Infectious Disease, Productivity, and Scale in Open and Closed Animal Production Systems

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Abstract

Comparative advantage motivates large trade flows in feeder animals throughout the world. Trade creates externalities when animal diseases can spread beyond the purchasing farm. When growers can choose between open and closed production systems, Nash equilibrium will likely involve socially excessive trading. Supply response to an increase in marginal costs may be positive. While first-best involves marketwide adoption of either an open-trade or closed-farm system, equilibrium may entail heterogeneous systems. If this is the case, then the feeder trade should be banned. Within a farm, we show how risk of infectious disease can create decreasing returns to scale when the technology is otherwise increasing in returns to scale. Control of disease risk through bilateral contracts or damage-control technologies will increase scale of production in fattening, while better sorting in feeder animal markets will have ambiguous effects on scale.

Keywords*:* feeder trade, industrialization, information, Nash equilibrium, vertical integration, welfare.

JEL Classification: D62, H23, L22

INFECTIOUS DISEASE, PRODUCTIVITY, AND SCALE IN OPEN AND CLOSED ANIMAL PRODUCTION SYSTEMS

Introduction

In December 2003, a Washington State dairy cow was identified as having bovine spongiform encephalopathy. The cow had originally entered the United States in an 81-animal shipment from Canada. An investigation by the U.S. Department of Agriculture (USDA) into what ultimately happened to this shipment concluded after accounting for only 29 animals (APHIS 2004). Recent inquiries by Skaggs et al. (2004) into the subsequent histories of Mexican live cattle imports to the United States also point to data inadequacies. These cases give testimony to the openness of bovine agriculture in North America and to the information problems this openness can generate. Tables 1 and 2 summarize the extent of internal flows (United States) and international flows of some of the main husbanded species.

Today, world trade is significantly larger than it was in the 1970s. For pigs, U.S. domestic and international flows have increased markedly since 1990. There are many reasons for these flows, including policy motives, regional economic growth patterns, and cultural issues. One important reason is that animal feed requirements change with animal maturity. Feed is bulky, and it is often more profitable to move young animals closer to the feed source rather than transport the feed. In recent years, U.S. feeder pigs have tended to move from the Atlantic South (North Carolina) and Canada to be fattened in the Corn Belt. Feeder cattle tend to move from the sparse West to the Midwest and Great Plains (Shields and Matthews 2003). There are also specialist trades in replacement dairy cows, poultry chicks, and laying hens. These animal flows are subject to some regulations, in large part because of disease risks that affect productivity and sometimes may affect human health.

Our main thesis in this paper is that openness matters in the presence of communicable diseases. We address two related issues having to do with the extent of openness in

TABLE 1. Internal (state-to-state) shipments of live animals—United States

Item	1960	1970	1980	1990	2001	2002
			(Million Head)			
Cattle	13.5	22.9	20.0	22.5	21.8	
Pigs	2.5	3.2	4.6	3.6	26.9	
Sheep	6.1	4.0	2.2	2.2	1.5	

Source: Shields and Mathews 2003.

TABLE 2. Exports of live animals—world

Item	1960	1970	1980	1990	2001	2002
			(Million Head)			
Cattle	4.9	6.7	7.1	8.0	8.3	8.8
Pigs	2.6	4.6	10.5	12.9	15.5	17.0
Sheep	6.5	8.5	16.4	19.0	17.1	18.4
Goats	1.3	1.5	2.1	2.0	3.1	2.6
Chickens	0.1	0.1	0.3	0.4	0.8	0.8
Turkeys	0.0	0.003	0.01	0.02	0.05	0.06

Source: FAO, Agricultural Data, Agriculture and Food Trade, April 2004.

animal production systems. We provide a simple formal model of the tension between regional comparative advantage as a motive for animal trade and efficiency losses due to higher incidence of infectious diseases under open trade. The model characterizes the Nash equilibrium (NE) set and provides suggestions on when it would be socially optimal to regulate the feeder animal trade. There can be a unique equilibrium or multiple stable equilibria, depending on how the extent of infection affects the productivity of the closed system relative to the open system. Supply response to a cost increase can be positive. In our model, too, it is optimal to restrict trade whenever market equilibrium comprises a mix of open and closed system farms.

We then turn to the consequences of sourcing, sorting, and disease husbandry decisions for efficiency and scale in fattening. We find that the risk of realizing a communicable disease within a feedlot discourages the exploitation of technical economies of scale. But the relationship between scale and animal health class may not be monotone, a consequence of interanimal dependencies when animals infect each other. These dependencies

also motivate sorting in feeder animal markets. Contracts to procure animals through private parties, rather than through open market sourcing, will likely decrease systemic disease risk and increase production scale. In addition, the model allows us to conclude that a ban on a damage-control input may decrease lot scale. After providing case studies of managing communicable animal disease, the paper concludes with a brief discussion.

Animal Movements and Communicable Disease Externalities

Countries coordinate efforts to eradicate diseases that pose the most significant threats to animal production systems and human health (Otte, Nugent, and McLeod 2004). Table 3 summarizes some of the more important transboundary animal diseases. Institutions involved in global efforts to control communicable animal diseases include public veterinary services at the national and regional levels, the Food and Agriculture Organization (FAO) of the United Nations, and the Office International des Épizooties (OIE).

To be successful in eradicating an infectious disease, the most effective strategy is often to cull all herds with infected animals, leave the production facilities idle for a sufficiently long duration, and strictly observe herds in a given radius around an infected farm. Controls often include forbidding transportation of animals from the farm and restricting trade with a region or country. Human travel may also be discouraged, perhaps by denying permission to hold sports events, animal fairs, cultural events, and elections. Because events surrounding outbreaks of diseases (listed in Table 3) can directly affect the daily lives of whole societies, outbreaks are widely reported. Many other infectious animal diseases that are not as widely publicized also cause considerable economic losses. There are several ways in which animal diseases affect the productivity of a herd. Apart from mortality, depressed productivity may lead to low feed conversion efficiency, reproductive losses, poor product quality, early culling of breeding and dairy stock, and reduced efficiency of management effort.

The formal economics literature on animal disease externalities is sparse. Most studies have been done in the field of veterinary science and consist in estimating the cost of production losses due to a disease. An early FAO (1962) study estimated that losses due to disease amounted to 15 percent of total livestock output in developed countries and 30 percent in less-developed countries. A study of health and fertility problems in dairying

TABLE 3. Important transboundary animal diseases and recent outbreaks

Disease	Affected Species and Countries	Epidemiology/Transmission	Economic Impact (actual outbreak values)
Foot-and-Mouth Disease	Cloven-hoofed livestock; Europe, South-America, Asia	Highly contagious virus. Spread by movement of infected animals, animal products, contaminated objects, and wind. Vaccination complicated because of multiple virus traits and loss of disease-free status.	February 2001, UK: cost $3.6-$11.6 billion[a] 1997, Philippines: Direct costs $25 million[b]
Bovine Spongiform Encephalopathy	Cattle; Europe, Japan, North America	Prion disease. Link to new-variant Creutzfeldt-Jakob disease. Likely transmission through inclusion of infected animals in feed.	Since mid-1980s, UK: Overall losses: $5.8 billion[a]
Classical Swine Fever	Pigs; Europe, South Asia, Latin America	Virus. Effects vary from subclinical to sudden death. Transmission by feed, respiration, semen, and manure spreading.	1997-98, Netherlands: Short-term costs $23 million[c]
Newcastle Disease	Birds	Virus. Primarily spread from bird to bird but also through contaminated feed, water, and clothing.	2000, Mexico: Major outbreak, 13.6 million birds were destroyed[d]
Avian Influenza	Poultry; outbreaks in Asia, Americas, Europe	Highly lethal virus. Probably ubiquitous in wild waterfowl. Wide range of disease symptoms, ranging from mild to severe.	1999-2001, Italy: 13 million birds killed. Also Asia (1997-98, 2003, 2004), Netherlands (2003)[e]

[a] Mathews and Buzby 2001.
[b] Perry et al. 1999.
[c] Horst et al. 1999.
[d] Otte, Nugent, and McLeod 2004.
[e] World Health Organization 2004.

(Dijkhuizen 1990) assessed losses equal to 10 percent of gross production value. Bennett (2003) estimated the annual value of output loss and input expenditure with treatment and prevention costs for 30 diseases of livestock in Great Britain at about 3.2 percent of the value of animal production.

McInerney (1996) developed a model that recognizes that the cost of disease is a composite loss due to disease and cost of treatment. Animal diseases are modeled in the primal production function as lowering the productivity index, and we follow this approach. The model has been applied in the study of controlling infectious disease on dairy farms in Canada by Chi et al. (2002). A limitation for policy purposes is that these models do not address the role of disease externalities, namely, the divergence between private and public consequences of actions.[1] This is important because if equilibrium is to be understood in the presence of contagion then one must establish when private actions are consistent with the actions of others. Economic inquiries into control of infectious diseases among humans have related equilibrium rates of infection under selfish behavior to economic policies (Geoffard and Philipson 1996, 1997; Kremer 1996). However, their models provide little guidance for animal health maintenance because humans are not traded and are largely free to behave as they will.

Empirical models to simulate the spread of contagious transboundary diseases include McCauley et al. (1979); Mangen, Nielsen, and Burrell (2002); Matthews et al. (2003); Mintiens et al. (2003); and Schoenbaum and Disney (2003). While useful in forecasting the spread of disease and analyzing the effectiveness of different intervention strategies, these models do not directly recognize the decision problem at the farm level—that private incentives must be in place to promote private decisions that are in the public interest.[2] To close the gap that exists in the disease literature, we develop a simple model that provides insight into regulating communicable animal diseases.

Model

Two types of commercial animal production systems exist. These are a closed (integrated) system that fattens animals from birth, and an open (specialist) system in which feeder animals are produced at one location and fattened at a second. Both systems involve a continuum of competitive firms that produce fattened animals who are at risk for

contracting a communicable chronic disease that reduces output. There is free entry from a large pool of potential producers and each producing firm has unit capacity. Fraction $\mu \in [0,1]$ of producing firms uses an open system while the residual uses a closed system. We will model the closed and open systems in turn.

