Self-Protection, Strategic Interactions, and the Relative Endogeneity of Disease Risks*

Carson J. Reeling  
Graduate Research Assistant  
Agricultural, Food, and Resource Economics  
Michigan State University  
reelingc@msu.edu

Richard D. Horan  
Professor  
Agricultural, Food, and Resource Economics  
Michigan State University  
horan@msu.edu

* This paper is currently under review at the American Journal of Agricultural Economics. Please do not cite or quote.
Abstract

Self-protection is a key behavior influencing infectious disease risks. 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 and the role of expectations. Moreover, we show that traditional notions of optimal incentive design to ensure the first-best level of self-protection is a Nash equilibrium may not address the potential for coordination failure. We derive conditions for a true first-best instrument that achieves the first-best outcome and eliminates the risk 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.

Keywords: disease; multiple equilibria; relative endogeneity of risk; spillover; strategic interactions; policy design
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 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 Hallet 2013). This may be insufficient to guarantee the first-best outcome when there is a potential for coordination failure. We present an approach that incentivizes the first-best level of self-protection as 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.

**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 \( P_I(\rho) \). ¹ We assume \( P_I'(\rho) < 0 \) and \( P_I''(\rho) > 0 \), where \( \rho \in [0,1] \) is the producer’s self-protection or biosecurity effort.² The signs on the partial derivatives
indicate that disease protection, defined as \( [1 - P_I(\rho)] \), is increasing in \( \rho \) 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 \( \rho \), but also the self-protection of his neighbors. Let \( \sigma \in [0,1] \)

¹ 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 et al. 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.

² 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.
denote the biosecurity effort of the other producers in the region. We treat $\sigma$ as a scalar for simplicity (since all neighbors are homogeneous and make identical choices), but the basic strategic relations and results in our static model are qualitatively unchanged if instead $\sigma$ were a vector of heterogeneous neighbors’ effort levels.\textsuperscript{3} The probability that the representative producer becomes infected via spread, conditional on being uninfected, is written $P^S(\rho, \sigma)$, with $P^S_i < 0$ and $P^S_{ii} > 0$ for $i = \rho, \sigma$. The function $P^S(\rho, \sigma)$ is quite general, and so $\sigma$ 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 these pathways, can be written

\begin{equation}
P(\rho, \sigma) = P^I(\rho) + [1 - P^I(\rho)]P^S(\rho, \sigma).
\end{equation}

**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 $\rho$. The producer’s problem is

\begin{equation}
\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{equation}

\textsuperscript{3} Heterogeneity of economic and/or technical relations, including heterogeneous disease risks over time, will generate heterogeneous behaviors. These heterogeneous behaviors will have quantitative impacts on economic and epidemiological outcomes (Rahmandad and Sternman 2008). However, the strategic relationships arise from a qualitative relationship between a producer’s marginal incentives for biosecurity and his (heterogeneous) neighbors’ biosecurity effort choices. As we describe below, the type of strategic relationship that arises depends on whether the producer’s marginal incentives are increasing or decreasing in his neighbors’ biosecurity. Therefore, the qualitative strategic relationships and associated results are preserved under producer heterogeneities that only influence disease risks quantitatively (e.g., Vives 2005).
where $\Lambda = (R^H - R^{NH}) > 0$ represents losses or damages due to infection. Problem (2) is solved as a Nash-Cournot game: the producer chooses his or her own biosecurity $\rho$ while taking others’ effort $\sigma$ as given. Other producers behave analogously.

The first-order condition of problem (2) is $E[\pi_{\rho}] = 0$, which implies

$$-\Lambda P_{\rho}(\rho, \sigma) = c_{\rho}(\rho). \tag{3}$$

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 the optimum, this is equal to the marginal cost of biosecurity, denoted by the right-hand side (RHS) term.

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

$$P_{\rho}(\rho, \sigma) = P^I_{\rho}(\rho) - P^I_{\rho}(\rho)P^S(\rho, \sigma) + [1 - P^I(\rho)]P^S_{\rho}(\rho, \sigma). \tag{4}$$

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 (4). The first RHS term in (4), which is negative, represents the marginal effect of $\rho$ on reducing introduction risks, holding spread risks constant. The second term represents the effect of $\rho$ 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). 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 (4), which is negative, reflects the effect of $\rho$ on the risk of spread, holding introduction risks constant.
Incentives Arising from Strategic Interactions

Condition (3) indicates that a producer’s marginal incentives for biosecurity depend on neighbors’ biosecurity, $\sigma$. The implicit solution to (3) is given by the best-response function $\rho(\sigma)$, with $\rho_\sigma = E\{\pi_{p\sigma}\}/(-E\{\pi_{p\rho}\})$. Note that $E\{\pi_{p\rho}\} < 0$ if $\rho(\sigma)$ maximizes expected profits. The sign of $\rho_\sigma$ therefore depends on the sign of $E\{\pi_{p\sigma}\}$, which indicates how the producer’s marginal incentives for $\rho$ change as neighbors increase their biosecurity $\sigma$. Hence, the slope of $\rho(\sigma)$ reflects the strategic relation between producers.

