MANAGING BIOLOGICAL AND LINKED POLLUTANTS

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ABSTRACT

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This dissertation contains three chapters, each of which examine the management of environmental pollutants. The first chapter considers livestock producers' incentives for self-protection against infectious disease, which can be viewed as a type of "biological pollutant." Spillovers in disease protection create different types of strategic interactions. Under certain conditions, multiple Nash equilibria may arise with the possibility of coordination failure involving excessively low self-protection, in which case individuals' expectations of others' efforts determine which outcome arises. In prior studies, assumed technical relations between self-protection and infection probabilities drive the strategic interactions. We demonstrate that strategic relations can be endogenously determined and depend on the relative endogeneity of risk (RER), defined here as the degree to which individuals can take control of their own risks in a strategic setting. The potential for coordination failure may arise when RER is sufficiently small, whereas larger levels of RER may eliminate this possibility to ensure larger levels of self-protection. We find that imposing a behaviorally-dependent indemnity may increase RER to eliminate the possibility of coordination failure. We apply our analysis to the problem of livestock disease and illustrate the theory using a numerical example of the 2001 United Kingdom foot-and-mouth disease epidemic.

The second chapter examines the problem of biological pollution from live animal movements. Prior literature asserts trade-related biological pollution externalities arise from the movement of contaminated goods. However, this literature ignores (i) importers' ability to

reduce disease spillovers via private risk management choices and (ii) the potential for strategic interactions to arise when an importer's risk management measures simultaneously protect himself and others. This paper explores the design of efficient disease prevention policies when importers can mitigate disease risks to others. We demonstrate that the biological pollution externality extends beyond trade-related activities—in contrast to prior work—and derive efficient policy incentives to internalize the externality. We also find spillovers between importers may be characterized by strategic complementarities, leading to multiple Nash-equilibrium levels of risk-mitigating activities. Additional command-and-control policies may be needed alongside of incentives to achieve efficiency.

The final chapter analyzes the management of linked environmental pollutants. Advances in the understanding of pollutant generation, transport, and fate has increased researchers' knowledge of the linkages between pollutants. A striking example involves reactive nitrogen (Nr), a family of pollutants which, once emitted into the environment, passes through multiple environmental media (e.g., air and water), causing economic damages in each. Traditional market-based policies manage different Nr species separately, and policy parameters, including pollutant caps, are often set independently (exogenously) of other market parameters. This paper examines the efficiency gains from an integrated market that allows trading across Nr species, both analytically and also using a numerical model of pollutant trading in the Susquehanna River Basin. When permit caps are exogenous, it is demonstrated that efficiency can be increased by integrating otherwise distinct markets for greenhouse gas and nutrient water pollutants. Specifically, integration allows regulated polluters to trade across pollutants at an optimally-chosen interpollutant exchange rate. Numerical results indicate the economic gains from integrating markets may be more than $200 million, depending on the levels of the permit caps.

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KEY TO ABBREVIATIONS

CO_2	Carbon dioxide
FMD	Foot-and-mouth disease
FOC	First-order condition
GHG	Greenhouse gas
LHS	Left-hand side
N_2O	Nitrous oxide
NO_3	Nitrate
Nr	Reactive nitrogen
RER	Relative endogeneity of risk
RHS	Right-hand side
SNE	Symmetric Nash equilibrium
SRB	Susquehanna River Basin

CHAPTER 1 SELF-PROTECTION, STRATEGIC INTERACTIONS, AND THE RELATIVE ENDOGENEITY OF DISEASE RISKS[1]

1.1 Introduction

A key behavior influencing the introduction and spread of infectious disease is the degree to which individuals self-protect from disease risks (e.g., via immunization or biosecurity effort). Strategic interactions may arise in making self-protection choices, as individuals' disease risks can depend on the self-protection behavior of others. Indeed, self-protection is an example of a private behavior that generates positive spillovers affecting the supply of a public good, i.e., infectious disease prevention (Buchanan and Kafoglis 1963; Olson and Zeckhauser 1970).

Different types of strategic relations may arise. Of particular consequence is when self-protection takes the form of a strategic complement, whereby an individual's marginal incentives for self-protection increase with the self-protection of others. Relatively strong complementarities in the neighborhood of one equilibrium can lead to multiple Nash equilibria, with the potential for coordination failure in which all individuals adopt low levels of self-protection (Echenique 2004; Vives 2011). Self-protection could also take the form of a strategic substitute, whereby an individual's marginal incentives for self-protection decline with the self-protection of others. Coordination failure is not a concern for this type of relationship (Hefti 2011).

In prior studies of disease and strategic interactions, the type of strategic interaction is driven by assumed technical relations defining how one's infection probability is jointly affected

[1] A version of this chapter was published in Reeling, C.J. and R.D. Horan. 2015. "Self-Protection, Strategic Interactions, and the Relative Endogeneity of Diseaes Risks." *American Journal of Agricultural Economics* 97(2): 422–468.

by the self-protection efforts of oneself and others. These technical relations generally depend on the type of transmission. For instance, Barrett (2004) and Hennessy (2007b) find that self-protection to reduce spread of an existing livestock disease between herds is a strategic substitute, while Hennessy (2008) finds that self-protection to prevent the introduction of a new livestock disease into a region is a strategic complement. Chen (2012) finds that social distancing is a strategic complement (substitute) when the rate of contact between individuals is decreasing (increasing) in the number of individuals. In all of this work, and in other work where strategic interactions exist but are not explicitly investigated (e.g., Fenichel et al. 2011), individuals' strategic behaviors are made in response to the spillovers created by particular technical relations that are taken as given rather than endogenously determined. This is true even in existing dynamic models in which strategic behaviors are both affected by and affect future risks (e.g., Fenichel et al. 2011; Chen 2012). Behavior in those models (i.e., social distancing or, equivalently, public avoidance, which could be viewed as a form of biosecurity) does not affect the nature of the technical relations or related spillovers that determine whether behaviors are strategic substitutes or complements, although behaviors can dynamically affect the magnitude of the spillovers (via effects on future states) to alter the strength of the strategic relations.

We examine a more general case in which disease can be transmitted over two modes, introduction and spread, where the predominant technical relations (stemming from the type of risk exposure, introduction versus spread) and the associated spillover effects endogenously depend on self-protection efforts. Therefore, in contrast to prior work, the strategic relations are also endogenously determined. We show these relations depend on a new concept we refer to as the relative endogeneity of risk (RER), defined here as the degree to which an individual can take control of his or her own risks within a strategic setting where others' actions also influence

one's risks.

When individuals' self-protection gives them sufficient control over their own risks (so that an individual is not very reliant on spillovers from others' self-protection), then self-protection is a weak strategic complement or strategic substitute and there will be at most one Nash equilibrium and no risk of coordination failure. Alternatively, when individuals' self-protection does not yield sufficient control over their own risks (so that an individual is more reliant on spillovers from others' self-protection), then self-protection is a strategic complement and coordination failure may become a concern.

The policy implications arising from strategic interactions have largely gone unaddressed, although Chen (2012) does consider the potential consequences of coordination failure. We find that properly designed disease prevention and control policies can increase RER to remove the possibility of coordination failure while also increasing equilibrium levels of self-protection. In particular, we show such results can arise from indemnity payments that increase with the individual's level of self-protection. The optimal design of these indemnities when there is a risk of coordination failure differs from traditional notions of optimal incentive design in strategic settings, however. Traditional approaches involve setting instrument parameters to ensure the first-best outcome is a Nash equilibrium (e.g., Acocella, Di Bartolomeo and Hughes-Hallett 2013). This may be insufficient to guarantee the first-best outcome when there is a potential for coordination failure. We present an approach in which the first-best level of self-protection is a globally stable Nash equilibrium.

In the sections that follow, we derive an analytical model of self-protection against disease risks and investigate how RER influences the strategic interactions among at-risk individuals. We then demonstrate how disease prevention policy can influence RER and, thus,

strategic relationships. A numerical model of the 2001 UK foot-and-mouth disease (FMD) outbreak demonstrates the theory, and is then used to explore optimal policy design.

1.2 A Model of Infection Risks

We develop a simple model of endogenous infection risks to illustrate the incentives for self-protection. The model is framed in terms of livestock producers whose herds are at risk from the introduction and spread of either an existing or emerging infectious disease. However, the essential features of the model—self-protection from infectious contacts along introduction and spread pathways, with strategic interactions arising along the spread pathway—are also relevant to disease problems among humans and human-managed ecosystems. For simplicity, we adopt a one-period model of these processes. This simple framework captures the same sorts of risks and choices that would arise in more complex dynamic models, while allowing us to focus on strategic elements of the problem.

Suppose a region contains a number of homogeneous livestock producers. Each producer's herd faces disease risks along two pathways: introduction and spread. Consider the risks faced by a particular producer whose herd takes on the binary status of being either infected or uninfected. The probability that this producer's herd becomes infected from sources outside the region (i.e., pathogen introduction) is $P^I(\rho)$.[2] We assume $P^I_\rho(\rho_i) < 0$ and $P^I_{\rho\rho}(\rho) > 0$, where $\rho \in [0,1]$ is the producer's self-protection or biosecurity effort.[3] The signs on the partial derivatives

[2] Disease ecology models often model transmission either with the number of animals as being the primary unit of analysis (e.g., Heesterbeek and Roberts 1995), or with the number of "patches" or "sub-populations" being the primary unit (Hess 1996; Gog, Woodroffe and Swinton 2002; McCallum and Dobson 2002). The latter case, often referred to as a metapopulation model, implicitly models population sizes within a patch as being fixed. Our approach, in which the farm is the primary unit of analysis, follows this latter approach.

[3] All probabilities in the model are defined as conditional probabilities, i.e., conditional on the biosecurity decisions of the producer and his neighbors. Subscripts represent partial derivatives.

indicate that disease protection, defined as $[1 - P^I(\rho)]$, is increasing in ρ at a decreasing rate. Introduction could occur via the importation of animals into the region.

The producer's herd is at risk of infection from direct or indirect contact from infected animals of neighboring producers (i.e., pathogen spread). Herd contact may occur via people or wildlife acting as disease transmission vectors, via direct contacts between animals in communal grazing areas or livestock exhibitions, or even via airborne droplets of water containing infectious agents. The probability that a producer's herd becomes infected by spread depends not only on his own self-protection ρ, but also the self-protection of his neighbors. Let $\sigma \in [0, 1]$ denote the biosecurity effort of the other producers in the region. We treat σ as a scalar for simplicity since all neighbors are homogeneous and make identical choices.[4] Accordingly, σ can be thought of as either the effort level of another producer in a duopolistic setting, or as the average effort of other producers in the region. This follows the convention used by Vives (2005) for multiple, homogeneous firms. The probability that the representative producer becomes infected via spread, conditional on being uninfected, is written $P^S(\rho, \sigma)$, with $P_i^S < 0$ and $P_{ii}^S > 0$ for $i = \rho, \sigma$. The function $P^S(\rho, \sigma)$ is quite general, and so σ could reduce spread risks both directly (e.g., via biosecurity to prevent transmission across farms) and indirectly (e.g., by reducing the likelihood of other producers becoming infected).

The probability that the producer becomes infected via either the introduction or spread pathway, assuming independence of the underlying random processes governing infection along

[4] Heterogeneity in economic and/or technical relations, including heterogeneous disease risks over time and space, may arise in some settings to generate heterogeneous behaviors (Rahmandad and Sterman 2008). In analyzing strategic economic behavior among many firms in non-disease settings, Vives (2005) demonstrates that the qualitative nature of strategic relationships and associated outcomes are preserved when heterogeneities are introduced without altering the underlying economic relations qualitatively (e.g., the heterogeneities do not affect whether a producer's marginal incentives for an action are increasing or decreasing in neighbors' actions). Accordingly, while producer heterogeneities will certainly have quantitative impacts on economic and epidemiological outcomes, there may be only a limited impact on qualitative results.

these pathways, can be written[5]

$$P(\rho,\sigma) = P^I(\rho) + [1 - P^I(\rho)]P^S(\rho,\sigma). \tag{1.1}$$

1.3 Economic Model

Suppose the producer chooses his biosecurity effort to maximize expected profit, denoted $E\{\pi\}$. The producer earns profits of $\pi^H = R^H - c(\rho)$ when his herd is in the susceptible (i.e., non-infected or healthy) state and $\pi^{NH} = R^{NH} - c(\rho)$ in the infected (or not healthy) state, where $R^H > R^{NH}$. Assume $c(\rho)$ is increasing and convex in ρ. The producer's problem is

$$\begin{aligned} \max_{\rho} \; E\{\pi\} &= [1 - P(\rho,\sigma)]R^H + P(\rho,\sigma)R^{NH} - c(\rho) \\ &= R^H - \Lambda P(\rho,\sigma) - c(\rho) \end{aligned} \tag{1.2}$$

where $\Lambda = (R^H - R^{NH}) > 0$ represents losses or damages due to infection. Problem (1.2) is solved as a Nash-Cournot game: the producer chooses his or her own biosecurity ρ while taking others' effort σ as given. Other producers behave analogously.

Assuming an interior solution, the first-order condition of problem (1.2) is $E\{\pi_\rho\} = 0$, which implies

$$-\Lambda P_\rho(\rho,\sigma) = c_\rho(\rho). \tag{1.3}$$

The left-hand side (LHS) represents the marginal benefits of biosecurity, which is the economic loss experienced in the infected state multiplied by the marginal impact of biosecurity on the probability of infection. At an interior optimum, this is equal to the marginal cost of biosecurity, denoted by the right-hand side (RHS) term. Note that a corner solution involving $\rho = 0$ is likely to be uncommon because the marginal cost of taking some defensive action is likely to be quite

[5] The probabilities of spread and introduction may be positively correlated in some settings. If so, then assuming independence will bias the modeled probability of infection downward relative to the true level.

low relative to the marginal benefit. However, it may be possible to have a corner solution involving $\rho = 1$, which we define as closing off one's farm from all outside contact, and we explore this possibility below.

Additional insight arises from examining the marginal impact of biosecurity on the probability of infection,

$$P_\rho(\rho,\sigma) = P_\rho^I(\rho) - P_\rho^I(\rho)P^S(\rho,\sigma) + [1 - P^I(\rho)]P_\rho^S(\rho,\sigma). \tag{1.4}$$

Increasing one's biosecurity effort has three effects on the probability of infection, as indicated by the three right hand side (RHS) terms in equation (1.4). The first RHS term in (1.4), which is negative, represents the marginal effect of ρ on reducing introduction risks, holding spread risks constant. The second term represents the effect of ρ on reallocating risk from disease introduction to disease spread. Mathematically, the reduction in $P^I(\rho)$ is accompanied by an increase in $1 - P^I(\rho)$, thereby placing more weight on the spread term $P^S(\cdot)$ in equation (1.1). Intuitively, the risk of infection via spread becomes relatively more important as introduction risks are reduced and spread becomes the more likely pathway of infection. Hence, this term is positive so as to partially offset the first RHS term. The final RHS term in (1.4), which is negative, reflects the effect of ρ on the risk of spread, holding introduction risks constant.

1.3.1 Incentives Arising from Strategic Interactions

Condition (1.3) indicates that a producer's marginal incentives for biosecurity depend on neighbors' biosecurity, σ. The implicit solution to (1.3) is given by the best-response function $\rho(\sigma)$, with $\rho_\sigma = E\{\pi_{\rho\sigma}\}/(-E\{\pi_{\rho\rho}\})$.[6] Note that $E\{\pi_{\rho\rho}\} < 0$ if $\rho(\sigma)$ maximizes expected profits.

[6] Define $F(\rho,\sigma) = \partial E\{\pi\}/\partial\rho$ so that the producer's best-response function $\rho(\sigma)$ implicitly solves $F(\rho,\sigma) = 0$. Totally differentiating this expression, $\rho'(\sigma) = -(\partial F/\partial\sigma)/(\partial F/\partial\rho)$.

The sign of ρ_σ therefore depends on the sign of $E\{\pi_{\rho\sigma}\}$, which indicates how the producer's marginal incentives for ρ change as neighbors increase their biosecurity σ. Hence, the slope of $\rho(\sigma)$ reflects the strategic relation between producers.

If $E\{\pi_{\rho\sigma}\} > 0\ \forall \rho, \sigma$, then expected profit is supermodular and biosecurity efforts among producers are global strategic complements (Fudenberg and Tirole 1995): a producer will increase (decrease) his efforts as his neighbors increase (decrease) theirs. Strategic complementarities result in multiple symmetric Nash equilibria (SNE) if the complementarities are relatively strong at one SNE, with $\rho_\sigma > 1$ so that this SNE is unstable. This case is presented in Figure 1.1a. Three SNE occur where the producer's best-response function $\rho(\sigma)$ crosses the 45° line. The SNE can be Pareto-ranked, with the high-effort equilibrium *B* being privately preferred since expected profits are increasing in σ (Vives 2005; Van Zandt and Vives 2007). Equilibria *A* and *B* are locally stable, as $\rho_\sigma < 1$ in the neighborhood of each point.[7] SNE *C* is unstable (as $\rho_\sigma > 1$ at this point), with the value $\sigma = \sigma_T$ representing an "expectational threshold" between *A* and *C*. Here, a producer's Nash equilibrium biosecurity effort depends on his expectations about his neighbors' effort. A common way of envisioning this within a static model is to think of a quasi-dynamic adjustment process, or tâtonnement process, in which ρ is adjusted based on initial expectations about σ (Krugman 1991). If the producer initially expects his neighbors to choose $\sigma > \sigma_T$, then the system will be in the high-effort basin and proceed via the tâtonnement process to *B*. Alternatively, if he initially expects his neighbors to choose $\sigma < \sigma_T$, then the system will be in the low-effort basin and proceed to *A*. This latter outcome represents coordination failure, a term that we use as in Cooper et al. (1990) to define a situation where the

[7] If the coordinates of *A* lay below the unit interval, then the origin would be a locally stable SNE. If the coordinates of *B* lie above the unit interval, then (1,1) would be a locally stable SNE. This is the case in our numerical example.

producers' privately-preferred outcome *B* is not attained. Producers' expectations therefore play a critical role in disease management outcomes when multiple SNE are present.

Strategic complementarities can also coincide with a unique, globally stable SNE if the complementarities are relatively weak at this equilibrium, with $\rho_\sigma < 1$ so that the SNE is stable (Vives 2011; Panagariya and Shibata 2000). This case is presented in Figure 1.1b. Expectations do not matter in this case, as the system will always converge to point *A*.

If $E\{\pi_{\rho\sigma}\} < 0 \; \forall \rho, \sigma$, then expected profit is submodular and biosecurity efforts are global strategic substitutes. In this case, a producer will decrease (increase) his effort as his neighbors increase (decrease) theirs. SNE are unique and globally stable in this case when $|\rho_\sigma| < 1 \; \forall \; \sigma$, as illustrated in Figure 1.1c, and so expectations do not matter.[8]

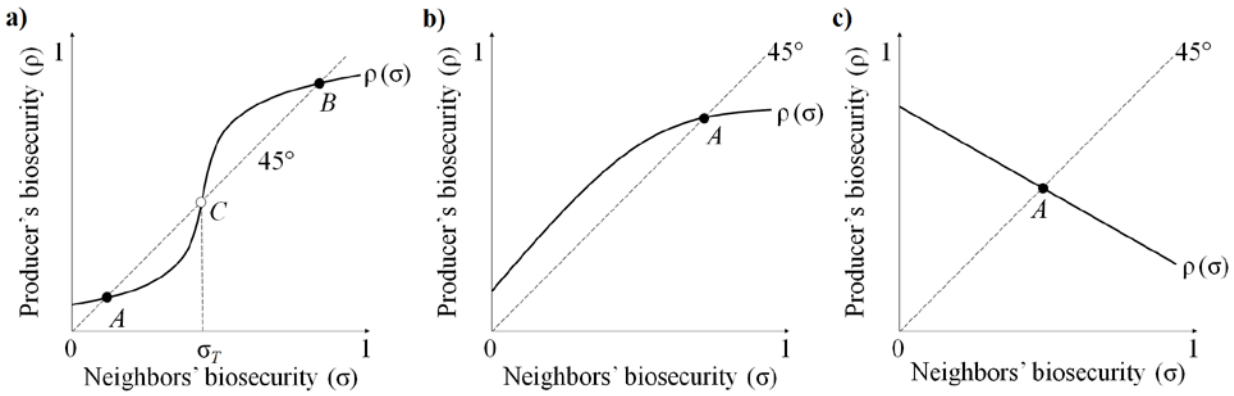

Figure 1.1 Best-response functions

Finally, it is possible that biosecurity may only exhibit strategic complementarity or substitution relations locally (i.e., $E\{\pi_{\rho\sigma}\} < 0$ or $E\{\pi_{\rho\sigma}\} > 0$ for sub-intervals of the domain for σ). Multiple equilibria may arise in such cases as well, with the stability properties described above

[8] The symmetric equilibrium becomes unstable, resulting in asymmetric equilibria, if $|\rho_\sigma(\sigma)| > 1$ at the symmetric equilibrium (Hefti 2011). Such an outcome is different from coordination failure because it involves some producers choosing high levels of biosecurity effort.

being defined locally rather than globally.

The strategic relationships depend on how neighbors' biosecurity effort affects the marginal technical effectiveness of a producer's own biosecurity. If $-P_{\rho\sigma} < 0$, so that a neighbor's biosecurity is a technical substitute (i.e., σ reduces the marginal technical effectiveness of a producer's own effort), then $E\{\pi_{\rho\sigma}\} < 0$ and biosecurity is also a strategic substitute (Aronsson and Koskela 2011). Conversely, if $-P_{\rho\sigma} > 0$, so that a neighbor's biosecurity is a technical complement (i.e., σ increases the marginal technical effectiveness of a producer's own biosecurity), then $E\{\pi_{\rho\sigma}\} > 0$ and biosecurity is also a strategic complement.

Further insight is obtained by considering the strategic relationship from an alternative—yet mathematically equivalent—perspective that follows from Young's theorem, $E\{\pi_{\rho\sigma}\} = E\{\pi_{\sigma\rho}\}$. Consider first the marginal impact of σ on the producer's risks, $\Lambda P(\rho, \sigma)$, noting that all impacts of σ occur along the spread pathway: $\Lambda P_{\sigma}(\rho,\sigma) = \Lambda\left[1 - P^{I}(\rho)\right]P_{\sigma}^{S}(\rho,\sigma) < 0$. This expression indicates that σ reduces the producer's spread risks by reducing the probability of spread. Accordingly, we can say that the negative value of the above expression, $E\{\pi_{\sigma}\} = -\Lambda P_{\sigma}(\rho, \sigma) > 0$, measures the extent to which the producer is reliant on neighbors for controlling his spread risks, with a larger value indicating greater reliance.

Now consider how ρ affects the producer's reliance on neighbors for controlling spread risks:

$$E\{\pi_{\sigma\rho}\} = -\Lambda P_{\sigma\rho}(\rho,\sigma) = \Lambda\left\{P_{\rho}^{I}(\rho)P_{\sigma}^{S}(\rho,\sigma) + \left[1 - P^{I}(\rho)\right]\left[-P_{\sigma\rho}^{S}(\rho,\sigma)\right]\right\} \tag{1.5}$$

The first RHS term in braces, which is positive, represents the degree to which the producer's biosecurity ρ reduces import risks, thereby increasing the relative risks associated with spread and hence the reliance on others to control those risks. The second RHS term in braces indicates the degree to which the producer's biosecurity ρ affects the positive spillover effects of others'

efforts in controlling spread risks. The overall sign of (1.5) is ambiguous. A positive value of (1.5) means that a larger ρ makes the producer more reliant on neighbors for protection from spread risks. In other words, by increasing ρ, the producer effectively cedes more control of his spread risks to his neighbors. This case arises when ρ increases spillover effects, i.e., $-P^S_{\sigma\rho}(\rho,\sigma) > 0$ (e.g., when jointly preventing wildlife that may act as a disease reservoir from entering common grazing areas) or when $-P^S_{\sigma\rho}(\rho,\sigma) \leq 0$ but sufficiently small in magnitude (e.g., when cleaning shared equipment before bringing it on the premises neutralizes the effect of σ on the producer's spread risks). A negative value of (1.5) (i.e., when $-P^S_{\sigma\rho}(\rho,\sigma) < 0$ and sufficiently large in absolute magnitude) means that a larger ρ makes the producer less reliant on neighbors for protection. In other words, by increasing ρ, the producer takes control of his spread risks away from his neighbors.