Closed System

In the closed system, it costs c_{fe} to produce each feeder animal internally and c_{fa} to fatten the animal. A chronic disease affecting productivity and mean output for a firm in the closed system is $G[I] \in [0,1]$, where $I \in [0,1]$ is an index of disease severity. In order to capture the consequences of animal congestion for the spread of disease in a region, disease severity is assumed to be strictly increasing in the region's output, Q. Disease severity is also assumed to be strictly increasing in the fraction of firms that produces under the open system. Thus, disease severity index $I(\mu, Q) : [0,1] \times \bar{\mathbb{R}}_{+} \to [0,1]$ is continuously differentiable and strictly increasing in both arguments.[3] In addition, $G[I]$ is continuously differentiable with $G_I[I] < 0$ to capture productivity losses. Closed-system mean profit per unit capacity is

$$\pi^{cs} = PG[I] - c_{fe} - c_{fa}, \tag{1}$$

where P is the product market price. The market demand function is $P(Q)$ with $P_Q(Q) < 0$. The number of producing firms adjusts to match supply with demand.

Open System

In the open system it costs w to procure a feeder animal. Procurement may be in the spot market, perhaps by live auction where feeder animals from a large number of farms mingle. Procurement may also be by bilateral contract. Inequality $w < c_{fe}$ is assumed so that specialization efficiencies could give rise to trade opportunities. It costs c_{fa} to fatten the animal, as in the closed system. Product sells for the same price, but disease losses differ relative to the closed system. Mean yield on a given open-system farm is $H[I] > 0$ where $H[I]$ has the same analytic properties as $G[I]$. In addition,

$H[I] < G[I] \; \forall I \in [0,1]$ because the open system is more exposed to communicable disease risk. Open-system mean profit per unit capacity is

$$\pi^{os} = PH[I] - w - c_{fa}, \tag{2}$$

where product price is as under the closed system and free entry balances supply with demand.

Competition and Equilibrium

The context is an infinite-player, two-strategy game in which the growers choose between closed-system and open-system procurement. The respective payoffs are as given in equations (1) and (2). We consider only pure strategy NE because a non-trivial, mixed strategy will always be dominated by a pure strategy.

Equilibrium may be described as a pair $\{\mu^*, P^*\}$ subject to $P^* > 0$ and $\mu^* \in [0,1]$. The market-level output consistent with P^* is Q^* and equilibrium can alternatively be described as $\{\mu^*, Q^*\}$. Equilibrium incidence of disease is $I^* = I(\mu^*, Q^*)$. There is free entry to each system so that economic profits are null, that is, $\pi^{cs} \leq 0$ and $\pi^{os} \leq 0$, where one weak inequality is an equality if production occurs. These conditions can be stated as

$$P^* \leq P^{cs} \equiv \frac{c_{fe} + c_{fa}}{G[I^*]}; \qquad P^* \leq P^{os} \equiv \frac{w + c_{fa}}{H[I^*]}; \tag{3}$$

with at least one an equality. Define

$$\theta = \frac{c_{fe} + c_{fa}}{w + c_{fa}}; \qquad M[I] = \frac{G[I]}{H[I]}, \tag{4}$$

where $\theta > 1$ is assured. Expression θ may be thought of as the ratio of internal private production costs to external private production costs.

If profits are to be equal across systems, and both are equal to zero given free entry, then $\theta = M[I^*]$. For zero profits, too, the law of demand requires that both $P(Q^*) = (c_{fe} + c_{fa}) / G[I^*]$ and $P(Q^*) = (w + c_{fa}) / H[I^*]$ where $P^{-1}(\cdot)$ is the inverse demand function. Summarizing, label the three conditions as

$$L1: \theta = M[I^*]; \qquad L2: Q^* = P^{-1}\left(\frac{c_{fe}+c_{fa}}{G[I^*]}\right); \qquad L3: Q^* = P^{-1}\left(\frac{w+c_{fa}}{H[I^*]}\right). \tag{5}$$

Only two of $L1$-$L3$ are independent, and any two solve to identify an equilibrium pair $\{\mu^*, P^*\}$ with associated market quantity Q^*. Our conditions on $M[\cdot]$ do not require that there be a unique solution to $L1$ on the domain of I. Notice that $L1$ is free of the demand function. Consequently, it is sometimes convenient to study $L1$ and one of $L2$ or $L3$ rather than the pair $L2$ and $L3$.

Characterizing Equilibrium

To better understand where $L1$-$L3$ could intersect, differentiate each in variables Q and μ:

$$\frac{dQ}{d\mu}|_{L1} = -\frac{I_\mu}{I_Q} < 0; \qquad \frac{dQ}{d\mu}|_{L2} = -\frac{\frac{I_\mu G_I}{G}}{\frac{P_Q}{P}+\frac{I_Q G_I}{G}} < 0; \qquad \frac{dQ}{d\mu}|_{L3} = -\frac{\frac{I_\mu H_I}{H}}{\frac{P_Q}{P}+\frac{I_Q H_I}{H}} < 0. \tag{6}$$

Furthermore, at any given point (μ, Q), $P_Q / P < 0$, and so

$$\frac{dQ}{d\mu}|_{L1} < \frac{dQ}{d\mu}|_{L2}, \qquad \frac{dQ}{d\mu}|_{L1} < \frac{dQ}{d\mu}|_{L3}. \tag{7}$$

This means that if, say, $L1$ (for a given value of I^*) and $L2$ cross at all, then they cross just once. Comparing the two zero profit curves, we have $(dQ/d\mu)|_{L2} \geq (\leq)\ (dQ/d\mu)|_{L3}$ if

$$\frac{d\text{Ln}(G[I])}{d\text{Ln}(I)} \leq (\geq) \frac{d\text{Ln}(H[I])}{d\text{Ln}(I)}, \tag{8}$$

that is, the elasticity of production with respect to disease severity in the closed system is less (greater) than that in the open system.

An example is provided in Figure 1, where a single crossing point exists (all three curves must cross there) and this NE crossover point is at (μ^*, Q^*). In it we have made

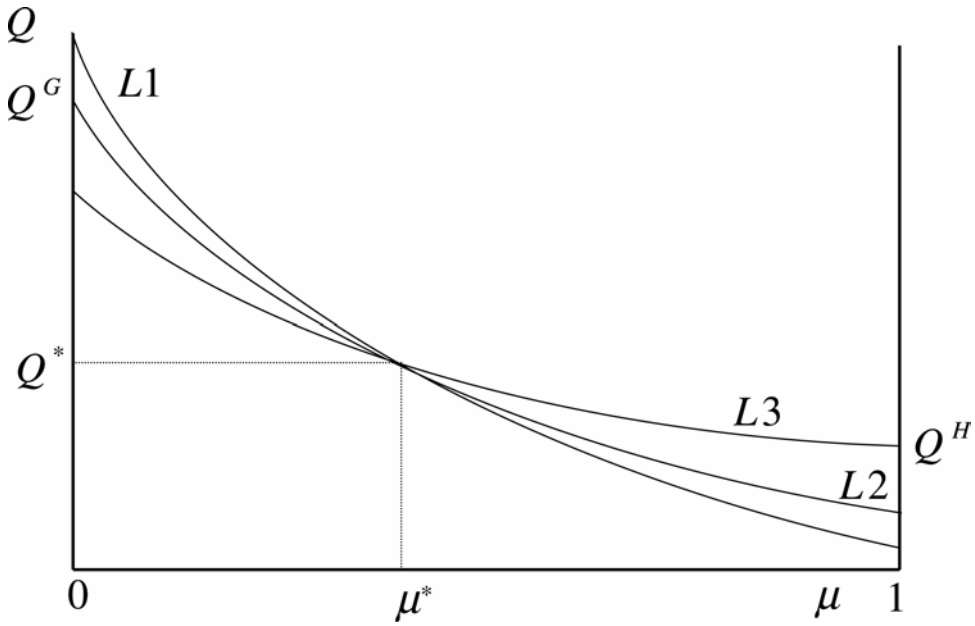

FIGURE 1. Heterogeneous system equilibrium

the assumption that $(dQ/d\mu)|_{L2} \leq (dQ/d\mu)|_{L3}$. Point $(0,Q^G)$ is the point on $L2$ such that $\mu = 0$, while $(1,Q^H)$ is the point on $L3$ such that $\mu = 1$.

A concern with understanding Figure 1 is whether the solution is unique, stable, and interior. There may not be an interior solution I^* to equation $L1$; there may be multiple solutions; and a solution may not be locally stable, that is, robust to a local perturbation. Suppose first that $\theta < M[I(\mu,Q)] \; \forall (\mu,Q) \in L2$. Then no firm would ever have a private incentive to choose the open system and so equilibrium requires $\mu^* = 0$. Under free entry, market output must be Q^G, the unique solution to $P(Q^*)G[I(0,Q^*)] = c_{fe} + c_{fa}$. Suppose instead that $\theta > M[I(\mu,Q)] \; \forall (\mu,Q) \in L2$ and no firm would ever have a private incentive to choose the closed system. Then $\mu^* = 1$ and market output must be Q^H, the unique solution to $P(Q^*)H[I(1,Q^*)] = w + c_{fa}$.

PROPOSITION 1. *(a) If* $\theta < M[I(\mu,Q)] \; \forall (\mu,Q) \in L2$*, then the unique NE is* $(\mu^*,Q^*) = (0,Q^G)$.

(b) If $\theta > M[I(\mu,Q)] \; \forall (\mu,Q) \in L2$*, then the unique NE is* $(\mu^*,Q^*) = (1,Q^H)$.

Now consider when there is at least one solution to $L1$. Suppose $M[\cdot]$ is locally increasing in I and a perturbation to an I^* renders the perturbed value of I to be too large to satisfy $L1$. Then, $M[I] > \theta$ and firms deviate to the more profitable closed system so that the value of μ decreases. This does indeed reduce the level of infection so that one can iterate toward equilibrium. Suppose instead that $M[\cdot]$ is decreasing and a perturbation to an I^* renders the perturbed value of I to be too large to satisfy $L1$ as $M[I] < \theta$. By $L1$, firms deviate to the more profitable open system, μ increases, and the infection index becomes larger. So only locally stable equilibria occur where $M[\cdot]$ is increasing.