If $E\{\pi_{p\rho}\} > 0 \forall \rho, \sigma$, 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 1a. Three SNE occur where the producer’s best-response function $\rho(\sigma)$ crosses the 45° line. Equilibria $A$ and $B$ are locally stable, as $\rho_\sigma < 1$ in the neighborhood of each point. 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 $\rho$ is adjusted based on initial expectations about $\sigma$ (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 $\sigma < \sigma_T$,

---

4 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, $\rho'(\sigma) = -(\partial F/\partial \sigma)/(\partial F/\partial \rho)$.

5 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.
then the system will be in the low-effort basin and proceed to A. This latter outcome represents coordination failure such that the producers’ universally preferred outcome B may not be 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 1b. Expectations do not matter in this case, as the system will always converge to point A.

If $E\{\pi_{\rho \sigma}\} < 0$ ∀$\rho, \sigma$, 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 $|\rho_\sigma| < 1$ ∀$\sigma$, as illustrated in figure 1c, and so expectations do not matter.\(^6\)

![FIGURE 1a–c]

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 $\sigma$). Multiple equilibria may arise in such cases as well, with the stability properties described above 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., $\sigma$ 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

\(^6\) 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.
complement (i.e., $\sigma$ 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 $\sigma$ on the producer’s risks, $\Lambda P(\rho, \sigma)$, noting that all impacts of $\sigma$ occur along the spread pathway: $\Lambda P_{\sigma}(\rho, \sigma) = \Lambda [1 - P'(\rho)]P^S(\rho, \sigma) < 0$. This expression indicates that $\sigma$ 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 $\rho$ affects the producer’s reliance on neighbors for controlling spread risks:

\begin{equation}
E[\pi_{\sigma\rho}] = -\Lambda P_{\sigma\rho}(\rho, \sigma) = \Lambda [P'(\rho)P^S_{\sigma}(\rho, \sigma) + [1 - P'(\rho)] - P^S_{\sigma}(\rho, \sigma)]
\end{equation}

The first RHS term in braces, which is positive, represents the degree to which the producer’s biosecurity $\rho$ 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 $\rho$ affects the positive spillover effects of others’ efforts in controlling spread risks. The overall sign of (5) is ambiguous. A positive value of (5) means that a larger $\rho$ makes the producer more reliant on neighbors for protection from spread risks. In other words, by adopting $\rho$, the producer effectively cedes more control of his spread risks to his neighbors. This case arises when $\rho$ increases spillover effects, i.e., $- P^S_{\alpha\rho}(\rho, \sigma) > 0$ (e.g., when jointly preventing wildlife that may act as a disease reservoir from entering common grazing areas) or when $- P^S_{\alpha\rho}(\rho, \sigma) \leq 0$ but sufficiently small in magnitude (e.g., when cleaning
shared equipment before bringing it on the premises neutralizes the effect of $\sigma$ on the producer’s spread risks. A negative value of (5) (i.e., when $-P^S_{\infty}(\rho,\sigma) < 0$ and sufficiently large in absolute magnitude) means that a larger $\rho$ makes the producer less reliant on neighbors for protection. In other words, by adopting $\rho$, the producer takes control of his spread risks away from his neighbors.

Expression (5) can be rewritten as

$$E[\pi_{\alpha}] = \Lambda P^I_\rho(\rho) P^S_\sigma(\rho,\sigma)(1 - \Omega^\rho),$$

where $\Omega^\rho = \eta^\rho / \varepsilon$, $\eta^\rho = -\frac{\partial(c(1 - P^S)/\partial\sigma)}{\partial(c(1 - P^S)/\partial\sigma)}\frac{\rho}{1 - P^I}$, and $\varepsilon = \frac{\partial(1 - P^I)}{\partial\rho} \frac{\rho}{1 - P^I} > 0$. We use the term $\Omega^\rho$ 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.\(^7\) 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, $\eta^\rho$ measures 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 adopting greater $\rho$, the producer takes control over his spread risks away from his neighbors, whereas a negative value implies the opposite. The term $\eta^\rho$ is then scaled by one’s self-protection impacts on exposure, $\varepsilon > 0$; the smaller is $\varepsilon$, the less exposure will there be to spread risks (for a given $\rho$) and hence the relative amount of control over these risks is less of a concern.\(^8\)

\(^7\) We use the term “relative” here in the same sense as Pratt’s (1964) coefficient of relative risk aversion, which is also an elasticity.

