SYPHILIS CYCLES

by

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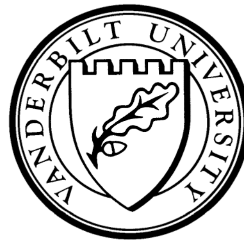

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Syphilis Cycles

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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 on the rise and has cycled around endemic levels for decades. We investigate syphilis dynamics by extending the classic SIRS epidemiological model to incorporate forward-looking, rational individuals and the AIDS epidemic. The integrated economic-epidemiological model shows 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 model also provides insights into two failed attempts by the U.S. government to eradicate syphilis from the U.S. population.

JEL Codes: D1, I1.

Keywords: syphilis, AIDS, 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 1930s and 1940s, 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 (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 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 1940s 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, the trend appears to have reversed. Infection rates for syphilis are rising in North America, Western Europe and Australia (Fenton et al. (2008)).

Syphilis also remains a persistent health threat in lesser developed countries. The World Health Organization (WHO) estimates that approximately 12 million new worldwide 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 and AIDS (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 1990s (Chen et al. (2007)).

In the face of these concerns, 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

[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 starting in 1932 (Nakashima et al. (1996)). In 1997, the U.S. government formally apologized for the incident.

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 incidence of syphilis in the U.S. has nearly doubled 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 because all sexually transmitted diseases (STDs) are driven by the same risky behavior. If the increases in the prevalence of syphilis indicate greater sexual promiscuity and reduced use of safe-sex practices, they could warn of a future rise in other STDs including HIV. Figure 1 demonstrates the similarity between the pattern of AIDS incidence and that of syphilis.

It is precisely these reasons (epidemic cycling; widespread public health concern, and transmission risks inseparable from other STDs) that syphilis is a pertinent and interesting research topic. There has also been a recent high profile debate over the determinants of the oscillations. At its core, the debate has revolved around whether the origins lie with the biology of the disease (Grassly, Fraser and Garnett (2005)) or with changes in societal behavior and treatment intensities (Breban et al. (2008)). Both sides of the debate use mathematical epidemiological (ME) models that fix human responses to disease risk. This runs counter to the findings of economists who have demonstrated the importance of individuals' ability to respond to changes in risk (Ehrlich and Becker (1972); Peltzman (1975); Rosen (1981); Viscusi (1990); Shogren and Crocker (1991)). Building on the principle that individuals respond to risk, we specify an integrated economic-epidemiological (EE) model where humans and disease biology work together to determine disease dynamics. Following the 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 joint model of syphilis and AIDS dynamics.[2] To the best of our knowledge, we are the first to model

[2] This integrated model is derived directly from the behavior of rational individuals. The resulting dynamic system closely resembles classic epidemiological models (Murray (2002)) with one major difference. In the integrated model, the traditional infection parameters are not fixed but vary over time and depend 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

more than one infectious disease within an optimization framework.

We highlight two findings from our research: (1) the existence and nature of syphilis cycles depend critically on human preferences over sexual activity and health and (2) policies designed to eradicate syphilis are likely to fall short of their objective. First, 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 a 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 first demonstrated by Kremer (1996). Here, we examine the conditions under which fatalism extends to a dynamic setting and to more than one disease. The potential of fatalism in a dynamic context is 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*.[3]

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. Assuming reasonable rates of transmission and rational, self-interested individuals, our model predicts that programs aimed at eradication are likely to fall short of the desired objective. The intuition is

natural rates 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.

[3]This effect is in contrast to the effect of coherence resonance (see for example, Dushoff et al. (2004)). Coherence resonance can amplify cycles and is derived from the interaction of the mean infection period and the average duration of immunity. In the modeling of the effect, the contact rate is specified *a priori* by a sinusoidal function with no behavioral basis.

straightforward: when prevalence of the disease is low and nearing eradication, this is precisely when sexually-active, rational individuals will choose to increase their number of partners and perpetuate the disease. Successful eradication is theoretically possible but it requires implausibly high degrees of altruism for those infected with syphilis and AIDS.

Of course, eradication policies can still have a positive impact. 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. To some degree, the 1999 syphilis eradication plan had a greater chance of success due to the prominence of AIDS risk among sexually active individuals. However, the window of opportunity to eradicate syphilis may be closing due to the discovery of new drug therapies that lower the health risks of AIDS and encourage more risky sexual behavior. This likely explains the recent rise in the prevalence of syphilis.

2 Syphilis Epidemiology

Syphilis is an 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)). 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 and is outlined in Figure 2.

The defining feature of aggregate syphilis dynamics is the regular cycle in disease prevalence (see Figure 1). As argued by Grassly et al. (2005), 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 also cause cycles to persist well past any initial driving impulse. AIDS and gonorrhea, for example, share the same method of contraction as syphilis but lack transitory host immunity and do not oscillate. Using gonorrhea as a comparison, Grassly et al. (2005) draw the conclusion that syphilis cycles during the three-decade period following 1960 must be due to disease biology rather than popular explanations involving the sexual liberation of the 1960s and the crack cocaine epidemic of the mid-to-late 1980s.

To the casual observer, syphilis is a 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, Talbot and Morton (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. Yet, these numbers mask serious policy issues.

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

arise with gay men (CDC (2006)). Second, statistics in the underdeveloped world are grim. As mentioned in the Introduction, there are approximately 12 million new worldwide syphilis infections per year and over 1.5 million cases of congenital syphilis in Sub-Saharan Africa alone. Finally, lesions caused by syphilis act as a conduit for other STDs and has been shown to significantly increase the chance of acquiring HIV (Chesson and Pinkerton (2000)).[4]

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 Integrated General Equilibrium Model

Following work by Philipson and Posner (1993), we specify an integrated epidemiological and economic model to describe syphilis dynamics. Sexual activity brings multiple risks that the individual cannot choose between, with AIDS being the most serious. Therefore to understand syphilis dynamics over the past three decades, we specify a joint SIRS/SI epidemiological model of syphilis and AIDS dynamics. The model is set in discrete time with t indexing annual decision intervals.[5] There is a constant population of N individuals, which are all identical except for their state of the disease.

3.1 Epidemiology

The epidemiological portion of the model describes the evolution of six mutually exclusive disease categories: susceptible to both diseases (s), infected with syphilis only (in^S), infected with AIDS only (in^A), infected with syphilis and AIDS (in^{SA}), immune to syphilis (r), and immune to syphilis while infected with AIDS (r^A). Each disease category is measured as a proportion of the overall

[4] Chesson, Dee and Aral (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 1990s.

[5] The SIRS and SI models are 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.

population with the sum of the categories equal to one. The model collapses to a traditional SIRS model when $in_t^A = in_t^{SA} = r_t^A = 0$ and to a traditional SI model when $in_t^S = in_t^{SA} = r_t = r_t^A = 0$.

Individual behavior and the population disease dynamics depend on the transition probabilities. Assuming that individuals independently choose x_t partners and engage in a fixed number of sexual acts (a) with each partner, the probability that susceptible individuals become infected with syphilis or AIDS is

$$p_t^S = \Pr(\text{contract syphilis}) = 1 - [1 - \lambda_p^S(in_t^S + in_t^{SA})]^{x_t} \quad (1)$$

$$p_t^A = \Pr(\text{contract AIDS}) = 1 - [1 - \lambda_p^A(in_t^A + in_t^{SA} + r_t^A)]^{x_t}, \quad (2)$$

where $\lambda_p^j = 1 - (1 - \lambda_a^j)^a$ is the probability of contracting disease $j \in \{S, A\}$ from a single infected partner, and λ_a^j is the probability of contracting the disease from a single sexual act. The conditional probabilities for those infected with one disease are:

$$p_t^{S|A} = \Pr(\text{contract syphilis} \mid \text{infected with AIDS}) = 1 - [1 - \lambda_p^{S|A}(in_t^S + in_t^{SA})]^{x_t^A} \quad (3)$$

$$p_t^{A|S} = \Pr(\text{contract AIDS} \mid \text{infected with syphilis}) = 1 - [1 - \lambda_p^{A|S}(in_t^A + in_t^{SA} + r_t^A)]^{x_t^S}, \quad (4)$$

where x_t^S (x_t^A) is the number of partners chosen by those infected with syphilis (AIDS). Individuals infected with syphilis or AIDS are allowed to have a different natural probability of infection, $\lambda_p^{S|A}$ and $\lambda_p^{A|S}$, than those without a disease. As mentioned above, those with primary or secondary syphilis have an elevated probability of acquiring HIV (i.e., $\lambda_p^{A|S} > \lambda_p^A$). The dependence on the chosen number of partners distinguishes the analysis from standard mathematical epidemiology.

The complete epidemiological model is represented by the following six equations:

$$
\begin{aligned}
s_{t+1} &= \mu + [(1-p_t^S)(1-p_t^A) - \mu]s_t + \gamma(1-p_t^{A|S})r_t && (5) \\
in_{t+1}^S &= -\mu in_t^S + p_t^S(1-p_t^A)s_t && (6) \\
in_{t+1}^A &= (1-\mu-p_t^{S|A})in_t^A + p_t^A(1-p_t^S)s_t + \gamma p_t^{A|S} r_t + \gamma r_t^A && (7) \\
in_{t+1}^{SA} &= -\mu in_t^{SA} + p_t^{S|A} in_t^A + p_t^S p_t^A s_t && (8) \\
r_{t+1} &= [(1-p_t^{A|S})(1-\gamma) - \mu]r_t + (1-p_t^{A|S})in_t^S && (9) \\
r_{t+1}^A &= (1-\mu-\gamma)r_t^A + (1-\gamma)p_t^{A|S} r_t + p_t^{A|S} in_t^S + in_t^{SA}, && (10)
\end{aligned}
$$

where μ is the common birth/death rate, $1/\gamma$ is the average duration of syphilis immunity, and the treatment rate for syphilis is 100%. The transition matrix between disease categories used to derive the epidemiological model is shown in the Appendix.

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

3.2 Economics

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

$$E \sum_{j=0}^{\infty} \beta^{t+j}[\ln(x_{i,t+j}) + h_{i,t+j}] \qquad (11)$$

where $0 \leq \beta \leq 1$ is the discount factor, E represents an individual's expectation of future outcomes and $\bar{x}$ is the maximum number of partners in a single 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 sexual partners bring immediate satisfaction but also the risk of future infection. Infection in turn causes a deterioration of health.[6]

[6] The risk of contracting an STD can be manipulated by varying the level of protection or the number of partners. Geoffard and Philipson (1996) and Toxvaerd (2010) are examples of studies where the control variable is costly prevention, such as using prophylaxis. Kremer (1996) and Auld (2003) are examples where the control variable is the number of partners. Both methods capture the essential tradeoff that risk of infection can be reduced by costly behavior, either increased protection or taking fewer partners.

In any period t, individual i must be in one of the six epidemiological states: susceptible ($s_{i,t}$), infected with syphilis ($in^S_{i,t}$), infected with AIDS ($in^A_{i,t}$), infected with syphilis and AIDS ($in^{SA}_{i,t}$), recovered and immune from syphilis ($r_{i,t}$) or recovered and immune from syphilis with AIDS ($r^A_{i,t}$). For example, if an individual is susceptible to both diseases then $s_{i,t} = 1$ and $in^S_{i,t} = in^A_{i,t} = in^{SA}_{i,t} = r_{i,t} = r^A_{i,t} = 0$. Because an individual can only be in one state at any time, $s_{i,t} + in^S_{i,t} + in^A_{i,t} + in^{SA}_{i,t} + r_{i,t} + r^A_{i,t} = 1$ for all i and 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, we drop the i subscript and consider a single representative individual in each disease category.

The biology of syphilis immunity in humans is complicated and difficult for individuals to detect (Garnett et al. (1997); LaFond and Lukehart (2006)). Therefore, although there are six epidemiological categories, the two recovered states are not relevant for decision making because individuals are unable to identify whether they are immune to syphilis. The value functions evaluated at the optimal number of partners for each of the four remaining categories – susceptible (V_t), infected with syphilis (V^S_t), infected with AIDS (V^A_t), and infected with syphilis and AIDS (V^{SA}_t) – are given by

$$\begin{aligned}
V_t &= \ln(x_t) + h + \\
&\quad \beta[p^S_t(1-p^A_t)V^S_{t+1} + p^A_t(1-p^S_t)V^A_{t+1} + p^S_t p^A_t V^{SA}_{t+1} + (1-p^S_t)(1-p^A_t)V_{t+1}] \quad (12) \\
V^S_t &= \ln(x^S_t) + h^S + \beta[p^{A|S}_t V^A_{t+1} + (1-p^{A|S}_t)V^S_{t+1}] \quad (13) \\
V^A_t &= \ln(x^A_t) + h^A + \beta[p^{S|A}_t V^{SA}_{t+1} + (1-p^{S|A}_t)V^A_{t+1}] \quad (14) \\
V^{SA}_t &= \ln(\bar{x}) + \beta V^A_{t+1}, \quad (15)
\end{aligned}$$

where the health parameters for susceptible (h), infected with syphilis (h^S) and infected with AIDS (h^A) individuals satisfy $h \geq h^S \geq h^A \geq 0$. The health parameter for individuals infected with both syphilis and AIDS is normalized to zero.

In our baseline model, all individuals regardless of infection status are self-interested and max-

imize (11) without concern for the welfare of the general population. Rational, self-interested individuals infected with syphilis and AIDS will therefore choose the maximum number of partner, $\bar{x}$, because they face no risk of immediate infection (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 propagates the disease through the population and causes susceptible individuals to choose a suboptimal number of sexual partners.[7] Conversely, an altruistic population of infected individuals (or a benevolent social planner guiding the actions of infected individuals) would sharply decrease the number of sexual encounters so the disease could quickly be eradicated. We allow for possible altruism by infected individuals (Philipson and Posner (1993); Gersovitz (2004)) by considering a range of values for $\bar{x}$.

Individuals are forward looking and concerned about future benefits and risks. We consider two types of expectation mechanisms in assessing these future benefits and risks. First, we assume individuals form naïve expectations where all future risks and benefits are expected to remain at their current values. This simplification seems reasonable given the many layers of incomplete information individuals face when attempting to forecast future disease risk. Survey and experimental evidence also shows that individuals often use simpler heuristics or "rules of thumb" to forecast uncertain future variables (Conlisk (1996)). Second, we consider a rational expectations forecast of future variables, whereby individuals have complete knowledge of the laws of motion for disease states and understand the risk-benefit tradeoffs faced by other individuals. Under rational expectations individuals make forecast errors, but they are unrelated to any available current information. Below, we focus on the results for naïve individuals because the role of economic choice on disease dynamics is more transparent. However, we solve for the equilibrium paths under both types of expectations and present the rational expectations results in the Appendix. The main qualitative findings of the paper hold under either expectations mechanism.

Assuming an interior solution, the Euler equations for the number of partners (x_t, x_t^S and x_t^A)

[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.

are

$$
\begin{aligned}
x_t^{-1} &= \beta p_{x,t}^{S}[(1-p_t^A)V_{t+1} - (1-p_t^A)V_{t+1}^S + p_t^A V_{t+1}^A - p_t^A V_{t+1}^{SA}] + \\
&\quad \beta p_{x,t}^{A}[(1-p_t^S)V_{t+1} - (1-p_t^S)V_{t+1}^A + p_t^S V_{t+1}^S - p_t^S V_{t+1}^{SA}] \qquad (16) \\
(x_t^S)^{-1} &= \beta p_{x,t}^{A|S}[V_{t+1} - V_{t+1}^A] \qquad (17) \\
(x_t^A)^{-1} &= \beta p_{x,t}^{S|A}[V_{t+1}^A - V_{t+1}^{SA}]. \qquad (18)
\end{aligned}
$$

where the partial derivative for the probability of infection with respect to the number of partners is of the form $p_{x,t} = -\ln(1-p_t)(1-p_t)/x_t$.[8]

To better understand the Euler equations, consider an individual who is infected with syphilis but is susceptible to AIDS. The basic problem facing the individual is how many partners to choose under the risk of future AIDS infection. Equation (17) represents a standard solution for dynamic expected-utility maximization problems of this type: continue to add partners (x^S) 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 future risk 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 do so at a decreasing rate and cause the incremental costs of sexual activity to fall as more partners are added.

These characteristics create an interesting optimization problem. Because individuals exhibit diminishing marginal utility in x, marginal benefits decline over all x. For a given disease prevalence, marginal costs also decline with x as the marginal probability of infection falls with additional partners. If individuals' relative concern for their health is low, marginal benefits will exceed marginal costs for all choices of x and individuals will choose the maximum number of sexual partners,

[8] The second-order sufficient conditions are presented in the Appendix. The sufficiency conditions 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.

$\bar{x}$. If individuals' concern for their health is high, marginal costs will exceed marginal benefits for all choices of x and the individual will instead abstain from sexual activity. But, if individuals have an intermediate concern for their health and sufficient curvature in utility, the marginal benefit and cost curves intersect twice (once for a low number of partners and once for a high number of partners). While both intersections satisfy the necessary condition for an optimal choice, only the low-partner intersection satisfies the sufficient condition and is the optimal choice (see the Appendix for more details on the second-order conditions).