Assume that only isolated points solve $M_I[I] = 0$.[4] Assign solutions to $M[I] = \theta$ such that $M[I] - \theta$ does not change sign locally as unstable.[5] Define the set of stable solutions to $M[I] = \theta$ as $S^*(M, \theta)$. Denote condition $M[I(0, Q^G)] < \theta$ as C1. The condition ensures that when all firms are closed, then there is strictly positive profit under the open system and so $(0, Q^G)$ cannot be NE. If $M[I(0, Q^G)] > \theta$ then assert that C2 applies. Similarly, denote condition $M[I(1, Q^H)] > \theta$ as C3. When all firms are open and C3 applies, then there is strictly positive profit under the closed system and so $(1, Q^H)$ cannot be NE either. If $M[I(1, Q^H)] < \theta$ then assert that C4 applies.

PROPOSITION 2. *Under conditions*

(a) *C1 and C3, then the set of stable NE is*

$$\{\{\mu^*, (c_{fe} + c_{fa}) / G[I(\mu^*, Q^*)]\} : (\mu^*, Q^*) \in S^*(M, \theta)\}.$$

(b) *C1 and C4, then the set of stable NE is as in (a) in union with*

$$\{1, (w + c_{fa}) / H[I(1, Q^H)]\}.$$

(c) *C2 and C3, then the set of stable NE is as in (a) in union with*

$$\{0, (c_{fe} + c_{fa}) / G[I(0, Q^G)]\}.$$

(d) *C2 and C4, then the set of stable NE is as in (b) in union with*

$$\{0, (c_{fe} + c_{fa}) / G[I(0, Q^G)]\}.$$

Under part (a), only interior equilibria result. Given that $G[I]$ and $H[I]$ are strictly

positive and continuous, ratio $M[I]$ must be too. As C1 and C3 require that $M[I]-\theta$ changes sign, continuity ensures there is at least one crossing from below as μ changes so that a stable equilibrium exists. Under part (b), there is no guarantee that a crossing occurs at all so that part (b) in Proposition 1 could apply. With part (c), it may be that no crossing occurs either and then part (a) of Proposition 1 applies. With part (d), a crossing occurs and at least one crossing point has negative value for $M_I[I]$. In that situation, we cannot rule out either the polar market-wide closed system or the polar market-wide open system. The situation where the only stable equilibria are these two polar cases is of particular interest, and we will return to it.

Concerning interior solutions, notice that $d\theta / dc_{fe} > 0$, $d\theta / dw < 0$, and $d\theta / dc_{fa} < 0$. Remembering that $M_I > 0$ for a locally stable equilibrium, Figure 2 illustrates the effect of an increase in the value of w on curve $L1$ and thus on equilibrium. In general, one can assert the following.

PROPOSITION 3. *For any element of* $\{\{\mu^*, (c_{fe}+c_{fa})/G[I(\mu^*,Q^*)]\} : (\mu^*,Q^*) \in S^*(M,\theta)\}$,

(a) μ^* *is increasing in* c_{fe}. *Equilibrium product price is increasing in* c_{fe}.

(b) μ^* *is decreasing in* w *and* c_{fa}. *Equilibrium product price is decreasing in* w *and* c_{fa}.

The most noteworthy effects are the positive supply responses to increasing costs in part (b). The rationale is as follows. An increase in the internalized relative cost of the open system elicits a shift toward the closed system, a decrease in equilibrium disease incidence, enhanced productivity for all firms, and an increase in market output.

An interesting situation arises when $M[I]$ is strictly decreasing on the domain of I while $M[I(0,Q^G)] > \theta > M[I(1,Q^H)]$. Then there are two stable NE: either all firms use the open system or all use the closed system. In this case, an increase in w (e.g., a per-head tax at barn auction sales) either has no effect or it causes a discrete switch from solution $\{1,(w+c_{fa})/H[I(1,Q^H)]\}$ to solution $\{0,(c_{fe}+c_{fa})/G[I(0,Q^G)]\}$. The switchover

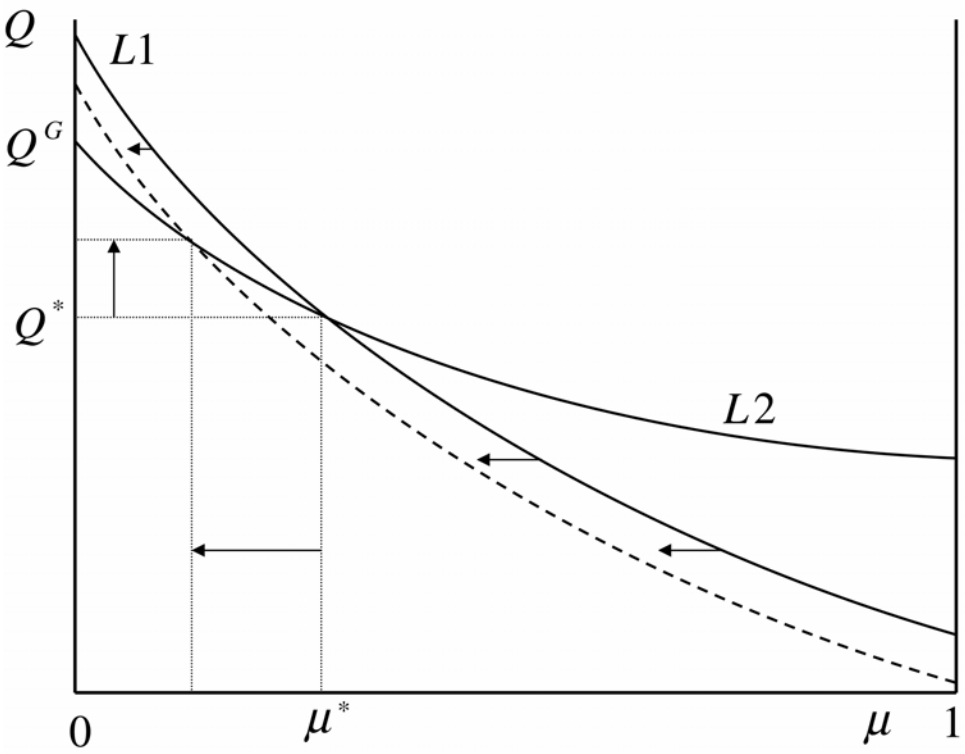

FIGURE 2. Increase in purchase cost shifts stable equilibria northwest

occurs when $P^* = (w + c_{fa}) / H[I(1, Q^H)] = (c_{fe} + c_{fa}) / G[I(1, Q^H)]$ and w then increases infinitesimally. But use equation (7) for different values of I to conclude that $G[I(0, Q^G)] > G[I(1, Q^H)]$, so that a discrete decrease in the value of P^* occurs.

PROPOSITION 4. *Suppose* $M[I(0, Q^G)] > \theta > M[I(1, Q^H)]$ *and* $M[I]$ *is strictly decreasing. Then any change in (stable) equilibrium in response to an increase in either* w *or* c_{fa} *(or a decrease in* c_{fe}*) involves a shift of all finishers from the open system to the closed system, a discrete increase in market output, and a discrete decrease in product price.*

Proposition 4 raises the possibility that a rapid change in the structure of animal production arises from a modest change in the price environment. The elasticity condition that $H_I / H > G_I / G$ on the domain of I (see relation [8] above) is neither intuitive nor unduly restrictive. Proposition 4 also motivates the idea of a disease eradication program in which movement controls temporarily raise unit cost w toward infinity until a new equilibrium is

established. Indeed, in an ideal world, after a disease has been eradicated one could trade and still achieve output $G[I = 0]$. Unfortunately, our model attaches system openness status to a production function and so we cannot pursue the idea of eradication in a formal manner.

Welfare

As the NE settled upon is not necessarily optimal when multiple equilibria exist, our interest in this section is to establish how to arrive at a superior equilibrium. To understand the equilibrium sought, note that, under free entry, fatteners receive no economic surplus. All surplus accrues to consumers, and so social surplus increases in equilibrium output Q^*. In addition, even if $(0, Q^G)$ is not an NE, it is preferred to any interior NE because equation (6) ensures that market output has to be larger when all firms are closed.

PROPOSITION 5. *(a) If* $(0, Q^G)$ *is an NE, then it is the NE that supports the largest surplus. (b) If there exists any interior NE, i.e.,* $\mu^* \in (0,1)$ *be it stable or unstable, then it is welfare improving to ban the feeder trade.*

The prescription to ban trade should not be taken literally. The real issue is the degree of openness of the market-level production system. Quarantine laws and other movement controls are intermediate approaches to reducing system openness. In addition, an information system can be viewed as a substitute for movement controls.

A concern that remains is to characterize when the open system is optimal. This is true whenever $Q^H > Q^G$. Define $w = w^H$ such that $Q^H = Q^G$, i.e., $P(Q^H) = P(Q^G)$ so that

$$w^H = (c_{fe} + c_{fa}) \frac{H[I(1, Q^H)]}{G[I(0, Q^H)]} - c_{fa}. \tag{9}$$

If $w < w^H$, then equation (6) assures that the unique equilibrium is $(1, Q^H)$. Because $Q^H > Q^G$ under this value of w, this equilibrium is also first-best. We have already shown that no intermediate NE is optimum.

PROPOSITION 6. *If* $w < w^H$*, then equilibrium is efficient. If* $w \geq w^H$*, then any tax* τ *on trade in feeder animals such that* $\tau > c_{fe} - w$ *supports the first-best equilibrium* $(0, Q^G)$.

Figure 3 depicts when the feeder trade is both optimal and the unique NE. At that point, w is sufficiently low so that the least value of Q on the closed-system zero-profit line $L3$ (i.e., Q^H) exceeds the greatest value of Q on the open-system zero-profit line $L2$ (i.e., Q^G). If $Q^H < Q^G$ instead, then $(1, Q^H)$ cannot be the unique NE and $(0, Q^G)$ is first-best.

Internal Management of Communicable Disease Risk

In addition to inter-farm disease externalities, there are also intra-farm communicable disease problems. In order to understand better the economics of intra-farm communicable disease problems, we will ignore inter-farm externalities for the remainder of the paper. The central trade-off will be between technical scale economies in fattening and the risk in large feedlots that some animal may cause lot-wide damage.

