DISCUSSION PAPER SERIES

No. 8112

RECURRENT INFECTION AND EXTERNALITIES IN PREVENTION

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INDUSTRIAL ORGANIZATION

Centre for Economic Policy Research

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ISSN 0265-8003

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November 2010

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CEPR Discussion Paper No. 8112

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ABSTRACT

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JEL Classification: C73 and I18
Keywords: economic epidemiology, preventive behavior, rational disinhibition and risk compensation

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* I am grateful to Robert Rowthorn, Joshua Ross, Frederick Chen, Mark Gersovitz, Peyton Young, Drew Fudenberg, Chryssi Giannitsarou, Christopher Harris, Tomas Philipson, Larry Samuelson, Jakub Steiner, Jess Benhabib, David Levine, Rakesh Vohra, David Bardey, Michael Mandler, David Myatt and Habtu Weldegebriel for constructive conversations and feedback. I also thank seminar participants at Columbia University, Wake Forest University and participants at the 2009 Annual Meetings of the American Economic Association, San Francisco and at the Journées Louis-André Gérard-Varet, Marseille (2010).

Submitted 02 November 2010

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1. Introduction

Since 1981, the AIDS epidemic has claimed more than 25,000,000 lives and ruined the livelihood of innumerably more.[1] In a historical perspective, these magnitudes are not unusual. In 1348-1350, the bubonic plague (the Black Death) is estimated to have caused the death of up to a third of Europe's total population of 100,000,000 while the influenza epidemic (the Spanish flu) in 1918-1920 claimed a world death toll estimated at 50,000,000.[2] In more recent times, the advent of bird flu and the ongoing swine flu pandemic have served as salutary reminders that communicable diseases are of first order importance as a matter of public policy and constitute a promising and worthwhile field of inquiry for economists and behavioral scientists alike.

While the study of infectious diseases has traditionally belonged to the domain of biology and medical science, policy and welfare concerns have always been a raison d'être

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[1] See UNAIDS 2008 Report on the Global AIDS Epidemic.

[2] See Langer (1970) and Johnson and Mueller (2002).

of epidemiological inquiry. In motivating his formal analysis of infectious diseases, the father of mathematical epidemiology, Daniel Bernoulli (1766) stated that

> "I simply wish that, in a matter which so closely concerns the well-being of mankind, no decision shall be made without all the knowledge which a little analysis and calculation can provide."

In a standard text on epidemiology, Daley and Gani (2001) state that

> "One of the purposes of modelling epidemics is to provide a rational basis for policies designed to control the spread of a disease."

Despite this stated goal, classical epidemiology has been largely devoid of behavioral considerations, a simplification which may be more appropriate for the study of animal populations than human ones. In the present work, I follow the literature on economic epidemiology in redressing this shortcoming by considering a model in which individuals in a closed population, through contact with other individuals, are exposed to an infectious disease. While recovery from infection is possible, the rate at which this occurs is beyond the influence of the individuals. On the other hand, individuals can influence the rate at which they are infected by engaging in privately costly preventive behavior. Importantly, in order to effectively prevent infection, this preventive behavior must be sustained through time (as in the use of condoms).

Formally, I analyze a simple economic extension of the classical *susceptible-infected-susceptible* (or SIS) model in which agents may recover from infection to become susceptible to future infection. After briefly reviewing the classical framework and describing the economic setting, I consider two different scenarios. First, I derive the optimal policy in a centralized setting where a benevolent social planner dictates individual exposure levels. This analysis serves as a benchmark for the analysis of the equilibrium set of the second scenario, namely a decentralized setting in which individuals act non-cooperatively in a strategically sophisticated manner. I also derive a number of important comparative statics results and relate these to concrete welfare and policy questions.

