Syphilis Cycles

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Abstract

Syphilis has re-emerged as a global public health issue. In lesser developed countries, millions of people are contracting the disease, which can be fatal without access to proper treatment. In developed countries, prevalence is much lower but has cycled around endemic levels for decades. The authors of a recent high-profile article in the journal *Nature* argue that these regular fluctuations in syphilis prevalence are driven primarily by endogenous disease dynamics rather than social or behavioral factors, as often theorized. We explore this hypothesis by extending the classic SIRS epidemiological model to incorporate forward-looking, rational individuals. This economic SIRS model (or E-SIRS) model is consistent with microeconomic fundamentals as it is derived from the behavioral equations of rational individuals. In contrast to the *Nature* article, the E-SIRS model predicts that human preferences over health and sexual activity are central to the nature of syphilis cycles. We find that low-activity individuals will behave in a manner that significantly dampen the cycles, while high-activity individuals will tend to exacerbate the cycles, a phenomenon we refer to as *rational dynamic resonance*. The economic SIRS model also provides additional insights into two failed attempts by the U.S. government to eradicate syphilis from the U.S. population.

JEL Codes: D1, I1.

Keywords: syphilis, disease, eradication, cycles, fatalism, dynamic resonance, SIRS

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1 Introduction

Syphilis is back on center stage as a global health issue. In the 1930's and 1940's, syphilis was perhaps the most prominent public health issue in the U.S., with more federal dollars spent on syphilis than any other infectious disease (St.Louis (1996); Brown (1971)).[1] In 1937, Surgeon General Thomas Parran estimated that 10 percent of all adults in the U.S. would be infected with syphilis during their lifetimes (Parran (1937)). However, with the introduction of antibiotics and the beginning of the HIV/AIDS epidemic, syphilis largely disappeared from the public's eye. Figure 1 shows the dramatic fall in U.S. infection rates for primary and secondary syphilis during the 1940's and the subsequent oscillations around a much lower rate of incidence. Despite the successful reduction in syphilis in the U.S. and other developed countries over the last half century, several recent events have worked together to bring syphilis back to the forefront of the global health scene.

First, syphilis remains a persistent health threat in lesser developed countries. The World Health Organization (WHO) estimates that approximately 12 million new syphilis infections occur each year, many of which go untreated (WHO (2004)). Congenital syphilis, in particular, is estimated to inflict over 1.5 million pregnant women in Sub-Saharan Africa with approximately 60% of the acute cases leading to fetal death. This amounts to nearly 500,000 infant deaths from syphilis in sub-Saharan Africa alone, rivaling those due to HIV (Schmid (2004)). Rapidly developing countries have also seen increases in the incidence of syphilis. Syphilis rates in China, for instance, have skyrocketed 25 fold since the early 1990's (Chen *et al.* (2007)).

Second, the WHO and the U.S. Center for Disease Control and Prevention (CDC) have been actively publicizing plans to eliminate syphilis. The WHO recently introduced its global initiative to eliminate congenital syphilis (WHO (2007)). Their plan advocates improved antenatal care, universal testing for pregnant women and partners, rapid treatment, promotion of condom use, and enhanced synergies with HIV prevention programs. The CDC's National Plan to Eradicate Syphilis, first introduced in 1999, is an attempt to capitalize on historically low levels of prevalence and finally rid the U.S. of the disease (CDC (1999)). The plan emphasizes improved reporting and data gathering, rapid diagnosis and treatment of outbreaks, and a concerted effort to increase awareness of the health consequences of sexual activity. But the U.S. plan has not worked. The

[1] Syphilis is remembered by many for the infamous Tuskagee experiments where poor, Southern black men were misleadingly infected with the disease and studied by the U.S. Public Health Service over a period of 40 years (Nakashima *et al.* (1996)) starting in 1932. In 1997, the U.S. government formally apologized for the incident.

incidence of syphilis in the U.S. has increased by nearly 25 percent since 2000, with similar increases occurring in parts of Europe and Asia (Nicoll and Hamers (2002); Fenton and Lowndes (2004); Renton *et al.* (2006); Reynolds *et al.* (2006)). These increases are alarming as they may suggest greater sexual promiscuity and less employment of safe-sex practices (Nakashima *et al.* (1996)). If true, this could warn of a future rise in other sexually transmitted diseases including HIV.

Third and finally, the authors of a recent highly publicized epidemiological study in the journal *Nature* argue that cycles in the prevalence of syphilis are not the result of social or behavioral factors, but instead are the result of the disease's biology (Grassly *et al.* (2005)).[2] If this is interpreted as implying that human behavioral responses play little-to-no role in the spread of the disease, such findings could steer policy in a dangerous direction, possibly contributing to the future spread of syphilis and other sexually transmitted diseases. Economists have repeatedly demonstrated the policy importance of individuals' ability to respond to changes in risk (e.g., Ehrlich and Becker (1972), Peltzman (1975), Rosen (1981), Viscusi (1990), and Shogren and Crocker (1991)).

Our paper builds on the principle that individuals will respond to risk by specifying an integrated economic-epidemiological model where humans and disease biology work together to determine syphilis dynamics. Following the pioneering research of Geoffard and Philipson (1996), Kremer (1996), Auld (2003), and Gersovitz and Hammer (2004), we consider an explicit role for optimizing human behavior within a model of infectious disease dynamics.[3] In most epidemiological models, such as the Susceptible-Infected-Recovered-Susceptible (SIRS) model, human responses to disease risk are captured by exogenous, time-invariant parameters governing rates of infection, recovery and susceptibility. Instead, we propose a modified SIRS model that allows for complex human responses to the risk of disease. This economic SIRS, or E-SIRS model, is derived directly from the behavior of rational individuals. The resulting SIRS dynamic system closely resembles the classic SIRS equations (Murray (2002)) with one major difference. In the E-SIRS model, the

[2] The article received significant media attention from National Public Radio, ABC News, and MSNBC, to mention a few. However, Grassly et al.'s findings have not been without controversy. In a recent letter to editors of major media outlets, John Douglas, the Director of the STD Prevention Office of the CDC has questioned Grassly et al.'s results over concerns that it oversimplifies the causes of syphilis and understates the role of human behavior in explaining syphilis cycles (Douglas (2005)). The implicit message taken away from the media's coverage of Grassly et al.'s findings, according to Dr. Douglas and colleagues at the CDC, is that because syphilis cycles are claimed to be driven primarily by the biological characteristics of the disease dynamics rather than human behavior, policy makers may be led to believe that prevention is of secondary importance and will have less incentive to promote preventive methods.

[3] Given the global devastation of HIV and AIDS, most recent economic studies of infectious disease have focused on HIV. One exception is Momota *et al.* (2005), who present a simple macroeconomic overlapping generations model of infectious disease. Their model is applied to syphilis, however, it does not explicitly model the epidemiology of the disease and employs unrealistically long time periods.

traditional infection parameter is not fixed but varies over time and depends on the optimally chosen number of sexual partners, the number of sex acts with each partner, the overall infection rate in the population, and the natural rate of infection. Consequently, predictions of individuals' collective responses to changes in the risk of disease transmission (e.g., through education campaigns emphasizing prevention and treatment) will be more robust than predictions from traditional models with fixed parameters and no behavioral responses. For instance, policies designed to reduce the transmission of the disease may fail if individuals choose to offset reductions in the risk of infection by engaging in increased amounts of sexual activity.

We highlight two main findings from our research: (1) human preferences over sexual activity and health are critical to the existence and nature of syphilis cycles and (2) policies designed to eradicate the disease are likely to fail. The first finding is at odds with Grassly *et al.* (2005). Depending on individual partner elasticities with respect to prevalence, syphilis cycles can be significantly dampened or accentuated by the collective actions of rational individuals**.** For individuals who take a modest number of sexual partners, the incentives are to choose fewer partners when infection rates rise and more partners when infection rates fall as noted by Geoffard and Philipson (1996). As a consequence, peaks in aggregate infections are lower, troughs are shallower, and cycles die out more rapidly. The response of low-activity individuals serves to dampen the cyclical fluctuations of the disease. For individuals who take a high number of partners, the probability of infection is sufficiently high that additional partners have negligible impact on the probability of infection. Under these circumstances an increase in prevalence causes a decrease in the marginal probability of infection, leading a rational individual to choose more partners. This type of rational fatalism was demonstrated by Kremer (1996). Here, the potential of fatalism in a dynamic context is derived in terms of human preferences over sexual activity and health, and shown to contribute to syphilis cycles by causing them to be exacerbated in their amplitude and persistence, a phenomenon we refer to as *rational dynamic resonance*.

In our second finding, we offer new insights into two failed campaigns by the U.S. government to eradicate syphilis: the 1964 campaign headed by William Brown, past director of the VD division of the CDC (Brown (1971)), and the more recent 1999 National Plan to Eradicate Syphilis.[4] Assuming reasonable rates of transmission and rational, self-interested individuals, our model predicts that programs aimed at eradication are likely to fall short of their desired objectives. The intuition is

[4]The plans include strategies to 1) enhance surveillance; 2) strengthen community involvement and partnerships; 3) respond rapidly to outbreaks; 4) expand clinical and laboratory services; and 5) enhance health promotion.

straightforward: when prevalence of the disease is low and nearing eradication, this is precisely the time when sexually-active, rational individuals will choose to increase their number of partners and perpetuate the disease. Of course, eradication policies can still have a positive impact. In fact, our model predicts that policies aimed at reducing the risk of infection for high-activity individuals, either through reductions in the number of partners or through increased protection, can lower long-run endemic equilibria and stabilize cycles.

2 Syphilis – Epidemiology and Statistics

2.1 Epidemiology

Syphilis is a sexually transmitted disease (STD) caused by the spiral microorganism *Treponema pallidum*. The disease is unique in its slow tempo of progression through infected individuals, but if left untreated may eventually cripple or kill one in four of those infected. The point of infection eventually becomes characterized by an ulceratic chancre signalling the beginning of what is known as the primary stage of the disease. Without treatment the disease progresses to a secondary stage observed by a skin rash and mucous membrane lesions. Following secondary symptoms the disease moves to the latent stage, and although inapparent, the infection remains within the body and can reappear or eventually damage internal organs with crippling effects and possible mortality (CDC (2006)). Individuals are infectious whenever surface lesions are present, in both primary and secondary stages of the disease. In the early latent stage individuals may return to the infectious stages, whereas in the late latent stage there are three potential outcomes for the infection. In the first, the infection is biologically eradicated within the body over a number of years. The second outcome finds the infection remaining within the individual over the course of their lifetime, but the internal damage is slight enough to be imperceptible. The final outcome is where the infection progresses slowly to cause organ damage and can be fatal (Cecil (1948)). The progression from susceptible to various stages of infection to immune and back to susceptible is outlined in Figure 2.

Syphilis has two further effects that deserve mention. First, infected women can transmit the disease to a fetus throughout the various stages of the disease. Known as congenital syphilis, this can cause numerous complications through pregnancy including miscarriages, stillborns, and fatally ill infants. Second, the lesions caused by syphilis can act as a conduit for other STDs, most notably the HIV infection. The presence of syphilis has been shown to increase the chance

of acquiring the HIV infection by 2 to 5 times (Chesson and Pinkerton (2000); CDC (2006)).[5]

While there is no vaccine for syphilis, treatment in its early stages (through an intramuscular injection of penicillin) will cure the individual, and repeated treatments will eliminate the infection in late stages. Following treatment and recovery from the infection, individuals may develop transitory immunity to reinfection before again becoming susceptible. This progression from susceptible to infected to recovered (and immune) to susceptible fits the general form of the classic SIRS model.