3.3 Equilibria

An equilibrium for the economic epidemiological system is characterized by a sequence of values $\{x_t, x_t^A, x_t^S, s_t, in_t^A, in_t^A, in_t^{SA}, r_t, r_t^A\}_{t=0}^{\infty}$ that solve the individual's optimization problem and satisfy (5 - 10) for all t, subject to the initial values $s_0, in_0^A, in_0^A, in_0^{SA}, r_0$ and r_0^A. 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 turn our attention to the transition path and short-run equilibrium. For clarity, we only present the equilibrium prior to the AIDS epidemic (the analysis of the system after the AIDS epidemic is presented in the Appendix). Since syphilis is the sole disease of interest, we omit the disease superscripts.

3.3.1 Long-run Equilibria

The long-run equilibrium is obtained when there are no disturbances and the system is allowed to gravitate to its steady state. In general, there are two possible steady states: an endemic equilibrium characterized by low prevalence of syphilis and an eradication equilibrium where syphilis has been eliminated from the population. The steady-state endemic equilibrium is represented by

the following four equations:

$$\begin{aligned}
s &= R_0^{-1} && (19)\\
in &= \frac{(1-s)(\mu+\gamma)}{1+\mu+\gamma} && (20)\\
r &= \frac{(1-s)}{1+\mu+\gamma} && (21)\\
x^{-1} &= \beta p_x(V-V^S) && (22)
\end{aligned}$$

where $R_0 = p[in(1+\mu)]^{-1}$ is the basic reproductive number. R_0 measures the number of susceptible individuals who contract the disease from a single infected person in an otherwise uninfected population (Anderson and May (1991)). In the classic SIRS model (i.e., $h = 0; x = 1$), R_0 is an exogenous constant and the key parameter for determining stability of the eradication steady state. R_0 is also key to the stability of the EE steady state but is endogenous and depends on individual choices. As a result, the dynamics around the EE steady states are linked to individuals' underlying preferences for sexual activity and health.

The baseline values for the parameters and the implied steady states are shown in Table 1[9]:

Table 1. Baseline Parameters and Steady-State Values

Parameters							
β	γ	μ	a	λ_a	h	$\bar{x}$	λ_p
0.96	0.2	0.05	40	0.023	8.1	10	0.60

Endemic Steady-State Values					
x	in	s	r	p	R_0
3.642	0.096	0.519	0.385	0.195	1.927

Eradication Steady-State Values					
x	in	s	r	p	R_0
10	0.000	1.000	0.000	0.000	5.714

In the endemic steady state, approximately 10% of the sexually active population are infected with syphilis and 38.5% are recovered and immune. The probability of infection with a single partner is nearly 20%. In the eradication steady state, individuals take the maximum number of partners, $\bar{x} = 10$, because there is no risk of infection ($p = 0$). The basic reproductive number, R_0, is greater than one indicating that eradication is locally unstable.

3.3.2 Short-Run Equilibrium and Transition Dynamics

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

[9] The calibration exercise is described in the Appendix.

approximation of the system around each steady state. The pre-AIDS EE system collapses to

$$
\begin{aligned}
s_{t+1} &= \mu + (1 - p_t - \mu)s_t + \gamma r_t && (23)\\
in_{t+1} &= -\mu in_t + p_t s_t && (24)\\
r_{t+1} &= (1 - \gamma - \mu)r_t + in_t && (25)\\
\frac{1}{p_{x,t}x_t} &= \beta E\left(\ln(x_{t+1}/\bar{x}) + (h - h^S) - \frac{p_{t+1}}{x_{t+1}p_{x,t+1}}\right), && (26)
\end{aligned}
$$

where E is the expectations operator and the value functions have been substituted out of the Euler equation. After linearizing the EE system and imposing naïve expectations, the system reduces to

$$
\begin{aligned}
\hat{x}_t &= [\kappa x/in]\widehat{in}_t && (27)\\
\widehat{in}_{t+1} &= (sp_{in} - \mu)\widehat{in}_t + sp_x\hat{x}_t + p\hat{s}_t && (28)\\
\hat{r}_{t+1} &= (1 - \gamma - \mu)\hat{r}_t + \widehat{in}_t, && (29)
\end{aligned}
$$

where carets ($\hat{}$) over variables indicate deviations from their steady-state values, κ is the elasticity of partner change (x) with respect to syphilis prevalence (in), and $p_{in} = x\lambda_p(1 - \lambda_p in)^{x-1}$ 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 bivariate dynamic system:

$$
\begin{bmatrix} \widehat{in}_{t+1} \\ \hat{r}_{t+1} \end{bmatrix} = \begin{bmatrix} \phi - (\mu + p) & -p \\ 1 & 1 - \gamma - \mu \end{bmatrix} \begin{bmatrix} \widehat{in}_t \\ \hat{r}_t \end{bmatrix}, \quad (30)
$$

where $\phi = s(p_{in} + p_x\kappa x/in)$ is the sum of two effects on syphilis prevalence. The first effect is standard in mathematical epidemiology and measures how a change in prevalence impacts the probability of infection, holding the number of partners fixed. The second, an economic effect, measures how a change in prevalence impacts the probability of infection through a change in the optimal number of partners. These two effects can work together or in opposite directions depending on individuals' preferences for sexual activity and health.

The equations in (30) determine the transition dynamics around the steady state and the local

stability of the system. Local stability is determined by the magnitude of the following two eigenvalues:

$$-\mu + 0.5\left[1 + (\phi - \gamma - p) \pm \sqrt{1 + (p - \gamma - \phi)^2 - 2(p + \gamma + \phi)}\right]. \quad (31)$$

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 equation (31), we see that the system displays stable cycles if the eigenvalues have modulus less than one and $2(p+\gamma+\phi) > 1 + (p-\gamma-\phi)^2$.[10] Using the parameter values in Table 1, the eigenvalues are $0.474 \pm 0.344i$, showing that the baseline EE system exhibits stable cycles with a ten-year period.

Figure 3 presents a numerically derived phase diagram for the EE system. The "Epidemiology" locus represents combinations of x_t and in_t that produce time-invariant values for in_t from the pre-AIDS SIRS equations. The "Economics" locus represents combination of x_t and in_t that produce time-invariant values for x_t from the Euler equation (26). The intersection of the two loci determines the long-run equilibrium of the EE system, one that exhibits stable cycles for the given parameter values.

3.3.3 Contrasting Economic and Mathematical Epidemiological Models

The primary distinction between the mathematical and economic epidemiological models is the ability to react to changes in the risk of infection. If individuals ignore the health consequences of risky behavior (i.e., $h = 0$), thus choosing the maximum number of partners each period, the EE model collapses to the ME model with infection probability

$$p_t = 1 - (1 - \lambda_p in_t)^{\bar{x}}. \quad (32)$$

[10] If the eigenvalues have an imaginary part, then they come in complex conjugates $a \pm ci$ with period equal to (Hamilton (1994))

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

and persistence equal to

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

The only difference between the traditional ME model and the EE model with $h = 0$ 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 model collapses to the traditional SIRS model with a time-invariant infection parameter, λ_p.

The linearized dynamic SIRS system is

$$\begin{bmatrix} \widehat{in}_{t+1} \\ \hat{r}_{t+1} \end{bmatrix} = \begin{bmatrix} sp_{in} - (\mu + p) & -p \\ 1 & 1 - \gamma - \mu \end{bmatrix} \begin{bmatrix} \widehat{in}_t \\ \hat{r}_t \end{bmatrix}. \tag{33}$$

SIRS individuals will not alter the number of partners they choose, even in response to significant variation in disease prevalence. In the EE 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 (30) and (33). The two matrices differ by the $sp_x \kappa x/in$ term in the (1,1) position. This term captures the effect of changes in current infection rates on the probability of infection through the choice 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) and the behavioral response demonstrated in Geoffard and Philipson (1996). In the ME model, $\kappa = 0$ because susceptible individuals do not respond to changes in the risk of infection, resulting in transition dynamics given by (33). In the EE model, κ can take on a range of values depending on the biological parameters and individual preferences over x and h. Following the linearization of (26), this elasticity can be expressed as

$$\kappa = \frac{\partial x}{\partial in} \frac{in}{x} = \frac{\lambda_p inx}{(1 - \lambda_p in)} \left[\frac{1}{\ln(1-p)} + \frac{1}{1 + \ln(1-p)} \frac{1+\beta}{1+\beta p} \right]. \tag{34}$$

The partner elasticity κ is the sum of two parts. The first part relates to the probability of infection, while the second part relates to expected changes in lifetime utility. Together, they capture the influence of human responses on the dynamics of the system and may cause cycles to either be dampened or accentuated.

3.3.4 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.[11] As a result, the original increase in the infection rate is tempered, a phenomenon we refer to as *rational dynamic dampening*.

Panel A of Figure 4 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 h 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 both pivots clockwise due to $p_{x,t}$ and shifts down due to $(V_{t+1} - V_{t+1}^S)$.

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. The associated critical probability is $p_c = 1 - (1/e) \approx 0.63$.[12] This pivot is represented by the lighter, dashed line. The increase in prevalence also shifts the marginal cost curve. Together, the pivot and shift lead to a 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.

To compare the dynamics of the ME and EE models under dynamic dampening, each model is subjected to a one-time, five percentage point increase in prevalence. The top panels of Figure 5 show the dynamic responses of prevalence to the unanticipated perturbation. The difference in the persistence is clear when comparing the models. In the ME model, the system produces

[11] This is similar to the behavioral response cited in Geoffard and Philipson (1996) where the hazard rate (probability of infection) decreases as prevalence increases. The implication of $\kappa < 0$ is not that the probability of infection must fall with an increase in prevalence, rather that the change in probability of infection is smaller than if individuals did not alter their choice of partners.

[12] The critical probability, p_c, is found by taking the cross partial derivative of p with respect to x and in, setting the expression equal to zero, and solving for p (Kremer (1996)). The relevant equation is $\partial p_{in}/\partial x = p_{in}/x + p_{in} \ln(1-p)/x = 0$, which reduces to $\ln(1-p) = -1$ or $p_c = 1 - (1/e)$.

cyclical responses with period equal to 11.7 years and persistence equal to 0.86; the cyclical response continues well past the forcing shock. The EE cycles have a slightly reduced ten-year periodicity but are significantly dampened with persistence of 0.59. Cycles are nearly imperceptible ten years after the driving shock.

The differences in ME and EE cycles reflect the number of chosen partners, shown in the bottom panels of Figure 5. In the ME model, the number of sexual partners is fixed implying that syphilis cycles are exclusively due to biological dynamics.[13] However, when individuals are free to choose their number of partners and $\kappa < 0$, cycles are significantly dampened.[14] 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 prevalence. 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.

3.3.5 Rational Dynamic Resonance

The opposite occurs when $\kappa > 0$ and the responses of humans and disease biology are in sync. Individuals become fatalistic and increase the 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 these individuals, the increased prevalence causes a decrease in the marginal cost of infection, leading a rational individual to choose more partners. In the context of a dynamic SIRS model, this behavior amplifies cycles – a phenomenon we refer to as *rational dynamic resonance.* This is a formalization of Kremer's (1996) rational fatalism applied to a dynamic setting and an

[13] For purposes of comparison, we set x in the ME model equal to the endogenous solution for x from the EE model. This also implies that p and in will be equal across the two models.

[14] For the baseline parameter values, the elasticity of partner change with respect to prevalence is $\kappa = -0.56$. Furthermore, if we hold x fixed at its steady-state value, the probability of infection at the steady state is 0.19, increasing to 0.28 with the five percentage point increase in prevalence.

SIRS disease.

Panel B of Figure 4 illustrates the problem facing an individual with rational fatalism. The parameter values in Panels A and B are precisely the same, except for the lower value of h in Panel B. 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 6 contrasts the dynamic responses of the ME and EE models to a one-time, five percentage point increase in prevalence. The parameter values in Figures 5 and 6 are identical except for the health parameter h, which is decreased about 38% from 8.1 to 5.0. The new steady-state implies an increase in partners from 3.6 to 5.8, 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.[15]

Are fatalism and rational dynamic resonance simply a theoretical curiosity? The answer appears to be no. There is some 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. In the developed world, syphilis prevalence is likely too low to induce fatalism. However, that has not always been the case. In the late 15th century, a syphilis epidemic spread throughout Europe leading to millions of deaths (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

[15] For the baseline parameter values, the elasticity of partner change with respect to prevalence is $\kappa = 0.25$. Furthermore, if we hold x fixed at its steady-state value, the probability of infection at the steady state is 0.37, increasing to 0.48 with the five percentage point increase in prevalence.

to the idea of contracting an STD and take additional partners in response to increases in disease prevalence.

The final piece of evidence for rational dynamic resonance comes from the EE model and surveys of the sexual behavior for high-risk individuals. Using the baseline parameter values, the EE model predicts that the threshold number of partners required to induce rational fatalism and dynamic resonance is approximately five partners per year. Several studies indicate that the rate of partner change among high-risk individuals exceeds this number. 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.[16] 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, amplifying syphilis cycles.

3.3.6 Dampening, Resonance and Syphilis Cycles in the Post-AIDS Era

The beginning of the AIDS epidemic in the early 1980s drastically changed the risks of sexual activity. Sexually active individuals are now primarily concerned with AIDS, rather than syphilis or other STDs. Annual deaths due to AIDS in the U.S. jumped from 135 individuals in 1981 to a peak of over 48,000 in 1995 (CDC (2007)). The annual mortality rate for AIDS has since declined to under 15,000 due to the introduction and widespread availability of effective antiretroviral therapies (Boily et al. (2005)). The effect of AIDS on the dynamics of syphilis prevalence in the U.S. can be seen in Figure 1. Starting in 1990, the overall number of primary and secondary syphilis infections in the U.S. gradually fell over the decade and has been gradually rising since 2000. The nearly two decade U-shaped pattern in syphilis prevalence is significantly different than the ten-

[16] In their ME model, Grassly et al. (2005) implicitly chose the number of partners per year to be 14.5. (Breban et al. (2008)) found that cycles only occur if individuals take more than 9.8 new partners per year. We find a much lower threshold in the EE model due to the behavioral responses.

year oscillations marking the period between the introduction of penicillin and the beginning of the AIDS epidemic. To better understand the changing dynamics in syphilis prevalence, we explore the predictions of the EE model after the introduction of AIDS.

For brevity, we outline our main findings here, leaving the details of the steady state and transition dynamics for the post-AIDS EE model in the Appendix. In a setting with a relatively high health parameter so that individuals choose fewer sexual partners, a one-time increase in syphilis prevalence has little impact on the optimal number of partners or the dynamics of syphilis prevalence because AIDS, not syphilis, is the primary health concern. The primary impact of higher syphilis prevalence is to increase the risk of AIDS through the higher natural probability of infection ($\lambda_p^{A|S} > \lambda_p^A$). A one-time increase in AIDS prevalence leads to a greater initial reduction in the number of partners but monotonically returns to the steady state. The dynamics of syphilis infections are similar to those from the ME model. This similarity occurs because individuals are responding to a portfolio of risks, which is dominated by the lifetime consequences of contracting AIDS.

Repeating the exercise for a low health parameter causes individuals to choose a higher number of partners and fatalism sets in. With an increase in either syphilis or AIDS prevalence, individuals choose a higher number of partners, as the marginal probability of AIDS infection declines. The higher number of partners exacerbates the initial increase in syphilis or AIDS prevalence. This interplay between the marginal probability of contracting AIDS and the chosen number of partners continues over time, amplifying and stretching out syphilis cycles. The cycles in syphilis prevalence spillover into AIDS dynamics through the higher natural probability of AIDS infection. This is rational dynamic resonance in the post-AIDS era and it occurs in both SIRS and SI diseases.

We now turn our attention to the eradication of syphilis.

4 Syphilis Eradication

Encouraged by historically low prevalence 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 the 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 plausible that policy makers at the CDC could 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 and are 81% higher in 2007 than in 2000. Why did these plans fall short of their desired objectives? To answer this question, we investigate the dynamics of the EE system near eradication.

4.1 Pre-AIDS

Begin with the pre-AIDS eradication plan. To analyze the plan, we investigate the stability properties of the eradication equilibrium. The transition matrix around the eradication steady state simplifies to

$$\begin{bmatrix} \bar{x}\lambda_p - \mu & 0 \\ 1 & 1-\gamma-\mu \end{bmatrix} \tag{35}$$

with eigenvalues $(\bar{x}\lambda_p - \mu)$ and $(1-\gamma-\mu)$.[17] 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, $\bar{x}\lambda_p - \mu$, equal to one.[18] Any value greater than one will cause eradication to be unstable. The critical number of partners that makes eradication stable is

$$\bar{x}^* = \frac{1+\mu}{\lambda_p}. \tag{36}$$

[17] To derive the transition matrix around the eradication steady state, evaluate (33) at the eradication steady state. The Euler equation for x is not relevant because when the system is near the eradication boundary, individuals will optimally choose $x_t = \bar{x}$ for all t.