The main results are as follows. With decentralized decision making, optimal play is necessarily *Markovian*, *stationary* and *myopic* on the transition path to steady state. This is because of the fact that there are no contemporaneous externalities on the transition path, strategic or otherwise and because agents cannot commit to future preventive effort. In choosing the (privately) optimal exposure level at some point in time, all that matters to an agent is the level of disease prevalence at that same instant, since this is what determines the costs and benefits of exposure at that moment.[3] On the transition path, players use pure strategies in equilibrium, choosing full exposure for sufficiently low disease prevalence and no exposure for sufficiently high prevalence. In steady state, players necessarily use mixed (or interior) strategies. As a result, the unique steady state is necessarily interior, i.e. infection is endemic and is never eradicated. Although there are no contemporaneous externalities on the transition path, in steady state there are strategic substitutabilities and therefore subsidies to the preventive effort of some agents will tend to crowd out the preventive effort of others.

[3] As shall be discussed in more detail later, this is a main factor differentiating this type of preventive behavior from vaccination.

With centralized decision making, the planner chooses a positive amount of infection in steady state and a positive level of aggregate exposure, akin to the mixed strategy equilibrium of the decentralized setting. Importantly though, the planner chooses a strictly lower level of exposure and steady state infection level than do individuals in the non-cooperative equilibrium. This implies that steady state welfare under decentralized decision making is lower than the first-best level. This is an outcome of the fact that individual decision makers do not internalize the positive externalities that their preventive effort has on the rest of the population.

There is an important exception to these findings, namely the scenario in which there is no possibility of recovery from the disease and the model reduces to that of a *simple epidemic* (also known as the SI model). In this special case, all differences between the decentralized equilibrium outcomes and the first-best centralized solution vanish. The predictions of this special case are similar to those derived by Geoffard and Philipson (1996), although they do not distinguish between centralized and decentralized decision making or perform explicit welfare analysis. There is also a curious relation to results on optimal vaccination by Francis (1997), who shows that in homogeneous populations, there are no externalities in vaccination decisions in equilibrium.[4]

There are a number of natural questions to ask of such an analysis. First, what's the nature of externalities and to what extent do these influence strategic interaction? Second, what are the equilibrium and welfare implications of these externalities? Third, given that the decentralized equilibrium outcome is socially sub-optimal, which policy measures can and should be considered? Possibilities include subsidies to preventive behavior (such as condom use) or measures that reduce the infectivity of the disease, such as pre-exposure or post-exposure prophylaxis (PrEP or PEP), which are known to effectively reduce the rate of transmission of the HIV virus.

In addressing these questions, a number of important policy implications ensue:

(i) The differences between the centralized outcome and the decentralized equilibrium are entirely parametrized by the rate of spontaneous recovery. For diseases such as HIV/AIDS where recovery is presently not possible, the non-cooperative equilibrium is shown to be first-best optimal. That is, in both settings, full exposure is optimal until a critical measure of infected individuals is reached. At that point, the optimal policy switches and full prevention becomes optimal forever after and no further infection takes place. When recovery is possible, the decentralized equilibrium is inefficient as individuals do not internalize the (beneficial) effects of their preventive effort on other (current and future) susceptible individuals.

(ii) Conditional on wanting to bring down infection, subsidies to prevention may not be cost-effective as they partially crowd out existing preventive measures.[5] This is an effect of preventive behavior being strategic substitutes in steady state. Instead, one may want to subsidize treatment, as therapeutic measures are strategic complements and a subsidy to an individual's treatment may make other individuals more inclined to treating themselves (see Toxvaerd, 2009).[6]

[4] The relation of the present work to this type of result will be elaborated in depth in Section 6.

[5] This feature was also noted by Geoffard and Philipson (1997) in a model of vaccination.

[6] But note that this argument compares a model where only protection is available with a model in which only treatment is available. Also, note that the steady states of these two models in general differ. For a model in which both policies coexist in a representative agent setup, see Gersovitz and Hammer (2004).