2.2 Statistics

The defining feature of syphilis is the regular cycle in disease prevalence (see Figure 1). As argued by Grassly *et al.* (2005), these cycles occur as synchronized waves of recovered individuals lose their temporary host immunity and re-enter the susceptible population. The ebb and flow of susceptible (S), infected (I) and immune/recovered (R) populations can also cause cycles to persist well past any initial driving impulse. Gonorrhea, for example, shares the same method of contraction but lacks transitory host immunity and in contrast to syphilis does not exhibit cycles. Grassly *et al.* (2005) therefore draw the conclusion that observed syphilis cycles during the three-decade period following 1960 must be due to disease biology rather than popular explanations involving the sexual liberation movement in the 1960s and the crack cocaine epidemic of the mid-to-late 1980s.

To the casual observer syphilis appears to be a fairly benign social problem in developed countries. Syphilis can be rapidly and effectively treated with penicillin. Furthermore, the reported cases of syphilis have fallen dramatically in the developed world during the past century (Green *et al.* (2001)). For example, there were only 7,980 cases of primary and secondary syphilis reported in the U.S. in 2004, representing 2.7 cases per 100,000 population (CDC (2006)). By contrast, there were nearly five times as many newly reported cases of AIDS in the U.S. in 2004 with potentially more severe health consequences. Yet, these numbers mask serious policy issues.

First, syphilis strikes the population in a very disproportionate manner, with substantially higher prevalence in urban areas, blacks and gay men. The CDC estimates that over 50% of all infections occurred in just 16 counties and 1 city, blacks are five times more likely to contract syphilis than whites, and nearly 65% of all primary and secondary syphilis cases arise with gay men

[5] Chesson *et al.* (2003) argue that the causality may also run in the other direction. They show that high rates of AIDS mortality in high-risk men were responsible, at least in part, for the decline in the prevalence of syphilis in the U.S. during the 1990's.

(CDC (2006)).

Second, statistics in the underdeveloped world are grim. A recent study by the WHO estimates there are approximately 12 million new syphilis infections per year, many of which go untreated (WHO (2004)). Congenital syphilis is estimated to inflict over 1.5 million pregnant women in Sub-Saharan Africa with an estimated 60% of the acute cases leading to fetal death. This amounts to nearly 500,000 infant deaths from syphilis in sub-Saharan Africa alone, rivaling those due to HIV (Schmid (2004)).

Finally, as noted above syphilis acts as a conduit for other STDs and has been shown to increase the chance of acquiring the HIV infection. Syphilis remains a threat to public health in the U.S. and societies across the globe. In order to provide policy makers with better insight into its control, we undertake a careful mathematical characterization of the disease's dynamics and the associated behavioral implications.

3 Theoretical Model

The E-SIRS model is set in discrete time with t indexing annual decision intervals.[6] There is a constant (net of births and deaths) population of N individuals, all of which are identical except for their state of the disease. The utility function is defined over sexual partners $x_{i,t}$ and the health stock $h_{i,t}$. The objective for the i^{th} individual is to choose a sequence $\{x_{i,t}\}_{t=0}^{\infty}$ to maximize

$$E\sum\nolimits_{s=0}^{\infty}\beta^{t+s}u(x_{i,t+s},h_{i,t+s}) \tag{1}$$

where $0 \leq \beta \leq 1$ is the discount factor and E represents an individual's expectation of future outcomes. The control variable $x_{i,t}$ is interpreted as the number of sexual partners chosen by agent i in period t. For analytical convenience the instantaneous utility function is specified as:

$$u(x_{i,t},h_{i,t}) = (x_{i,t}^{\alpha}/\alpha) + bh_{i,t} \tag{2}$$

where the maximum number of feasible partners in a single period is $\bar{x}$, α is the curvature parameter, b is a parameter reflecting the relative importance of the health stock, and $\bar{h}$ is the upper bound on the health stock. The specification in (2) has the advantage that the linearized dynamic system

[6] The SIRS model is traditionally modeled in continuous time, but the discrete time version is more convenient for specifying lead and lag relationships, selecting the timing of driving shocks, and for contrasting predictions of the model with the annually observed U.S. syphilis data.

can be reduced to a two-equation system with concise dynamic roots that govern the cyclical fluctuations. Also, with $b = 0$ and $\bar{x} = 1$, the E-SIRS model collapses to the SIRS model, making it straightforward to contrast predictions from the E-SIRS model with those from previous research. Note that although utility is linear in the health stock, h is ultimately related to the chosen number of partners (x) in a concave fashion through the probability of infection.

The core tradeoff in the model is that additional sexual partners bring immediate satisfaction but also the risk of future infection. Infection in turn causes a deterioration of the health stock. Following Grossman (1972), the law of motion for the health stock is

$$h_{i,t+1} = h' + (1 - \delta)h_{i,t} - in_{i,t+1} \tag{3}$$

where $0 \leq \delta \leq 1$ is the depreciation rate for health and $\bar{h} = h'/\delta$ is the steady-state level of the health stock for an uninfected individual.

In any period t, individual i can be in one of three states: susceptible ($s_{i,t}$), infected ($in_{i,t}$) or recovered ($r_{i,t}$). If the agent is susceptible then $s_{i,t} = 1$ and $in_{i,t} = r_{i,t} = 0$; if infected, then $in_{i,t} = 1$ and $s_{i,t} = r_{i,t} = 0$; and if recovered, then $r_{i,t} = 1$ and $in_{i,t} = s_{i,t} = 0$. Because an individual can only be in one state at any time, $s_{i,t} + in_{i,t} + r_{i,t} = 1$ for all t. The fractions of susceptible, infected and recovered individuals in the entire population are then given by

$$s_t = \frac{1}{N}\sum\nolimits_{i=1}^{N} s_{i,t};\ in_t = \frac{1}{N}\sum\nolimits_{i=1}^{N} in_{i,t};\ \text{and } r_t = \frac{1}{N}\sum\nolimits_{i=1}^{N} r_{i,t}, \tag{4}$$

where $s_t + in_t + r_t = 1$.

Individual behavior and aggregate disease dynamics depend on the transition probabilities between disease states. These probabilities are shown in Table 1.

Table 1. Individual Transition Probabilities

State Transition	Probability
Susceptible ($s_{i,t}$) $\longrightarrow$ Infected ($in_{i,t+1}$)	$p_{i,t} = 1 - (1 - \lambda_p in_t)^{x_{i,t}}$
Infected ($in_{i,t}$) $\longrightarrow$ Recovered ($r_{i,t+1}$)	ν
Recovered ($r_{i,t}$) $\longrightarrow$ Susceptible ($s_{i,t+1}$)	γ

As in the classic SIRS model, the probability that an infected individual recovers and the probability that a recovered individual loses immunity are time-invariant, exogenous and given by ν and

γ, respectively. The probability that a susceptible individual becomes infected $p_{i,t}$, however, is endogenous and depends on the natural probability of contracting syphilis from a single infected partner λ_p, the number of chosen sexual partners $x_{i,t}$, and the fraction of individuals in the population that are infected in_t. Assuming that individuals choose $x_{i,t}$ independent partners per period and engage in a fixed number of sexual acts (a) with each partner, the probability that an individual remains susceptible is

$$\Pr(s_{i,t} \longrightarrow s_{i,t+1}) = [in_t(1-\lambda_a)^a + (1-in_t)]^{x_{i,t}},$$

where λ_a is the natural probability of infection from a single sexual act with an infected partner. With a sufficiently large population, in_t measures the probability that a susceptible individual will randomly choose an infected partner and risk contracting the disease. Because in_t is a population measure, each susceptible individual takes it as given when deciding how many sexual partners to choose and does not take into account the impact of their choice on others' probability of infection. The probability of becoming infected is then

$$p_{i,t} = \Pr(s_{i,t} \longrightarrow in_{i,t+1}) = 1 - \Pr(s_{i,t} \longrightarrow s_{i,t+1}) = 1 - [1-(1-(1-\lambda_a)^a)in_t]^{x_{i,t}}.$$

Letting $\lambda_p = 1-(1-\lambda_a)^a$, the probability then simplifies to the expression in Table 1.

Aggregation of the transition probabilities across all individuals produces the laws of motion for each disease category. The result is the classic SIRS model (Murray (2002)), modified to incorporate births, deaths, discrete time and endogenous choice of sexual partners:

$$\begin{aligned} s_{t+1} - s_t &= \mu - (\lambda in_t + \mu)s_t + \gamma r_t && (5) \\ in_{t+1} - in_t &= \lambda in_t s_t - (\nu + \mu)in_t && (6) \\ r_{t+1} - r_t &= \nu in_t - (\gamma + \mu)r_t, && (7) \end{aligned}$$

where μ measures both the birth and death rates and $\lambda = p_t/in_t$. (See Appendix A for the complete derivation of equations (5) through (7)). The system is straightforward to interpret. Equation (5) states that changes in the susceptible proportion of the population come from births less deaths ($\mu - \mu s_t$), recovered individuals that lose immunity and re-enter the susceptible state (γr_t), and susceptible individuals that become infected ($-\lambda in_t s_t$). The latter term is derived by averaging the individual probability of infection ($p_t = \lambda in_t$) across all susceptible agents. The evolution

of the infected proportion of the population is given by equation (6). The proportion of infected individuals increases due to newly infected individuals ($\lambda in_t s_t$) but decreases due to deaths ($-\mu in_t$) and recovery from the disease ($-\nu in_t$). These newly recovered individuals generate an increase in the recovered proportion as shown in equation (7), while deaths of the recovered individuals ($-\mu r_t$) and those that lose immunity and transition back into the susceptible state ($-\gamma r_t$) reduce the fraction recovered. The overall population is constant over time as deaths across the three states ($-\mu s_t - \mu in_t - \mu r_t = -\mu$) exactly offset the birth of new susceptible individuals (μ).

To the best of our knowledge this is the first attempt to provide a micro-level derivation for the aggregate SIRS epidemiological model that is consistent with optimizing individual behavior. The key insight from this exercise is that the infection parameter from the SIRS model, $\lambda = \lambda(\lambda_p, x_t, in_t)$, is now time varying and depends on the endogenous risky behavior of agents, as well as the overall prevalence of the disease. The derived model is consistent with the classical SIRS model because if $x_t = 1$ for all periods, so that each susceptible agent chooses only one partner per period, the infection parameter collapses to $\lambda = \lambda_p$. Our micro-level derivation of the SIRS model also highlights why previous researchers have needed to calibrate their models with values for λ in excess of one in order to match the dynamics of actual syphilis infections (e.g., Garnett *et al.* (1997); Grassly *et al.* (2005)).

3.1 Optimal Decision Making

In our baseline model, all individuals – regardless of their infection status – are assumed to be self-interested and maximize (1) without concern for the welfare of the general population. Under these conditions, the optimal choice for recovered and infected individuals is straightforward because they face no risk of immediate infection (or re-infection), and as a result, they will choose the maximum number of partners $x_t = \bar{x}$ (Geoffard and Philipson (1996)). The choice to engage in the maximum amount of risky behavior while infected imposes a negative externality on the rest of the population because it continues to propagate the disease through the population and causes susceptible individuals to choose a suboptimal number of sexual partners.[7] Conversely, an entirely altruistic population of infected individuals (or a benevolent social planner guiding the actions of

[7] The consequences and policies associated with the externalities imposed by infected individuals have been studied in depth for the SIS epidemiological model by Goldman and Lightwood (2002), Gersovitz and Hammer (2004) and Gersovitz and Hammer (2005). Their work focuses on the design of optimal tax policies to encourage effective treatment and prevention of the disease. We instead focus on explaining cyclical fluctuations in a SIRS framework and the ability of policy makers to eradicate the disease from the general population.

infected individuals) would sharply decrease the number of sexual encounters so the disease could quickly be eradicated.

