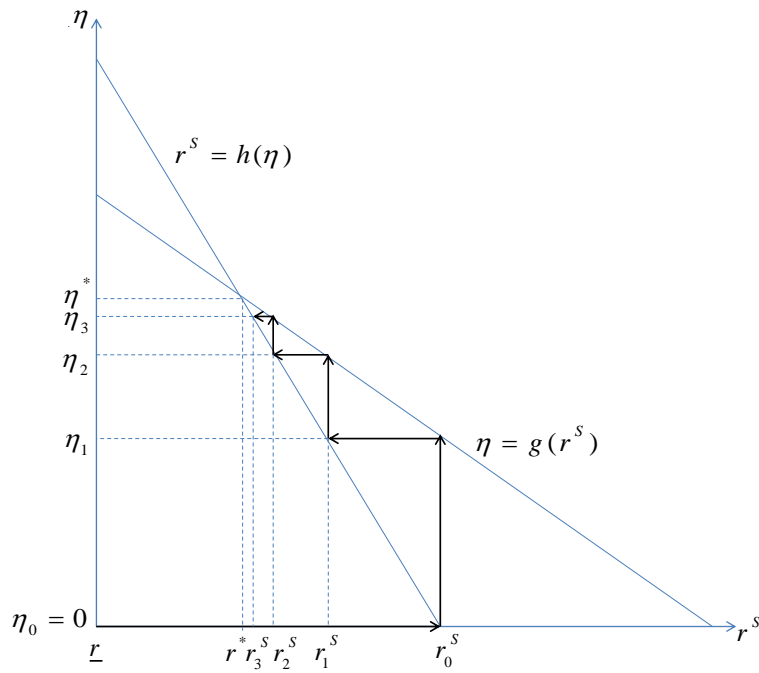


Figure 4.3 (c). Cobweb Effect---Partial Participation

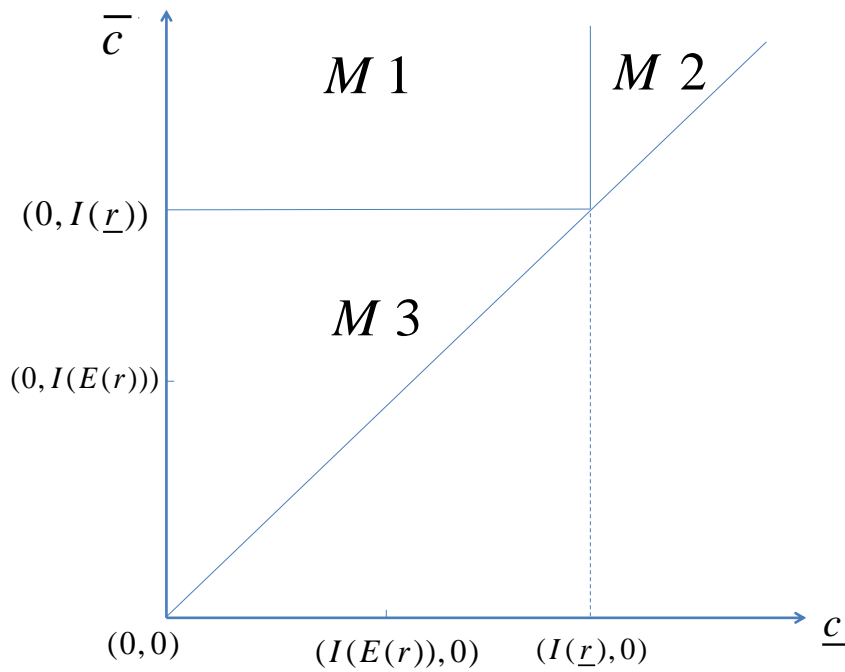


Figure 4.4. Participant Types under Different Costs---Mandatory Program

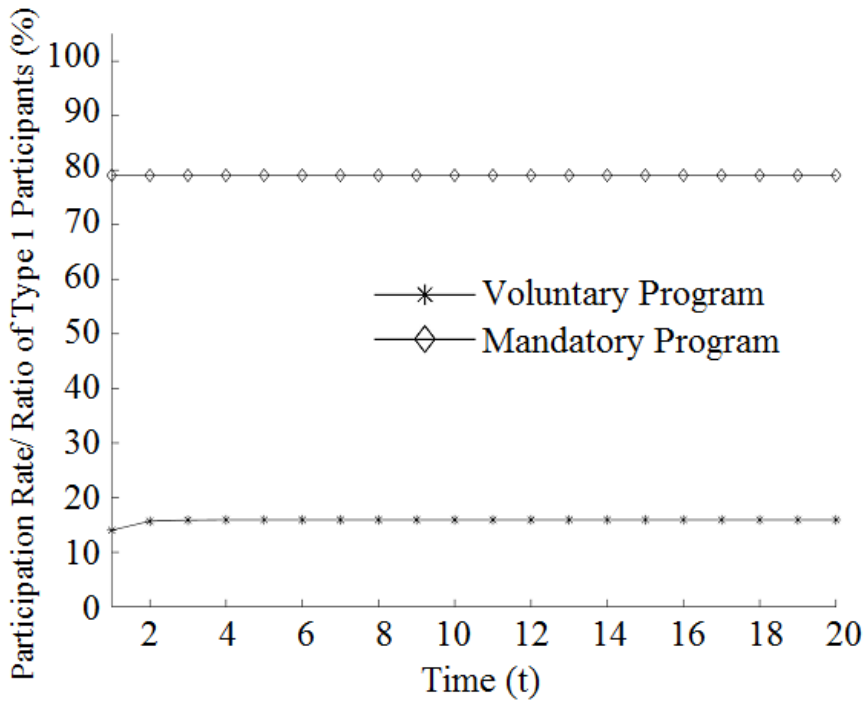


Figure 4.5. Participation rate and ratio of motivated Type A producers to all producers.

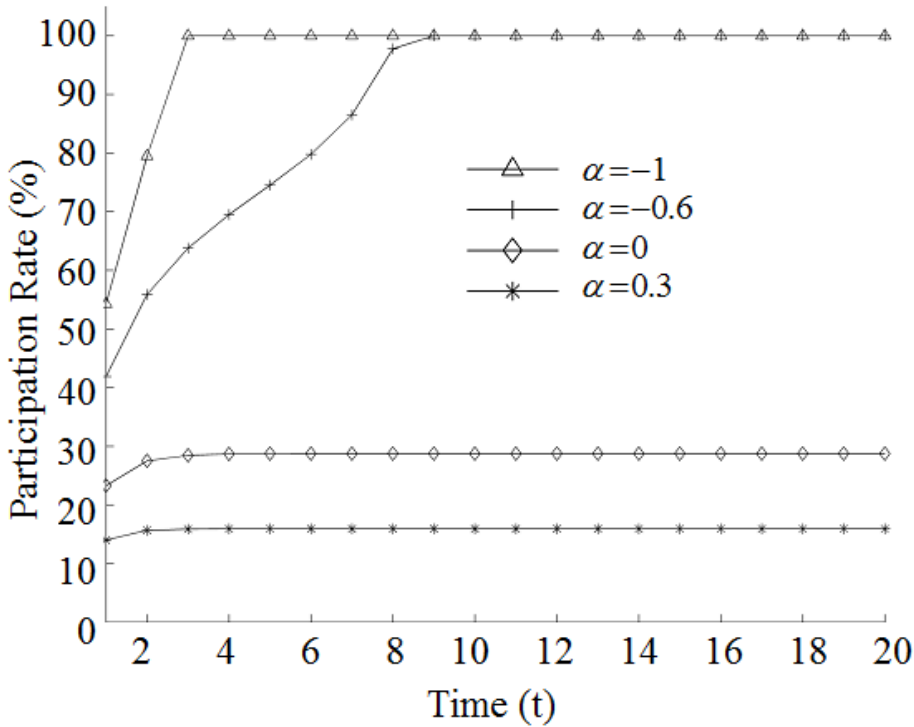


Figure 4.6 (a). Participation rates under different values of α .

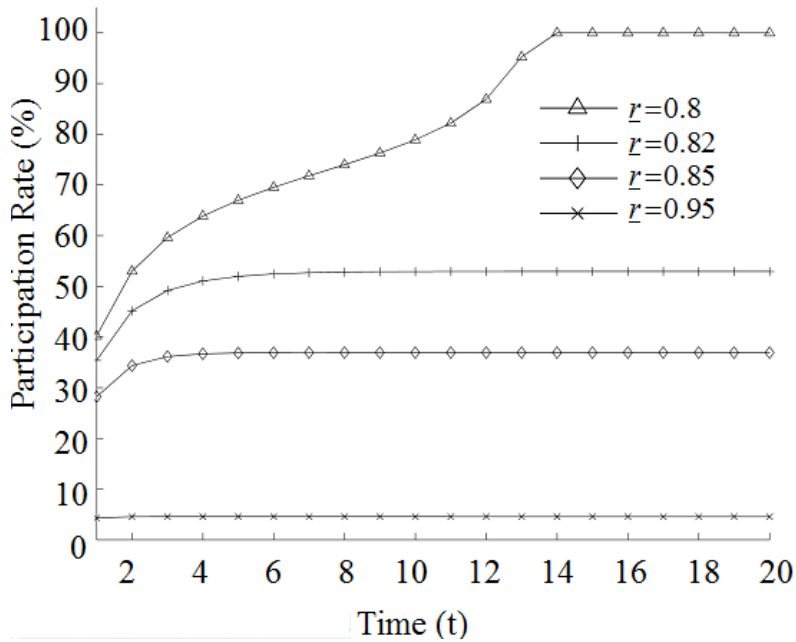


Figure 4.6 (b). Participation rates under different values of r .

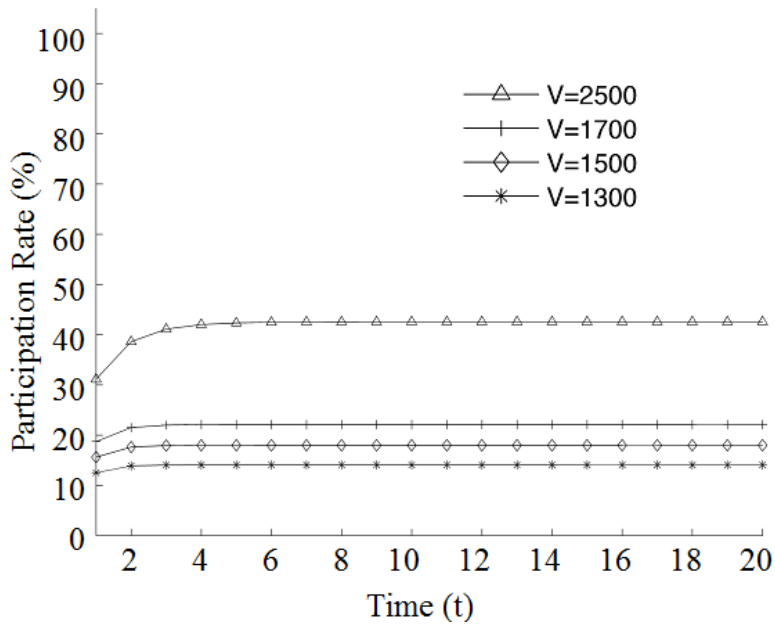


Figure 4.6(c). Participation rates under different values of V .

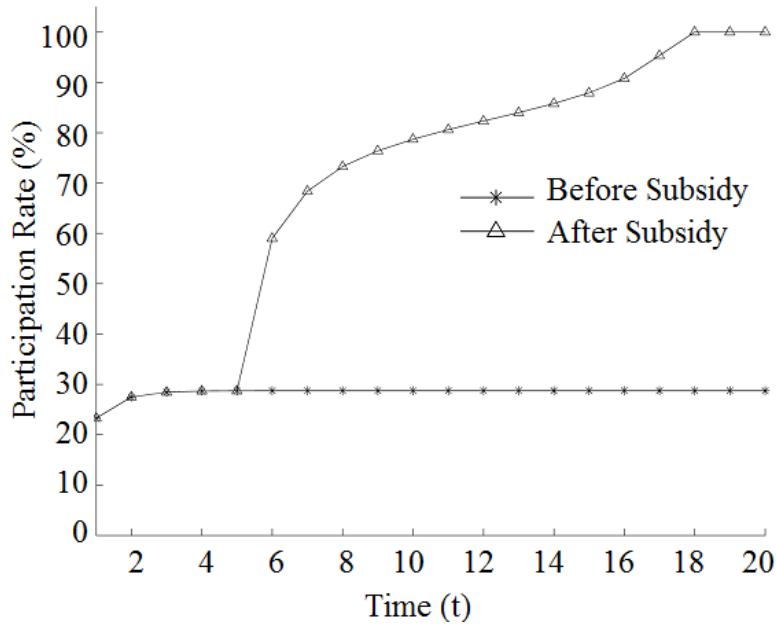


Figure 4.7(a). Subsidy and tipping— $\alpha = 0$.

