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Abstract

In this paper, we investigate the nature of rational expectations equilibria for economic epidemiological models. Unlike mathematical epidemiological models, economic epidemiological models can produce regions of indeterminacy or instability around the endemic steady state. We consider SI, SIS, SIR and SIRS versions of economic compartmental models and show how well-intentioned public policy may contribute to disease instability and uncertainty.

JEL Codes: D1, I1.

Keywords: economic epidemiology, equilibria, dynamics, disease, indeterminacy, rational expectations

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1 Introduction

In this paper we investigate the dynamic properties of rational expectations economic epidemiological (EE) models. The economic epidemiology field integrates traditional mathematical epidemiology (mathematical modeling of disease transmission; Anderson and May (1991)) and economic choice (rational decision making). Economic research in this area began in response to the AIDS epidemic and has led to an improved understanding of how decision making by individuals and policymakers influences infectious disease dynamics. For example, policymakers may have limited ability to eradicate infectious diseases if rational individuals respond to lower prevalence by reducing protection (Geoffard and Philipson (1996)) or may increase disease prevalence and induce fatalistic behavior with the introduction of imperfect vaccines (Kremer (1996)). These examples highlight the need to understand how economic incentives can alter policy prescriptions in the presence of infectious diseases.

Our focus is on the stability properties of rational expectations EE equilibria and the relationship to public health policy.[1] Similar to the macroeconomic literature on the stability properties of monetary and fiscal policy (Guo and Lansing (1998); Clarida, Gali, and Gertler (2000); Fatás and Mihov (2003)), we show that well-intentioned public policy has the potential to contribute to aggregate instability and volatility. For instance, government policy designed to lower the transmission probability or raise the quality-of-life associated with infectious diseases can push the EE system from a stable equilibrium path to ones exhibiting instability or indeterminate equilibrium paths. The latter also have the potential of contributing to self-fulfilling "sunspot" equilibria. To the best of our knowledge, this is a new finding in the EE literature and an additional reason for policymakers to consider the predictions of integrated economic and epidemiological models.

2 Economic Epidemiological Model

Following work by Philipson and Posner (1993), we specify an integrated economic epidemiological model to describe communicable disease dynamics. The model is set in discrete time (Auld (2003)), where t indexes the decision interval.[2] There is a constant population of N individuals, which are all identical except for their state of the disease.

[1] The stability properties of continuous-time epidemiological systems have been studied in detail (see e.g., Korobeinikov and Wake (2002)). In general, the endemic equilibrium has been found to be globally stable.

[2] Allen (1994) finds that endemic equilibria from discrete-time epidemiological models have the potential to be stable, exhibit periodicity or be chaotic. Instability tends to be driven by high contact rates and high birth/death rates per time interval.

2.1 Epidemiology

The epidemiological portion of the model describes the evolution of three mutually exclusive disease categories: susceptible (s), infected (in), and recovered with immunity (r). This is the classical SIRS model (Anderson and May (1991)) where individuals transition from being susceptible to infected to recovered (and immune) and then back to susceptible. The SIRS model has previously been used to model infectious diseases such as syphilis and whooping cough (Grassly, Fraser, and Garnett (2005); Rohani, Zhong, and King (2010)). The SIRS model is sufficiently general to handle cases with permanent infection (SI diseases such HIV/AIDS), diseases with recovery but no immunity (SIS diseases such as the common cold), and diseases with permanent immunity (SIR diseases such as measles and chicken pox).

Each disease category is measured as a proportion of the overall population with the categories summing to one. The epidemiological model is represented by three equations:

$$s_{t+1} = \mu + (1 - p_t - \mu)s_t + \gamma r_t \tag{1}$$

$$in_{t+1} = (1 - v - \mu)in_t + p_t s_t \tag{2}$$

$$r_{t+1} = (1 - \gamma - \mu)r_t + vin_t, \tag{3}$$

where μ is the common birth/death rate, $1/\gamma$ is the average duration of immunity, v is the recovery rate, and p_t is the probability of infection. The SIR model sets $\gamma = 0$ so that individuals are permanently recovered and immune to the disease. The SIS and SI models omit the immunity category and are treated in the Appendix.

Assuming that individuals independently choose x_t contacts and engage in a fixed number of interactions (a) with each contact, the probability that susceptible individuals become infected is

$$p_t = \Pr(\text{infection}) = 1 - (1 - \lambda_p in_t)^{x_t}, \tag{4}$$

where $\lambda_p = 1 - (1 - \lambda_a)^a$ is the probability of contracting the disease from a single infected contact, and λ_a is the probability of contracting the disease from a single interaction with an infected contact (Kaplan (1990); Oster (2005)). The dependence on the chosen number of contacts distinguishes the analysis from standard mathematical epidemiology.

We now turn our attention to the economic analysis and the optimal choice of contacts.

2.2 Economics

Representative individual i maximizes expected lifetime utility by choosing the number of contacts, $x_{i,t}$. The objective function is

$$E_t \sum_{j=0}^{\infty} \beta^j [\ln(x_{i,t+j}) + h_{i,t+j}] \tag{5}$$

where $0 < \beta < 1$ is the discount factor, E_t represents an individual's rational expectation of future outcomes conditional on all information dated t and earlier, and $\bar{x}$ is the maximum number of contacts per period. The parameter $h_{i,t}$ captures the individual's health status with infected individuals experiencing lower values of h. The core tradeoff in the model is that additional contacts bring immediate satisfaction but also the risk of future infection. Infection in turn causes a deterioration of health.