Expression (1.5) can be rewritten as

$$E\{\pi_{\sigma\rho}\} = \Lambda P^I_\rho(\rho) P^S_\sigma(\rho,\sigma)\left(1-\Omega^P\right), \tag{1.6}$$

where $\Omega^P = \eta^P/\varepsilon$, $\eta^P = -\dfrac{\partial\left[\partial\left(1-P^S\right)/\partial\sigma\right]}{\partial\rho}\dfrac{\rho}{\partial\left(1-P^S\right)/\partial\sigma}$, and $\varepsilon = \dfrac{\partial\left(1-P^I\right)}{\partial\rho}\dfrac{\rho}{\left(1-P^I\right)} > 0$. We use the term Ω^P to quantify the (scaled) *relative endogeneity of risk* (RER) of infection via spread, defined here as the degree to which an individual can take control of his or her own risks within a strategic setting where others' actions also influence one's risks.[9] The concept of RER extends more traditional notions of risk endogeneity (Shogren 1991) that focus on an individual's ability to control the risk he or she faces, without regard for strategic effects. Specifically, η^P measures

[9] We use the term "relative" here in the same sense as Pratt's (1964) coefficient of relative risk aversion, which is also an elasticity.

the extent to which the producer can take control of his own spread risks away from his neighbors, i.e., the degree to which spillover effects are endogenous to the producer. A positive value means that, by raising ρ, the producer takes control over his spread risks away from his neighbors, whereas a negative value implies the opposite. The term η^P is then scaled by one's self-protection impacts on exposure, $\varepsilon > 0$; the smaller is ε, the less exposure will there be to spread risks (for a given ρ) and hence the relative amount of control over these risks is less of a concern.[10]

By Young's Theorem, we see that strategic relations and RER are fundamentally related:

$$\text{(1.7)} \qquad \text{self-protection is a strategic} \begin{cases} \text{substitute} \\ \text{complement} \end{cases} \text{ iff } \text{RER} = \Omega^P \begin{cases} > \\ < \end{cases} 1.$$

If Ω^P is sufficiently large, then the producer can take control of his risks by substituting his own ρ for decreases in neighbors' σ: biosecurity is a strategic substitute, the magnitude of which is increasing in Ω^P, other things equal. If Ω^P is sufficiently small, then the producer enhances neighbors' control over his spread risks by supplementing increases in σ with increases in ρ: biosecurity is a strategic complement, the magnitude of which is decreasing in Ω^P, other things equal. These results, along with our earlier results on the stability of SNE, suggest that a larger value of Ω^P is likely to reduce the risk of coordination failure.

Finally, note that Ω^P only reflects a producer's relative ability to control the probabilistic (not economic) component of spread risks, hence the superscript *P* applied to Ω. The next section illustrates how the use of economic policy may modify expressions (1.6) and (1.7) to also reflect control over economic components of risk.

[10] The measure Ω^P could also be said to measure the relative degrees of control along the two pathways: spread and introduction. While this is also a valid perspective, we believe the focus on the relative degree to which one can endogenously control one's own risk vis-à-vis the control exerted by neighbors is a more insightful and useful perspective. For instance, the endogenous risk perspective highlights the potential role of policy mechanisms that may offer more opportunities to control one's economic risks.

1.4 Disease Prevention Policy and Strategic Interactions

Up to this point, strategic interactions between producers have depended on technological relationships. This is a common finding in the literature (e.g., Barrett 2004; Hennessy 2007b, 2008; Chen 2012). However, little attention has been paid to the potential effects of disease prevention policy on these strategic interactions. In this section we consider the effect of disease prevention policy on producers' marginal incentives for biosecurity and its effect on RER. For concreteness, assume the policy is an indemnity, which is the most commonly used instrument in practice (Hoag, Thilmany and Koontz 2006). This is not an insurance program, as no premiums are paid. Rather, these are used as safety nets and to motivate producers to report disease outbreaks on their farm to regulatory authorities, limiting the potential for disease spread. Prior work has shown, however, that indemnities may reduce the incentives for biosecurity (e.g., Muhammad and Jones 2008). We examine how a particular class of indemnity can enhance biosecurity incentives, thereby reducing adverse spillovers. Issues related to optimal policy design are explored following the numerical example below.

Returning to our model from equations (1.1) and (1.2), assume now that infected producers are compensated with an indemnity payment $\phi(\rho)\Lambda$. The relation $\phi(\rho) \in [0,1]$, denotes the share of damages for which the producer receives compensation.[11] The case of $\phi_\rho(\rho) \neq 0$ represents a behaviorally-dependent indemnity, so that compensation is greater when producers apply more biosecurity efforts. The case of $\phi_\rho(\rho) = 0$ (so that $\phi(\rho)$ is a constant) represents a behaviorally-independent indemnity, so compensation does not depend on producers' biosecurity efforts.

The representative producer's expected-profit maximization problem (1.2) is now

[11] We relax the assumption that $\phi(\rho) \geq 0$ in our analysis of optimal policy design.

$\max_{\rho} E\{\pi\} = R^H - \Lambda[1-\phi(\rho)]P(\rho,\sigma) - c(\rho)$, with first-order condition

$$-\Lambda[1-\phi(\rho)]P_\rho(\rho,\sigma) + \Lambda\phi_\rho(\rho)P(\rho,\sigma) = c_\rho(\rho). \tag{1.8}$$

Consider first the special case of a behaviorally-independent indemnity ($\phi_\rho(\rho) = 0$), which is the form indemnities typically take in practice (Hoag et al. 2006). Here, the second LHS term vanishes from (1.8), and so the indemnity reduces the marginal expected benefits from ρ, relative to condition (1.3). Other things equal, this results in lower biosecurity effort, as is consistent with most prior work on indemnity payments (Bicknell, Wilen and Howitt 1999; Kuchler and Hamm 2000; Hennessy 2007a; Muhammad and Jones 2008).

Now consider the case of a behaviorally-dependent indemnity ($\phi_\rho(\rho) > 0$). The second LHS term in (1.8) is positive so as to increase the incentives for biosecurity. However, the overall incentives provided by the indemnity are ambiguous. Subtract condition (1.3) from (1.8) to obtain the difference in the marginal benefits of biosecurity with and without the indemnity:

$$\Lambda\phi(\rho)\left[\left(1-P^S(\rho,\sigma)\right)P_\rho^I(\rho) + \left(1-P^I(\rho)\right)P_\rho^S(\rho,\sigma)\right] + \Lambda\phi_\rho(\rho)P(\rho,\sigma). \tag{1.9}$$

This difference is related to the expected indemnity payment: $\Lambda\phi(\rho)P(\rho,\sigma)$. The first term in (1.9), which is negative, is the marginal effect of ρ on the expected indemnity payment, holding the indemnity level fixed (i.e., only considering the impact of ρ on the overall probability of infection, P). The second term in (1.9), which is positive, is the marginal effect of ρ on the expected indemnity payment, holding the probability of infection fixed (i.e., only considering the impact of ρ on the payment rate, ϕ). Together, these effects represent ρ's impact on both the probabilistic and economic components of risk. The net effect is ambiguous in sign.

Expression (1.9) is positive, so that indemnities increase one's incentives for self-protection, when ρ increases the expected indemnity payment $\Lambda\phi(\rho)P(\rho,\sigma)$. In contrast, most

prior literature finds that indemnities decrease one's incentives for self-protection (e.g., Muhammad and Jones 2008). Our result differs because the effect of ρ on reducing the probability of infection may be smaller than its effect on increasing the payment amount. Gramig, Horan, and Wolf (2009) also find that biosecurity incentives can be increased by basing indemnities on observable disease outcomes that are correlated to biosecurity efforts (e.g. disease prevalence within a herd). However, they do not model strategic effects, which play an important role here.

The effect of indemnities on the strategic interactions can be seen by differentiating (1.8) with respect to σ:

$$E\{\pi_{\rho\sigma}\} = -\Lambda\left[1-\phi(\rho)\right]P_{\rho\sigma}(\rho,\sigma) + \Lambda\phi_{\rho}(\rho)P_{\sigma}(\rho,\sigma). \tag{1.10}$$

Consider first the case of a behaviorally-independent indemnity ($\phi_\rho(\rho) = 0$), in which case the final RHS term vanishes. The indemnity does not impact the prevailing type of strategic relationship, as it does not affect the sign of $E\{\pi_{\rho\sigma}\}$ relative to equation (1.5). However, relative to equation (1.5), the indemnity produces a scaling effect that reduces the magnitude of any complementarities or increases the magnitude of substitution. For instance, suppose there were strategic complementarities involving multiple equilibria prior to the indemnity. In reducing the magnitude of the complementarities, $\rho_\sigma(\sigma)$ is reduced and so the unstable SNE may be eliminated. This means the behaviorally-independent indemnity may result in a unique SNE, which would likely be a small value of ρ since this indemnity reduces biosecurity incentives. Our numerical example below illustrates this might be the case with a large indemnity. Multiple equilibria will persist if the reduction in complementarities is not too great, although the basin of attraction for the low-effort SNE may increase due to the reduced biosecurity incentives under the indemnity. Hennessy (2007a) shows that the potential for coordination failure may increase

with a small indemnity, thereby increasing the importance of expectations.

Next consider the case of a behaviorally-dependent indemnity ($\phi_\rho(\rho) > 0$) so that the final RHS term in (1.10) does not vanish. Now the sign and magnitude of $E\{\pi_{\rho\sigma}\}$ depends on both a (technologically-defined) probabilistic relationship, $P_{\rho\sigma}(\rho,\sigma)$, and an economic relationship, $\phi_\rho(\rho)$. Specifically, the term $\Lambda\phi_\rho(\rho)P_\sigma(\rho, \sigma)$ reflects the larger indemnity that can be earned in response to a larger ρ. This term is negative: an increase in σ decreases the probability of infection, and thus decreases the producer's expected marginal indemnity benefits from ρ.

If ρ and σ were strategic substitutes prior to the indemnity, they will remain so after the indemnity. If they were strategic complements prior to the indemnity, the post-indemnity outcome is ambiguous and depends on the relative magnitude of the probabilistic and economic effects described above. At a minimum, the behaviorally-dependent indemnity will weaken the complementarities, and it could even change the sign of $E\{\pi_{\rho\sigma}\}$ relative to the case of no indemnity or a behaviorally-independent indemnity. For instance, suppose there were strategic complementarities involving multiple equilibria prior to the indemnity. In reducing the magnitude of the complementarities, $\rho_\sigma(\sigma)$ is reduced and so the unstable SNE may be eliminated. This means the behaviorally-dependent indemnity may result in a unique SNE. But, in contrast to the case of the behaviorally-independent indemnity, the SNE may be at a large value of ρ since the behaviorally-dependent indemnity increases biosecurity incentives. Thus, a behaviorally-dependent indemnity may eliminate both the possibility of coordination failure and the role of expectations. Such a case is explored in the numerical example below.

The strategic impacts of a behaviorally-dependent indemnity also affect our measure of RER. Specifically, we can derive the following condition:

(1.11) self-protection is a strategic $\begin{cases} \text{substitute} \\ \text{complement} \end{cases}$ iff $\text{RER} = \Omega^P + \Omega^E \begin{cases} > \\ < \end{cases} 1$,

where $\Omega^E = \eta^E/\varepsilon$ and $\eta^E = -[\partial(1-\phi)/\partial\rho][\rho/(1-\phi)] > 0$ when $\phi_\rho > 0$. The numerator η^E reflects the producer's ability to take control over his infection risks via the indemnity. The denominator of Ω^E once again reflects exposure to spread risks via the term ε.

Condition (1.11) indicates that RER is unchanged for a behaviorally-independent indemnity, since $\Omega^E = 0$ in this case so that $\text{RER} = \Omega^P$ as in condition (1.7). In the case of a behaviorally-dependent indemnity, however, condition (1.11) indicates that RER is increased by the term Ω^E. This new measure of RER quantifies the producer's ability to take control over both components of spread risk: the probability of infection via spread (reflected by Ω^P) and the economic impact of infection (reflected by Ω^E). The more able a producer is to control his economic risks, the less likely are there to be strong strategic complementarities that generate the potential for coordination failure.

1.5 Numerical Example: The 2001 UK Foot-and-Mouth Disease Epidemic

We now explore our model through the use of an illustrative numerical example based on the 2001 UK FMD epidemic. The outbreak began in Northumberland County in northeastern England in early February. The source of the outbreak was traced to a pig that had been fed infected meat (Segarra and Rawson 2001). The disease initially spread throughout Great Britain via animal movements, contaminated vehicles, and farm workers (Gibbens et al. 2001). Nationwide movement restrictions were implemented in late February, at which point the virus continued to spread via airborne transmission (Donaldson and Alexandersen 2002). At its greatest extent, the outbreak had spread throughout Great Britain and parts of France, Ireland, and the Netherlands. By the time the outbreak was over, more than six million cattle, sheep, pigs,

and other animals had been slaughtered in the UK alone, with economic damages to producers, government, and the tourism sector totaling over £3 billion ($4.4 billion; Thompson et al. 2002).

We apply our model to the case of dairy farmers in Cumbria County, located in northwestern England. Cumbria was the most heavily affected county during the 2001 outbreak, experiencing 44 percent of the total number of cases reported nationwide (Convery et al. 2005). The next section describes the model specification. We then calculate Nash equilibria for two scenarios: (i) a baseline case in which no disease prevention policy exists and (ii) the case of behaviorally-dependent and -independent policies.

1.5.1 Model Specification

Consider a region of Cumbria County that is initially disease-free but at risk of infection from outside sources. We assume producers in this region are homogeneous in herd size, costs, and prices. The analysis is therefore based on the perspective of a representative producer. The representative producer's herd is at risk from infection along two pathways: (i) primary infection via sources outside the region (i.e., pathogen introduction) and (ii) secondary infection via direct or indirect contact with neighboring producers who have had the pathogen introduced to their herds (i.e., pathogen spread). For simplicity, we adopt a static, one-period, non-spatial model of these processes, thereby representing behavior at the earliest stages of an outbreak. This framework is consistent with prior work (Hennessy 2008), except that we allow producers to choose biosecurity to protect their herds from both pathogen introduction and spread.[12]

[12] Hennessy (2007b) also considers tertiary infections, i.e., when a producer infects another producer who infects another producer. This was the exclusive pathway through which neighbors' biosecurity effort choices enter the representative producer's profit-maximization problem. In contrast, we account for positive spillovers from neighbors' efforts to prevent spread via reducing the effective number of contacts between herds. This means of controlling spread has not been examined previously in economic studies. Our assumption that herds are not at risk from tertiary infections is a simplifying one, although it is unlikely to affect the general results of the model.

First consider the introduction pathway. Let the probability that the pathogen is introduced into the representative producer's herd be $P^I(\rho) = \xi(1 - \mu\rho)$. Here, ξ is the probability of pathogen introduction when no biosecurity is adopted, and μ denotes the reduction in this probability per unit of biosecurity.

Now consider spread from neighboring herds that have become infected. The pathogen is introduced into a neighboring herd with probability $P^I(\sigma) = \xi(1 - \mu\sigma)$. Spread to the representative producer's herd may then occur via direct or indirect contact. Direct contact may occur in communal grazing areas or livestock exhibitions, whereas indirect contacts may involve disease transmission vectors such as people, wildlife, or airborne droplets of water containing infectious agents. Let $N \geq 1$ denote the number of herds in the region that may directly or indirectly contact the representative producer's herd in the absence of measures to avoid those contacts. Biosecurity effort by either the representative producer or his neighbors can reduce the effective number of contacts between herds, e.g., by limiting access to farm workers who travel between farms, by cleaning shared equipment, or by prohibiting the movement of animals between neighboring farms. Suppose biosecurity reduces the producer's effective number of contacts to $\hat{N}(\rho, \sigma) = N(1 - \alpha\rho)(1 - \alpha\sigma)$, where α represents the reduction in contacts per unit of biosecurity adopted.[13] This specification implies that neighbors' biosecurity efforts are as effective in preventing contacts as the producer's own efforts, e.g., because the biosecurity

[13] Absent biosecurity, the representative producer can potentially make contact with each of his N neighbors. This represents a case of uniform mixing over the landscape. This is a somewhat extreme assumption, but it is commonly used in epidemiological modeling, particularly in metapopulation models (e.g., Watts et al. 2005). It seems somewhat realistic here, in the absence of movement restrictions, since the disease is easily spread by movement of people and equipment across farms, which may be somewhat randomly distributed over the landscape. As biosecurity is adopted, more weight would have to be put on more localized (e.g., aerial) transmission. This is accomplished by adopting a larger value of α, which effectively puts less weight on the total surrounding population N. Epidemiological models often adopt a similar approach to modeling spatial transmission by putting less weight on the "susceptibility function", which describes the susceptible portion of the population that is likely to come into contact with infected animals (Barlow 1995).

technology used by each producer is the same.

Given that the producer's herd is uninfected and contact does occur with an infected neighboring herd, let $k(\rho) = \kappa(1 - \nu\rho)$ represent the probability that such contact spreads the pathogen to the producer's herd. Here, κ is the probability that contact with an infected herd results in spread absent biosecurity, and ν is the reduction in this probability per unit of biosecurity adopted. The probability the producer's herd becomes infected from contact with a particular neighbor's herd, conditional on that herd being infected, is then $k(\rho)P^I(\sigma)$. Note that $k(\rho)$ does not depend on σ, as it is assumed that neighbors have no incentive to prevent disease from leaving their farm. Even so, neighbors' biosecurity efforts produce positive spillovers in reducing spread, as their efforts reduce both the probability of pathogen introduction to the region and the number of contacts between herds, as described above.

Given this specification, the probability that a producer's herd becomes infected via spread from any of his neighbors—conditional on the producer's herd being uninfected—can be modeled as a Bernoulli process:[14]

$$P^S(\rho,\sigma) = 1 - [1 - k(\rho)P^I(\sigma)]^{\hat{N}(\rho,\sigma)}. \tag{1.12}$$

The expression $[1 - k(\rho)P^I(\sigma)]$ is the probability that a particular neighbor's herd does not infect the producer's herd, and so $[1 - k(\rho)P^I(\sigma)]^{\hat{N}(\rho,\sigma)}$ represents the probability that the producer's herd does not get infected by any of his neighbors' herds. Thus, $P^S(\cdot)$ is the probability that at least one infectious contact occurs via a neighbor's herd. Note that $P^S(\cdot)$ is increasing in N; as the number of producers in a region increases, the likelihood of an infectious contact with at least

[14] Note that it is likely that the exponent $\hat{N}(\cdot)$ will take a non-integer value. In the strictest sense, a Bernoulli process requires the number of trials to be in the set of nonnegative natural numbers, $\mathbb{N}_+$. However, allowing non-integer values in equation (1.12) serves as a reasonable approximation of the probability of infection and the differentiability allowed by this functional form allows for a greater level of analysis while preserving realistic assumptions about how biosecurity affects contact rates between herds.

one neighbor's herd increases for the representative producer. Likewise, $P^S(\cdot)$ is decreasing in ρ and σ. Finally, the probability that a producer's herd becomes infected via either pathway (introduction or spread) is found by substituting $P^I(\rho)$ and $P^S(\rho,\sigma)$ into equation (1.1).

The producer's problem (1.2) is parameterized using values taken or derived from economic and epidemiological studies of the 2001 UK FMD outbreak. It is assumed that the parameters are homogeneous among all N producers. Allowing the parameters to vary among producers will affect the quantitative results of our example but will not qualitatively change how biosecurity affects infection risks and economic incentives. Thus, the insights into the strategic interactions provided by this numerical example will be unaffected. Parameter values are presented in Table 1.1. The sources and derivation of each parameter are detailed in Appendix

Table 1.1 Functional Forms and Parameters Used for the Numerical Example

	Domain	Form/Value in Numerical Example[a]	Description	Units
Parameters				
ξ	[0, 1]	0.3125[*#]	Probability of introducing infected animal, no biosecurity	Unitless
μ	[0, 1]	1	% reduction in probability of introduction per unit of biosecurity	Unitless
κ	[0, 1]	0.9[*§]	Probability of infection given contact occurs	Unitless
ν	[0, 1]	0.15	% reduction in probability of infection from contact per unit of biosecurity	Unitless
χ	≥ 0	1,328[*]	Cost parameter	$
N	≥ 2	80[†]	Number of neighboring herds	Herds
α	[0, 1]	0.75[*†♠+]	% reduction in contacts per unit of biosecurity	Unitless
$\Lambda = R^H - R^{NH}$	$[0, \infty)$	26,404[‡]	Cost of infection	$
Variables				
ρ	[0, 1]	—	The producer's biosecurity effort	Units of effort
σ	[0, 1]	—	Neighboring producers' biosecurity effort	Units of effort
Functions				
$P^I(\rho), P^I(\sigma)$	[0, 1]	$P^I(\rho) = \xi(1 - \mu\rho), P^I(\sigma) = \xi(1 - \mu\sigma)$	Probability of introducing infected animal to farm	Unitless
$P^S(\rho, \sigma)$	[0, 1]	$P^S(\rho, \sigma) = 1 - [1 - k(\rho)P^I(\sigma)]^{N(\rho, \sigma)}$	Probability of infection from spread	Unitless
$k(\rho)$	[0, 1]	$k = \kappa(1 - \nu\rho)$	Probability herd becomes infected given contact with infected herd	Unitless
$\hat{N}(\rho, \sigma)$	$[0, N]$	$\hat{N}(\rho, \sigma) = (N - 1)(1 - \alpha\rho)(1 - \alpha\sigma)$	Effective number of neighboring farms	Farms
$E\{\pi\}$	$\mathbb{R}$	$E\{\pi\} = R^H - \Lambda P(\rho,\sigma) - c(\rho)$	Expected profit	$
$c(\rho)$	$[0, \infty)$	$c(\rho) = \chi\rho^2$	Cost of biosecurity effort	$

[a] Note: [*] Chi et al. (2002); [#] Green et al. (2006); [§] Schley et al. (2009); [†] Brennan et al. (2008); [♠] Defra (2011); [‡] Thompson et al. (2002); [+] Ferguson et al. (2001). Entries without superscripts are assumed values

A. Note that biosecurity is assumed to be only marginally effective in reducing the infectivity of contact $k(\rho)$ between herds. This is because FMD is highly contagious and is capable of aerial transmission over relatively long distances (Mikkelsen et al. 2003; Ferguson et al. 2001). Also because of aerial transmission, we assume biosecurity can only eliminate contact from neighbors located outside of a 2 km radius of the representative farm; within this radius, there is always positive probability of aerial transmission (Ferguson et al. 2001).

1.5.2 Scenario (i): Baseline Case with No Disease Prevention Policy

We first consider strategic interactions arising from the baseline case when no indemnity or other disease prevention policy is present, i.e., the baseline case. We solve first-order condition (1.3) numerically using *Mathematica 7.0* (Wolfram Research, Inc. 2008) to derive the producer's best-response function, $\rho(\sigma)$. This function is depicted by the solid, discontinuous curve in Figure 1.2. The only two equilibria present in the model are the two symmetric Nash equilibria (SNE), *A* and *B* in Figure 1.2.[15] The SNE can be Pareto-ranked, with the high-effort equilibrium *B* being privately preferred (Van Zandt and Vives 2007).[16]

The SNE *A* and *B* are each locally stable, with $\rho_\sigma < 1$ at both points. The dashed vertical line running through point *C* represents a discontinuity in the response function that acts like an unstable threshold, dividing the graph into two basins of attraction: a low-effort basin with a stable SNE at *A* and a high-effort basin with a stable SNE at *B*.[17] Expectations matter in Figure

[15] We consider only pure-strategy Nash equilibria. Mixed strategies are often unstable (Harsanyi 1973; Echenique and Edlin 2004), and can therefore be unreliable predictors of behavior.