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

Using the baseline parameters from Table 1 ($\mu = 0.05$ and $\lambda_p = 0.60$), the stability threshold implies that individuals must average less than 1.75 partners per year for eradication to be locally stable. Even two partners per person will cause eradication to become unstable and the EE system to gravitate toward an endemic equilibrium.[19]

Alternatively, the stability threshold (36) for eradication can be interpreted in terms of the basic reproduction number $R_0 = p[(1+\mu)in]^{-1}$, which using L'Hôpital's rule reduces to $R_0 = \lambda_p \bar{x}/(1+\mu)$. 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 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 ($1 + \mu$).

4.2 Post-AIDS

Now consider the stability of eradication after the onset of AIDS. Here, the chance of syphilis eradication is improved because susceptible individuals will take fewer partners due to the fear of AIDS. To examine the stability of syphilis eradication, we calculate the basic reproductive number for syphilis around the eradication steady state. The basic reproductive number is given by

$$R_0 = \frac{s\lambda_p^S x + in^A \lambda_p^{S|A} x^A}{1+\mu}. \tag{37}$$

Note the similarities of R_0 to the pre-AIDS period. The first term in the numerator measures the rate at which individuals from the susceptible pool are becoming infected. This term is weighted by s, the proportion of the susceptible syphilis population without AIDS, and involves x rather than $\bar{x}$ because susceptible individuals will not take the maximum number of partners due to the risk of AIDS. The second term involves the proportion of individuals that are susceptible to syphilis but have AIDS, in^A. These individuals will choose more partners because they are already infected

[19] The eradication stability frontier is independent of h implying that changes in the relative concern for health do not impact the local stability of eradication. This occurs because h affects the transition dynamics exclusively through its impact on in (see matrix system (30)). When stability is evaluated at the eradication steady state (implying that $in = 0$), h has no role in the transition dynamics and local stability.

with AIDS and the health risks of syphilis are relatively low. As in the pre-AIDS period, the denominator $(1 + \mu)$ captures the rate at which individuals are leaving the infected pool, either through treatment (100%) or death (μ).

One way to contrast syphilis eradication pre- and post-AIDS is to calculate the necessary degree of altruism (maximum number of allowable partners) for successful eradication. Table 2 shows the degree of altruism needed for syphilis eradication to be locally stable.

Table 2. Altruism and Stability of Syphilis Eradication

			Number of Partners	
Period	Type	Fraction of Population	Self-Interested Choice	Required Altruism
Before AIDS	s	100%	$\bar{x} = 10$	1.75
After AIDS	s	85%	1.87	1.87
After AIDS	in^A	15%	$\bar{x} = 10$	0.95

Before the AIDS epidemic, the EE model shows that a high degree of altruism is necessary for eradication to be stable and keep the system from gravitating toward an endemic equilibrium: the susceptible population must reduce their number of partners by 83% (from $\bar{x} = 10$ to less than two partners per year). For the sexually active population under consideration, this is an extreme degree of altruism (Andrus et al. (1990)). After the AIDS epidemic, individuals are primarily concerned with the risk of contracting AIDS, not syphilis. Those susceptible to AIDS will voluntarily take fewer partners, $x = 1.87$ for our calibration, not out of concern for the general population but rather out of self-interest. The remaining portion of the population (those with AIDS) will need to reduce their number of partners to less than one partner per year ($x^A \leq 0.95$) for syphilis eradication to be successful. Overall, this is a much smaller degree of required altruism because the majority of the population voluntarily reduced the number of partners due to the risk

of AIDS. Yet, those with AIDS are still required to take no more than one partner per year and display a high level of altruism for syphilis to be eradicated.

4.3 Policy Evaluation

Why have the eradication plans failed to reach their objectives? The answer lies not with the biology of the disease but rather with economic principles. In the late 1950s, when U.S. syphilis 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 is that for reasonable values of the transmission and population growth parameters, anything more than two partners per year will make eradication infeasible. This number is well below commonly accepted estimates of partner frequency per year for those who have an elevated risk of syphilis (Andrus et al. (1990)). The goal of eradicating syphilis in the late 1950s predictably failed.

After the introduction of AIDS, syphilis was no longer a primary concern of sexually active individuals. Prior to 1999, syphilis rates in the U.S. were dropping because sexually active individuals were reacting to the risk of AIDS by using protection or taking fewer partners. However, as drug therapies were discovered that lengthened and improved the quality of life for those infected with AIDS, the incentive to protect or take fewer partners diminished. Individuals began to take more risk and offset the efforts of the 1999 syphilis eradication campaign (Boily et al. (2005)).

The primary lesson here is that when infection rates are near eradication and the health risks of other sexually transmitted diseases are limited – as was the case in the U.S. in 1956 and 2000 – is precisely the time when individuals will choose the highest number of partners and take the most risks. As a result, the disease remains within the general population and continues to fluctuate around its endemic long-run rate.

5 Conclusion

Our research has both methodological and policy significance. Methodologically, we develop an integrated economic-ecological model of infectious disease dynamics in the spirit of Philipson (1995), Gersovitz and Hammer (2004), Geoffard and Philipson (1996) and Kremer (1996). The model is unique in focusing on an SIRS disease – syphilis – and developing a joint model with AIDS. To the best of our knowledge, this is the first attempt to model two sexually transmitted diseases within a single optimization framework. In the process, we extend Kremer's (1996) fatalism result to a dynamic setting and demonstrate how human responses may either dampen or exacerbate the magnitude and duration of infectious disease cycles.

The implications from the model can also inform policy. A key part of designing and implementing effective public health policy for infectious diseases is understanding the role of human behavior. For syphilis, Grassly et al. (2005) argue convincingly that 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. In contrast, our analysis shows that behavioral responses are central to the nature of syphilis dynamics. This gives credence to public health policies that aim to change sexual practices in an attempt to control the disease.

Perhaps the most striking implication of the integrated EE framework is that eradication is nearly impossible. When the disease is near eradication, this is precisely the time when a rational susceptible individual will choose to take more partners because the chance of matching up with an infected individual is very low. Prior to the AIDS epidemic, our model predicts that the sexually active population under consideration would need to scale back their number of partners to less than two per year – an extreme degree of altruism for the relevant population – for eradication to be successful. After the introduction of AIDS, syphilis eradication is more plausible as susceptible individuals will rationally reduce their number of partners in response to AIDS risk. However, this window of opportunity to eradicate syphilis may have closed. The discovery of new drug therapies have drastically improved the quality of life for those infected with AIDS but has the unintended

consequence of making syphilis eradication much more difficult. As a result, public health officials may be better served by directing their efforts away from eradication and toward finding the best mix of education, prevention and treatment policies to reach a more desirable endemic equilibrium.

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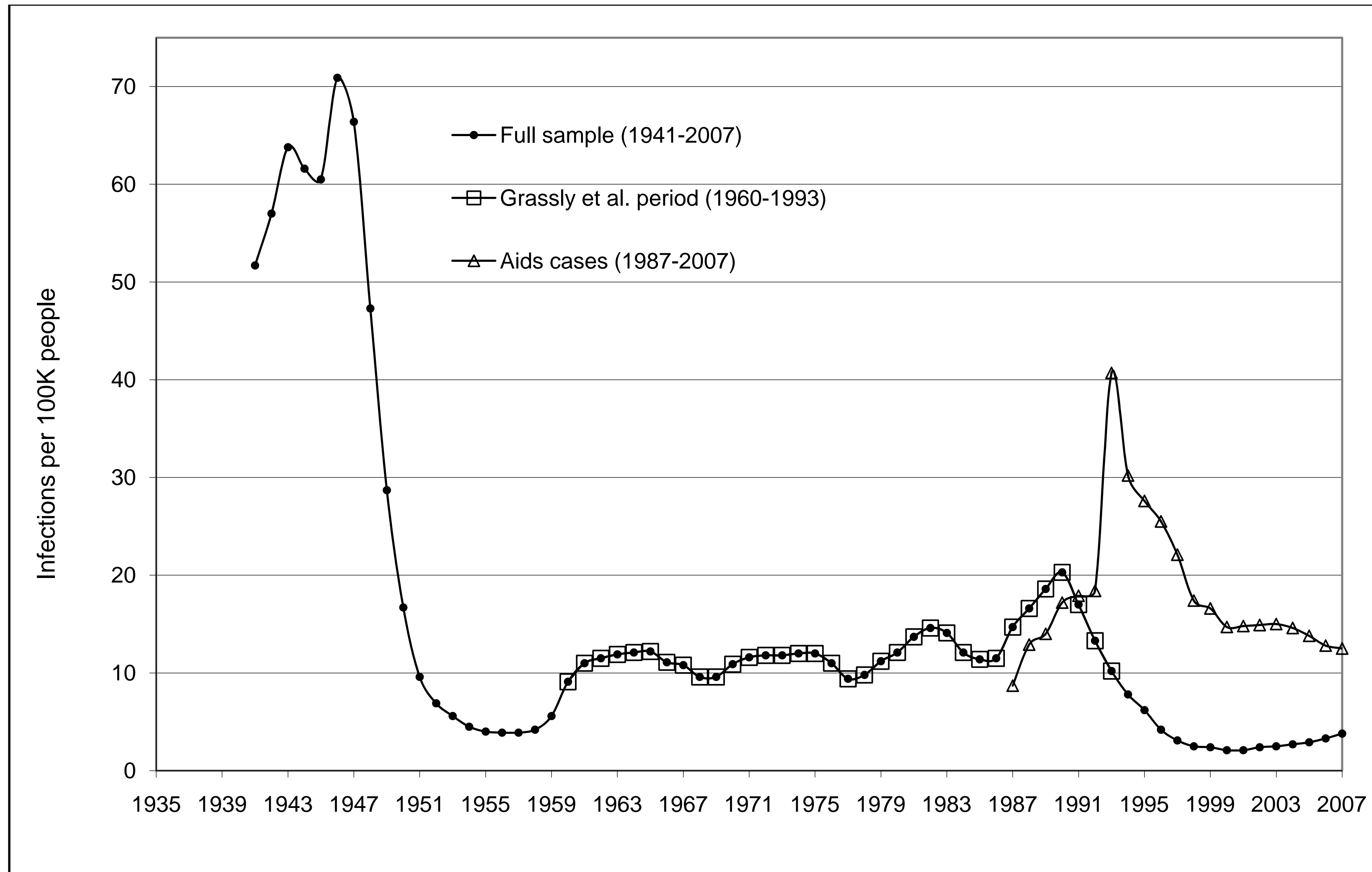
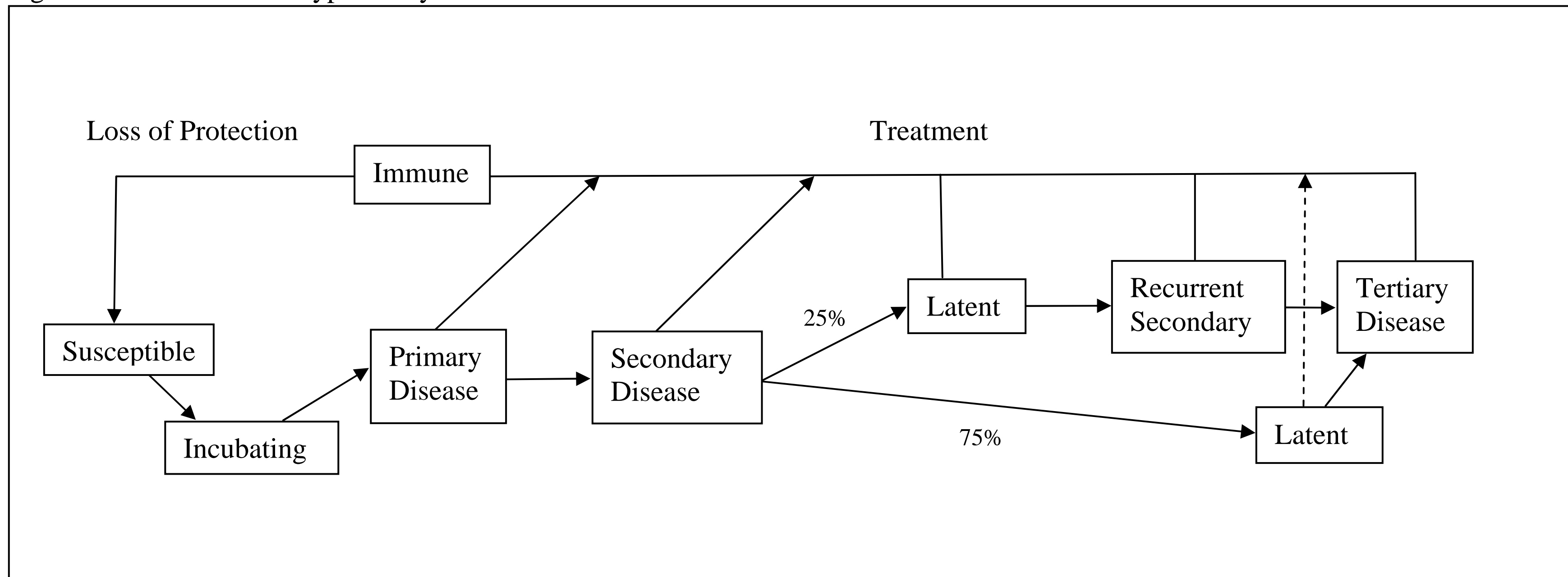
Figure 1. U.S. Cases of (Primary & Secondary) Syphilis and AIDS



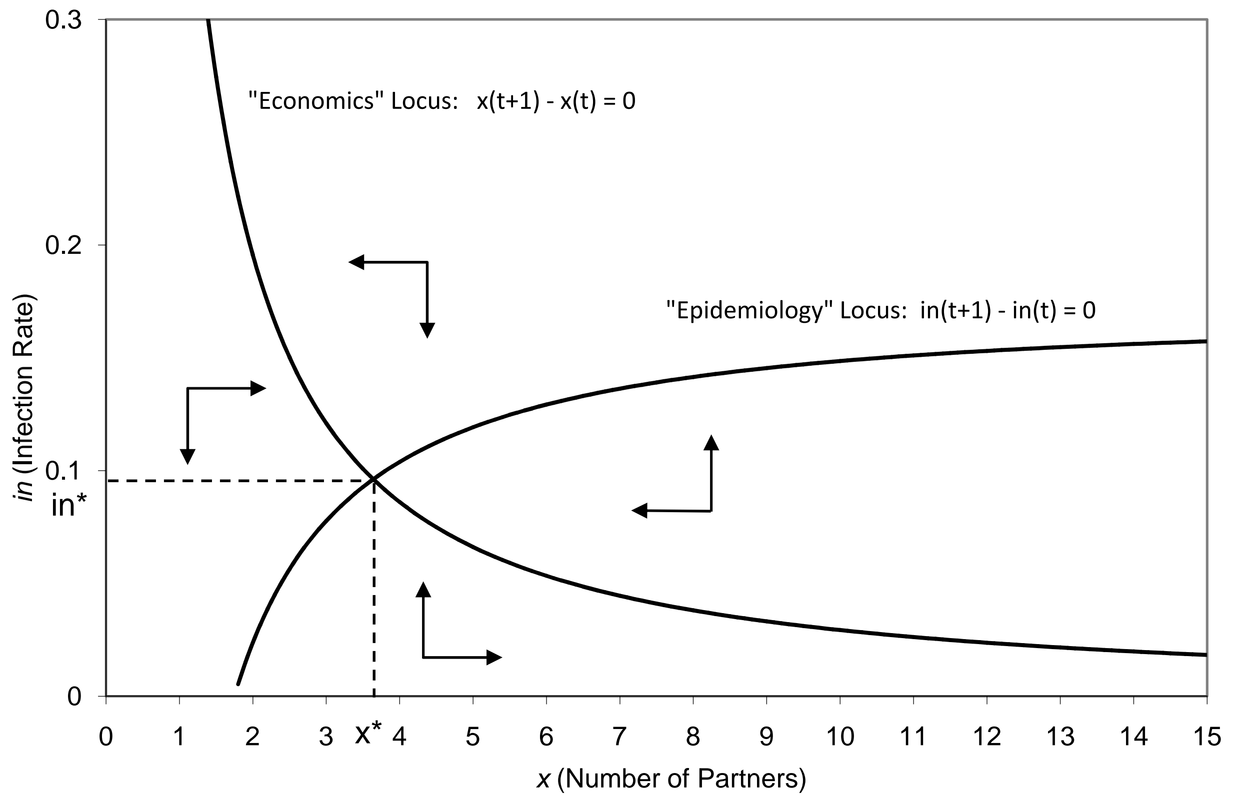
Source: Center for Disease Control and Prevention (www.cdc.gov/std/).