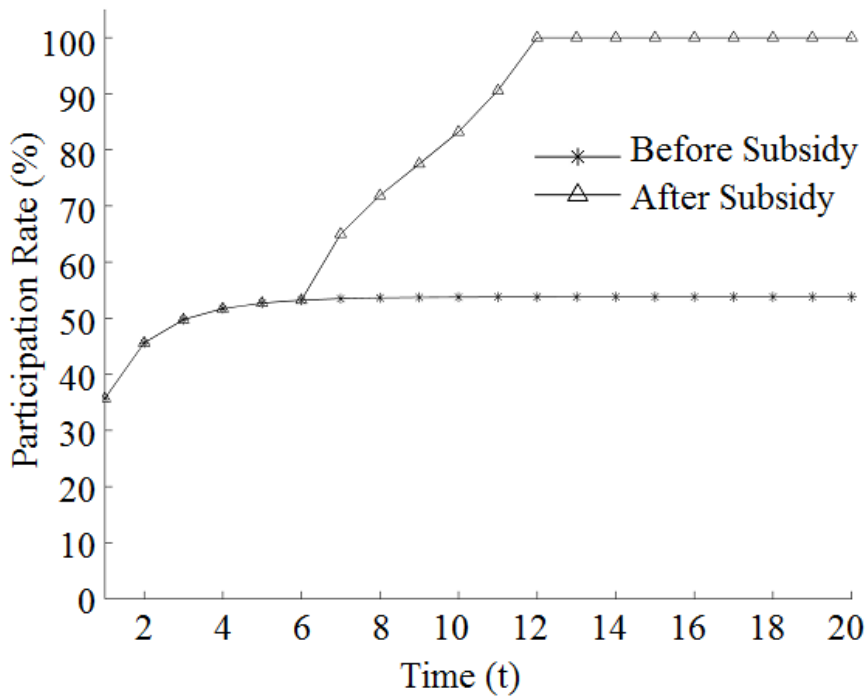


Figure 4.7(b). Subsidy and tipping— $\alpha = -0.4$.

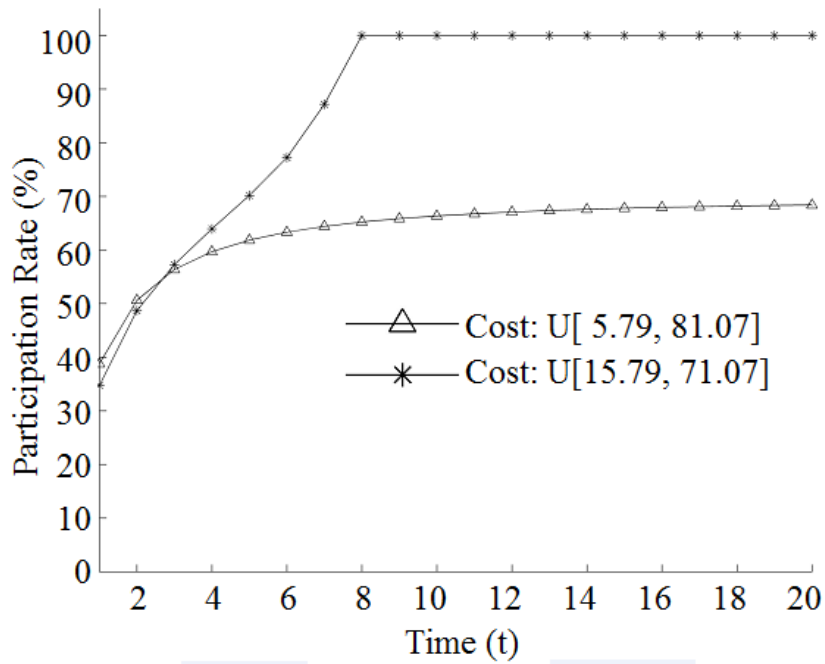


Figure 4.8(a). Cost heterogeneity— $\alpha = -0.5$.

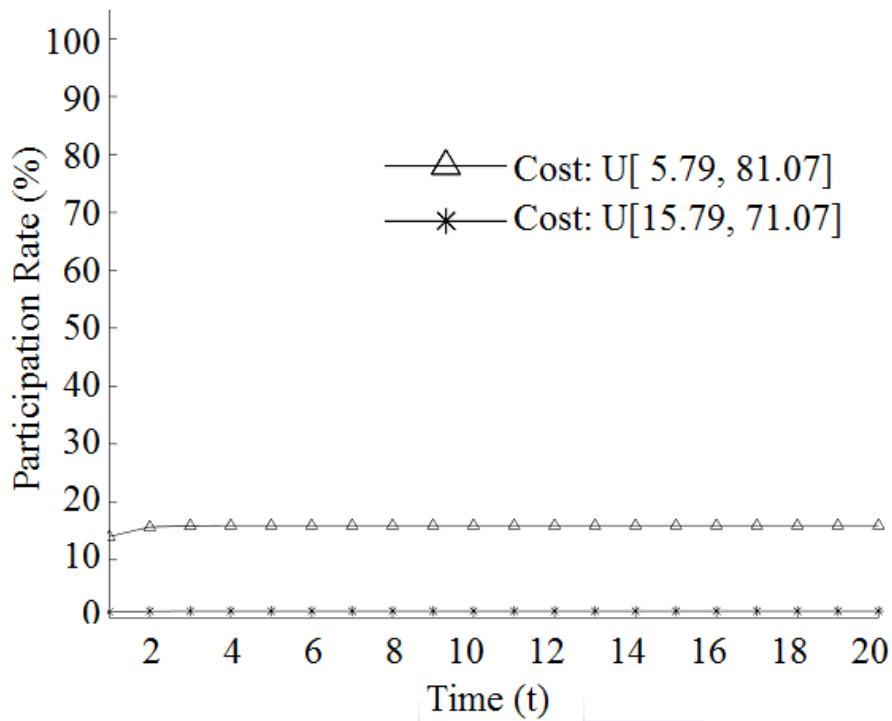


Figure 4.8(b). Cost heterogeneity— $\alpha = 0.3$.

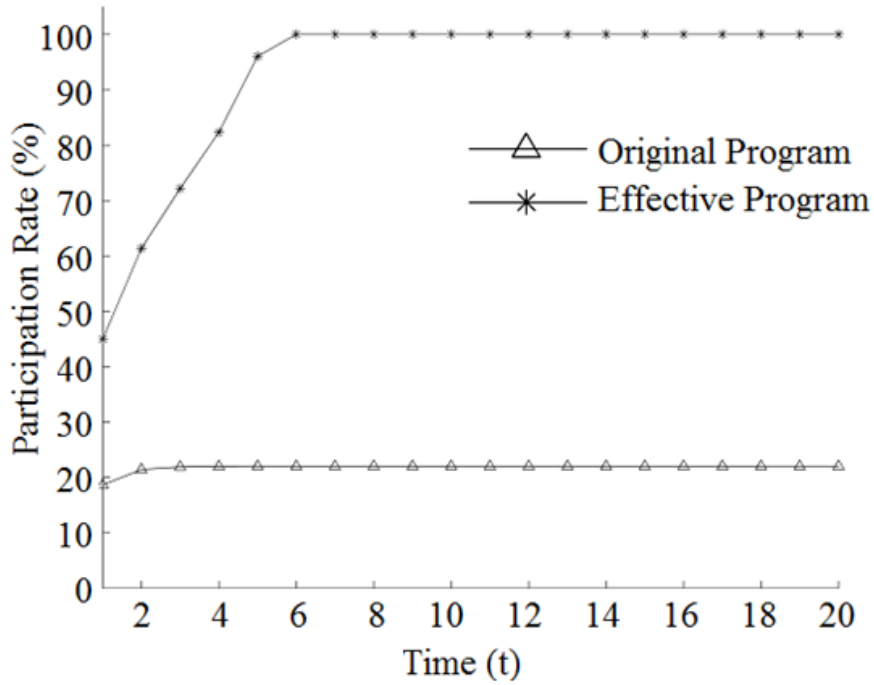


Figure 4.9. Effective program vs. baseline program.

SUPPLEMENTAL MATERIALS

Voluntary Program---Coarse Grading

In this section we will present our model under the alternative assumption of a coarse grading test. A coarse grading test only distinguishes two possible cases. Producers obtain a certificate when their disease-free rate is above a threshold \hat{r} and no certificate otherwise.

The unit livestock price of a non-participating herd is $p_t^1 = r_t^S V + (1 - r_t^S) \alpha V$, while the realized unit price of one that participates takes the following form:

$$p_t^2 = \begin{cases} r_t^S V + (1 - r_t^S) \alpha V & \text{whenever } r \leq \hat{r}, \\ E[r | r > \hat{r}] V + (1 - E[r | r > \hat{r}]) \alpha V & \text{whenever } r > \hat{r}. \end{cases}$$

Therefore the realized premium received by a producer who participates is:

$$p_t^2 - p_t^1 = \begin{cases} 0, & \text{whenever } r_t \leq \hat{r}; \\ \gamma(E[r | r > \hat{r}] - r_t^S) & \text{whenever } r_t > \hat{r}. \end{cases}$$

The expected price premium from participating is thus:

$$\begin{aligned} I_t^c(r_t^S, \hat{r}) &= \gamma \int_{\hat{r}}^1 (E[r | r > \hat{r}] - r_t^S) dF(r) + \int_0^{\hat{r}} 0 f(r) dr \\ &= \gamma(E[r | r > \hat{r}] - r_t^S)[1 - F(\hat{r})]. \end{aligned} \tag{SM-1}$$

Here $I^c(\cdot)$ represents the price premium function in the coarse grading case. Next, we will show that in the coarse grading system, price premium depends only on the lowest threshold. The number of thresholds and the value of other thresholds will not affect the premium. Therefore without loss of generality, we will focus on the single threshold case.

Proposition SMI: Suppose there are two coarse grading programs with $\hat{r}^1 < \hat{r}^2$. Program 1 offers the certificate whenever the tested disease-free rate $r > \hat{r}^1$; program 2 offers level 1 certificate whenever the tested disease-free rate satisfies $r \in [\hat{r}^1, \hat{r}^2)$, level 2 certificate whenever the tested disease-free rate satisfies $r > \hat{r}^2$. The expected premiums generated by the two programs are the same.

Proof: According to (SM-1), the expected price premium for program 1 is:

$$I^c(r_t^S, \hat{r}) = \gamma(E[r | r > \hat{r}^1] - r_t^S)[1 - F(\hat{r}^1)] \quad (\text{SM-2})$$

The realized price charged by a producer participating in program 2 takes the following form:

$$p_t^2 = \begin{cases} r_t^S V + (1 - r_t^S) \alpha V, & \text{whenever } r \leq \hat{r}^1; \\ E[r | \hat{r}^1 < r \leq \hat{r}^2] V + (1 - E[r | \hat{r}^1 < r \leq \hat{r}^2]) \alpha V, & \text{whenever } \hat{r}^1 < r \leq \hat{r}^2; \\ E[r | r > \hat{r}^2] V + (1 - E[r | r > \hat{r}^2]) \alpha V, & \text{whenever } r > \hat{r}^2. \end{cases}$$

Since the unit livestock price from non-participating is $p_1 = r^S V + (1 - r^S) \alpha V$, we can calculate the realized premium from participating in program 2 as:

$$p_t^1 - p_t^1 = \begin{cases} 0, & \text{whenever } r_t \leq \hat{r}^1; \\ \gamma(E[r | \hat{r}^1 < r \leq \hat{r}^2] - r_t^S), & \text{whenever } \hat{r}^1 < r_t \leq \hat{r}^2; \\ \gamma(E[r | r > \hat{r}^2] - r_t^S), & \text{whenever } r_t > \hat{r}^2. \end{cases}$$

The expected price premium from participating in program 2 is:

$$\begin{aligned} I_c(r_t^S, \hat{r}^1, \hat{r}^2) &= \gamma \int_{\hat{r}^1}^{\hat{r}^2} (E[r | \hat{r}^1 < r \leq \hat{r}^2] - r_t^S) dF(r) + \gamma \int_{\hat{r}^2}^1 (E[r | r > \hat{r}^2] - r_t^S) dF(r) \\ &= \gamma(E[r | \hat{r}^1 < r \leq \hat{r}^2] - r_t^S)(F(\hat{r}^2) - F(\hat{r}^1)) + \gamma(E[r | r > \hat{r}^2] - r_t^S)(1 - F(\hat{r}^2)) \\ &= \gamma[(E[r | \hat{r}^1 < r \leq \hat{r}^2])(F(\hat{r}^2) - F(\hat{r}^1)) + (E[r | r > \hat{r}^2])(1 - F(\hat{r}^2))] - \gamma r_t^S (1 - F(\hat{r}^1)) \quad (\text{SM-3}) \\ &= \gamma(E[r | r > \hat{r}^1])[1 - F(\hat{r}^1)] - \gamma r_t^S [1 - F(\hat{r}^1)] \quad (\text{By the Law of Iterated Expectations}) \\ &= \gamma(E[r | r > \hat{r}^1] - r_t^S)[1 - F(\hat{r}^1)]. \end{aligned}$$

This is exactly the expected price premium generated by program 1, as shown in (SM-2). \square

In the coarse grading case, silent producers are comprised of two groups: participants who fail to obtain the certificate and the non-participants. Similar to the equilibrium defined in the fine grading case, we will define the equilibrium under coarse grading as:

$$\begin{aligned} r^* &= \phi(r^*)E(r | r \leq \hat{r}) + [1 - \phi(r^*)]E(r); \\ \phi(r^*) &= \frac{\eta^* F(\hat{r})}{\eta^* F(\hat{r}) + 1 - \eta^*}; \quad \eta^* = G(I^c(r^*, \hat{r})). \end{aligned} \quad (\text{SM-4})$$

As a counterpart to Figure 2 in the voluntary fine grading case, Figure SM1 displays all

possible types of equilibria when cost structures differ. As an example, we will show by Proposition SM2 that there are two possible equilibria in region R5. The proof resembles that in Proposition 2.