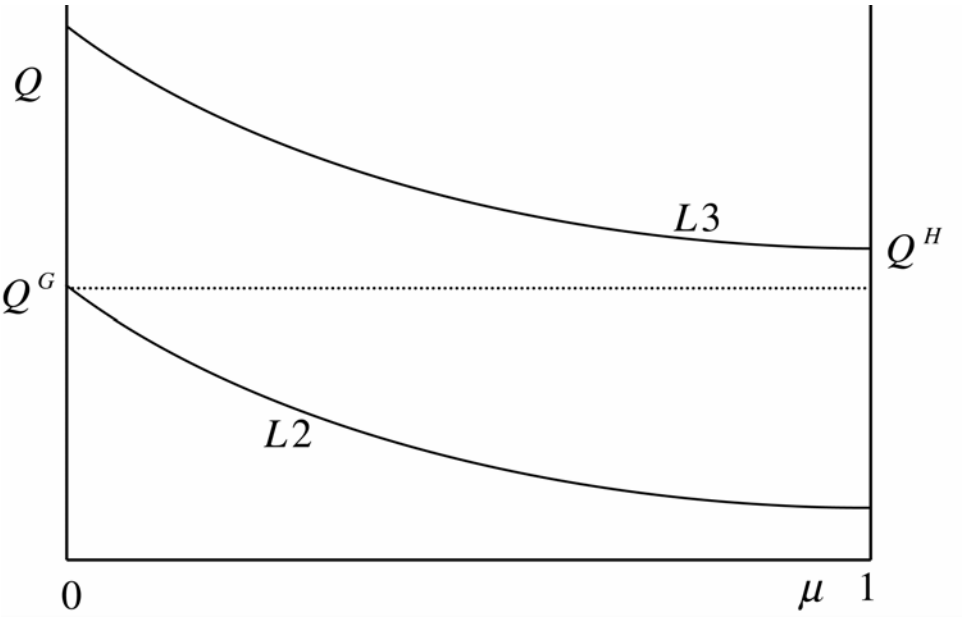

FIGURE 3. When feeder trade is optimal, unique equilibrium occurs

Our basic template in modeling firm-level production under communicable disease risk is Kremer's (1993) O-ring production function. Ignoring disease costs for the moment, the technical cost of fattening q animals is given by the twice continuously differentiable function $C(q)\colon\ [0,\infty)\to[0,\infty)$. There is a positive probability that a contagious disease affects the production unit. For simplicity we assume that if any animal becomes infected then all animals in the unit become infected. If infected, then the magnitude of loss per animal is δ. The probability that one animal *does not* become infected is $p\in(0,1]$. Disease probabilities are independent across animals, an assumption we will revisit later. The probability that a lot of q animals, each with the same p value, does not become infected is $p^q = e^{q\eta}, \eta = \mathrm{Ln}(p) \le 0$.[6]

Sorting by Health Classes

Now consider two lots of animals where all animals are purchased in the feeder market. The lots are comprised of mixed health classes. Lot I has q_1^I animals each with probability p_1 (of class p_1) that it is healthy and q_2^I animals each with probability $p_2, 0 < p_2 < p_1$, that it is healthy. These probabilities are known to all, so that there are no information externalities. Lot II has q_1^{II} animals of class p_1 and q_2^{II} animals of class p_2. Let revenue per animal be R, while the value of each health class p_i animal is $w(p_i)$, called the feeder price-health schedule. We will solve for the value of $w(p_i)$ later. Expected profit aggregated over both lots is

$$\begin{aligned}\pi &= (R-\delta)(q_1^I + q_2^I + q_1^{II} + q_2^{II}) - C(q_1^I + q_2^I) - C(q_1^{II} + q_2^{II}) \\ &+(q_1^I + q_2^I)\delta p_1^{q_1^I} p_2^{q_2^I} + (q_1^{II} + q_2^{II})\delta p_1^{q_1^{II}} p_2^{q_2^{II}} - (q_1^I + q_1^{II})w(p_1) - (q_2^I + q_2^{II})w(p_2).\end{aligned} \tag{10}$$

Define lot I as the high exposure lot if $F_I \equiv (q_1^I + q_2^I)p_1^{q_1^I} p_2^{q_2^I} \ge (q_1^{II} + q_2^{II})p_1^{q_1^{II}} p_2^{q_2^{II}} \equiv F_{II}$. Suppose, without loss of generality, that $F_I \ge F_{II}$. If q_1^I increases by one and q_2^I decreases by one, then the value of $C(q_1^I + q_2^I)$ does not change while F_I increases in value. Obtain these animals by a unit decrease in q_1^{II} and a unit increase in q_2^{II} so that sums $q_1^I + q_1^{II}$ and $q_2^I + q_2^{II}$ are preserved. Note that under the exchange,

$$F_I + F_{II} \rightarrow F_I(p_1 / p_2) + F_{II}(p_2 / p_1) > F_I + F_{II}. \quad (11)$$

This is because $F_I p_1^2 + F_{II} p_2^2 = (F_I - F_{II}) p_1^2 + F_{II} p_1^2 + F_{II} p_2^2$, $(F_I - F_{II}) p_1^2 > (F_I - F_{II}) p_1 p_2$, $F_{II} p_1^2 + F_{II} p_2^2 > 2F_{II} p_1 p_2$, and $2F_{II} p_1 p_2 + (F_I - F_{II}) p_1 p_2 = F_I p_1 p_2 + F_{II} p_1 p_2$ so that $F_I p_1^2 + F_{II} p_2^2 > F_I p_1 p_2 + F_{II} p_1 p_2$, where the last statement is equivalent to equation (11). The animal exchange increases profit because terms in equation (10) other than F_I and F_{II} are invariant to the exchange. This notional animal exchange does not actually occur. Rather, growers recognize the inefficiency and never actually make the inappropriate placement.

In general, and regardless of how many health classes exist or how many production-fattening lots are under consideration, it can readily be shown that an exchange similar to that supporting inequality (11) always generates an increase in expected revenue for the same level of cost. This means that it is always efficient to exchange a lower-risk animal initially in a lower exposure lot for a higher-risk animal initially in a higher exposure lot. This process will continue until lots have become, as far as possible, sorted by health class.

PROPOSITION 7. *If aggregate expected profit is maximized, then animals of two different health classes, say, p_1 and p_2 with $p_1 \neq p_2$, can exist in, at most, one production lot.*

Kremer (1993) established the analogous result for formation of worker teams where team members have heterogeneous competencies, each contributing a single task to a project such that failure in one task means project failure.

Unit Costs

Henceforth we invoke Proposition 7 to assume that in-lot animals are of the same health class and we drop the health class notation. The unit cost of fattening is then

$$U(q) = \frac{C(q)e^{q\eta} + [C(q) + \delta q](1 - e^{q\eta})}{q} = A(q) + S(q); \quad A(q) = \frac{C(q)}{q}; \quad S(q) = \delta - \delta e^{q\eta}. \quad (12)$$

Define $A(q)$ as the unit technical cost and $S(q)$ as the unit disease cost. For a lot with given p, lot size in competitive equilibrium will be chosen to minimize the average cost

of fattening a feeder animal. Were average cost not minimized then economic profits would be other than zero. Feeder animal price $w(p)$ will satisfy $w(p) = R - \min_q U(q)$, that is, it is the Ricardian rent.

Incentive to Isolate

If unit technical cost $A(q)$ is constant, then unit cost of fattening is increasing in scale, that is, $dU(q)/dq = -\delta\eta e^{q\eta} > 0$ for $p < 1$.

PROPOSITION 8. *If* $A'(q) = 0 \; \forall q \in [0, \infty)$*, then optimum lot scale is* $q = 1$.

When $A(q)$ is decreasing and convex, then $U(q)$ is the sum of a decreasing, convex function and increasing, concave function $S(q)$. One cannot be a priori sure that any local minimum is a global minimum. Even if $A(q)$ is U-shaped (i.e., basin-shaped with interior minimum), one can only be sure that the minimizer of $U(q)$ is to the left of the minimizer of $A(q)$.

Figure 4 decomposes the unit cost function into unit technical cost and unit disease cost. Function $A(q)$ is decreasing at $q = 0$ and convex on $q \in [0, \infty)$ with interior minimum. But when p is comparatively low, then $U(q)$ is increasing for low positive lot scale before peaking and assuming convex curvature at higher lot scale. As drawn, isolation minimizes unit cost. When p is comparatively high, then $U(q)$, as drawn, is decreasing at $q = 0$ and convex on $q \in [0, \infty)$ with interior minimum. There are many other forms that $U(q)$ might take.

Interior Lot Scale

In order to rule out the isolation lot scale solution, we assert two related requirements.

Assumption 1. $U(q)$ is strictly convex in q, that is, $A''(q) > \delta\eta^2 e^{q\eta} \; \forall q \in [0, \infty)$. In addition, optimal solution q^* satisfies $q^* > 1$.

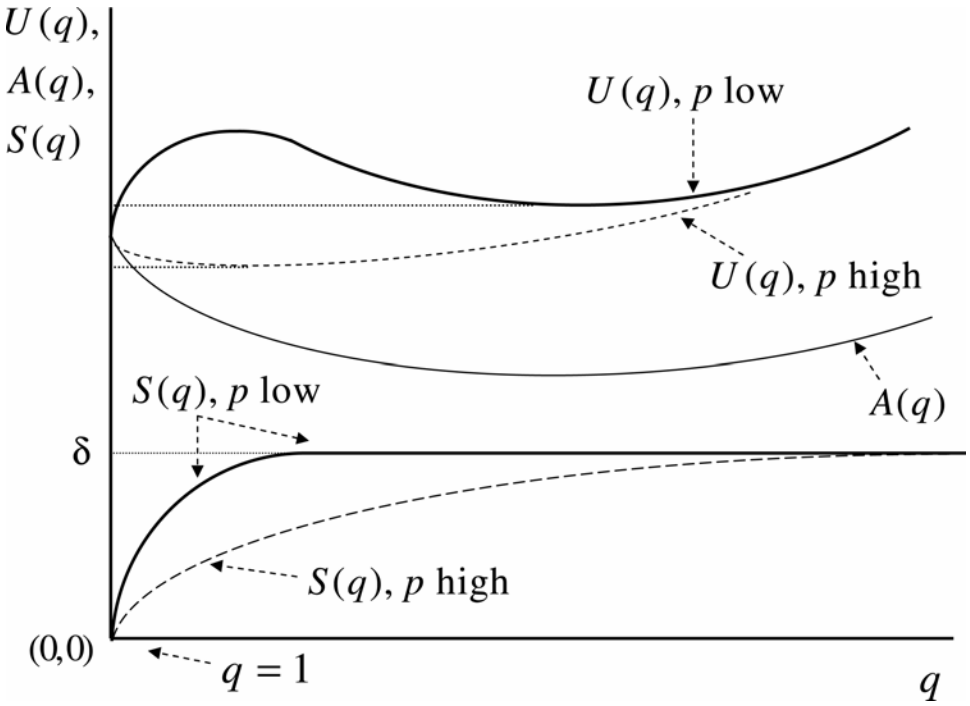

FIGURE 4. Decomposition of unit cost function for fattening

The convexity assumption ensures that any solution is a unique, global equilibrium. As we have already seen, the convexity condition is less likely to apply when p is comparatively small. Upon differentiating, unit-cost-minimizing lot scale q^* is determined by

$$A'(q^*) = \delta\eta e^{q^*\eta}. \tag{13}$$

Notice that the right-hand side is non-positive so that $A'(q^*) \leq 0$. Notice too that, as Figure 4 has already illustrated, when $A''(q) \geq 0 \; \forall q \in \overline{\mathbb{R}}_+$ then both left-hand and right-hand sides of equation (13) are increasing in the choice argument and a unique solution is not assured. Relation (13) also allows for an interpretation of when convexity applies in the locality of an optimum. Inserting (13) into $A''(q^*) > \delta\eta^2 e^{q^*\eta}$ generates $A''(q^*)/A'(q^*) < \text{Ln}(p)$ so that the coefficient of relative curvature for unit technical costs is bounded from above by the natural log of the animal health class. The curvature bound becomes less demanding as p increases.