(iii) Infectivity-reducing policies such as the introduction of PrEP and PEP unambiguously lead to lower steady state welfare under decentralized decision making. The same is true under centralized decision making for sufficiently low recovery rate or for sufficiently high discount rate. The reason is as follows. When infectivity becomes lower, exposure becomes less risky and therefore its level is optimally increased. As a consequence, steady state disease prevalence increases which in turn leads to lower steady state welfare. The upshot of this result is that in order for overall welfare to increase due to lower infectivity, substantial welfare gains must accrue during the transition to the new (and higher) steady state level of disease prevalence. Since a social planner can always induce the same path of disease prevalence as before the introduction of such measures but at a lower cost, overall welfare cannot be reduced. Thus all benefits of infectivity reduction stem solely from the benefits of increased exposure that accrue on the path towards the new steady state. Once in steady state, welfare is lower than in the original (and lower) steady state.

In the decentralized setting, the overall effects on welfare are ambiguous. In this model, there is *rational disinhibition*, which makes higher exposure to infection an individually rational response to decreased infectivity. But since all individuals find it privately optimal to increase exposure, aggregate exposure may increase to an extent that is detrimental to aggregate welfare.

(iv) Continual preventive measures are not the right tool to eradicate a disease. In fact, were prevention the only available measure against infection, then it would never be optimal to eradicate the disease (as long as prevention is costly). Therefore, in order to eradicate a disease one must do so through alternative measures, e.g. through treatment and/or vaccination (if available).

1.1. Related Literature. The existing theoretical literature on economic epidemiology falls into two broad categories. First, there is a relatively small literature that considers different models of treatment, including Sanders (1971), Sethi (1974), Sethi and Staats (1978) Goldman and Lightwood (1995, 2002), Rowthorn (2006) and Toxvaerd (2009a,b). Second, and more directly related to the present analysis, is the larger and growing literature on the effects and desirability of preventive measures such as quarantines, prophylaxis, vaccines and promiscuity/partner change. Main contributions include Sethi (1978), Geoffard and Philipson (1996, 1997), Kremer (1996), Auld (2003), Aadland et al. (2010), Francis (1997, 2004), Gersovitz and Hammer (2004), Boulier et al. (2007), Brito et al. (1991), Barrett (2003) and Reluga (2009).

Last, there is an important body of work on the empirics of infectious disease, such as that on prevalence elasticity, partner choice etc. These include Ahituv et al. (1996), Philipson (2000), Auld (2006), Gibbison (2006), Dupas (2007) and Oster (2005, 2007).

Especially worthy of mention are the contributions by Brito et al. (1991), Geoffard and Philipson (1997), Goldman and Lightwood (1995, 2002), Francis (1997, 2004) and Gersovitz and Hammer (2004) who all explicitly consider decentralized decision making and compare the outcomes with that chosen by a central planner. The aim of this paper is to contribute to this strand of literature by considering a model of recurrent infection that is tractable enough to make sharp analysis and conclusions and to contribute to the ongoing policy debate on the management of infection diseases.

Although the aforementioned papers all study interventions that fall under the broad category of "preventive measures" that may seem superficially similar, there are very

important differences between them in terms of predictions and policy implications. It is therefore of crucial importance for practical applications to understand the delicate relations between them. In models of abstinence, such as those studied by Kremer (1996) and Auld (2003), agents' decisions are whether to enter the pool (or market) for sexual interaction. In such models, there are strategic complementarities. This is because when a susceptible individual chooses to enter the pool, he/she reduces the probability that other susceptible individuals become infected from entering the pool. But this makes entering the pool a more appealing proposition, thereby reinforcing the decision to enter the pool. Strategic complementarities of this sort may create multiple steady states.