To allow for possible altruism by infected individuals (Philipson and Posner (1993); Gersovitz (2004)), we introduce the parameter $\theta \in [0,1]$ which scales the number of sexual partners chosen by the representative infected individual. For example, if $\theta = 1$, infected individuals are entirely self-interested and will choose $x_t = \theta\bar{x} = \bar{x}$ partners. If $\theta = 0$, the individuals are perfectly altruistic and will abstain from sexual activity $x_t = \theta\bar{x} = 0$. This simple mechanism for capturing altruistic behavior allows for varying degrees of altruism in the population. In the baseline model, infected individuals are assumed to be entirely self-interested ($\theta = 1$). The implications of relaxing this assumption are discussed below.

The optimization problem for the representative susceptible individual is to maximize (1) subject to (3) and the individual transition probabilities in Table 1. The resulting Euler equation is

$$u_x(x_t, h_t) = -\beta u_h(x_{t+1}, h_{t+1}) \frac{\partial h_{t+1}}{\partial in_{t+1}} \frac{\partial \Pr(s_t \to in_{t+1})}{\partial x_t}, \tag{8}$$

where subscripts on the utility function refer to partial derivatives. Equation (8) requires susceptible individuals to add partners until the marginal utility from an additional partner just equals the discounted future expected disutility of contracting the disease. (The second-order sufficient conditions are presented in Appendix B.) Applying the explicit functional forms in (2) and (3), the Euler equation collapses to

$$x_t^{\alpha-1} = \beta b p_{x,t}, \tag{9}$$

where $p_{x,t} = \partial \Pr(s_t \to in_{t+1})/\partial x_t = -\ln(1-\lambda_p in_t)(1-\lambda_p in_t)^{x_t}$. Note that because x_t enters $p_{x,t}$, this is not an explicit solution so that steady-state and dynamic values for x_t are only recoverable through numerical methods.

3.1.1 Behavioral Underpinnings of the Euler Equation

Begin from the perspective of a susceptible individual, who treats the infection rate as fixed. The basic problem facing each susceptible individual is how many partners to choose under the risk of future infection. Equation (8) yields a standard solution for dynamic expected-utility maximization problems: continue to add partners (x) until the marginal benefits from an additional partner just offset the discounted expected disutility of contracting the disease in the future (hereafter, marginal cost). However, unlike standard expected utility maximization problems (von Neumann and

Morgenstern (1944)), here the risk of future infection is endogenous (Ehrlich and Becker (1972)). The more partners are chosen, the greater the probability of infection. Yet, the probability of infection is also bounded above by one. This implies that although additional partners will increase the risk of infection, they must do so at a decreasing rate and marginal costs will be declining over all x.

Consider the following numerical example. Let the natural transmission rate be $\lambda_p = 0.8$ and the overall infection rate fixed at $in = 0.25$. Following Table 1, the probability of infection with one partner per year is $p = 0.20$ and with two partners it is nearly double at $p = 0.36$, an increase in probability of 0.16. With seven partners, however, the probability is $p = 0.79$ but only increases to $p = 0.83$ with the eighth partner, an increase of only 0.04. Thus, the incremental costs of sexual activity clearly fall as more partners are added.

In Figure 3, we illustrate the marginal benefit and marginal cost curves for the E-SIRS system when prevalence is endogenous and varies with x. Because individuals exhibit diminishing marginal utility in x, marginal benefits (black line) are declining over all x. Marginal costs initially increase with x and then decline. At low levels of x, the marginal cost curve slopes up because an increase in partners causes prevalence to rise sharply. But as the number of partners increases, additional partners no longer have much of an effect on prevalence and marginal costs decline with x.

The optimal choice is given by the intersection of the marginal benefit and cost curves. If individuals' relative concern for their health is too low, marginal benefits will exceed marginal costs for all choices of x. As a result, there will be no interior solution and individuals will rationally choose the maximum number of sexual partners, $\bar{x}$, in each period. But, if individuals have a greater concern for their health (as shown in Figure 3), the marginal benefit and cost curves intersect twice. While both intersections satisfy the necessary condition for an optimal choice, only the left-hand equilibrium satisfies the second-order sufficient condition. (The sufficiency condition in Appendix B shows that either the marginal cost curve must slope up or if it slopes down, it must be locally flatter than the marginal benefit curve for the choice to be optimal.) Because the sufficiency condition is not satisfied at the right-hand intersection, lifetime utility is not maximized and it is not a viable equilibrium choice.

4 Equilibria and Dynamics

An equilibrium for the E-SIRS system is characterized by a sequence of values $\{x_t, h_t, s_t, in_t, r_t\}_{t=0}^{\infty}$ that satisfy (3), (5), (6), (7), and (9) for all t, subject to the initial values s_0, in_0, r_0 and h_0. Given the complexity of the system, an analytical solution for the optimal path is not possible. Instead, we solve the steady-state conditions numerically and use standard linearization methods to evaluate the stability and transition dynamics around each steady state. We first examine the long-run equilibrium and then later turn our attention to the short-run equilibrium.

4.1 Long-Run Equilibrium

There are two possible steady-state equilibria – an eradication and an endemic equilibrium. The eradication equilibrium, where the infectious disease is eliminated from the population, is given by

$$\{x = \bar{x}, h = \bar{h}, s = 1, in = 0, r = 0\}. \tag{10}$$

The eradication equilibrium is feasible for all parameter values (although not necessarily stable). The properties of the eradication equilibrium are important to policy makers who wish to transition from an endemic equilibrium to eradication. These properties are considered in detail following a characterization of the endemic equilibrium and the transition dynamics.

The endemic equilibrium is found by solving time-invariant versions of (3), (5), (6), (7), and (9) in terms of the fundamental parameters. The endemic steady-state system is

$$\begin{aligned}
x^{\alpha-1} &= \beta b p_x && \text{(11a)}\\
s &= R_0^{-1} && \text{(11b)}\\
in &= (\gamma+\mu)(1-s)(\gamma+\nu+\mu)^{-1} && \text{(11c)}\\
r &= \nu(1-s)(\gamma+\nu+\mu)^{-1} && \text{(11d)}\\
h &= \delta^{-1}(h' - in) && \text{(11e)}
\end{aligned}$$

where $R_0 = \lambda/(\nu+\mu)$ is the basic reproductive number. The basic reproductive number measures how many susceptible individuals contract the disease from a single infected person (Garnett *et al.* (1997)). In the classic SIRS model (i.e., $b = 0; \bar{x} = 1$), R_0 is an exogenous constant and the key parameter for determining stability of the eradication and endemic steady states. R_0 is also key

to the stability of the E-SIRS steady states, but is endogenous and depends on individual choices. As a result, the dynamics around the E-SIRS steady states are linked to individuals' underlying preferences for sexual activity and health.

Figure 4 shows the steady-state endemic infection rates as a function of b (relative concern for health) and ν (treatment rate). These are two parameters policy makers are likely to be able to influence. The health parameter b can be influenced through public health education informing individuals of the dangers and consequences of risky sexual behavior. Influence over treatment rates ν is linked to serological screening practices, contact tracing and the speed of treatment provided by public and private health services (Green *et al.* (2001)).

Figure 4 highlights several consequences of b and ν on steady-state endemic infection rates (prevalence). First, steady-state prevalence is independent of b when concern for health is less than b^*, the threshold value for susceptible individuals to choose the maximum number of partners, $\bar{x}$.[8] When $b \leq b^*$, prevalence is exclusively a function of ν and biological parameters such as γ and λ_a. This indicates that the initial returns to public health education programs may be low, particularly in lesser developed countries where public health budgets are relatively small and the health consequences of infection may not be fully understood. Second, when concern for health is greater than b^*, the infection contours are decreasing in b and ν. In other words, an increased concern for one's health or higher treatment rates correspond to a reduced long-run prevalence of the disease. Third, the infection contours grow increasingly far apart as the treatment rate rises. As the treatment rate increases, the population of infected individuals falls so that increases in the treatment rate impact a smaller and smaller fraction of the population; there are diminishing returns to treatment in the aggregate.

4.2 Short-Run Equilibrium and Stability

An analytical solution for the transition path of the E-SIRS system is not available. Therefore, we investigate the stability of the system by taking a first-order Taylor series approximation of the

[8] The threshold value b^* can be identified by the "kink" in the infection contours in Figure 4 and is characterized by the value that assures marginal costs are just tangent to marginal benefits (see Figure 3).

system around each steady state:

$$\begin{aligned}
\hat{x}_t &= [\kappa x/in]\widehat{in}_t && (12)\\
\hat{h}_{t+1} &= (1-\delta)\hat{h}_t - \widehat{in}_{t+1} && (13)\\
\widehat{in}_{t+1} &= [1-(\nu+\mu)+sp_{in}]\widehat{in}_t + sp_x\hat{x}_t + p\hat{s}_t && (14)\\
\hat{r}_{t+1} &= (1-\gamma-\mu)\hat{r}_t + \nu\widehat{in}_t, && (15)
\end{aligned}$$

where carets ($\hat{}$) over variables indicate deviations from their steady-state values, κ is the elasticity of partner change (x) with respect to aggregate infections (in), and p_{in} is the partial derivative of p with respect to in. After substituting out the control variable $\hat{x}_t$ and using the restriction $\hat{s}_t + \widehat{in}_t + \hat{r}_t = 0$, the system can be reduced to the following dynamic matrix system:

$$\begin{bmatrix} \widehat{in}_{t+1} \\ \hat{r}_{t+1} \end{bmatrix} = \begin{bmatrix} 1-\nu-\mu-p+\phi & -p \\ \nu & 1-\gamma-\mu \end{bmatrix} \begin{bmatrix} \widehat{in}_t \\ \hat{r}_t \end{bmatrix}, \tag{16}$$

where $\phi = s[p_{in} + (\kappa p_x x/in)]$. The system of equations in (16) determines the transition dynamics around the steady state and can be used to evaluate the local stability of the model.

The local stability of the steady state is determined by the eigenvalues (ψ_1, ψ_2) for the linearized E-SIRS system:

$$\psi_{1,2} = 0.5\left[2(1-\mu) - \gamma - \nu + \phi \pm \sqrt{(\phi+\gamma-\nu)^2 - 4\nu p}\right]. \tag{17}$$

The magnitude of the eigenvalues determines whether the system converges to the steady state. If both eigenvalues are inside the unit circle then the system is locally stable, returning to the steady state for small perturbations. If the eigenvalues also have an imaginary part, then the system will exhibit stable cycles. Using (17), we see that the E-SIRS system displays stable cycles if $|\psi| < 1$ and $4\nu p > (\phi+\gamma-\nu)^2$.[9] From a policy perspective, a primary concern is how parameters such as b and ν affect the stability of the E-SIRS system. To examine this issue, the larger of the

[9] If the eigenvalues have an imaginary part, then they come in complex conjugates $\psi_{1,2} = a \pm ci$ with period equal to (Hamilton (1994))

$$\frac{2\pi}{\cos^{-1}\left[a/\sqrt{a^2+c^2}\right]}$$

and persistence equal to

$$R = Mod[\psi_{1,2}] = \sqrt{a^2+c^2}.$$

two eigenvalues is set equal to one and the sensitivity of this stability frontier to changes in policy parameters is investigated. The stability threshold for the eradication steady state is discussed in Section 5.

Figure 5 presents a numerically derived phase diagram for the E-SIRS system. The black locus represents combinations of x_t and in_t that produce time-invariant values for in_t from the modified SIRS equations. The red locus represents combination of x_t and in_t that produce time-invariant values for x_t from the Euler equation (9). The intersection of these two loci determines the long-run equilibrium of the E-SIRS system, one that exhibits stable cycles for the given parameter values.