Proposition SM2: Under the voluntary program coarse grading case, NP and FP are two possible equilibria when $I^c(E(r), \hat{r}) \leq \underline{c} \leq \bar{c} \leq I^c(E(r | r \leq \hat{r}), \hat{r})$.

We seek now to prove Proposition SM3, a counterpart to Proposition 4 in the fine grading case. In order to do so we will first show in Lemma SM1 that a counterpart of Assumption 2 always holds in the coarse grading case.

Lemma SM1: In the coarse grading case, an equivalent to Assumption 2 of the fine grading case will always hold. That is $J(r_t^S, \hat{r}) = \phi(r_t^S, \hat{r})(E(r) - E(r | r \leq \hat{r}))$ is decreasing in $r_t^S \in [\underline{r}, E(r)]$ where $\phi(r_t^S, \hat{r}) = G(I^c(r_t^S, \hat{r}))F(\hat{r}) / [G(I^c(r_t^S, \hat{r}))F(\hat{r}) + 1 - G(I^c(r_t^S, \hat{r}))]$.

Proof: As $E(r) - E(r | r \leq \hat{r})$ is a constant, we only need $\phi(r_t^S, \hat{r})$ to be decreasing in $r_t^S \in [\underline{r}, E(r)]$. This is obvious as $G(I^c(r_t^S, \hat{r}))$ is decreasing in r_t^S and $F(\hat{r})$ is fixed. \square

Proposition SM3: In coarse grading case, the following three inferences always apply: i)

Average disease-free rate of silent producers will be non-increasing over time, i.e.,

$r_0^S \geq r_1^S \geq \dots \geq r_k^S$; ii) Premium from participation will be non-decreasing over time, i.e.,

$I_0 \leq I_1 \leq \dots \leq I_k$ ($I_k \equiv I^c(r_k^S)$); iii) Participation rate will be non-decreasing over time, i.e.,

$\eta_0 \leq \eta_1 \leq \dots \leq \eta_k$.

Lastly we will briefly revisit Example 1 in the coarse grading case, where an additional threshold parameter takes value of $\hat{r} = 0.98$. It follows that:

$$\begin{aligned}
I^c(E(r_t | r_t \leq \hat{r}), \hat{r}) &= \gamma \int_{\hat{r}}^1 [E(r_t | r_t > 0.98) - E(r_t | r_t \leq 0.98)] dF(r) \\
&= 0.8 * 500 \int_{0.98}^1 \frac{[E(r_t | r_t > 0.98) - E(r_t | r_t \leq 0.98)]}{1 - 0.90} dr = 4.
\end{aligned}$$

Therefore $\bar{c} \geq \underline{c} \geq I^c(E(r_t | r_t \leq \hat{r}), \hat{r})$. Now the case of Example 1 is located in region R3 of Figure SM1, where the only equilibrium is NP and tipping will not occur. Therefore strategic complementarity as provided in Proposition SM3, may not lead to tipping. Next we will show that in a more general setup, coarse grading generates a smaller expected price premium, i.e., less incentive for producers to participate.

Proposition SM4: Assume that $\hat{r} > E(r)$. Then price premium under the coarse grading system $I^c(r_t^S, \hat{r})$ is no greater than that under the fine grading system, $I(r_t^S)$ for any given $r_t^S \in [0, 1]$.

Proof: Calculate

$$\begin{aligned}
I^c(r_t^S, \hat{r}) &= \gamma \int_{\hat{r}}^1 (E[r | r \geq \hat{r}] - r_t^S) dF(r) \\
&= \gamma E[r | r \geq \hat{r}] \int_{\hat{r}}^1 dF(r) - \gamma \int_{\hat{r}}^1 r_t^S dF(r) = \gamma \int_{\hat{r}}^1 r f(r) dr - \gamma \int_{\hat{r}}^1 r_t^S dF(r) \\
&= \gamma \int_{\hat{r}}^1 (r - r_t^S) f(r) dr \\
&< \gamma \int_{r_t^S}^1 (r - r_t^S) f(r) dr \quad (\text{By } r_t^S \leq E(r) < \hat{r}) \\
&= I(r_t^S). \quad \square
\end{aligned}$$

Note that condition $\hat{r} > E(r)$, which means that participants only obtain a certificate when their disease-free rate is above the average, is a sufficient condition for Proposition SM4 to hold. This is because $r_t^S \leq r_0^S = E(r)$ (by equation (5)), thus producers with disease-free rate $r_t \in (r_t^S, \hat{r}]$ will obtain a premium in fine grading case, but not in a coarse grading case. Thus ex-ante a producer has a lower expected premium in the coarse grading system.

Mandatory Program---Coarse Grading

The average disease-free rate for the silent producers, who are comprised solely of the producers that obtain no certificate, is $E(r | r \leq \hat{r})$. Thus by (SM-1) the expected price premium from participation is:

$$I^c(E(r | r \leq \hat{r}), \hat{r}) = \gamma[1 - F(\hat{r})][E(r | r > \hat{r}) - E(r | r \leq \hat{r})]. \quad (\text{SM-5})$$

Similar to Figure 4, Figure SM2 also displays three possible cases. These are:

1. $\underline{c} \leq I^c(E(r | r \leq \hat{r}), \hat{r}) \leq \bar{c}$. This cost structure is represented by region M1, where both types of participants exist. Those participants with $c \in [\underline{c}, I^c(E(r | r \leq \hat{r}), \hat{r})]$ are motivated type A participants, and those with $c \in (I^c(E(r | r \leq \hat{r}), \hat{r}), \bar{c}]$ are type B participants;
2. $\underline{c} \geq I^c(E(r | r \leq \hat{r}), \hat{r})$. This is represented by region M2, where the market is solely comprised of unmotivated type B participants;
3. $\bar{c} \leq I^c(E(r | r \leq \hat{r}), \hat{r})$. This is represented by region M3, where the market is solely comprised of motivated type A participants.

As $\underline{c} = 5.5 \geq I^c(E(r | r \leq \hat{r}), \hat{r}) = 4$, we know that the case in Example 1 is now located in M2. This means that the market is comprised solely of type B participants, who would choose not to participate if the government spends no effort on auditing. As

$I^c(E(r | r \leq \hat{r}), \hat{r}) \leq I(\underline{c})$,²⁰ region M3 of Figure 4 takes a larger area than M3 of Figure SM2.

Therefore ceteris paribus, the coarse grading incentive structure entails no less implementation cost than the fine grading one.

For the purposes of simulation we assume that the average disease-free rate is uniformly

²⁰ This follows as a combination of results in Proposition SM4 and Proposition 1. The former implies that $I^c(E(r | r \leq \hat{r}), \hat{r}) \leq I(E(r | r \leq \hat{r}))$, while from the latter we know that $I(E(r | r \leq \hat{r})) \leq I(\underline{c})$.

distributed as $r \sim U[\underline{r}, 1]$ and participation cost is uniformly distributed as $c \sim U[\underline{c}, \bar{c}]$.

Voluntary program: Coarse grading

By (SM-1), the expected price premium from participating in the program in period t is:

$$I^c(r_t^S, \hat{r}) = \gamma \int_{\hat{r}}^1 \frac{(E[r | r \geq \hat{r}] - r_t^S)}{1 - \underline{r}} dr = \gamma(0.5(1 + \hat{r}) - r_t^S) \frac{1 - \hat{r}}{1 - \underline{r}}. \quad (\text{SM-6})$$

Similar to (10), the disease-free rate among silent sellers in period $t+1$ is:

$$\begin{aligned} r_{t+1}^S &= \phi(r_t^S, \hat{r})E(r | r \leq \hat{r}) + [1 - \phi(r_t^S, \hat{r})]E(r); \\ \phi(r_t^S, \hat{r}) &= \frac{G(I^c(r_t^S, \hat{r}))F(\hat{r})}{G(I^c(r_t^S, \hat{r}))F(\hat{r}) + 1 - G(I^c(r_t^S, \hat{r}))}; \\ G(I^c(r_t^S, \hat{r})) &= \frac{I^c(r_t^S, \hat{r}) - \underline{c}}{\bar{c} - \underline{c}}; & F(\hat{r}) &= \frac{\hat{r} - \underline{r}}{1 - \underline{r}}; \\ E(r | r < \hat{r}) &= \frac{\hat{r} + \underline{r}}{2}; & E(r) &= \frac{1 + \underline{r}}{2}. \end{aligned} \quad (\text{SM-7})$$

From initial values $r_0^S = E(r)$ and \hat{r} , we can calculate the premium $I_0 = I(r_0^S, \hat{r})$ by (SM-6). Then given r_0^S and I_0 , we can solve for r_1^S by (SM-7). Similarly for $t \geq 1$, I_t and r_{t+1}^S will be solved. The participation rate $G(I^c(r_t^S, \hat{r}))$ in each period can also be obtained.

Mandatory program: Coarse grading

By (SM-5), the expected price premium from participating in the program is:

$$\begin{aligned} I^c(E(r | r \leq \hat{r}), \hat{r}) &= \gamma \int_{\hat{r}}^1 \frac{E[r | r \geq \hat{r}] - E[r | r < \hat{r}]}{1 - \underline{r}} dr \\ &= \gamma \frac{(E[r | r \geq \hat{r}] - E[r | r < \hat{r}])(1 - \hat{r})}{1 - \underline{r}} \\ &= \gamma \frac{[0.5(1 + \hat{r}) - 0.5(\hat{r} + \underline{r})](1 - \hat{r})}{1 - \underline{r}} = 0.5\gamma(1 - \hat{r}). \end{aligned} \quad (\text{SM-8})$$

Thus among market participants the proportion with the incentive to participate is:

$$G(0.5\gamma(1 - \hat{r})) = \frac{0.5\gamma(1 - \hat{r}) - \underline{c}}{\bar{c} - \underline{c}}. \quad (\text{SM-9})$$

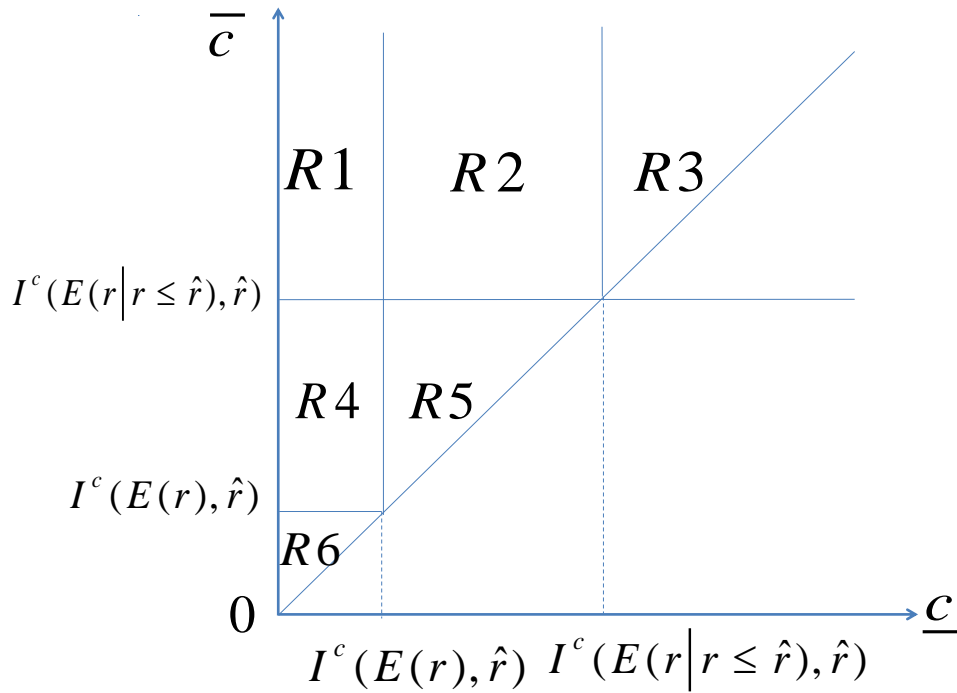


Figure 4.SM1. Equilibria under Different Cost Structures—Voluntary Program, Coarse Grading.

