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. 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 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)). 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. U.S. infection rates for primary and secondary syphilis fell dramatically during the 1940s and began to oscillate around a much lower rate of incidence (see Figure 1). 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)). The sharpest rise has occurred in men who have sex with men (MSM), accounting for over 60% of primary and secondary U.S. syphilis cases in 2004 as compared to only 4% of U.S. cases in 2000. This is an alarming demographic shift given the high-risk sexual behavior and elevated chance for HIV infection among MSM (Chen et al. (2002); Heffelfinger et al. (2007)).

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

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1Syphilis is remembered by many for the infamous Tuskegee 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.
its global initiative to eliminate congenital syphilis (WHO (2007)). Their plan advocates improved antenatal care, universal testing for pregnant women and partners, rapid treatment, promotion of condom use, and enhanced synergies with HIV prevention programs. The CDC’s National Plan to Eradicate Syphilis, first introduced in 1999, is an attempt to capitalize on historically low levels of prevalence and finally rid the U.S. of the disease (CDC (1999)). The plan emphasizes improved reporting and data gathering, rapid diagnosis and treatment of outbreaks, and a concerted effort to increase awareness of the health consequences of sexual activity. But the U.S. plan has not worked. The 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)).

Our research offers new insights into the failure of the National Plan to Eradicate Syphilis. Our model predicts that the eradication plan had a greater chance of success in its first years due to the prominence of AIDS risk among sexually active individuals. However, the window of opportunity to eradicate syphilis has likely closed due to the discovery of new drug therapies that lower the health risks of AIDS and encourage more risky sexual behavior, in turn explaining the recent rise in the prevalence of syphilis. Although eradication is still theoretically possible, it requires implausibly high degrees of altruism for those infected with syphilis or AIDS.3

In addition to the eradication plan, there is an ongoing debate over the determinants of oscillations in the prevalence of syphilis. 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 disease risk (e.g., Geoffard and Philipson (1996); Kremer (1996); Auld (2003); Gersovitz and Hammer (2004)) and various economic risks (e.g., Ehrlich and Becker

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2 For more details on the plan, see www.cdc.gov/stopsyphilis/.
3 Of course, eradication policies can still have a positive impact. 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.
Although our research does not resolve the debate, it does highlights how the existence and nature of syphilis cycles depend critically on human preferences over sexual activity and health.

Using an integrated economic-epidemiological (EE) model, we show how syphilis cycles can either 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. 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.

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 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.

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.
2 Syphilis Epidemiology

Syphilis is a sexually transmitted disease (STD) caused by the spiral microorganism *Treponema pallidum*. The disease is unique in its slow tempo of progression through infected individuals, but if left untreated may eventually cripple or kill one in four of those infected. The point of infection eventually becomes characterized by an ulcerative 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 MSM (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. Although our model is ultimately calibrated to U.S. data and therefore directly applicable to developed countries, we expect the same general behavioral responses to risk should apply to individuals from the developing world. 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)).

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.

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6Chesson, 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.
3 Integrated General Equilibrium Model

Following work by Philipson and Posner (1993), we specify an integrated epidemiological and economic model to describe syphilis dynamics. The model is set in discrete time with \( t \) indexing annual decision intervals.\(^7\) There is a constant population of \( N \) individuals, which are all identical except for their state of the disease. Sexual activity brings multiple STD risks that individuals cannot choose between. For transparency, we present a single-disease model that is appropriate when the health risks of other diseases are low or relatively stable. A more sophisticated SIRS/SI epidemiological model of syphilis and AIDS dynamics is presented in the Appendix.

3.1 Epidemiology

The epidemiological portion of the model describes the evolution of three mutually exclusive disease categories: susceptible (\( s \)), infected (\( in \)), and immune to syphilis (\( r \)). Each disease category is measured as a proportion of the overall population with the sum of the categories equal to one.