Figure 2. Flow Chart for Syphilis Dynamics



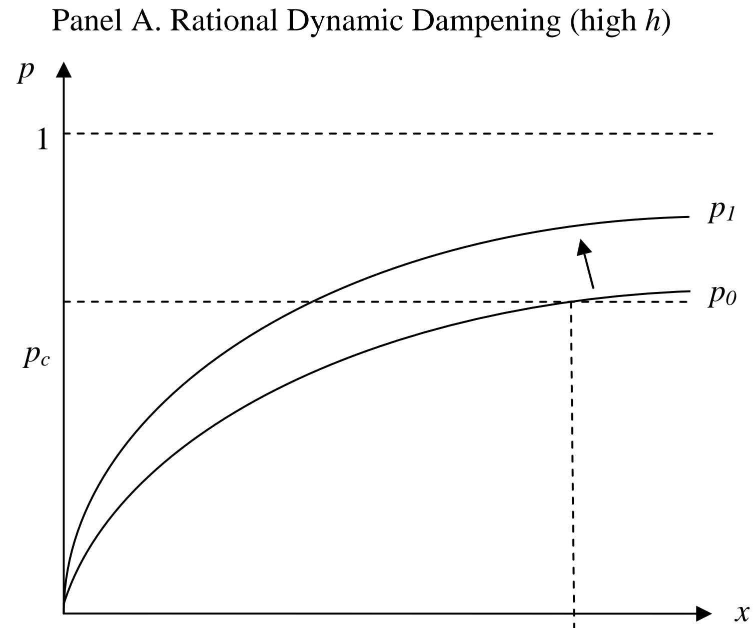
Reproduced from Garnett et al. (1997).

Figure 3. Phase Diagram for the EE System



Notes. The parameter values are set at $\beta = 0.96$, $\gamma = 0.2$, $\lambda_a = 0.023$, $a = 40$, $\mu = 0.05$, $h = 8.1$ and $\bar{x} = 10$. The steady state of the EE model, (in^*, x^*), is found at the intersection of the time invariant loci for *in* and *x*.

Figure 4. Individual Optimal Choice and the Probability of Infection: Exogenous Increase in Prevalence



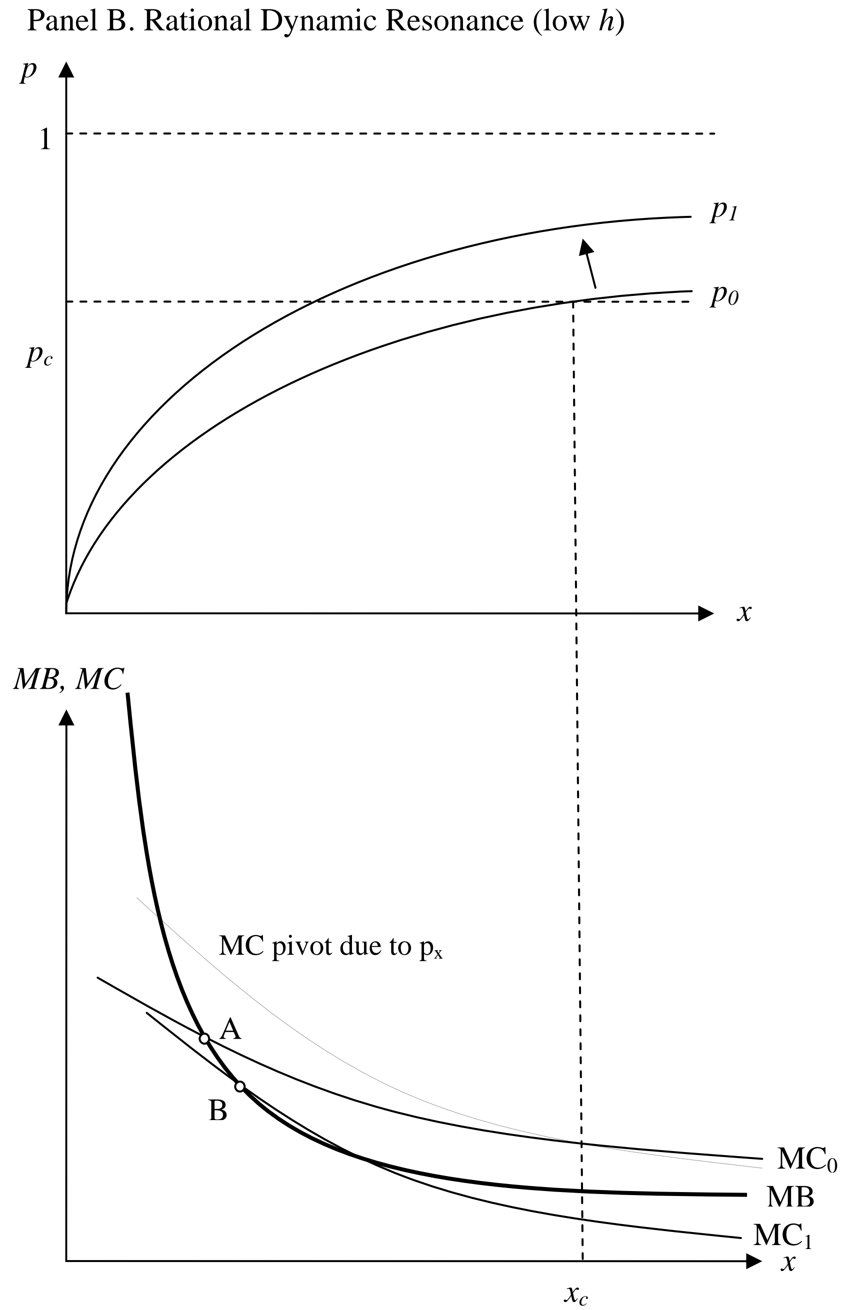
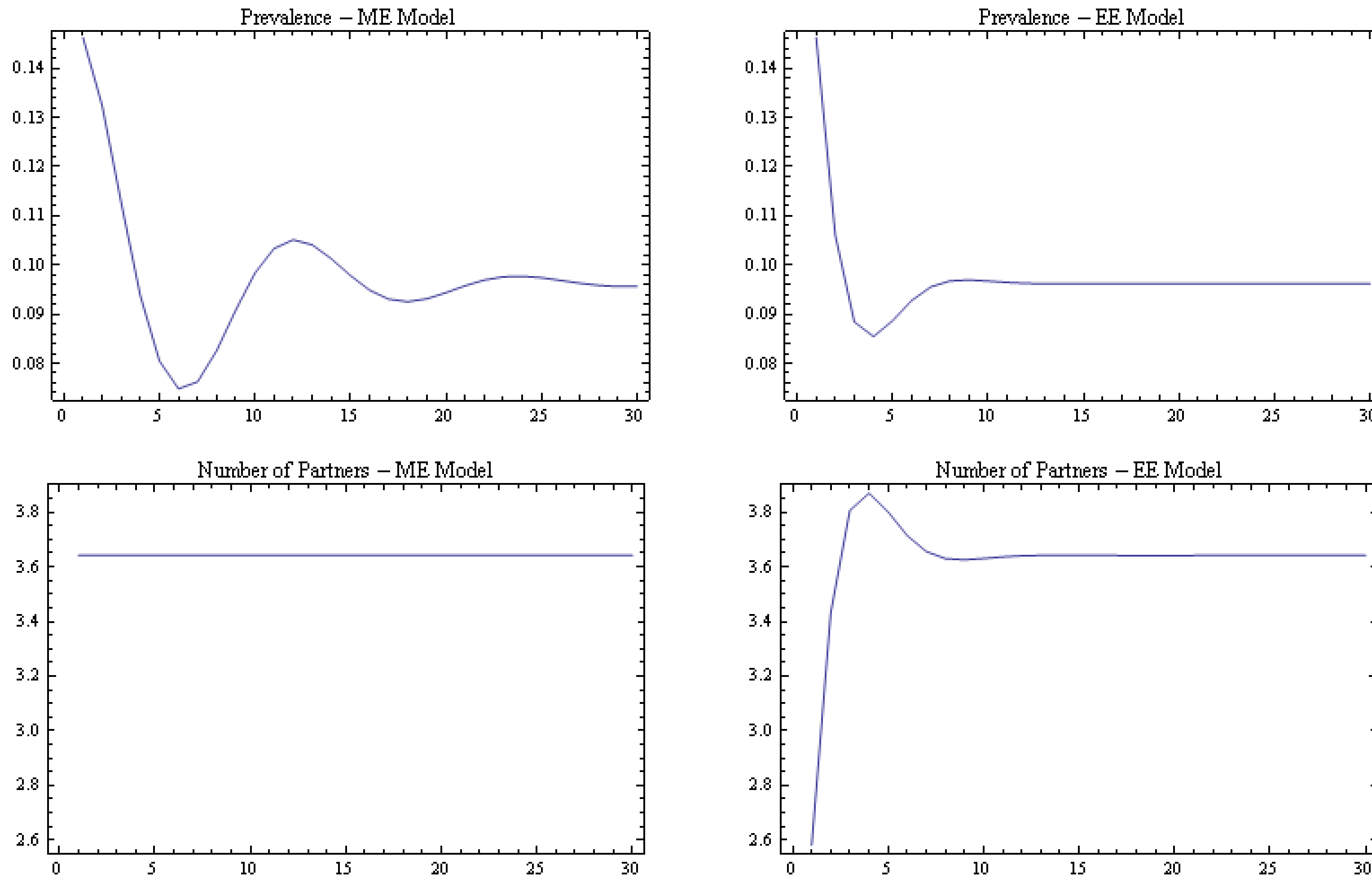
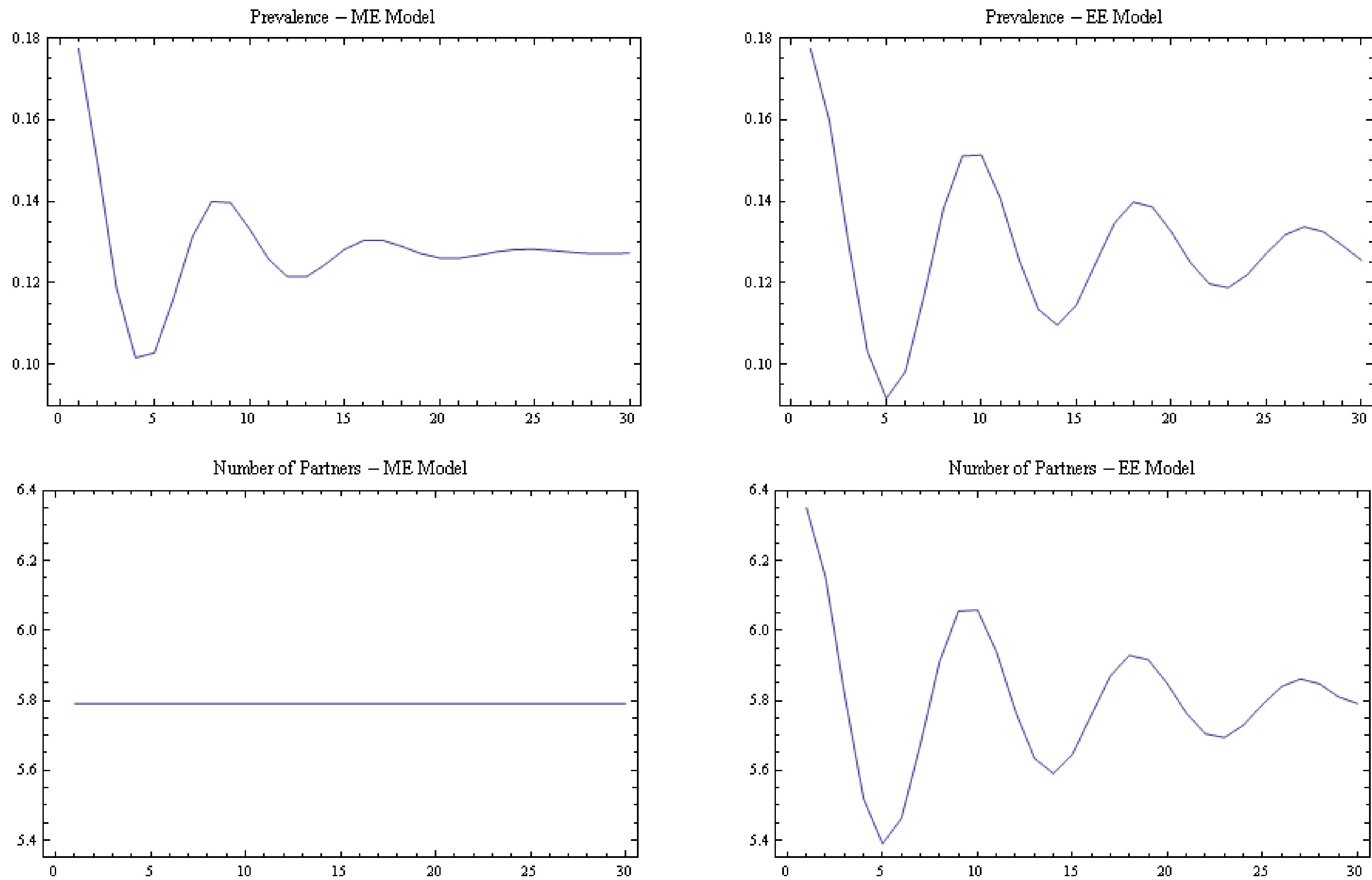


Figure 5. Impulse Response Functions for the ME and EE Systems – Rational Dynamic Dampening



Notes. The fundamental parameters in the EE system are set at $\beta = 0.96$, $\mu = 0.05$, $\gamma = 0.2$, $h = 8.1$, $\lambda_a = 0.023$, $a = 40$ and $\bar{x} = 10$. For comparison purposes, we set the steady-state number of partners (x) in the ME model equal to the endogenously solved for number of partners in the EE model. As a result, the steady-state prevalence is also equal in the ME and EE models.

Figure 6. Impulse Response Functions for the ME and EE Systems – Rational Dynamic Resonance



Notes. The fundamental parameters in the EE system are set at $\beta = 0.96$, $\mu = 0.05$, $\gamma = 0.2$, $h = 5.4$, $\lambda_a = 0.023$, $a = 40$, and $\bar{x} = 10$. For comparison purposes, we set the steady-state number of partners (x) in the ME model equal to the endogenously solved for number of partners in the EE model. As a result, the steady-state prevalence is also equal in the ME and EE models.

Appendix

May 2010

To accompany the manuscript "Syphilis Cycles"

David Aadland, David Finnoff and Kevin X.D. Huang

In this appendix, we present the technical details of the integrated economic-epidemiological (EE) model in the pre- and post-AIDS eras. We start by presenting the joint syphilis-AIDS model after the introduction of AIDS and then present the details of the model prior to the introduction of AIDS.

1 POST-AIDS ERA

1.1 SYPHILIS-AIDS EPIDEMIOLOGICAL MODEL

The joint syphilis-AIDS (SIRS-SI) population model contains six mutually exclusive categories: susceptible to both diseases (s), infected with syphilis only (in^S), infected with AIDS only (in^A), infected with syphilis and AIDS (in^{SA}), immune to syphilis (r), and immune to syphilis while infected with AIDS (r^A). We start by presenting the transition matrix between these categories in Table A1:

Table A1. Transition Matrix for Disease Categories

	s_{t+1}	in^S_{t+1}	in^A_{t+1}	in^{SA}_{t+1}	r_{t+1}	r^A_{t+1}
s_t	$(1-p^S_t)(1-p^A_t)$	$p^S_t(1-p^A_t)$	$p^A_t(1-p^S_t)$	$p^S_t p^A_t$	0	0
in^S_t	0	0	0	0	$1-p^{A\|S}_t$	$p^{A\|S}_t$
in^A_t	0	0	$1-p^{S\|A}_t$	$p^{S\|A}_t$	0	0
in^{SA}_t	0	0	0	0	0	1
r_t	$\gamma(1-p^{A\|S}_t)$	0	$\gamma p^{A\|S}_t$	0	$(1-p^{A\|S}_t)(1-\gamma)$	$(1-\gamma)p^{A\|S}_t$
r^A_t	0	0	γ	0	0	$1-\gamma$

Using the transition probabilities and a 100% syphilis treatment rate, the equations of motion for the disease categories are

$$
\begin{aligned}
s_{t+1} &= \mu + [(1-p^S_t)(1-p^A_t) - \mu]s_t + \gamma(1-p^{A|S}_t)r_t \\
in^S_{t+1} &= -\mu in^S_t + p^S_t(1-p^A_t)s_t \\
in^A_{t+1} &= (1-\mu-p^{S|A}_t)in^A_t + p^A_t(1-p^S_t)s_t + \gamma p^{A|S}_t r_t + \gamma r^A_t \\
in^{SA}_{t+1} &= -\mu in^{SA}_t + p^{S|A}_t in^A_t + p^S_t p^A_t s_t \\
r_{t+1} &= [(1-p^{A|S}_t)(1-\gamma) - \mu]r_t + (1-p^{A|S}_t)in^S_t \\
r^A_{t+1} &= (1-\mu-\gamma)r^A_t + (1-\gamma)p^{A|S}_t r_t + p^{A|S}_t in^S_t + in^{SA}_t,
\end{aligned}
$$

where the marginal and conditional probabilities of contracting syphilis or AIDS are

$$
\begin{aligned}
p_t^S &= \Pr(\text{contract syphilis}) = 1 - [1 - \lambda_p^S(in_t^S + in_t^{SA})]^{x_t} \\
p_t^A &= \Pr(\text{contract AIDS}) = 1 - [1 - \lambda_p^A(in_t^A + in_t^{SA} + r_t^A)]^{x_t} \\
p_t^{S|A} &= \Pr(\text{contract syphilis} \mid \text{infected with AIDS}) = 1 - [1 - \lambda_p^{S|A}(in_t^S + in_t^{SA})]^{x_t^A} \\
p_t^{A|S} &= \Pr(\text{contract AIDS} \mid \text{infected with syphilis}) = 1 - [1 - \lambda_p^{A|S}(in_t^A + in_t^{SA} + r_t^A)]^{x_t^S}.
\end{aligned}
$$