Figure 6 depicts the length of the cycles around the endemic steady state. The bottom region reflects combinations of ν and b that lead to stable monotonic approach paths. The top regions reflect stable oscillatory approach paths. Cycle length is independent of b when $x = \bar{x}$. When $b > b^*$, the periodicity depends on both parameters with the length of the cycle being an increasing function of b, but a non-monotonic function of ν. For a fixed treatment rate, Figure 6 is especially helpful in calibrating b to match the empirical cycles. For a treatment rate of $\nu = 0.9$, the health parameter b must be in the 1.5 to 3 range to match the periodicity of U.S. syphilis cycles (Grassly *et al.* (2005)).[10]

4.3 Contrasting the SIRS and E-SIRS Models

The primary distinction between the SIRS and E-SIRS models is the ability to react to changes in the risk of infection. If individuals ignore the health consequences of risky behavior (i.e., $b = 0$), thus choosing the maximum number of partners each period, the E-SIRS model collapses to the SIRS model with infection parameter

$$\lambda = [1 - (1 - \lambda_p in_t)^{\bar{x}}]/in_t. \tag{18}$$

The only difference between the traditional SIRS model and the E-SIRS model with $b = 0$ is that the former has a constant infection parameter while the latter has the parameter varying with in_t. If the additional restriction $\bar{x} = 1$ is imposed, the E-SIRS model collapses to the traditional SIRS

[10] The treatment parameter ν captures both the rate of diagnosis and treatment. The treatment effectiveness for syphilis appears to be close to 100%. For example, Alexander *et al.* (1999) found treatment effectiveness for pregnant women to be 100% for primary and late latent syphilis, 95% for secondary syphilis, and 98% for early latent syphilis. Assuming that $\nu = 0.9$ is therefore tantamount to assuming that 90% of all infected individuals are seeking treatment with the remaining 10% either unaware they are infected or choosing not to seek treatment.

model with a time-invariant infection parameter.[11]

The linearized dynamic SIRS system is

$$\begin{bmatrix} \widehat{in}_{t+1} \\ \hat{r}_{t+1} \end{bmatrix} = \begin{bmatrix} 1-\nu-\mu-p+sp_{in} & -p \\ \nu & 1-\gamma-\mu \end{bmatrix} \begin{bmatrix} \widehat{in}_{t} \\ \hat{r}_{t} \end{bmatrix}. \tag{19}$$

SIRS individuals will not alter the number of partners they choose, even in response to significant variation in disease prevalence. In the E-SIRS model, however, individuals vary the number of partners whenever the risk of infection deviates from normal levels.

The difference in dynamics between the two models can be seen by contrasting the transition matrices in (16) and (19). The two matrices differ by the $(sx\kappa p_x/in)$ term in the (1,1) position. This term captures the effect of changes in current infection rates on infection rates in the following period, through responses in the chosen number of partners. The key parameter is κ, the elasticity of partner change (x) with respect to aggregate infections (in), which is the dynamic counterpart to the behavioral elasticity discussed by Kremer (1996). Following the linearization of (9), this elasticity can be expressed as

$$\kappa = \frac{\partial x}{\partial in}\frac{in}{x} = \frac{\lambda_p in[1+\ln(1-\lambda_p in)^x]}{(1-\lambda_p in)\ln(1-\lambda_p in)[1+\ln(1-\lambda_p in)^x-\alpha]}. \tag{20}$$

In the SIRS model, $\kappa = 0$ because susceptible individuals have no ability to respond to changes in the risk of infection, resulting in transition dynamics given by (19). In the E-SIRS model, κ can take on a range of values depending on the biological parameters and individual preferences over x and h. The value of κ captures the influence of human responses on the dynamics of the system and causes cycles to either be dampened or accentuated.

4.3.1 Rational Dynamic Dampening

Consider an exogenous increase in prevalence. When $\kappa < 0$, the increased risk of infection causes susceptible individuals to choose fewer partners. The reduction in partners in turn lowers the prevalence of the disease and the risk of infection. As a result, the original increase in the infection rate is tempered, a phenomenon we refer to as *rational dynamic dampening.*

[11] Notice that setting $\bar{x} = 1$ implies that $\lambda = \lambda_p$, the probability of contracting the disease from a single infected partner. However, in order to match actual syphilis cycles, the SIRS model typically requires a value for λ in excess of one (Grassly *et al.* (2005)). This is an advantage of the E-SIRS model over the standard SIRS model – an infection parameter greater than one is a natural implication of the model rather than an ad hoc assumption.

To gain further insight into dynamic dampening, let $\alpha = 0$ so that utility is logarithmic in x. In this case, κ reduces to

$$\kappa = \frac{\lambda_p in}{(1 - \lambda_p in)\ln(1 - \lambda_p in)} < 0,$$

which after substituting into (16) produces the transition matrix

$$\begin{bmatrix} 1 - \nu - \mu - p & -p \\ \nu & 1 - \gamma - \mu \end{bmatrix}.$$

Any increase in the risk of infection is therefore exactly offset by reductions in the number of partners (i.e., $p_{in} = -p_x \frac{\partial x}{\partial in}$ or $\phi = 0$). In the more general case where α is less than zero and there is less willingness to substitute partners across time, the number of partners will be reduced but not enough to completely offset the increase in the probability of infection (i.e., $p_{in} > -p_x \frac{\partial x}{\partial in}$).

Panel A of Figure 7 illustrates how an individual with dynamic dampening will respond to an exogenous increase in disease prevalence. The upper graph shows the probability of infection facing an individual while the lower graph shows the marginal benefits and costs of sexual activity. Marginal costs are drawn for a high value of b such that there is a relatively high concern for health. The optimal choice of partners corresponds to point A where the marginal benefit curve MB first crosses the marginal cost curve MC_0. In response to an exogenous increase in prevalence the probability curve shifts up from p_0 to p_1. The marginal cost curve pivots clockwise at the point x_c, the critical or threshold number of partners at which an increase in prevalence leaves the slope of the probability curve unchanged. This pivot is represented as the movement from MC_0 to MC_1. The individual then chooses to take fewer partners, moving to point B where the MB curve intersects the MC_1 curve.

The associated threshold probability, p_c, is the value for which the marginal cost of infection is unchanged and the partner elasticity with respect to prevalence, κ, is equal to zero. Solving this equation produces $p_c = 1 - (1/e) \approx 0.63$, a critical probability that is independent of any economic or epidemiological conditions. For relatively high values of b (panel A of Figure 7), the optimal choices are to the left of x_c and result in higher marginal probabilities of infection, higher expected marginal costs, and a reduction in partners. This corresponds to a movement from point A to point B in panel A of Figure 7. Optimal choices to the right of x_c lead to lower marginal probabilities of infection, lower expected marginal costs and more partners in response to increases in prevalence. As a result, p_c defines the critical probability determining whether or not the individual exhibits

dynamic dampening.

To compare the dynamics of the SIRS and E-SIRS models under dynamic dampening, each model is subjected to a one-time, five percentage point increase in prevalence. The top panels of Figure 8 show the dynamic responses of aggregate infection rates to the unanticipated perturbation. The difference in the persistence is clear when comparing the models. In the SIRS model, the system produces cyclical responses with period equal to 8.8 years and persistence equal to 0.84; the cyclical response continues well past the forcing shock. The E-SIRS cycles have a similar periodicity but are significantly dampened with persistence of only 0.51. Cycles are nearly imperceptible ten years after the driving shock.

The differences in cycles across the SIRS and E-SIRS models are a direct reflection of the number of partners, shown in the bottom panels of Figure 8. In the SIRS model, the number of sexual partners is fixed implying that syphilis cycles are exclusively due to biological dynamics.[12] However, when individuals are free to choose their number of partners and have a $\kappa < 0$, cycles are significantly dampened.[13] With the initial increase in prevalence, the risk to each susceptible individual rises causing them to rationally scale back their number of partners. This in turn places downward pressure on rising infection rates. As the newly infected individuals get treated and transition to recovery, prevalence falls and the risk of infection wanes. Susceptible individuals then rationally increase their number of partners, preventing infections from falling as sharply. The result of this interplay between human responses and biological dynamics causes cycles to be smaller and less persistent than if they were driven solely by biology.

4.3.2 Rational Dynamic Resonance

The opposite occurs when $\kappa > 0$ and the responses of humans and disease biology are in sync. When the probability of infection is above p_c, individuals will become fatalistic and will choose to increase their number of partners in response to an increase in the prevalence of the disease. This behavior is not driven by emotions, but rather by rational decision making. For high-activity individuals, the increased prevalence of the disease causes a decrease in the marginal probability of infection, leading a rational individual to choose more partners. In the context of a dynamic SIRS

[12] For purposes of comparison, we set x in the SIRS model equal to the endogenous solution for x from the E-SIRS model. This also implies that λ and in will be equal across the two models.

[13] For the parameter values in Figure 8, the elasticity of partner change with respect to aggregate infections is $\kappa = -0.73$. Furthermore, if we hold x fixed at its steady-state value, the probability of infection at the steady state is 0.40, increasing to 0.55 with the five percentage point increase in the aggregate infection rate.

model, this behavior will tend to amplify cycles – a phenomenon we refer to as *rational dynamic resonance*.

Panel B of Figure 7 illustrates the problem facing an individual with rational dynamic resonance. The parameter values in Panels A and B are precisely the same, except for the lower value of b in Panel B. With a lower value for b the marginal cost curves intersect the marginal benefit curve to the right of x_c. As a consequence, the increase in prevalence leads to a reduction in the expected marginal cost of infection and an increase in the number of partners from point A to point B.

Figure 9 demonstrates how combinations of the preference parameters α and b map into various values of κ and associated behavioral responses. The lower-right region with high b and low α leads to $\kappa < 0$ and an endemic equilibrium with dynamic dampening (corresponding to Panel A in Figure 7). The upper-left region with low b and high α results in susceptible individuals choosing the maximum number of partners each period regardless of changes in prevalence. This occurs because the with a sufficiently low value for b, the marginal cost curves in Figure 7 will be everywhere below the marginal benefit curve. The band between the two regions has $\kappa > 0$ and results in rational dynamic resonance (corresponding to Panel B in Figure 7).

Figure 10 contrasts the dynamic responses of the E-SIRS and SIRS models to a one-time, five percentage point increase in the infection rate. The parameter values in Figures 8 and 10 are identical except for the health parameter b, which is decreased about 25% from 2.25 to 1.66. The new steady-state implies an increase in partners from eight to 15, causing fatalism to set in. When prevalence is rising, individuals choose more partners forcing prevalence even higher; when prevalence is falling, individuals choose fewer partners forcing prevalence even lower. This resonance between the initial change in infection rates and optimal partner choice causes cycles to be amplified and drawn out.[14]

Are fatalism and rational dynamic resonance simply a theoretical curiosity? The answer appears to be no. There is some indirect evidence that fatalism and rational dynamic resonance may exist. Kremer (1996) cites anecdotal evidence that individuals have displayed fatalism with respect to AIDS in high prevalence regions of Uganda. Particularly in the developed world, syphilis is not currently as grave of a concern as AIDS. However, that has not always been the case. In the late 15th century, a syphilis epidemic spread throughout Europe leading to millions of deaths

[14] For the parameter values in Figure 10, the elasticity of partner change with respect to aggregate infections is $\kappa = 0.69$. Furthermore, if we hold x fixed at its steady-state value, the probability of infection at the steady state is 0.66, increasing to 0.80 with the five percentage point increase in the aggregate infection rate.

(Hayden (2003)). Into the 20th century syphilis continued to be one of society's primary health concerns, accounting for "10% of public health expenditures in the U.S., 1 in 14 of all mental hospital admissions and 20,000 annual deaths" in 1936 (Green *et al.* (2001)). Brown (1971) also estimated that because many cases of syphilis escape detection, the actual number of cases may be more than five times higher than reported numbers. Furthermore, when you factor in the probability of contracting the suite of other STDs such as gonorrhea, chlamydia and HIV, high-risk individuals may become resigned to the idea of contracting a sexually transmitted disease and take on additional partners in response to increases in disease prevalence.