[16] Note that the high-effort equilibrium is also socially-preferred in this case since it represents the maximum level of biosecurity available. This is not a general result, however; if the highest SNE value of σ were strictly less than 1, then it would not be Pareto optimal (Milgrom and Roberts 1990).

[17] The stability properties of the system follow from the fact that, according to the numerical results, $E\{\pi_\rho\} < 0$ for σ below the discontinuity and $E\{\pi_\rho\} > 0$ for σ above the discontinuity. Intuitively, when two stable equilibria are divided by a threshold, the threshold must be unstable. It is therefore straightforward to hypothesize a tâtonnement process of adjustment to *A* for any expectation by the representative producer that places σ to the left of the

1.2. If producers expect their neighbors initially choose biosecurity $\sigma > \sigma_T$, then the system proceeds via tâtonnement to *B*. Conversely, if producers expect their neighbors initially choose biosecurity $\sigma < \sigma_T$, then coordination failure occurs and the system proceeds via tâtonnement to *A*.[18]

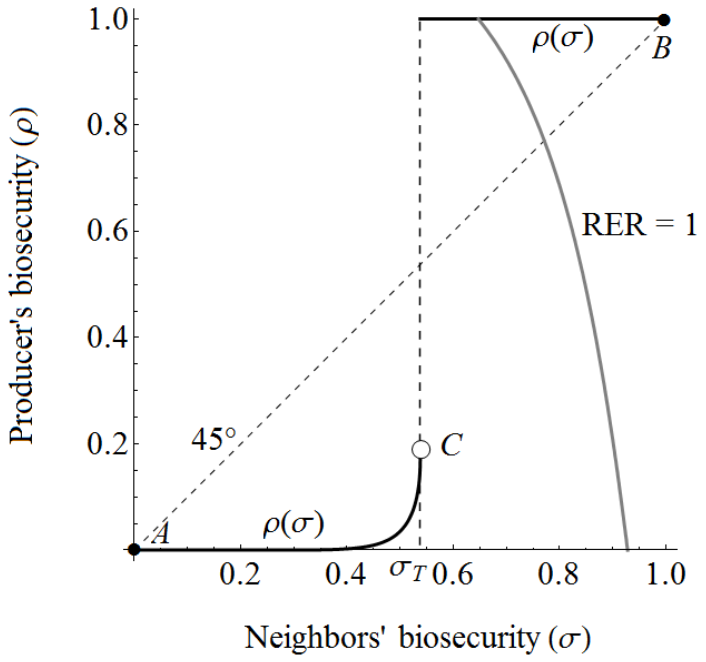

Figure 1.2 The representative producer's best-response function with no policy

The multiple, stable SNE in Figure 1.2 arise due to strong strategic complementarities within the neighborhood of the discontinuity. Strategic complementarities in this region can be seen in two ways. First is the positive slope of $\rho(\sigma)$ for all points between *A* and *C*. Second, the curve labeled RER = 1 in Figure 1.2 is a contour line denoting the locus of points for which Ω^P =

discontinuity or to *B* for any expectation that puts σ to the right of the discontinuity. Note also that if a 45° line extending from the origin intersected the positively-sloped portion of ρ(σ), the resulting SNE would also be unstable as $\rho_\sigma > 1$ at such a point.

[18] We perform a sensitivity analysis for the baseline parameterization. For conciseness, we present the analysis in Appendix A.

1. To the left of this curve, $\Omega^P < 1$, and thus by condition (1.7), biosecurity is a local strategic complement at *A*, as well as up to and beyond the expectational threshold. To the right of this curve, $\Omega^P > 1$, and thus biosecurity is a local strategic substitute at *B*.

1.5.3 Scenario (ii): Indemnities

Consider next the case in which an infected producer receives an indemnity $\phi(\rho)$ that offsets his losses due to disease. We begin by analyzing behavior under a behaviorally-dependent indemnity, i.e., $\phi_\rho(\rho) > 0$. Assume for simplicity that $\phi(\rho) = \zeta\rho$, where $\zeta = 0.68$, i.e., the producer receives compensation equal to 68 percent of his losses from infection if he fully self-protects. This value of ζ is chosen because it yields $\rho(1) = 1$ as an interior solution so that the producer is indifferent to fully self-protecting; a larger amount would actually reduce his biosecurity incentives (see below). Using the relation for $\phi(\rho)$, we numerically solve the first-order condition (1.3) for the best-response function $\rho(\sigma)$, with a maximum of $\rho(\sigma) = 1$. The resulting function is represented by the solid curve in Figure 1.3.

The behaviorally-dependent indemnity drastically changes the producer's best-response function relative to Figure 1.2. The indemnity leads to greater incentives for ρ when σ is small, shifting the expectational threshold leftward to $\sigma = 0$ such that the low-effort basin—and the low-effort equilibrium *A*—is eliminated. The only SNE remaining in Figure 1.3 is the high-effort equilibrium *B*.

The expectational threshold and the low-effort equilibrium have been eliminated in Figure 1.3 because the indemnity has increased RER for each value of σ, weakening strategic complementarities at lower effort levels. Indeed, curve RER = 1 now lies to the left of the RER = 1 curve from Figure 1.2, as the RER = 1 curve is now a contour line denoting the locus of points for which $\Omega^P + \Omega^E = 1$ (see condition (1.11)). Since the unique SNE is located to the right of

RER = 1 in Figure 1.3, biosecurity is a strategic substitute at equilibrium, and *B* is globally stable: expectations do not matter. A larger marginal indemnity rate ζ produces similar qualitative results, although the high-effort equilibrium *B* is reduced slightly. Intuitively, a higher rate would increase the producers' control over his spread risks enough such that he has incentives to scale back his biosecurity (see the tradeoffs described in relation to expression (1.9)).

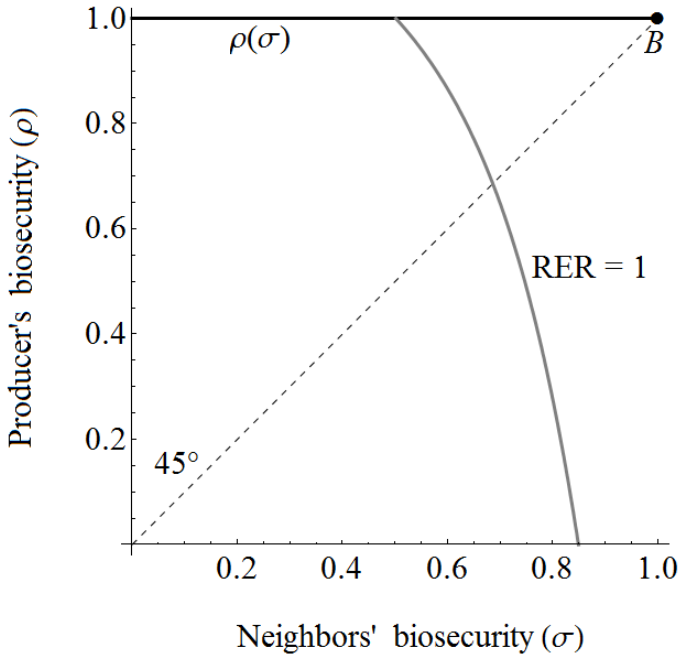

Figure 1.3 A behaviorally-dependent indemnity

Finally, consider a behaviorally-independent indemnity. For the sake of comparison, let the indemnity take the form $\phi(\rho) = 0.68$ such that it is equal to the equilibrium indemnity earned in the behaviorally-dependent case above. Plugging this indemnity into the producer's expected profit maximization problem, (1.8) is again solved for the best-response function ρ(σ), shown in Figure 1.4a. Decoupling the indemnity from behavior has lowered the producer's incentive to

adopt biosecurity for any value of σ, shifting ρ(σ) rightward and downward relative to Figure 1.2. Also, although RER is unaffected—the measure of RER under a behaviorally-independent indemnity is once again given by condition (1.7)—the magnitude of the strategic complementarities has been reduced (i.e., the best response curve is less steeply sloped along the interval from points *A* to *C* relative to Figure 1.2). The reduced complementarities along with reduced biosecurity incentives has expanded the low-effort basin by shifting the expectational threshold σ_T to the right relative to Figure 1.2, increasing the risk of coordination failure. A larger behaviorally-independent indemnity causes the expectational threshold to vanish, as in Figure 1.4b. The only SNE remaining in Figure 1.4b is the low-effort equilibrium *A*, which is globally stable since biosecurity is a weak strategic complement at this point, and so expectations do not matter.

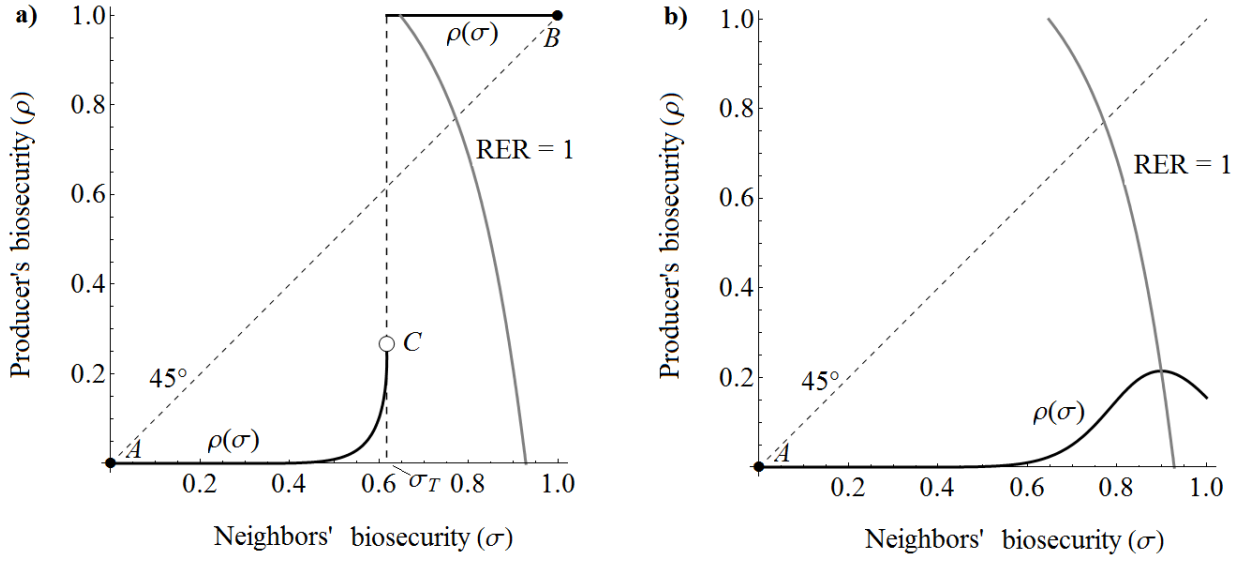

Figure 1.4 a) A small and b) large behaviorally-independent indemnity

1.6 Optimal Indemnity Design

As a final consideration, we explore the social planner's problem of choosing the optimal indemnity to maximize the net economic surplus arising from self-protection. We begin by designing a linear indemnity of the form $\phi(\rho) = \zeta\rho$, where the parameter ζ is derived based on the first-best optimality conditions. Therefore, we first characterize the first-best outcome.

Our specification of social net economic surplus is based on the simplifying assumptions that the disease has no zoonotic spillovers on human health, that the regulated producers do not have a collective impact on input or output prices, and that society is risk neutral. Also, we continue to assume producers are homogeneous. Given these assumptions, social net economic surplus is simply the aggregate profit to livestock producers:

$$E\{\Pi\} = \max_{\rho}\left\{(N+1)\left[R^{H} - \Lambda P(\rho,\sigma) - c(\rho)\right]_{\sigma=\rho}\right\}. \tag{1.13}$$

The first term in brackets is the representative producer's expected profit and the second term in brackets represents the aggregate profits of his *N* neighbors.

For an interior solution, the optimal level of biosecurity for the representative producer solves $\partial E\{\Pi\}/\partial\rho = 0$, or

$$-\Lambda P_{\rho}(\rho,\sigma)\big|_{\sigma=\rho} - \Lambda P_{\sigma}(\rho,\sigma)\big|_{\sigma=\rho} = c_{\rho}(\rho). \tag{1.14}$$

The first LHS term and the RHS term of equation (1.14) are the same as in the producer's decentralized first order condition (1.3) for the case of no indemnity. The second LHS term ($-\Lambda P_{\sigma} > 0$) accounts for the positive spillovers from ρ on others' disease risks; given symmetry, $\sigma = \rho$. These spillovers cause the social incentives for biosecurity to exceed the private incentives. Intuitively, individual producers do not take account of the positive spillovers generated by their biosecurity. When the decentralized optimality condition (1.3) is satisfied as an equality (an interior solution), producers exert too little effort relative to the first-best. The

optimal indemnity must therefore give producers an incentive to internalize the spillovers arising from their choice of ρ. A decentralized outcome involving a corner solution of $\rho = \sigma = 1$ will coincide with the first-best. Hence, the socially-optimal level of ρ is only weakly greater than the level chosen in the decentralized case.

Consider an interior first-best outcome. We can set parameter ζ to equate the producer's first-order condition for the case of a decentralized indemnity (equation 1.8) to that of the command optimum (equation 1.14) evaluated at the first-best outcome:[19]

$$-\Lambda P_{\sigma}\left(\rho^{*},\sigma\right)\Big|_{\sigma=\rho^{*}} = \zeta\left[\Lambda P\left(\rho,\sigma\right)\left(1+\delta\right)\right]\Big|_{\sigma=\rho=\rho^{*}}, \tag{1.15}$$

where ρ^{*} is the first-best outcome and $\delta = (\partial P/\partial\rho)(\rho/P) < 0$ is the elasticity of the probability of infection with respect to self-protection. The LHS of expression of (1.15) represents the positive spillover effects of biosecurity. The RHS of (1.15) is the marginal incentive provided by the indemnity.

The first-best value ζ^{*} is derived from expression (1.15) as $\zeta^{*} = [\partial(1 - P^{*})/\partial\sigma]/[P^{*}(1 + \delta^{*})]$, where the superscript * means all variables are evaluated at $\sigma = \rho = \rho^{*}$. The numerator of ζ^{*}, which is positive, is the aggregate marginal reduction in infection probability from biosecurity. This is normalized by the biosecurity effectiveness term $(1 + \delta^{*}) > 0$.[20] Note that ζ^{*} is unaffected by whether biosecurity is a strategic complement or substitute.

The indemnity rate ζ^{*} corrects the inefficiency arising from the spillover effects of one's self-protection on others' expected profits. However, we argue that ζ^{*} is first-best only when this

[19] An alternative but equivalent approach involves choosing policy instrument parameters to maximize net economic surplus, conditional on producers' optimal response to the policy in the decentralized setting (Acocella, Di Bartolomeo and Hughes-Hallett 2013).

[20] To see that $(1 + \delta^{*}) > 0$, assume otherwise, i.e., that $(1 + \delta) \leq 0$. This condition requires that $(P/\rho + P_{\rho}) \leq 0$ since $(1 + \delta) = (P + \rho P_{\rho})/P = (P/\rho + P_{\rho})\rho/P$, where the first equality follows from the definition of δ. However, this implies a contradiction since $(P/\rho - [-P_{\rho}])$ is strictly positive given that P is decreasing and convex in ρ, requiring that the average impact of ρ on P exceeds the absolute value of the marginal impact.

rate yields a globally stable SNE at the first-best outcome. Otherwise, in the case of multiple, locally stable equilibria (of which one is the first-best outcome), ζ^* does not guarantee the first-best outcome. The reason is that the linear indemnity rate ζ^* does not address an additional source of inefficiency that is generally present in this setting: the problem of coordinating on the first-best equilibrium. This inefficiency can manifest itself when there are multiple decentralized SNE, which may occur when biosecurity is a relatively strong strategic complement.

An indemnity with an additional parameter is needed to address this additional source of inefficiency (Tinbergen 1952). We consider a general indemnity $\phi(\rho; \zeta_1, \zeta_2)$, where ζ_i are parameters to be chosen optimally. The producer's decentralized first-order condition (1.8) may be implicitly solved for the symmetric best-response function conditional on the indemnity, denoted $\rho(\sigma, \zeta_1, \zeta_2)|_{\sigma=\rho*}$. The first-best policy parameters are then chosen to simultaneously solve the following conditions:

(1.16a) $$\rho(\sigma,\zeta_1,\zeta_2)|_{\sigma=\rho^*} = \rho^*$$

(1.16b) $$\int_0^{\rho^*} \rho(\sigma,\zeta_1,\zeta_2)\,d\sigma > \int_0^{\rho^*} \sigma\,d\sigma \quad \forall\,\sigma\,.$$

Condition (1.16a) addresses the positive spillovers from biosecurity. Specifically, it states that the best response curve intersects the first-best level of biosecurity to generate a SNE at ρ^*. This is the sole condition that must be satisfied under traditional notions of optimal indemnity design, and was addressed by our linear indemnity in condition (1.15).

Condition (1.16b) addresses the risk of coordination failure. Specifically, this condition requires the area beneath the producer's best-response function (the LHS) to be greater than the area beneath the 45° curve (the RHS), over the range $\sigma = 0$ to $\sigma = \rho^*$. This ensures there are no

other SNE at values of $\sigma < \rho^*$, thereby eliminating the risk of coordination failure.[21] Note that (1.16b) is automatically satisfied when biosecurity is a weak strategic complement or a global strategic substitute.

Consider first the effect of the optimal indemnity for the general case in which interior equilibria exist prior to the indemnity. There are three cases, as pictured in Figures 1.1a–c. In each case, the first-best outcome would lie on the 45° curve above and to the right of the high-effort SNE (i.e., point *B* in figure 1a, or point *A* in Figures 1.1b or 1.1c). An optimal indemnity will shift the best-response function upwards through the first-best outcome. In the case of multiple equilibria in Figure 1.1a, the optimal indemnity would also rotate the best response function clockwise so that it no longer intersects the 45° curve at any point other than the first-best outcome. Note that condition (1.16b) also ensures the new best-response function will intersect the 45° curve from above, guaranteeing that the solution is globally stable. [22]

Finally, consider the special case in which the first-best outcome is a corner solution, i.e., $(\rho, \sigma) = (1, 1)$. This is the case for our baseline numerical example. Point *B* in the no-policy case of Figure 1.1a is a SNE corner solution of (1, 1) that coincides with the first-best optimum. Thus, no indemnity or other instrument is needed under traditional notions of first-best policy design which consider sub-optimal self-protection as the sole source of inefficiency. However, the first-

[21] This condition is the same as that which characterizes first-order stochastic dominance (Mas-Colell, Whinston and Green 1995). This condition is sufficient to guarantee a unique SNE. To see this, suppose that there exist multiple SNE. This implies the presence of strategic complementarities. In this case, each SNE can be Pareto ranked, with the largest one being socially preferred. Since ρ^* must be weakly greater than any decentralized equilibrium, there cannot be a preferred equilibrium above the first-best level, ρ^*. Thus, there cannot exist additional SNE above ρ^* if condition (1.16b) is satisfied.

[22] For instance, an indemnity of the form $\phi(\rho) = \zeta_1 + \zeta_2\rho$ may achieve this outcome. Specifically, a negative value of the lump sum component, ζ_1 , and a positive value of ζ_2 will shift the producer's best-response function upwards for all σ, increasing his incentives for biosecurity. At the same time, a positive value of ζ_2 will increase RER to help eliminate the risk of coordination failure. Together, these values can be set to accomplish the two objectives defined by conditions (1.16). An additional parameter, ζ_3, may be required to satisfy $\partial\rho(\sigma, \zeta_1, \zeta_2, \zeta_3)/\partial\sigma > -1 \; \forall\sigma$, so as to avoid particularly strong strategic substitute relations that can generate asymmetric equilibria.

best outcome only arises if producers' expect $\sigma > \sigma_T$. Thus, despite the fact that the first-best outcome is a SNE, the potential exists for producers to equilibrate at a locally stable, sub-optimal outcome (i.e., point *A*, which is the origin in our model). An indemnity that eliminates the expectational threshold and makes the first-best SNE globally stable can therefore ensure a welfare improvement.

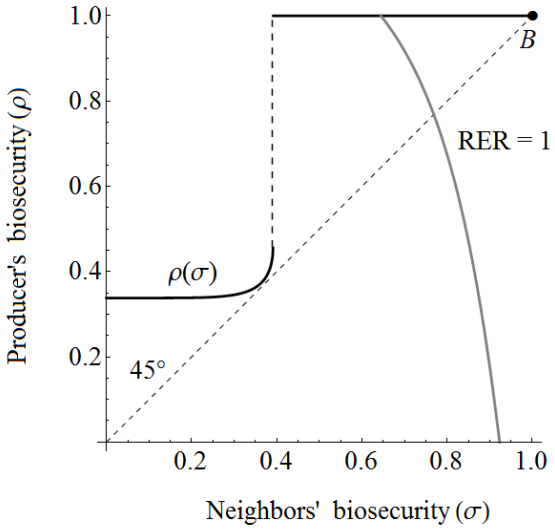

Figure 1.5 A best-response function under an optimal indemnity

Figure 1.5 depicts the effect of a small, behaviorally-dependent indemnity (i.e., $\phi(\rho) = 0.034\rho$). Like the no-policy case in Figure 1.2, the first-best optimum (point *B*) remains an SNE under this indemnity. The indemnity in Figure 1.5 specifically addresses the second source of inefficiency described above: it has increased RER and, with it, producer incentives for self-protection at low levels of σ, shifting the best-response function above the 45° line. Here, the first-best outcome *B* is now a unique SNE: by increasing RER, the optimal indemnity provides producers with just enough control over their economic risks so as to eliminate the risk of coordination failure and ensure the long-run sustainability of the first-best outcome. A larger

behaviorally-dependent indemnity is unnecessary and may even reduce the SNE level of self-protection and make it unstable (see footnote 7).

1.7 Discussion and Conclusion

Understanding that environmental risks are endogenous has had important implications for managing environmental systems (e.g., Archer and Shogren 1996; Kane and Shogren 2000; Treich 2010). When these risks involve positive spillovers from self-protection, strategic interactions among at-risk individuals may endogenously affect individual risks. In this chapter, we have attempted to provide insight as to how various strategic relationships endogenously arise and how decision-makers can influence them to enhance disease management.

Our analysis of endogenously- and strategically-determined risk has led us to identify a new concept linking these elements: the *relative endogeneity of risk*, which measures individuals' ability to take control over their own risks. A smaller RER results in self-protection being strategic complements where coordination failure may occur, whereas a larger RER yields a more stable strategic relation (e.g., weak strategic complements or strategic substitutes) with potentially greater levels of self-protection. Furthermore, disease prevention policies can influence RER, altering strategic relationships and the resulting equilibrium outcomes.

Although our numerical analysis is based on a very simple model (i.e., a single period with homogenous agents and non-spatial interactions) to highlight the link between RER and the strategic interactions between at-risk individuals, we should emphasize that our analytical framework is quite general and could be expanded to incorporate heterogeneous agents interacting via various types of spatial disease transmission encompassed by P^S (e.g., aerial transmission, direct or indirect contact between herds, etc.). Also, the underlying disease processes may be dynamic, although disease dynamics are not modeled explicitly. Making the

model fully dynamic in this context would primarily involve making the infection probabilities non-stationary, but it would not alter the behavioral response mechanisms assuming that individuals' behavior is myopic. Myopic behavior makes sense when self-protection efforts do not involve capital accumulation but instead involve the amount of care exerted in each period—which is true of many livestock diseases. Still, the lack of explicit disease dynamics is limiting in the sense that we are unable to see how strategic behaviors affect epidemiological dynamics and how these may affect future strategic interactions and, hence, future RER. We leave for future research any extensions to a fully dynamic model, and also to non-myopic behavior for which the strategic interactions would have clear analogues in differential games.