\(^8\) The measure $\Omega^\rho$ 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
By Young’s Theorem, we see that strategic relations and RER are fundamentally related:

\[
(7) \quad \text{self-protection is a strategic}\begin{cases} \text{substitute} & \text{iff } \text{RER} = \Omega^p \geq 1 \\ \text{complement} & \text{iff } \text{RER} = \Omega^p < 1. \end{cases}
\]

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

Finally, note that \(\Omega^p\) only reflects a producer’s relative ability to control the probabilistic (not economic) component of spread risks, hence the superscript \(P\) applied to \(\Omega\). The next section illustrates how the use of economic policy may modify expressions (6) and (7) to also reflect control over economic components of risk.

**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 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.
practice (Hoag, Thimany, and Koontz 2006). This is not an insurance program, as no premiums are paid. Rather, these are used as safety nets and to incentivize 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. 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) and (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.\(^9\) The case of $\phi(\rho) \neq 0$ represents a behaviorally-dependent indemnity, so that compensation is greater when producers apply more biosecurity efforts. The case of $\phi(\rho) = 0$ (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 (2) is now

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

with first-order condition

$$- \Lambda[1 - \phi(\rho)]P_{\rho}(\rho, \sigma) + \Lambda \phi_{\rho}(\rho)P(\rho, \sigma) = c_{\rho}(\rho).\tag{8}$$

Consider first the special case of a behaviorally-independent indemnity ($\phi(\rho) = 0$), which is the form indemnities typically take in practice (Hoag, Thimany, and Koontz 2006). Here, the second LHS term vanishes from (8), and so the indemnity reduces the marginal expected benefits from $\rho$, relative to condition (3). Other things equal, this results in lower biosecurity effort, as is consistent with most prior work on indemnity payments (e.g., Bicknell, Wilen, and Howitt 1999; Kuchler and Hamm 2000; Hennessy 2007a; Muhammad and Jones 2008).

\(^9\) We relax the assumption that $\phi(\rho) \geq 0$ in our analysis of optimal policy design.
Now consider the case of a behaviorally-dependent indemnity ($\phi_p(\rho) > 0$). The second LHS term in (8) is positive so as to increase the incentives for biosecurity. However, the overall incentives provided by the indemnity are ambiguous. Subtract condition (3) from (8) to obtain the difference in the marginal benefits of biosecurity with and without the indemnity:

$$ (9) \quad \Lambda \phi(\rho) \left[ (1 - P^S(\rho, \sigma)) P^I(\rho) + (1 - P^I(\rho)) P^S(\rho, \sigma) \right] + \Lambda \phi_p(\rho) P(\rho, \sigma). $$

This difference is related to the expected indemnity payment: $\Lambda \phi(\rho) P(\rho, \sigma)$. The first term in (9), which is negative, is the marginal effect of $\rho$ on the expected indemnity payment, holding the indemnity level fixed (i.e., only considering the impact of $\rho$ on the overall probability of infection, $P$). The second term in (9), which is positive, is the marginal effect of $\rho$ on the expected indemnity payment, holding the probability of infection fixed (i.e., only considering the impact of $\rho$ on the payment rate, $\phi$). Together, these effects represent $\rho$’s impact on both the probabilistic and economic components of risk. The net effect is ambiguous in sign.

Expression (9) is positive, so that indemnities increase one’s incentives for self-protection, when $\rho$ increases the expected indemnity payment $\Lambda \phi(\rho) P(\rho, \sigma)$. This result, which is in contrast to most prior literature, occurs when the effect of $\rho$ on reducing the probability of infection is 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 (8) with respect to $\sigma$:

$$ (10) \quad E[\pi_{\sigma}] = -\Lambda \left[ 1 - \phi(\rho) \right] P_{\sigma}(\rho, \sigma) + \Lambda \phi_p(\rho) P_{\sigma}(\rho, \sigma). $$

Consider first the case of a behaviorally-independent indemnity ($\phi_p(\rho) = 0$), 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 (5). However, relative to equation (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 $\rho$ 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 (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 $\rho$. This term is negative: an increase in $\sigma$ decreases the probability of infection, and thus decreases the producer’s expected marginal indemnity benefits from $\rho$.

If $\rho$ and $\sigma$ 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 $E\{\pi_\rho\}$ 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 $\rho$ 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 while also improving ex ante social welfare. 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:

$$(11) \quad \text{self-protection is a strategic } \begin{cases} \text{substitute} & \text{iff } RER = \Omega^P + \Omega^E > 1, \\ \text{complement} & \text{iff } RER = \Omega^P + \Omega^E < 1, \end{cases}$$

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

Condition (11) indicates that RER is unchanged for a behaviorally-independent indemnity, since $\Omega^E = 0$ in this case so that $RER = \Omega^P$ as in condition (7). In the case of a behaviorally-dependent indemnity, however, condition (11) indicates that RER is increased by the term $\Omega^E$. This new measure of RER measures the producer’s ability to take control over both components of spread risk: the probability of infection via spread (reflected by $\Omega^P$) and the economic impact of infection (reflected by $\Omega^E$). 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.