Two interesting cross-derivatives are

$$\frac{d^2U(q^*)}{dqdp} = -\delta(1+q^*\eta)e^{(q^*-1)\eta}; \qquad \frac{d^2U(q^*)}{dqd\delta} = -\eta e^{q^*\eta}. \tag{14}$$

The first of these has the sign of $-(1+q^*\eta)$, while the second is positive.[7] From Topkis' (1995) theory of submodular cost functions, if Assumption 1 applies and the unique minimizing argument satisfies $q^* \leq -1/\mathrm{Ln}(p)$ then $U(q)$ is submodular in q and p in the proximity of the unique optimizing lot scale.[8] A small increase in p shifts q^* rightward. If $q^* \geq -1/\mathrm{Ln}(p)$ then a small increase in p shifts q^* leftward. Unit cost is also submodular in q and $-\delta$.

PROPOSITION 9. *Make Assumption 1. Then the unit-cost-minimizing lot scale is smaller in the presence of disease risk than absent disease risk (i.e., with $\eta = 0$ or $\delta = 0$). Unit-cost-minimizing lot scale is decreasing in magnitude of disease loss. If $q^* \leq (\geq) -1/\mathrm{Ln}(p)$ then a small increase in herd animal health class leads to an increase (decrease) in optimal lot scale.*

No matter how η changes, though, q^* cannot exceed in value the solution to $A'(q) = 0$. An example is worthy of note.

Example 1. Consider the negative exponential unit technical cost function $A(q) = A_0 + A_1 e^{-\lambda q}$, $A_0 \geq 0, A_1 > 0, \lambda > 0$, so that $\mathrm{Lim}_{q\to\infty} A(q) = A_0$ and (13) solves as

$$q^* = \frac{1}{\lambda+\eta}\mathrm{Ln}\left(\frac{-\lambda A_1}{\delta\eta}\right). \tag{15}$$

Absent disease considerations, that is, $\delta\eta = 0$, optimal lot scale would be unbounded. Because $A'(0) = -\lambda A_1$ and $S'(0) = -\delta\eta$, inequality $\mathrm{Ln}[-\lambda A_1/(\delta\eta)] > 0$ holds if and only if unit technical cost decreases at a more rapid rate at $q = 0$ than unit disease cost in-

creases at $q = 0$. Even if this were true, it is possible that $A'(q) + S'(q) \neq 0$ for any positive finite value of q. This possibility is ruled out when $A(q)$ is sufficiently convex, specifically, $\lambda + \eta > 0$. If the value η is sufficiently large (i.e., sufficiently close to 0) then $\text{Ln}[-\lambda A_1/(\delta\eta)] > 0$ and $\lambda + \eta > 0$, so that the unique solution is interior.

Note that $A''(q) > \delta\eta^2 e^{q\eta}$ requires $[1/(\lambda+\eta)]Ln(\lambda^2 A_1/\delta\eta^2) > q$ when $\lambda + \eta > 0$. This bound on q asserts that if there is an interior minimizer for $U(q)$ then $U(q)$ is not convex on all of $q \in [0, \infty)$. Assumption 1 is not satisfied for the negative exponential technology, but it was unduly restrictive in any case. Unit cost is only convex on $[0, \hat{q}]$, $\hat{q} \equiv [1/(\lambda+\eta)]Ln(\lambda^2 A_1/\delta\eta^2) = q^* + [1/(\lambda+\eta)]Ln(-\lambda/\eta) > q^*$. Convexity at q^* when q^* is the unique solution to $A'(q^*) + S'(q^*) = 0$ ensures that q^* is indeed the unique minimizer. At high values of *q*, unit cost must become concave because $U(q)$ increases toward asymptote $A_0 + \delta$.

From equation (15), $dq^*/d\eta = -(q^*\eta + 1)/[(\lambda+\eta)\eta]$ and the derivative sign is as posited in Proposition 9. To verify that the derivative may take either sign, fix the value of $q^*\eta + 1$ at zero and write $q^*\eta + 1 = [\lambda + \eta + \eta\text{Ln}(-\lambda A_1/\delta\eta)]/[\lambda+\eta]$. With $y = -\lambda/\eta$ and $\kappa = A_1/\delta$, then $q^*\eta + 1 = 0$ becomes $\kappa = e^{y-1}/y$, a curve in parameter space. Slope is $d\kappa/dy = (y-1)e^{y-1}/y^2 > 0$ because $y > 1$. Curvature satisfies $d^2\kappa/dy^2 \overset{sign}{=} (y-1)^2 + 1 > 0$. If $\kappa > e^{y-1}/y$, $y \in (1, \infty)$, then $q^*\eta + 1 < 0$ and lot scale decreases with an increase in η, while if $\kappa < e^{y-1}/y$ on $y \in (1, \infty)$ then $q^*\eta + 1 > 0$ and lot scale increases with an increase in η. If one varies p over $y \in (1, \infty)$ such that $\kappa y > 1$, then q^* is minimized when $\kappa = e^{y-1}/y$.

The situation is depicted in Figure 5. On the vertical axis at $y = 1$ then $\text{Lim}_{-\eta\uparrow\lambda} q^* = \infty$ so output can only decline as y increases away from $y = 1$. But $\text{Lim}_{-\eta\downarrow 0} q^* = \infty$ also, so that lot scale is only finite at intermediate health class values. On the vertical axis at $y = 1$, lot scale is unbounded to take advantage of increasing technical returns to scale when the magnitude of disease loss is acceptably low (i.e., κ high). At high y, lot scale

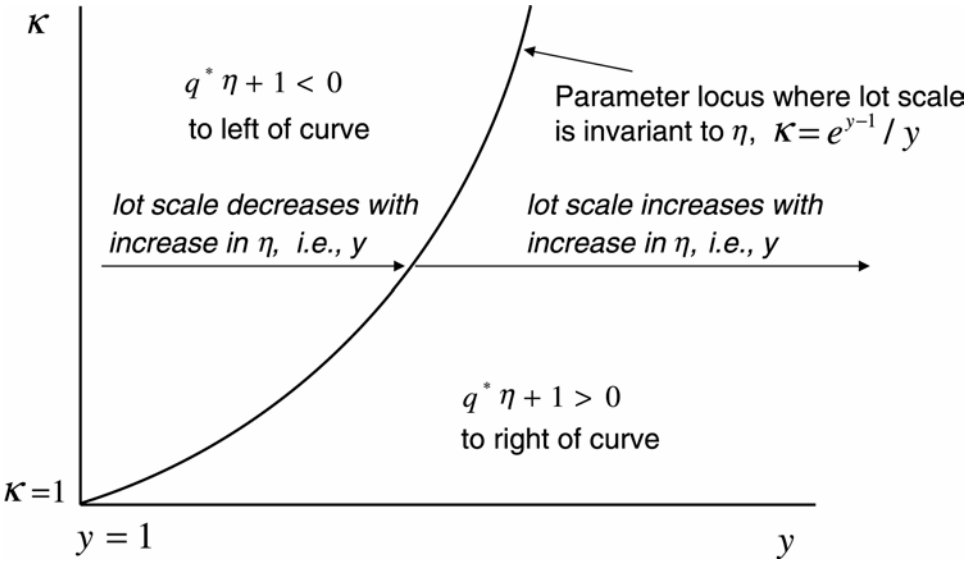

FIGURE 5. Lot scale response to health class for negative exponential unit technical cost

is unbounded because p is sufficiently large that disease loss is again acceptably low at large lot scale.

Feeder Animal Price

The feeder animal price-health class schedule is the Ricardian rent

$$w(p) = R - A(q^*) - \delta + \delta e^{q^* \eta}. \tag{16}$$

In the case of Example 1, an explicit form can be obtained by direct substitution. In general, the envelope theorem provides $dw(p)/dp = \delta q^* e^{(q^*-1)\eta}$ so that

$$\frac{d^2 w(p)}{dp^2} = \delta q^* (q^* - 1) e^{(q^*-2)\eta} + (1 + q^* \eta) \delta e^{(q^*-2)\eta} \frac{dq^*}{d\eta}. \tag{17}$$

Proposition 9 has shown that $(1 + q^* \eta) dq^* / d\eta \geq 0$ regardless of the sign of $1 + q^* \eta$. In addition, some work confirms that

$$d^2 w(p) / dp d\delta = [A''(q^*) q^* + A'(q^*)] e^{(q^*-1)\eta} / [A''(q^*) - \delta \eta^2 e^{q^* \eta}].$$

PROPOSITION 10. *Make Assumption 1. Then schedule* $w(p)$ *is convex in* p. *The schedule becomes more steeply increasing when the magnitude of disease loss increases (decreases) if* $-q^*A''(q^*)/A'(q^*) > (<) 1$.