Finally, RER may be of value as a metric. Metrics are commonly used in epidemiology, with R_0 (defined as the average number of cases an infected individual generates over the course of its infectious period) being the key metric of interest. R_0 typically does not capture economic relationships that may be important in disease transmission (Fenichel et al. 2011). RER may therefore serve as an economic metric that provides greater insight into both infection risks and the incentives individuals have to self-protect against these risks. We are aware of only one prior study that econometrically estimates the strategic relationships governing self-protection from disease (Kobayashi and Melkonyan 2011). Future work utilizing structural econometric models may be useful in estimating the technological and economic relationships that determine RER, leading to greater efficacy of disease prevention efforts.

CHAPTER 2 ECONOMIC INCENTIVES FOR MANAGING BIOLOGICAL POLLUTION RISKS FROM TRADE

2.1 Introduction

The movement of live animals is a key driver behind the spread of infectious livestock diseases (Perry, Grace and Sones 2011), which can be considered a form of "biological pollution" akin to invasive species (Daszak, Cunningham and Hyatt 2000; Horan et al. 2002). The economic impact of infectious disease is staggering. For example, the costs of the 2001 foot-and-mouth disease outbreak in the United Kingdom—which was initially spread throughout Great Britain via animal movements—totaled over $4 billion (Thompson et al. 2002). Mechanisms to mitigate the externalities associated with live animal movements have thus gained considerable attention.

Prior literature asserts that trade-related biological pollution externalities arise not from trade per se, but rather from trade of contaminated goods (e.g., Paarlberg and Lee 1998; McAusland and Costello 2004; Mérel and Carter 2008). This literature acknowledges that it is generally not possible or cost-effective to perfectly identify each contaminated good in trade flows, so trade-based policies comprising tariffs (to internalize expected damages from imported goods) and inspection regimes (to sort out some contaminated goods) are derived to internalize these externalities.

All of this prior work implicitly assumes that (i) individual importers have neither the ability nor the incentive to mitigate externalities from the importation of potentially contaminated goods and (ii) social damages from biological pollution are determined from unilateral spillovers (i.e., those who generate social risk are not themselves at risk from others' actions). Realistically, however, the risk posed by trade-related biological pollution problems is a "filterable externality" (Shogren and Crocker 1991): an individual importer can reduce disease

spillovers via multiple, private risk management choices. For example, a livestock producer who quarantines newly-imported animals reduces his own disease risks as well as those to neighboring susceptible herds. Producers can also protect others directly via risk-abatement measures such as properly disinfecting shared equipment. Social damages in this context may result from bilateral spillovers (i.e., those who generate social risk are themselves at risk from others' actions), with strategic interactions arising to the extent that a producer's risk management measures simultaneously protect himself and neighbors. Similar issues may arise with biological pollutants other than livestock diseases, such as invasive species that adversely affect the nursery industry (Knowler and Barbier 2005).

The myriad choices involved in generating external risks, combined with stochasticity and an inability to observe biological pollution at an acceptable cost, suggest the problem of managing trade-related biological pollution risks is analogous to nonpoint source pollution problems (Horan et al. 2002). In this context, the importation of contaminated goods and private risk management and abatement activities (e.g., inspection effort, biosecurity to prevent spread) can be thought of as inputs to a biological pollution production function.

The nonpoint source literature suggests efficient pollution management policies can be based on the biological pollution production function (which serves as a proxy for the externality) or else on the inputs to this function (e.g., Griffin and Bromley 1982). Prior work on managing risks from trade has instead focused on trade-based policies. These can be efficient if trade and surveillance choices are the only inputs to the biological pollution production function. However, trade-based policies are inefficient if biological pollution depends on other, non-trade-based inputs such as importers' risk abatement effort.

This paper explores the design of efficient disease prevention policies when importers can mitigate disease risks to others, thereby generalizing prior work (McAusland and Costello 2004; Mérel and Carter 2008; Paarlberg and Lee 1998). Specifically, we reframe the problem of managing disease risks from trade as a nonpoint source pollution problem, following the approach of Horan et al. (2002) and Horan and Lupi (2005a; 2005b). We begin by examining the first-best allocation of disease control efforts in the case of unilateral spillovers. We then compare this allocation with the privately-optimal allocation and derive policy instruments that incentivize first-best disease control efforts. In contrast to prior work, we find the externality extends beyond trade in contaminated goods. We demonstrate that efficient incentive-based mechanisms must target the externality, or each of the choices contributing to the externality, rather than just the trade-related activities. We also show the magnitude of these efficient incentives decreases when producers have greater private risk management incentives and a greater ability to directly protect others.

Next, we consider the case when social damages stem from bilateral disease spillovers between importers. Prior work on trade-related biological pollution has not considered these types of spillovers. In a related study that does not model import decisions, Reeling and Horan (2015) examine bilateral externalities arising from a single biosecurity choice that protects against infection risks from both imports and spread among neighbors. Likewise, Wang and Hennessy (2015) consider the allocation of government and producer effort to prevent and control the spread of disease within a region. These authors consider bilateral spillovers but do not explore efficient policy design. We extend these analyses by allowing for multiple risk management choices, including import decisions, and by focusing on efficient policy design. Within this setting, we find that spillovers between importers may lead to multiple Nash

equilibria in the decentralized outcome. These equilibria include one or more sub-optimal equilibria in addition to the first-best equilibrium, and producer's expectations about their neighbors' strategies determine which outcome is pursued. We show that incentives alone may not be sufficient to achieve efficiency; additional command-and-control policies (e.g., regulations) may also be needed alongside incentives (Anderson and Francois 1997).

2.2 Trade and Infection Risks Under Unilateral Spillovers

We begin by deriving a simple model of trade and infection risks, which we frame in terms of livestock trade for concreteness. Suppose there exists a region that contains a fixed number of livestock importers, indexed by $i = 1, \ldots, N$, each of whom are price takers operating in competitive input and output markets. Each importer purchases a fixed quantity of live animals, X_i, from outside the region to raise on his farm. Each importer may purchase animals from either a risk-free source known to be devoid of infected animals or from a lower-cost risky source where infected animals are present with a known probability. Denote the share of the animals purchased from the risk-free source as $x_i \in [0, 1]$. The probability the importer purchases an infected animal is denoted $P_i^I(x_i)$, where $\partial P_i^I / \partial x_i < 0$ and $P_i^I(1) = 0$. If any imported animal is infected, then we assume all other animals purchased from the risky source will also become infected during transport.

The importer can inspect and quarantine animals purchased from the risky source prior to introducing them into his herd. If an infection is detected, then all imported animals from the risky source, $(1 - x_i)X_i$, will be culled at per-unit cost δ_i to the importer. Denote the probability that infected animals are caught via inspection as $P_i^C(z_i)$, with $\partial P_i^C / \partial z_i > 0$, where $z_i \in [0, 1]$ is importer i's inspection effort. Infected animals that are not caught via inspection are assumed to

infect the importer's entire herd, generating a per-unit cost $\lambda_i > \delta_i$ to the importer. We assume for now that a given importer is not at risk from infection due to neighboring importers' choices.[23] These additional risks are considered later. Taken together, importer *i*'s expected losses from disease are $\Lambda_i(x_i, z_i) = P_i^I(x_i)[P_i^C(z_i)(1-x_i)X_i\delta_i + (1-P_i^C(z_i))X_i\lambda_i]$. The first term in Λ_i is the importer's expected losses given that he imports an infected animal, identifies it via inspection, and has to cull all of the animals imported from the risky region. The second term in Λ_i is the importer's expected losses given that he imports an infected animal but does not detect it, and must therefore cull his entire herd.

Given our specification for importer *i*'s own expected losses, and assuming the importers are risk-neutral, we can define importer *i*'s private net benefits by

$$\pi_i(x_i, z_i, a_i) = B_i - c_i(x_i, z_i, a_i) - \Lambda_i(x_i, z_i). \tag{2.1}$$

The first right-hand-side (RHS) term, B_i, is the net revenue earned by selling the imported animals. The second RHS term, $c_i(x_i, z_i, a_i, b_i) = w(1 - x_i)X_i h_i(z_i) + vx_iX_i, + f_i(a_i)$ represents the importer's costs unrelated to infection. Specifically, the cost of purchasing animals is $w(1 - x_i)X_i$ $h(z_i) + vx_iX_i$, where w and v are the exogenous per-unit costs of animals from the risky and risk-free sources, respectively, with $w < v$. The function $h_i(\cdot) \geq 1$, with h_i', $h_i'' > 0$ and $h_i(0) = 1$ reflects inspection costs. The term $f_i(a_i)$ is the cost of abatement, where $a_i \in [0, 1]$ is abatement effort to reduce the probability of disease spread off the farm. We assume increasing, convex

[23] Spillovers from introduction are unilateral in the sense that the importer's choices impose damages on society, and not the other way around. Examples of such damages include those arising from diseases which spread from an importer's herd into a wildlife reservoir. For instance, bovine tuberculosis (bTb; *Mycobacterium bovis*) was introduced with cattle into Australia and New Zealand and quickly became established in feral deer and possum populations (Tweddle and Livingstone 1994). Exposure to bTb, e.g., via hunting and field-dressing infected deer, can result in tuberculosis in humans (Wilkins et al. 2003). More broadly, the analysis of one-way spillovers is also appropriate for analyzing risks from other invasive species, e.g., salt cedar (*Tamarix spp.*), an invasive shrub that was introduced via the nursery trade and has become widely established in the Southwestern U.S. (Whitcraft et al. 2007).

abatement costs, f_i', $f_i'' > 0$, with $f_i(0) = f_i'(0) = 0$. Our assumptions imply c_i is linear in x_i and increasing and convex in z_i and a_i, with $\partial c_i(x_i, z_i, 0)/\partial a_i = 0$. In what follows, we focus on $c_i(\cdot)$ rather than on its individual components. The profit function is concave in both x_i and z_i.

An infected animal that goes undetected in importer i's herd may transmit the disease to other, non-importing local farms, causing social damage Θ. As indicated above, the importer can invest in abatement effort, a_i, to reduce the probability disease leaves his farm, $P_i^L(a_i)$, with $\partial P_i^L / \partial a_i < 0$. The probability the disease spreads from importer j's herd is then

$$e_j(x_j, z_j, a_j) = P_j^I(x_j)[1 - P_j^C(z_j)]P_j^L(a_j). \tag{2.2}$$

Let $\mathbf{e} = (e_1, \ldots, e_N)$. The probability at least one importer transmits the disease to non-importers, so that damages occur, is then given by

$$P^D(\mathbf{e}) = 1 - \prod_i \left[1 - e_i(x_i, z_i, a_i)\right], \tag{2.3}$$

so that expected social damages from disease spread are $D(\mathbf{e}) = \Theta P^D(\mathbf{e})$. Social net benefits are defined as the sum of the N importers' profits less expected social damages.

2.2.1 The Efficient and Privately-Optimal Outcomes under Unilateral Spillovers

We begin by comparing the privately-optimal and efficient outcomes, which clearly indicate the biological pollution externality extends beyond trade in contaminated goods. We then examine how private risk management activities contribute to the externality, with a particular focus on the relative contribution of trade and non-trade related activities.

The importer's private expected profit maximization problem involves choosing x_i, z_i, and a_i to maximize $\pi_i(\cdot)$. Assuming an interior solution, the importer's private first order conditions (FOCs) are

(2.4) $$\frac{\partial \pi_i}{\partial v_i} = -\frac{\partial c_i}{\partial v_i} - \frac{\partial \Lambda_i}{\partial v_i} = 0, \quad v \in \{x, z, a\}.$$

The FOCs in (2.4) imply the importer's private marginal benefits from disease mitigation equal his private marginal costs at the privately-optimal solution. The importer's privately-optimal choice, v_i^0, $v \in \{x, z, a\}$, is illustrated in Figure 2.1 at the intersection of the curve labeled $\partial c_i/\partial v_i + \partial \Lambda_i/\partial v_i$ and the horizontal axis. Note that $\partial \pi_i/\partial a_i = -c_i' = 0$ (since $\partial \Lambda_i/\partial a_i = 0$), implying $a_i^0 = 0$: the individual importer has no incentive to invest in abatement effort since it does not influence his own disease risks. In contrast, the importer's choices of x_i^0 and z_i^0 are weakly positive since these choices do influence his own disease risks.

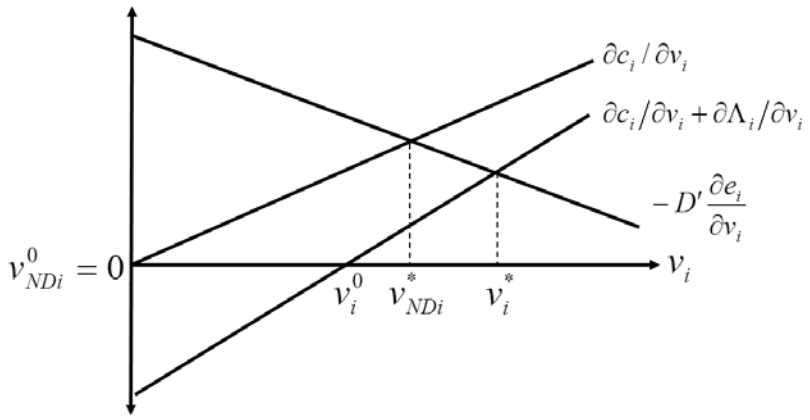

Figure 2.1 Comparison of privately- and socially-optimal risk mitigation

Consider next the efficient outcome, for which the share of risky imports, x_i, inspection effort, z_i, and risk abatement effort, a_i, are chosen to maximize importers' aggregate expected profits less expected social damages. Assuming for simplicity that importers within the region do not have a collective influence on input or output prices, and taking N as fixed (i.e., ignoring the potential for entry and exit), the social planner's problem is

(2.5) $$\max_{x_i, z_i, a_i \forall i} V = \sum_{i=1}^{N} \pi_i(x_i, z_i, a_i) - D(\mathbf{e}).$$

The first-best choices x_i^*, z_i^*, and a_i^* solve the three FOCs

(2.6) $$\frac{\partial V}{\partial v_i} = \frac{\partial \pi_i}{\partial v_i} - D' \frac{\partial e_i}{\partial v_i} = 0$$
$$\Rightarrow \frac{\partial c_i}{\partial v_i} + \frac{\partial \Lambda_i}{\partial v_i} = -D' \frac{\partial e_i}{\partial v_i} \quad \forall i;\ v \in \{x, z, a\}.$$

The second line of condition (2.6) shows that the marginal net private costs of v_i, given by $\partial c_i / \partial v_i + \partial \Lambda_i / \partial v_i$, optimally equals the marginal expected external benefits from i's choice of $v \in \{x, z, a\}$, i.e., the marginal avoided social damages stemming from risk mitigation, $-D'[\partial e_i / \partial v_i]$. As $-D'[\partial e_i / \partial v_i] = -\Theta(\partial P^D / \partial e_i)(\partial e_i / \partial v_i) > 0$, condition (2.6) requires that $\partial c_i / \partial v_i + \partial \Lambda_i / \partial v_i > 0\ \forall v \in \{x,z,a\}$ when evaluated at the first-best outcome. This implies importer i's first-best choice of v, denoted v_i^*, is greater than his privately-optimal choice, v_i^0, which does not account for the marginal expected external benefits of v (Figure 2.1).

Comparison of FOCs (2.4) and (2.6) clearly indicates that externalities exist, where the source of the externality created by importer i is the probability that infection will be transmitted, $e_i(\cdot)$. This function can be thought of as a biological pollution production function that measures the external pressures being generated by the importer, and it is the probabilistic analogue of the pollution production functions used in more traditional nonpoint source pollution models. Note that this function depends only partly on trade decisions, in contrast to prior work that focuses exclusively on trade decisions involving contaminated goods as the externality (McAusland and Costello 2004; Mérel and Carter 2008).

Note also that the magnitude of the externality is reduced when risks are filterable. To see this, consider the extreme case where individual importers do not face disease risks such that Λ_i

= 0 and condition (2.4) becomes $-\partial c_i/\partial v_i = 0\ \forall v \in \{x, z, a\}$, with importer i's privately optimal choice being $v_{NDi}^0 = 0\ \forall v$. The planner's optimality condition (2.6) becomes

$$\frac{\partial V}{\partial v_i} = 0 \Rightarrow \frac{\partial c_i}{\partial v_i} = -D' \frac{\partial e_i}{\partial v_i} \quad \forall i;\ v \in \{x, z, a\}. \tag{2.7}$$

Comparing (2.7) with (2.6) implies that the social optimum occurs at $v_{NDi}^* < v_i^*$, as illustrated in Figure 2.1.

The magnitude of trade-related disease spillovers is smaller when risks are filterable, i.e., $v_{NDi}^* - v_{NDi}^0 = v_{NDi}^* > v_i^* - v_i^0$ in Figure 2.1. Intuitively, importers' private incentives for risk mitigation cause them to make choices that reduce the disease risks they face via their own imports. These choices in turn reduce the throughput of disease risks from the importers' farms to non-importers (i.e., these choices "filter" the biological pollution externality). We show in Appendix B that this result holds generally. Hence, the marginal benefits from mitigation beyond privately-optimal levels are reduced, at least for those activities for which $-\partial \Lambda_i/\partial v_i > 0$. This finding suggests a need to direct a greater policy focus at the margin on inputs for which individuals have fewer or no private incentives to provide on their own (e.g., risk abatement activities), and to reduce the policy focus on inputs where there are greater private incentives to mitigate risk. This result has not arisen in prior work in which trade does not subject individuals to private risks.

2.2.2 Incentives for Efficient Disease Control

We now consider how economic incentives might be designed and implemented to eliminate the externalities described above. Incentives for managing disease transmission would ideally be based on the actual emissions of biological pollutants, but there are difficulties with this approach because disease transmission is stochastic and the actual spread of infection to others is

generally unobservable (at least, without considerable cost to trace the source of infection; Elbakidze 2007).

Disease control incentives must be based on an alternative construct in the face of unobservable emissions. Griffin and Bromley (1982) suggest two alternatives. The first is to base policies on a nonpoint pollution production function that links a firm's choices to emissions, thereby acting as a performance proxy. The analogous construct for biological pollution is the probability of disease transmission, $e_i(\cdot)$. Such a measure can be thought of as a risk-based performance proxy. The second approach is to target the individual choices or inputs that contribute to external infection risks through the use of input-based incentives. Both approaches have been analyzed and applied for the case of invasive species (Horan and Lupi 2005a; Horan and Lupi 2005b). We now examine each in turn.

2.2.2.1 Risk-Based Incentives

Suppose a tax t_i is levied on the probability of disease transmission, e_i, which can be calculated from a predictive model based on observations of the importer's decisions.[24] The importer's problem under this risk-based tax is

$$J_i = \max_{x_i, z_i, a_i} \pi_i(x_i, z_i, a_i) - t_i e_i(x_i, z_i, a_i), \tag{2.8}$$

with FOCs

$$\frac{\partial J_i}{\partial v_i} = \frac{\partial \pi_i}{\partial v_i} - t_i \frac{\partial e_i}{\partial v_i} = 0, \quad v \in \{x, z, a\}. \tag{2.9}$$

[24] Assuming risk neutrality and perfect traceability, an equivalent approach is to charge importers a fine if an outbreak occurs and the infection can be traced back to their trade decisions. The optimal fine will be larger if traceability is imperfect (i.e., if outbreaks can be traced back to the original importer with probability less than one).

Comparison of (2.9) with the social planner's FOCs (2.6) indicates that importer *i*'s optimal choices under the risk-based tax are first-best as long as the tax is set equal to the expected marginal social damages from spread off his farm, i.e., if, for all *i*,

(2.10) $$t_i = D'\big|_{x_i^*, z_i^*, a_i^* \forall i}.$$

Intuitively, taxing the probability that disease spreads from the importer's farm—a proxy for the true externality of disease transmission—incentivizes the importer to internalize the consequences of all of his choices on the disease risks to his neighbors. Note also that efficiency can only be attained if the risk-based tax is specific to the importer: the tax would be overdetermined—and thus at most second-best—if instead a regulatory authority were to apply a uniform tax rate to all importers.

2.2.2.2 Input-Based Incentives

Consider next the outcome under input-based incentives. Specifically, suppose regulators can charge a tariff, τ_i, on all animals purchased from the risky source. Suppose also that the importer receives per-unit subsidies σ_{zi} and σ_{ai} for each unit of inspection effort and abatement effort he undertakes on his farm, respectively.[25] The importer's problem in this case is

(2.11) $$J_i = \max_{x_i, z_i, a_i} \pi_i(x_i, z_i, a_i) - \tau_i(1 - x_i)X_i + \sigma_{zi} z_i + \sigma_{ai} a_i$$

with FOCs

(2.12) $$\frac{\partial J}{\partial x_i} = \frac{\partial \pi_i}{\partial x_i} + \tau_i X_i = 0,$$

[25] The availability of a subsidy for importers' inspection effort assumes that z_i is observable. Alternatively (and equivalently), we could assume that z_i is unobservable but that a benevolent regulatory authority can also provide inspection effort—say, z—in addition to the importer's own z_i. If we assume that z_i and z are perfect substitutes, then the aggregate level of inspection effort $z_i + z$ chosen by the importer and regulator (perhaps according to a Stackelberg game where the importer chooses his optimal z_i given the regulator's z) will be the same as that chosen using an optimally-derived subsidy.

(2.13) $$\frac{\partial J}{\partial z_i} = \frac{\partial \pi_i}{\partial z_i} + \sigma_{zi} = 0,$$

(2.14) $$\frac{\partial J}{\partial a_i} = \frac{\partial \pi_i}{\partial a_i} + \sigma_{ai} = 0.$$

Comparison of (2.12)–(2.14) with (2.6) indicates that input-based incentives can achieve the efficient outcome if τ_i, σ_{zi}, and σ_{ai} are each set such that

(2.15) $$\tau_i = -D' \frac{\partial e_i}{\partial x_i} X_i^{-1} \Big|_{x_i^*, z_i^*, a_i^* \forall i} \quad \forall i,$$

(2.16) $$\sigma_{zi} = -D' \frac{\partial e_i}{\partial z_i} \Big|_{x_i^*, z_i^*, a_i^* \forall i} \quad \forall i,$$

(2.17) $$\sigma_{ai} = -D' \frac{\partial e_i}{\partial a_i} \Big|_{x_i^*, z_i^*, a_i^* \forall i} \quad \forall i.$$

The first-best instruments equal the marginal social damage from the externality, D', multiplied by the marginal effectiveness of each input in mitigating the externality, $\partial e_i/\partial v_i$ for $v \in \{z,a\}$ and $(\partial e_i/\partial v_i)/X_i$ for $v = x$ (i.e., the marginal product of $v \in \{x, z, a\}$ in the biological pollution production function). Our earlier results on filterable externalities (see Figure 2.1) suggest the first-best instruments will be smaller the larger are importers' private incentives for risk mitigation and abatement activities because the magnitude of the externality will be smaller. Note also that the first-best instruments must be applied to each input contributing to e_i.

These results differ from the literature on trade in goods potentially contaminated with invasive species (e.g., McAusland and Costello 2004; Mérel and Carter 2008; Paarlberg and Lee 1998) in two ways. First, this prior work does not consider non-trade inputs to the biological pollution production function. Failure to provide incentives for non-trade inputs (e.g., because these inputs are unobservable) means their levels will be set to zero at the private optimum (i.e.,

$a_i = 0$), and hence biological pollution control can be at most second-best. It is straightforward to show the second-best τ_i and σ_{zi} are the same as those in (2.15) and (2.16), except a_i is evaluated at zero. It is unclear whether the second-best incentives are larger or smaller than their first-best counterparts, but these instruments will generally distort trade flows and private incentives for surveillance relative to the first-best outcome.

Second, prior work finds uniform incentives to be efficient due to a focus on a single, representative importer. We show efficiency requires the instruments be applied at importer-specific rates since each choice has an importer-specific marginal contribution to e_i, which in turn has an importer-specific marginal contribution to D. In the present context, uniform incentives will result in further efficiency losses as importers with a small effect on expected social damages will receive incentives that are too large, and vice versa (Shortle, Abler and Horan 1998). The nonpoint source pollution literature indicates gains can be made from better targeting the design of pollution control incentives (e.g., Babcock et al. 1997; Westra, Easter and Olson 2002); the same is likely to be true for biological pollution.