**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.

**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.10

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, $\xi$ is the probability of pathogen introduction when no biosecurity is adopted, and $\mu$ 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

---

10 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.
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 $N(\rho, \sigma) = N(1 - \alpha \rho)(1 - \alpha \sigma)$, where $\alpha$ represents the reduction in contacts per unit of biosecurity adopted.\(^{11}\) This specification implies that neighbors’ biosecurity efforts are as effective in preventing contacts as the producer’s own efforts, e.g., because the biosecurity 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, $\kappa$ is the probability that contact with an infected herd results in spread absent biosecurity, and $\nu$ 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 $\sigma$, 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

---

\(^{11}\) 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 $\alpha$, 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).
modeled as a Bernoulli process:\textsuperscript{12} 

\begin{equation}
P^S(\rho, \sigma) = 1 - \left[ 1 - k(\rho)P^I(\sigma) \right]^{\tilde{N}(\rho, \sigma)}.
\end{equation}

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 $[1 - k(\rho)P^I(\sigma)]^{\tilde{N}(\rho, \sigma)}$ 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 one neighbor’s herd increases for the representative producer. Likewise, $P^S(\cdot)$ is decreasing in $\rho$ and $\sigma$. 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).

\text{[Table 1]}

The producer’s problem (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. The sources and derivation of each parameter are detailed in the supplementary appendix online. 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

\text{\textsuperscript{12} Note that it is likely that the exponent $\tilde{N}(\cdot)$ 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 (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.}
contagious and is capable of aerial transmission over relatively long distances (Mikkelsen et al. 2003; Ferguson, Donnelly, and Anderson 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, Donnelly, and Anderson 2001).

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 (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 2.

The only two equilibria present in the model are the two symmetric Nash equilibria (SNE), \( A \) and \( B \) in figure 2.\(^{13}\) The SNE can be Pareto-ranked, with the high-effort equilibrium \( B \) being privately preferred (Van Zandt and Vives 2007).\(^{14}\)

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 \( B \).\(^{15}\) Expectations matter in figure 2.

If producers expect their neighbors initially choose biosecurity \( \sigma > \sigma_T \), then the system proceeds

\(^{13}\) 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.

\(^{14}\) 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 \( \sigma \) were strictly less than 1, then it would not be Pareto optimal (Milgrom and Roberts 1990).

\(^{15}\) The stability properties of the system follow from the fact that, according to the numerical results, \( E[\pi_\sigma] < 0 \) for \( \sigma \) below the discontinuity and \( E[\pi_\sigma] > 0 \) for \( \sigma \) 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 \( \sigma \) to the left of the discontinuity or to \( B \) for any expectation that puts \( \sigma \) to the right of the discontinuity. Note also that if a 45° line extending from the origin intersected the positively-sloped portion of \( \rho(\sigma) \), the resulting SNE would also be unstable as \( \rho_\sigma > 1 \) at such a point.
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.\(^{16}\)

The multiple, stable SNE in figure 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 \( \text{RER} = 1 \) in figure 2 is a contour line denoting the locus of points for which \( \Omega^p = 1 \).

To the left of this curve, \( \Omega^p < 1 \), and thus by condition (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 \( B \).

[Figure 2]

**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 \( \zeta = 0.55 \), i.e., the producer receives compensation equal to 55 percent of his losses from infection if he fully self-protects. This value of \( \zeta \) 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 (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 3.

The behaviorally-dependent indemnity drastically changes the producer’s best-response

---

\(^{16}\) We have performed a sensitivity analysis for the baseline parameterization shown in table 1. All else equal, multiple SNE give way to a single SNE when: a) \( \xi < 0.11 \); b) \( \kappa < 0.44 \); or c) \( N < 37 \). Changes in the other parameters listed in table 1 do not affect the number of equilibria nor are there other significant qualitative changes to the results.
function relative to figure 2. The indemnity leads to greater incentives for $\rho$ when $\sigma$ 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 3 is the high-effort equilibrium $B$.

The expectational threshold and the low-effort equilibrium have been eliminated in figure 3 because the indemnity has increased RER for each value of $\sigma$, weakening strategic complementarities at lower effort levels. Indeed, curve RER = 1 now lies to the left of the RER = 1 curve from figure 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 (11)). Since the unique SNE is located to the right of RER = 1 in figure 3, biosecurity is a strategic substitute at equilibrium, and $B$ is globally stable: expectations do not matter. A larger marginal indemnity rate $\zeta$ 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 (9)).