Anther type of protective measure is vaccination, which are commonly thought to lead to an environment of strategic substitutes. The conventional thinking is that when one individual is vaccinated, he/she cannot pass on the disease to other individuals, thereby obviating the need for them to vaccinate. This type of thinking may in part stem from the lessons of static analyses such as those by Brito et al. (1991) and Barrett (2003), who shows that under decentralized decision making, there is under-vaccination compared to the first-best. As shown in a striking paper by Francis (1997), once a fully dynamic setting is considered, externalities may disappear altogether. Similarly surprising, when vaccines are imperfect, vaccination may become strategic complements, as shown by Chen and Cottrell (2009). Another related paper is that by Geoffard and Philipson (1997), which discusses the desirability and feasibility of subsidizing vaccines in order to align private and public incentives. Last, in models of continual prevention without acquired immunity, such as the homogeneous susceptible-infected type model studied by Geoffard and Philipson (1996), there are no externalities at all. This point will be elaborated further when the special case of the present model in which there is no recovery is discussed.

In Section 2, I set out the classical and economic models and briefly summarize the classical results. In Section 3, I analyze the problem faced by a benevolent central planner, the solution of which serves as a benchmark. In Section 4, I analyze the equilibrium under non-cooperative decentralized decision making. In Section 5, I discuss welfare, derive comparative statics results and draw policy conclusions. Section 6 contains a detailed comparison of the present model and related dynamic models of vaccination. In Section 7 I conclude.

2. THE CLASSICAL AND ECONOMIC MODELS

To make the exposition self-contained, I start by expounding the classical epidemiological version of the model in some detail. This will not only aid in understanding the economic models that follow, but also highlight the contrast in predictions based on the separate modeling approaches.

The classical susceptible-infected-susceptible model is simple to describe.[7] Time is continuous and runs indefinitely. A population $\mathcal{P} = [0, 1]$ consists of a continuum of infinitely lived individuals who can at each instant t each be in one of two states, namely *susceptible* or *infected*. The set of infected individuals is denoted by $\mathcal{I}(t)$ and has measure $I(t)$, while the set of susceptible individuals is denoted by $\mathcal{S}(t)$ and has measure $S(t)$. Because the population size has been normalized to unity, these measures can be

[7]See Anderson and May (1991), Daley and Gani (2001) or Keeling and Rohani (2008) for good introductions and applications.

interpreted as fractions. Henceforth, $I(t)$ shall be referred to as *disease prevalence*.

At each instant, the population mixes homogeneously. This corresponds to pairwise random matching where each individual has an equal chance of meeting any other individual, irrespective of the health status of the two matched individuals. Whereas a match between two infected individuals or two susceptible individuals does not create any new infection, a match between an infected and a susceptible individual may. The rate at which infection is transferred in such a match is denoted by $\beta > 0$. This parameter captures the infectivity of the disease. Coupled with the assumption of homogeneous mixing, this means that the rate at which susceptible individuals become infected is given by the simple expression $\beta I(t)S(t)$. Thus the rate of new infection, or *disease incidence*, is proportional to disease prevalence.[8] Note that while disease incidence is a flow value, disease prevalence is a stock value.

Infected individuals recover spontaneously at rate $\alpha \geq 0$. This means that the rate at which infected individuals become susceptible is given by $\alpha I(t)$. The dynamics of the model are thus described by the following system of differential equations:

$$\dot{S}(t) = I(t)\left[\alpha - \beta S(t)\right] \tag{1}$$
$$\dot{I}(t) = I(t)\left[\beta S(t) - \alpha\right] \tag{2}$$
$$I(t) = 1 - S(t), \quad I(0) = I_0 \tag{3}$$

Using the normalization, this system reduces to the following simple logistic growth equation:

$$\dot{I}(t) = I(t)\left[\beta(1 - I(t)) - \alpha\right], \quad I(0) = I_0 \tag{4}$$

The steady states of this system are

$$I^* = 0, \quad I^* = \frac{\beta - \alpha}{\beta} \tag{5}$$

For $\beta > \alpha$, the stable steady state is endemic (or persistent), while for $\beta < \alpha$, the stable steady state involves eradication. In other words, if the rate at which individuals become infected surpasses the rate at which they recover, then some positive fraction of the population will always be infected. If recovery is not possible, the entire population ends up being infected. On the other hand, if individuals recover at a higher rate than the rate at which they become infected, then the disease eventually dies out. This completes the description of the classical SIS model.