2.3 Trade and Infection Risks under Bilateral Spillovers

We now extend the model to consider the case in which disease spillovers are bilateral in the sense that an importing producer's herd is also at risk of becoming infected via spread from other importers. We show that alternative, command-and-control policies may be needed alongside of economic incentives to achieve efficiency.

Suppose importer i's herd can be infected via spread from any of the $N - 1$ neighboring importers' herds, should they become infected. Importer i can protect his own herd from this

spread of infection by investing in biosecurity effort $b_i \in [0, 1]$.[26] We can then write the probability importer i's herd becomes infected from any of the $N - 1$ neighboring importers as

$$P_i^O(b_i;\mathbf{e}_{-i}) = P_i^S(b_i)\left(1-\prod_{j\neq i}\left[1-e_j(x_j,z_j,a_j)\right]\right), \tag{2.18}$$

where $\mathbf{e}_{-i} = (e_1, \ldots, e_{i-1}, e_{i+1}, \ldots, e_N)$ and $P_i^S(b_i)$ is the probability the disease becomes established in i's herd, conditional on spread from at least one of his neighbors. We assume $\partial P_i^S(b_i)/\partial b_i < 0$, so P_i^O is decreasing in each argument. Importer i's expected disease losses are

$$\begin{aligned} &\underbrace{P_i^I\left[P_i^C(1-x_i)X_i\delta_i + (1-P_i^C)X_i\lambda_i\right]}_{\Lambda_i} \\ &+\underbrace{\left(P_i^I P_i^C\left[X_i\lambda_i - (1-x_i)X_i\delta_i\right] + (1-P_i^I)X_i\lambda_i\right)P_i^O}_{\Omega_i}, \end{aligned} \tag{2.19}$$

where function arguments are omitted for conciseness. The term Λ_i again represents importer i's expected losses stemming from his own choices. The term Ω_i—which does not arise with unilateral spillovers—is importer i's expected losses resulting from others' choices. The first term in Ω_i is the importer i's expected losses given that he imports an infected animal and identifies it via inspection, but is infected via spread anyway. The second term in Ω_i is the importer's expected losses given that he does not import an infected animal, but is infected via spread anyway. We continue to assume importers can spread the disease to other, non-importing producers, causing expected damage $D(\mathbf{e})$ that are in addition to the damages to neighboring importers (i.e., the Ω_i terms).

Taken together, importer i's expected profits can be written

$$\pi_i(x_i,z_i,a_i,b_i;\mathbf{e}_{-i}) = B_i - c_i(x_i,z_i,a_i,b_i) - \Lambda_i(x_i,z_i) - \Omega_i(x_i,z_i,b_i;\mathbf{e}_{-i}), \tag{2.20}$$

[26] We assume the actions that comprise b_i are distinct from the actions that comprise a_i, which only protect neighboring herds.

where $c_i(\cdot)$ takes the same form as in (2.4) except that it now also includes biosecurity costs, which we denote as $g_i(b_i)$, with $\partial g_i/\partial b_i > 0$.

We can simplify the remaining analysis by rewriting (2.20) as a restricted profit function in b_i and e_i: $\pi_i^R(e_i, b_i; \mathbf{e}_{-i})$ (see Appendix B for derivation). This means e_i will be treated as a choice in what follows, which is common in pollution problems (e.g., Tietenberg 1985).

2.3.1 The Efficient and Privately-Optimal Outcomes under Bilateral Externalities

The inefficiency arising from biological pollution under bilateral spillovers can again be seen by comparing the efficient and privately-optimal outcomes. Importer *i*'s private problem is to maximize his expected profits, π_i^R. This problem is solved as a Nash-Cournot game, i.e., b_i and e_i are chosen to maximize expected profits, taking the externalities generated by other importers ($\mathbf{e}_{-i}$) as given. Formally, importer *i*'s problem is $\max_{b_i, e_i} \pi_i^R(b_i, e_i; \mathbf{e}_{-i})$. Assume for now that a unique Nash equilibrium arises as an interior solution, with the privately-optimal choices, b_i^0 and e_i^0, solving [27]

$$\frac{\partial \pi_i^R}{\partial b_i} = 0 \tag{2.21}$$

$$\frac{\partial \pi_i^R}{\partial e_i} = 0. \tag{2.22}$$

Conditions (2.21) and (2.22) imply the familiar result that marginal expected net benefits from self-protection and emitting biological pollution, respectively, are zero.

[27] Existence of at least one Nash equilibrium follows from a straightforward application of Brouwer's Fixed Point Theorem (Mas-Colell et al. 1995).

Consider next the social planner's problem, which is to choose b_i and e_i $\forall i$ to maximize aggregate importer profits less damages to non-importers. Formally, the social planner's problem is $\max_{b_i, e_i \forall i} \sum_i \pi_i^R(b_i, e_i; \mathbf{e}_{-i}) - D(\mathbf{e})$. Assuming an interior solution, the first-best choices, b_i^* and e_i^*, solve the necessary conditions

$$\frac{\partial \pi_i^R}{\partial b_i} = 0 \tag{2.23}$$

$$\frac{\partial \pi_i^R}{\partial e_i} + \sum_{j \neq i} \frac{\partial \pi_j^R}{\partial e_i} - D' = 0 \quad \forall i, \tag{2.24}$$

where the term $\sum_{j \neq i} \partial \pi_j^R / \partial e_i$ reflects expected marginal losses to neighboring importers due to spread from importer *i*'s herd. Comparing (2.23) and (2.24) with (2.21) and (2.22) shows that—as with the externality to non-importers—the externality to importers arises from e_i. In contrast, importers will freely choose b_i^0 at the first-best level whenever policies are implemented to ensure $e_i^0 = e_i^*$. The reason is that b_i does not affect the production of biological pollution, and hence no spillovers arise from its under-provision. However, b_i^0 will be inefficiently large when $e_i^0 > e_i^*$.

2.3.2 Incentives for Efficient Disease Control with Bilateral Externalities

Consider how economic incentives might be designed and implemented to eliminate the bilateral externalities described above. Efficiency can be attained through either risk- or input-based incentives, as in the unilateral case. For conciseness, we derive only the optimal risk-based tax; deriving the optimal input-based incentives provides little additional insight beyond that discussed for the unilateral case.

Suppose a tax, t_i, is levied on importer i if disease is transmitted from his farm, so that the risk-based tax payment for importer i is $t_i e_i$. Importer i's problem under this risk-based tax is $\max_{b_i, e_i} \pi_i^R(b_i, e_i; \mathbf{e}_{-i}) - t_i e_i$, with necessary conditions

$$\frac{\partial \pi_i^R}{\partial b_i} = 0 \tag{2.25}$$

$$\frac{\partial \pi_i^R}{\partial e_i} - t_i = 0, \tag{2.26}$$

Comparing (2.26) to (2.24) shows that efficiency can be attained by setting

$$t_i = \sum_{j \neq i} \frac{\partial \Omega_j}{\partial e_i} + D' \Big|_{b_i^{**}, e_i^{**} \forall i} \quad \forall i, \tag{2.27}$$

which is similar to the tax rate identified in (2.10) except that now the optimal tax rate incentivizes importers i to internalize the external marginal damages from his choices to neighboring importers and non-importers.

2.3.3 Efficient Incentives in the Presence of Multiple Equilibria

The foregoing assumes the Nash equilibrium of the importer's problem is unique. This need not be the case in general, as prior work suggests trade-related choices (e.g., import and surveillance decisions) may result in multiple Nash equilibria (Hennessy 2008; Reeling and Horan 2015). Multiple Nash equilibria arise from non-convexities brought on by strategic complementarities between producers' self-protection choices (i.e., an importer's marginal incentives for self-protection increase with his neighbors' self-protection). We show in Appendix B that strategic complementarities between e_i and e_{-i} may exist both before and after implementation of the efficient risk-based tax.

The existence of multiple equilibria complicates incentive design due to the potential for coordination failure. Specifically, which equilibrium outcome arises depends on importers' expectations regarding their neighbors' choices. If enough importers believe their neighbors will make choices consistent with the Pareto dominant outcome, which is the efficient equilibrium in the case of an efficient tax (Milgrom and Roberts 1990; Vives 2005), then the system will converge to this outcome. Coordination failure arises when an insufficient number of importers' expect neighbors will make efficient choices, so that the system instead equilibrates at a Pareto-dominated equilibrium exhibiting lower risk mitigation levels.

The problem of coordination failure is formalized as follows. Suppose there are multiple roots to the importer's necessary conditions in (2.25) and (2.26), resulting in an efficient, Pareto-dominant Nash equilibrium, denoted $(\mathbf{b}^*, \mathbf{e}^*) = ((b_1^*, e_1^*), ..., (b_N^*, e_N^*))$, and $K > 1$ Pareto-dominated Nash equilibria, $(\mathbf{b}_k^\#, \mathbf{e}_k^\#) = ((b_{1k}^\#, e_{1k}^\#), ..., (b_{Nk}^\#, e_{Nk}^\#)) > (\mathbf{b}^*, \mathbf{e}^*)$ for $k \in \{1, \ldots, K\}$. Prices alone cannot generally ensure the system converges to the efficient equilibrium in the presence of multiple equilibria and coordination failure risks (Dasgupta and Mäler 2003), which means importers may instead arrive at one of the Pareto-dominated outcomes.

Following Anderson and Francois (1997), the efficient outcome can be ensured through a combination of the efficient risk-based tax in (2.27) and command-and-control regulations on e_i. Specifically, suppose a regulatory authority can set and enforce a maximum allowable e_i, say, $\bar{e}_i$. $> e_i^*$.[28] Efficiency can then be attained by setting $\bar{e}_i$ sufficiently close to e_i^*, with $\bar{e}_i < e_{ik}^\#$ for $e_{ik}^\# \leq e_{ik''}^\#$ $\forall k' = 1, \ldots, K$. Importers will then expect their neighbors to make efficient decisions. In

[28] In practice, these regulations may be achieved through restrictions on the inputs to e_i. Specifically, suppose that the regulator can set and enforce regulations designating a minimum share of animals, $\underline{x}_i$, that can be purchased from the risk-free region. Suppose also the regulator can provide supplemental surveillance, $\underline{z}_i$, of all animals from the risky region whenever $x_i \neq 1$. If we let $\bar{e}_i = e_i(\underline{x}_i, \underline{z}_i, a_i)$, then these input-based regulations have the same effect as placing a maximum bound on e_i.

other words, this regulation eliminates importers' expectations that can lead to coordination failure: all Pareto-dominated equilibria are precluded such that the only remaining equilibrium coincides with the efficient outcome. Note also that the regulation $\overline{e_i}$ is non-binding in the sense that importers will have private incentives to reduce e_i beyond this level.

Our results contrast with prior literature in managing trade-related biological pollution spillovers (e.g., McAusland and Costello 2004; Mérel and Carter 2008; Paarlberg and Lee 1998) by showing that incentives alone may not achieve efficiency in the presence of bilateral spillovers. Alternative policy interventions (e.g., regulations) may be needed in addition to price-based incentives to achieve efficiency. This finding also contrasts with the nonpoint source pollution literature (e.g., Griffin and Bromley 1982), which finds regulations or incentives can be used interchangeably to achieve efficiency. The difference here arises from the fact that importers' disease risks depend on choices made by neighboring importers, leading to strategic interactions that may produce non-convexities and the potential for multiple equilibria; in contrast, the nonpoint literature typically does not assume others' pollution enters the emitter's payoff function.

2.4 Discussion and Conclusion

Trade-related disease externalities impose considerable economic costs on society, yet the drivers of these externalities are not well understood. Prior work focuses primarily on trade-related decisions (e.g., import and surveillance choices), which conflates the externality problem with a trade problem. However, other decisions are also relevant. In particular, individual importers' private risk abatement choices also contribute to disease externalities. Prior work has not considered these choices.

We reframe the issue of trade and the spread of an infectious disease as a biological pollution problem, akin to a classic nonpoint source pollution problem. In this context, we show spillovers depend both on trade decisions and on importers' behaviors and choices once infected animals enter the importing region. Disease spillovers are analogous to those arising from more traditional nonpoint source pollutants, suggesting the spread of disease via animal movements is better viewed as a pollution problem rather than a trade problem. This finding casts importers as the perpetrators of biological pollution externalities, rather than the victims, suggesting a greater role for disease control and prevention efforts within an importing region. This contrasts with prior work in which the onus is on keeping infected animals and goods from entering the region in the first place (McAusland and Costello 2004; Mérel and Carter 2008; Paarlberg and Lee 1998).

We use our approach to derive first-best incentives in the face of unilateral spillovers comprising importer-specific incentives on either (i) the probability disease spreads from his or her farm or (ii) on all choices that contribute to the probability of spread. The optimal incentives are smaller the greater are the importer's private incentives for risk mitigation; hence, policy interventions will have greater marginal benefits when individuals have fewer incentives for private behavior. This result does not arise in prior work that focuses primarily on the choices of a single, representative importer.

We also show that multiple equilibria—and the risk of coordination failure—may arise when spillovers are bilateral. Additional command-and-control policies (e.g., regulations) may be needed alongside incentives to avoid coordination failure and suboptimal disease outcomes. Prior work in both the trade and nonpoint source pollution literature does not consider bilateral spillovers, and so policy recommendations do not reflect the potential for coordination failure.

Our model of biological pollution is deliberately simple; disease spillovers are viewed as static, with non-spatial interactions between at-risk individuals. This approach is consistent with—and allows us to glean insights from—the traditional nonpoint source literature (e.g., Griffin and Bromley 1982). Our results are likely to be relevant in more complex settings, including those where disease spillovers are dynamic. To see this, note that in a dynamic setting the importation of an infected animal and disease transmission from the importer's herd (the actual externality) occur sequentially in distinct time periods. This might suggest policy intervention is only required to prevent transmission in the latter period. However, the probability of transmission at any given time will depend on the stock of infected animals, which in turn depends on trade choices made in a previous period. Disease spillovers therefore depend on both trade choices and private risk abatement choices (e.g., biosecurity) in a dynamic setting, as in our static model.

Optimal policy instruments are derived here under the implicit assumption that non-uniform instruments can be imposed on importers. In reality, significant information asymmetries due to unobservable private disease mitigation choices may preclude the use of non-uniform incentives. International trade agreements that prohibit discriminatory policy instruments are likely to further impede the use of importer-specific tariffs. Biological pollution control will be at most second-best in the face of informational and policy constraints. We leave the extension of second-best instrument design for future work.

CHAPTER 3 INTEGRATING POLLUTION MARKETS ACROSS ENVIRONMENTAL MEDIA: THE CASE OF REACTIVE NITROGEN

3.1 Introduction

An intriguing feature of some environmental markets is the ability to trade across pollutants. This is particularly common in greenhouse gas (GHG) emission markets like California's Cap-and-Trade Program (CA AB 32). Under these "multipollutant markets," a regulated polluter that abates emissions of nitrous oxide (N_2O), for example, can sell its corresponding permits to another regulated polluter needing to offset its CO_2 emissions subject to an inter-pollutant exchange rate. Provisions for multipollutant trading have also existed for local air quality markets (e.g., California's South Coast Air Quality Management District allows new emissions sources to offset their emissions by paying for reductions in emissions of different air pollutants on a case-by-case basis; see Rule 1309 (h)), although these provisions have rarely, if ever, been exploited (Montero 2001) .

Prior work focuses largely on identifying optimal policy parameters for inter-pollutant exchange in greenhouse gas or air quality trading programs (e.g., Reilly and Richards 1993; Muller and Mendelsohn 2009) or water quality programs (e.g., Hung and Shaw 2005). Other work has demonstrated that trading across pollutants or other environmental services can increase social welfare relative to having separate markets. For example, Montero (2001) uses a simple but general theoretical model to demonstrate that integrated markets can be welfare-enhancing relative to separate markets when abatement cost curves are relatively steeper than marginal benefit curves. Horan, Shortle, and Shogren (2015) consider the case of a fishery where productivity is degraded by both overfishing and pollution. They show that a market in which

fishermen and polluters trade harvest rights for the right to pollute and vice versa can achieve a first-best allocation of fishing and pollution abatement.

Consideration of multipollutant trading has—both in theory and practice—been restricted to different pollutants that all affect the same resource or environmental medium.[29] However, recent advances in our understanding of pollutant generation, transport, and fate have increased our knowledge of the linkages between environmental pollutants across environmental media. A striking example involves reactive nitrogen (Nr), a chemical form of nitrogen that is readily converted to different species (i.e., different forms of Nr, such as NO_X, N_2O, NO_3). A single molecule of Nr released to the environment from disparate activities like agricultural fertilizer use, fossil fuel combustion, or wastewater treatment can travel through multiple environmental media (Galloway et al. 2003), causing environmental and economic damage in each medium it passes through (Birch et al. 2011). The purpose of this essay is to examine multi-pollutant trading when the pollutants impact different media and when one source (agriculture) is a significant contributor to the various pollutants and hence affects multiple media.

Several features of Nr pollution suggest there may be gains from trading across pollutants (i.e., the different Nr species) and environmental media via integrated markets. First, fluxes of different Nr species like N_2O (a potent GHG with 300 times the global warming potential of CO_2) and NO_3 (a water pollutant that contributes to eutrophication and hypoxia) are generated as complements from agricultural production choices like nitrogen fertilizer application. Another justification arises from the vast heterogeneity in the costs and benefits of abatement of different Nr species. For example, Birch et al. (2011) find the economic damage per unit of Nr emitted in the Chesapeake Bay Watershed can vary by a factor of 100 across the various Nr species, and the

[29] Montero's (2001) model is highly general, but was developed in the context of air pollution trading.

marginal abatement costs can vary by a factor of 20 across Nr sources. This heterogeneity in the costs and benefits of Nr abatement suggests integrated markets may decrease the cost of pollution control by offering regulated polluters additional flexibility in meeting their abatement targets. A final justification for integrated markets lies in the highly mobile nature of Nr, which suggests it may not matter from a biogeochemical perspective in which environmental medium Nr abatement occurs, only *that* abatement occurs (Galloway et al. 2003).[30]

Whether—and to what degree—integrated markets are an improvement over separate markets is an empirical question. Prior work on the design of pollution control policies involving multiple pollutants suggests market design choices, especially trade exchange ratios defining how pollutants substitute for one another and the level of emissions caps for each pollutant, will determine the gains from a more integrated approach to pollutant trading (Woodward 2011; Muller 2012). However, emissions caps are often not chosen independently of permit trading programs involving agriculture—let alone programs that might allow trade across pollutants. For instance, agricultural emissions are generally not regulated unless farmers voluntarily participate in a program that pays for abatement. In contrast, point sources are generally regulated, such as through National Permit Discharge Elimination System (NPDES) permits for water emissions. Existing and proposed permit markets involving agriculture are designed to allow point sources to achieve their permit levels by purchasing credits generated via nonpoint source abatement. Within this setting, it is unlikely that caps would be chosen efficiently absent coordination of the

[30] We do not model this feature of Nr emissions for simplicity and because empirical estimates of Nr interconversion rates between species are not well-known, but note only that abatement of Nr fluxes in one environmental medium will ultimately influence fluxes of Nr to other media.

distinct regulatory agencies that may manage different Nr fluxes to different environmental media.[31]

Given sub-optimal pollutant caps, integrated markets provide an additional policy parameter that may be used to improve the performance of environmental management: an interpollutant exchange rate, or the rate at which abatement of one pollutant can be substituted for abatement of another pollutant. Prior work has attempted to estimate efficient interpollutant exchange rates among a single class of pollutants (e.g., greenhouse gases; Reilly and Richards 1993), but no prior work considers rules for exchanging jointly produced pollutants across different environmental media.

The purpose of this essay is to investigate whether integrated markets improve the efficiency of Nr control relative to separate pollutant- and media-specific markets when permit caps are set independently from the trading rules (e.g., the trade ratio). Using an analytical and numerical model of Nr trading in the Susquehanna River Basin (SRB), the largest source of nutrient inputs to the Chesapeake Bay Watershed in the Mid-Atlantic U.S., we show that integrated markets improve the efficiency of pollution control relative to separate markets when pollution caps are set arbitrarily. Greater economic performance arises under integrated markets from increased flexibility in meeting pollution targets.

The following section develops an analytical model of Nr pollution. The next two sections derive the conditions for efficient Nr abatement and shows only integrated markets that allow multi-pollutant trading can be cost-effective in general. We then develop a numerical

[31] A piecemeal approach to environmental regulation is common (Yaffee 1997). In some cases, a single pollutant is managed by different agencies. For example, NO_3 emissions from point sources in the United States are regulated by the USEPA under the Clean Water Act, but NO_3 emissions from nonpoint sources are typically managed via conservation programs run by the USDA.

application of interpollutant trading in the SRB. Numerical results are discussed, and the final section concludes.

3.2 A Model of Nr Pollution

Suppose for simplicity there exist three polluting sectors, each of which emits a different species of Nr.[32] The first sector comprises point source emitters of N_2O to the atmosphere and is referred to heuristically as the industrial sector (indexed by *I*). The second sector comprises point source emitters of NO_3 to aquatic ecosystems and is referred to heuristically as the wastewater treatment sector (indexed by *W*). The final sector comprises nonpoint source emitters of both N_2O to the atmosphere and NO_3 to aquatic ecosystems and is referred to heuristically as the agricultural sector (indexed by *A*).

We assume for simplicity that pollution by all sources is deterministic.[33] Point sources choose their pollution levels directly; denote N_2O emissions by the industrial sector as e_I and NO_3 loadings (or emissions that are delivered to a water body) by the wastewater treatment sector as r_W. In contrast, the agricultural sector generates both N_2O and NO_3 via the use of a vector of polluting inputs, $\mathbf{z}$; agricultural emissions and loadings are thus $e_A = g_e(\mathbf{z})$ and $r_A = g_r(\mathbf{z})$, respectively. Moving forward, it will be simpler to work with abatement, rather than pollution levels. Define abatement by the industrial sector as $a_{eI} = e_{I0} - e_I$, where the subscript "0" denotes initial emissions prior to abatement. Likewise, wastewater treatment abatement is $a_{rW} = r_{W0} - r_W$,

[32] We restrict our focus on NO_3 and N_2O for simplicity, but note that the model can easily be generalized to accommodate additional Nr species, including NO_X and NH_3.

[33] More realistically, farmers choose a vector of inputs that influences *the distribution of their pollution*, since emissions and loadings from nonpoint sources are random functions of weather and other factors (Shortle and Dunn 1986). We assume for now that farm emissions are deterministic to focus attention on the issue of market integration. We consider an extended model with uncertainty later.

and agricultural emissions and loadings abatement are $a_{eA} = e_{A0} - e_A$ and $a_{rA} = r_{A0} - r_A$, respectively.

The industrial and wastewater treatment sectors can abate their emissions by investing in pollution control technologies. The industrial abatement cost function is $C_I(a_{eI})$, where $C_I', C_I'' > 0$. The water treatment sector's abatement cost function is defined analogously as $C_W(a_{rW})$. The agricultural sector abates their emissions and loadings by altering input use. We specify the agricultural sector's abatement cost function as

$$C_A(a_{eA}, a_{rA}) = \min_{\mathbf{z}} \pi(\mathbf{z}_0) - \pi(\mathbf{z}) \text{ subject to } a_{eA} \leq g_e(\mathbf{z}),\ a_{rA} \leq g_r(\mathbf{z}), \tag{3.1}$$

with $\partial C_A/\partial a_{sA}$, $\partial^2 C_A/(\partial a_{eA})^2 > 0\ \forall\ s \in \{e, r\}$. We follow Woodward (2011) and assume complementarity in abatement, i.e., $\partial^2 C_A/\partial a_{eA}\partial a_{rA} < 0$.