From Example 1, $-q^*A''(q^*)/A'(q^*) = [\lambda/(\lambda+\eta)]\text{Ln}(-\lambda A_1/\delta\eta)$. With $\eta/\lambda = -0.1$, then $-q^*A''(q^*)/A'(q^*) = (0.9)^{-1}\text{Ln}(10A_1/\delta) > (<) 1$ whenever $A_1/\delta > (<)\ 0.1e^{0.9} = 0.246$. This last number, being larger than $-\eta/\lambda = 0.1$, is consistent with positive output so that $A_1/\delta \in (0.1, 0.246)$ is possible and so is the peculiar circumstance that $d^2w(p)/dpd\delta < 0$.

Information and Bayesian Conditioning

Quality of information will affect lot scale. To show how, let purchasers and sellers have symmetric but imperfect information on two animal types, as given in Table 4. If the animal has health class p_1 [with $\eta_1 = \text{Ln}(p_1)$] then the signal, say animal coat condition, is *h* with probability α. If the animal has class p_2, $p_2 < p_1$, then the signal is *h* with probability β. If α increases or β decreases, then we say that the information structure has become more informative. The true fraction of class p_1 animals in the pool of feeder animals is $\rho \in (0,1)$. The signal-conditioned probabilities that an animal has class p_1 are

$$\text{Prob}(p_1 \mid h) = r_h = \frac{\rho\alpha}{\rho\alpha + (1-\rho)\beta}; \qquad \text{Prob}(p_1 \mid l) = r_l = \frac{\rho(1-\alpha)}{\rho(1-\alpha) + (1-\rho)(1-\beta)}. \quad (18)$$

Following Proposition 7, the firm purchases only uniform-signal feeder animals. Write the $i \in \{h,l\}$ signal lot exposure as $qp_1^{qr_i}p_2^{q(1-r_i)} = qe^{q(r_i\eta_1 + (1-r_i)\eta_2)}$ so that signal-conditioned unit costs are

$$\begin{aligned} U(q;v_h) &= A(q) + \delta - \delta e^{qv_h}, \qquad v_h = \frac{\rho\alpha\eta_1 + (1-\rho)\beta\eta_2}{\rho\alpha + (1-\rho)\beta}; \\ U(q;v_l) &= A(q) + \delta - \delta e^{qv_l}, \qquad v_l = \frac{\rho(1-\alpha)\eta_1 + (1-\rho)(1-\beta)\eta_2}{\rho(1-\alpha) + (1-\rho)(1-\beta)}. \end{aligned} \quad (19)$$

TABLE 4. Probabilities in detecting feeder animal health class

Given that	Signal is *h*	Signal is *l*
animals have health class p_1	α	$1-\alpha$
animals have health class p_2	β	$1-\beta$

Note that $v_h > v_l$ if and only if $(\alpha-\beta)(\eta_1-\eta_2)>0$, that is, $\alpha>\beta$, and we make this assumption.[9]

With respective minimizing arguments in equation (19) as q_h^* and q_l^*, the envelope theorem yields

$$\begin{aligned}
\frac{dU(q_h^*;v_h)}{d\alpha} &= -\frac{\beta}{\alpha}\frac{dU(q_h^*;v_h)}{d\beta} = -\delta q_h^* e^{q_h^* v_h}\,\frac{\rho(1-\rho)\beta(\eta_1-\eta_2)}{[\rho\alpha+(1-\rho)\beta]^2} \le 0;\\
\frac{dU(q_l^*;v_l)}{d\alpha} &= -\frac{(1-\beta)}{(1-\alpha)}\frac{dU(q_l^*;v_l)}{d\beta} = -\delta q_l^* e^{q_l^* v_l}\,\frac{\rho(1-\rho)(1-\beta)(\eta_2-\eta_1)}{[\rho(1-\alpha)+(1-\rho)(1-\beta)]^2} \ge 0.
\end{aligned} \tag{20}$$

Better quality information of either sort reduces unit cost for high-signal lots because the fraction of poor-health animals declines. By contrast, better information increases unit cost for low-signal lots. This is because the fraction of good-health animals declines upon improved sorting in the marketplace. Referring to equation (16), the feeder price schedule difference $w(e^{v_h}) - w(e^{v_l})\,[\equiv U(q_l^*;v_l) - U(q_h^*;v_h)]$ should increase with better information.

The finding in equation (20) conveys nothing about the role of information on lot scale. Consider the cross-derivatives

$$\begin{aligned}
\frac{d^2U(q_h^*;v_h)}{d\alpha dq} &\overset{sign}{=} -(1+q_h^* v_h); \qquad \frac{d^2U(q_h^*;v_h)}{d\beta dq} \overset{sign}{=} 1+q_h^* v_h;\\
\frac{d^2U(q_l^*;v_l)}{d\alpha dq} &\overset{sign}{=} 1+q_l^* v_l; \qquad \frac{d^2U(q_l^*;v_l)}{d\beta dq} \overset{sign}{=} -(1+q_l^* v_l).
\end{aligned} \tag{21}$$

More information decreases (increases) the value of $dU(q_h^*;v_h)/dq$ if $1+q_h^* v_h \ge (\le)\, 0$ so that q_h^* will shift rightward (leftward) upon the advent of better (symmetric) information. More information increases (decreases) the value of $dU(q_l^*;v_l)/dq$ if $1+q_l^* v_l \ge (\le)\, 0$ so

that q_l^* will shift leftward (rightward) upon the advent of better information. Given the technology in Example 1, there always exists a v_h above which more information increases scale q_h^* and a v_l above which more information decreases scale q_l^*.

PROPOSITION 11. *As the signal becomes slightly more informative (i.e., a small increase in α or a small decrease in β) then unit cost in high-signal lots decreases while unit cost in low-signal lots increases. If $1+q_h^*v_h \geq (\leq)\, 0$, then an increase in signal informativeness increases (decreases) lot scale for high-signal lots. If $1+q_l^*v_l \leq (\geq)\, 0$, then an increase in signal informativeness increases (decreases) lot scale for low-signal lots.*

Given $0 \geq v_h > v_l$, Example 1 shows that it is not inconsistent for $1+q_h^*v_h$ and $1+q_l^*v_l$ to have different signs. Varying the value of y for a given κ value in Figure 5, the possibilities are $\text{sign}(1+q_h^*v_h, 1+q_l^*v_l) \in \{(-,-),(+,-),(+,+)\}$. If $\text{sign}(1+q_h^*v_h, 1+q_l^*v_l) = (+,+)$ then more information increases the gap between lot scales: apply Proposition 9, remembering that $v_h > v_l$ and $dv_h/d\alpha > 0 > dv_l/d\alpha$. High-signal lots become even larger while low-signal lots become even smaller. If $\text{sign}(1+q_h^*v_h, 1+q_l^*v_l) = (-,-)$ then lot scales also diverge, but now low-signal lots become larger and high-signal lots become smaller. The case of $(+,-)$ is ambiguous.

Dependent Disease Risks

To this point we have assumed that the health probabilities for animals in a given lot are independent. We relax this assumption by allowing for idiosyncratic and systemic components to an animal's health probabilities. The probability that one animal does not become infected, $p \in (0,1]$, is decomposed into a systemic component ν and an independent idiosyncratic component φ, $p = \nu\varphi$. The relative sizes of the multiplied probabilities may be viewed as being determined by the degree of similarity in backgrounds of the lot animals; if very similar, then ν is low and φ is high for a given value of p.

At a specified value of p, primary disease source risk is said to be fixed. For systemic component ν, one cannot attribute disease risk to any particular animal. The systemic components are perfectly correlated across the lot while the idiosyncratic components are independent. The probability that the lot does not become infected is then $\nu\varphi^q = p\varphi^{q-1} = pe^{(q-1)\vartheta}$, $\vartheta = \mathrm{Ln}(\varphi)$.[10] As φ increases then animals at a given health class p become more similar in the sense that most of the risk falls on common component ν.[11] The envelope theorem suggests that unit cost is decreasing in ϑ for a given value of p, $dU(q^*)/d\vartheta = -\delta p(q^* - 1)e^{(q^*-1)\vartheta} \leq 0$ under Assumption 1. In addition, equation (13) becomes $A'(q^*) = p\delta\vartheta e^{(q^*-1)\vartheta}$ so that

$$\frac{dq^*}{d\vartheta} = \frac{1 + (q^* - 1)\vartheta}{A''(q^*) - \vartheta A'(q^*)} p\delta e^{(q^*-1)\vartheta}. \tag{22}$$

The numerator and denominator are both assuredly positive under Assumption 1 whenever ϑ is sufficiently close to 0.

PROPOSITION 12. *Make Assumption 1 and fix health class p. Unit cost decreases with an increase in φ. For φ that are sufficiently large, optimal lot scale increases with an increase in φ.*

Proposition 12 suggests that one should procure feeder stock from as similar a background, be it in nature or nurture, as possible. Livestock auctions are unlikely to perform well in that regard. Closed-system feeder stock procurement from a single-feeder animal source would perform better.

Damage Control

To model expenditures on controlling loss, let control input level x per animal be used at unit cost t. The input reduces loss in that δ is a decreasing function, $\delta'(x) \leq 0$. Then equation (12) becomes

$$U(q, x) = A(q) + \delta(x) - \delta(x)e^{q\eta} + tx. \tag{23}$$

Second cross-derivatives are $d^2U(\cdot)/dqdx = -\delta'(x)\eta e^{q\eta} \leq 0$, $d^2U(\cdot)/dqd(-t) = 0$ and $d^2U(\cdot)/dxd(-t) = -1 \leq 0$.[12] The system is submodular in argument set $\{x, q, -t\}$. Standard deductions from submodularity theory support the intuition that an increase in the price of the control input decreases both input use per animal and lot scale.

Internal Production

Suppose one could produce internally such that there is zero probability of disease. Let $q^{**} = \arg\min A(q)$ and write $c_{fe} = A(q^{**})$ in the notation of the earlier model. Then the critical health class for external procurement is the solution to $R - c_{fe} = R - \delta - A(q^*) + \delta e^{q^*\eta}$. If the external procurement p value satisfies $p < \hat{p}$, where $\hat{p} = [(\delta + A(q^*) - c_{fe})/\delta]^{1/q^*}$, then one should produce internally rather than buy feeder animals.