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 is

\[
p_t = \Pr(\text{contract syphilis}) = 1 - (1 - \lambda_p)^{x_t}
\]

where \( \lambda_p = 1 - (1 - \lambda_a) \) is the probability of contracting the disease from a single infected partner, and \( \lambda_a \) is the probability of contracting the disease from a single sexual act (Kaplan (1990); Oster (2005)). The dependence on the endogenous number of partners distinguishes the analysis from standard mathematical epidemiology.

The epidemiological model is represented by the following three equations:

\(^7\)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.
\[ s_{t+1} = \mu + (1 - \lambda in_t - \mu)s_t + \gamma r_t \]  
\[ in_{t+1} = \lambda in_t s_t + (1 - \nu - \mu)in_t \]  
\[ r_{t+1} = \nu in_t + (1 - \gamma - \mu)r_t, \]  

where \( \mu \) is the common birth/death rate, \( 1/\gamma \) is the average duration of immunity, and the treatment rate is 100\%. 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

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

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.\(^8\)

In any period \( t \), individual \( i \) must be in one of the three epidemiological states: susceptible \( (s_{i,t}) \), infected \( (in_{i,t}) \), or recovered and immune \( (r_{i,t}) \). For example, if an individual is susceptible then \( s_{i,t} = 1 \) and \( in_{i,t} = r_{i,t} = 0 \). Because an individual can only be in one state at any time, \( s_{i,t} + in_{i,t} + r_{i,t} = 1 \) for all \( i \) and \( t \). The proportions of susceptible, infected and recovered individuals

\(^8\)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 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 three epidemiological categories, the recovered state is 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 the other two categories – susceptible (\(V^S_t\)) and infected (\(V^{IN}_t\)) – are given by

\[
V^S_t = \ln(x_t) + h + \beta[p_t V^{IN}_{t+1} + (1 - p_t) V^S_{t+1}]
\]

\[
V^{IN}_t = \ln(\bar{x}) + \beta V^S_{t+1},
\]

where the health parameter for infected individuals is normalized to zero.

In our baseline model, all individuals regardless of infection status are self-interested and maximize (5) without concern for the welfare of the general population. Rational, self-interested infected individuals 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.\(^9\) 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 \(\bar{x}\).

\(^9\)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.
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. For a more thorough investigation of rational expectation epidemiological equilibria using a similar modeling framework, see the working paper Aadland, Finnoff and Huang (2011).

Assuming an interior solution, the Euler equations for the number of partners \( x_t \) is

\[
x_t^{-1} = \beta p_{x,t} [V^S_{t+1} - V^I_{t+1}],
\]

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

To better understand the Euler equation, consider a susceptible individual who is deciding how many partners to choose under the risk of future infection. Equation (8) represents a standard solution for dynamic expected-utility maximization problems of this type: continue to add partners (\( x \)) until the marginal benefits from an additional partner just offset the discounted expected disutility of contracting the disease in the future (hereafter, marginal cost). However, unlike standard expected utility maximization problems (von Neumann and Morgenstern (1944)), here the 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, \( \bar{x} \). 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). For the range of parameter values considered in this paper, the high-partner intersection of marginal benefits and costs well exceeds the upper limit \( \bar{x} \).

### 3.3 Equilibria

An equilibrium for the economic epidemiological system is characterized by a sequence of values \( \{x_t, s_t, m_t, r_t\}_{t=0}^{\infty} \) that solve the individual’s optimization problem and satisfy (2 - 4) for all \( t \), subject to the initial values \( s_0, m_0 \) and \( r_0 \). Given the complexity of the system, an analytical solution for the optimal path is not possible. Instead, we solve the steady-state conditions numerically and use standard linearization methods to evaluate the stability and transition dynamics around each steady state.\(^{10}\) We first examine the long-run equilibrium and then turn our attention to the transition path and short-run equilibrium.