In any period t, individual i is in one of three epidemiological states as measured by the binary variables: susceptible ($s_{i,t}$), infected ($in_{i,t}$), or recovered and immune ($r_{i,t}$). The proportions of susceptible, infected and recovered individuals in the entire population are given by averaging over all i. Because all individuals are identical other than disease state and health level, we drop the i subscript and consider a single representative individual in each disease category.

The value functions for each category – susceptible, infected, and recovered – are given by

$$V_t^S = \ln(x_t) + h^S + \beta E_t[p_t V_{t+1}^{IN} + (1 - p_t) V_{t+1}^S] \tag{6}$$

$$V_t^{IN} = \ln(\bar{x}) + h^{IN} + \beta E_t[v V_{t+1}^R + (1 - v) V_{t+1}^{IN}] \tag{7}$$

$$V_t^R = \ln(\bar{x}) + h^S + \beta E_t[\gamma V_{t+1}^S + (1 - \gamma) V_{t+1}^R], \tag{8}$$

where $h^S > h^{IN}$.

All individuals maximize (5) without concern for the welfare of the general population. Infected and immune individuals will therefore choose the maximum number of contacts, $\bar{x}$, because they face no risk of immediate infection (Geoffard and Philipson (1996)). Assuming an interior solution, susceptible individuals will choose the number of contacts to satisfy the Euler equation:

$$x_t^{-1} = \beta p_{x,t} E_t[V_{t+1}^S - V_{t+1}^{IN}], \tag{9}$$

where the partial derivative of p_t with respect to the number of contacts is $p_{x,t} = -\ln(1 - p_t)(1 - p_t)/x_t$.[3]

[3] The second-order sufficiency conditions require that the marginal cost curve with respect to contacts (right side of equation (9)) must slope up or if it slopes down, it must be locally flatter than the marginal benefit curve (left side of equation (9)).

The contact rate in mathematical epidemiological models is typically constant or varies deterministically (Korobeinikov (2006)). As equation (9) shows, the contact rate in EE models is instead based on behavioral responses to changes in disease risk**.** We look at two cases depending on individuals' ability to observe their own host immunity.

2.2.1 Unobservable Host Immunity

In this case, individuals with host immunity believe they are susceptible. Therefore, equation (8) is not relevant and equation (7) becomes

$$V_t^{IN} = \ln(\bar{x}) + h^{IN} + \beta E_t[vV_{t+1}^S + (1-v)V_{t+1}^{IN}]. \tag{10}$$

Substituting out the value functions (V_{t+1}^S and V_{t+1}^{IN}), equation (9) can be rewritten as

$$x_t^{-1} = \beta p_{x,t} E_t \left[\ln(x_{t+1}/\bar{x}) + h + \frac{(1-v-p_{t+1})}{x_{t+1}p_{x,t+1}}\right], \tag{11}$$

where $h = h^S - h^{IN}$ is the health gap between being susceptible and infected. This equation states that rational individuals choose the number of contacts to balance the marginal benefits (left side) with the discounted, expected costs (right side).

2.2.2 Observable Host Immunity

When individuals observe their own immunity, they rationally choose the maximum number of contacts $\bar{x}$ and have health level h^S. Susceptible individuals, on the other hand, choose x_t to satisfy

$$x_t^{-1} = \beta p_{x,t} E_t \left[\ln(x_{t+1}/\bar{x}) + h + \frac{(1-v-p_{t+1})}{x_{t+1}p_{x,t+1}} - \beta\Delta_{t+2}\right], \tag{12}$$

where

$$\Delta_{t+2} = \frac{v\gamma}{x_{t+2}p_{x,t+2}} + (1-v-\gamma)\left[\ln\left(\frac{x_{t+2}}{\bar{x}}\right) + \frac{1-p_{t+2}}{x_{t+2}p_{x,t+2}}\right] + (1-\gamma)\left[h - \frac{1}{\beta x_{t+1}p_{x,t+1}}\right].$$

The Euler equations in (11) and (12) are identical except for Δ_{t+2}. This term captures the expected future "costs" of infection associated with observed acquired immunity. Because Δ_{t+2} enters the right side of (12) with a negative sign, the possibility of future immunity is a benefit of becoming infected.

3 Equilibria

We focus on the nature of the transition dynamics around the endemic EE steady state.[4]

3.1 Steady State

The endemic steady state solves time-invariant versions of (1), (2), (3) and the Euler equation. The Euler equation either takes the form of (11) when the indicator variable ϕ is zero or the form of (12) when $\phi = 1$. The steady-state system can therefore be rewritten as four equations:

$$
\begin{aligned}
s &= (\mu + \gamma r)/(1-p) && (13)\\
in &= ps/(v+\mu) && (14)\\
r &= vin/(\gamma+\mu) && (15)\\
x^{-1} &= \beta[p_x(\ln(x/\bar{x}) + h - \phi\beta\Delta) + (1-v-p)/x] && (16)
\end{aligned}
$$

in four unknown variables (s, r, in, x) where

$$
\Delta = \frac{1}{p_x x}[v\gamma + (1-v-\gamma)(1-p) - (1-\gamma)/\beta] + (1-v-\gamma)\ln(x/\bar{x}) + (1-\gamma)h.
$$

3.2 Transition Dynamics

To analyze the transition dynamics, we linearize around the endemic steady state:

$$
\begin{aligned}
\hat{s}_{t+1} &= (1-p-\mu)\hat{s}_t + \gamma\hat{r}_t - s\hat{p}_t && (17)\\
\widehat{in}_{t+1} &= (1-v-\mu)\widehat{in}_t + s\hat{p}_t + p\hat{s}_t && (18)\\
\hat{r}_{t+1} &= (1-\gamma-\mu)\hat{r}_t + v\widehat{in}_t, && (19)
\end{aligned}
$$