The final piece of evidence for rational dynamic resonance comes from the E-SIRS model and surveys of the sexual behavior of high-risk individuals. Using the parameter values from Figure 10, the E-SIRS model predicts that the threshold number of partners, x_c, required to induce rational fatalism and dynamic resonance is approximately 15 partners per year. Several studies indicate that the rate of partner change among the population of high-risk individuals exceeds $x_c = 15$. For example, McKusick *et al.* (1985) report that from a sample of 454 high-risk homosexual men, over 50% have had more than 24 partners in a year, with an average exceeding 40. Koblin *et al.* (2003), based on a non-HIV sample of approximately 4,300 homosexual men across 6 major U.S. cities, find that over half the sample report having more than 15 partners per year; nearly half report more than 20 partners per year. At current rates of prevalence, the rational response for these individuals is to resign themselves to the likelihood of contracting the disease and behave in a fatalistic manner. That is, individuals will take on additional partners when prevalence rises and take on fewer partners when prevalence falls, contributing to the amplification of syphilis cycles.

5 Eradication

The stability properties of the eradication equilibrium are critical to policy makers who wish to rid society of the disease. The transition matrix around the eradication steady state simplifies to

$$\begin{bmatrix} 1-\nu-\mu+\bar{x}\lambda_p & 0 \\ \nu & 1-\gamma-\mu \end{bmatrix} \tag{21}$$

with eigenvalues $(1-\nu-\mu+\bar{x}\lambda_p)$ and $(1-\gamma-\mu)$.[15] These two roots are always real so when the system is stable, it converges monotonically to the eradication steady state. The stability frontier is found by setting the first eigenvalue, $1-\nu-\mu+\bar{x}\lambda_p$, equal to one.[16] Any value greater than one will cause eradication to be unstable. The critical value of ν that just makes eradication stable is given by

$$\nu^* = \bar{x}\lambda_p - \mu, \tag{22}$$

which shows that treatment rate must be at least as large as the rate of new infections (net of new susceptibles) for eradication to be locally stable. The eradication stability frontier is independent of b implying that changes in the relative concern for health do not impact the local stability of eradication. This occurs because b affects the transition dynamics exclusively through its impact on in (see matrix system (16)). When stability is evaluated at the eradication steady state (implying that $in = 0$), b has no role in the transition dynamics and local stability.

Alternatively, the stability threshold (22) for eradication can be interpreted in terms of the basic reproduction number $R_0 = \lambda/(\mu+\nu)$, which using L'Hôpital's rule reduces to $R_0 = \lambda_p\bar{x}/(\mu+\nu)$. The standard result in the epidemiological literature is that eradication is locally stable if R_0 is less than one (Anderson and May (1991)). The intuition for this result is straightforward – for eradication to be stable, the rate at which people are entering the infection pool ($\lambda_p\bar{x}$) must be less than the rate at which people are leaving the infected pool ($\mu+\nu$).

The remarkable policy implication of (22) is that eradication is nearly impossible if individuals are forward-looking and self-interested. When syphilis is near eradication, this is precisely the time when a rational susceptible individual will choose to the maximum number of sexual partners because the risk of matching up with an infected individual is very low. Garnett *et al.* (1997) survey several studies and write that "the one potentially unbiased study suggests that the transmission probability is around 60%." Using a partner transmission probability of $\lambda_p = 0.6$, a treatment rate of $\nu = 0.9$ and a 3% population growth rate, anything other than complete abstinence or monogamy (choosing a maximum of one partner per year) will cause eradication to be unstable and the system will gravitate toward an endemic steady state.

[15] To derive the transition matrix around the eradication steady state, take equations (14) and (15) and substitute in the linearized constraint $\hat{s}_t + \widehat{in}_t + \hat{r}_t = 0$. Equation (12) is not relevant at eradication because the individual is at a boundary solution and chooses $x_t = \bar{x}$ in each period, which also implies that $\hat{x}_t = 0$ and b is of no consequence.

[16] The other eigenvalue will be less than one in magnitude because our calibrations always satisfy $\gamma + \mu < 1$.

5.1 U.S. Plans to Eradicate Syphilis

Encouraged by historically low infection rates in the late 1950s and in the late 1990s, the CDC has twice unveiled formal plans to eradicate syphilis from the general population (U.S. Department of Health and Welfare (1963); CDC (1999)). Both plans emphasized improved reporting and data gathering, rapid diagnosis and treatment of outbreaks, and a concerted effort to increase individuals awareness of the health consequences of sexual activity. It is easy to understand the motivation for these eradication plans. For example, in 1956 the reported number of primary and secondary syphilis cases had fallen to 6,392 or approximately one infection for every 26,000 persons (see Figure 1). Similarly, in 1999, the reported number of cases was 5,797 or approximately one infection for every 45,000 persons. With proper education regarding prevention and treatment, it seems reasonable that policy makers at the CDC should be able to continue the downward trend and eventually eliminate the disease altogether. Yet syphilis rates did not fall. In fact, after the 1999 Eradication Plan, rates of primary and secondary syphilis incidence rose with infections in 2005 being 46% higher than in 2000.

Why did these plans fall short of their desired objectives? The answer lies not with the biology of the disease but rather with economic principles. In the late 1950s and the late 1990s, when U.S. infection rates were very low, susceptible individuals realized that they faced a relatively low risk of matching with an infected partner and contracting the disease. Economic theory predicts that self-interested, rational individuals will react to this reduced risk by increasing the number of sexual partners until the benefits of additional partners are balanced by the additional risks of infection. The surprising result from the stability analysis above is that for reasonable values of the transmission, treatment and population growth parameters, anything other than abstinence or monogamy will make eradication infeasible. Even if we consider a reasonably altruistic infected population (e.g., $\theta = 0.5$), such that half the infected population abstains from sexual activity, eradication still becomes unstable with anything more than three partners per year. This number is still well below commonly accepted estimates of partner frequency per year for those at risk of syphilis (Andrus *et al.* (1990)).

The primary lesson here is that when infection rates drop to such low levels, such as those seen in the U.S. in 1956 and 2000, is precisely the time when individuals will choose the highest number of partners. As a result, the disease remains within the general population and continues to fluctuate around its endemic long-run rate.

6 Conclusion

Understanding the role of human behavior in infectious disease dynamics is a key part of designing and implementing effective public health policy. For the case of syphilis, Grassly *et al.* (2005) claim social and behavioral responses play a secondary role in the evolution of the disease. This implies that strategies directed towards changing sexual practices may be of limited use in controlling the disease. Using a dynamic model that melds together optimizing human behavior and infectious disease dynamics, we demonstrate the importance of human responses on the dynamics of the disease. In particular, we show that human responses may either dampen or exacerbate the magnitude and duration of syphilis cycles**.** This finding gives credence to public health policies that aim to change sexual practices in an attempt to control the disease.

In addition, the method developed in this paper establishes the micro-level foundations for the classic SIRS epidemiological model. The derived system closely resembles the classic SIRS model apart from the traditional infection parameter, which is instead time-varying and endogenous.

Perhaps the most striking implication of incorporating human behavior into the SIRS syphilis model is that eradication of the disease is nearly impossible. When syphilis is near eradication, this is precisely the time when a rational susceptible individual will choose to be as risky as possible because the chance of matching up with an infected individual is very low. If individuals are driven by self-interest, any response other than complete abstinence or monogamy will cause eradication to be locally unstable and the system will gravitate toward an endemic steady state. As a result, public health officials may be better served by directing their efforts toward finding the best mix of education, prevention and treatment policies to reach a more desirable endemic equilibrium.

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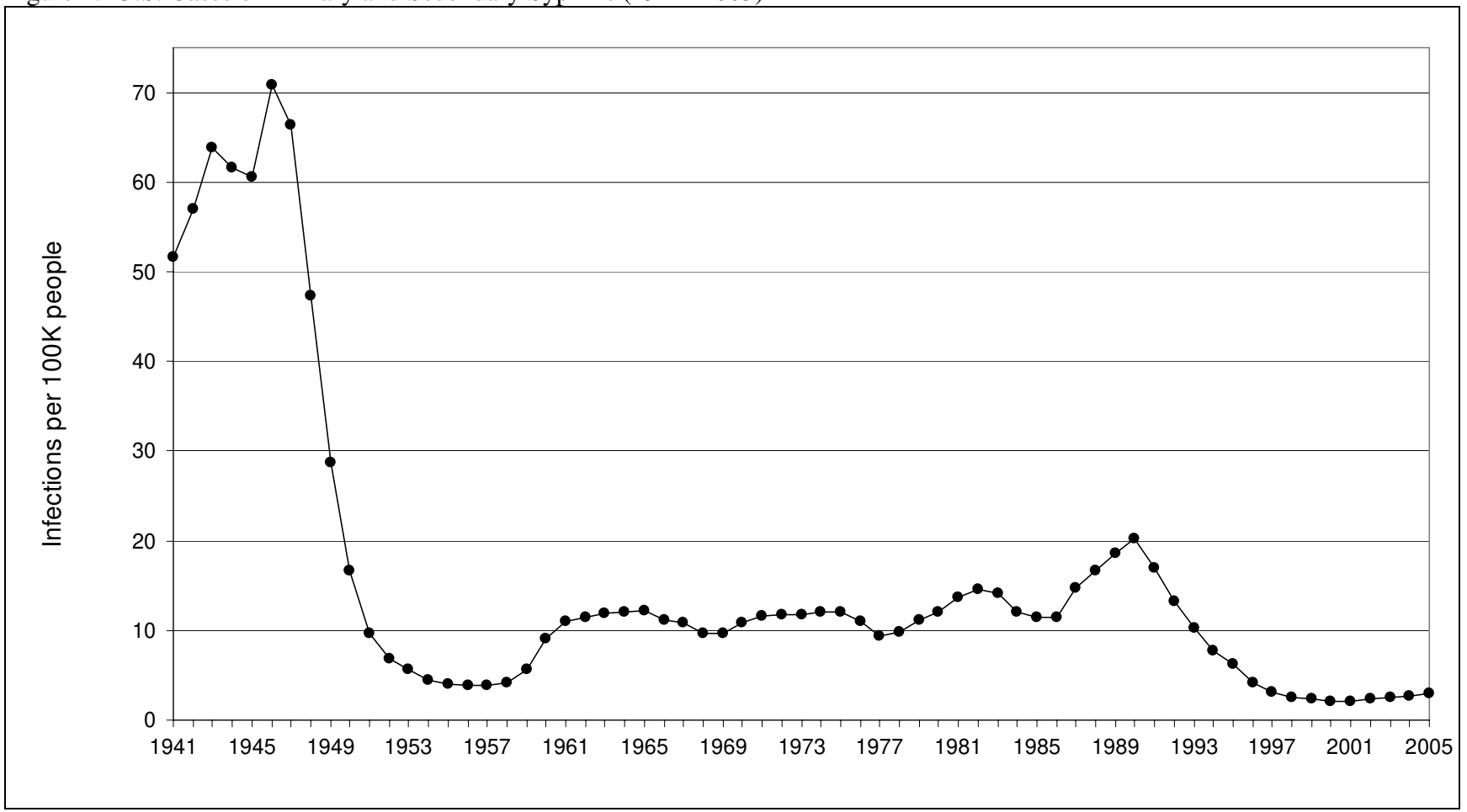
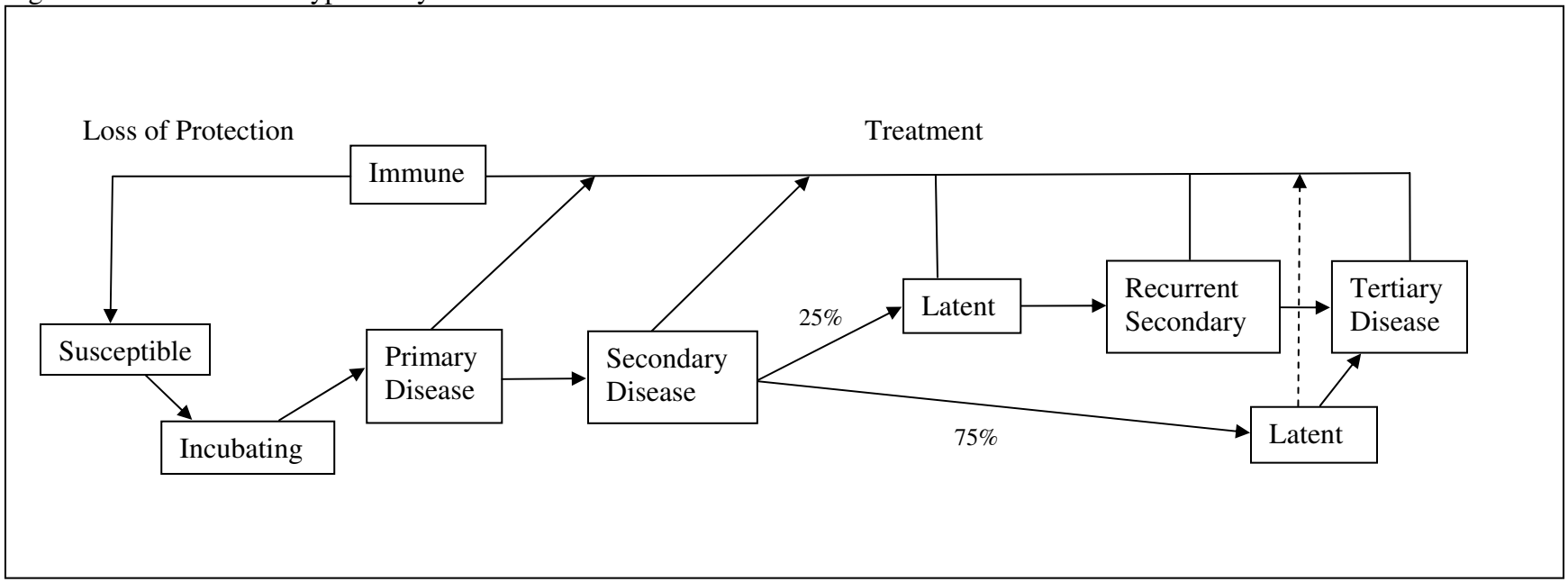
Figure 1. U.S. Cases of Primary and Secondary Syphilis (1941- 2005)