\(^{10}\)Simulations were also performed on the non-linear system using GAMS. Comparisons of the results to those from the linearized system are reported in the Appendix.
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:

\[
\begin{align*}
    s & = R_0^{-1} \\
    in & = \frac{(1 - s)(\mu + \gamma)}{1 + \mu + \gamma} \\
    r & = \frac{(1 - s)}{1 + \mu + \gamma} \\
    x^{-1} & = \beta p_x (V - V^S)
\end{align*}
\]

where \( R_0 = p[\ln(1 + \mu)]^{-1} \) 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:\textsuperscript{11}:

<table>
<thead>
<tr>
<th>Parameters</th>
<th>$\beta$</th>
<th>$\gamma$</th>
<th>$\mu$</th>
<th>$a$</th>
<th>$\lambda_a$</th>
<th>$h$</th>
<th>$\bar{x}$</th>
<th>$\lambda_p$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.96</td>
<td>0.2</td>
<td>0.05</td>
<td>40</td>
<td>0.023</td>
<td>7.21</td>
<td>10</td>
<td>0.60</td>
</tr>
</tbody>
</table>

**Endemic Steady-State Values**

<table>
<thead>
<tr>
<th>$x$</th>
<th>$in$</th>
<th>$s$</th>
<th>$r$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>4.042</td>
<td>0.105</td>
<td>0.477</td>
<td>0.419</td>
<td>0.231</td>
</tr>
</tbody>
</table>

**Eradication Steady-State Values**

<table>
<thead>
<tr>
<th>$x$</th>
<th>$in$</th>
<th>$s$</th>
<th>$r$</th>
<th>$p$</th>
<th>$R_0$</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>0.000</td>
<td>1.000</td>
<td>0.000</td>
<td>0.000</td>
<td>5.714</td>
</tr>
</tbody>
</table>

In the endemic steady state, approximately 10% of the sexually active population are infected with syphilis and 42% are recovered and immune. The probability of infection with a single partner is 23%. 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

\textsuperscript{11}The calibration exercise is described in the Appendix.
approximation of the system around each steady state. The EE system collapses to

\begin{align*}
    s_{t+1} &= \mu + (1 - p_t - \mu)s_t + \gamma r_t \\
    \ln_{t+1} &= -\mu \ln_{t} + p_t s_t \\
    r_{t+1} &= (1 - \gamma - \mu)r_t + \ln_t \\
    \frac{1}{p_{x,t}^x} &= \beta E \left( \ln(x_{t+1}/\bar{x}) + h - \frac{p_{t+1}}{x_{t+1}p_{x,t+1}} \right),
\end{align*}

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{align*}
    \hat{x}_t &= [\kappa x/\ln] \ln_{t} \\
    \ln_{t+1} &= (sp_{in} - \mu) \ln_{t} + sp_{x} \hat{x}_t + p \hat{s}_t \\
    \hat{r}_{t+1} &= (1 - \gamma - \mu) \hat{r}_t + \ln_{t},
\end{align*}

where carets (\(^\)\) over variables indicate deviations from their steady-state values, \( \kappa \) is the elasticity of partner change (\( x \)) with respect to syphilis prevalence (\( \ln \)), and \( p_{in} = x\lambda_{p}(1 - \lambda_{p}\ln)^{-1} \) is the partial derivative of \( p \) with respect to \( \ln \). After substituting out the control variable \( \hat{x}_t \) and using the restriction \( \hat{s}_t + \ln_{t} + \hat{r}_t = 0 \), the system can be reduced to the following bivariate dynamic system:

\begin{align*}
    \begin{bmatrix} \ln_{t+1} \\ \hat{r}_{t+1} \end{bmatrix} &= \begin{bmatrix} \phi - (\mu + p) & -p \\ 1 & 1 - \gamma - \mu \end{bmatrix} \begin{bmatrix} \ln_{t} \\ \hat{r}_{t} \end{bmatrix},
\end{align*}

where \( \phi = s(p_{in} + p_{x}\kappa x/\ln) \) 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 (20) 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:

\[-\mu + 0.5 \left[ 1 + (\phi - \gamma - p) \pm \sqrt{1 + (p - \gamma - \phi)^2 - 2(p + \gamma + \phi)} \right]. \tag{21}\]