[4] There is also an eradication steady state where $in = r = 0$, $s = 1$, and $x = \bar{x}$. In general, the economic eradication steady state is locally unstable because susceptible individuals have no incentive to reduce the number of contacts or engage in preventative behavior.

where hats ($\hat{}$) over the variables indicate deviation from the steady state. The linearized Euler equation is:

$$\begin{aligned} p_x\hat{x}_t + x\hat{p}_{x,t} &= \beta p_x(1-v-p-xp_x)E_t\hat{x}_{t+1} + \beta x(1-v-p)E_t\hat{p}_{x,t+1} + \beta x p_x E_t\hat{p}_{t+1} \\ &+\phi\beta^2 E_t \left\{ \begin{array}{c} p_x[v\gamma + (1-v-\gamma)(1-p-xp_x)]\hat{x}_{t+2} + x[v\gamma + (1-v-\gamma)(1-p)]\hat{p}_{x,t+2} + \\ [(1-v-\gamma)xp_x]\hat{p}_{t+2} - [(1-\gamma)p_x/\beta]\hat{x}_{t+1} - [(1-\gamma)x/\beta]\hat{p}_{x,t+1} \end{array} \right\} \end{aligned} \tag{20}$$

where

$$\hat{p}_t = p_{in}\widehat{in}_t + p_x\hat{x}_t \tag{21}$$

$$\hat{p}_{x,t} = [(1+\ln[1-p])/x]\hat{p}_t - (p_x/x)\hat{x}_t \tag{22}$$

and

$$p_{in} = x\lambda_p(1-\lambda_p in)^{x-1} \tag{23}$$

$$p_x = -\ln(1-p)(1-p)/x. \tag{24}$$

Using the restriction $\hat{s}_t = -\hat{r}_t - \widehat{in}_t$ and imposing perfect foresight, the $\phi = 0$ linearized EE matrix system is:

$$\begin{aligned} &\underbrace{\begin{bmatrix} 0 & 1-v-\mu-p & -p \\ 0 & v & 1-\gamma-\mu \\ p_x & 0 & 0 \end{bmatrix}}_{A} \begin{bmatrix} \hat{x}_t \\ \widehat{in}_t \\ \hat{r}_t \end{bmatrix} + \underbrace{\begin{bmatrix} s & 0 \\ 0 & 0 \\ 0 & x \end{bmatrix}}_{B} \begin{bmatrix} \hat{p}_t \\ \hat{p}_{x,t} \end{bmatrix} \\ &= \underbrace{\begin{bmatrix} 0 & 1 & 0 \\ 0 & 0 & 1 \\ \beta p_x(1-v-p-xp_x) & 0 & 0 \end{bmatrix}}_{C} \begin{bmatrix} \hat{x}_{t+1} \\ \widehat{in}_{t+1} \\ \hat{r}_{t+1} \end{bmatrix} + \underbrace{\begin{bmatrix} 0 & 0 \\ 0 & 0 \\ \beta x p_x & \beta x(1-v-p) \end{bmatrix}}_{D} \begin{bmatrix} \hat{p}_{t+1} \\ \hat{p}_{x,t+1} \end{bmatrix} \end{aligned} \tag{25}$$

and

$$\underbrace{\begin{bmatrix} -1 & 0 \\ -(1+\ln(1-p))/x & 1 \end{bmatrix}}_{F} \begin{bmatrix} \hat{p}_t \\ \hat{p}_{x,t} \end{bmatrix} = -\underbrace{\begin{bmatrix} p_x & p_{in} & 0 \\ p_x/x & 0 & 0 \end{bmatrix}}_{G} \begin{bmatrix} \hat{x}_t \\ \widehat{in}_t \\ \hat{r}_t \end{bmatrix}. \tag{26}$$

When $\phi = 1$, we have

$$\underbrace{\begin{bmatrix} 0 & 1-v-\mu-p & -p & 0 & 0 \\ 0 & v & 1-\mu-\gamma & 0 & 0 \\ p_x & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 1 \end{bmatrix}}_{A} \begin{bmatrix} \hat{x}_t \\ \widehat{in}_t \\ \hat{r}_t \\ \hat{x}_{t+1} \\ \widehat{in}_{t+1} \end{bmatrix} + \underbrace{\begin{bmatrix} s & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & x & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}}_{B} \begin{bmatrix} \hat{p}_t \\ \hat{p}_{x,t} \\ \hat{p}_{t+1} \\ \hat{p}_{x,t+1} \end{bmatrix} \tag{27}$$

$$= \underbrace{\begin{bmatrix} 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 \\ \beta p_x(1-v-p-xp_x) - \beta^2[(1-\gamma)p_x/\beta] & 0 & 0 & \beta^2 p_x[v\gamma + (1-v-\gamma)(1-p-xp_x)] & 0 \\ 1 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 \end{bmatrix}}_{C} \begin{bmatrix} \hat{x}_{t+1} \\ \widehat{in}_{t+1} \\ \hat{r}_{t+1} \\ \hat{x}_{t+2} \\ \widehat{in}_{t+2} \end{bmatrix} +$$

$$\underbrace{\begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ \beta x p_x & \beta x(1-v-p) - \beta^2[(1-\gamma)x/\beta] & \beta^2(1-v-\gamma)xp_x & \beta^2 x[v\gamma + (1-v-\gamma)(1-p)] \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}}_{D} \begin{bmatrix} \hat{p}_{t+1} \\ \hat{p}_{x,t+1} \\ \hat{p}_{t+2} \\ \hat{p}_{x,t+2} \end{bmatrix}$$

and

$$\underbrace{\begin{bmatrix} -1 & 0 & 0 & 0 \\ -(1+\ln(1-p))/x & 1 & 0 & 0 \\ 0 & 0 & -1 & 0 \\ 0 & 0 & -(1+\ln(1-p))/x & 1 \end{bmatrix}}_{F} \begin{bmatrix} \hat{p}_t \\ \hat{p}_{x,t} \\ \hat{p}_{t+1} \\ \hat{p}_{x,t+1} \end{bmatrix} = - \underbrace{\begin{bmatrix} p_x & p_{in} & 0 & 0 & 0 \\ p_x/x & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & p_x & p_{in} \\ 0 & 0 & 0 & p_x/x & 0 \end{bmatrix}}_{G} \begin{bmatrix} \hat{x}_t \\ \widehat{in}_t \\ \hat{r}_t \\ \hat{x}_{t+1} \\ \widehat{in}_{t+1} \end{bmatrix}. \tag{28}$$