[Figure 3]

Finally, consider a behaviorally-independent indemnity. For the sake of comparison, let the indemnity take the form $\phi(\rho) = 0.55$ 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, (8) is again solved for the best-response function $\rho(\sigma)$, shown in figure 4a. Decoupling the indemnity from behavior has lowered the producer’s incentive to adopt biosecurity for any value of $\sigma$, shifting $\rho(\sigma)$ rightward and downward relative to figure 2. Also, although RER is unaffected—the measure of RER under a behaviorally-independent indemnity is once again given by condition (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 2). The reduced complementarities along with reduced biosecurity incentives has expanded the low-effort basin by shifting the expectational threshold $\sigma_T$ to the right relative to figure 2, increasing the risk of coordination failure. A larger behaviorally-independent indemnity causes the expectational threshold to vanish, as in figure 4b. The only SNE remaining in figure 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 4a–b]

**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 $\zeta$ 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:

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

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
The first LHS term and the RHS term of equation (14) are the same as in the producer’s decentralized first order condition (3) for the case of no indemnity. The second LHS term \(-\Lambda P_\sigma\) accounts for the positive spillovers from \(\rho\) 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 (3) is satisfied as an equality (an interior solution), producers exert too little effort relative to the first-best. The optimal indemnity must therefore incentivize producers to internalize the spillovers arising from their choice of \(\rho\). A decentralized outcome involving a corner solution of \(\rho = \sigma = 1\) will coincide with the first-best. Hence, the socially-optimal level of \(\rho\) is only weakly greater than the level chosen in the decentralized case.

Consider an interior first-best outcome. We can set parameter \(\zeta\) to equate the producer’s first order condition for the case of a decentralized indemnity (equation 8) to that of the command optimum (equation 14) evaluated at the first-best outcome:

\[
(15) \quad -\Lambda P_\sigma(\rho, \sigma)_{\sigma = \rho} - \Lambda P_\sigma(\rho, \sigma)_{\sigma = \rho} = c_\rho(\rho).
\]

where \(\rho^*\) 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 (15) represents the positive spillover effects of biosecurity. The RHS of (15) is the marginal incentive provided by the indemnity.

\[\text{An alternative but equivalent approach involves choosing policy instrument parameters to maximize net economic surplus, conditional on producers’ optimal response to the policy in the decentralized setting (Acocella, Di Bartolomeo, and Hallet 2013).}\]
The first-best value $\zeta^*$ is derived from expression (15) as $\zeta^* = \left[ \partial(1 - P^*)/\partial \sigma \right] / [P^*(1 + \delta^*)]$, where the superscript * means all variables are evaluated at $\sigma = \rho = \rho^*$. The numerator of $\zeta^*$, which is positive, is the aggregate marginal reduction in infection probability from biosecurity. This is normalized by the biosecurity effectiveness term $(1 + \delta^*)$, which may be positive or negative. The optimal rate $\zeta^*$ may therefore be positive or negative, where a negative indemnity is actually a tax on biosecurity that is only imposed in the case of infection (i.e., a state-dependent tax). Note that $\zeta^*$ is unaffected by whether biosecurity is a strategic complement or substitute.

Whether a positive or negative indemnity is required to provide the first-best incentives for biosecurity depends on the magnitude of $\delta^*$. For instance, individuals can be indemnified in the case of an outbreak, with $\zeta^* > 0$. However, the incentives will be weak if the increased self-protection reduces the probability of an outbreak to such an extent that the indemnity is unlikely to be paid. The degree to which the incentives are weakened is given by $\delta^*$, which reflects the effectiveness of self-protection in reducing the likelihood of infection. If $(1 + \delta^*) < 0$, then a positive indemnity will not have the desired effect. In this case, self-protection is incentivized via a negative indemnity, $\zeta^* < 0$. Here, producers have incentives to self-protect to avoid the tax.

The indemnity rate $\zeta^*$ corrects the inefficiency arising from the spillover effects of one’s self-protection on others’ expected profits. However, we argue that $\zeta^*$ is first-best only when this 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), $\zeta^*$ does not guarantee the first-best outcome. The reason is that the linear indemnity rate $\zeta^*$ 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 \( \zeta_i \) are parameters to be chosen optimally. The producer’s decentralized first-order condition (8) may be implicitly solved for the symmetric best-response function conditional on the indemnity, denoted \( \rho(\sigma, \zeta_1, \zeta_2)\big|_{\sigma=\rho^*} \). The first-best policy parameters are then chosen to simultaneously solve the following conditions:

\[
(16a) \quad \rho(\sigma, \zeta_1, \zeta_2)\big|_{\sigma=\rho^*} = \rho^*
\]

\[
(16b) \quad \int_0^{\rho^*} \rho(\sigma, \zeta_1, \zeta_2) d\sigma > \int_0^{\rho^*} \sigma d\sigma \quad \forall \sigma.
\]

Condition (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 \( \rho^* \). 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 (15).