1.2 VALUE FUNCTIONS

The four value functions apply to individuals: (1) susceptible to both diseases, $V_t = V(z_t)$; (2) infected with syphilis only, $V_t^S = V^S(z_t)$; (3) infected with AIDS only, $V_t^A = V^A(z_t)$; and (4) infected with syphilis and AIDS, $V_t^{SA} = V^{SA}(z_t)$, where $z_t \equiv (s_t, in_t^S, in_t^A, in_t^{SA}, r_t, r_t^A)'$ is the vector of states. There are no value functions for those recovered (and immune) to syphilis because we assume the recovered stage cannot be observed by individuals. The value functions are

$$
\begin{aligned}
V_t &= \max_{x_t}\{u(x_t, h) + \beta[p_t^S(1-p_t^A)V_{t+1}^S + p_t^A(1-p_t^S)V_{t+1}^A + p_t^S p_t^A V_{t+1}^{SA} + (1-p_t^S)(1-p_t^A)V_{t+1}]\} & (1)\\
V_t^S &= \max_{x_t^S}\{u(x_t^S, h^S) + \beta[p_t^{A|S}V_{t+1}^A + (1-p_t^{A|S})V_{t+1}]\} & (2)\\
V_t^A &= \max_{x_t^A}\{u(x_t^A, h^A) + \beta[p_t^{S|A}V_{t+1}^{SA} + (1-p_t^{S|A})V_{t+1}^A]\} & (3)\\
V_t^{SA} &= u(\bar{x}, h^{SA}) + \beta V_{t+1}^A. & (4)
\end{aligned}
$$

1.3 EULER EQUATIONS

The necessary first-order conditions for s, in^S, and in^A individuals are:

$$
\begin{aligned}
x_t^{-1} &= \beta p_{x,t}^S[(1-p_t^A)V_{t+1} - (1-p_t^A)V_{t+1}^S + p_t^A V_{t+1}^A - p_t^A V_{t+1}^{SA}] + \\
&\quad \beta p_{x,t}^A[(1-p_t^S)V_{t+1} - (1-p_t^S)V_{t+1}^A + p_t^S V_{t+1}^S - p_t^S V_{t+1}^{SA}] & (5)\\
(x_t^S)^{-1} &= \beta p_{x,t}^{A|S}(V_{t+1}^S - V_{t+1}^A) & (6)\\
(x_t^A)^{-1} &= \beta p_{x,t}^{S|A}(V_{t+1}^A - V_{t+1}^{SA}), & (7)
\end{aligned}
$$

where the x subscript on the probabilities refers to the partial derivatives with respect to the appropriate x. The left side of the Euler equations is the marginal utility or benefit (MB) and the right side is the marginal disutility or cost (MC) associated with the chosen number of partners. Using (1) through (4) to substitute out the optimized value functions, the

Euler equations become

$$\frac{1}{p_{x,t}^{S|A} x_t^A} + \frac{1}{p_{x,t}^{A|S} x_t^S} = \beta E\left(u_{t+1} - u_{t+1}^{SA} + p_{t+1}^S (1 - p_{t+1}^A) \delta_{1,t+1} \delta_{2,t+1} - \frac{p_{t+1}^S p_{t+1}^A}{x_{t+1}^A p_{x,t+1}^{S|A}} + \frac{1 - p_{t+1}^A}{x_{t+1}^S p_{x,t+1}^{A|S}} \right) \tag{8}$$

$$\frac{1}{p_{x,t}^{S|A} x_t^A} + \frac{1}{p_{x,t}^{A|S} x_t^S} = -\delta_{1,t}\delta_{2,t} + \beta E\left(u_{t+1}^S - u_{t+1}^{SA} + \frac{1 - p_{t+1}^{A|S}}{x_{t+1}^S p_{x,t+1}^{A|S}} \right) \tag{9}$$

$$\frac{1}{p_{x,t}^{S|A} x_t^A} = \beta E\left(u_{t+1}^A - u_{t+1}^{SA} - \frac{p_{t+1}^{S|A}}{x_{t+1}^A p_{x,t+1}^{S|A}} \right), \tag{10}$$

where

$$\delta_{1,t} = \left(\frac{p_{x,t}^S p_t^A + p_{x,t}^A p_t^S}{x_t^A p_{x,t}^{S|A}} + \frac{p_{x,t}^A}{x_t^S p_{x,t}^{A|S}} - \frac{1}{x_t} \right) \text{ and}$$

$$\delta_{2,t} = \frac{1}{p_{x,t}^S (1 - p_t^A) - p_t^S p_{x,t}^A}.$$

The second-order conditions for an optimal program require

$$\frac{\partial MB}{\partial x} - \frac{\partial MC}{\partial x} < 0,$$

for equations (5), (6) and (7). Since the marginal benefits decline with x, an optimal program requires an upward sloping marginal cost curve (i.e., $\frac{\partial MC}{\partial x} > 0$), or if it slopes down, it must be locally flatter than the MB curve (i.e., $\left|\frac{\partial MB}{\partial x}\right| > \left|\frac{\partial MC}{\partial x}\right|$).

1.4 EXPECTATIONS

We consider two types of expectations by individuals: naïve and rational. Under naïve expectations, the expectation of all future variables is set equal to the associated current value. Under rational expectations, E is the mathematical expectations operator conditional on all information dated time t and earlier. With rational expectations, individuals have complete information on the laws of motion for the aggregate disease variables and the optimal choices of other individuals.

1.5 STEADY STATE

The endemic steady-state solves for nine variables, $\{s, in^S, in^A, in^{SA}, r, r^A, x, x^S, x^A\}$, from the following nine equations:

$$
\begin{aligned}
s &= \frac{\mu + \gamma(1 - p^{A|S})r}{\mu + p^S + p^A - p^S p^A} \\
in^S &= \frac{p^S(1 - p^A)s}{1 + \mu} \\
in^A &= \frac{p^A(1 - p^S)s + \gamma(p^{A|S}r + r^A)}{\mu + p^{S|A}} \\
in^{SA} &= \frac{p^{S|A} in^A + p^S p^A s}{1 + \mu} \\
r &= \frac{(1 - p^{A|S}) in^S}{\mu + \gamma(1 - p^{A|S}) + p^{A|S}} \\
r^A &= \frac{(1 - \gamma)p^{A|S}r + p^{A|S} in^S + in^{SA}}{\mu + \gamma} \\
x^{-1} &= \beta p_x^S[(1 - p^A)V - (1 - p_t^A)V^S + p^A V^A - p^A V^{SA}] + \\
&\quad \beta p_x^A[(1 - p^S)V - (1 - p^S)V^A + p^S V^S - p^S V^{SA}] \\
(x^S)^{-1} &= \beta p_x^{A|S}(V^S - V^A) \\
(x^A)^{-1} &= \beta p_x^{S|A}(V^A - V^{SA}).
\end{aligned}
$$

1.6 LINEARIZATION

We start by linearizing the SIRS and SI epidemiological equations around the endemic steady state. Variables with hats refer to deviations from the steady state (e.g., $\hat{s}_t = s_t - s$):

$$
\begin{aligned}
\hat{s}_{t+1} &= [-\mu + (1 - p^S)(1 - p^A)]\hat{s}_t + \gamma(1 - p^{A|S})\hat{r}_t - s(1 - p^A)\hat{p}_t^S - s(1 - p^S)\hat{p}_t^A - \gamma r \hat{p}_t^{A|S} \\
\widehat{in}_{t+1}^S &= -\mu \widehat{in}_t^S + p^S(1 - p^A)\hat{s}_t + (1 - p^A)s\hat{p}_t^S - p^S s\hat{p}_t^A \\
\widehat{in}_{t+1}^A &= (1 - \mu - p^{S|A})\widehat{in}_t^A - in^A \hat{p}_t^{S|A} + p^A(1 - p^S)\hat{s}_t - sp^A\hat{p}_t^S + s(1 - p^S)\hat{p}_t^A + \gamma p^{A|S}\hat{r}_t + \gamma r\hat{p}_t^{A|S} + \gamma \hat{r}_t^A \\
\widehat{in}_{t+1}^{SA} &= -\mu \widehat{in}_t^{SA} + p^{SA}\widehat{in}_t^A + in^A\hat{p}_t^{S|A} + p^S p^A \hat{s}_t + p^S s\hat{p}_t^A + p^A s\hat{p}_t^S \\
\hat{r}_{t+1} &= [1 - \mu - \gamma(1 - p^{A|S}) - p^{A|S}]\hat{r}_t - [r(1 - \gamma) + in^S]\hat{p}_t^{A|S} + (1 - p^{A|S})\widehat{in}_t^S \\
\hat{r}_{t+1}^A &= (1 - \mu - \gamma)\hat{r}_t^A + (1 - \gamma)p^{A|S}\hat{r}_t + [in^S + (1 - \gamma)r]\hat{p}_t^{A|S} + p^{A|S}\widehat{in}_t^S + \widehat{in}_t^{SA}.
\end{aligned}
$$

The linearized equations for the probabilities (and the derivatives of the probabilities with respect to partners) are given by:

$$\begin{aligned}
\hat{p}_t^S &= p_{in}^S \widehat{in}_t^S + p_{in}^S \widehat{in}_t^{SA} + p_x^S \hat{x}_t \\
\hat{p}_t^A &= p_{in}^A \widehat{in}_t^A + p_{in}^A \widehat{in}_t^{SA} + p_{in}^A \hat{r}_t^A + p_x^A \hat{x}_t \\
\hat{p}_t^{S|A} &= p_{in}^{S|A} \widehat{in}_t^S + p_{in}^{S|A} \widehat{in}_t^{SA} + p_x^{S|A} \hat{x}_t^A \\
\hat{p}_t^{A|S} &= p_{in}^{A|S} \widehat{in}_t^A + p_{in}^{A|S} \widehat{in}_t^{SA} + p_{in}^{A|S} \hat{r}_t^A + p_x^{A|S} \hat{x}_t^S \\
\hat{p}_{x,t}^S &= [(1 + \ln[1 - p^S])/x]\hat{p}_t^S - (p_x^S/x)\hat{x}_t \\
\hat{p}_{x,t}^A &= [(1 + \ln[1 - p^A])/x]\hat{p}_t^A - (p_x^A/x)\hat{x}_t \\
\hat{p}_{x,t}^{S|A} &= [(1 + \ln[1 - p^{S|A}])/x^A]\hat{p}_t^{S|A} - (p_x^{S|A}/x^A)\hat{x}_t^A \\
\hat{p}_{x,t}^{A|S} &= [(1 + \ln[1 - p^{A|S}])/x^S]\hat{p}_t^{A|S} - (p_x^{A|S}/x^S)\hat{x}_t^S
\end{aligned}$$

where

$$\begin{aligned}
p_{in}^S &= p_{in^S}^S = p_{in^{SA}}^S = x\lambda_p^S(1 - \lambda_p^S(in^S + in^{SA}))^{x-1} \\
p_{in}^A &= p_{in^A}^A = p_{in^{SA}}^A = p_{r^A}^A = x\lambda_p^A(1 - \lambda_p^A(in^A + in^{SA} + r^A))^{x-1} \\
p_{in}^{S|A} &= p_{in^S}^{S|A} = p_{in^{SA}}^{S|A} = x^A\lambda_p^{S|A}(1 - \lambda_p^{S|A}(in^S + in^{SA}))^{x^A-1} \\
p_{in}^{A|S} &= p_{in^A}^{A|S} = p_{in^{SA}}^{A|S} = p_{r^A}^{A|S} = x^S\lambda_p^{A|S}(1 - \lambda_p^{A|S}(in^A + in^{SA} + r^A))^{x^S-1} \\
p_x^S &= -\ln[1 - p^S](1 - p^S)/x \\
p_x^A &= -\ln[1 - p^A](1 - p^A)/x \\
p_x^{S|A} &= -\ln[1 - p^{S|A}](1 - p^{S|A})/x^A \\
p_x^{A|S} &= -\ln[1 - p^{A|S}](1 - p^{A|S})/x^S.
\end{aligned}$$

Summarizing, the linearized EE system is

SIRS/SI System

$$\begin{aligned}
\hat{s}_{t+1} &= a_1\hat{s}_t + a_2\hat{r}_t + a_3\hat{p}_t^S + a_4\hat{p}_t^A + a_5\hat{p}_t^{A|S} \\
\widehat{in}_{t+1}^S &= a_6\widehat{in}_t^S + a_7\hat{s}_t + a_8\hat{p}_t^S + a_9\hat{p}_t^A \\
\widehat{in}_{t+1}^A &= a_{10}\widehat{in}_t^A + a_{11}\hat{p}_t^{S|A} + a_{12}\hat{s}_t + a_{13}\hat{p}_t^S + a_{14}\hat{p}_t^A + a_{15}\hat{r}_t + a_{16}\hat{p}_t^{A|S} + a_{17}\hat{r}_t^A \\
\widehat{in}_{t+1}^{SA} &= a_{18}\widehat{in}_t^{SA} + a_{19}\widehat{in}_t^A + a_{20}\hat{p}_t^{S|A} + a_{21}\hat{s}_t + a_{22}\hat{p}_t^A + a_{23}\hat{p}_t^S \\
\hat{r}_{t+1} &= a_{24}\hat{r}_t + a_{25}\hat{p}_t^{A|S} + a_{26}\widehat{in}_t^S \\
\hat{r}_{t+1}^A &= a_{27}\hat{r}_t^A + a_{28}\hat{r}_t + a_{29}\hat{p}_t^{A|S} + p^{A|S}\widehat{in}_t^S + \widehat{in}_t^{SA}
\end{aligned}$$

Probabilities

$$\begin{aligned}
\hat{p}_t^S &= p_{in}^S\widehat{in}_t^S + p_{in}^S\widehat{in}_t^{SA} + p_x^S\hat{x}_t \\
\hat{p}_t^A &= p_{in}^A\widehat{in}_t^A + p_{in}^A\widehat{in}_t^{SA} + p_{in}^A\hat{r}_t^A + p_x^A\hat{x}_t \\
\hat{p}_t^{S|A} &= p_{in}^{S|A}\widehat{in}_t^S + p_{in}^{S|A}\widehat{in}_t^{SA} + p_x^{S|A}\hat{x}_t^A \\
\hat{p}_t^{A|S} &= p_{in}^{A|S}\widehat{in}_t^A + p_{in}^{A|S}\widehat{in}_t^{SA} + p_{in}^{A|S}\hat{r}_t^A + p_x^{A|S}\hat{x}_t^S \\
\hat{p}_{x,t}^S &= a_{30}\hat{p}_t^S + a_{31}\hat{x}_t \\
\hat{p}_{x,t}^A &= a_{32}\hat{p}_t^A + a_{33}\hat{x}_t \\
\hat{p}_{x,t}^{S|A} &= a_{34}\hat{p}_t^{S|A} + a_{35}\hat{x}_t^A \\
\hat{p}_{x,t}^{A|S} &= a_{36}\hat{p}_t^{A|S} + a_{37}\hat{x}_t^S
\end{aligned}$$