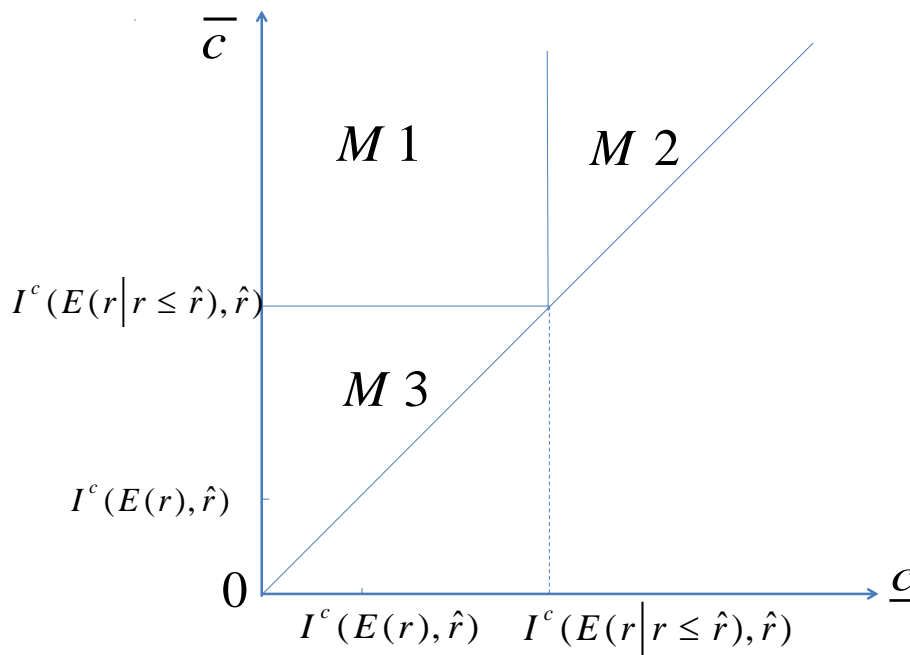


Figure 4.SM2. Participant Types under Different Costs—Mandatory Program, Coarse Grading.

CHAPTER 5: ROLE OF STRATEGIC INTERACTIONS IN DISEASE PREVENTION AND CURE EFFORT CHOICES

A paper submitted to *Health Economics*

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ABSTRACT

For infectious diseases that can be cured but do not provide permanent immunity, incentives to prevent and cure interact in complex ways. There are intra-person temporal interactions. Incentives to prevent illness will increase as cure becomes more costly while incentives to cure will decrease as prevention becomes more costly. There are also inter-person contemporaneous interactions. Incentives to cure decreases in light of free riding as the benefits from cure are shared. Incentives to prevent increase in light of strategic complementarities as diseases likely enter at the weakest link. This paper provides a succinct two-agent model to explore these interactions. Notwithstanding opposing interactions, we suggest that equilibrium levels of prevention and cure efforts are likely to increase as the magnitude of loss from disease increases. Public prevention effort complements both private prevention and private cure efforts. However, public cure effort substitutes for both private cure and private prevention efforts.

INTRODUCTION

The list of bacteria that are pathogenic to humans include Whooping cough, Diphtheria, Enteropathogenic E. coli (leading to diarrhea in infants), *Helicobacter pylori* (believed to cause the majority of peptic ulcers), Leprosy and Tuberculosis. Many sexually transmitted infection (STI) are caused by bacteria. Examples include chlamydia, gonorrhea, syphilis and trichomoniasis. The list of parasitic diseases includes intestinal helminths. Hookworm, whipworm and other helminth diseases are estimated to infect hundreds of millions of

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humans in the developing world, being a major factor in morbidity and malnutrition (Miguel and Kremer 2004), and evidence suggests that eradication can have significant social welfare impacts (Bleakley 2007).

All these diseases are curable. All are susceptible until they become infected, and become susceptible again after an infection is cured. The epidemiology modeling literature refers to these as SIS diseases.²³ In addition, prevention and cure involve externalities in that the behavior of one's neighbors matters too. Zoonoses are another disease class of concern to human health. Many of these have implications for the management of livestock farms. At the individual animal level a disease may not be of SIS with externalities form, but at the farm level it may be. Bovine tuberculosis and brucellosis are zoonotic diseases such that farms are either susceptible or infected and behavior on other farms affects any given farm's prospects for prevention and cure. For both of these diseases and many others, governments in developed countries have waged lengthy campaigns with intent to eradicate the disease (Olmstead and Rhode 2007; Olmstead 2009).

It is important to recognize that prevention and cure interventions are taken in different states. Prevention actions are taken in the susceptible state while cure actions are taken in the infected state. There is of course reason to believe that these decisions are linked. Hennessy (2008a), for example, studies the relationship between these two actions taken by the same individual. De Preux (2011) considers the impact of a change in the loss when diseased, through enhanced insurance availability in the form of Medicare, on prevention activities. Such analyses fall short when we think of the contagious nature of the most infectious diseases, for which the efforts of other parties should not be ignored when seeking to

²³ Other types of disease include susceptible-infected (SI) and susceptible-infected-recovered (SIR). Diseases such as AIDS fall into the SI category (Kremer 1996). Vaccination models such as Geoffard and Philipson (1997) and Francis (2004) are modified SIR models in that susceptible individuals who vaccinate will also acquire permanent immunity.

understand disease prevention and cure.

In this paper, we will develop a two-agent model of an SIS disease where the disease is passed on to a second agent instantaneously whenever one agent contracts it. The focus of our paper is on strategic interactions across disease states and across agents. For example, how does one agent's prevention effort affect the other agent's cure effort? Or how does an exogenous shock to cure effectiveness affect prevention actions? We find that the nature of interactions is such that responses may not be monotonic. However, we see reason to believe that incentives to take cure actions are likely to be weak. In particular, the own-price response could be negative under reasonable circumstances. Also, an increase in public cure efforts will lead to a decrease in private cure and private prevention efforts while an increase in public prevention efforts will lead to an increase in these efforts.

The general model on how prevention and cure decisions interact in the presence of disease externalities is first developed and analyzed as far as a general analysis will allow us to go. We then provide a more specific and tractable version of the model to allow for explicit solutions and further development. We conclude with some brief comments.

MODEL

Setup

This is a continuous time model. There are two possible states, namely the susceptible (S) and infected (I) states. In the susceptible state, each agent earns an income w . There are also two agents, labeled 1 and 2. Agent j will take a disease prevention effort of magnitude a_j for $j \in \{1, 2\}$. The continuous flow cost of taking effort level a_j is $C(a_j)$ where, as is standard, it is assumed that $\partial C(a_j) / \partial a_j \equiv C_1(a_j) \geq 0$ and $\partial^2 C(a_j) / \partial a_j^2 \equiv C_2(a_j) \geq 0$. Agent j receives a utility of $\pi^{S,j} \equiv U^S(w - C(a_j))$ in the susceptible state. Here, also as is standard, we assume that $\partial U^S(\cdot) / \partial w \equiv U_1^S(\cdot) \geq 0$ and $U_2^S(\cdot) \leq 0$.

Define x_1 and x_2 as the number of years passed before agents 1 and 2 contract the disease, respectively. In addition, assume that if one agent contracts the disease, then the other agent will contract it at the same time. The agent that takes least precautions is likely to be the weakest link. Therefore it will take $\min[x_1, x_2]$ years for both agents 1 and 2 to contract the disease. Write the joint survival function beyond x_1 and x_2 given respective prevention actions a_1 and a_2 as $\bar{F}(x_1, x_2; a_1, a_2)$. Our intent is to develop a tractable model in which state transitions depend on actions only, i.e., they are time invariant. Write $\lambda^j(a_j) > 0$ as the time invariant hazard rate at which the disease is contracted by Agent j individually while $\lambda > 0$ is the hazard rate at which the disease is contracted by both agents simultaneously, perhaps due to external forces. We assume that $\lambda_1^j(a_j) < 0$ and $\lambda_2^j(a_j) \geq 0$, i.e., Agent j can lower its own hazard rate at a decreasing rate by increasing its prevention action.

The unique continuous univariate distribution with constant hazard rate is the exponential. In the bivariate case a variety of distributions satisfy the condition, where we choose the well-known and widely applied Marshall and Olkin (1967) distribution:

$$\bar{F}(x_1, x_2; a_1, a_2) = e^{-\lambda^1(a_1)x_1 - \lambda^2(a_2)x_2 - \lambda \max[x_1, x_2]} \quad (1)$$

One way of interpreting this distribution is to specify $x_1 = \min[\omega_1, \varepsilon]$ and $x_2 = \min[\omega_2, \varepsilon]$ where ω_1 , ω_2 and ε are independent random variables with negative exponential distributions having respective parameters $\lambda^1(a_1)$, $\lambda^2(a_2)$ and λ . In this light ε can be viewed as a common external shock causing simultaneous infection of both agents.

From Marshall and Olkin (1967) we know that, perhaps surprisingly, $\min[x_1, x_2]$ is exponentially distributed with parameter $\phi = \lambda^1(a_1) + \lambda^2(a_2) + \lambda$, and so

$$E\{\min[x_1, x_2]\} = \frac{1}{\lambda^1(a_1) + \lambda^2(a_2) + \lambda} \quad (2)$$

Ceteris paribus an increase in prevention effort by either agent lowers hazard rate ϕ .

In the infected state each agent receives amount $w - L$ where L is the loss from disease. We assume that the disease will not be eradicated unless both agents get rid of the disease. Denote the cure effort taken by Agent j as magnitude b_j for $j \in \{1, 2\}$ while the efficiency of government is given as b_0 . The probability rate for recovery is $\eta(b_0, b_1, b_2)$. Of course the effort has some effect, i.e., $\eta_{b_j} \geq 0 \forall j \in \{0, 1, 2\}$. We will also require the form of $\eta(\cdot)$ to be such that b_1 and b_2 substitute as success involves a joint effort to recover.²⁴ The cost of b_j is $m(b_j)$ with $m_1(b_j) \geq 0$ and $m_2(b_j) \geq 0$. Therefore, in the infected state Agent j receives a utility of $\pi^{I,j} \equiv U^I(w - L - m(b_j))$, which is again smoothly increasing and concave in w .

Lifetime expected utility

We define $V^{S,j}$ and $V^{I,j}$ as Agent j 's lifetime expected utility when in the susceptible and infected states, respectively. With r as the continuous time discount rate, the fundamental valuation equations are as given by (Shapiro and Stiglitz 1984):

$$rV^{S,j} = \pi^{S,j} + \phi \times (V^{I,j} - V^{S,j}); \quad rV^{I,j} = \pi^{I,j} + \eta \times (V^{S,j} - V^{I,j}). \quad (3)$$

Each equation takes the form that interest rate times lifetime expected utility equals the instantaneous utility plus the expected gains or losses from a transition of state. That is, utility flow from being in the given state equals instantaneous utility given the present state

²⁴Consider the case of hookworm, typically spread by infected individuals defecating in common areas where others tread barefoot. Prevention would involve installing a toilet or at least incurring the cost of moving to a more secluded place. There is a pool of infection where it doesn't really matter who contributes to reducing the pool's size, so efforts substitute.

plus the valuation implication of an instantaneous state transition.

The solution to (3) is

$$V^{S,j} = \frac{(r + \eta)\pi^{S,j} + \phi\pi^{I,j}}{r\psi}; \quad V^{I,j} = \frac{\eta\pi^{S,j} + (r + \phi)\pi^{I,j}}{r\psi}; \quad \psi = r + \eta + \phi. \quad (4)$$

The difference in the expected lifetime utility between two different states is:

$$V^{S,j} - V^{I,j} = \frac{\pi^{S,j} - \pi^{I,j}}{\psi}. \quad (5)$$

We can see that the surplus value in the susceptible state can be viewed as a bond paying utility flow $\pi^{S,j} - \pi^{I,j}$ at discount rate ψ (Hennessy 2007).