Finally, suppose pollution abatement prevents economic damage that varies with the aggregate abatement of each pollutant. Let $E_e = e_{I0} - a_{eI} + e_{A0} - a_{eA}$ and $E_r = r_{W0} - a_{rW} + r_{A0} - a_{rA}$ represent the ambient concentration of N_2O and NO_3, respectively.[34] Aggregate abatement of N_2O and NO_3 is then $a_e = E_{e0} - E_e$ and $a_r = E_{r0} - E_r$, where the subscript "0" denotes unregulated pollution (i.e., with zero abatement). The benefit from abating each species—expressed as avoided economic damage—is then denoted $B_e(a_e) = D_e(E_{e0}) - D_e(E_e)$ and $B_r(a_r) = D_r(E_{r0}) - D_r(E_r)$, where D represents economic damages from ambient pollution.

[34] We assume uniform mixing of pollutants for simplicity. Note also that, by defining r as loadings we already account for spatial heterogeneity in NO_3 delivery across sources.

3.3 First-Best Nr Control

We first consider the efficient, or first-best, allocation of pollution control effort as a baseline for comparison with market outcomes. The efficient outcome is defined as an allocation of pollution control effort that maximizes social net benefits

$$\max_{a_{eI},a_{rW},a_{eA},a_{rW}} V = B_e(a_e) + B_r(a_r) - C_I(a_{eI}) - C_W(a_{rW}) - C_A(a_{eA}, a_{rA}). \tag{3.2}$$

Assuming an interior solution, the first-order conditions (FOCs) for problem (3.2) are

$$\frac{\partial V}{\partial e_I} = 0 \Rightarrow C_I' = B_e', \tag{3.3}$$

$$\frac{\partial V}{\partial r_W} = 0 \Rightarrow C_W' = B_r', \tag{3.4}$$

$$\frac{\partial V}{\partial e_A} = 0 \Rightarrow \frac{\partial C_A}{\partial a_{eA}} = B_e', \tag{3.5}$$

$$\frac{\partial V}{\partial r_A} = 0 \Rightarrow \frac{\partial C_A}{\partial a_{rA}} = B_r' \tag{3.6}$$

The FOCs (3.3)–(3.6) state the familiar result that, at the first-best abatement levels a_{eI}^*, a_{rW}^*, a_{eA}^*, and a_{rA}^*, each agent's marginal abatement costs equal the marginal benefits from abatement.

Additional insight can be had by manipulating (3.3)–(3.6) to yield the following modified equi-marginal condition

$$\frac{C_I'}{B_e'} = \frac{\partial C_A / \partial a_{eA}}{B_e'} = \frac{C_W'}{B_r'} = \frac{\partial C_A / \partial a_{rA}}{B_r'}, \tag{3.7}$$

so that the effective marginal cost of abatement—measured by the marginal cost normalized by the marginal avoided damages from abatement—is equalized across all sources under the efficient outcome. Note that this assumes the damages caused by each pollutant are fungible: the social planner treats avoided damages from emissions abatement as a perfect substitute for

avoided damages from loadings abatement. This contrasts with current approaches to environmental regulation which treat abatement of distinct pollutants (e.g., NO_2 and NO_3) as non-substitutable, even when those pollutants arise from the same source.

3.4 Nr Abatement under Market Trading Schemes

We now consider the outcome under two alternative Nr trading schemes. The first considers the outcome under separate markets for each pollutant and reflects current approaches to Nr management. We then compare this with the outcome under an integrated market that allows trading across environmental media via multipollutant markets.

3.4.1 Separate NO_3 and N_2O Markets when Caps are Chosen Efficiently

Assume N_2O emissions and NO_3 loadings are regulated separately by different regulatory agencies that coordinate in setting permit levels, or emissions caps, for each Nr species to promote efficiency. Denote emissions permits by $\hat{e}_I$, with each permit representing the right to pollute a single unit of N_2O. Likewise, denote loadings permits that cap NO_3 by $\hat{r}_W$. Let the initial emissions permits allocated to the industrial sector be $\hat{e}_{I0}$ and the initial loadings permits allocated to the wastewater treatment sector be $\hat{r}_{W0}$. In contrast, the agricultural sector is endowed with a fixed quantity of offsets corresponding to its unregulated emissions and loadings: $\hat{e}_{A0} = e_A(\mathbf{z}_0)$ and $\hat{r}_{A0} = r_A(\mathbf{z}_0)$, where $\mathbf{z}_0$ is their input choice vector prior to abatement. Point sources are regulated such that they must hold one permit or offset for every unit of emissions or loadings they emit.[35] In contrast, farmers have an implicit right to pollute, but can voluntarily supply

[35] In practice, trades between point and nonpoint sources would be subject to a species-specific trading ratio τ^s. Point sources of s must purchase τ^s permits from nonpoint sources to offset one unit of their own emissions. The trading ratio reflects imperfect substitutability between point and nonpoint source abatement due to stochastic nonpoint source emissions (Malik et al. 1993). We assume perfect substitutability here to focus our attention on substitution across pollutants, but note that our model could be generalized to account for imperfect substitution.

offsets for each pollutant to point source polluters.

The price of emissions and loadings permits are denoted p^e and p^r, respectively. The industrial sector chooses a_{eI} minimize abatement costs, less the cost of purchasing offsets from the agricultural sector. Prior work (e.g., Horan and Shortle 2005) has shown that this problem can be formulated as

(3.8) $$\min_{a_{eI}} C_I(a_{eI}) + p^e[e_{I0} - a_{eI} - \hat{e}_{I0}]$$

Substituting the constraint into the objective function and assuming an interior condition, the FOC for problem (3.8) implies

(3.9) $$C_I' = p^e.$$

Condition (3.9) simply states that, at the optimum, the marginal cost of abatement by the industrial sector (left-hand side [LHS] term) equals the marginal cost of an agricultural offset. The wastewater treatment sector's problem is analogous to the industrial sector's; the cost-minimizing level of abatement satisfies

(3.10) $$C_W' = p^r.$$

The interpretation of (3.10) is analogous to that of (3.9).

The agricultural sector's problem is to choose a_{eA} and a_{rA} to minimize abatement cost less revenue from offset sales:

(3.11) $$\min_{a_{eA}, a_{rA}} C_A(a_{eA}, a_{rA}) - p^e a_{eA} - p^r a_{rA}.$$

The FOCs imply

(3.12) $$\frac{\partial C_A}{\partial a_{eA}} = p^e,$$

(3.13) $$\frac{\partial C_A}{\partial a_{rA}} = p^r,$$

which state that the marginal abatement cost (LHS terms in (3.12) and (3.13)) equals the marginal permit revenues for each species (right-hand side [RHS] terms) at the optimal abatement levels.

Conditions (3.9), (3.10), (3.12) and (3.13), along with the following market-clearing conditions that require post-trade emissions and loadings equal the caps on these pollutants:

$$\hat{e}_{I0} + \hat{e}_{A0} \geq (e_{I0} - a_{eI}) + (e_{A0} - a_{eA}) \text{ and } \hat{r}_{W0} + \hat{r}_{A0} \geq (r_{W0} - a_{rW}) + (r_{A0} - a_{rA}),$$

form the equilibrium system under separate markets. The separate market equilibrium comprises a vector of prices, p^{eS} and p^{rS} and pollution abatement allocations, a_{eI}^S, a_{eA}^S, a_{rW}^S, and a_{rA}^S, that satisfy this system (where the superscript "S" denotes the separate market outcome). Comparing conditions (3.9), (3.10), (3.12) and (3.13) with the efficient FOCs in (3.3)–(3.6) shows the first-best abatement arises when the pollutant caps for emissions and loadings are chosen such that $p^e = B_e'|_{a_{eI}^*, a_{eA}^*}$ and $p^r = B_r'|_{a_{rW}^*, a_{rA}^*}$.[36] Specifically, let $E_e^* = (e_{I0} - a_{eI}^*) + (e_{A0} - a_{eA}^*)$ and $E_r^* = (r_{W0} - a_{rW}^*) + (r_{A0} - a_{rA}^*)$ be the efficient levels of total emissions and loadings, respectively. Assuming agricultural sources are not initially regulated so that they have implicit initial permit caps of e_{A0} and r_{A0}, then the efficient permit caps for the emissions and loadings by the industrial and wastewater treatment sectors, respectively, are $\hat{e}_{e0}^* = E_e^* - e_{A0}$ and $\hat{r}_{W0}^* = E_r^* - r_{A0}$.

[36] We assume that unregulated emissions by the agricultural sector are sufficiently small that the caps on point sources can, in fact, be chosen such that the market operates efficiently. Generally, the larger are unregulated agricultural sector emissions, the smaller must be the initial permit allocations to the industrial and wastewater treatment sectors to attain efficiency.

3.4.2 An Integrated Nr Market when Caps are Chosen Efficiently

Now consider the case in which the markets for each species are integrated so that regulated emitters of either species are allowed to offset their emissions by purchasing permits or offsets representing either N_2O or NO_3. We continue to assume the agency can choose permit caps to promote market efficiency. These caps can be set as in non-integrated markets.

The first point that must be addressed in designing an integrated Nr market is the commodity to be traded. Without loss of generality, we choose emissions of N_2O as a numeraire. Let τ denote the inter-pollutant exchange rate governing trades between emitters of NO_3 and N_2O. As before, denote the prices of emissions and loadings as p^e and p^r. The total pollutant cap—denominated in units of N_2O—will then be $\hat{e}_{I0} + \hat{e}_{A0} + [\hat{r}_{W0} + \hat{r}_{A0}]/\tau$.

The industrial sector's problem is again to minimize the cost of abating emissions. In contrast to the separate-market case above, the constraint on the industrial sector's emissions can be satisfied by holding permits or offsets for either emissions or loadings, as long as the industrial sector holds $1/\tau$ loading permits or offsets for every unit of N_2O they emit. The wastewater treatment sector's problem is analogous to the industrial sector's problem. The constraint on this sector's loadings can be satisfied by holding permits or offsets for either emissions or loadings, as long as it holds $1/\tau$ loading permits or offsets for every unit of NO_3 they emit. Agricultural emissions and loadings are only constrained by the offsets that they sell.

Prior work on trades involving a trade ratio (e.g., Horan and Shortle 2005) indicate that the following condition must hold in a market equilibrium,

$$p^r = \frac{p^e}{\tau} \Rightarrow \tau = \frac{p^e}{p^r} = \frac{C_I'}{C_W'} = \frac{\partial C_A / \partial a_{eA}}{\partial C_A / \partial a_{rA}} \tag{3.14}$$

so that each source is indifferent between purchasing permits and offsets from any other source. Given relation (3.14), the industrial sector's problem reduces to problem (3.8), with the FOCs

satisfying condition (3.9) above. Analogously, the wastewater treatment sector's problem reduces to problem (3.9), with the FOCs being condition (3.10). The farms' problem reduces to (3.11), with the FOCs being condition (3.13). The FOCs (3.9), (3.10), (3.12), and (3.13), the inter-pollutant exchange rate given by (3.14), along with the market clearing condition

$$\hat{e}_{I0} + \hat{e}_{A0} + \frac{\hat{r}_{W0} + \hat{r}_{A0}}{\tau} \geq \left(e_{I0} - a_{eI}\right) + \left(e_{A0} - a_{eA}\right) + \frac{\left(r_{W0} - a_{rW}\right) + \left(r_{A0} - a_{rA}\right)}{\tau}, \tag{3.15}$$

form the equilibrium system under an integrated market. Equilibrium comprises a vector of prices, p^{e**} and p^{r**}, and pollution abatement allocations a_{eI}^{**}, a_{eA}^{**}, a_{rW}^{**} and a_{rA}^{**}, that satisfy this system. Comparing conditions (3.9), (3.10), (3.12), and (3.13) with the cost-effective FOCs in (3.3)–(3.6) shows the first-best abatement arises if the permit cap is chosen so that $p^e = B_e' \big|_{a_{eI}^*, a_{eA}^*}$ as before. The optimal value of p^r then follows from (3.15), implying the optimal inter-pollutant exchange rate is the ratio of marginal damages from atmospheric Nr fluxes to the marginal damages from aquatic Nr fluxes, or

$$\tau^* = B_e' / B_r' \big|_{a_{eI}^*, a_{eA}^*, a_{rW}^*, a_{rA}^*}. \tag{3.16}$$

This result contrasts with current multi-pollutant markets, which typically determine the inter-pollutant exchange rate according to their physical or chemical qualities.[37]

[37] For example, different types of GHGs are traded based on their global warming potential, denominated in units of "carbon equivalents." Trades governed by these types of exchange rates are unlikely to be cost-effective as they ignore the economic characteristics of pollution that vary across pollutant species (Muller 2012; Schmalensee 1993). An economically-optimal trading ratio accounts for these economic characteristics, as illustrated in (3.15).

3.4.3 The Relative Efficiency of an Integrated Market when Caps are Exogenous to the Market

Both separate and integrated markets are first-best under the optimal caps and exchange rates derived above. However, it is unlikely in practice that markets will be designed efficiently because the caps are not determined as a component of the optimal market design. In this case, gains may arise from trading across pollutants since integration offers an additional policy parameter—the inter-pollutant exchange rate—that can be optimally chosen to attain second-best pollution abatement.[38]

We derive the second-best inter-pollutant exchange rate τ by finding the value of τ that maximizes social net benefits from abatement, subject to the polluters' privately optimal abatement choices in the market equilibrium. As τ is the only policy parameter affecting the market equilibrium, the polluters' FOCs (3.9), (3.10), (3.12), and (3.13) can be implicitly solved for the abatement supplies as functions of τ, i.e., $a_{eI}(\tau)$, $a_{rW}(\tau)$, $a_{eA}(\tau)$, and $a_{rA}(\tau)$.

Identifying the sign of the derivative of each of these supply functions requires evaluating a 5×5 Hessian matrix, which is analytically intractable. Prior work (Horan and Shortle 2015) indicates the sign of each derivative depends on two effects. A cost effect stems from the final two equalities in equilibrium relation (3.14): a larger τ implies a larger relative marginal abatement cost, and hence more abatement, in the emissions sector, other things equal (and vice versa for the loadings sector). A quantity effect arises from condition (3.16): a larger τ means loadings abatement exchanges for fewer permits of the numeraire commodity (emissions), so that more abatement of loadings is required to produce the same number of emissions permits

[38] This is a straightforward consequence of Le Châtelier's Principle, i.e. that auxiliary constraints (e.g., the constraint that pollutants cannot be exchanged under separate markets) reduce responses to parameter changes, and hence the efficiency of a system at equilibrium (Samuelson 1947).

(and vice versa for emissions permits being exchanged for loadings permits). The effects suggest $a'_{eI} > 0$ and $a'_{eA} > 0$, as the cost and quantity effects operate in the same direction. In contrast, the sign of a'_{rW} and a'_{rA} are ambiguous due to opposing cost and quantity effects. We will consider outcomes where the price effect dominates (so that a'_{rW}, $a'_{rA} < 0$) and where the quantity effect dominates (so that a'_{rW}, $a'_{rA} > 0$) below.

Substituting the abatement supply functions into the social planner's objective function (3.2) and maximizing yields the FOC

$$[B'_r - C'_W]a'_{rW} + \left[B'_e - \frac{\partial C_A}{\partial a_{eA}}\right]a'_{eA} + \left[B'_r - \frac{\partial C_A}{\partial a_{rA}}\right]a'_{rA} = 0 . \tag{3.17}$$

Noting that the marginal cost of abatement each pollutant equals the permit or offset price of each pollutant at the second-best outcome, (3.17) can be simplified:

$$[B'_e - C'_I][a'_{eI} + a'_{eA}] + \left[B'_r - \frac{C'_I}{\tau}\right][a'_{rW} + a'_{rA}] = 0 . \tag{3.17′}$$

Solving for τ yields the second-best inter-pollutant exchange rate

$$\tau^{**} = \frac{C'_I}{B'_r + \Phi\rho} = \frac{C'_I}{C'_W} = \frac{\partial C_A / \partial a_{eA}}{\partial C_A / \partial a_{rA}} \tag{3.18}$$

where $\Phi = [B'_e - C'_I]$ is the marginal net benefits from abating emissions and $\rho = [a'_{eI} + a'_{eA}]/[a'_{rW} + a'_{rA}]$ is the marginal rate of substitution between emissions and loadings. We have argued that the numerator of ρ is positive, whereas the denominator is ambiguous in sign: $\rho > 0$ if the quantity effect dominates the cost effect in the loadings sector, and $\rho < 0$ otherwise. The second and third equalities in (3.17) stem from the market equilibrium conditions $\tau = p_e / p_r$, $p_e = C'_I = \partial C_A / \partial a_{eA}$, and $p_r = C'_W = \partial C_A / \partial a_{rA}$.

The second-best ratio τ^{**} is the same as the first-best ratio when $\Phi = 0$ such that $B'_e = C'_I = \partial C_A / \partial a_{eA}$, in which case (3.18) implies $B'_r = C'_W = \partial C_A / \partial a_{rA}$. However, a single policy tool is unlikely to be able to correct all the externalities to produce the first-best outcome. This means $\Phi \neq 0$ so that $C'_I \neq B'_e$, and hence the second-best trade ratio will yield $B'_r \neq C'_W = \partial C_A / \partial a_{rA}$.

Suppose first that $\Phi > 0$, such that $B'_e > C'_I = \partial C_A / \partial a_{eA}$. If $\rho > 0$ (such that a larger τ increases abatement in each sector), then $\Phi\rho > 0$ and the denominators of the two middle terms in (3.18) indicate that $B'_r < C'_W = \partial C_A / \partial a_{rA}$. These results suggest that $\tau^{**} = \dfrac{C'_I}{B'_r + \Phi\rho} < \dfrac{B'_e}{B'_r} = \tau^{*}$. It would not be efficient to increase τ^{**} towards τ^{*}. While a larger τ^{**} would enhance efficiency in the emissions sector by reducing the difference $\Phi = B'_e - C'_I$, this would come at a cost stemming from an increased difference $C'_W - B'_r$.

Alternatively, if $\rho < 0$ (such that a larger τ increases [reduces] abatement in the emissions [loadings] sector), then $\Phi\rho < 0$ and the denominators of the two middle terms in (3.18) indicate that $B'_r > C'_W = \partial C_A / \partial a_{rA}$. These results suggest an ambiguous relation between τ^{**} and τ^{*}. Suppose $\tau^{**} < \tau^{*}$. Here, increasing τ^{**} towards τ^{*} would enhance efficiency in the emissions sector by reducing the difference $\Phi = B'_e - C'_I$, but this would come at a cost stemming from an increased difference $B'_r - C'_W$. Now consider the alternative, supposing instead that $\tau^{**} > \tau^{*}$. In this case, reducing τ^{**} towards τ^{*} would reduce efficiency in the emissions sector by increasing the difference $\Phi = B'_e - C'_I$, while at the same time generating benefits stemming from a smaller difference $B'_r - C'_W$. Therefore, either $\tau^{**} < \tau^{*}$ or $\tau^{**} > \tau^{*}$ may emerge as an equilibrium outcome when $\Phi > 0$ and $\rho < 0$.

Suppose next that $\Phi < 0$ so that $B'_e < C'_I = \partial C_A / \partial a_{eA}$. If $\rho > 0$ then $\Phi\rho < 0$ such that $B'_r > C'_W = \partial C_A / \partial a_{rA}$ by (3.18). This suggests the relationship between τ^{**} and τ^* is ambiguous. If $\tau^{**} < \tau^*$, then increasing τ^{**} towards τ^* increases abatement in both sectors. This increases efficiency in the loading sector by bringing C'_W closer to B'_r. However, this gain is offset by a loss in efficiency from additional abatement in the emissions sector, leading to a greater difference $C'_I - B'_e$. If $\tau^{**} > \tau^*$, then decreasing τ^{**} towards τ^* will increase efficiency in the emissions sector by decreasing emissions abatement, reducing the difference $C'_I - B'_e$. These gains will be offset by a decrease in loading abatement, increasing the difference $B'_r - C'_W$.

Alternatively, if $\rho < 0$ then $\Phi\rho > 0$ such that $B'_r < C'_W = \partial C_A / \partial a_{rA}$. This suggests $\tau^{**} > \tau^*$. In this case, reducing τ^{**} towards τ^* would increase efficiency in the emissions sector by reducing the difference $C'_I - B'_e$. However, these gains would be offset by the resulting increase in abatement by the loading sector that would increase the difference $C'_W - B'_r$.

It is unlikely that pollution caps are set efficiently under separate markets. Hence, gains may arise from integration. We now turn to a numerical example of a hypothetical Nr trading program to illustrate the gains from integration in a real-world setting.

3.5 Numerical Model: Inter-pollutant Trading in the Susquehanna River Basin

We now illustrate the theory using an illustrative model of inter-pollutant trading in the Pennsylvania portion of the SRB. Nr pollution in the form of greenhouse gas emissions and water quality loadings poses a major ecological and economic threat to environmental quality in the Chesapeake Bay and surrounding airshed (Birch et al. 2011). We focus on the Pennsylvania portion due to data availability and because Pennsylvania accounts for the greatest share of land

area and pollution in the SRB. The SRB features numerous point sources of GHGs and water pollution. In addition, Ribaudo and Nickerson (2009) identify several subbasins of the Pennsylvania portion of the SRB as promising for the establishment of point-nonpoint water quality trading markets. Hence, the SRB is an economically interesting example for analyzing the potential for integrated markets. We now provide a brief description of the simulation.

Data for the simulation come primarily from secondary sources. Specifically, data describing aggregate loadings, abatement and marginal abatement costs for point and nonpoint water pollution sources in the SRB are taken from the Chesapeake Bay TMDL (Kaufman et al. 2014). We continue to aggregate all point sources of N loadings into the "wastewater treatment" sector, although our data includes loadings from other types of polluters in addition to wastewater treatment plants. Likewise, we aggregate nonpoint sources of N loadings into the "agricultural sector."

Data describing initial GHG emissions for point sources in the SRB are taken from the US EPA's flight database. As with the wastewater treatment sector, we aggregate all point sources of emissions into a single "industrial" sector, but note that our data includes emissions from power plants and other regulated point sources. Marginal abatement cost data are taken from RGGI (2014). Finally, agricultural GHG emissions and marginal abatement costs from corn production are estimated following Reeling and Gramig (2012).

All sectors' abatement cost functions are assumed to be cubic in abatement. Specifically, let $C_I(a_{eI}) = \phi a_{eI}^3$ and $C_W(a_{rW}) = \psi a_{rW}^3$ be the industrial and wastewater treatment sectors, where $\phi > 0$ and $\psi > 0$ are parameters calibrated for the SRB. Following Woodward (2011), let $C_A(a_{eA}, a_{rA}) = (\alpha/2)a_{eA}^3 + (\beta/2)a_{rA}^3 - \gamma a_{eA}a_{rA}$ represent the agricultural sources' abatement cost function, where $\alpha, \beta, \gamma > 0$ are parameters and γ represents the degree of complementarity in

agricultural sector emissions and loadings abatement.

The marginal damages from emissions are assumed to be constant since GHGs are a globally mixed pollutant. Hence, the damage function for emissions takes the form $D_e(E_e) = \varepsilon E_e$ so that the (constant) marginal damage from emissions is ε (Tol 2005). The damage from loadings is assumed to take the form $D_r(E_r) = \nu E_r^2$, where ν is calibrated using data from the Chesapeake TMDL (Kaufman et al. 2014).

3.5.1 Simulation Results

The numerical model of the SRB is used to simulate abatement costs under separate and integrated markets using calibrated and simulated in *Mathematica 7.0* (Wolfram, Inc. 2008). Calibrated parameters can be found in Table 3.1.