Euler Equations

$$\begin{aligned}
a_{38}\hat{p}_{x,t}^{S|A} + a_{39}\hat{x}_t^A + a_{40}\hat{p}_{x,t}^{A|S} + a_{41}\hat{x}_t^S &= a_{42}E\hat{x}_{t+1} + a_{43}E\hat{p}_{t+1}^S + a_{44}E\hat{p}_{t+1}^A + a_{45}E\hat{p}_{x,t+1}^S + \\
&\quad a_{46}E\hat{p}_{x,t+1}^A + a_{47}E\hat{x}_{t+1}^A + a_{48}E\hat{p}_{x,t+1}^{S|A} + a_{49}E\hat{x}_{t+1}^S + a_{50}E\hat{p}_{x,t+1}^{A|S} \\
a_{51}\hat{p}_{x,t}^{S|A} + a_{52}\hat{x}_t^A + a_{53}\hat{p}_{x,t}^{A|S} + a_{54}\hat{x}_t^S &= -a_{55}\hat{p}_{x,t}^S - a_{56}\hat{p}_t^A - a_{57}\hat{p}_t^S - a_{58}\hat{p}_{x,t}^A - a_{59}\hat{x}_t + \\
&\quad a_{60}E\hat{x}_{t+1}^S + a_{61}E\hat{p}_{t+1}^{A|S} + a_{62}E\hat{p}_{x,t+1}^{A|S} \\
a_{63}\hat{x}_t^A &= a_{64}E\hat{x}_{t+1}^A + a_{65}E\hat{p}_{t+1}^{S|A} + a_{66}E\hat{p}_{x,t+1}^{S|A}
\end{aligned}$$

with coefficients

$$
\begin{aligned}
a_1 &= 1-\mu-p^S-p^A+p^Sp^A;\ a_2=\gamma(1-p^{A|S});\ a_3=-s(1-p^A);\ a_4=-s(1-p^S);\ a_5=-\gamma r;\ a_6=-\mu;\\
a_7 &= p^S(1-p^A);\ a_8=(1-p^A)s;\ a_9=-p^Ss;\ a_{10}=1-\mu-p^{S|A};\ a_{11}=-in^A;\ a_{12}=(1-p^S)p^A;\\
a_{13} &= -sp^A;\ a_{14}=s(1-p^S);\ a_{15}=\gamma p^{A|S};\ a_{16}=\gamma r;\ a_{17}=\gamma;\ a_{18}=-\mu;\ a_{19}=p^{S|A};\ a_{20}=in^A;\\
a_{21} &= p^Sp^A;\ a_{22}=p^Ss;\ a_{23}=p^As;\ a_{24}=1-\mu-\gamma(1-p^{A|S})-p^{A|S};\ a_{25}=-r(1-\gamma)-in^S;\\
a_{26} &= 1-p^{A|S};\ a_{27}=1-\mu-\gamma;\ a_{28}=(1-\gamma)p^{A|S};\ a_{29}=in^S+(1-\gamma)r;\ a_{30}=(1+\ln[1-p^S])/x;\\
a_{31} &= -p^S_x/x;\ a_{32}=(1+\ln[1-p^A])/x;\ a_{33}=-p^A_x/x;\ a_{34}=(1+\ln[1-p^{S|A}])/x^A;\ a_{35}=-p^{S|A}_x/x^A;\\
a_{36} &= (1+\ln[1-p^{A|S}])/x^S;\ a_{37}=-p^{A|S}_x/x^S;\ a_{38}=-(p^{S|A}_x)^{-2}(x^A)^{-1};\ a_{39}=-(p^{S|A}_x)^{-1}(x^A)^{-2};\\
a_{40} &= -(p^{A|S}_x)^{-2}(x^S)^{-1};\ a_{41}=-(p^{A|S}_x)^{-1}(x^S)^{-2};\ a_{42}=\beta[x^{-1}+p^S(1-p^A)\delta_2x^{-2}];\\
a_{43} &= \beta[(1-p^A)\delta_1\delta_2+p^S(1-p^A)p^A_x\delta_1\delta_2^2+p^S(1-p^A)p^A_x\delta_2(x^Ap^{S|A}_x)^{-1}-p^A(x^Ap^{S|A}_x)^{-1}];\\
a_{44} &= \beta[-p^S\delta_2\delta_1+p^S(1-p^A)\delta_2^2\delta_1p^S_x+p^S(1-p^A)\delta_2p^S_x(x^Ap^{S|A}_x)^{-1}-p^S(x^Ap^{S|A}_x)^{-1}-(x^Sp^{A|S}_x)^{-1}];\\
a_{45} &= \beta[-p^S(1-p^A)^2\delta_2^2\delta_1+p^S(1-p^A)\delta_2p^A(x^Ap^{S|A}_x)^{-1}];\\
a_{46} &= \beta[(p^S)^2(1-p^A)\delta_2^2\delta_1+(p^S)^2(1-p^A)\delta_2(x^Ap^{S|A}_x)^{-1}+p^S(1-p^A)\delta_2(x^Sp^{A|S}_x)^{-1}];\\
a_{47} &= \beta[-p^S(1-p^A)\delta_2(p^S_xp^A+p^A_xp^S)(p^{S|A}_x)^{-1}(x^A)^{-2}+p^Sp^A(p^{S|A}_x)^{-1}(x^A)^{-2}];\\
a_{48} &= \beta[-p^S(1-p^A)\delta_2(p^S_xp^A+p^A_xp^S)(p^{S|A}_x)^{-2}(x^A)^{-1}+p^Sp^A(p^{S|A}_x)^{-2}(x^A)^{-1}];\\
a_{49} &= \beta[-p^S(1-p^A)\delta_2p^A_x(p^{A|S}_x)^{-1}(x^S)^{-2}-(1-p^A)(p^{A|S}_x)^{-1}(x^S)^{-2}];\\
a_{50} &= \beta[-p^S(1-p^A)\delta_2p^A_x(x^S)^{-1}(p^{A|S}_x)^{-2}-(1-p^A)(p^{A|S}_x)^{-2}(x^S)^{-1}];\\
a_{51} &= -(p^{S|A}_x)^{-2}(x^A)^{-1}-\delta_2(p^S_xp^A+p^A_xp^S)(p^{S|A}_x)^{-2}(x^A)^{-1};\\
a_{52} &= -(p^{S|A}_x)^{-1}(x^A)^{-2}-\delta_2(p^S_xp^A+p^A_xp^S)(p^{S|A}_x)^{-1}(x^A)^{-2};\\
a_{53} &= -(p^{A|S}_x)^{-2}(x^S)^{-1}-\delta_2p^A_x(p^{A|S}_x)^{-2}(x^S)^{-1};\ a_{54}=-(p^{A|S}_x)^{-1}(x^S)^{-2}-\delta_2p^A_x(p^{A|S}_x)^{-1}(x^S)^{-2};\\
a_{55} &= -\delta_1\delta_2^2(1-p^A)+\delta_2p^A(x^Ap^{S|A}_x)^{-1};\\
a_{56} &= \delta_1\delta_2^2p^S_x+\delta_2p^S_x(x^Ap^{S|A}_x)^{-1};\ a_{57}=\delta_1\delta_2^2p^A_x+\delta_2p^A_x(x^Ap^{S|A}_x)^{-1};\ a_{58}=\delta_1\delta_2^2p^S+\delta_2p^S(x^Ap^{S|A}_x)^{-1}+\delta_2(x^Sp^{A|S}_x)^{-1};\\
a_{59} &= \delta_2x^{-2};\ a_{60}=\beta[(x^S)^{-1}-(1-p^{A|S})(x^S)^{-2}(p^{A|S}_x)^{-1}];a_{61}=\beta[-(x^Sp^{A|S}_x)^{-1}];\ a_{62}=\beta[-(1-p^{A|S})(x^S)^{-1}(p^{A|S}_x)^{-2}];\\
a_{63} &= a_{38};a_{64}=\beta[(x^A)^{-1}+p^{S|A}(p^{S|A}_x)^{-1}(x^A)^{-2}];\ a_{65}=\beta[-(x^Ap^{S|A}_x)^{-1}];\ a_{66}=\beta[p^{S|A}(x^A)^{-1}(p^{S|A}_x)^{-2}].
\end{aligned}
$$

1.7 LINEARIZED MATRIX SYSTEM

The linearized EE system in matrix form is

$$\underbrace{\begin{bmatrix} a_1 & 0 & 0 & 0 & a_2 & 0 & 0 & 0 \\ a_7 & a_6 & 0 & 0 & 0 & 0 & 0 & 0 \\ a_{12} & 0 & a_{10} & 0 & a_{15} & a_{17} & 0 & 0 \\ a_{21} & 0 & a_{19} & a_{18} & 0 & 0 & 0 & 0 \\ 0 & a_{26} & 0 & 0 & a_{24} & 0 & 0 & 0 \\ 1 & 1 & 1 & 1 & 1 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_{41} \\ 0 & 0 & 0 & 0 & 0 & 0 & a_{59} & a_{54} \end{bmatrix} \begin{bmatrix} \hat{s}_t \\ \widehat{in}_t^S \\ \widehat{in}_t^A \\ \widehat{in}_t^{SA} \\ \hat{r}_t \\ \hat{r}_t^A \\ \hat{x}_t \\ \hat{x}_t^S \end{bmatrix}}_{A\hat{y}_t} + \underbrace{\begin{bmatrix} a_3 & a_4 & 0 & a_5 & 0 & 0 & 0 & 0 \\ a_8 & a_9 & 0 & 0 & 0 & 0 & 0 & 0 \\ a_{13} & a_{14} & a_{11} & a_{16} & 0 & 0 & 0 & 0 \\ a_{23} & a_{22} & a_{20} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & a_{25} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & a_{38} & a_{40} \\ a_{57} & a_{56} & 0 & 0 & a_{55} & a_{58} & a_{51} & a_{53} \end{bmatrix} \begin{bmatrix} \hat{p}_t^S \\ \hat{p}_t^A \\ \hat{p}_t^{S|A} \\ \hat{p}_t^{A|S} \\ \hat{p}_{x,t}^S \\ \hat{p}_{x,t}^A \\ \hat{p}_{x,t}^{S|A} \\ \hat{p}_{x,t}^{A|S} \end{bmatrix}}_{B\hat{w}_t}$$

$$= \underbrace{\begin{bmatrix} 1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & a_{42} & a_{49} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_{60} \end{bmatrix} \begin{bmatrix} \hat{s}_{t+1} \\ \widehat{in}_{t+1}^S \\ \widehat{in}_{t+1}^A \\ \widehat{in}_{t+1}^{SA} \\ \hat{r}_{t+1} \\ \hat{r}_{t+1}^A \\ E\hat{x}_{t+1} \\ E\hat{x}_{t+1}^S \end{bmatrix}}_{C\hat{y}_{t+1}} + \underbrace{\begin{bmatrix} 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ a_{43} & a_{44} & 0 & 0 & a_{45} & a_{46} & a_{48} & a_{50} \\ 0 & 0 & 0 & a_{61} & 0 & 0 & 0 & a_{62} \end{bmatrix} \begin{bmatrix} E\hat{p}_{t+1}^S \\ E\hat{p}_{t+1}^A \\ E\hat{p}_{t+1}^{S|A} \\ E\hat{p}_{t+1}^{A|S} \\ E\hat{p}_{x,t+1}^S \\ E\hat{p}_{x,t+1}^A \\ E\hat{p}_{x,t+1}^{S|A} \\ E\hat{p}_{x,t+1}^{A|S} \end{bmatrix}}_{D\hat{w}_{t+1}}$$

and

$$\underbrace{\begin{bmatrix} 1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 & 0 & 0 \\ -a_{30} & 0 & 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & -a_{32} & 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & -a_{34} & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & -a_{36} & 0 & 0 & 0 & 1 \end{bmatrix} \begin{bmatrix} \hat{p}_t^S \\ \hat{p}_t^A \\ \hat{p}_t^{S|A} \\ \hat{p}_t^{A|S} \\ \hat{p}_{x,t}^S \\ \hat{p}_{x,t}^A \\ \hat{p}_{x,t}^{S|A} \\ \hat{p}_{x,t}^{A|S} \end{bmatrix}}_{F\hat{w}_t} = \underbrace{\begin{bmatrix} 0 & p_{in}^S & 0 & p_{in}^S & 0 & 0 & p_x^S & 0 \\ 0 & 0 & p_{in}^A & p_{in}^A & 0 & p_{in}^A & p_x^A & 0 \\ 0 & p_{in}^{S|A} & 0 & p_{in}^{S|A} & 0 & 0 & 0 & 0 \\ 0 & 0 & p_{in}^{A|S} & p_{in}^{A|S} & 0 & p_{in}^{A|S} & 0 & p_x^{A|S} \\ 0 & 0 & 0 & 0 & 0 & 0 & a_{31} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & a_{33} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_{37} \end{bmatrix} \begin{bmatrix} \hat{s}_t \\ \widehat{in}_t^S \\ \widehat{in}_t^A \\ \widehat{in}_t^{SA} \\ \hat{r}_t \\ \hat{r}_t^A \\ \hat{x}_t \\ \hat{x}_t^S \end{bmatrix}}_{G\hat{y}_t},$$

where, for our parameter choices, individuals with only AIDS always choose the maximum number of partners ($x_t^A = \bar{x}$). This implies that $\hat{x}_t^A = 0$ for all t.

Writing the matrix EE system in compact form, we get

$$\begin{aligned} A\hat{y}_t + B\hat{w}_t &= C\hat{y}_{t+1} + D\hat{w}_{t+1} \\ F\hat{w}_t &= G\hat{y}_t \end{aligned}$$

or

$$\hat{y}_t = J\hat{y}_{t+1},$$

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

1.8 RATIONAL EXPECTATIONS EQUILIBRIUM (REE)

We use the method of Blanchard and Kahn (1980) to solve for the REE. The vector $\hat{y}_t$ contains six predetermined and two jump variables. There are two forward-stable roots of J so the system displays saddle-path stability and a unique endemic REE. The equilibrium under naïve expectations is calculated by setting $E\hat{x}_{t+1} = \hat{x}_t$, $E\hat{x}_{t+1}^S = \hat{x}_t^S$, and $E\hat{w}_{t+1} = \hat{w}_t$.

1.9 PARAMETERS AND STEADY-STATE VALUES

Table A2 shows the baseline parameter values and the implied steady-state values.

Table A2. Baseline Parameters and Steady-State Values

Parameters												
β	γ	μ	a	h^{SA}	h^A	h^S	h	$\bar{x}$	λ_a^S	λ_a^A	$\lambda_a^{A\|S}$	$\lambda_a^{S\|A}$
0.96	0.2	0.05	40	0	0	5	5	10	0.023	0.0008	0.024	0.023

Endemic Steady-State Values												
x	x^S	x^A	s	in^S	in^A	in^{SA}	r	r^A	p^S	p^A	$p^{S\|A}$	$p^{A\|S}$
1.941	0.116	10	0.562	0.043	0.081	0.028	0.161	0.125	0.082	0.014	0.358	0.018

Syphilis Eradication Steady-State Values													
x	x^S	x^A	s	in^S	in^A	in^{SA}	r	r^A	p^S	p^A	$p^{S\|A}$	$p^{A\|S}$	R_0
2.141	–	10	0.745	0	0.255	0	0	0	0	0.017	0	–	2.393

We now justify our choice of parameter values, which can be placed into epidemiological and economic categories.

1.9.1 Epidemiological Parameters

- $\lambda_a^S = 0.023$ (Probability of contracting syphilis with an infected partner, one act)
- $\lambda_p^S = 0.60 = 1 - (1 - \lambda_a^S)^a$ (Probability of contracting syphilis with an infected partner, all acts)
- $\lambda_a^A = 0.0008$ (Probability of contracting AIDS with an infected partner, one act)
- $\lambda_a^{A|S} = 0.024$ (Probability of contracting AIDS with syphilis and an infected partner, one act)
- $v = 1$ (Syphilis treatment rate)
- $\gamma = 0.2$ (Syphilis loss of host immunity rate)
- $\mu = 0.05$ (Birth/death rate)

For the syphilis parameters, Garnett et al. (1997) suggests that $\lambda_p^S = 0.6$ is a potentially "unbiased estimate" (page 189) for the partner probability of syphilis transmission. If we assume that a susceptible individual has $a = 40$ sexual acts with

each partner, the implied probability of contracting syphilis from a single act is $\lambda_a^S = 0.023$. For the AIDS parameters, Chesson and Pinkerton (2000) document mean per act probabilities of AIDS transmission to be 0.001 for male-to-female transmission and 0.0006 for female-to-male transmission. We employ the average of these in our gender-neutral per-act AIDS transmission probability of $\lambda_a^A = 0.0008$. Chesson and Pinkerton (2000) also provide an estimate of the probability that an individual who has syphilis will contract AIDS from a single act with an infected partner ($\lambda_a^{A|S} = 0.024$). The treatment parameter for syphilis v captures both the rate of diagnosis and treatment. The treatment effectiveness for syphilis appears to be close to 100% (Alexander et al. (1999)) so that $v = 1$. Following Garnett et al. (1997) we assume an average duration of host immunity to syphilis of 5 years, implying a value of $\gamma = 0.2$. The population is assumed to have a birth/death rate of $\mu = 0.05$ as in Garnett et al. (1997).

1.9.2 Economic Parameters

- $a = 40$ (Number of sexual acts per partner)
- $h^{SA} = 0$ (Health parameter with syphilis and AIDS)
- $h^A = h^{SA}$ (Health parameter with AIDS only)
- $h^S = 5$ (Health parameter with syphilis only)
- $h = h^S$ (Health parameter without syphilis or AIDS)
- $\beta = 0.96$ (Discount factor)
- $\bar{x} = 10$ (Maximum number of partners per period)

Sexual acts per partner is set at $a = 40$. Chesson and Gift (2000) set the total number of sexual acts per year at 100. Smith (1994) cite a figure of 62 total sexual acts per year, on average across the adult population. Using our steady state of approximately two partners per year, the implied total number of sexual acts ($40 \times 2 = 80$) is a midpoint of these two estimates. We normalize the utility health parameter with syphilis and AIDS (h^{SA}) to zero. The health parameter for individuals with AIDS but not syphilis is also set at zero. This captures the notion that the health risks of AIDS dominate those of syphilis. Contracting syphilis is still a concern to susceptible individuals because it significantly increases the risk of contracting AIDS. The health parameter without AIDS or syphilis (h) or without AIDS but with syphilis (h^S) is set at 5. This value produces dynamic dampening and is chosen to produce syphilis cycles with an approximate ten-year period under naïve expectations (Grassly, Fraser and Garnett (2005)). A discount factor of $\beta = 0.96$ is standard for annual data and is consistent with a 4% real rate of return. The value of $\bar{x} = 10$ was inferred from a number of sources. Andrus et al. (1990) reports an average number of partners for those infected with syphilis of 6.3 partners during the infectious period. Koblin

et al. (2003) found that in a non-HIV sample of approximately 4,300 homosexual men across six major U.S. cities, over half the sample report having more than 15 partners per year. The value for $\bar{x}$ is also varied to capture possible altruism by infected individuals.