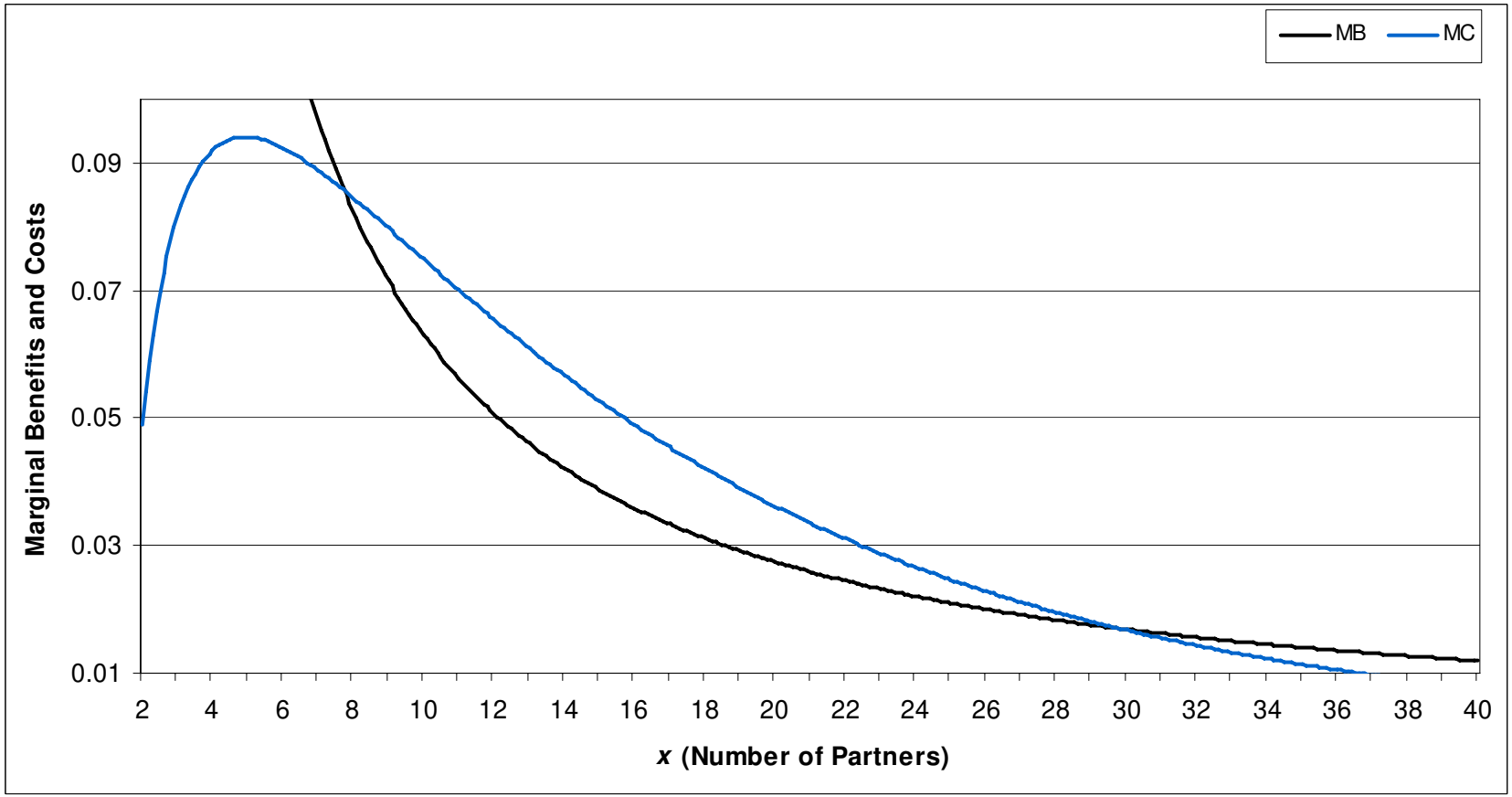
Source: Center for Disease Control and Prevention (www.cdc.gov/std/syphilis/).

Figure 2. Flow Chart for Syphilis Dynamics



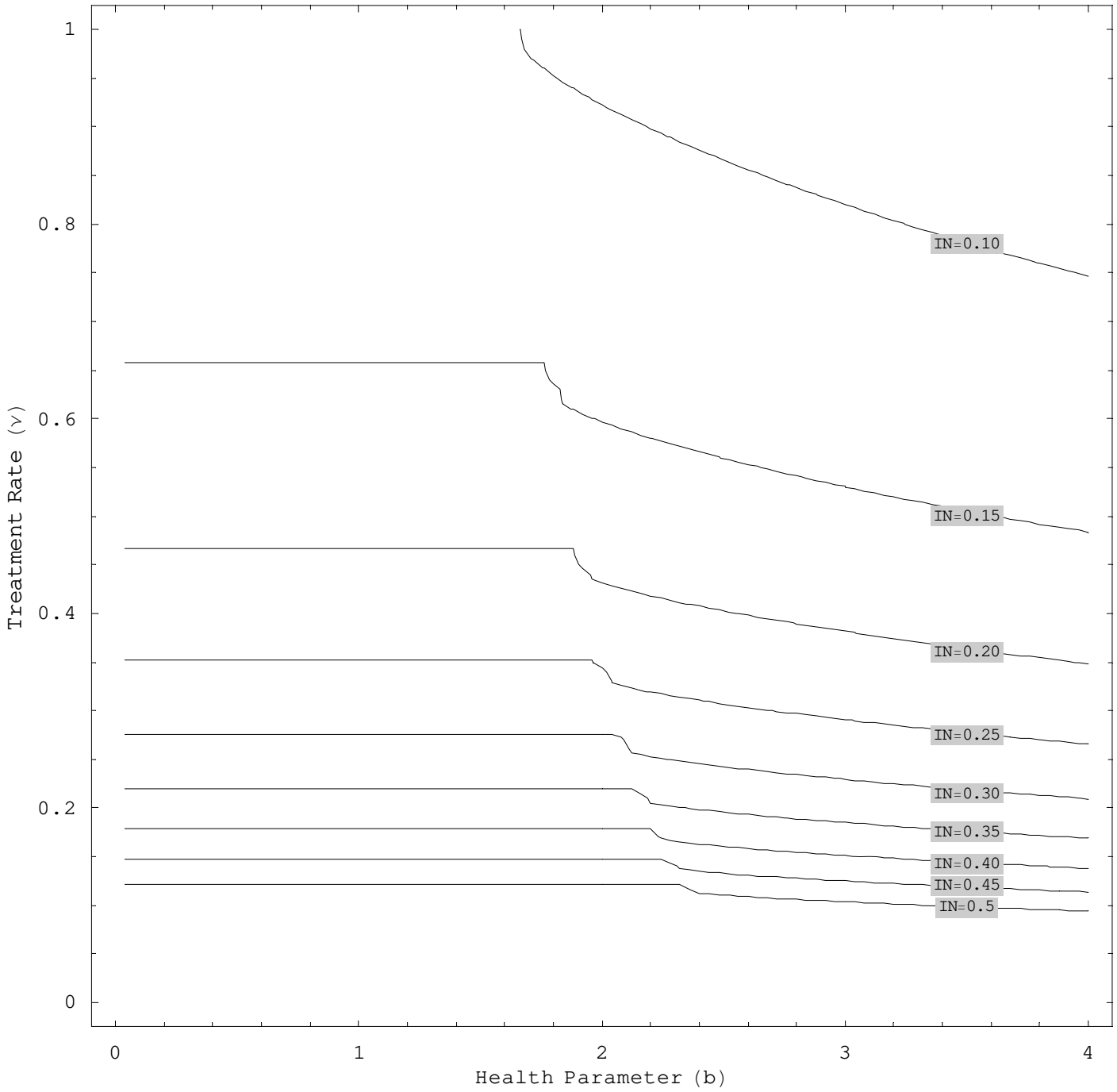
Reproduced from Garnett et al. (1997).