Consider first the efficient outcome as a baseline for comparison with second best abatement allocations. The efficient outcome can arise from the social planner's problem (3.2), or from efficiently designed integrated or separate pollution markets (i.e., where the initial emissions and loading caps, $\hat{e}_{I0}$ and $\hat{r}_{W0}$, are chosen to satisfy $p^e = B'_e|_{a^*_{eI}, a^*_{eA}}$ and $p^r = B'_r|_{a^*_{rW}, a^*_{rA}}$, given that nonpoint sources have an implicit right to pollute). Table 3.2 shows the gains from optimally managing emissions and loadings can be significant—social net benefits total nearly $141.5 million for the Pennsylvania portion of the SRB. The efficient inter-pollutant exchange rate is small and suggests the marginal benefits (and hence the marginal costs) from abating loadings are more than 430 times those from abating emissions at the first best outcome. Point sources abate the majority of emissions in the efficient allocation. This contrasts with the

Table 3.1 Simulation Parameters

Parameter	Description	Value	Source
ϕ	Marginal cost parameter, industrial sector	0.0021	Reeling and Gramig (2012)
ψ	Marginal cost parameter, wastewater treatment sector	2.98×10^{-14}	Kaufman et al. (2014)
α	Marginal cost parameter, agricultural sector	0.61×10^{-11}	Reeling and Gramig (2012)
β	Marginal cost parameter, agricultural sector	2.43×10^{-4}	Reeling and Gramig (2012)
γ	Complementarity parameter, agricultural sector	5×10^{-6}	Assumption
ε	Marginal damage from emissions ($/mtCO$_2$e)	14	Tol (2005)
ν	Marginal damage parameter, loadings	0.08	Kaufman et al. 2014
e_{I0}	Initial industrial emissions (million mtCO2$_e$)	153.5	US EPA FLIGHT (2013)
e_{A0}	Initial agricultural emissions (million mtCO2$_e$)	5.54	Reeling and Gramig (2012)
r_{W0}	Initial wastewater treatment loadings (thousand mtN)	12	Kaufman et al. (2014)
r_{A0}	Initial agricultural loadings (thousand mtN)	28	Kaufman et al. (2014)

loadings sector, in which agriculture abates the majority of pollution. The difference arises due to the agricultural sector's relatively higher costs of abating emissions, but relatively lower cost of abating loadings.

We examine two second-best scenarios in which the permit caps for each pollutant have been set sub-optimally since these choices are generally made distinctly in each sector, by different agencies, and they typically do not account for agricultural sources which are not initially regulated and therefore have implicit initial permit caps of e_{A0} and r_{A0}. Specifically, let the permit caps for the emissions and loadings by the industrial and wastewater treatment sectors, respectively, be $\hat{e}_{e0} = \zeta_e \hat{e}^*_{e0}$ and $\hat{r}_{W0} = \zeta_r \hat{r}^*_{W0}$, where ζ_s (for $s \in \{e, r\}$) is a parameter equal to one in the efficient case. A value of $\zeta_s < 1$ (> 1) implies an inefficiently (lax) cap on

Table 3.2 Results from Hypothetical Pollutant Trading Scenarios, SRB[a]

	Social net benefits ($ million)	τ	Emissions abatement		Loadings abatement	
			Industry	Agriculture	WWT[b]	Agriculture
First-best	141.5	0.0023	12.52	0.88	0.96	2.86
Separate 2nd-best, $\zeta_e = \zeta_r = 0.9$[c]	–136.22	N/A	25.62	1.80	1.17	3.47
Integrated 2nd-best, $\zeta_e = \zeta_r = 0.9$	72.77	0.0002	7.92	0.56	2.11	6.26
Change from integration	*208.99*	——	*–17.70*	*–1.24*	*0.94*	*2.79*
Separate 2nd-best, $\zeta_e = \zeta_r = 1.05$	98.89	N/A	5.98	0.42	0.86	2.56
Integrated 2nd-best, $\zeta_e = \zeta_r = 1.05$	103.2	0.001	6.50	0.46	0.68	2.02
Change from integration	*4.31*	——	*0.52*	*0.04*	*–0.18*	*–0.54*

[a] All emissions figures are expressed in millions of metric tons of carbon dioxide equivalents ($mtCO_2e$). All loadings figures are expressed in thousands of metric tons of nitrogen. τ is the required reduction in loadings (in thousands of metric tons of nitrogen) for a one-unit increase in emissions (in millions of metric tons of $C0_2$ equivalents).

[b] WWT = Wastewater treatment sector.

[c] Efficient emissions cap = 140.1 million $mtCO_2e$; efficient loadings cap = 8200 mtN.

pollution from point sources of *s*. We continue to assume the agricultural sector has an implicit right to pollute such that ζ_s does not affect the initial allocation of agricultural offsets.

Consider first the second-best outcome in which $\zeta_e = \zeta_r = 0.9$ such that the permit caps on point source pollution are set exogenously at 90 percent of their efficient levels. Such an outcome may arise if policymakers implement excessive point source controls since they are unable to regulate nonpoint sources. The social net benefits from pollution abatement decrease dramatically under both market institutions. Consider first the outcome under separate markets. The inefficiently strict caps result in greater abatement by all sources relative to the efficient outcome. Excessive abatement drives marginal abatement costs above the marginal benefits from abatement for all sectors. Social net benefits are negative under separate markets and total nearly $280 million less than the first-best level.

In contrast, the effects of excessively strict permit caps are more muted under integration. Social net benefits decline by nearly half relative to the first-best outcome but are still positive and large, totaling nearly $73 million. The second-best inter-pollutant trade ratio τ^{**} is an order of magnitude smaller than the first-best level, meaning that the *effective* initial allocation of loadings permits (given by $\hat{r}_{W0}/\tau^{**}$) actually increases, despite the decrease in the absolute cap level. This encourages trade (and, hence, abatement) by the loadings sources, and reduces abatement by the emissions sources.

Consider finally the second-best outcome in which $\zeta_e = \zeta_r = 1.05$. (An emissions cap much beyond this level results in initial permit allocations greater than initial industrial emissions.) Such an outcome may arise if policymakers fail to account for the complementarity between agricultural emissions and loadings abatement (Feng and Kling 2005). Social net benefits decline by 28–30 percent under both second-best market outcomes. Under separate

markets, excessively large initial permit caps lead to reduced abatement in each market, particularly in the emissions market, where total abatement declines by more than 50 percent from first-best levels.[39] The reduction in abatement is much smaller for the loadings sector.

Integration again leads to additional gains relative to separate markets, although the gains are smaller in this case where the caps have been made less stringent. Integration also results in substitution of emissions abatement for loadings abatement. This is in contrast to the case where caps were overly stringent, as would be expected.

Table 3.3 Pollutant Caps under Each Market Setting

	Emissions (million mt CO_2e)		Loadings (thousand mtN)			Effective Loadings (million mt CO_2e)[a]	
	Industry	Agriculture	WWT	Agriculture	τ	WWT	Agriculture
Efficient	140.1	5.54	8.17	28	0.0023	3.48	11.92
Second-best:							
$\zeta_e = \zeta_r = 0.9$	126.1	5.54	7.36	28	0.0002	37.45	142.46
$\zeta_e = \zeta_r = 1.05$	147.1	5.54	8.58	28	0.0013	6.72	21.92

[a] Effective loadings caps are the loadings caps divided by τ, so that the cap is denominated in emissions units. This measure only applies to integrated markets.

3.6 Discussion and Conclusion

Our results suggest the gains from integrating separate pollution markets may be substantial. Despite this, and despite our increasing knowledge of the linkages between pollutants, little research considers the potential for linking environmental markets across pollutants and environmental media, nor do examples of such markets exist in practice.

Alternative approaches for holistic environmental management are being pursued. One example is "credit bundling," i.e., selling a package of credits, each representing a distinct

[39] A 50 percent decrease in abatement from a 5 percent increase in the cap may appear excessive. However, note that abatement and emissions are different measures, with emissions generally being much larger than abatement. Hence, a small change in allowable emissions generates a relatively large change in abatement.

environmental service, as a single commodity (Robertson et al. 2014). Credit bundling is used in current ecosystem service markets (e.g., the Willamette Partnership in Oregon; Deal, Cochran and LaRocco 2012). However, bundling differs from integration in that it treats environmental services as non-separable. Trading of bundled credits therefore requires sellers to search for buyers interested in a particular bundle of environmental services. Costs related to this search may limit trading activity under credit bundling. In contrast, integrated markets treat environmental services as separable (e.g., the agricultural sector sells emissions offsets separately from loadings offsets, even though abatement of both pollutants may arise from the same action). This may increase the liquidity of credits or offsets and increase trading, while simultaneously accounting for linkages between pollutants. The extent to which integration enhances market participation is left to future research.

Several practical issues may influence the success of integrated markets. First, integration assumes damages from pollution are fungible, meaning the allocation of damages to different stakeholder groups spread out across the landscape does not matter. However, trading across pollutants and environmental media may result in abatement allocations that vary spatially and/or put different populations at risk. This may be problematic when the effects of pollution are highly localized. The distribution of rents from pollution trading will also change with the flow of permit payments across sectors and/or the landscape.

The efficiency and functionality of integrated markets will also depend on several factors not considered here, including transaction costs and uncertainty about the costs and benefits of pollution abatement. Integrated markets may feature lower transaction costs relative to separate markets. Different Nr fluxes affect different environmental media whose protection may fall under the purview of different government agencies. Attaining efficiency under separate caps

may be a challenge when doing so requires multiple agencies to coordinate the choice of each pollution cap. In contrast, a single cap need be chosen under an integrated market. There may be an advantage to integrated markets in that they reduce the number of policy parameters that need be negotiated (Weitzman 2014).

Our understanding of the linkages between environmental pollutants is increasing. New approaches will improve the efficiency and sustainability of managing these pollutants. This paper is the first to explore the potential for utilizing integrated markets to manage distinct environmental pollutants across environmental media. That said, much about the dynamics of many pollutants and their implications for efficient market design remains unknown. Further disciplinary and interdisciplinary work is needed to combine insights from economics and the natural sciences to more effectively manage linked pollutants.

APPENDICES

Appendix A Supplementary Information for Chapter 1

In this appendix, we describe the calibration of the numerical model and provide a sensitivity analysis of the numerical model.

A.1 Numerical Model Calibration

We begin by deriving ρ as an index of biosecurity investment, where this index is tied to management practices of varied effectiveness. We then use this index, along with the costs of the practices, to construct the cost relation $c(\rho)$. Chi et al. (2002) calculate the costs of ten biosecurity practices that include various combinations of background checks on purchased livestock, the decision to raise livestock on-farm, and vaccination. Since vaccines for FMD are not commonly used and were not used during the UK FMD epidemic, we ignore all practices involving vaccination. The remaining practices are listed in Table A1. We rank these practices according to relative effectiveness, assigning a value of 0 to the least effective practice (i.e., purchasing cattle at auction without background checks or vaccination) and 4 to the most effective (closing one's farm off to introduced animals). Next, we assume the correspondence between practice number and ρ takes the form $\rho = 1 - e^{-\lambda \cdot practice\#}$, where *practice#* corresponds to the ranking in Table A1. This nonlinear relationship is based on the assumption that more effective practices increase the degree of protection at a decreasing rate. The parameter λ is set to 1.15, as this value yields $\rho \approx 1$ when *practice#* = 4, which corresponds with full investment in biosecurity. We then used the calibrated values of ρ, along the costs as reported by Chi et al. (2002) to estimate the cost function $c(\rho) = \chi\rho^2$. Using OLS, we estimated $\ln(c) = \beta_0 + \beta_1 \ln(\rho)$ and obtained a value for $\chi = 1328$ ($\beta_0 = 7.19$, *p-value* 0.001) and $\beta_1 = 1.9$ (*p-value* = 0.00), with $R^2 = 0.99$. As β_1 was not significantly different from 2, we used the value of 2 for as in the text.[40]

[40] Note that we calibrated ρ and estimated the cost relation for several different relations between ρ and *practice#*, and we found the goodness of fit to be the best under the current specification for ρ.

Next, consider the probability of infection for a given farmer (equation 1.1, main text). We begin by parameterizing $P^I(\rho) = \xi(1 - \mu\rho)$. First, since full biosecurity involves closing off one's farm (so that no animals will be introduced), the maximum achievable reduction in introduction risks achievable through biosecurity is 100 percent when $\rho = 1$. That is, $P^I(\rho)|_{\rho = 1} = 0$. We therefore set $\mu = 1$ to ensure this outcome. Next, we calibrate the value ξ. Green et al. (2006) assume one in ten animals purchased from these markets are infected in modeling the initial spread of FMD from animal movements from livestock markets in northern England. This implies $P^I = 0.1$ for some value of ρ. We assume this probability corresponds to typical biosecurity investment, which we define as conducting background checks on all animals purchased from a market (practice 1, Table A1, or $\rho = 0.68$). Given this assumption, we set $P^I(\rho)|_{\rho = 0.68} = 0.1$ to derive $\xi = 0.3125$.

Table A.1 Description of Biosecurity Practices Used in Numerical Example

ρ	Practice #	Farm Access	Source of Livestock	Background Checks	Cost (£)
0.99	4	Closed	N/A	N/A	2,446
0.97	3	Open	Other producer	Yes	1,694
0.90	2	Open	Other producer	No	1,519
0.68	1	Open	Dealer/auction	Yes	173
0.00	0	Open	Dealer/auction	No	0

The infectivity of contact between herds is given by $k(\rho) = \kappa(1 - \nu\rho)$. The parameter ν represents the per-unit reduction in infectivity per unit of biosecurity investment. Biosecurity is likely to be only marginally effective in reducing the probability that infection spreads between infected and susceptible herds, especially in the absence of vaccination. We therefore assume $\nu = 0.15$. We assume that the probability that a herd gets infected by a neighboring herd under typical biosecurity (practice 1, Table A1) is $k(\rho)|_{\rho = 0.68} = 0.8$. This large probability reflects the

large transmission risks in Cumbria County due to the high farm density in this county and also the relatively high transmissibility of FMD (Schley et al. 2009). Then, given our value of v = 0.15, we use the relation $k(\rho)|_{\rho = 0.68} = \kappa(1 - 0.15 \times 0.68) = 0.8$ to solve for $\kappa = 0.9$.

The number of neighboring producers N is estimated from Brennan et al. (2008). The authors use network and cluster analysis to identify connections among farms in northwestern England. The authors estimate that indirect contacts occur between 50 of the 56 farms in the 10 km × 10 km area they cover in their analysis. This does not account for connections to farms outside the area of study, so the pool of farms over which these contacts occur may be somewhat larger. We set $N = 80$ to account for these additional contacts.

The effective number of neighboring producers depends on the extent to which the representative producer can protect himself from direct and indirect contacts with these herds. Full biosecurity is not perfectly effective in reducing contact between herds due to aerosol transmission of the FMD virus. Ferguson et al. (2001) utilize another kernel density to approximate the probability that aerial transmission occurs from an infected premises within a given distance. Beyond 2 km, the risk of aerial transmission is negligible. Combining this transmission kernel with herd density data for Cumbria County (Defra 2011), it was estimated that an average of five herds will be located within a 2 km radius of a given farm, and thus a representative producer could be exposed to aerosol transmission from five neighboring producers, should they become infected. Recalling that the effective number of neighboring producers is $\hat{N}(\rho, \sigma) = (N - 1)(1 - \alpha\rho)(1 - \alpha\sigma)$, we set $\hat{N}(\cdot) = 5$ and $\rho = \sigma = 1$. This expression is then solved for $\alpha = 0.75$.

Finally, we calculate the representative producer's cost of infection, Λ, which we take to be the value of the herd (assuming all animals within an infected herd are culled) and idling

factors of production. There were 6.124 million animals slaughtered in the UK during the outbreak, at a total cost to producers of £1.155 billion. This value results in a cost per animal slaughtered of £188.6. We multiply this value by 140, which is the average herd size in Cumbria county (Defra 2011). This results in a total cost to the producer of Λ = £26,404.

A.2 Sensitivity Analysis

We now present the results of a sensitivity analysis (see Table A2) that examines how sensitive the our results are to changes in the model's parameters. The first column in Table A2 indicates the parameter being considered, as well as the baseline value of the indicated parameter. The remaining columns indicate the scenario being considered. We consider three scenarios: (a) the baseline model with no policy, (b) the behaviorally-independent indemnity of $\phi(\rho) = 0.68$, and (c) the behaviorally-dependent indemnity of $\phi(\rho) = 0.68\rho$ (note that this is the indemnity associated with Figure 1.3, not the first-best indemnity). Recall that scenarios (a) and (b) each yield multiple SNE, whereas scenario (c) yielded a unique SNE. Our sensitivity analysis explores the robustness of these qualitative results. Specifically, for scenarios (a) and (b), we identify the minimum required percentage change in the indicated parameter value, *ceteris paribus*, that will yield a unique SNE. The opposite is performed for scenario (c): we identify the minimum required percentage change in the indicated parameter value, *ceteris paribus*, that will yield multiple SNE.

For scenarios (a) and (b), a unique SNE arises when the parameters are changed to sufficiently increase individuals' relative control over their spread risks (the opposite arises for our analysis of scenario (c), in which we are trying to produce multiple SNEs). This means changing parameters to reduce spread risks (i.e., smaller κ or N, or larger ν or α) or introduction

risks (as the risk of introduction by others contributes to spread risks; i.e., smaller ξ or larger μ [although the baseline value of μ is already maximized at $\mu = 1$]). Multiple equilibria can also be eliminated by reducing Δ, although in this case fewer economic losses means a unique equilibria at a low value of ρ is more likely.

Overall, the results in Table A2 indicate the qualitative results of the main article are insensitive to changes in a single parameter. Qualitative changes are only possible for six of the eight parameters in scenarios (a) and (b), and for only three of the seven variables in scenario (c). Where a qualitative change is possible, the required percentage change in the indicated parameter is substantial.

Table A.2 Sensitivity Analysis Results

Parameter[a]	Baseline (a)[b]	Behaviorally-independent indemnity (b)	Behaviorally-dependent indemnity (c)
ξ (0.3125)	-68%	-71%	-39%
μ (1)	-97%	-74%	—
κ (0.9)	-60%	-73%	—
ν (0.15)	—	—	—
α (0.75)	—	—	—
χ (1,328)	+907%	+222%	+292%
N (80)	-70%	-78%	—
Δ (26,404)	-89%	-69%	-75%

[a] The initial value of the parameter is in parentheses.
[b] An entry of — indicates that multiple equilibria arise for all possible parameter values.

Appendix B Supplementary Information for Chapter 2

B.1 Proof that $v^*_{NDi} > v^*_i - v^0_i$

There are two cases to consider. First, suppose $-D'(\partial e_i / \partial v_i) > \partial \Lambda_i / \partial v_i \ \forall v$. The proof proceeds in two steps. The first establishes that $v^0_i < v^*_{NDi} < v^*_i$. Consider the first inequality. We know from equations (2.4) and (2.7) that v^0_i and v^*_{NDi} solve, respectively,

(B1) $$-\frac{\partial \Lambda_i}{\partial v_i} = \frac{\partial c_i}{\partial v_i}, \quad v \in \{x, z, b\}$$

(B2) $$-D'\frac{\partial e_i}{\partial v_i} = \frac{\partial c_i}{\partial v_i} \quad v \in \{x, z, b\}.$$

The LHS in (B2) is greater than the LHS in (B1) by assumption, implying $v^0_i < v^*_{NDi}$ by convexity of c_i. Likewise, from equation (2.6) we know v^*_i solves

(B3) $$-\frac{\partial \Lambda_i}{\partial v_i} - D'\frac{\partial e_i}{\partial v_i} = \frac{\partial c_i}{\partial v_i} \quad v \in \{x, z, b\}.$$

The LHS in (B3) is greater than the LHS in (B2), implying $v^*_{NDi} < v^*_i$ by convexity of c_i. Hence, $v^0_i < v^*_{NDi} < v^*_i$.

The second step establishes that $v^*_{NDi} > v^*_i - v^0_i$. By convexity of $D(\cdot)$, $\Lambda_i(\cdot)$, and $c(\cdot)$, we know $v^0_i > v^*_i - v^0_i$ (see Figure 2.1, main text). Further, $v^*_{NDi} > v^0_i$, implying $v^*_{NDi} > v^*_i - v^0_i$.

For the second case, suppose $-D'(\partial e_i / \partial v_i) < \partial \Lambda_i / \partial v_i$, implying $v^0_i \geq v^*_{NDi}$ (see Figure B1). Importer i's private marginal costs with and without risks are given by $MC_i = \partial c_i/\partial v_i + \partial \Lambda_i/\partial v_i$ and $MC_{NDi} = \partial c_i/\partial v_i$, respectively. Differentiating with respect to v_i,

$$\frac{\partial MC_i}{\partial v_i} = \frac{\partial^2 \Lambda_i}{\partial v_i^2} + \frac{\partial^2 c_i}{\partial v_i^2} > \frac{\partial^2 c_i}{\partial v_i^2} = \frac{\partial MC_{NDi}}{\partial v_i}$$

by convexity of Λ_i as shown in Figure B1. It must be the case that $v_i^* - v_i^0 < v_{NDi}^*$ by convexity of marginal damages. ■

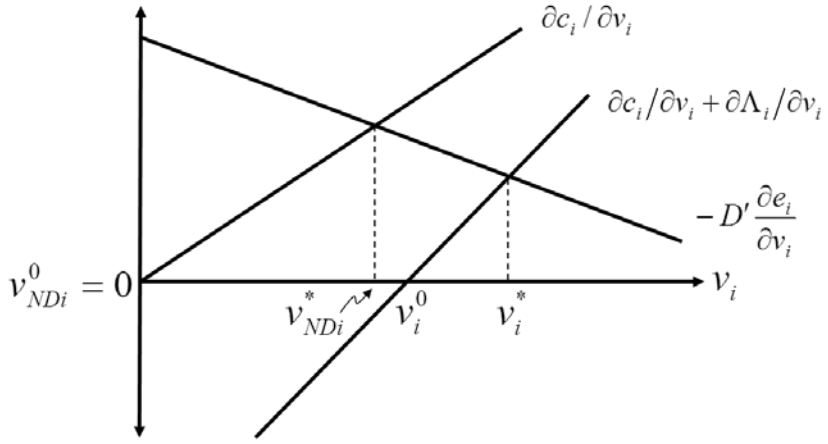

Figure B.1 Comparison of privately- and socially-optimal risk mitigation when $v_i^0 > v_{NDi}^*$

B.2 The Restricted Profit Function and its Properties

In this section we derive the restricted profit function $\pi_i^R(e_i, b_i; \mathbf{e}_{-i})$ and demonstrate that it may be non-convex in the biological pollution externalities from others, $\mathbf{e}_{-i}$.