1.10 DYNAMICS AND IMPULSE RESPONSE FUNCTIONS

Figures A1 and A2 show the dynamic responses to a 0.05 increase in the fraction of the population infected with syphilis only (in^S) and a 0.05 increase in the fraction of the population infected with AIDS only (in^A). Figure A1 uses the baseline parameters in Table A2 and displays dynamic dampening for both naïve and rational expectations. Figure A2 uses identical parameter values except the health parameter, $h^S = h$, is reduced to 1.95. This causes fatalism to set in for naïve individuals, leading to dynamic resonance. Further reductions in $h^S = h$ are necessary for fatalism and dynamic resonance to occur with rational expectations. The nature of the dynamic responses and the comparison to the pre-AIDS era are discussed in the main paper.

2 PRE-AIDS ERA

2.1 SYPHILIS EPIDEMIOLOGICAL MODEL

The syphilis (SIRS) population model contains three mutually exclusive categories: susceptible to syphilis (s), infected with syphilis (in), and immune to syphilis (r). We start by presenting the transition matrix between these categories in Table A3:

Table A3. Transition Matrix for Disease Categories

	s_{t+1}	in_{t+1}	r_{t+1}
s_t	$1 - p_t$	p_t	0
in_t	0	0	1
r_t	γ	0	$1 - \gamma$

Using the transition probabilities and a 100% syphilis treatment rate, the equations of motion for the disease categories are

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

where the probability of contracting syphilis is

$$p_t = \Pr(\text{contract syphilis}) = 1 - (1 - \lambda_p in_t)^{x_t}.$$

2.2 VALUE FUNCTIONS

The value functions apply to individuals: (1) susceptible to syphilis, V_t and (2) infected with syphilis, V_t^S. There is no value function for those recovered (and immune) to syphilis because we assume individuals cannot distinguish the susceptible state from the recovered and immune state. The value functions are

$$V_t = \max_{x_t}\{u(x_t, h) + \beta[p_t V_{t+1}^S + (1 - p_t)V_{t+1}]\} \tag{11}$$

$$V_t^S = u(\bar{x}, h^S) + \beta V_{t+1}. \tag{12}$$

2.3 EULER EQUATIONS

Assuming an interior solution, the necessary first-order condition for s individuals is

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

where

$$p_{x,t} = \frac{\partial p_t}{\partial x_t} = -\ln(1 - p_t)(1 - p_t)/x_t.$$

Using (11) and (12) to substitute out the optimized value functions, the Euler equation become

$$\frac{1}{p_{x,t}x_t} = \beta E\left(u_{t+1} - u_{t+1}^S - \frac{p_{t+1}}{x_{t+1}p_{x,t+1}}\right). \tag{13}$$

2.4 STEADY STATE

The endemic steady state solves for four variables, $\{s, in, r, x\}$, from the following four equations:

$$s = in(1 + \mu)/p \tag{14}$$

$$in = \frac{(1 - s)(\mu + \gamma)}{1 + \mu + \gamma} \tag{15}$$

$$r = \frac{(1 - s)}{1 + \mu + \gamma} \tag{16}$$

$$1 = \beta[p_x x(u - u^S) - p]. \tag{17}$$

2.5 LINEARIZATION

We start by linearizing the SIRS epidemiological equations around the endemic steady state:

$$\begin{aligned}\hat{s}_{t+1} &= (1-p-\mu)\hat{s}_t + \gamma\hat{r}_t - s\hat{p}_t \\ \widehat{in}_{t+1} &= (sp_{in}-\mu)\widehat{in}_t + sp_x\hat{x}_t + p\hat{s}_t \\ \hat{r}_{t+1} &= (1-\gamma-\mu)\hat{r}_t + \widehat{in}_t.\end{aligned}$$

Next, linearize the probabilities (and derivative of the probability with respect to the number of partners):

$$\hat{p}_t = p_{in}\widehat{in}_t + p_x\hat{x}_t \tag{18}$$

$$\hat{p}_{x,t} = [(1+\ln[1-p])/x]\hat{p}_t - (p_x/x)\hat{x}_t \tag{19}$$

where

$$\begin{aligned}p_{in} &= x\lambda_p(1-\lambda_p in)^{x-1} \\ p_x &= -\ln(1-p)(1-p)/x.\end{aligned}$$

The linearized Euler equation is:

$$x^{-1}\hat{x}_t + p_x^{-1}\hat{p}_{x,t} = -\beta[p_x + px^{-1}]E\hat{x}_{t+1} + \beta E\hat{p}_{t+1} - \beta pp_x^{-1}E\hat{p}_{x,t+1}. \tag{20}$$

Summarizing, the linearized EE system with (18) and (19) substituted into (20) is

SIRS System

$$\begin{aligned}\widehat{in}_{t+1} &= b_1\widehat{in}_t + sp_x\hat{x}_t - p\hat{r}_t \\ \hat{r}_{t+1} &= b_2\hat{r}_t + \widehat{in}_t\end{aligned}$$

Probabilities

$$\begin{aligned}\hat{p}_t &= p_{in}\widehat{in}_t + p_x\hat{x}_t \\ \hat{p}_{x,t} &= b_3\hat{p}_t + b_4\hat{x}_t\end{aligned}$$

Euler Equation

$$b_5\hat{x}_t + b_6\widehat{in}_t = b_7E\hat{x}_{t+1} + b_8E\widehat{in}_{t+1}$$

with coefficients

$$\begin{aligned}b_1 &= sp_{in} - \mu - p;\ b_2 = 1-\gamma-\mu;\ b_3 = [(1+\ln[1-p])/x];\ b_4 = -p_x/x; \\ b_5 &= x^{-1} + (b_3 + b_4 p_x^{-1});\ b_6 = p_x^{-1}b_3p_{in};\ b_7 = -\beta[px^{-1} - p(b_3 + b_4p_x^{-1})]; \\ b_8 &= \beta(p_{in} - pp_x^{-1}b_3p_{in}).\end{aligned}$$

2.6 LINEARIZED MATRIX SYSTEM

The linearized EE system in matrix form is

$$\underbrace{\begin{bmatrix} b_1 & -p & sp_x \\ 1 & b_2 & 0 \\ b_6 & 0 & b_5 \end{bmatrix}}_{\tilde{A}} \begin{bmatrix} \widehat{in}_t \\ \hat{r}_t \\ \hat{x}_t \end{bmatrix} = \underbrace{\begin{bmatrix} 1 & 0 & 0 \\ 0 & 1 & 0 \\ b_8 & 0 & b_7 \end{bmatrix}}_{\tilde{B}} \begin{bmatrix} \widehat{in}_{t+1} \\ \hat{r}_{t+1} \\ E\hat{x}_{t+1} \end{bmatrix}. \tag{21}$$

The matrix system includes the restriction $\hat{s}_t = -\hat{r}_t - \widehat{in}_t$ and the maximum choice of partners for those with syphilis, $x_t^S = \bar{x}$.

2.7 RATIONAL EXPECTATIONS EQUILIBRIUM (REE)

The pre-AIDS EE system also exhibits saddle-path stability with one jump variable, two predetermined variables and one forward-stable root. Again, using the method of Blanchard and Kahn (1980), we solve for a contemporaneous relationship between the jump variable and the two state variables:

$$\hat{x}_t = b_9 \widehat{in}_t + b_{10} \hat{r}_t \tag{22}$$

where

$$\begin{aligned} b_9 &= -Q_{31}^{-1}/Q_{33}^{-1}; \\ b_{10} &= -Q_{32}^{-1}/Q_{33}^{-1}, \end{aligned}$$

Q_{ij}^{-1} refers to the (i,j) element of the inverse of the matrix of stacked eigenvectors for $\tilde{A}^{-1}\tilde{B}$, and the $i = 3$ eigenvalue of $\tilde{A}^{-1}\tilde{B}$ is the forward stable root. Using (22) to solve for the reduced-form representation gives

$$\begin{bmatrix} \widehat{in}_{t+1} \\ \hat{r}_{t+1} \end{bmatrix} = \begin{bmatrix} b_1 + sp_x b_9 & -p + sp_x b_{10} \\ 1 & b_2 \end{bmatrix} \begin{bmatrix} \widehat{in}_t \\ \hat{r}_t \end{bmatrix}. \tag{23}$$

2.8 DYNAMICS AND IMPULSE RESPONSE FUNCTIONS

Figures A3, A4 and A5 show the dynamic responses to a 0.05 increase in syphilis prevalence. Figure A3 uses the baseline parameter values from Table 1 in the main paper; Figure A4 uses identical parameter values except the health parameter, h, is reduced to 5.4; and Figure A5 further reduces h to 4.54. Figure A3 shows that individuals with naïve or rational expectations both display dynamic dampening. Figure A4 shows that for naïve individuals the reduction to $h = 5.4$ decreases the health benefit of being disease free, increases the steady-state number of partners, and leads to fatalistic behavior. Individuals with rational expectations continue to exhibit dampening behavior when $h = 5.4$. Figure A5 shows that a further reduction to $h = 4.54$ induces individuals with rational expectations to be fatalistic and cause dynamic resonance.

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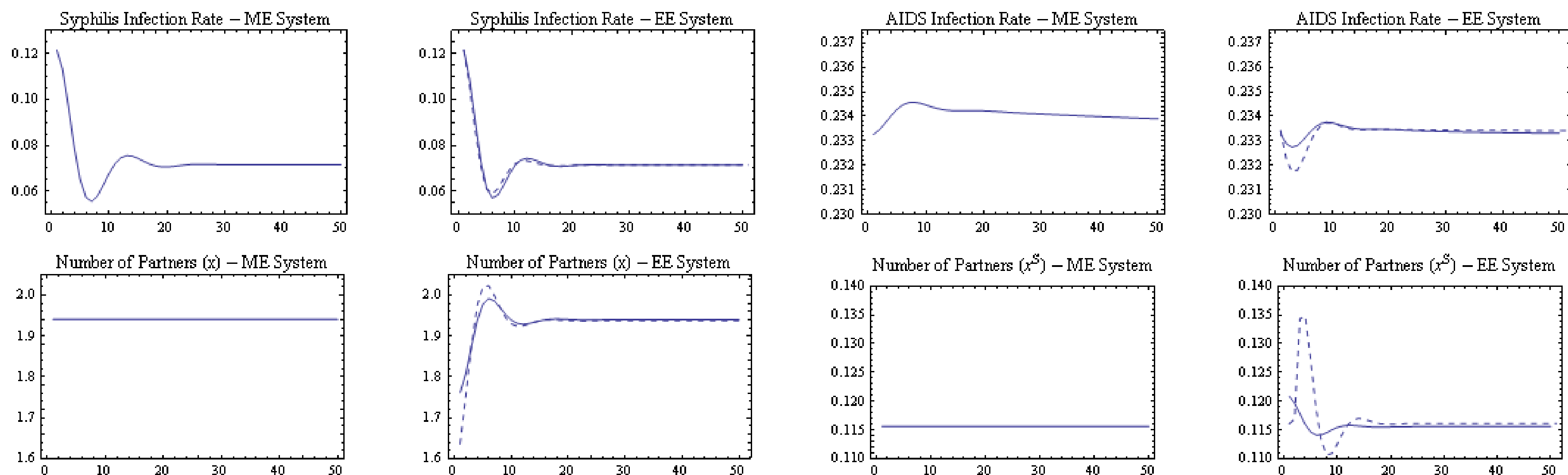
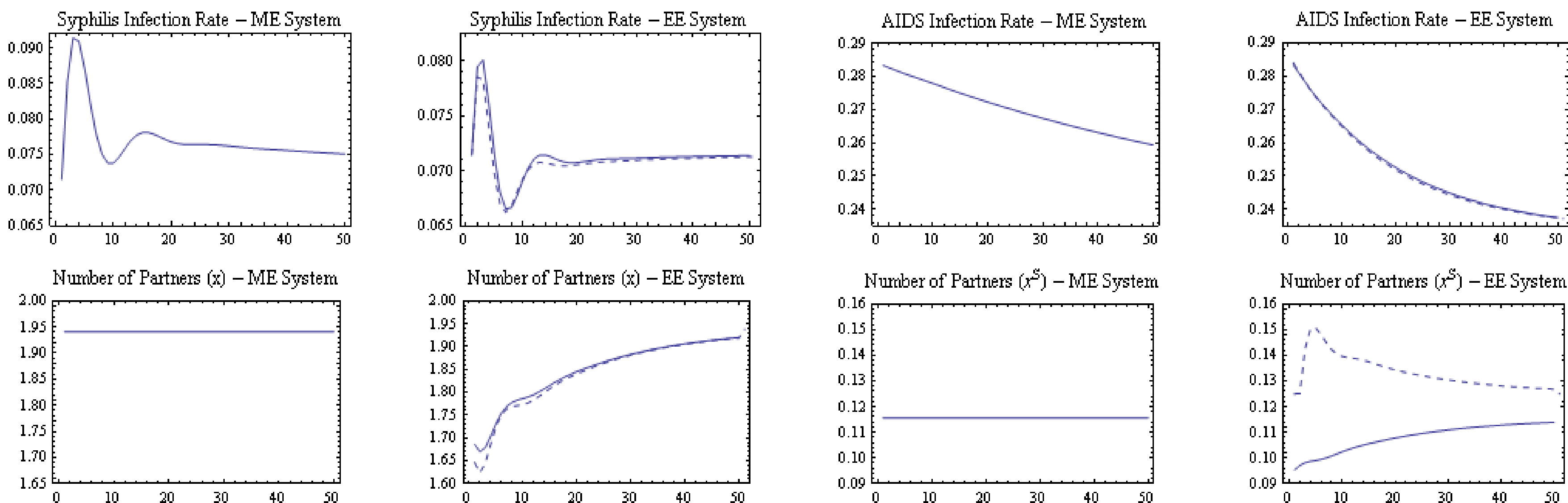
Figure A1. Impulse Response Functions for the ME and EE Systems in the Post-AIDS Period – Rational Dynamic Dampening

Syphilis Shock: One-time 0.05 increase in syphilis prevalence (in^S), (solid = naïve expectations, dashed = rational expectations)



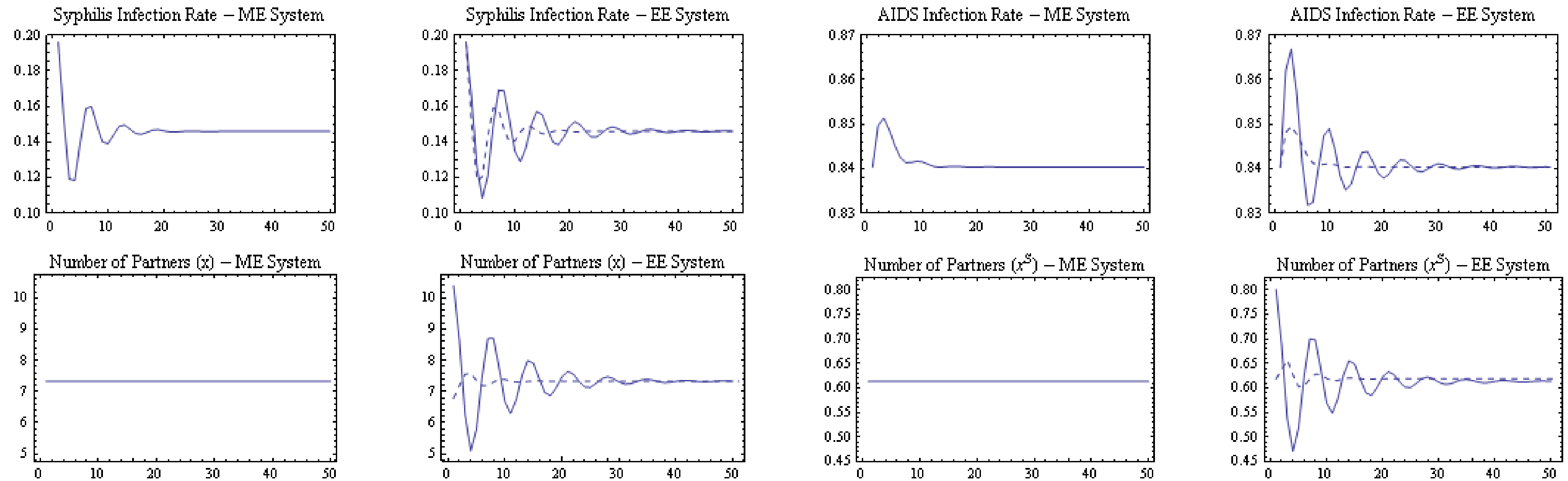
AIDS Shock: One-time 0.05 increase in AIDS prevalence (in^A), (solid = naïve expectations, dashed = rational expectations)