If we let $\hat{z}_t = (\hat{x}_t, \widehat{in}_t, \hat{r}_t)'$ or $\hat{z}_t = (\hat{x}_t, \widehat{in}_t, \hat{r}_t, \hat{x}_{t+1}, \widehat{in}_{t+1})'$ then the EE system reduces to

$$\hat{z}_t = J\, \hat{z}_{t+1} \tag{29}$$

where

$$J = (A - BF^{-1}G)^{-1}(C - DF^{-1}G).$$

We use the method of Blanchard and Kahn (1980) to analyze the nature of the rational expectation EE equilibrium. The three-variable system (29) contains one jump ($\hat{x}_t$) and two predetermined ($\widehat{in}_t$ and $\hat{r}_t$) variables. The system will exhibit saddle-path stability if there are two eigenvalues of J outside the unit circle, indeterminate multiple stable paths if there are no forward stable eigenvalues, and explosive paths if there is more than one forward-stable eigenvalue. The five-variable system contains three jump ($\hat{x}_t$, $\hat{x}_{t+1}$ and $\widehat{in}_{t+1}$) and two predetermined ($\widehat{in}_t$ and $\hat{r}_t$) variables. The fifth equation in (27) is an identity for $\widehat{in}_{t+1}$with a zero eigenvalue. Considering the other four eigenvalues, the system will exhibit saddle-path stability if exactly two of the eigenvalues are outside the unit circle, indeterminate multiple stable paths if there are three or more eigenvalues outside the unit circle, and explosive paths if there is less than two eigenvalues outside the unit circle.

3.3 Parameter Values and Mathematical Program

The parameter values in Table 1 are fixed and not calibrated to a particular disease.

Table 1. Fixed Parameter Values

Parameters	β	μ	v	γ	$\bar{x}$
Value	0.96	0.05	1	0.2	10

The value of β implies a 4% annual discount rate, μ gives a 5% birth and death rate for the population, v implies a 100% recovery rate within a year of infection, γ gives an expected 5-year immunity duration, and the maximum number of feasible annual partners is 10.

4 Results and Policy Implications

Figures 1-3 show the types of dynamic paths for the EE and ME models under a range of values for the health gap (h) and infection rate (λ_p). These two parameters represent possible public health policy targets. The health gap parameter ($h = h^S - h^{IN}$) can be lowered through the discovery and introduction of drug treatments, while the infection rate (λ_p) can be lowered through the introduction of vaccines or new protection technologies.

Figure 1 shows the map of path types for the SI and SIS models around the endemic steady state. The top panels show the type of localized dynamic paths for the economic and mathematical SI models with

no available treatment, $v = 0$. The majority of the parameter space for the economic SI model is defined by saddle-path equilibria. For a given initial prevalence level (in_0), there is a unique initial contact choice (x_0) that puts the system on a convergent equilibrium path to the endemic steady state. All other initial contact levels lead to divergent paths that violate non-negativity or non-explosion conditions. Parameter combinations with low values for the health gap h and high values of the infection parameter λ_p lead to stable equilibrium where individuals choose the maximum number of contacts. Because individuals in this parameter region do not vary their choice of contacts in response to changes in disease prevalence, the model collapses to a mathematical epidemiological SI system. The mathematical SI model (upper right panel) is characterized by stable dynamic paths with a fixed number of contacts and no dependence on the health gap, h. The bottom panels of Figure 1 show the path types for the SIS models where infected individuals have access to perfectly effective treatment ($v = 1$) and return to the susceptible pool after treatment. In addition to the saddle-path region, the economic SIS model also contains a parameter region where the dynamic path near the endemic steady state is explosive. Public health policy aimed at improving the health of infected individuals could inadvertently move the system from a stable saddle-path region to an explosive system with higher prevalence as individuals rationally take more risk.

To gain intuition for the types of dynamic paths in the economic and mathematical SIS models, consider a simple heuristic, din_{t+1}/din_t, relating changes in future prevalence to a change in current prevalence. Nearby the endemic steady state, this metric is given by

$$\frac{d\widehat{in}_{t+1}}{d\widehat{in}_t} = (1 - v - \mu) + (1 - in)\left(p_{in} + \kappa p_x x/in\right), \tag{30}$$

where κ is the contact elasticity with respect to prevalence. Prevalence elasticity measures the percentage change in contacts for a one-percent change in prevalence. This elasticity is generally negative, indicating that susceptible individuals respond to the increased risk of infection by choosing fewer contacts.[5] Whether the economic SIS system depicted in the lower left panel of Figure 1 is saddle-path stable or explosive depends on the magnitude of κ. The critical prevalence elasticity along the stable-explosive boundary can be found by setting (30) equal to -1 and solving for κ:

$$\kappa_c = -\frac{in}{p_x x}\frac{[1 + (1 - v - \mu) + p_{in}(1 - in)]}{1 - in}.$$

[5] Kremer (1996) discusses the possibility of a positive prevalence elasticity and fatalistic behavior.