The restricted profit function $\pi_i^R(\cdot)$ is the solution to the problem

(B4) $$\max_{x_i, z_i, a_i} \pi_i(x_i, z_i, a_i, b_i; \mathbf{e}_{-i}) \text{ subject to } e_i = e_i(x_i, z_i, a_i).$$

The associated Lagrangian is

(B5) $$L_i = \pi_i(x_i, z_i, a_i, b_i; \mathbf{e}_{-i}) + \eta_i[e_i - e_i(x_i, z_i, a_i)],$$

where η_i is the Lagrange multiplier. The FOCs from L_i can be solved for the response functions $x_i(e_i, b_i; \mathbf{e}_{-i})$, $z_i(e_i, b_i; \mathbf{e}_{-i})$, $a_i(e_i, b_i; \mathbf{e}_{-i})$, and $\eta_i(e_i, b_i; \mathbf{e}_{-i})$. Substituting these responses into the objective function in (B4) yields

(B6) $$\pi_i(x_i(e_i, b_i; \mathbf{e}_{-i}), z_i(e_i, b_i; \mathbf{e}_{-i}), a_i(e_i, b_i; \mathbf{e}_{-i}), b_i; \mathbf{e}_{-i}) = \pi_i^R(e_i, b_i; \mathbf{e}_{-i}).$$

We now show the after-tax problem, $\pi_i^R(\cdot) - t_i e_i$, may exhibit strategic complementarities—and hence non-convexities—resulting in the possibility of multiple Nash equilibria. Specifically, strategic complementarities require $\partial^2(\pi_i^R - t_i e_i)/\partial v_i \partial e_j \geq 0$, $v \in \{e, b\}$ and $\partial^2(\pi_i^R - t_i e_i)/\partial b_i \partial e_i \geq 0$; Vives (2005)). Relations (B5) and (B6) imply

(B7) $$\frac{\partial^2(\pi_i^R - t_i e_i)}{\partial e_i \partial e_j} = \frac{\partial^2 L_i}{\partial e_i \partial e_j} = \frac{\partial \eta_i}{\partial e_j} \gtreqless 0$$

(B8) $$\frac{\partial^2(\pi_i^R - t_i e_i)}{\partial b_i \partial e_j} = \frac{\partial^2 L_i}{\partial b_i \partial e_j} = \frac{\partial^2 \pi_i}{\partial b_i \partial e_j} = -\frac{\partial^2 \Omega}{\partial b_i \partial e_j} > 0 \quad \text{(by (2.20))}$$

(B9) $$\frac{\partial^2(\pi_i^R - t_i e_i)}{\partial b_i \partial e_i} = \frac{\partial^2 \pi_i^R}{\partial e_i \partial b_i} = \frac{\partial^2 L_i}{\partial e_i \partial b_i} = \frac{\partial \eta_i}{\partial b_i} \gtreqless 0.$$

Solving for $\partial\eta_i/\partial e_j$ and $\partial\eta_i/\partial b_i$ involves evaluating the determinant of the 5 × 5 bordered Hessian matrix for the Lagrangean in (B5). This is analytically intractable, so we instead use *Mathematica 7.0* (Wolfram Research, Inc. 2008) to calculate $\partial\eta_i/\partial e_j$ and $\partial\eta_i/\partial b_i$ numerically for an illustrative numerical example to show that complementarities may exist. For simplicity, we assume homogenous importers, with the functional forms and parameters used in the example summarized in Table B1. The expressions (B7) and (B9) are both positive ($\partial\eta_i/\partial e_j$ = 0.355, $\partial\eta_i/\partial b_i$ = 0.162), implying strategic complementarity. The results are robust to significant perturbations in parameter values.

Table B.1 Functional Forms and Parameter Values used in Illustrative Example

	Domain	Form/Value in Numerical Example	Description	Units
Functions				
$P_i^I(x_i)$	[0, 1]	$P_i^I(x_i) = \gamma(1 - x_i)$	Probability of importing infected animal from risky region	Unitless
$P_i^C(z_i)$	[0, 1]	$P_i^C(z_i) = \alpha z_i$	Probability of detecting infection via surveillance	Unitless
$P_i^L(a_i)$	[0, 1]	$P_i^L(a_i) = 1 - \rho a_i$	Probability of infection leaving importer's herd	Unitless
$P_i^S(b_i)$	[0, 1]	$P_i^S(b_i) = \varepsilon(1 - \mu b_i)$	Probability of infection spreading to importer's herd from others	Unitless
$c_i(x_i, z_i, a_i, b_i)$	[0, 1]	$c_i(x_i, z_i, a_i, b_i) = w(1 - x_i)X \times (1 + hz_i^2) + vx_iX + fa^2 + gb^2$	Costs unrelated to disease	$
Parameters				
N	[0, ∞)	5	Number of neighboring importers	Herds
γ	[0, 1]	0.9	Probability of importing infected animal if all animals purchased from risky region	Unitless
α	[0, 1]	0.5	Probability of detecting infected animal with full surveillance effort	Unitless
ρ	[0, 1]	0.2	% reduction in probability of disease leaving one's herd per unit of abatement effort	Unitless
ε	[0, 1]	0.9	Probability of disease spreading to one's herd in the absence of biosecurity	Unitless
μ	[0, 1]	0.9	% reduction in probability of disease spreading to one's herd per unit of biosecurity	Unitless
X	[0, ∞)	1	Feedlot capacity	Animals
δ	[0, ∞)	0.6	Losses from culling infected animals before introduction to herd	$/animal
λ	[0, ∞)	0.8	Losses from culling infected animals after introduction to herd	$/animal
w	[0, ∞)	0.2	Cost of animals from risky source	$/animal

Table B1 (cont'd)

	Domain	Form/Value in Numerical Example	Description	Units
v	$[0, \infty)$	0.5	Cost of animals from risk-free source	$/animal
h	≥ 1	1	Surveillance cost parameter	Unitless
f	$[0, \infty)$	1	Abatement cost parameter	Unitless
g	$[0, \infty)$	1	Biosecurity cost parameter	Unitless

REFERENCES

REFERENCES

Acocella, N., G. Di Bartolomeo, and A. Hughes-Hallett. 2013. *The Theory of Economic Policy in a Strategic Context*. New York: Cambridge University Press.

Anderson, S., and P. Francois. 1997. "Environmental Cleanliness as a Public Good: Welfare and Policy Implications of Nonconvex Preferences." *Journal of Environmental Economics and Management* 34(3):256–274.

Archer, D.W., and J.F. Shogren. 1996. "Endogenous Risk in Weed Control Management." *Agricultural Economics* 14(2):103–122.

Aronsson, T., and E. Koskela. 2011. "Redistributive Income Taxation, Outsourcing and Foreign Direct Investment." *Economics Letters* 110(2):86–89.

Babcock, B.A., P.G. Lakshminarayan, J. Wu, and D. Zilberman. 1997. "Targeting Tools for the Purchase of Environmental Amenities." *Land Economics* 73(3):325–339.

Barlow, N.D. 1995. "Critical Evaluation of Wildlife Disease Models." In B. T. Grenfell and A. P. Dobson, eds. *Ecology of Infectious Diseases in Natural Populations*. New York: Cambridge University Press.

Barrett, S. 2004. "Eradication versus Control: The Economics of Global Infectious Disease Policies." *Bulletin of the World Health Organization* 82(9):683–688.

Bicknell, K.B., J.E. Wilen, and R.E. Howitt. 1999. "Public Policy and Private Incentives for Livestock Disease Control." *Australian Journal of Agricultural and Resource Economics* 45:501–521.

Birch, M.B.L., B.M. Gramig, W.R. Moomaw, O.C. Doering III, and C.J. Reeling. 2011. "Why Metrics Matter: Evaluating Policy Choices for Reactive Nitrogen in the Chesapeake Bay Watershed." *Environmental Science & Technology* 45(1):168.

Brennan, M.L., R. Kemp, and R.M. Christley. 2008. "Direct and Indirect Contacts Between Cattle Farms in North-West England." *Preventive Veterinary Medicine* 84:242–260.

Buchanan, J.M., and M.Z. Kafoglis. 1963. "A Note on Public Goods Supply." *The American Economic Review* 53(3):403–414.

CA AB 32. *California Global Warming Solutions Act of 2006*. Available at: http://www.arb.ca.gov/cc/ab32/ab32.htm [Accessed June 15, 2015].

Chen, F. 2012. "A Mathematical Analysis of Public Avoidance Behavior during Epidemics using Game Theory." *Journal of Theoretical Biology* 302:18–28.

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* 50:237–256.

Convery, I., C. Bailey, M. Mort, and J. Baxter. 2005. "Death in the Wrong Place? Emotional Geographies of the UK 2001 Foot and Mouth Disease Epidemic." *Journal of Rural Studies* 21(1):99–109.

Cooper, R.W., D.V. DeJong, R. Forsythe, and T.W. Ross. 1990. "Selection Criteria in Coordination Games: Some Experimental Results." *The American Economic Review* 80(1):218–233.

Dasgupta, P., and K.-G. Mäler. 2003. "The Economics of Non-Convex Ecosystems: Introduction." *Environmental and Resource Economics* 26(4):499–525.

Daszak, P., A.A. Cunningham, and A.D. Hyatt. 2000. "Emerging Infectious Diseases of Wildlife-- Threats to Biodiversity and Human Health." *Science* 287(5452):443–449.

Deal, R.L., B. Cochran, and G. LaRocco. 2012. "Bundling of Ecosystem Services to Increase Forestland Value and Enhance Sustainable Forest Management." *Forest Policy and Management* 17: 69–76.

Department for Environment Food and Rural Affairs (DEFRA). 2011. "Land Use and Livestock on Agricultural Holdings." Available at: http://archive.defra.gov.uk/evidence/statistics/foodfarm/landuselivestock/junesurvey/results.htm [Accessed May 10, 2013].

Donaldson, A.I., and S. Alexandersen. 2002. "Predicting the Spread of Foot and Mouth Disease by Airborne Virus." *Revue Scientifique et Technique - Office International des Epizooties* 21(3):569–575.

Echenique, F. 2004. "A Characterization of Strategic Complementarities." *Games and Economic Behavior* 46(2):325–347.

Echenique, F., and A.S. Edlin. 2004. "Mixed Equilibria are Unstable in Games of Strategic Complements." *Journal of Economic Theory* 118(1):61–79.

Elbakidze, L. 2007. "Economic Benefits of Animal Tracing in the Cattle Production Sector." *Journal of Agricultural and Resource Economics* 32(1):169–180.

Feng, H. and C.L. Kling. 2005. "The Consequences of Cobenefits for the Efficient Design of Carbon Sequestration Programs." *Canadian Journal of Agricultural Economics* 53(4): 461–476.

Fenichel, E.P., C. Castillo-Chavez, M.G. Ceddia, G. Chowell, P.A. Gonzalez Parra, G.J. Hickling, G. Holloway, R. Horan, B. Morin, C. Perrings, M. Springborn, L. Velazquez, and C. Villalobos. 2011. "Adaptive Human Behavior in Epidemiological Models." *Proceedings of the National Academy of Sciences* 180(15):6306–6311.

Ferguson, N.M., C.A. Donnelly, and R.M. Anderson. 2001. "The Foot-and-Mouth Epidemic in Great Britain: Pattern of Spread and Impact of Interventions." *Science* 292:1155–1160.

Fudenberg, D., and J. Tirole. 1995. *Game Theory*. Cambridge, Massachusetts: The MIT Press.

Galloway, J.N., J.D. Aber, J.W. Erisman, S.P. Seitzinger, R.W. Howarth, E.B. Cowling, and B.J. Cosby. 2003. "The Nitrogen Cascade." *BioScience* 54:341–356.

Gibbens, J.C., C.E. Sharpe, J.W. Wilesmith, L.M. Mansley, E. Michalopoulou, J.B. Ryan, and M. Hudson. 2001. "Descriptive Epidemiology of the 2001 Foot-and-Mouth Disease Epidemic in Great Britain: The First Five Months." *The Veterinary Record* 149(24):729–743.

Gog, J., R. Woodroffe, and J. Swinton. 2002. "Disease in Endangered Metapopulations: The Importance of Alternative Hosts." *Proceedings of the Royal Society B: Biological Sciences* 269(1492):671–676.

Gramig, B.M., R.D. Horan, and C.A. Wolf. 2009. "Livestock Disease Indemnity Design When Moral Hazard Is Followed by Adverse Selection." *American Journal of Agricultural Economics* 91(3):627–641.

Green, D.M., I.Z. Kiss, and R.R. Kao. 2006. "Modelling the Initial Spread of Foot-and-Mouth Disease Through Animal Movements." *Proceedings of the Royal Society B* 273:2729–2735.

Griffin, R.C., and D.W. Bromley. 1982. "Agricultural Runoff as a Nonpoint Externality: A Theoretical Development." *American Journal of Agricultural Economics* 64(3):547–552.

Harsanyi, J.C. 1973. "Games with Randomly Disturbed Payoffs: A New Rationale for Mixed-Strategy Equilibrium Points." *International Journal of Game Theory* 2(1):1–23.

Heesterbeek, J.A.P., and M.G. Roberts. 1995. "Mathematical Models for Microparasites of Wildlife." In B. T. Grenfell and A. P. Dobson, eds. *Ecology of Infectious Diseases in Natural Populations*. New York: Cambridge University Press, pp. 90–122.

Hefti, A. 2011. "On Uniqueness and Stability of Symmetric Equilibria in Differentiable Symmetric Games." *Working Paper No. 18, University of Zurich Department of Economics*.

Hennessy, D.A. 2007a. "Behavioral Incentives, Equilibrium Endemic Disease, and Health Management Policy for Farmed Animals." *American Journal of Agricultural Economics* 89(3):698–711.

Hennessy, D.A. 2007b. "Biosecurity and Spread of an Infectious Animal Disease." *American Journal of Agricultural Economics* 89(5):1226–1231.

Hennessy, D.A. 2008. "Biosecurity Incentives, Network Effects, and Entry of a Rapidly Spreading Pest." *Ecological Economics* 68:230–239.

Hess, G. 1996. “Disease in Metapopulation Models: Implications for Conservation.” *Ecology* 77(5):1617–1632.

Hoag, D.L., D.D. Thilmany, and S.R. Koontz. 2006. “Economics of Livestock Disease Insurance—Principles, Issues and Worldwide Cases.” In D. L. Hoag, D. D. Thilmany, and J. W. Green, eds. *The Economics of Livestock Disease Insurance: Concepts, Issues and International Case Studies*. Wallingford, UK: CABI, pp. 1–18.

Horan, R.D., and F. Lupi. 2005a. “Economic Incentives for Controlling Trade-Related Biological Invasions in the Great Lakes.” *Agricultural and Resource Economics Review* 34(1):75–89.

Horan, R.D., and F. Lupi. 2005b. “Tradeable Risk Permits to Prevent Future Introductions of Invasive Alien Species into the Great Lakes.” *Ecological Economics* 52(3):289–304.

Horan, R.D. and J.S. Shortle. 2005. “When Two Wrongs Make a Right: Second-Best Point-Nonpoint Trading Ratios.” *American Journal of Agricultural Economics* 87(2): 340–352.

Horan, R.D., J.S. Shortle, and J.F. Shogren. 2015. “Multi-Market Trading for Cooperative Resource Management: An Application to Water Pollution and Fisheries.” Working Paper, Michigan State University.

Horan, R., C. Perrings, F. Lupi, and E.H. Bulte. 2002. “Biological Pollution Prevention Strategies Under Ignorance: The Case of Invasive Species.” *American Journal of Agricultural Economics* 84(5):1303–1310.

Hung, M.F. and D. Shaw. 2005. “A Trading-Ratio System for Trading Water Pollution Discharge Permits.” *Journal of Environmental Economics and Management* 49(1): 83–102.

Kane, S., and J.F. Shogren. 2000. “Linking Adaptation and Mitigation in Climate Change Policy.” *Climatic Change* 45(1):75–102.

Kaufman, Z., D. Abler, J. Shortle, J. Harper, and J. Hamlett, 2014. “The Agricultural Costs of the Chesapeake Bay TMDL.” *Environmental Science and Technology*. 48 (24), pp 14131–14138.

Knowler, D., and E. Barbier. 2005. “Importing Exotic Plants and the Risk of Invasion: Are Market-Based Instruments Adequate?” *Ecological Economics* 52(3):341–354.

Kobayashi, M., and T. Melkonyan. 2011. “Strategic Incentives in Biosecurity Actions: Theoretical and Empirical Analyses.” *Journal of Agricultural and Resource Economics* 36(242-262).

Krugman, P. 1991. “History versus Expectations.” *The Quarterly Journal of Economics* 106(2):651–667.

Kuchler, F., and S. Hamm. 2000. “Animal Disease Incidence and Indemnity Eradication Programs.” *Agricultural Economics* 20(299-308).

Malik, A.S., D. Letson, and S.R. Crutchfield. 1993. “Point/Nonpoint Source Trading of Pollution Abatement: Choosing the Right Trading Ratio.” *American Journal of Agricultural Economics* 75(4):959–967.

Mas-Colell, A., M. Whinston, and J.R. Green. 1995. *Microeconomic Theory*. New York: Oxford University Press.

McAusland, C., and C. Costello. 2004. “Avoiding Invasives: Trade-Related Policies for Controlling Unintentional Exotic Species Introductions.” *Journal of Environmental Economics and Management* 48:954–977.

McCallum, H., and A. Dobson. 2002. “Disease, Habitat Fragmentation and Conservation.” *Proceedings of the Royal Society B: Biological Sciences* 269(1504):2041–2049.

Mérel, P.R., and C.A. Carter. 2008. “A Second Look at Managing Import Risk from Invasive Species.” *Journal of Environmental Economics and Management* 56(3):286–290.

Mikkelsen, T., S. Alexandersen, P. Astrup, H.J. Champion, A.I. Donaldson, F.N. Dunkerley, J. Gloster, J.H. Sørensen, and S. Thykier-Nielsen. 2003. “Investigation of Airborne Foot-and-Mouth Disease Virus Transmission During Low-Wind Conditions in the Early Phase of the UK 2001 Epidemic.” *Atmospheric Chemistry and Physics* 3(6):2101–2110.

Milgrom, P., and J. Roberts. 1990. “Rationalizability, Learning, and Equilibrium in Games with Strategic Complementarities.” *Econometrica* 58(6):1255–1277.

Montero, J.-P. 2001. “Multipollutant Markets.” *The RAND Journal of Economics* 32(4):762–774.

Muhammad, A., and K. Jones. 2008. “The Impact of Federal Indemnification on Livestock Biosecurity.” *Economics Bulletin* 17(10):1–9.

Muller, N.Z. 2012. “The Design of Optimal Climate Policy with Air Pollution Co-Benefits.” *Resource and Energy Economics* 34(4):696–722.

Muller, N.Z. and R. Mendelsohn. “Efficient Pollution Regulation: Getting the Prices Right.” *American Economic Review* 99(5): 1714–1739.

Olson, M., Jr., and R. Zeckhauser. 1970. “The Efficient Production of External Economies.” *The American Economic Review* 60(3):512–517.

Paarlberg, P.L., and J.G. Lee. 1998. “Import Restrictions in the Presence of a Health Risk: An Illustration Using FMD.” *American Journal of Agricultural Economics* 80(1):175–183.

Panagariya, A., and H. Shibata. 2000. “Defense and Welfare under Rivalry.” *International Economic Review* 41(4):951–969.

Perry, B.D., D. Grace, and K. Sones. 2011. “Current Drivers and Future Directions of Global Livestock Disease Dynamics.” *Proceedings of the National Academies of Science* 110(52):20871–20877.

Pratt, J.W. 1964. “Risk Aversion in the Small and in the Large.” *Econometrica* 32(1-2):122–136.

Rahmandad, H., and J. Sterman. 2008. “Heterogeneity and Network Structure in the Dynamics of Diffusion: Comparing Agent-Based and Differential Equation Models.” *Management Science* 54(5):998–1014.

Reeling, C.J., and B.M. Gramig. 2012. “A Novel Framework for Analysis of Cross-Media Environmental Effects from Agricultural Conservation Practices.” *Agriculture, Ecosystems & Environment* 146:44–51.

Reeling, C.J., and R.D. Horan. 2015. “Self-Protection, Strategic Interactions, and the Relative Endogeneity of Disease Risks.” *American Journal of Agricultural Economics* 97(2):452–468.

Regional Greenhouse Gas Initiative. 2014. “CO2 Auctions.” Available at: http://www.rggi.org/market/co2_auctions [Accessed December 8, 2014].

Reilly, J.M. and K.R. Richards. 1993. “Climate Change Damage and the Trace Gas Index Issue.” *Environmental and Resource Economics* 3:41–61.

Ribaudo, M.O., and C.J. Nickerson. 2009. “Agriculture and Water Quality Trading: Exploring the Possibilities.” *Journal of Soil and Water Conservation* 64(1):1–7.

Robertson, M., T.K. BenDor, R. Lave, A. Riggsbee, J.B. Ruhl, and M. Doyle. 2014. “Stacking Ecosystem Services.” *Frontiers in Ecology and the Environment* 12: 186–193.

Samuelson, P.A. 1947. *The Foundations of Economic Analysis*. Cambridge: Harvard University Press.

Schley, D., S. Gubbins, and D.J. Paton. 2009. “Quantifying the Risk of Localised Animal Movement Bans for Foot-and-Mouth Disease.” *PLoS ONE* 4(5):e5481.

Schmalensee, R. 1993. “Comparing Greenhouse Gases for Policy Purposes.” *The Energy Journal* 14:245–255.

Segarra, A.E., and J.M. Rawson. 2001. “Foot and Mouth Disease: A Threat to U.S. Agriculture.” Available at: http://www.policyarchive.org/handle/10207/bitstreams/3466.pdf [Accessed May 7, 2013].

Shogren, J. 1991. “Endogenous Risk and Protection Premiums.” *Theory and Decision* 31(2). Available at: http://search.proquest.com.proxy1.cl.msu.edu/docview/1303221316?accountid=12598 [Accessed January 30, 2015].

Shogren, J.F., and T.D. Crocker. 1991. “Cooperative and Noncooperative Protection Against Transferable and Filterable Externalities.” *Environmental and Resource Economics* 1:195–214.

Shortle, J., D. Abler, and R. Horan. 1998. “Research Issues in Nonpoint Pollution Control.” *Environmental and Resource Economics* 11(3):571–585.

Shortle, J.S., and J.W. Dunn. 1986. “The Relative Efficiency of Agricultural Source Water Pollution Control Policies.” *American Journal of Agricultural Economics* 68(3):668–677.

Thompson, D., P. Muriel, D. Russell, P. Osborne, A. Bromley, M. Rowland, S. Creigh-Tyte, and C. Brown. 2002. “Economic Costs of the Foot and Mouth Disease Outbreak in the United Kingdom in 2001.” *Revue Scientifique et Technique -- Office International des Epizooties* 21(3):675–687.

Tietenberg, T.H. 1985. *Emissions Trading, an Exercise in Reforming Pollution Policy*. Washington, D.C.: RFF Press.

Tinbergen, J. 1952. *On the Theory of Economic Policy*. Amsterdam: North Holland.

Tol, R.S.. 2005. “The Marginal Damage Costs of Carbon Dioxide Emissions: An Assessment of the Uncertainties.” *Energy Policy* 33:2064–2074.

Treich, N. 2010. “The Value of a Statistical Life Under Ambiguity Aversion.” *Journal of Environmental Economics and Management* 59(1):15–26.

Tweddle, N.E., and P. Livingstone. 1994. “Bovine Tuberculosis Control and Eradication Programs in Australia and New Zealand.” *Veterinary Microbiology* 40(1–2):23–39.

US Environmental Protection Agency (US EPA). 2013. “EPA Facility Level GHG Emissions Data.” Available at: http://ghgdata.epa.gov/ghgp/main.do [Accessed December 8, 2014].

Vives, X. 2005. “Complementarities and Games: New Developments.” *Journal of Economic Literature* 43(2):437–479.

Vives, X. 2011. “Strategic Complementarity, Fragility, and Regulation.” *CESifo Working Paper: Monetary Policy and International Finance No. 3507.* Accessed 18 January 2013.

Wang, T., and D.A. Hennessy. 2015. “Strategic Interactions Among Private and Public Efforts When Preventing and Stamping Out a Highly Infectious Animal Disease.” *American Journal of Agricultural Economics* 97(2):435–451.

Watts, D.J., R. Muhamad, D.C. Medina, and P.S. Dodds. 2005. “Multiscale, Resurgent Epidemics in a Hierarchical Metapopulation Model.” *Proceedings of the National Academy of Sciences of the United States of America* 102(32):11157–11162.

Westra, J.V., K.W. Easter, and K.D. Olson. 2002. “Targeting Nonpoint Source Pollution Control: Phosphorus in the Minnesota River Basin.” *JAWRA Journal of the American Water Resources Association* 38(2):493–505.

Whitcraft, C.R., D.M. Talley, J.A. Crooks, J. Boland, and J. Gaskin. 2007. “Invasion of Tamarisk (Tamarix spp.) in a Southern California Salt Marsh.” *Biological Invasions* 9(7):875–879.

Woodward, R.T. 2011. “Double-Dipping in Environmental Markets.” *Journal of Environmental Economics and Management* 61(2): 153–169.

Yaffee, S.L. 1997. “Why Environmental Nightmares Recur.” *Conservation Biology* 11(2): 328–337.

Weitzman, M.L. 2014. “Can Negotiating a Uniform Carbon Price Help to Internalize the Global Warming Externality?” *Journal of the Association of Environmental and Resource Economists* 1(1/2): 29–49.

Wolfram, Inc. *Mathematica 7.0*. Champaign, IL.