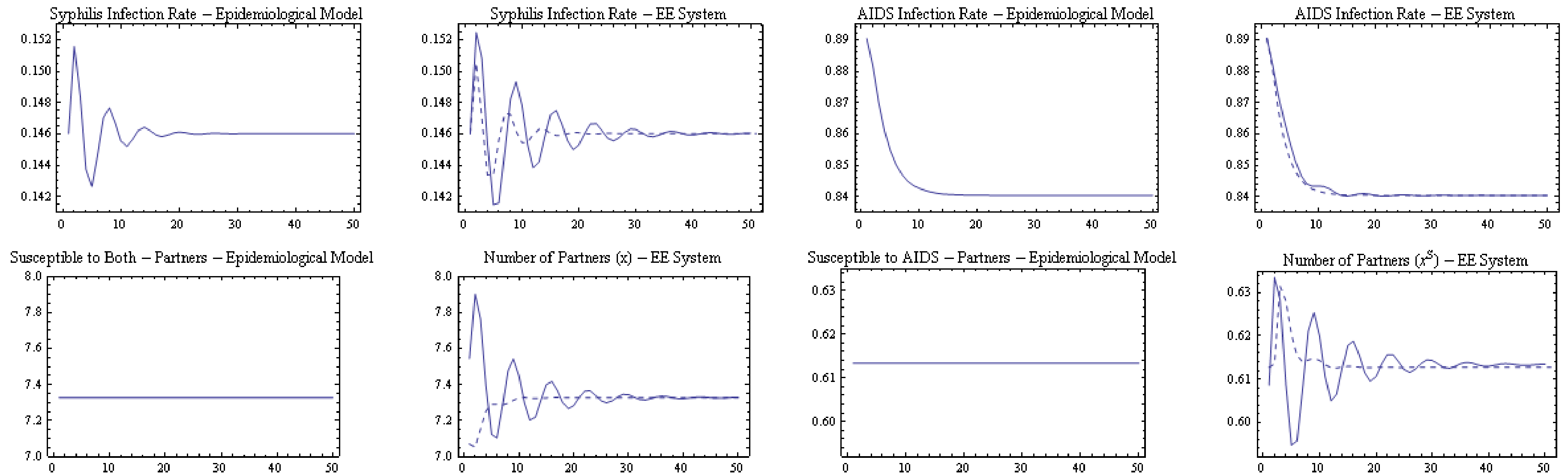
Notes. EE fundamental parameters: $\beta = 0.96$, $\mu = 0.05$, $\gamma = 0.2$, $h^{SA} = h^A = 0$, $h^S = h = 5$, $\lambda_a^S = \lambda_a^{S|A} = 0.023$, $\lambda_a^A = 0.0008$, $\lambda_a^{A|S} = 0.024$, $\lambda_a^S = 0.023$, $a = 40$ and $\bar{x} = 10$.

Figure A2. Impulse Response Functions for the ME and EE Systems in the Post-AIDS Period – Rational Dynamic Resonance

Syphilis Shock: One-time 0.05 increase in syphilis prevalence (in^S) , (solid = naïve expectations, dashed = rational expectations)

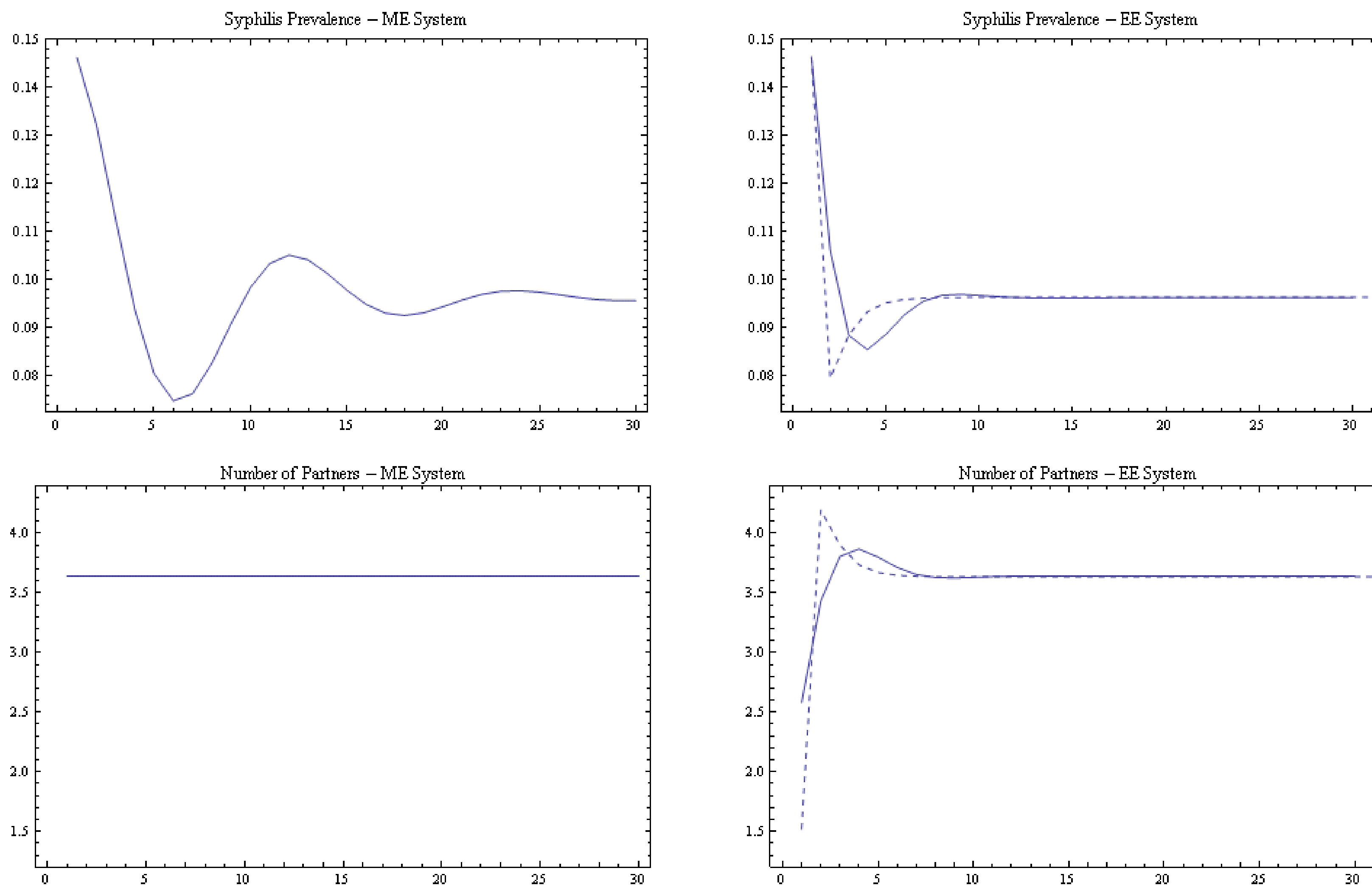


AIDS Shock: One-time 0.05 increase in AIDS prevalence (in^A) , (solid = naïve expectations, dashed = rational expectations)



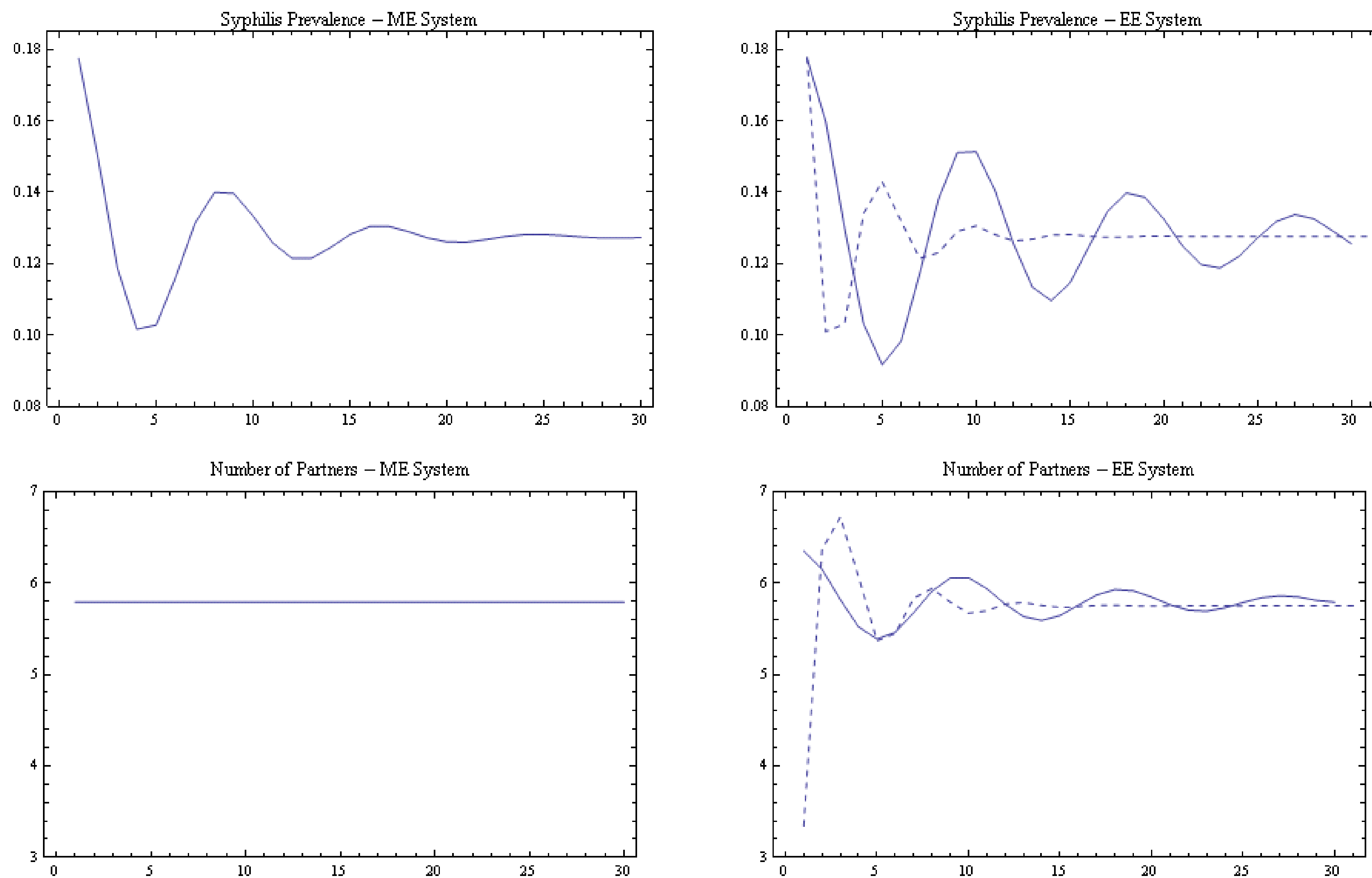
Notes. EE fundamental parameters: $\beta = 0.96$, $\mu = 0.05$, $\gamma = 0.2$, $h^{SA} = h^{A} = 0$, $h^{S} = h = 1.95$, $\lambda_a^S = \lambda_a^{S|A} = 0.023$, $\lambda_a^A = 0.0008$, $\lambda_a^{A|S} = 0.024$, $\lambda_a^S = 0.023$, $a = 40$ and $\bar{x} = 10$.

Figure A3. Impulse Response Functions for the ME and EE Systems – Rational Dynamic Dampening
(solid = naïve expectations, dashed = rational expectations)



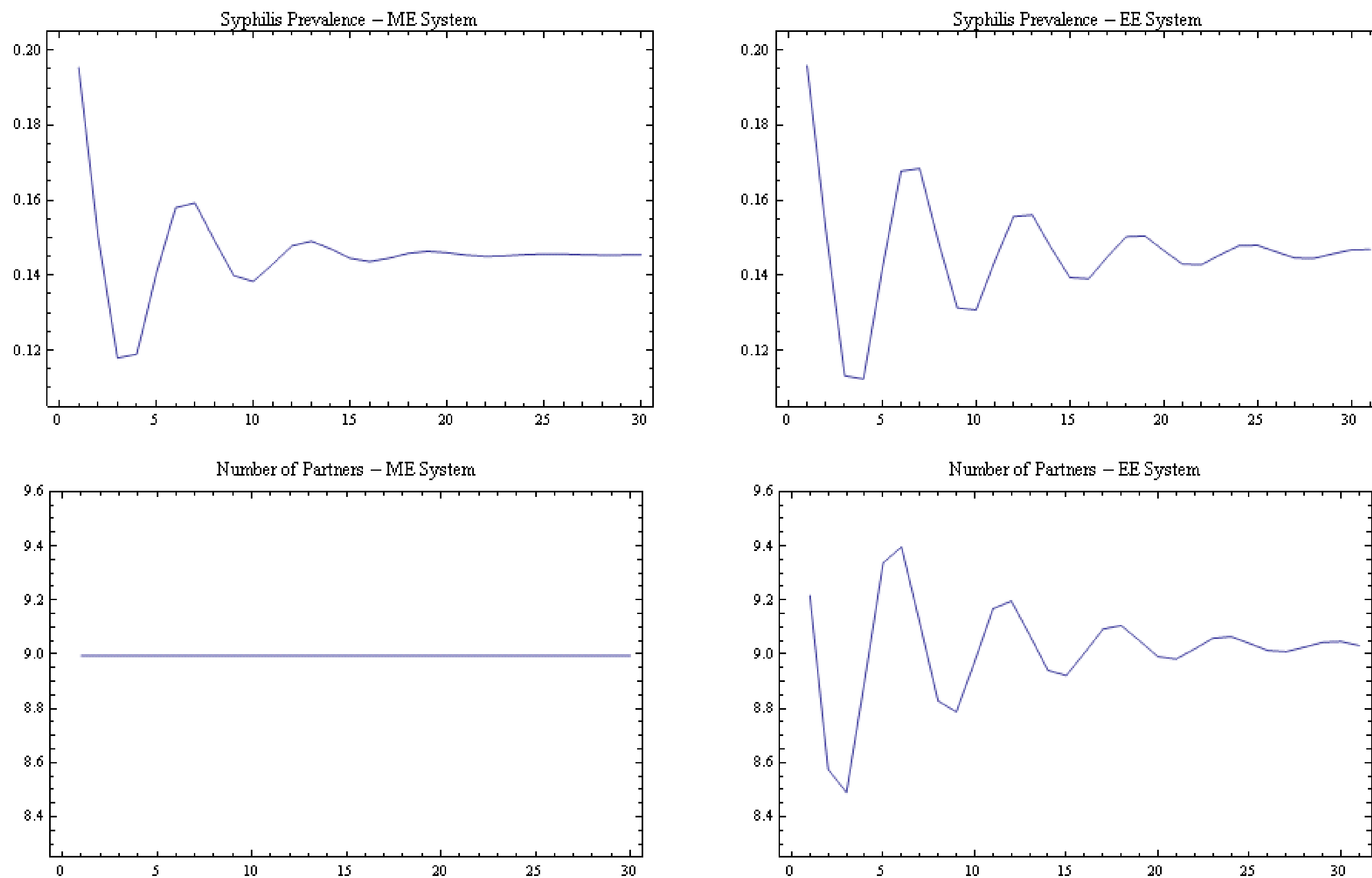
Notes. The fundamental parameters in the EE system are set at $\beta = 0.96$, $\mu = 0.05$, $\gamma = 0.2$, $h = 8.1$, $\lambda_a = 0.023$, $a = 40$ and $\bar{x} = 10$. For comparison purposes, we set the steady-state number of partners (x) in the ME model equal to the endogenously solved for number of partners in the EE model. As a result, the steady-state prevalence is also equal in the ME and EE models.

Figure A4. Impulse Response Functions for the ME and EE Systems – Rational Dynamic Resonance
(solid = naïve expectations, dashed = rational expectations)



Notes. The fundamental parameters in the EE system are set at $\beta = 0.96$, $\mu = 0.05$, $\gamma = 0.2$, $h = 5.4$, $\lambda_a = 0.023$, $a = 40$ and $\bar{x} = 10$. For comparison purposes, we set the steady-state number of partners (x) in the ME model equal to the endogenously solved for number of partners in the EE model. As a result, the steady-state prevalence is also equal in the ME and EE models.

Figure A5. Impulse Response Functions for the ME and EE Systems – Rational Dynamic Resonance (Rational Expectations)



Notes. The fundamental parameters in the EE system are set at $\beta = 0.96$, $\mu = 0.05$, $\gamma = 0.2$, $h = 4.54$, $\lambda_a = 0.023$, $a = 40$ and $\bar{x} = 10$. For comparison purposes, we set the steady-state number of partners (x) in the ME model equal to the endogenously solved for number of partners in the EE model. As a result, the steady-state prevalence is also equal in the ME and EE models.