ANALYSIS

In the susceptible state Agent j 's objective is to choose the optimal prevention measure a_j to maximize $V^{S,j}$, or $\max_{a_j} V^{S,j}$. The first-order condition (F.O.C.) is, $\forall j \in \{1, 2\}$,

$$\frac{\partial V^{S,j}}{\partial a_j} \equiv -\left\{(\pi^{S,j} - \pi^{I,j})\phi_{a_j} + \psi U_1^S(w - C(a_j))C_1(a_j)\right\} \frac{(r + \eta)}{r\psi^2} = 0. \quad (6)$$

In the infected state the agent's objective is to choose cure action level b_j to maximize $V^{I,j}$, or $\max_{b_j} V^{I,j}$. The first-order condition is, $\forall j \in \{1, 2\}$,

$$\frac{\partial V^{I,j}}{\partial b_j} \equiv \left\{(\pi^{S,j} - \pi^{I,j})\eta_{b_j} - \psi U_1^I(w - L - m(b_j))m_1(b_j)\right\} \frac{(r + \phi)}{r\psi^2} = 0. \quad (7)$$

A pure strategy Nash Equilibrium (NE) will be any $(a_1, b_1, a_2, b_2) = (a_1^n, b_1^n, a_2^n, b_2^n)$ that satisfies the four conditions given in (6)-(7).

Note that $\phi_{a_j} \leq 0$, $\eta_{b_j} \geq 0$, $U_1^S(\cdot)C_1(\cdot) \geq 0$ and $U_1^I(\cdot)m_1(\cdot) \geq 0$. Therefore $\pi^{S,j} \geq \pi^{I,j}$ for agents 1 and 2 when evaluated at any NE, i.e., the equilibrium utility in the susceptible state is greater than that in the infected state. Equilibrium conditions (6)-(7) can be rearranged to

be

$$\begin{aligned}
\frac{(\pi^{S,1} - \pi^{I,1})\eta_{b_1}}{U_1^I(w - L - m(b_1^n))m_1(b_1^n)} &= -\frac{(\pi^{S,1} - \pi^{I,1})\phi_{a_1}}{U_1^S(w - C(a_1^n))C_1(a_1^n)} = \psi; \\
\frac{(\pi^{S,2} - \pi^{I,2})\eta_{b_2}}{U_1^I(w - L - m(b_2^n))m_1(b_2^n)} &= -\frac{(\pi^{S,2} - \pi^{I,2})\phi_{a_2}}{U_1^S(w - C(a_2^n))C_1(a_2^n)} = \psi; \\
-\frac{(\pi^{S,1} - \pi^{I,1})\phi_{a_1}}{U_1^S(w - C(a_1^n))C_1(a_1^n)} &= -\frac{(\pi^{S,2} - \pi^{I,2})\phi_{a_2}}{U_1^S(w - C(a_2^n))C_1(a_2^n)} = \psi; \\
\frac{(\pi^{S,1} - \pi^{I,1})\eta_{b_1}}{U_1^I(w - L - m(b_1^n))m_1(b_1^n)} &= \frac{(\pi^{S,2} - \pi^{I,2})\eta_{b_2}}{U_1^I(w - L - m(b_2^n))m_1(b_2^n)} = \psi.
\end{aligned} \tag{8}$$

Here system (8) defines a set of four conditions that characterize NE. The first pair of conditions are intra-personal. They equate the expected marginal cost of effort across states for the respective agents. Were this not true then lifetime expected utility could be increased by reallocating efforts across states. The other two equations are inter-personal. They assert that while the choices made may differ across agents, the differences are driven by the change in utility flow that the decisions are intended to secure; i.e., $\pi^{S,1} - \pi^{I,1}$ and $\pi^{S,2} - \pi^{I,2}$. In all cases, the numerator can be viewed as a transition risk adjusted cashflow differential (i.e., susceptible less infected) over time that is being protected while the denominator is the present time cost so that the ratio should naturally be a hazard-rate augmented opportunity cost of capital, ψ .

Linear specification

From this point forward we make the assumption that cost and hazard functions are linear of the following forms: $C(a_j) = r_a a_j$ with r_a strictly positive; $m(b_j) = r_b b_j$ with r_b strictly positive; $\phi = h_0 - h_1 a_1 - h_2 a_2$ where h_0, h_1 are h_2 strictly positive; and $\eta = g_0 + g_1 b_1 + g_2 b_2$ where g_0, g_1 are g_2 strictly positive. The motivation for form $\phi = h_0 - h_1 a_1 - h_2 a_2$ is given

by eqn. (2) where $\lambda^1(a_1) = \lambda^{1*} - h_1 a_1$, $\lambda^2(a_2) = \lambda^{2*} - h_2 a_2$ and $\lambda = h_0 - \lambda^{1*} - \lambda^{2*}$. Expression $\eta = g_0 + g_1 b_1 + g_2 b_2$ does not have a similar structural motivation and should be viewed as a reduced form expression seeking to capture substitution between b_1 and b_2 .

Notice that an increase in either $h_j, j \in \{1, 2\}$, amounts to an increase in the marginal effectiveness of prevention actions, while an increase in either $g_j, j \in \{1, 2\}$, amounts to an increase in the marginal effectiveness of cure actions. A decrease in h_0 can be viewed as an exogenous increase in effort to prevent. As such it could reflect expenditure on a public health campaign or on border measures to reduce disease prevalence within a region. An increase in g_0 can be viewed as an exogenous increase in effort to cure. It could also be viewed as expenditure on a public health campaign, or effort to reduce the extent of background infection through channels other than inter-personal interactions.

We present first a symmetry result:

Result 1: A symmetric Nash equilibrium $(a_1^n, b_1^n) = (a_2^n, b_2^n)$ exists if and only if $h_1 = h_2$ and $g_1 = g_2$.

Proof: For the *if* part; when $h_1 = h_2$ and $g_1 = g_2$ then the last two equations of (8) generate:

$$\begin{aligned} \frac{U_1^S(w - r_a a_1^n)}{U^S(w - r_a a_1^n) - U^I(w - L - r_b b_1^n)} &= \frac{h_1}{\psi r_a}; \\ \frac{U_1^S(w - r_a a_2^n)}{U^S(w - r_a a_2^n) - U^I(w - L - r_b b_2^n)} &= \frac{h_2}{\psi r_a}; \\ \frac{U_1^I(w - L - r_b b_1^n)}{U^S(w - r_a a_1^n) - U^I(w - L - r_b b_1^n)} &= \frac{g_1}{\psi r_b}; \\ \frac{U_1^I(w - L - r_b b_2^n)}{U^S(w - r_a a_2^n) - U^I(w - L - r_b b_2^n)} &= \frac{g_2}{\psi r_b}. \end{aligned} \tag{9}$$

When $h_1 = h_2$ and $g_1 = g_2$ then symmetry in the agent's functional forms allows us to state

the following: For any (a_1^n, b_1^n) satisfying the conditions in (9), then setting $(a_2^n, b_2^n) = (a_1^n, b_1^n)$ ensures that (a_2^n, b_2^n) also satisfies these conditions.

For the *only if* part; were $h_1 \neq h_2$ and/or $g_1 \neq g_2$ then $(a_2^n, b_2^n) = (a_1^n, b_1^n)$ would lead to contradictions. For example the left-hand sides of the first two relations in (9) would be the same but the right-hand sides would not. \square

Result 1 shows that when the efforts by the two agents have the same effectiveness then there exists a NE in which the two agents make the same prevention and cure efforts, and vice versa. Can we state anything about asymmetric solutions to (9) when agent technology opportunities are identical? If asymmetric solutions exist, and we will affirm that they do with an example, then the next result provides a characterization. The next result might be viewed as rationalizing hypochondriac behavior, at least in a relative sense.

Result 2: Suppose that $h_1 = h_2$ and $g_1 = g_2$ and a non-symmetric Nash equilibrium

$(a_1^n, b_1^n), (a_2^n, b_2^n)$ exists. Then $(a_1^n - a_2^n)(b_1^n - b_2^n) \geq 0$; that is, the agent making the higher level of cure effort also makes the higher level of prevention effort.

Proof: Suppose instead that $(a_1^n - a_2^n)(b_1^n - b_2^n) < 0$. For example, suppose that $a_1^n > a_2^n$, while $b_1^n < b_2^n$. By $a_1^n > a_2^n$ and the first two equations of (9) we have:

$$U^S(w - r_a a_1^n) - U^I(w - L - r_b b_1^n) > U^S(w - r_a a_2^n) - U^I(w - L - r_b b_2^n) \quad (10)$$

By $b_1^n < b_2^n$ and the last two equations of (9) we have:

$$U^S(w - r_a a_1^n) - U^I(w - L - r_b b_1^n) < U^S(w - r_a a_2^n) - U^I(w - L - r_b b_2^n) \quad (11)$$

Note that (10) and (11) contradict. Similarly a contradiction will follow when we assume that $a_1^n < a_2^n$, while $b_1^n > b_2^n$. Therefore $(a_1^n - a_2^n)(b_1^n - b_2^n) < 0$ cannot be true, and our original

conclusion holds. \square

So an agent who invests more in prevention efforts will also invest more in cure efforts. In other words, it may be rational for agents to maintain either prudent or reckless behavior throughout their lives. Intuitively, the agent who acts prudently perceives a high utility difference between the two states, while the one who acts recklessly believes that the utility difference between the two states is small. Since we assume a concave utility function, the agent who spends more in both states will end up having a large utility difference. Therefore their actions at NE reinforce their original beliefs, which further support their actions at NE.

SUPERMODULARITY

In this section we will check whether the game we study is supermodular (Milgrom and Roberts 1990), i.e., whether the agents' strategies display the strategic complementarity property (Bulow, Geanakoplos and Klemperer 1985). This is of interest as then an exogenous adjustment to the system could impact all actions in a beneficial and reinforcing way. For example a subsidy on any one action would induce an increase in all NE choices under optimal behavior.²⁵

Result 3: $\partial^2 V^{s,1} / \partial a_1 \partial b_2 \leq 0$, $\partial^2 V^{s,2} / \partial a_2 \partial b_1 \leq 0$, $\partial^2 V^{I,1} / \partial a_2 \partial b_1 \geq 0$ and $\partial^2 V^{I,2} / \partial a_1 \partial b_2 \geq 0$.

That is, *i*) the marginal impact of own prevention effort on expected lifetime utility in the susceptible state is decreasing in the cure effort of the other agent, while *ii*) the marginal impact of own cure effort on expected lifetime utility in the infected state is increasing in the prevention effort of the other agent.

Result 3 holds that for two different agents, the disease prevention and cure efforts could

²⁵ Proofs of results 3-5 are provided in Appendix A.

either be strategic substitutes or strategic complements. To be specific, in the susceptible state, one agent will choose to decrease her prevention effort in NE if the other agent increases her cure effort in the infected state (strategic substitutes). There is an incentive to free ride. However, in the infected state one agent will choose to increase the cure effort in NE if the other agent increases her prevention effort in the susceptible state (strategic complements).²⁶ Here the other agent's additional prevention efforts increase the probability that own cure efforts are effective.

Result 4: $\partial^2 V^{S,j} / \partial a_1 \partial a_2 \geq 0 \forall j \in \{1,2\}$ and $\partial^2 V^{I,j} / \partial b_1 \partial b_2 \leq 0 \forall j \in \{1,2\}$. That is, *i*) the marginal impact of own prevention effort on expected lifetime utility in the susceptible state is increasing in the prevention effort of the other agent, while *ii*) the marginal impact of own cure effort on expected lifetime utility in the infected state is decreasing in the cure effort of the other agent.

From Result 4 we see that in the susceptible state, two agents' disease prevention efforts are strategic complements. Were Agent 2 to increase her prevention effort, then the probability that Agent 1 would contract the disease from Agent 2 decreases. Therefore Agent 1 will have an incentive to increase her prevention effort to ensure that she will not contract the disease first. Stated differently, were Agent 2 to decrease her prevention effort then Agent 1 will decrease her effort too since she is more likely to acquire the disease from Agent 2 anyway.

In the infected state, however, the two agents' cure efforts are strategic substitutes. If the total cure efforts increase, then the probability of transiting from *I* to *S* increases. Here when one agent contributes more to the total cure effort, the other agent will choose to free ride.

²⁶ All findings in Result 3 are inter-personal. Hennessy (2008a) demonstrated similar, but intra-personal, results.