For the parameter values in Table 1, the critical prevalence elasticity is approximately $\kappa_c = -2$. Parameter combinations in the SIS saddle-path region are associated with prevalence elasticities that have a smaller magnitude than κ_c while parameter combinations in the SIS explosive region are associated with prevalence elasticities larger in magnitude than κ_c. If an increase in disease prevalence triggers susceptible individuals to reduce the percentage of contacts by twice the initial increase in prevalence, then prevalence at $t+1$ falls more than the initial rise in prevalence at t. As a result, the system oscillates in an explosive manner. Conversely, prevalence elasticity is zero in the mathematical SIS model because susceptible individuals do not alter their behavior in response to changes in disease prevalence. This implies that

$$\frac{d\widehat{in}_{t+1}}{d\widehat{in}_t} = (1 - v - \mu) + (1 - in)p_{in},$$

which is positive and less than one for all values of the infection parameter λ_p in Figure 1. Increases in prevalence near the endemic steady state cause the mathematical SIS system to convergence monotonically back to the endemic steady state.

Figure 2 shows a similar map for the dynamic paths in SIR and SIRS models where immunity is unobservable. The top panels show the equilibrium types for the economic and mathematical SIR models with permanent immunity, $\gamma = 0$. For both the economic and mathematical model, the entire range of parameter combinations results in stable equilibria. For the economic epidemiological model, parameter combinations with high h and high λ_p produce stable saddle-path dynamics. In this region, individuals respond to the higher disease prevalence by rationally reducing their contacts below the maximum allowable contacts. The lower panels treat the economic and mathematical SIRS models with $\gamma = 0.2$ (i.e., average immunity duration of five years). Unlike the economic SIS model, the EE SIRS model produces a region of indeterminacy for high λ_p and moderate h where there are multiple equilibrium paths and the possibility of "sunspot" equilibria (Benhabib and Farmer (1999)). Sunspot equilibria are often associated with self-fulfilling prophecies and additional aggregate volatility.

Figure 3 depicts the SIRS counterpart to Figure 2 but with observable host immunity. Observable host immunity causes two primary changes. First, individuals in the EE SIR system take the maximum number of contacts for any parameter combination. Knowledge of perfectly effective treatment and permanent immunity greatly reduces the future cost of current risky behavior. Second, the indeterminacy region for the EE SIRS system now covers a large range of health gap parameters. Thus, public health policy aimed at improving the quality of life for individuals infected with diseases that have known temporary immunity

may encourage additional risk-taking and induce aggregate instability and indeterminacy.

5 Conclusion

Economic epidemiology has made significant advances in educating health officials about the behavioral implications of public policies. However, one area that has received little attention is how policy influences the nature of communicable disease dynamics as the system transitions toward the endemic long-run equilibrium. In this paper, we explore the nature of the short-run equilibrium dynamics for rational expectations economic epidemiological systems. The analysis digs beneath a comparison of fixed parameter values and demonstrates the behavioral origin for changes in the dynamical properties of the system. Indeed, we show that well-intentioned policy has the potential to create instability and indeterminacy when individuals behave rationally and in a self-interested manner. Future research should focus on providing precise policy recommendations by applying and calibrating the methods outlined in this paper to specific diseases.

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Appendix A. SIS Economic Epidemiological System

Here we describe the SIS economic epidemiological model. In the SIS model, infected individuals transition directly back to the susceptible category and do not experience a period of immunity. The SIS dynamic equations are

$$s_{t+1} = \mu + (1 - p_t - \mu)s_t + v in_t \tag{A.1}$$

$$in_{t+1} = (1 - v - \mu)in_t + p_t s_t, \tag{A.2}$$

while the steady-state values are

$$s = (v + \mu)/(p + v + \mu) \tag{A.3}$$

$$in = p/(p + v + \mu). \tag{A.4}$$

The SI model is defined by $v = 0$ so that infection is permanent. The value functions are

$$V_t^S = \ln(x_t) + h + \beta E_t[p_t V_{t+1}^{IN} + (1 - p_t)V_{t+1}^S] \tag{A.5}$$

$$V_t^{IN} = \ln(\bar{x}) + \beta E_t[v V_{t+1}^S + (1 - v)V_{t+1}^{IN}]. \tag{A.6}$$

The Euler equation for susceptible individuals is given by equation (9) in the main text. Using (A.5) and (A.6) to substitute out the value functions, the Euler equation can be rewritten as

$$x_t^{-1} = \beta p_{x,t} E_t \left[\ln(x_{t+1}/\bar{x}) + h + \frac{(1 - v - p_{t+1})}{x_{t+1} p_{x,t+1}}\right]. \tag{A.7}$$

The linearized EE system is

$$\widehat{in}_{t+1} = (1 - v - \mu)\widehat{in}_t + (1 - in)\hat{p}_t \tag{A.8}$$

$$p_x \hat{x}_t + x\hat{p}_{x,t} = \beta p_x (1 - v - p - x p_x) E_t \hat{x}_{t+1} + \beta x (1 - v - p) E_t \hat{p}_{x,t+1} + \beta x p_x E_t \hat{p}_{t+1} \tag{A.9}$$

along with equations (21) and (22). Assuming perfect foresight, equations (A.8) and (A.9) in matrix form are

$$\underbrace{\begin{bmatrix} 0 & 1-v-\mu-p \\ p_x & 0 \end{bmatrix}}_{A} \begin{bmatrix} \hat{x}_t \\ \widehat{in}_t \end{bmatrix} + \underbrace{\begin{bmatrix} 1-in & 0 \\ 0 & x \end{bmatrix}}_{B} \begin{bmatrix} \hat{p}_t \\ \hat{p}_{x,t} \end{bmatrix}$$

$$= \underbrace{\begin{bmatrix} 0 & 1 \\ \beta p_x(1-v-p-xp_x) & 0 \end{bmatrix}}_{C} \begin{bmatrix} \hat{x}_{t+1} \\ \widehat{in}_{t+1} \end{bmatrix} + \underbrace{\begin{bmatrix} 0 & 0 \\ \beta x p_x & \beta x(1-v-p) \end{bmatrix}}_{D} \begin{bmatrix} \hat{p}_{t+1} \\ \hat{p}_{x,t+1} \end{bmatrix}. \tag{A.10}$$