For later use, note the following comparative statics results. Increasing the recovery rate α decreases the endemic steady state level of infection I^* while increasing the infectivity rate β increases it.

At the aggregate level, there is no uncertainty and thus the probability that a randomly chosen individual is infected must coincide with the fraction of infected individuals. From the perspective of an infected individual, the transition to susceptibility is governed by a Poisson process with rate α, which is memoryless. Similarly, for a fixed level of ag-

[8]The term $\beta I(t)S(t)$ should be thought of as the rate at which susceptible individuals have contact with other individuals, multiplied by the probability of the contact being with an infectious individual, multiplied by the probability that the infection is transmitted in such a contact. See e.g. Keeling and Rohani (2008) for a detailed derivation.

gregate infection $I(t)$, the transition to infectivity for a susceptible individual is governed by a Poisson process with rate $\beta I(t)$. Thus transition probabilities are memoryless, a fact that greatly simplifies the analysis that follows.

2.1. Extension to an Economic Model. To turn the classical model into a fully fledged economic model, I will proceed by assigning payoffs to the different disease states and assume that time is discounted. Specifically, I will assume that individuals earn a flow payoff $\pi_{\mathcal{S}}$ per instant while susceptible and $\pi_{\mathcal{I}} < \pi_{\mathcal{S}}$ per instant while infected and that time is discounted at rate $\rho > 0$. For notational simplicity, let the quantity $\pi \equiv \pi_{\mathcal{S}} - \pi_{\mathcal{I}} > 0$ denote the health premium. The health premium should be thought of broadly as the benefits of not being infected, e.g. physical well-being, labor income and social esteem.

To model the possibility of engaging in preventive behavior, assume that the individuals can affect the rate of infection by controlling the rate at which they expose themselves to infection. In particular, at each instant t, each individual $i \in \mathcal{S}(t)$ non-cooperatively chooses exposure level $\varepsilon_i(t) \in [0,1]$, at personal cost $(1-\varepsilon_i(t))c \geq 0$.[9] Effectively, this reduces the rate of infection to $\varepsilon_i(t)\beta I(t)$. This formalization captures the notion that, ceteris paribus, exposure is desirable. Equivalently, this means that engaging in preventive behavior is privately costly. This completes the description of the economic version of the SIS model.

Before I analyze the economic model in more detail, it should be mentioned that the classical model presented here has a number of simplifying assumptions that are inherited by the economic version of the model. First, there is only one disease and one level (or severity) of infection.[10] In particular, this rules out the possibility of superinfection by different strains of the disease. Second, the moment an individual is infected coincides with the onset of symptoms such as the welfare loss brought about by infection (i.e. the *incubation period* has zero length), so no infected individual acts under the mistaken belief that he or she is susceptible. Last, once an individual becomes infected, he or she immediately becomes infectious to other individuals (i.e. the *latency period* has zero length).

For later use, the following assumptions are made:

Assumption 1

$$c < \pi\left(\frac{\beta}{\alpha+\beta+\rho}\right) \tag{6}$$

This assumption, which bounds the cost of prevention, ensures that prevention is potentially desirable from the perspective of both the planner and the individuals. Next, I will assume that

Assumption 2

$$\alpha < \beta \tag{7}$$

[9] This is formally equivalent to choosing protection level $p(t) \in [0,1]$ at cost $p(t)c$ which reduces the infection rate to $(1-p(t))\beta I(t)$.

[10] Thus the sets $\mathcal{S}(t)$ and $\mathcal{I}(t)$ are disjoint and exhaust $\mathcal{P}$.

This assumption implies that the eradication steady state is unstable and the relevant steady state is the endemic one. Note that if $\alpha > \beta$, then the infectious disease is eventually eradicated even if no-one engages in any preventive behavior at all. The only possible role for preventive measures would then be to speed up the inevitable eradication.