Condition (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.\(^{18}\) Note that (16b) is automatically satisfied when biosecurity is a weak strategic complement or a global strategic substitute.

---

\(^{18}\) 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 \( \rho^* \) must be weakly greater than any decentralized equilibrium, there cannot be a preferred equilibrium above the first-best level, \( \rho^* \). Thus, there cannot exist additional SNE above \( \rho^* \) if condition (16b) is satisfied.
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 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 1b or 1c). An optimal indemnity will shift the best-response function upwards through the first-best outcome. In the case of multiple equilibria in figure 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 (16b) also ensures the new best-response function will intersect the 45° curve from above, guaranteeing that the solution is globally stable.  

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 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-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 5 depicts the effect of a small, behaviorally-dependent indemnity (i.e., \(\phi(\rho) = \frac{1}{2} + \frac{1}{2} \rho\)). 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, \(\zeta_1\), and a positive value of \(\zeta_2\) will shift the producer’s best-response function upwards for all \(\sigma\), increasing his incentives for biosecurity. At the same time, a positive value of \(\zeta_2\) will increase RER to help eliminate the risk of coordination failure. Together, these values can be set to accomplish the two objectives defined by condition (16). An additional parameter, \(\zeta_3\), may be required to satisfy \(\hat{\varphi}(\sigma, \zeta_1, \zeta_2) / \partial \sigma > -1 \forall \sigma\), so as to avoid particularly strong strategic substitute relations that can generate asymmetric equilibria.

\(^{19}\) 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, \(\zeta_1\), and a positive value of \(\zeta_2\) will shift the producer’s best-response function upwards for all \(\sigma\), increasing his incentives for biosecurity. At the same time, a positive value of \(\zeta_2\) will increase RER to help eliminate the risk of coordination failure. Together, these values can be set to accomplish the two objectives defined by condition (16). An additional parameter, \(\zeta_3\), may be required to satisfy \(\hat{\varphi}(\sigma, \zeta_1, \zeta_2) / \partial \sigma > -1 \forall \sigma\), so as to avoid particularly strong strategic substitute relations that can generate asymmetric equilibria.
Like the no-policy case in figure 1, the first-best optimum (point $B$) remains an SNE under this indemnity. The indemnity in figure 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 $\sigma$, shifting the best-response function above the $45^\circ$ 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 will increase the budgetary burden, and may even reduce the SNE level of self-protection and make it unstable.

[Figure 5]

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 article, 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. Understanding these effects can lead to the development of more effective policies.

Another key insight from our analysis is that traditional means of identifying optimal incentive instruments (e.g., indemnities) may not fully address the multiple sources of inefficiency present in decentralized self-protection decisions. Specifically, traditionally-constructed incentives may not address the problem of coordination failure, which can result in sub-optimal self-protection even when the first-best outcome is one potential outcome. We have shown that true first-best indemnities both incentivize the first-best level of self-protection and eliminate the risk of coordination failure, ensuring the first-best outcome is a globally stable Nash equilibrium. When multiple equilibria are present in the decentralized, no-policy case, then global stability is achieved via instruments that increase RER to sufficiently weaken strategic complementarities and guarantee a unique, stable Nash equilibrium. In the specific case of indemnities, we find that the first-best may be achieved using indemnities that are behaviorally-dependent, where payments are increasing in the level of self-protection.

The inability to accurately observe self-protection may make behaviorally-dependent policies difficult to implement. Gramig, Horan, and Wolf (2009) explore the case of indemnification under moral hazard and adverse selection and may provide a guide to the implementation of such policies. More generally, if behaviorally-dependent policies are deemed infeasible, disease management under the possibility of coordination failure becomes more challenging. A regulatory authority could attempt to manage producers’ expectations regarding others’ self-protection. Here, information campaigns and extension efforts geared towards generating “focal points” (Schelling 1980), or conspicuous signals of expectations regarding others’ self-protection behavior, may be effective.
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.). In particular, our qualitative results do not depend on particular specifications for $P^S(\cdot)$ and are valid as long as $P^S(\cdot)$ is decreasing in the self-protection choices of others. 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.

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.