Result 5: $\partial^2 V^{s.1} / \partial a_1 \partial b_1 = 0$, $\partial^2 V^{s.2} / \partial a_2 \partial b_2 = 0$, and $\partial^2 V^{I.1} / \partial a_1 \partial b_1 = \partial^2 V^{I.2} / \partial a_2 \partial b_2 = 0$.

That is, for both expected lifetime utility in the susceptible state and in the infected state, the marginal impact of own prevention effort is unaffected by an increase in own cure effort.

Result 5 establishes that the disease prevention and cure efforts are strategically independent for any agent. Note that all decisions at issue are intra-personal. So Result 5 is consistent with eqn. (6) of Hennessy (2008a), and shows that local independence of intra-personal decisions across states extends to the case where there are inter-personal interactions. Intuitively, one agent's marginal increase in cure effort generates two effects for herself, current loss and future gain. These two effects exactly offset each other at NE and the lifetime expected utility in the infected state remains the same.²⁷

We turn attention now to the matter of whether structure allows for monotone comparative statics, as in Milgrom and Shannon (1994). To clarify what is meant here consider the following context. Agent 1 takes action y while Agent 2 takes action z . Payoffs are $W^1(y, z; \theta)$ for Agent 1 and $W^2(z; \theta)$ for Agent 2 where θ is an exogenous variable impacting both agents.

Suppose *i*) $\partial^2 W^1(y, z; \theta) / \partial y \partial z \geq 0$, $\partial^2 W^1(y, z; \theta) / \partial y \partial \theta \geq 0$ and $\partial^2 W^2(z; \theta) / \partial z \partial \theta \geq 0$.

Under standard assumptions it is not hard to show that the Agent 2 optimal choice of z will increase with an increase in θ , and furthermore that the Agent 1 optimal choice of y will also increase with an increase in θ . The latter response is due in part to the complementary spillover arising from the impact of θ on optimal z . However if instead *ii*)

$\partial^2 W^1(y, z; \theta) / \partial y \partial z \geq 0$, $\partial^2 W^1(y, z; \theta) / \partial y \partial \theta \geq 0$ and $\partial^2 W^2(z; \theta) / \partial z \partial \theta \leq 0$ then we cannot be sure what impact θ would have on y . While the direct effect of an increase in θ on y

²⁷ Were they not to offset each other at NE, the agent would have an incentive to increase one of her efforts. Thus the status quo efforts will not be a NE, a contradiction.

would be to increase its optimal value the indirect effect through z would oppose that effect.

Finally if *iii*) $\partial^2 W^1(y, z; \theta) / \partial y \partial z \leq 0$, $\partial^2 W^1(y, z; \theta) / \partial y \partial \theta \geq 0$ and $\partial^2 W^2(z; \theta) / \partial z \partial \theta \leq 0$

then we can sign the effect of an increase in θ on optimal y . To see this just write $z' \equiv -z$,

$\hat{W}^1(y, z'; \theta) \equiv W^1(y, z; \theta)$, and $\hat{W}^2(z'; \theta) \equiv W^2(z; \theta)$. The transformed objective functions

satisfy the properties laid out in *i*) above. However, such transformations are of no assistance in our problem.

Result 6: The system cannot not be transformed into a supermodular game.

Proof: We summarize Results 3 to 5 as:

$$\begin{array}{lll}
 \frac{\partial^2 V^{s,1}}{\partial a_1 \partial a_2} \geq 0; & \frac{\partial^2 V^{s,1}}{\partial a_1 \partial b_1} = 0; & \frac{\partial^2 V^{s,1}}{\partial a_1 \partial b_2} \leq 0; \\
 \frac{\partial^2 V^{l,1}}{\partial b_1 \partial b_2} \leq 0; & \frac{\partial^2 V^{l,1}}{\partial b_1 \partial a_1} = 0; & \frac{\partial^2 V^{l,1}}{\partial b_1 \partial a_2} \geq 0; \\
 \frac{\partial^2 V^{s,2}}{\partial a_2 \partial a_1} \geq 0; & \frac{\partial^2 V^{s,2}}{\partial a_2 \partial b_2} = 0; & \frac{\partial^2 V^{s,2}}{\partial a_2 \partial b_1} \leq 0; \\
 \frac{\partial^2 V^{l,2}}{\partial b_2 \partial b_1} \leq 0; & \frac{\partial^2 V^{l,2}}{\partial b_2 \partial a_2} = 0; & \frac{\partial^2 V^{l,2}}{\partial b_2 \partial a_1} \geq 0.
 \end{array} \tag{12}$$

We cannot transform arguments in this system such that derivatives are all nonnegative, as in

Milgrom and Shannon (1994, p. 172). This is because terms such as $\partial^2 V^{s,1} / \partial a_1 \partial b_2$ and

$\partial^2 V^{l,2} / \partial b_2 \partial a_1$ cannot be simultaneously positive no matter how we reverse the sign of the

a_j as a set or of the b_j as a set. \square

Result 6 implies that there is little hope of identifying a general result in which a subsidy for one class of action, be it prevention or cure, will have a clear monotone impact (in any direction) on all decisions in the linked system. By the reasoning in Result 6 it is also clear that a system containing any three out of four endogenous variables could not be transformed

into a supermodular system. It is obvious however, that if we view the cure efforts by both agents as exogenous, then the system with only two endogenous variables is now a supermodular one. That is, prevention efforts complement and encouraging prevention by one agent will encourage prevention by others. This point has been made elsewhere in Hennessy (2008b).

AN EXAMPLE

In this section, we will construct a specific example where NE is solvable under this setup. Similar to Hennessy (2008a), we assume CARA utility function for both states $U^S(x) = U^I(x) = -e^{-\rho x}$ with $\rho > 0$. As we have already shown, the NE solution $(a_1^n, b_1^n, a_2^n, b_2^n)$ is determined by equations (6) and (7). Under our linear technology specification and CARA, the equivalent for (6) and (7) are $\forall i \in \{1, 2\}$:

$$\begin{aligned} h_j [e^{-\rho(w-L-r_b b_j)} - e^{-\rho(w-r_a a_j)}] &= \psi e^{-\rho(w-r_a a_j)} \rho r_a; \\ g_j [e^{-\rho(w-L-r_b b_j)} - e^{-\rho(w-r_a a_j)}] &= \psi \rho r_b e^{-\rho(w-L-r_b b_j)}. \end{aligned} \quad (13)$$

Assume NE solutions are $(a_1^n, b_1^n, a_2^n, b_2^n)$. We can manipulate the system to be, $\forall i \in \{1, 2\}$:

$$\frac{h_j (1 - e^{-\rho(L-r_a a_j^n + r_b b_j^n)})}{\rho r_a \psi} = e^{-\rho(L-r_a a_j^n + r_b b_j^n)}; \quad \frac{g_j (1 - e^{-\rho(L-r_a a_j^n + r_b b_j^n)})}{\rho r_b \psi} = 1. \quad (14)$$

Thus $e^{-\rho(L-r_a a_j^n + r_b b_j^n)} = 1 - \rho r_b \psi / g_j < 0$ so that $L - r_a a_j^n + r_b b_j^n > 0$. We will explore next the equilibrium solution set.

Result 7: Necessary conditions for an interior NE to exist for all possible prices r_a and r_b are $g_1 = g_2$ and $h_1 = h_2$.

Proof: Difference the expressions in (14). As $e^{-\rho(L-r_a a_j^n + r_b b_j^n)} \neq 1$, division by $1 - e^{-\rho(L-r_a a_j^n + r_b b_j^n)}$ is admissible and establishes $(r_a g_j - r_b h_j) / (r_a r_b) = \rho \psi$, so that

$$\frac{h_1 - h_2}{r_a} = \frac{g_1 - g_2}{r_b}. \quad (15)$$

We can see from the above derivation that parameters r_a , r_b , h_1 , h_2 , g_1 and g_2 should satisfy (15) for system (14) to have a pure strategy NE solution. Suppose (15) holds for all possible prices r_a and r_b , then it is necessary that $g_1 = g_2 = g$ and $h_1 = h_2 = h$. \square

Note that in Result 1 we have shown that the same effectiveness of two agents' efforts is required for a symmetric NE to exist. Result 7 shows that, under our specific setup, for a NE to exist under all possible effort prices then the effectiveness of two agents' efforts should be the same. If $g_1 = g_2 = g$ and $h_1 = h_2 = h$, then relation $(r_a g_j - r_b h_j) / (r_a r_b) = \rho \psi$ implies that

$$\frac{g}{\rho r_b} - \frac{h}{\rho r_a} = r + g_0 + g(b_1^n + b_2^n) + h_0 - h(a_1^n + a_2^n). \quad (16)$$

Next, use (14) when evaluated at equilibrium to obtain:

$$\frac{hr_b}{gr_a} = e^{-\rho(L - r_a a_j^n + r_b b_j^n)}. \quad (17)$$

From (16) and (17) we can obtain equations containing only action sums as sufficient statistics. With $\mu = r + g_0 + h_0$ one can write:

$$\begin{aligned} g(b_1^n + b_2^n) - h(a_1^n + a_2^n) &= \frac{1}{\rho} \left(\frac{g}{r_b} - \frac{h}{r_a} \right) - \mu \equiv \Lambda_1; \\ r_b b_1^n - r_a a_1^n &= \frac{1}{\rho} \ln \left(\frac{gr_a}{hr_b} \right) - L \equiv \frac{\Lambda_2}{2}; \\ r_b b_2^n - r_a a_2^n &= \frac{1}{\rho} \ln \left(\frac{gr_a}{hr_b} \right) - L \equiv \frac{\Lambda_2}{2}. \end{aligned} \quad (18)$$

Elementary manipulation then establishes the NE solutions for $a_1^n + a_2^n$ and $b_1^n + b_2^n$ as²⁸

²⁸ The detailed derivation is in Appendix B.

$$\begin{aligned}
a_1^n + a_2^n &= \frac{1}{\rho r_a} - \frac{r_b \mu - 2gL}{r_a g - r_b h} - \frac{2g}{\rho} \frac{\ln[(r_a g)/(r_b h)]}{r_a g - r_b h} \equiv A; \\
b_1^n + b_2^n &= \frac{1}{\rho r_b} - \frac{r_a \mu - 2hL}{r_a g - r_b h} - \frac{2h}{\rho} \frac{\ln[(r_a g)/(r_b h)]}{r_a g - r_b h} \equiv B.
\end{aligned} \tag{19}$$

If we assume that $\rho, r_a, r_b, h_0, g_0, h, g$ and L take such values that both $A \geq 0$ and $B \geq 0$, then pure strategy NE exist. Due to the same effectiveness requirement, at any state only the sum of the two agents' efforts matters, as shown in (19). There are multiple Nash equilibria but all satisfy $a_1^n + a_2^n = A$ and $b_1^n + b_2^n = B$. From Result 2 and non-negativity, if we arbitrarily assume that $a_1^n \leq 0.5A$ then we know that $a_2^n \geq 0.5A$, $b_1^n \leq 0.5B$ and $b_2^n \geq 0.5B$.

Result 8: Assume that $g_1 = g_2 = g$ and $h_1 = h_2 = h$. Without further loss of generality assume that $a_1^n \leq 0.5A$. Then NE solutions take the form $(a_1^n, b_1^n, a_2^n, b_2^n) = 0.5(A - \Lambda_3 / r_a, B - \Lambda_3 / r_b, A + \Lambda_3 / r_a, B + \Lambda_3 / r_b)$ where constant $\Lambda_3 \in [0, \min\{Ar_a, Br_b\}]$.

Proof: As both equations in (13) hold for both agents 1 and 2, we can further obtain:

$$\begin{aligned}
\frac{e^{-\rho(w-L-r_b b_2^n)} - e^{-\rho(w-r_a a_2^n)}}{e^{-\rho(w-L-r_b b_1^n)} - e^{-\rho(w-r_a a_1^n)}} &= e^{\rho r_a (a_2^n - a_1^n)}; \\
\frac{e^{-\rho(w-L-r_b b_2^n)} - e^{-\rho(w-r_a a_2^n)}}{e^{-\rho(w-L-r_b b_1^n)} - e^{-\rho(w-r_a a_1^n)}} &= e^{\rho r_b (b_2^n - b_1^n)}.
\end{aligned} \tag{20}$$

It follows immediately from (20) that $r_a (a_2^n - a_1^n) = r_b (b_2^n - b_1^n) = \Lambda_3$, and so

$a_2^n = a_1^n + \Lambda_3 / r_a$ and $b_2^n = b_1^n + \Lambda_3 / r_b$. The result follows from the positivity requirement on actions. \square

We turn now to the effects of exogenous shocks to the system. Given that solutions exist on plane segments $a_1^n + a_2^n = A$ and $b_1^n + b_2^n = B$ with choice values bounded below by 0 but are otherwise indeterminate, we can only find the effect on aggregate action levels.