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, dampened cycles. Using equation (21), we see that the system displays stable cycles if the eigenvalues have modulus less than one and $2(p + \gamma + \phi) > 1 + (p - \gamma - \phi)^2$.\textsuperscript{12}

Using the parameter values in Table 1, the eigenvalues are $0.337 \pm 0.245i$, 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 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 (16). 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

\[p_t = 1 - (1 - \lambda_p in_t)^2. \tag{22}\]

\textsuperscript{12}If the eigenvalues have an imaginary part, then they come in complex conjugates $a \pm ci$ with period equal to (Hamilton (1994))

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

and persistence equal to

\[R = \text{Mod} [\psi_{1,2}] = \sqrt{a^2 + c^2}.\]
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, \( \lambda_p \).

The linearized dynamic SIRS system is

\[
\begin{bmatrix}
\hat{\text{i}}_{n+1} \\
\hat{\rho}_{t+1}
\end{bmatrix} = \begin{bmatrix}
s p_{in} - (\mu + p) & -p \\
1 & 1 - \gamma - \mu
\end{bmatrix} \begin{bmatrix}
\hat{\text{i}}_n \\
\hat{\rho}_t
\end{bmatrix}.
\]

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 (20) and (23). The two matrices differ by the \( sp_x \kappa x \) 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 \( \kappa \), 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 (23). In the EE model, \( \kappa \) can take on a range of values depending on the biological parameters and individual preferences over \( x \) and \( h \).

Following the linearization of (16), this elasticity can be expressed as

\[
\kappa = \frac{\partial x}{\partial \text{in}} \frac{\text{in}}{x} = \left[ -\frac{p_{in} \text{in}}{p_x x} \right] + \left[ \frac{p_{in} \text{in}}{1 + \ln(1 - p)} \frac{\beta}{1 + \beta p} \right] .
\]

The partner elasticity \( \kappa \) 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.\(^{13}\) 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 \( p_c = 1 - (1/e) \approx 0.63 \).\(^{14}\) This pivot is represented by the 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

\(^{13}\)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.

\(^{14}\)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 10.5 years and persistence equal to 0.85; the cyclical response continues well past the forcing shock. The EE cycles have a ten-year periodicity but are significantly dampened with persistence of 0.42. 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. However, when individuals are free to choose their number of partners and $\kappa < 0$, cycles are significantly dampened. 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

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15 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 $\nu$ will be equal across the two models.

16 For the baseline parameter values, the elasticity of partner change with respect to prevalence is $\kappa = -0.81$. Furthermore, if we hold $x$ fixed at its steady-state value, the probability of infection at the steady state is 0.23, increasing to 0.33 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 37% from 7.21 to 4.54. The new steady-state implies an increase in partners from 4.0 to 9.0, 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.\textsuperscript{17}

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

\textsuperscript{17}For the baseline parameter values, the elasticity of partner change with respect to prevalence is $\kappa = 0.28$. Furthermore, if we hold $x$ fixed at its steady-state value, the probability of infection at the steady state is 0.56, increasing to 0.67 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.\footnote{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.}

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.

We now turn our attention to the eradication of syphilis.

\section{Syphilis Eradication and AIDS Therapies}

Encouraged by historically low prevalence in the late 1990s, the CDC unveiled a formal plan to eradicate syphilis from the general population (CDC (1999)). The plan 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 plan. 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, rates of primary and secondary syphilis incidence rose after the plan’s introduction and are 81% higher in 2007 than in 2000. Why did the plan fall short of the desired objective? To answer this question, we investigate the stability properties of the EE system near eradication.