**Supplementary Information (for online publication)**

Here we describe the calibration of the numerical model. We first derive an index of biosecurity investment. Chi et al. (2002) calculate the costs of ten biosecurity practices which 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 organize 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). We assume the correspondence between practice number and \( \rho \) takes the exponential form \( \rho = 1 - \exp(-\lambda \cdot \text{practice#}) \), where \( \text{practice#} \) corresponds to the value assigned to each practice. The nonlinear relationship between \( \rho \) and practice number reflects the assumption that the degree of protection arising from biosecurity investment increases at a decreasing rate. The parameter \( \lambda \) is set to 1.15, as this is the value that sets the left-hand side (LHS) of (A1) approximately equal to 1 when \( \text{practice#} = 4 \), which corresponds with full investment in biosecurity.
Next, consider the probability of infection for a given farmer (equation 1, main article). We begin by parameterizing $P^I(\cdot)$. Green et al. (2006) model the initial spread of FMD from animal movements from livestock markets in northern England. The authors assume one in ten animals purchased from these markets are infected. We assume that this level of risk corresponds to typical biosecurity investment, which we define as conducting background checks on all animals purchased from a market (practice 1, Table A1). Further, 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. Given the functional form $P^I(\rho) = \xi(1 - \mu\rho)$, we set the LHS to 0.1 and set $\mu = 1$. This expression is then solved for $\xi = 0.22$.

The infectivity of contact between herds is given by $k(\rho) = \kappa(1 - \nu\rho)$. The parameter $\nu$ 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 $\nu = 0.15$. Schley et al. (2009) estimate a transmission kernel density describing the probability that a farm becomes infected by spread from an infected premises. Based on this kernel and the high density of farms in Cumbria county, we assume that the probability that a herd gets infected by a neighboring herd under typical biosecurity (practice 1, Table A1) is $k(\cdot) = 0.8$, implying $\kappa = 0.9.$

### Table A1. Description of Biosecurity Practices Used in Numerical Example (Source: Chi et al. 2002)

<table>
<thead>
<tr>
<th>$\rho$</th>
<th>Practice #</th>
<th>Farm</th>
<th>Source of Livestock</th>
<th>Background Checks</th>
<th>Cost (£)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.99</td>
<td>4</td>
<td>Closed</td>
<td>N/A</td>
<td>N/A</td>
<td>2,446</td>
</tr>
<tr>
<td>0.97</td>
<td>3</td>
<td>Open</td>
<td>Other producer</td>
<td>Yes</td>
<td>1,694</td>
</tr>
<tr>
<td>0.90</td>
<td>2</td>
<td>Open</td>
<td>Other producer</td>
<td>No</td>
<td>1,519</td>
</tr>
<tr>
<td>0.68</td>
<td>1</td>
<td>Open</td>
<td>Dealer/auction</td>
<td>Yes</td>
<td>173</td>
</tr>
<tr>
<td>0.00</td>
<td>0</td>
<td>Open</td>
<td>Dealer/auction</td>
<td>No</td>
<td>0</td>
</tr>
</tbody>
</table>
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 $\times$ 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 with 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 $\alpha = 0.75$.

Next, we calibrate the biosecurity cost function, $c(\rho) = \chi \rho^2$. The cost of each biosecurity practice is given in table A1. Taking the natural logarithm of both sides of $c(\rho)$ and assuming a lognormally-distributed error term $\omega$, we get an estimable function $\ln c = \hat{\gamma}_0 + \hat{\gamma}_1 \ln \rho + \hat{\alpha}$, where the symbol ($\hat{\cdot}$) denotes an estimated parameter. Note that $\ln \hat{\chi} = \hat{\gamma}_0$ and, by hypothesis, $\hat{\gamma}_1 = 2$. Using ordinary least squares, we estimate $\hat{\gamma}_0 = 7.19$ (s.e. = 0.44)—implying $\hat{\chi} = 1,328$—and $\hat{\gamma}_1$.

35
= 1.88 (s.e. = 0.11), which is not significantly different from 2 ($R^2 = 0.995, F = 299.64$).

Finally, we calculate the representative producer’s cost of infection, $\Lambda$, which we take to be the value of the herd (assuming all animals within an infected herd are culled) and idling factors of production. Thompson et al. (2002) estimate the total cost of the 2001 UK epidemic to producers at £1.2 billion and the total number of animals slaughtered at 6.124 million, so that the total cost per animal is £188.6. We multiply this figure by 140, which is the average herd size in Cumbria county (Defra 2011). This results in a total cost to the producer of $\Lambda = £26,404$. 
References


Gibbens, J.C., C.E. Sharpe, J.W. Wilesmith, C.E. Sharpe, L.M. Mansley, E. Michalopoulou,


Journal of Agricultural Economics 89:1226–1231.