*Result 9: i) $\partial(a_1^n + a_2^n) / \partial L \geq 0$ and $\partial(b_1^n + b_2^n) / \partial L \geq 0$. That is, the aggregate levels of private prevention and cure efforts both increase with an increase in the magnitude of loss from disease. ii) $\partial(a_1^n + a_2^n) / \partial h_0 \leq 0$ and $\partial(b_1^n + b_2^n) / \partial h_0 \leq 0$. That is, aggregate action levels both *increase* with an increase in public health expenditure on prevention. iii) $\partial(a_1^n + a_2^n) / \partial g_0 \leq 0$ and $\partial(b_1^n + b_2^n) / \partial g_0 \leq 0$. That is, aggregate action levels both *decrease* with an increase in public health expenditure on cure.*

Proof: We have already shown that $L + r_b b_j^n - r_a a_j^n > 0, \forall j \in \{1, 2\}$. Thus we know from (17) that $g r_a > h r_b$. Then differentiate (19). \square

From Result 9 we can see that public health prevention efforts complement private prevention and cure efforts but public cure efforts crowd out private prevention and cure efforts. This suggests a strategic motive to prefer public intervention to prevent over public intervention to cure. At least one is strengthening private incentives. We complete the analysis with a study of price shocks.²⁹

Result 10: i) $\partial(a_1^n + a_2^n) / \partial r_a \leq 0$ and $\partial(a_1^n + a_2^n) / \partial g \leq 0$. That is, the aggregate level of private prevention effort decreases whenever such effort becomes more costly or cure actions become more effective. ii) $\partial(a_1^n + a_2^n) / \partial r_b \geq 0$ and $\partial(a_1^n + a_2^n) / \partial h \geq 0$. That is, the aggregate level of private prevention effort increases whenever the cure action becomes more costly or prevention effort becomes more effective.

The findings are mostly in accord with standard intuition. The price response in part ii) asserts that private prevention effort complements cure effort in the sense that the sum of

²⁹ Proofs of results 10-11 are provided in Appendix B.

private prevention efforts increases with an increase in the unit cost of cure. This finding is consistent with Hennessy's (2008a) study of private actions absent externalities. The responses of private cure actions are somewhat more involved.

Result 11: i) $\partial(b_1^n + b_2^n) / \partial h \geq 0$. That is, aggregate private cure effort increases whenever prevention effort becomes more effective. ii) the signs of $\partial(b_1^n + b_2^n) / \partial g$, $\partial(b_1^n + b_2^n) / \partial r_a$ and $\partial(b_1^n + b_2^n) / \partial r_b$ cannot be determined without further information. However, if $2r_b h \geq r_a g$ then $\partial(b_1^n + b_2^n) / \partial g \leq 0$, $\partial(b_1^n + b_2^n) / \partial r_a \leq 0$ and $\partial(b_1^n + b_2^n) / \partial r_b \geq 0$.

From Result 11, we learn that higher levels of cure effort will be taken when preventative effort becomes more effective. Agents will invest more in curing a disease if they assume that the susceptible state can be more easily maintained. However, the cure effort will drop with a increase in cure effectiveness whenever $2r_b h \geq r_a g$, i.e., *ceteris paribus* the cost of cure is not very low. The own price effect of the cure input is interesting. The own price has two impacts. One involves a decrease in input demand given the disease state at hand, or a Hicksian substitution effect away from the input. The other involves a probabilistic switch in states as that is why the input is used. This latter state probability effect is similar to the income effect in Marshallian demand analysis. As Hennessy (2008a) has pointed out, the cure input could turn out to be a Giffen good when this state probability effect outweighs the substitution effect. In our example, the cure effort could increase with an increase in own price when the preventative effort is relatively effective and the cost of cure input is not very low. Both conditions lead to a high state probability effect.

CONCLUDING REMARKS

This paper shows that private healthcare choices across time and individuals interact in

involved ways, suggesting pessimism in regard to understanding any overall response to an exogenous shock. However in a simple model we do show that an exogenous improvement in prevention, perhaps through a government program, will encourage both private prevention actions and private cure actions. By contrast an exogenous improvement in cure will discourage all private actions. If the view is that private healthcare actions are lower than those that maximize social welfare then the analysis provides some evidence in favor of public prevention programs over public cure programs.

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APPENDIX A

Proof of Results 3 to 5: First, based on (6)-(7) we can solve for $\partial^2 V^{S,i} / \partial a_i \partial a_j$, $\partial^2 V^{S,i} / \partial a_i \partial b_i$ and $\partial^2 V^{S,i} / \partial a_i \partial b_j$ at the NE point:

$$\begin{aligned}
\frac{\partial^2 V^{S,i}}{\partial a_i \partial a_j} &= \frac{2\phi_{a_i} \phi_{a_j} (r + \eta)(\pi^{S,i} - \pi^{I,i})}{r\psi^3} + \frac{\phi_{a_i} (r + \eta)}{r\psi^2} U_1^S (w - C(a_i^n)) C_1(a_i^n) \\
&= \frac{2\phi_{a_i} \phi_{a_j} (r + \eta)(\pi^{S,i} - \pi^{I,i})}{r\psi^3} - \frac{\phi_{a_j} \phi_{a_i} (r + \eta)(\pi^{S,i} - \pi^{I,i})}{r\psi^3} \\
&= \frac{\phi_{a_i} \phi_{a_j} (r + \eta)(\pi^{S,i} - \pi^{I,i})}{\psi^3} \geq 0;
\end{aligned} \tag{A-1}$$

$$\begin{aligned}
\frac{\partial^2 V^{S,i}}{\partial a_i \partial b_i} &= \frac{\eta_{b_i} \psi - \eta_{b_i} (r + \eta)}{r\psi^2} U_1^S (w - C(a_i^n)) C_1(a_i^n) \\
&\quad - \frac{\phi_{a_i} \eta_{b_i} (\phi - r - \eta)(\pi^{S,i} - \pi^{I,i})}{r\psi^3} - \frac{\phi_{a_i} (r + \eta)}{r\psi^2} U_1^I (w - L - m(b_i^n)) m_1(b_i^n) \\
&= \frac{\eta_{b_i} \phi (\pi^{S,i} - \pi^{I,i}) \phi_{a_i}}{r\psi^3} - \frac{\phi_{a_i} \eta_{b_i} (\phi - r - \eta)(\pi^{S,i} - \pi^{I,i})}{r\psi^3} - \frac{\phi_{a_i} (r + \eta)(\pi^{S,i} - \pi^{I,i}) \eta_{b_i}}{r\psi^3} \\
&= \frac{\phi_{a_i} \eta_{b_i} (\pi^{S,i} - \pi^{I,i})}{r\psi^3} (\phi - \phi + r + \eta - r - \eta) = 0.
\end{aligned} \tag{A-2}$$

Note that $\partial^2 V^{S,i} / \partial a_i \partial b_j$ resembles the form of $\partial^2 V^{S,i} / \partial a_i \partial b_i$ but without the last term.

Therefore: $\partial^2 V^{S,i} / \partial a_i \partial b_j = \phi_{a_i} \eta_{b_j} (\pi^{S,i} - \pi^{I,i})(r + \eta) / (r\psi^3) \leq 0$.

Next, based on (7)-(8) we will solve $\partial^2 V^{I,i} / \partial b_i \partial b_j$, $\partial^2 V^{I,i} / \partial b_i \partial a_i$ and $\partial^2 V^{S,i} / \partial b_i \partial a_j$ at

the NE point:

$$\begin{aligned}
\frac{\partial^2 V^{I,i}}{\partial b_i \partial b_j} &= -\frac{2\eta_{b_i} \eta_{b_j} (r + \phi)(\pi^{S,i} - \pi^{I,i})}{r\psi^3} + U_1^I (w - L - m(b_i^n)) m_1(b_i^n) \frac{\eta_{b_j} (r + \phi)}{r\psi^2} \\
&= -\frac{2\eta_{b_i} \eta_{b_j} (r + \phi)(\pi^{S,i} - \pi^{I,i})}{r\psi^3} + \frac{\eta_{b_i} (\pi^{S,i} - \pi^{I,i}) \eta_{b_j} (r + \phi)}{r\psi^3} \\
&= -\frac{\eta_{b_i} \eta_{b_j} (r + \phi)(\pi^{S,i} - \pi^{I,i})}{r\psi^3} \leq 0;
\end{aligned} \tag{A-3}$$

$$\begin{aligned}
\frac{\partial^2 V^{I,i}}{\partial b_i \partial a_i} &= -\frac{(r + \phi)\eta_{b_i}}{r\psi^2} U_1^S (w - C(a_i^n)) C_1(a_i^n) + \frac{\eta_{b_i} \phi_{a_i} (\eta - \phi - r)(\pi^{S,i} - \pi^{I,i})}{r\psi^3} \\
&\quad - U_1^I (w - L - m(b_i^n)) m_1(b_i^n) \frac{\phi_{a_i} \eta}{r\psi^2} \\
&= \frac{(r + \phi)\eta_{b_i} (\pi^{S,i} - \pi^{I,i}) \phi_{a_i}}{r\psi^3} + \frac{\eta_{b_i} \phi_{a_i} (\eta - \phi - r)(\pi^{S,i} - \pi^{I,i})}{r\psi^3} - \frac{(\pi^{S,i} - \pi^{I,i}) \eta_{b_i} \phi_{a_i} \eta}{r\psi^3} \\
&= \frac{\eta_{b_i} \phi_{a_i} (\pi^{S,i} - \pi^{I,i})}{r\psi^3} (r + \phi + \eta - \phi - r - \eta) = 0.
\end{aligned} \tag{A-4}$$

Finally, note that $\partial^2 V^{I,i} / \partial b_i \partial a_j$ resembles the form of $\partial^2 V^{I,i} / \partial b_i \partial a_i$ but without the last term. Therefore $\partial^2 V^{I,i} / \partial b_i \partial a_j = \eta_{b_i} \phi_{a_j} (\pi^{S,i} - \pi^{I,i}) / (r\psi^3 \eta) \geq 0$. \square

APPENDIX B

System (18) can be manipulated into form:

$$r_b g (b_1^n + b_2^n) - r_b h (a_1^n + a_2^n) = r_b \Lambda_1; \quad r_b g (b_1^n + b_2^n) - r_a g (a_1^n + a_2^n) = g \Lambda_2. \tag{B-1}$$

We can solve for $a_1^n + a_2^n$ as:

$$\begin{aligned}
a_1^n + a_2^n &= \frac{r_b \Lambda_1 - g \Lambda_2}{r_a g - r_b h} \\
&= \frac{r_b}{r_a g - r_b h} \left[\frac{1}{\rho} \left(\frac{g}{r_b} - \frac{h}{r_a} \right) - \mu \right] - \frac{2g}{r_a g - r_b h} \left[\frac{1}{\rho} \ln \left(\frac{g r_a}{h r_b} \right) - L \right] \\
&= \frac{1}{\rho r_a} - \frac{r_b \mu - 2gL}{r_a g - r_b h} - \frac{2g}{\rho} \frac{\ln[(r_a g) / (r_b h)]}{r_a g - r_b h}.
\end{aligned} \tag{B-2}$$

Similarly $b_1^n + b_2^n$ at NE can be expressed as:

$$\begin{aligned}
b_1^n + b_2^n &= \frac{r_a A_1 - h A_2}{r_a g - r_b h} \\
&= \frac{r_a}{r_a g - r_b h} \left[\frac{1}{\rho} \left(\frac{g}{r_b} - \frac{h}{r_a} \right) - \mu \right] - \frac{2h}{r_a g - r_b h} \left[\frac{1}{\rho} \ln \left(\frac{g r_a}{h r_b} \right) - L \right] \\
&= \frac{1}{\rho r_b} - \frac{r_a \mu - 2hL}{r_a g - r_b h} - \frac{2h}{\rho} \frac{\ln[(r_a g) / (r_b h)]}{r_a g - r_b h}.
\end{aligned} \tag{B-3}$$