Closing the Model

When there is only one health class and N firms then the market equilibrium level of N, labeled N^*, and the equilibrium level of p, labeled p^*, can be determined as follows. Write $Q = Nq$ as the market supply of feeder animals and $Q^* = N^*q^*$ as the equilibrium market supply. The inverse demand function for fat animals is then $R(Q) = R(Nq)$. Write market cost of supplying Q feeder animals at health class p as $H(Q, p)$ with $dH(\cdot)/dQ \geq 0$ and $dH(\cdot)/dp \geq 0$. Then, from equation (16), $w(p^*) = R(Q^*) - A(q^*) - \delta + \delta(p^*)^{q^*} = dH(Q^*, p^*)/dQ$ while $Q^*dw(p^*)/dp = dH(Q^*, p^*)/dp$. These, together with equation (13), solve for p^*, Q^*, q^*, and so N^*. It is unlikely, however, that any system solution is unique. The likely convexity of $w(p)$, as demonstrated in Proposition 10, is one reason for this.

Openness and Communicable Animal Diseases

To illustrate the role of openness in disease management, we consider efforts to control three categories of communicable animal disease. These are babesiasis (Texas fever) in bovines, tuberculosis (TB) in bovines, and respiratory diseases in swine. The first is

tick-borne, most problematic in warmer climates, and of little health risk to humans. TB is bacterial, more difficult to detect when trading animals, and presents significant human health risks. The most troublesome swine respiratory diseases are viral diseases, which are often readily transmitted by air and do not pose significant human health risks. Each has somewhat distinct features regarding transmission and economic impacts.

Babesiasis

The ailment causes fever, jaundice, a decline in milk production, and abortion. It was a very serious threat to beef productivity from 1850 to 1890 in the U.S. Southwest and Great Plains. At that time, Texas grown cattle were herded north to railheads for slaughter in the Midwest as well as for the feeder trade in the Midwest and Great Plains. Homesteaders in Kansas, Missouri, Nebraska, and elsewhere suffered large losses through trampled crops, babesiasis, and rowdy cowboys. Similar to legislation elsewhere, in 1859 the Kansas Territorial Legislature prohibited the droving of sick cattle into the state and a quarantine law (no droving of any cattle into the state between April and November) followed in 1861. The legislation was ineffective and largely ignored.

In 1867 the Kansas state legislature imposed an east-to-west quarantine line within the state (Hutson 1994). The line shifted several times before the trade petered out. The risk of babesiasis was considered by progressive ranchers in the state to be a major deterrent to investing in herd quality enhancement. Even away from trails, Texas strays mingled with herds on open range and through broken fences. It was clear to most in the Kansas cattle industry by the late 1880s that the need to protect their own herd far outweighed gains from the Texas trade, and more stringent statewide quarantine laws were enforced. Similar events occurred in other states and effectively ended U.S. cattle trail droving.

The disease, though much diminished, remained a problem in Kansas until about 1930. The spatial externalities caused by babesiasis were among the major motives for setting up the USDA's Bureau of Animal Industry in 1884. The disease also provided the Bureau with one of its first successes when Bureau scientists proved the disease was tick-borne. In 1892, the federal government imposed a national quarantine line above which any southern cattle moved between January 15 and November 15 had to be by rail or boat and for immediate slaughter.

Bovine Tuberculosis

Unlike babesiasis, bovine TB is communicable to humans, and that fact was a determining issue in prioritizing the disease for eradication. Among cattle, it can be spread through bacterial contamination of water, bedding, feed, and shared air. Productivity losses typically amount to a 10 to 25 percent reduction in output from infected animals. At the herd level, the disease generally spreads through stock purchases, although herd contiguity (i.e., density of production), a common water supply, and wildlife vectors are also factors. Commencing in 1917, large resources were devoted by the U.S. government to eradicate the disease in bovines. Measures included a testing program, quarantine for animals entering the country, and movement controls on animals inside the United States, as well as a tracking system for moved animals (Smith 1958; Myers and Steele 1969). Test positive animals were destroyed, and this number peaked at 377,000 in 1935.

The early focus of the campaign was on breeding herds, in part because of the mobility of high-quality progeny. Breeding herds declared as TB-free were designated as accredited. Accreditation was later extended to counties, and a market premium for live animals from these herds and counties provided private incentives beyond direct productivity effects to secure accreditation. State-level legislation required out-of-state animals to pass tests before they were allowed entry. In-state testing regimes were put in place, reactors were slaughtered with compensation, and herds with reactors were quarantined pending subsequent tests. Opposition among some farmers was intense, indicating the gap between private and public benefits. The program is viewed as a great success and all counties in the United States had infection rates below 0.5 percent by 1941. Even ignoring the reduction of bovine TB infections in the human population, it was held that the program benefits far exceeded program costs in securing agricultural productivity.[13]

It was recognized early in federal animal disease eradication endeavors that system closure through movement control was critical. The bovine TB program illustrates two related central themes in the approach generally taken: imposing movement controls and designing robust information systems. Movement controls and information systems are to some extent substitutes. Broad movement controls may be necessary if the extent of a disease is known with a low degree of certainty. But the resulting cost would be high when there are strong private incentives to move animals. A good information system

may allow for targeted movement controls at low cost. The most important feature of an information system is the quality of testing procedures, and a TB test (though imperfect) for use on farms became available in 1892 (Myers and Steele 1969).

Swine Respiratory Diseases

Many swine diseases are contagious but transferable only by direct animal contact, as in our intra-farm model. The movement of animals between herds is an important condition for the spread of these diseases. But gains from trade are forgone when limits are placed on animal movements, while losses from restricting breeding selection are also important. Many breeding farms have moved to closed systems, while fattening farms remain relatively open (Plonait 2001). The introduction of new genetic material in breeding farms occurs by means of artificial insemination, embryo-transfers, and pre-natal harvest of piglets to ensure specified pathogen-free animals.

Besides the transmission of infection from other the animals, infections may occur through manure spread on land, wild animals, feed, and human contact. Aerial transmission is important for certain viral infections. Several costly respiratory diseases in swine illustrate the issues we have discussed; See Table 5.[14] Enzootic pneumonia (EP) can take a subclinical form up to an acute form with significant economic losses to the farm. It is often caused by the introduction of new animals in existing herds. EP can also be transferred at a distance in aerosol. This mode of transmission is highly dependent on farm and regional stocking density. Actinobacillus-pleuropneumonia, first discovered in 1964, is similar to EP. Infection mostly occurs by buying new animals but also through passive infection by clothing and airborne transmission. Its chronic form is often present in fattening farms and closed breeder-fattening systems.

Swine influenza has been endemic in the United States since it was first observed in 1918. Its later emergence in East Asia, South America, and Europe was likely through imported breeding pigs. It is introduced by the purchase of latent infected animals, but outbreaks usually occur only under adverse weather conditions. Porcine reproductive and respiratory syndrome is viral and was first reported in 1987 in the United States. The first European outbreak occurred in 1990 in Germany, and from there it spread rapidly throughout Europe (Nodelijk et al. 2003).

TABLE 5. Some important respiratory diseases in swine

Disease	Incubation Time	Effects	Morbidity	Mortality	Age Classes
Enzootic pneumonia – acute	Up to 3 weeks	Some coughing and weakened general condition	60%	Up to 10%	All
Enzootic pneumonia – chronic	Not applicable	Few observable impacts	80%	Chronic	3 weeks to 6 months
Pleuropneunomia – acute	2-5 days	Strong impact on general condition with severe respiratory symptoms	80%	Up to 50%	All
Pleuropneunomia – chronic	Not applicable	Some symptoms, including coughing	30%	Chronic	Weaner and feeder pigs
Swine Influenza	1-2 days	Strong impact on general condition with severe respiratory symptoms	Up to 100%	2-3%	All but piglets
Pseudorabies	Up to 20 days	Strong impact on general condition. Apathy, some coughing and respiratory problems	Up to 100%	Up to 10%	Weaner pigs
Porcine reproductive and respiratory syndrome	Up to 10 days	Strong impact on general condition, including abortion and severe respiratory symptoms	Up to 100%	Up to 10%	Fattening and breeding pigs

These diseases are most problematic in areas of dense swine populations. Veterinarians and animal scientists advocate that commercial production should occur in, as far as is practicable, closed systems. For small swine production units it is often difficult to implement a completely closed system because genetic upgrading requires the introduction of purchased stock. An industrial system, on the other hand, generally involves closer coordination to protect against disease. However, industrial systems need to recognize their disease vulnerability due to their larger scale. Many large-scale growers have adopted all-in/all-out systems, together with routine pharmaceutical prevention programs.[15]

Concluding Comments

In this article, we have identified some ways in which the extent of openness in an animal production system can affect system performance. Closed systems that forgo potential gains from trade can be more efficient when the risk of losses from communicable disease is significant. When communicable disease gives rise to inter-farm externalities, then temporary (at the least) public action to close the system may be necessary to improve industry performance. Indeed, to the extent that regional advantages in feed costs encourage feeder trade, our first model suggests that cheap feed may reduce competitiveness for the system as a whole. If advantage is to be taken of cheap feed, then the relatively closed contractual approach adopted among U.S. hogs may perform better than the more open feeder cattle trade approach even when price discovery is impeded.

Our model of intra-farm effects showed how communicable disease considerations impede exploitation of technical economies of scale. The relationship between health class and scale may not be simple. However, the reasonableness of the negative exponential technology suggests that when health class is sufficiently large then scale should increase with improved health class. It should not be surprising that industrial approaches to animal production, with high fixed cost capital requirements and scale economies, place emphasis on procuring animals of consistently superior health. Optimal lot scale may not otherwise be sufficiently large to clear a profit.

A common thread running through the analysis is the relevance of information in improving performance. Openness and information can hardly be considered separately. If traditional open approaches to animal production are to remain competitive then the

genuine information problems attending open systems must be solved. If a production system is to exploit the potential advantages of trade then comprehensive animal information infrastructure will be necessary. Indeed, plural information sets may be required. Governments need information to manage animal and human health externalities. Producers and processors have additional information needs that are unlikely to be met by any government endeavors. Record keeping can be burdensome, so any government system should be capable of extension to accommodate private sector needs.