The transition matrix around the eradication steady state simplifies to

\[
\begin{bmatrix}
\bar{x} \lambda_p - \mu & 0 \\
1 & 1 - \gamma - \mu
\end{bmatrix}
\]  
(25)

with eigenvalues \((\bar{x} \lambda_p - \mu)\) and \((1 - \gamma - \mu)\).\(^{19}\) 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.\(^{20}\) Any value greater than one will cause eradication to be unstable. The critical number of partners that makes eradication stable is\(^{21}\)

\[
\bar{x}^* = \frac{1 + \mu}{\lambda_p}.
\]  
(26)

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.

Why has the eradication plan failed? The analysis above shows that when the chance of syphilis infection is minimal and the health risks of other STDs are low, eradication requires sexually active individuals to sharply reduce their number of partners. The required 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)). Prior to the discovery of drug therapies for AIDS, susceptible

\(^{19}\)To derive the transition matrix around the eradication steady state, evaluate (23) 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 \(t\).

\(^{20}\)The other eigenvalue will be less than one in magnitude because our calibrations always satisfy \(\gamma + \mu < 1\).

\(^{21}\)Alternatively, the stability threshold (26) for eradication can be interpreted in terms of the basic reproduction number \(R_0 = p(1 + \mu)\bar{x}^{-1}\), which using L’Hopital’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 \((1 + \mu)\).
individuals rationally reduced their number of partners in response to the health risks of HIV/AIDS. As shown in the Appendix, before the introduction of effective drug therapies susceptible individuals are predicted to voluntarily reduce the number of partners to less than two partners per year. This number is near the partner threshold required for syphilis eradication, implying that syphilis eradication was indeed feasible. However, the introduction of effective drug therapies for AIDS has encouraged sexually active individuals to take more risk and stifle the efforts of the 1999 syphilis eradication campaign (Boily et al. (2005)).

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. 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. For example, our model predicts that the recent demographic shift in syphilis infections toward men that have sex with men (MSM) may amplify syphilis cycles, to the extent that the MSM group is practicing riskier sexual behavior. The MSM demographic shift also implies a higher rate of HIV incidence among the MSM group because syphilis infections sharply increase the likelihood of contracting HIV.

One of the more striking predictions of the integrated economic-epidemiological framework is
that syphilis eradication is now nearly impossible. While the discovery of new drug therapies has drastically improved the quality of life for those infected with AIDS, it also has the unintended consequence of encouraging more risk taking among the sexually active population. In 1999 when the syphilis eradication plan was introduced, susceptible individuals were rationally reducing their number of partners in response to AIDS risk, making it more likely that syphilis could be eradicated from the population. Now, individuals have the opportunity to lead long and productive lives while infected with AIDS. Rational individuals react by taking more sexual partners, implying that the window of opportunity to eradicate syphilis has likely closed.

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

Figure 2. Flow Chart for Syphilis Dynamics

Reproduced from Garnett et al. (1997).
Figure 3. Phase Diagram for the EE System

The parameter values are set at $\beta = 0.96$, $\gamma = 0.2$, $\lambda_a = 0.023$, $a = 40$, $\mu = 0.05$, $h = 7.21$ 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 $x$. 

Notes. The parameter values are set at $\beta = 0.96$, $\gamma = 0.2$, $\lambda_a = 0.023$, $a = 40$, $\mu = 0.05$, $h = 7.21$ 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 $x$. 

"Economics" Locus: $x(t+1) - x(t) = 0$

"Epidemiology" Locus: $in(t+1) - in(t) = 0$
Figure 4. Individual Optimal Choice and the Probability of Infection: Exogenous Increase in Prevalence

Panel A. Rational Dynamic Dampening (high $h$)

Panel B. Rational Dynamic Resonance (low $h$)
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 $\beta = 0.96$, $\mu = 0.05$, $\gamma = 0.2$, $h = 7.21$, $\lambda_a = 0.023$, $a = 40$ 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 $\beta = 0.96$, $\mu = 0.05$, $\gamma = 0.2$, $h = 4.54$, $\lambda_a = 0.023$, $a = 40$, 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.