3. Centralized Decision Making

In the centralized setup, a benevolent planner seeks to maximize the sum of the individuals' expected, discounted lifetime utilities through the direct control of the aggregate level of exposure $\varphi(t) \in [0,1]$. In solving this problem, the planner explicitly takes into account the fact that he influences aggregate disease incidence and prevalence through the control of aggregate exposure. This will turn out to be a central difference between the scenarios with centralized and decentralized decision making respectively, since each individual in the population is too small to influence the aggregate.

Formally, the planner's problem is to solve the following programme[11]:

$$\max_{\varphi(t)\in[0,1]} \int_0^\infty e^{-\rho t}[I(t)\pi_{\mathcal{I}} + S(t)\left[\pi_{\mathcal{S}} + (1-\varphi(t))c\right]]dt \tag{8}$$

$$s.t. \quad \dot{I}(t) = I(t)\left[\varphi(t)\beta S(t) - \alpha\right], \quad I(0) = I_0 \tag{9}$$

The constraint on the planner's problem is simply the logistic growth equation for the measure of infected individuals, suitably modified to take into account that the rate at which infection occurs is a function of the centrally chosen exposure level.

Instead of solving this problem, the following simplified but equivalent programme will be considered (which differs only by the constant $\pi_{\mathcal{S}}$):

$$\max_{\varphi(t)\in[0,1]} \int_0^\infty e^{-\rho t}\left[-I(t)\pi - (1-I(t))(1-\varphi(t))c\right]dt \tag{10}$$

$$s.t. \quad \dot{I}(t) = I(t)\left[\varphi(t)\beta(1-I(t)) - \alpha\right], \quad I(0) = I_0 \tag{11}$$

An admissible policy is a pair of functions $(I(t), \varphi(t))$ in which for all $t \geq 0$, $I(t)$ satisfies the logistic growth equation and where $\varphi(t) \in [0,1]$. Furthermore, $\varphi(t)$ must be piecewise continuous.

To solve the problem, let $\lambda(t)$ denote multiplier on the constraint. The multiplier is required to be piecewise continuously differentiable. The current-value Hamiltonian is then given by

$$\begin{aligned} H^C \equiv & -I(t)\pi - (1-I(t))(1-\varphi(t))c \\ & +\lambda(t)I(t)\left[\varphi(t)\beta(1-I(t)) - \alpha\right] \end{aligned} \tag{12}$$

The evolution of the multiplier is described by the differential equation

$$\dot{\lambda}(t) = \lambda(t)\left[\rho + \alpha + \varphi(t)\beta\left(2I(t)-1\right)\right] + \left[\pi - (1-\varphi(t))c\right] \tag{13}$$

Differentiating the current-value Hamiltonian with respect to $\varphi(t)$ (and supposing that

[11] Note that both the centralized and decentralized problems are autonomous and thus along any optimal path, it must be that the state variable is monotone in time (see Hartl, 1987).

$I(t) < 1$) yields the following necessary condition for optimality:

$$\lambda(t)\beta I(t) + c = 0 \tag{14}$$

The optimal policy for the planner is of the bang-bang type and given by

$$\varphi(t) = 0 \quad for \quad -\lambda(t)\beta I(t) > c \tag{15}$$
$$\varphi(t) \in [0,1] \quad for \quad -\lambda(t)\beta I(t) = c \tag{16}$$
$$\varphi(t) = 1 \quad for \quad -\lambda(t)\beta I(t) < c \tag{17}$$

This policy simply states that when the marginal cost of prevention is lower than the marginal benefit, then the planner fully prevents infection, i.e. he sets the exposure level to zero. Similarly, when the marginal benefit of prevention is lower than the marginal cost, then full exposure is optimal. Last, when the two coincide, then any policy yields the same welfare and hence is optimal.