Endnotes

1. Sumner (2003) explains in depth the public good aspects of agricultural diseases.
2. A notable exception is Kuchler and Hamm (2000), in which the issue is a bounty on reporting scrapie infections.
3. See Biggs (1985) on practical motivation for these monotonicity assumptions. Notice that our model has trivial spatial structure in that all production in the market is equally exposed to any given disease outbreak. Even so, the classical Reed-Frost algorithm for disease contagion is not spatial either (Thrusfield 1995). While more spatial structure would be critical in any epidemiological model seeking prediction, it is less relevant to a qualitative economic analysis of contagion. A significant deficiency in our model is that system openness is attached to a production function. But if a disease can be eradicated then an open system can have low disease incidence. Even then, the more open the system, the greater the risk of a subsequent outbreak.
4. This means that $M[I]$ is not flat in metric space neighborhoods. Our analysis could include such situations, but no additional insights would result.
5. They are unstable to a perturbance in one direction, i.e., either unstable when I increases or unstable when I decreases.
6. While we use calculus to optimize, it is recognized that lot scale takes integer values. Exponential transformation $p^q = e^{q\eta}$ will facilitate analysis later in the section.
7. Condition $q^*\eta \geq -1$ (i.e., $p^{q^*} \geq e^{-1}$) asserts the lot is diseased with probability of at least 0.368.
8. In this case, submodular means that $d^2U(q)/dqdp \leq 0$. On these methods in comparative statics, see Topkis 1995 or Milgrom and Shannon 1994.
9. If $\alpha < \beta$, then re-label α as β and β as α. If $\alpha = \beta$, then the signal bears no information.

10. If the lot consists of two animals, then there is probability $1-\nu$ that both succumb to a common cause. For each animal also, there is the probability $1-\varphi$ that the particular animal succumbs to an idiosyncratic cause and then contaminates the other. The three events are independent and the probability that none occur is $\nu\varphi^2 = p\varphi$.
11. Caution is warranted when interpreting systemic risk in this model. As the value of ν decreases, animals become more similar in risk exposure but lot systemic risk decreases.
12. The expression $d^2U(\cdot)/dqdx \leq 0$ suggests that farm-level conditions under which lot scale is high should also tend to be conditions under which x is high. A survey by APHIS (2000) found that large cattle feedlots ($\geq$ 8,000 head) spent an average of $16.26 per sick animal on (often communicable) respiratory diseases, compared with an average of $11.09 per sick animal in smaller feedlots ($<$ 8,000 head but $\geq$ 1,000 head). For digestive problems (excluding non-eaters) costs per sick animal were about the same: $6.27 on large lots and $6.14 on smaller lots. For problems of the central nervous system problems, large lots spent $11.29 per sick animal while smaller lots spent $11.61 per sick animal.
13. See p. 134 of Myers and Steele 1969.
14. The descriptions are largely from information in Zimmermann and Plonait 2001.
15. All-in/all-out management means that all animals are removed from a lot prior to restocking. The facilities are then cleaned and disinfected before restocking occurs.

References

APHIS (Animal and Plan Health Inspection Service). 2000. *Part III: Health Management and Biosecurity in U.S. Feedlots, 1999*. Washington, DC: U.S. Department of Agriculture, December.

———. 2004. *A Case of Bovine Spongiform Encephalopathy (BSE) in the United States*. http://www.aphis.usda.gov/lpa/issues/bse/BSE_tr_ban%20_ltr_enc_1.pdf (accessed July 2004) Washington, DC: USDA.

Bennett, R. 2003. "The 'Direct Costs' of Livestock Disease: The Development of a System of Models for the Analysis of 30 Endemic Livestock Diseases in Great Britain." *Journal of Agricultural Economics* 54(1): 55–71.

Biggs, P.M. 1985. "Infectious Animal Disease and its Control." *Philosophical Transactions of the Royal Society of London, Series B* 310(1144): 259–74.

Chi, J., A. Weersink, J.A. VanLeeuwen, and G.P. Keefe. 2002. "The Economics of Controlling Infectious Diseases on Dairy Farms." *Canadian Journal of Agricultural Economics* 50(November): 237–56.

Dijkhuizen, A.A. 1990. "The Profitability of Herd Health Control in Cattle." *Landbouwkundig Tijdschrift* 102: 12–16.

FAO (Food and Agriculture Organization). 1962. "The Economic Losses Caused by Animal Disease." In *Animal Health Yearbook*. FAO, United Nations, Rome.

———. 2004. The Global Livestock Production and Health Atlas (GLiPHA). Software, downloadable at http://www.fao.org/ag/againfo/resources/en/glipha/default.html (accessed July 2004).

Geoffard, P-Y., and T. Philipson. 1996. "Rational Epidemics and their Public Control." *International Economic Review* 37(August): 603–24.

———. 1997. "Disease Eradication: Private versus Public Vaccination." *American Economic Review* 87(March): 222–30.

Horst, H.S., C.J. de Vos, F.H.M. Tomassen, and J. Stelwagen. 1999. "The Economics of Evaluation of Control and Eradication of Epidemic Livestock Disease." *Scientific and Technical Review of the Office International des Épizooties* 18(2): 367–79.

Hutson, C.K. 1994. "Texas Fever in Kansas." *Agricultural History* 68(1): 74–104.

Kremer, M. 1993. "The O-Ring Theory of Economic Development." *Quarterly Journal of Economics* 108(August): 551–75.

———. 1996. "Integrating Behavioral Choice into Epidemiological Models of AIDS." *Quarterly Journal of Economics* 111(May): 549–73.

Kuchler, F., and S. Hamm. 2000. "Animal Disease Incidence and Indemnity Eradication Programs." *Agricultural Economics* 22(April): 299–308.

Mangen, M-J.J., M. Nielsen, and A.M. Burrell. 2002. "Simulated Effect of Pig-Population Density on Epidemic Size and Choice of Control Strategy for Classical Swine Fever Epidemics in The Netherlands." *Preventive Veterinary Medicine* 56(2002): 141–63.

Mathews, K., and J. Buzby. 2001. "Dissecting the Challenges of Mad Cow and Foot-and-Mouth Disease." *Agricultural Outlook,* August, pp. 4–6.

Matthews, L., D.T. Haydon, D.J. Shaw, M.E. Chase-Topping, M.J. Keeling, and M.E.J. Woolhouse. 2003. "Neighbourhood Control Policies and the Spread of Infectious Diseases." *Proceedings of the Royal Society of London, Series B* (270): 1659–66.

McCauley, E.H., J.C. New, N.A. Aulaqi, W.B. Sundquist, and W.M. Miller. 1979. "A Study of the Potential Economic Impact of Foot-and-Mouth Disease in the United States." Department of Agricultural Economics TB-1597, University of Minnesota, May.

McInerney, J.P. 1996. "Old Economics for New Problems–Livestock Disease: Presidential Address." *Journal of Agricultural Economics* 47(3): 295–314.

Milgrom, P., and C. Shannon. 1994. "Monotone Comparative Statics." *Econometrica* 62(January): 157–80.

Mintiens, K., H. Laevens, J. Dewulf, F. Boelaert, D. Verloo, and F. Koenen. 2003. "Risk Analysis of the Spread of Classical Swine Fever Virus through 'Neighbourhood Infections' for Different Regions in Belgium." *Preventive Veterinary Medicine* 60: 27–36.

Myers, J.A., and J.H. Steele. 1969. *Bovine Tuberculosis Control in Man and Animals*. St. Louis, MO: Warren H. Green.

Nodelijk, G., M. Nielen, M.C.M. De Jong, J.H.M. Verheijden. 2003. "A Review of Porcine Reproductive and Respiratory Syndrome Virus in Dutch Breeding Herds: Population Dynamics and Clinical Relevance." *Preventative Veterinary Medicine* 60: 37–52.

Otte, M.J., R. Nugent, and A. McLeod. 2004. "Transboundary Animal Diseases: Assessment of Socio-Economic Impacts and Institutional Reform." Food and Agriculture Organization, Livestock Information and Policy Branch, Rome, February.

Perry, B.D., W. Kalpravidh, P.G. Coleman, H.S. Horst, J.J. McDermott, T.F. Randolph, and L.J. Gleeson. 1999. "The Economic Impact of Foot and Mouth Disease and its Control in South-East Asia: A Preliminary Assessment with Special Reference to Thailand." *Scientific and Technical Review of the Office International des Épizooties* 18(2): 478–97.

Plonait, H. 2001. "Infektionsschutz, Sanierung und Planmäßige Bestandsbehandlung." In *Lehrbuch der Schweinekranheiten, 3rd ed*. Edited by K.H. Waldmann, M. Wendt, H. Polait, and K. Bickhardt, pp. 549-574. Berlin: Parey Publishers.

Schoenbaum, M.A., and W.T. Disney. 2003. "Modeling Alternative Mitigation Strategies for a Hypothetical Outbreak of Foot-and-Mouth Disease in the United States." *Preventive Veterinary Medicine* 58: 25–52.

Shields, D.A., and K.H. Mathews, Jr. 2003. "Interstate Livestock Movements." U.S. Department of Agriculture, Economic Research Service, LDP-M-108-01, Washington, DC, June.

Skaggs, R., R. Acuña, L.A. Torell, and L. Southard. 2004. "Live Cattle Exports from Mexico into the United States: Where Do the Cattle Come From and Where Do They Go?" *Choices,* Vol. 19, First Quarter, pp. 25–30.

Smith, H.R. 1958. *The Conquest of Bovine Tuberculosis in the United States*. Somerset, MI: Printed by author.

Sumner, D.A. 2003. "Economics of Policy for Exotic Pests and Diseases: Principles and Issues." In *Exotic Pests and Diseases: Biology and Economics for Biosecurity*. Edited by D.A. Sumner, pp. 9–18. Ames, IA: Iowa State University Press.

Thrusfield, M.V. 1995. *Veterinary Epidemiology, 2nd ed.* Oxford, UK: Blackwell.

Topkis, D.M. 1995. "Comparative Statics of the Firm." *Journal of Economic Theory* 67(December): 370–401.

World Health Organization. 2004. "Avian Influenza Fact Sheet." January 15. http://www.who.int/mediacentre/factsheets/avian_influenza/en/ (accessed July 2004).

Zimmerman, W., and H. Plonait. 2001. "Erkrankungen des Atmungsapparates." In *Lehrbuch der Schweinekranheiten, 3rd ed*. Edited by K.H. Waldmann, M. Wendt, H. Polait, and K. Bickhardt, pp. 111-150. Berlin: Parey Publishers.