Proof of Result 10: From the NE solutions defined in Result 8 we can calculate the

following:

$$\begin{aligned}
\frac{\partial(a_1^n + a_2^n)}{\partial g} &= \frac{\partial}{\partial g} \left\{ \frac{1}{\rho r_a} - \frac{r_b \mu}{r_a g - r_b h} + \frac{2gL}{r_a g - r_b h} - \frac{2g \ln[(r_a g)/(r_b h)]}{\rho(r_a g - r_b h)} \right\} \\
&= \frac{r_a r_b \mu}{(r_a g - r_b h)^2} + \frac{2[L(r_a g - r_b h) - gLr_a]}{(r_a g - r_b h)^2} - \frac{2 \ln[(r_a g)/(r_b h)]}{\rho(r_a g - r_b h)} \\
&\quad - 2 \frac{(r_a g - r_b h) - g r_a \ln[(r_a g)/(r_b h)]}{\rho(r_a g - r_b h)^2} \\
&= \frac{r_a r_b \mu}{(r_a g - r_b h)^2} - \frac{2Lh r_b}{(r_a g - r_b h)^2} - \frac{2 \ln[(r_a g)/(r_b h)]}{\rho(r_a g - r_b h)} \\
&\quad - 2 \frac{(r_a g - r_b h) - g r_a \ln[(r_a g)/(r_b h)]}{\rho(r_a g - r_b h)^2} \\
&= \frac{r_b}{r_a g - r_b h} \left[\frac{r_a \mu - 2hL}{r_a g - r_b h} - \frac{2}{\rho r_b} + \frac{2h \ln[(r_a g)/(r_b h)]}{\rho(r_a g - r_b h)} \right] = - \frac{r_b}{r_a g - r_b h} \left[B + \frac{1}{\rho r_b} \right] \leq 0;
\end{aligned} \tag{B-4}$$

$$\begin{aligned}
\frac{\partial(a_1^n + a_2^n)}{\partial h} &= \frac{\partial}{\partial h} \left\{ \frac{1}{\rho r_a} - \frac{r_b \mu}{r_a g - r_b h} + \frac{2gL}{r_a g - r_b h} - \frac{2g \ln[(r_a g)/(r_b h)]}{\rho(r_a g - r_b h)} \right\} \\
&= - \frac{r_b r_b \mu}{(r_a g - r_b h)^2} + \frac{2gLr_b}{(r_a g - r_b h)^2} + \frac{2g}{\rho} \frac{(r_a g - r_b h)/h - r_b \ln[(r_a g)/(r_b h)]}{(r_a g - r_b h)^2} \\
&= \frac{r_b}{r_a g - r_b h} \left[- \frac{r_b \mu - 2gL}{r_a g - r_b h} + \frac{2g/h}{\rho r_b} - \frac{2g \ln[(r_a g)/(r_b h)]}{\rho(r_a g - r_b h)} \right] \\
&= \frac{r_b}{r_a g - r_b h} \left[\frac{1}{\rho r_a} - \frac{r_b \mu - 2gL}{r_a g - r_b h} - \frac{2g \ln[(r_a g)/(r_b h)]}{\rho(r_a g - r_b h)} + \frac{2g/h}{\rho r_b} - \frac{1}{\rho r_a} \right] \\
&= \frac{r_b}{r_a g - r_b h} \left[A + \frac{r_a g/h + (r_a g/h - r_b)}{\rho r_b r_a} \right] \geq 0;
\end{aligned} \tag{B-5}$$

$$\begin{aligned}
\frac{\partial(a_1^n + a_2^n)}{\partial r_a} &= \frac{\partial}{\partial r_a} \left\{ \frac{1}{\rho r_a} - \frac{r_b \mu}{r_a g - r_b h} + \frac{2gL}{r_a g - r_b h} - \frac{2g \ln[(r_a g)/(r_b h)]}{\rho(r_a g - r_b h)} \right\} \\
&= -\frac{1}{\rho r_a^2} + \frac{r_b g \mu}{(r_a g - r_b h)^2} - \frac{2g^2 L}{(r_a g - r_b h)^2} - \frac{2g}{\rho} \frac{(r_a g - r_b h)/r_a - g \ln[(r_a g)/(r_b h)]}{(r_a g - r_b h)^2} \\
&= \frac{g}{r_a g - r_b h} \left[\frac{r_b \mu}{r_a g - r_b h} - \frac{2gL}{r_a g - r_b h} - \frac{2}{\rho r_a} + \frac{2g \ln[(r_a g)/(r_b h)]}{\rho(r_a g - r_b h)} \right] - \frac{1}{\rho r_a^2} \\
&= -\frac{g}{r_a g - r_b h} \left(A + \frac{1}{\rho r_a} \right) - \frac{1}{\rho r_a^2} \leq 0;
\end{aligned} \tag{B-6}$$

$$\begin{aligned}
\frac{\partial(a_1^n + a_2^n)}{\partial r_b} &= \frac{\partial}{\partial r_b} \left\{ \frac{1}{\rho r_a} - \frac{r_b \mu}{r_a g - r_b h} + \frac{2gL}{r_a g - r_b h} - \frac{2g \ln[(r_a g)/(r_b h)]}{\rho(r_a g - r_b h)} \right\} \\
&= -\frac{r_b h \mu}{(r_a g - r_b h)^2} + \frac{2ghL}{(r_a g - r_b h)^2} + \frac{2g}{\rho} \frac{(r_a g - r_b h)/r_b - h \ln[(r_a g)/(r_b h)]}{(r_a g - r_b h)^2} \\
&= \frac{h}{r_a g - r_b h} \left[-\frac{r_b \mu}{r_a g - r_b h} + \frac{2gL}{r_a g - r_b h} + \frac{2g}{\rho h r_b} - \frac{2g \ln[(r_a g)/(r_b h)]}{\rho(r_a g - r_b h)} \right] \\
&= \frac{h}{r_a g - r_b h} \left(A - \frac{1}{\rho r_a} + \frac{2g}{\rho h r_b} \right) \\
&= \frac{h}{r_a g - r_b h} \left(A + \frac{r_a g - r_b h}{\rho h r_b r_a} + \frac{r_a g}{\rho h r_b r_a} \right) \geq 0.
\end{aligned} \tag{B-7}$$

Proof of Result 11:

$$\begin{aligned}
\frac{\partial(b_1^n + b_2^n)}{\partial h} &= \frac{\partial}{\partial h} \left\{ \frac{1}{\rho r_b} - \frac{r_a \mu}{r_a g - r_b h} + \frac{2hL}{r_a g - r_b h} - \frac{2h \ln[(r_a g)/(r_b h)]}{\rho(r_a g - r_b h)} \right\} \\
&= -\frac{r_a r_b \mu}{(r_a g - r_b h)^2} + \frac{2[L(r_a g - r_b h) + r_b h L]}{(r_a g - r_b h)^2} - \frac{2 \ln[(r_a g)/(r_b h)]}{\rho(r_a g - r_b h)} \\
&\quad - \frac{2h - (1/h)(r_a g - r_b h) + r_b \ln[(r_a g)/(r_b h)]}{\rho (r_a g - r_b h)^2} \\
&= -\frac{r_a r_b \mu}{(r_a g - r_b h)^2} + \frac{2r_a g L}{(r_a g - r_b h)^2} - \frac{2 \ln[(r_a g)/(r_b h)]}{\rho (r_a g - r_b h)} \\
&\quad + \frac{2(r_a g - r_b h) - h r_b \ln[(r_a g)/(r_b h)]}{\rho (r_a g - r_b h)^2} \\
&= -\frac{r_a r_b \mu}{(r_a g - r_b h)^2} + \frac{2r_a g L}{(r_a g - r_b h)^2} + \frac{2(r_a g - r_b h) - r_a g \ln[(r_a g)/(r_b h)]}{\rho (r_a g - r_b h)^2} \\
&= \frac{r_a}{r_a g - r_b h} \left[-\frac{r_b \mu}{r_a g - r_b h} + \frac{2g L}{r_a g - r_b h} + \frac{2}{\rho r_a} - \frac{2}{\rho} \frac{g \ln[(r_a g)/(r_b h)]}{r_a g - r_b h} \right] \\
&= \frac{r_a}{r_a g - r_b h} \left(A + \frac{1}{\rho r_a} \right) \geq 0; \tag{B-9}
\end{aligned}$$

$$\begin{aligned}
\frac{\partial(b_1^n + b_2^n)}{\partial g} &= \frac{\partial}{\partial g} \left\{ \frac{1}{\rho r_b} - \frac{r_a \mu}{r_a g - r_b h} + \frac{2hL}{r_a g - r_b h} - \frac{2h \ln[(r_a g)/(r_b h)]}{\rho(r_a g - r_b h)} \right\} \\
&= \frac{r_a r_a \mu}{(r_a g - r_b h)^2} - \frac{2r_a h L}{(r_a g - r_b h)^2} - \frac{2h}{\rho} \frac{(1/g)(r_a g - r_b h) - r_a \ln[(r_a g)/(r_b h)]}{(r_a g - r_b h)^2} \\
&= \frac{r_a}{r_a g - r_b h} \left(\frac{r_a \mu}{r_a g - r_b h} - \frac{2hL}{r_a g - r_b h} - \frac{2h/g}{\rho r_a} + 2 \frac{h \ln[(r_a g)/(r_b h)]}{\rho(r_a g - r_b h)} \right) \\
&= \frac{r_a}{r_a g - r_b h} \left\{ \left[-\frac{1}{\rho r_b} + \frac{r_a \mu}{r_a g - r_b h} - \frac{2hL}{r_a g - r_b h} + 2 \frac{h \ln[(r_a g)/(r_b h)]}{\rho(r_a g - r_b h)} \right] \right. \\
&\quad \left. + \frac{1}{\rho r_b} - \frac{2h/g}{\rho r_a} \right\} \\
&= \frac{r_a}{r_a g - r_b h} \left(-B + \frac{r_a g - 2r_b h}{\rho r_a r_b g} \right) \leq 0; \tag{B-10}
\end{aligned}$$

The inequality in (B-10) holds whenever $2r_b h \geq r_a g$.

$$\begin{aligned}
\frac{\partial(b_1^n + b_2^n)}{\partial r_a} &= \frac{\partial}{\partial r_a} \left\{ \frac{1}{\rho r_b} - \frac{r_a \mu}{r_a g - r_b h} + \frac{2hL}{r_a g - r_b h} - \frac{2h \ln[(r_a g)/(r_b h)]}{\rho (r_a g - r_b h)} \right\} \\
&= \frac{r_a g \mu}{(r_a g - r_b h)^2} - \frac{\mu}{r_a g - r_b h} - \frac{2hgL}{(r_a g - r_b h)^2} \\
&\quad - \frac{2h (1/r_a)(r_a g - r_b h) - g \ln[(r_a g)/(r_b h)]}{\rho (r_a g - r_b h)^2} \\
&= \frac{g}{r_a g - r_b h} \left[\frac{r_a \mu}{r_a g - r_b h} - \frac{2hL}{r_a g - r_b h} - \frac{2h}{\rho r_a g} + \frac{2}{\rho} \frac{h \ln[(r_a g)/(r_b h)]}{r_a g - r_b h} \right] - \frac{\mu}{r_a g - r_b h} \\
&= \frac{g}{r_a g - r_b h} \left(-B + \frac{1}{\rho r_b} - \frac{2h}{\rho r_a g} \right) - \frac{\mu}{r_a g - r_b h} \\
&= \frac{\partial b_i^n}{\partial g} \frac{g}{r_a} - \frac{\mu}{r_a g - r_b h} \leq 0;
\end{aligned} \tag{B-11}$$

The inequality in (B-11) holds whenever the inequality in (B-11) holds, i.e., whenever

$$2r_b h \geq r_a g .$$

$$\begin{aligned}
\frac{\partial(b_1^n + b_2^n)}{\partial r_b} &= \frac{\partial}{\partial r_b} \left\{ \frac{1}{\rho r_b} - \frac{r_a \mu}{r_a g - r_b h} + \frac{2hL}{r_a g - r_b h} - \frac{2h \ln[(r_a g)/(r_b h)]}{\rho (r_a g - r_b h)} \right\} \\
&= -\frac{1}{\rho r_b^2} - \frac{r_a h \mu}{(r_a g - r_b h)^2} + \frac{2h^2 L}{(r_a g - r_b h)^2} \\
&\quad - \frac{2h - (1/r_b)(r_a g - r_b h) + h \ln[(r_a g)/(r_b h)]}{\rho (r_a g - r_b h)^2} \\
&= \frac{h}{r_a g - r_b h} \left(-\frac{r_a \mu}{r_a g - r_b h} + \frac{2hL}{r_a g - r_b h} + \frac{2}{\rho r_b} - \frac{2}{\rho} \frac{h \ln[(r_a g)/(r_b h)]}{r_a g - r_b h} \right) - \frac{1}{\rho r_b^2} \\
&= \frac{h}{r_a g - r_b h} \left(B + \frac{1}{\rho r_b} \right) - \frac{1}{\rho r_b^2} \\
&\geq \frac{h}{2r_b h - r_b h} \left(B + \frac{1}{\rho r_b} \right) - \frac{1}{\rho r_b^2} = \frac{B}{2r_b} \geq 0.
\end{aligned} \tag{B-12}$$

The inequality in (B-12) holds whenever $2r_b h \geq r_a g$. \square