Along with (26), the SIS economic epidemiological model can then be written in the form of equation (29) where the coefficient matrices A, B, C and D are redefined and $\hat{z}_t = (\hat{x}_t, \widehat{in}_t)$.

Appendix B. Derivation of the Economic SIRS Euler Equation with Observable Immunity

In Appendix B, we derive the Euler equation for the economic SIRS model with observable immunity. To begin, note that equations (7) and (8) imply

$$V_t^R - V_t^{IN} = h + \beta E_t \left[\gamma(V_{t+1}^S - V_{t+1}^R) + (1-v)(V_{t+1}^R - V_{t+1}^{IN})\right], \tag{A.11}$$

while equations (6) and (7) imply

$$V_t^S - V_t^{IN} = \ln(x_t/\bar{x}) + h + \beta E_t \left[(1-p_t)(V_{t+1}^S - V_{t+1}^{IN}) - v(V_{t+1}^R - V_{t+1}^{IN})\right]. \tag{A.12}$$

Using equation (9), we have

$$E_t(V_{t+1}^S - V_{t+1}^{IN}) = (\beta x_t p_{x,t})^{-1}, \tag{A.13}$$

for all t. Next, rearrange (A.12) as

$$V_{t+1}^R - V_{t+1}^{IN} = \frac{1}{\beta v}[\ln(x_t/\bar{x}) + h] + \frac{1}{v}(1-p_t)E_t(V_{t+1}^S - V_{t+1}^{IN}) - \frac{1}{\beta v}\left(V_t^S - V_t^{IN}\right). \tag{A.14}$$

Take E_{t-1} on both sides of (A.14) and substitute (A.13) to get

$$E_{t-1}(V_{t+1}^R - V_{t+1}^{IN}) = \frac{1}{\beta v}E_{t-1}[\ln(x_t/\bar{x}) + h] + \frac{1}{\beta v}E_{t-1}\left(\frac{1-p_t}{x_t p_{x,t}}\right) - \frac{1}{\beta^2 v}\left(\frac{1}{x_{t-1}p_{x,t-1}}\right). \tag{A.15}$$

Now rewrite equation (A.11) as

$$V_t^R - V_t^{IN} = h + \beta E_t \left[\gamma(V_{t+1}^S - V_{t+1}^{IN}) + (1-v-\gamma)(V_{t+1}^R - V_{t+1}^{IN})\right]. \tag{A.16}$$

Move (A.16) ahead one period, take E_{t-1} of both sides, and set equal to (A.15) to get

$$\begin{aligned} &\frac{1}{\beta v}E_{t-1}\left[\ln(x_t/\bar{x}) + h\right] + \frac{1}{\beta v}E_{t-1}\left(\frac{1-p_t}{x_t p_{x,t}}\right) - \frac{1}{\beta^2 v}\left(\frac{1}{x_{t-1}p_{x,t-1}}\right) \\ = \;& h + \beta E_{t-1}\left\{\gamma(\beta x_t p_{x,t})^{-1} + (1-v-\gamma)\left(\frac{1}{\beta v}\left[\ln(x_t/\bar{x}) + h\right] + \frac{1}{\beta v}\left(\frac{1-p_t}{x_t p_{x,t}}\right) - \frac{1}{\beta^2 v}\left(\frac{1}{x_{t-1}p_{x,t-1}}\right)\right)\right\} \end{aligned} \tag{A.17}$$