Table 1. Functional Forms and Parameters Used for the Numerical Example\(^a\)

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Domain</th>
<th>Form/Value in Numerical Example(^b)</th>
<th>Description</th>
<th>Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>ξ</td>
<td>[0, 1]</td>
<td>0.22(^*)#</td>
<td>Probability of introducing infected animal, no biosecurity</td>
<td>Unitless</td>
</tr>
<tr>
<td>μ</td>
<td>[0, 1]</td>
<td>1</td>
<td>% reduction in probability of introduction per unit of biosecurity</td>
<td>Unitless</td>
</tr>
<tr>
<td>κ</td>
<td>[0, 1]</td>
<td>0.9(^*)#</td>
<td>Probability of infection given contact occurs</td>
<td>Unitless</td>
</tr>
<tr>
<td>ν</td>
<td>[0, 1]</td>
<td>0.15</td>
<td>% reduction in probability of infection from contact per unit of biosecurity</td>
<td>Unitless</td>
</tr>
<tr>
<td>χ</td>
<td>≥ 0</td>
<td>1,328(^*)</td>
<td>Cost parameter</td>
<td>$</td>
</tr>
<tr>
<td>Λ = RH - RN(^N)</td>
<td>[0, ∞)</td>
<td>26,404(^i)</td>
<td>Cost of infection</td>
<td>$</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Variables</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>ρ</td>
<td>[0, 1]</td>
<td>—</td>
<td>The producer’s biosecurity effort</td>
<td>Units of effort</td>
</tr>
<tr>
<td>σ</td>
<td>[0, 1]</td>
<td>—</td>
<td>Neighboring producers’ biosecurity effort</td>
<td>Units of effort</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Functions</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>P(^I)(ρ), P(^I)(σ)</td>
<td>[0, 1]</td>
<td>(P(^I)(ρ) = ξ(1 – μρ), P(^I)(σ) = ξ(1 – μσ))</td>
<td>Probability of introducing infected animal to farm</td>
<td>Unitless</td>
</tr>
<tr>
<td>P(^S)(ρ, σ)</td>
<td>[0, 1]</td>
<td>(P(^S)(ρ, σ) = 1 – [1 – k(ρ)P(^I)(σ)]^N(ρ, σ))</td>
<td>Probability of infection from spread</td>
<td>Unitless</td>
</tr>
<tr>
<td>k(ρ)</td>
<td>[0, 1]</td>
<td>(k = κ(1 – νρ))</td>
<td>Probability herd becomes infected given contact with infected herd</td>
<td>Unitless</td>
</tr>
<tr>
<td>(N(ρ, σ))</td>
<td>[0, N]</td>
<td>(N(ρ, σ) = (N – 1)(1 – αρ)(1 – ασ))</td>
<td>Effective number of neighboring farms</td>
<td>Farms</td>
</tr>
<tr>
<td>(E{π})</td>
<td>(ℝ)</td>
<td>(E{π} = RH – ΛP(ρ, σ) – c(ρ))</td>
<td>Expected profit</td>
<td>$</td>
</tr>
<tr>
<td>c(ρ)</td>
<td>[0, ∞)</td>
<td>(c(ρ) = χρ^2)</td>
<td>Cost of biosecurity effort</td>
<td>$</td>
</tr>
</tbody>
</table>

\(^a\) See supplemental appendix online for derivation and discussion of parameter values and functional forms.

\(^b\) References: \(^*\) Chi et al. (2002); \(^#\) Green et al. (2006); \(^\#\) Schley et al. (2009); \(^\dagger\) Brennan et al. (2008); \(^\dagger\) Defra (2011); \(^\dagger\) Thompson et al. (2002); \(^\dagger\) Ferguson et al. (2001). Entries without superscripts are assumed values.
Figure 1. a) Multiple symmetric equilibria can arise when biosecurity is a strategic complement with relatively strong complementarities arising at SNE C, where $\rho_\sigma(\sigma) > 1$. In this case, low expectations of neighbors’ biosecurity efforts can lead to coordination failure (point A). Alternatively, a single, globally-stable SNE (A) can arise when biosecurity is b) a relatively weak strategic complement with $\rho_\sigma(\sigma) < 1$ at the SNE; or c) a strategic substitute and $|\rho_\sigma(\sigma)| < 1$
Figure 2. The representative producer’s best response function $\rho(\sigma)$ is discontinuous at the expectational threshold $\sigma_T$, which represents the minimum expected biosecurity effort that will achieve (via tâtonnement) the preferred symmetric Nash equilibrium $B$; biosecurity is a strategic substitute at $B$ (i.e., RER $> 1$) and a strategic complement at equilibrium $A$ (i.e., RER $< 1$)
Figure 3. A behaviorally-dependent indemnity increases RER and may eliminate the low-effort basin, making the preferred equilibrium $B$ globally stable.
Figure 4. a) A small, behaviorally-independent indemnity has no effect on RER but reduces producer incentives for biosecurity effort, resulting in a larger low-effort basin relative to the no-policy case; b) A large behaviorally-independent indemnity eliminates the expectational threshold, resulting in a unique Nash equilibrium at low levels of biosecurity.
Figure 5. A linear indemnity with a single, optimal chosen parameter will make the first-best outcome globally stable when both the decentralized and first-best equilibrium is a corner solution.