Figure 3. Equilibrium Outcome for the E-SIRS Model



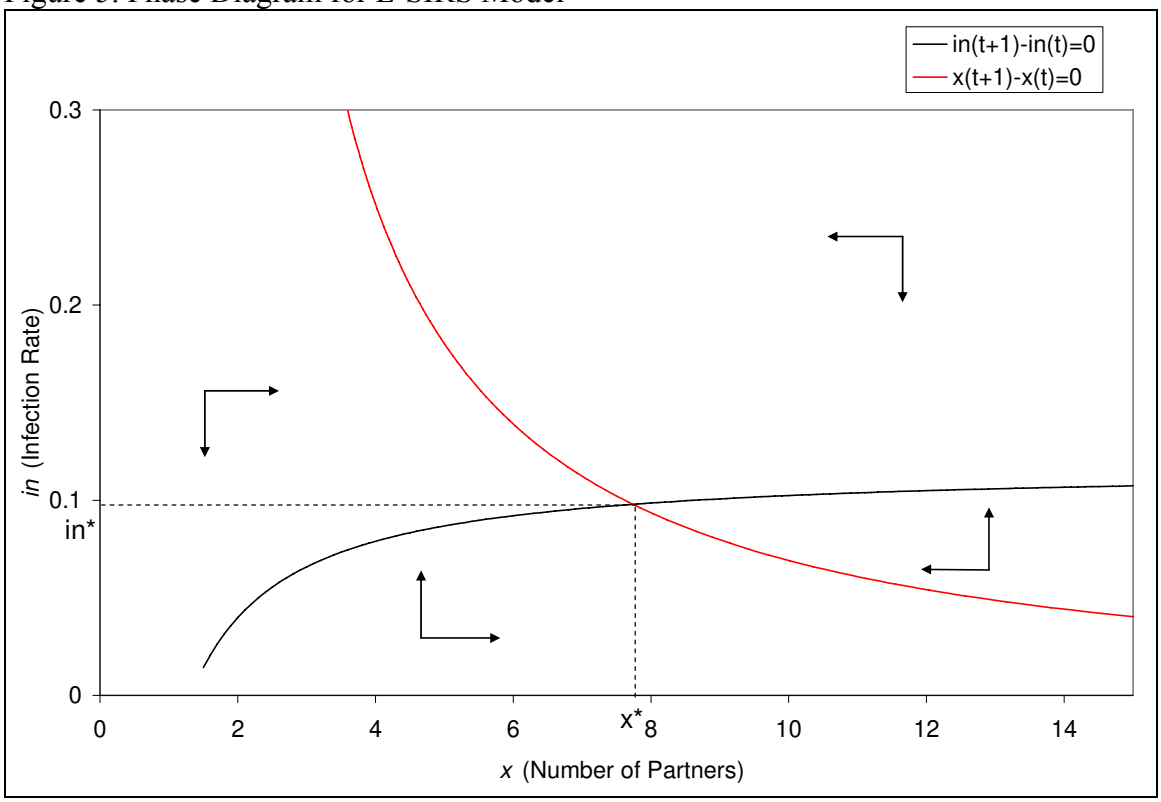
Notes: The parameter values are set at $\alpha = -0.2$, $b = 2.25$, $\beta = 0.96$, $\gamma = 0.1$, $\delta = 0.05$, $\mu = 0.03$, $\lambda_a = 0.3$, $a = 3$ and $\nu = 0.9$. The marginal cost curve is calculated by solving the steady-state SIRS system for aggregate infection rates at the various levels of x. MB ≡ marginal benefits. MC ≡ marginal costs.

Figure 4. Contour Map for Steady-State Infection Rates



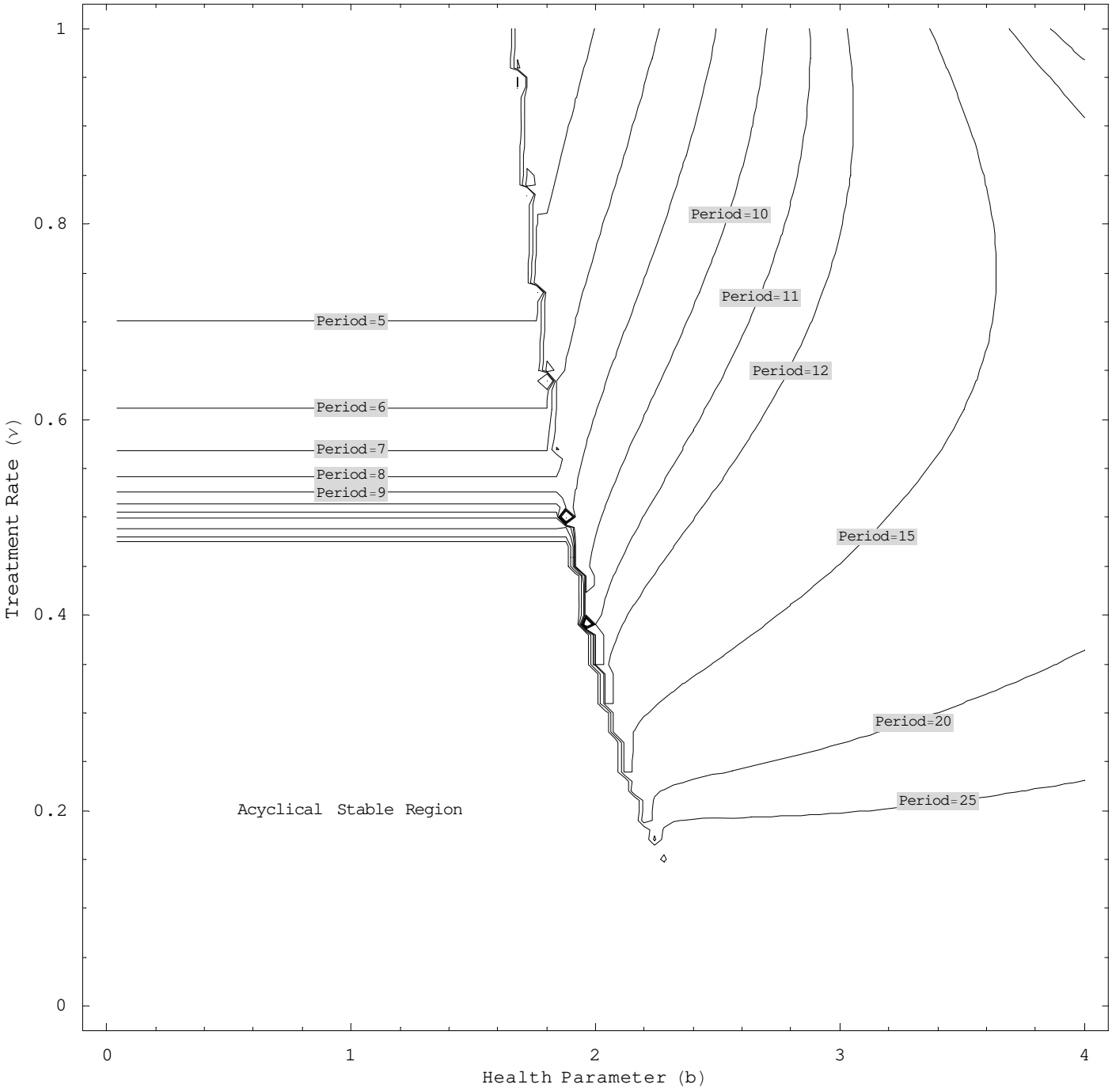
Notes. The fundamental parameters are set at $\alpha = -0.2$, $\beta = 0.96$, $\delta = 0.05$, $\mu = 0.03$, $\gamma = 0.1$, $\lambda_a = 0.3$ and $a = 3$.

Figure 5. Phase Diagram for E-SIRS Model



Notes. The parameter values are set at $\alpha = -0.2$, $\beta = 0.96$, $\gamma = 0.1$, $\delta = 0.05$, $\lambda_a = 0.3$, $a = 3$, $\mu = 0.03$, $b = 2.25$, and $\nu = 0.9$. The steady state of the E-SIRS model, (in^*, x^*), is found at the intersection of the time invariant loci for *in* and *x*.

Figure 6. Contour Graph for Periodicity around the Endemic Steady State



Notes. The fundamental parameters are set at $\alpha = -0.2$, $\beta = 0.96$, $\delta = 0.05$, $\mu = 0.03$, $\gamma = 0.1$, $\lambda_a = 0.3$ and $a = 3$.

Figure 7. Individual Optimal Choice and the Probability of Infection: Exogenous Increase in Prevalence