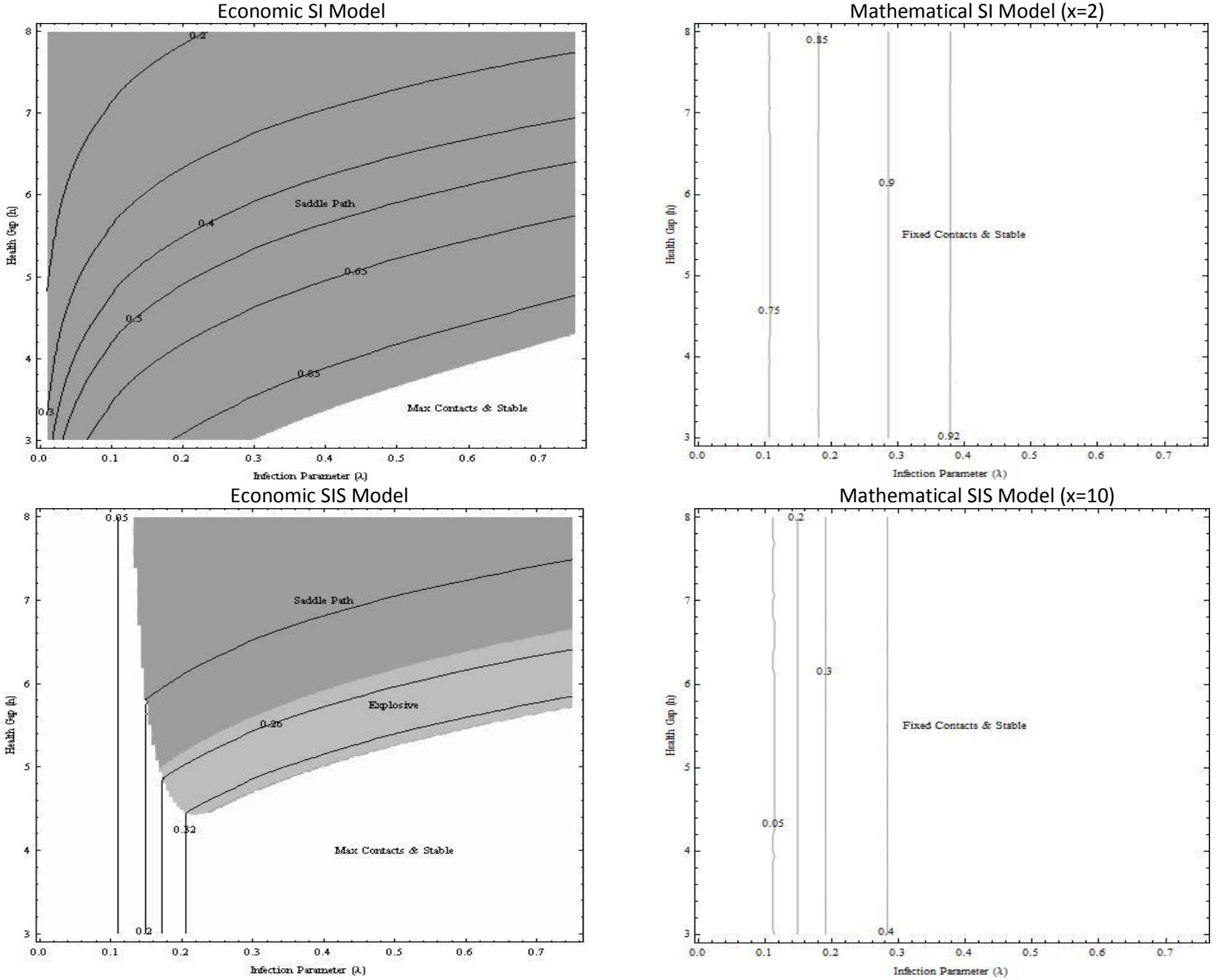
Impose perfect foresight, move ahead one period, and rearrange to get

$$x_t^{-1} = \beta p_{x,t}\left[\ln(x_{t+1}/\bar{x}) + h + \frac{(1-v-p_{t+1})}{x_{t+1}p_{x,t+1}} - \beta\Delta_{t+2}\right],$$

where

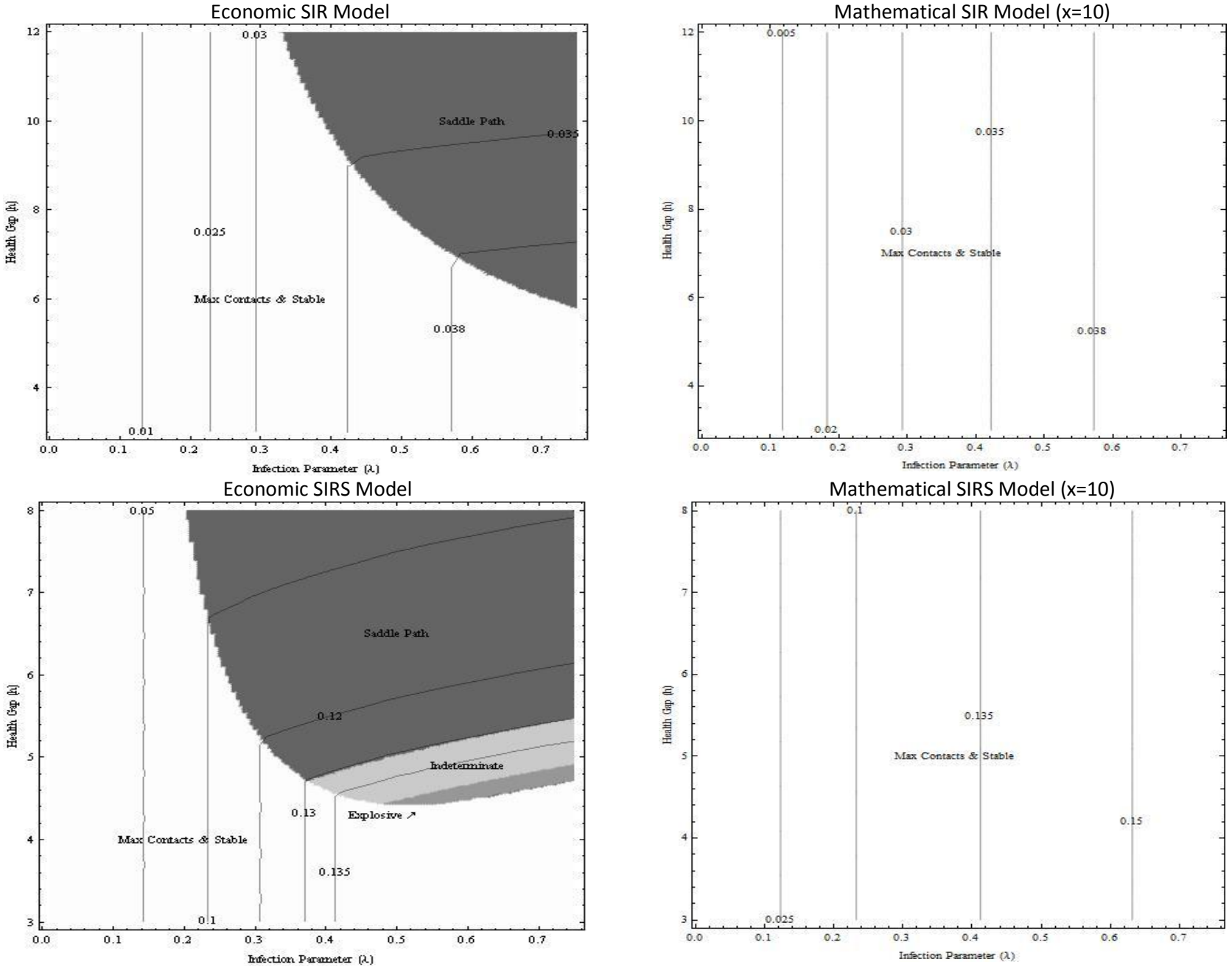
$$\Delta_{t+2} = \frac{v\gamma}{x_{t+2}p_{x,t+2}} + (1-v-\gamma)\left[\ln\left(\frac{x_{t+2}}{\bar{x}}\right) + \frac{1-p_{t+2}}{x_{t+2}p_{x,t+2}}\right] + (1-\gamma)\left[h - \frac{1}{\beta x_{t+1}p_{x,t+1}}\right].$$

Figure 1. Types of Dynamic Paths for Rational Expectations Economic SI and SIS Models



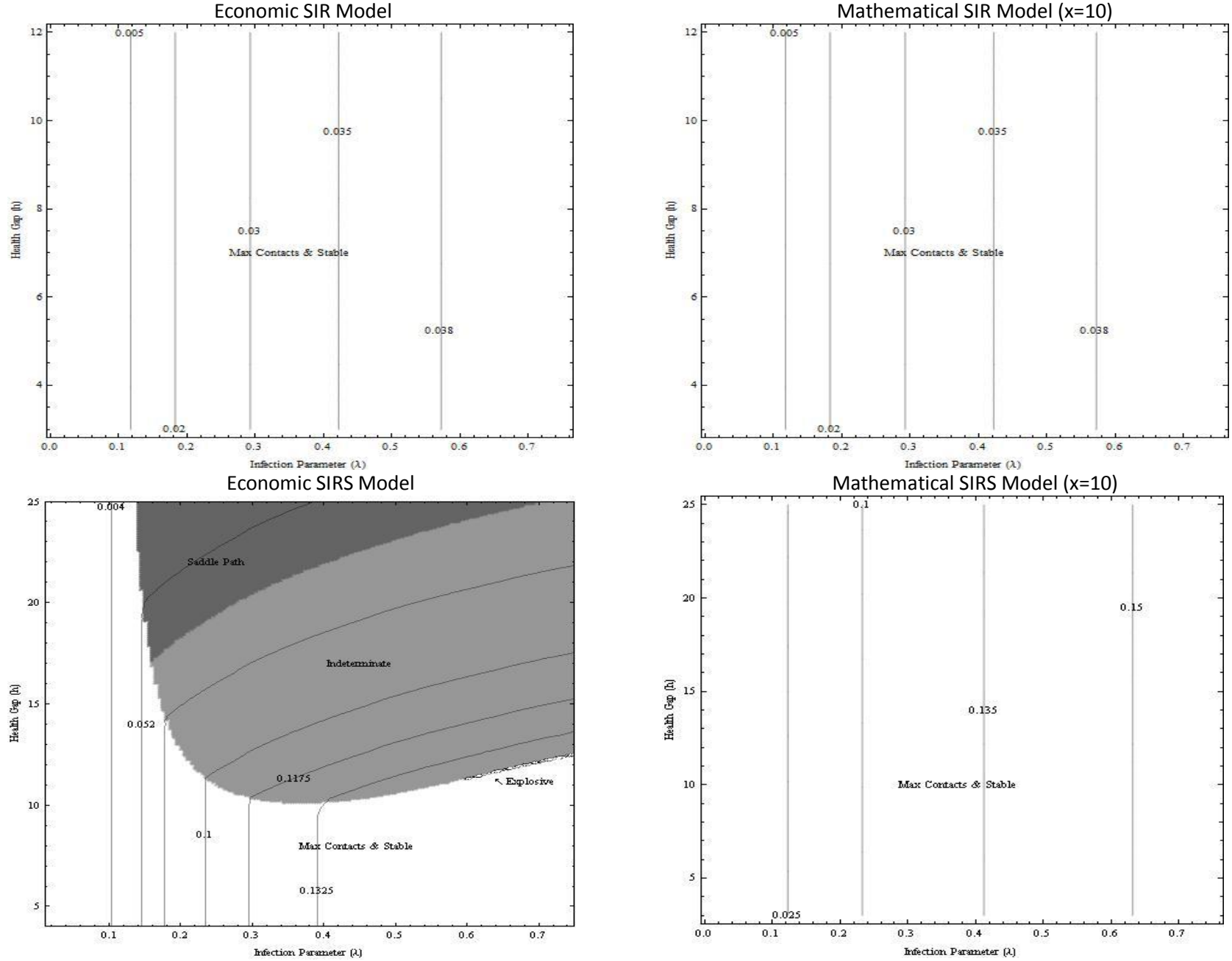
Notes: Contour lines indicate disease prevalence. Parameter values are given in Table 1.

Figure 2. Types of Dynamic Paths for Rational Expectations Economic SIR and SIRS Models (with Unobserved Immunity)



Notes: Contour lines indicate disease prevalence. Parameter values are given in Table 1.

Figure 3. Types of Dynamic Paths for Rational Expectations Economic SIR and SIRS Models (with Observed Immunity)



Notes: Contour lines indicate disease prevalence. Parameter values are given in Table 1.