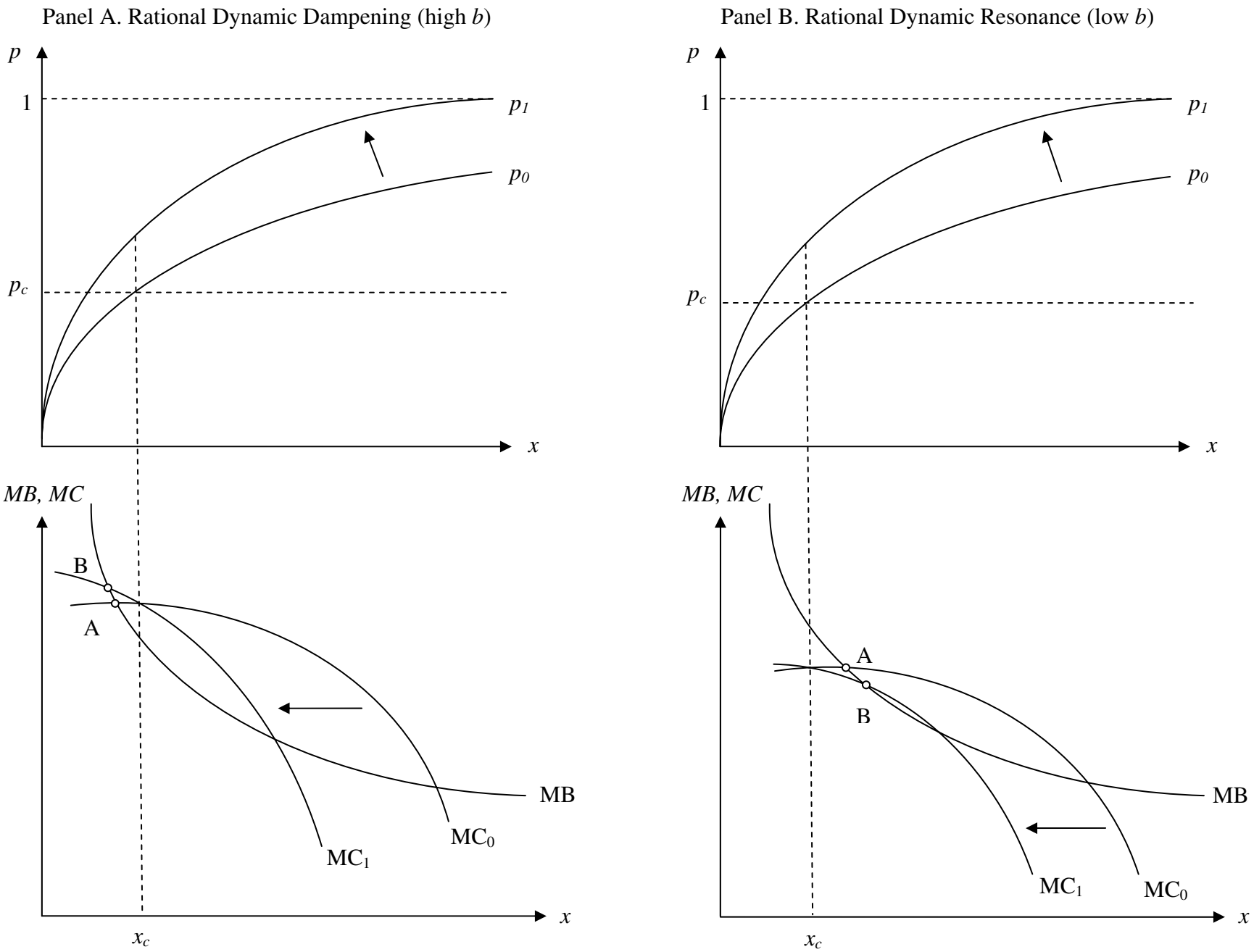
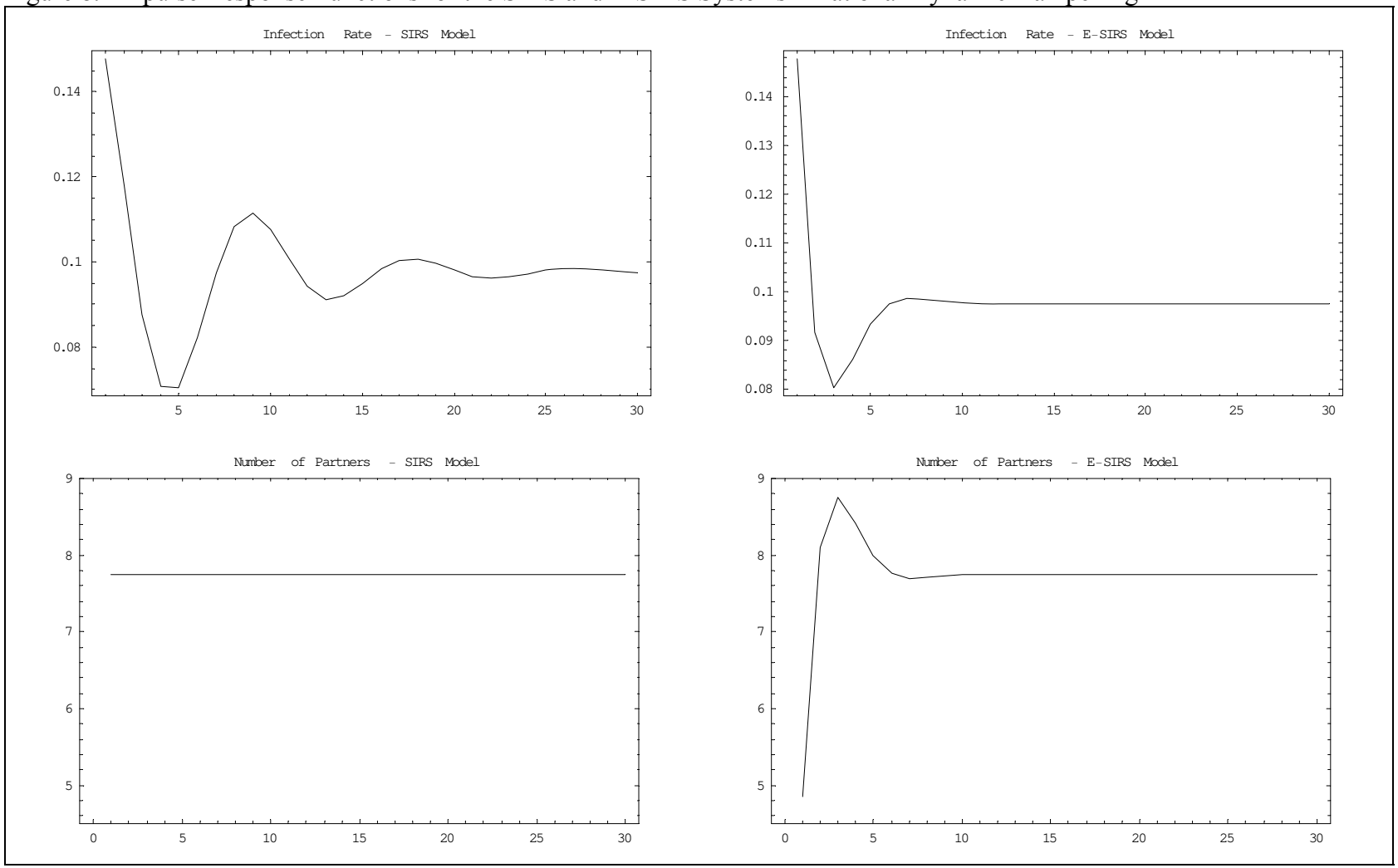
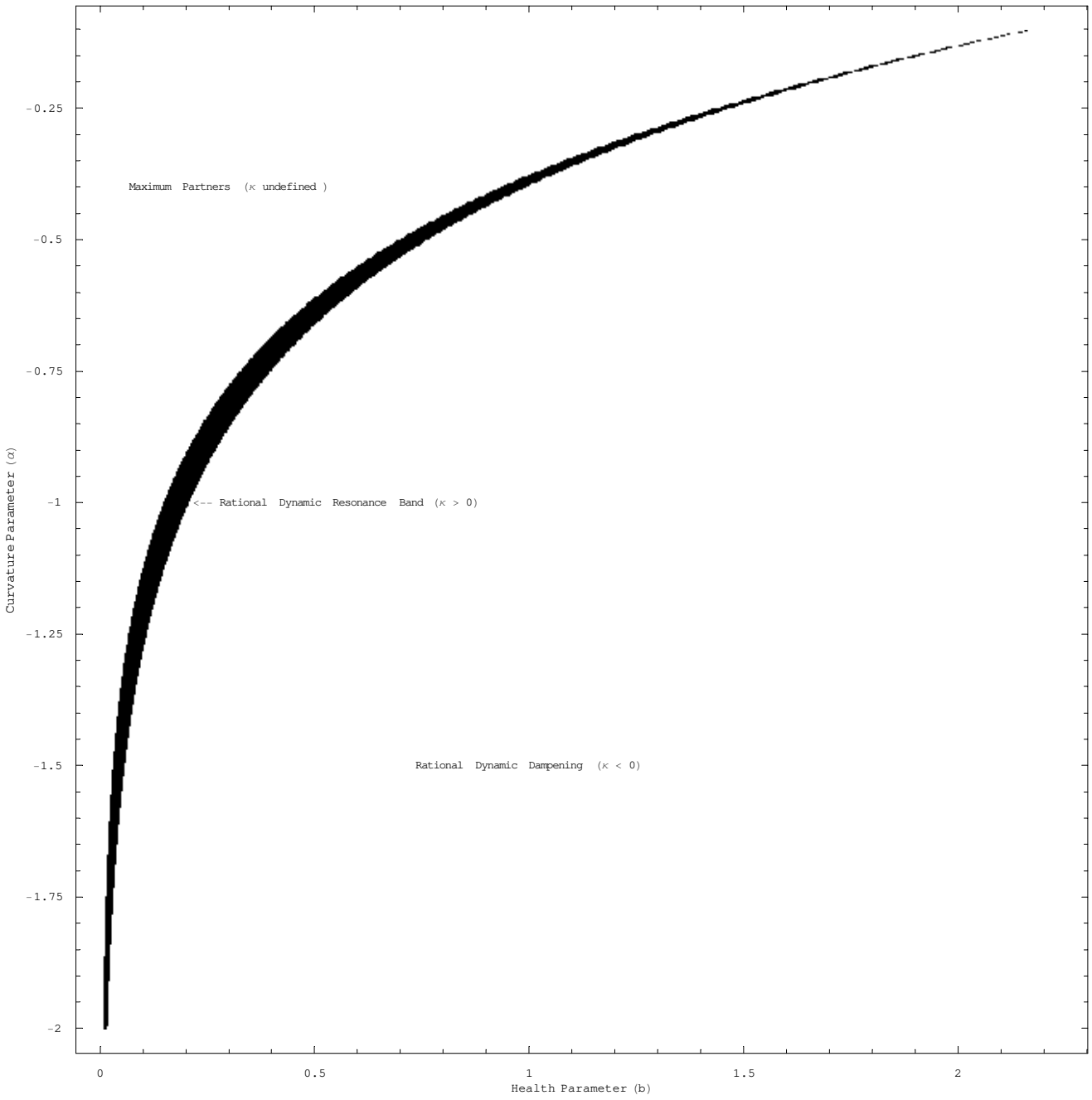


Figure 8. Impulse Response Functions for the SIRS and E-SIRS Systems – Rational Dynamic Dampening



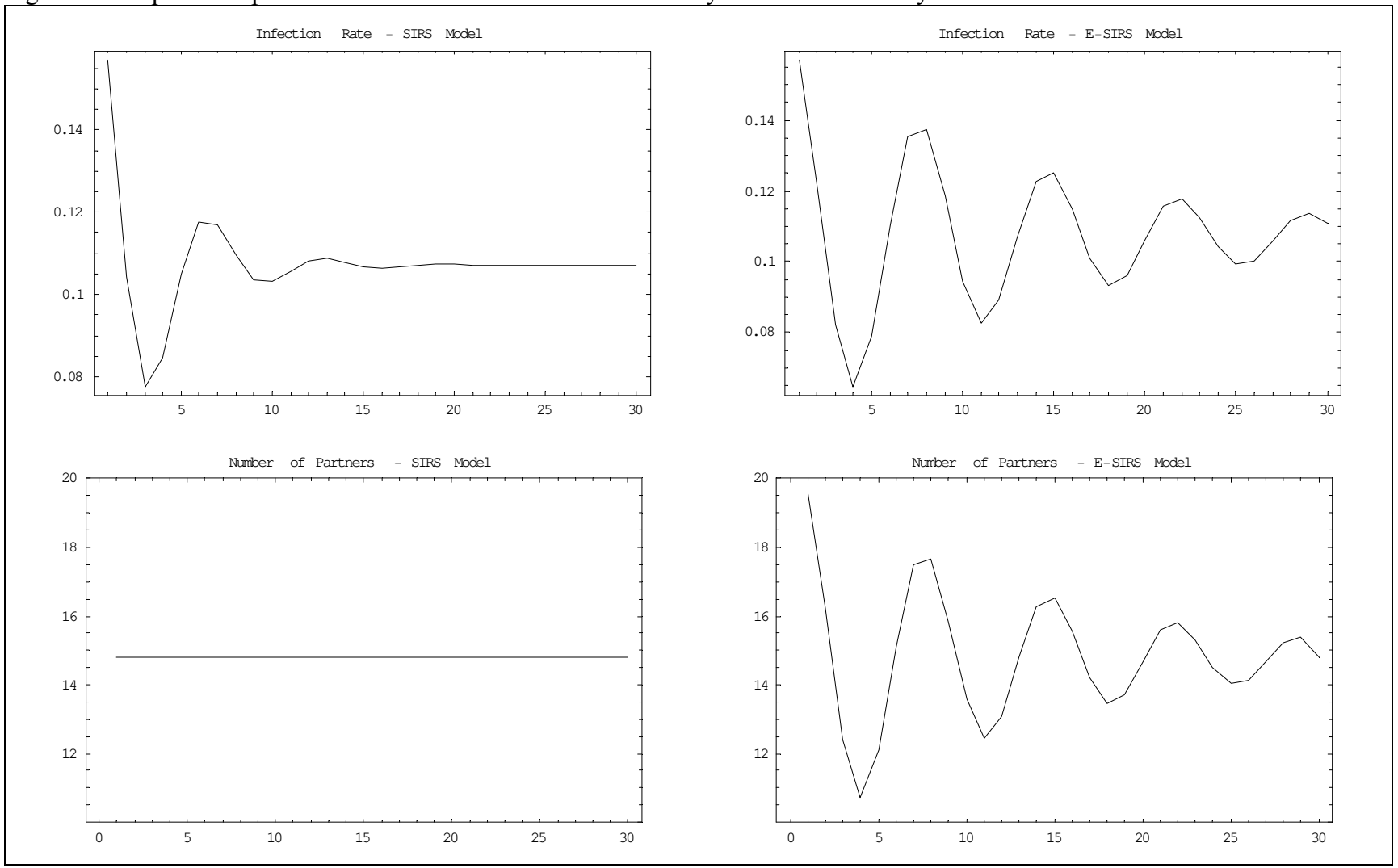
Notes. The fundamental parameters in the E-SIRS system are set at $\alpha = -0.2$, $\beta = 0.96$, $\delta = 0.05$, $\mu = 0.03$, $\gamma = 0.1$, $b = 2.25$, $\nu = 0.9$, $\lambda_a = 0.3$ and $a = 3$. For comparison purposes, we set the steady-state number of partners (x) in the SIRS model equal to the endogenously solved for number of partners in the E-SIRS model. As a result, the steady-state infection rates are also equal in the SIRS and E-SIRS models.

Figure 9. Preferences and Partner Elasticity Regions



Notes. The fundamental parameters are set at $\nu = 0.9$, $\beta = 0.96$, $\delta = 0.05$, $\mu = 0.03$, $\gamma = 0.1$, $\lambda_a = 0.3$ and $a = 3$.

Figure 10. Impulse Response Functions for the SIRS and E-SIRS Systems – Rational Dynamic Resonance



Notes. The fundamental parameters in the E-SIRS system are set at $\alpha = -0.2$, $\beta = 0.96$, $\delta = 0.05$, $\mu = 0.03$, $\gamma = 0.1$, $b = 1.66$, $\nu = 0.9$, $\lambda_a = 0.3$ and $a = 3$. For comparison purposes, we set the steady-state number of partners (x) in the SIRS model equal to the endogenously solved for number of partners in the E-SIRS model. As a result, the steady-state infection rates are also equal in the SIRS and E-SIRS models.