To characterize the singular steady state, I set $\dot{I}(t) = \dot{\lambda}(t) = 0$ and use the indifference condition to get the following steady state values:

$$\varphi_C^* \equiv \frac{\alpha(\pi - c)}{\beta\pi - (\beta + \rho)c} \tag{18}$$
$$I_C^* \equiv \frac{c\rho}{\beta(\pi - c)} \tag{19}$$
$$\lambda_C^* \equiv \frac{-(\pi - c)}{\rho} \tag{20}$$

This steady state is saddle path stable.[12]

As the following result shows, there is no boundary solution to the planner's problem:

Proposition 1: The centralized problem has a unique steady state, which is interior.

Proof: See Appendix A ■

This result has a straightforward intuition. When enough individuals are infected, the probability that an unprotected individual will become infected is so high that the cost of prevention is outweighed by the expected welfare loss of becoming infected. Similarly, when only a few individuals are infected, the probability of becoming infected is too low to warrant engaging in costly preventive behavior. Thus the optimal policy always forces disease prevalence towards the interior of its domain.

Having characterized the unique steady state, all that remains is to characterize the optimal path towards the steady state. It takes a particularly simple form, as the next result shows:

[12] The eigenvalues of the Jacobian are given by $\frac{\rho(-\beta\pi + c(\rho+\beta-\alpha))}{-\beta\pi + c(\rho+\beta)}$ and $\frac{\alpha\rho c}{-\beta\pi + c(\rho+\beta)}$ respectively. Under Assumption 1, these are always of opposite sign.

Proposition 2: The optimal path is of the most rapid approach type and given by

$$\varphi_C^* = 0 \quad for \quad I(t) > I_C^* \tag{21}$$

$$\varphi_C^* = \frac{\alpha(\pi - c)}{\beta\pi - (\beta + \rho)c} \quad for \quad I(t) = I_C^* \tag{22}$$

$$\varphi_C^* = 1 \quad for \quad I(t) < I_C^* \tag{23}$$

Proof: See Appendix B ■

This result follows since the necessary conditions for the optimality of a nearest approach path in Sethi (1977, Theorem 3.1) are met.[13]

Thus whenever disease prevalence is above the steady state level, the planner optimally reduces exposure to zero until the steady state level is reached. At this point, the planner switches to partial exposure so as to maintain the steady state disease prevalence. Similarly, whenever disease prevalence is below the steady state level, the planner optimally chooses full exposure until disease prevalence has increased to its steady state level, which is subsequently maintained through partial exposure.

4. Decentralized Decision Making

In this section, the decentralized problem is analyzed. I will proceed by analyzing the problem of an individual agent and then aggregate across the entire population. From the perspective of an individual, the path of disease prevalence is exogenously given.[14] It will be assumed that each individual has perfect foresight in the sense that conjectures about the evolution of the disease are confirmed in equilibrium.

For some fixed level of exposure ε and initial infection state Q_0, the individual's health state evolves according to a two-state continuous-time Markov process with the following transition rate matrix:

$$\mathbb{T} = \begin{pmatrix} -\varepsilon\beta I(t) & \varepsilon\beta I(t) \\ \alpha & -\alpha \end{pmatrix} \tag{24}$$

Let $Q(t) \in [0,1]$ be the probability that the individual is infected at instant t. This probability will be used as the state variable in the individual's optimal control problem and its evolution is governed by the rates in the transition rate matrix $\mathbb{T}$ (which in turn is a function of the strategies adopted by the individual and the population as a whole).

In what follows, I consider the simplest stationary Markov strategies $\varepsilon : [0,1] \to [0,1]$ in which each individual at each instant conditions his exposure level on the level of disease prevalence (while suitably taking into account expectations about the future evolution of the disease). A given individual's problem is therefore as follows:[15]

$$\max_{\varepsilon_i(t)\in[0,1]} \int_0^\infty e^{-\rho t} \left[-Q(t)\pi - (1 - Q(t))(1 - \varepsilon_i(t))c\right] dt \tag{25}$$

$$s.t. \quad \dot{Q}(t) = \varepsilon_i(t)\beta I(t)(1 - Q(t)) - \alpha Q(t), \quad Q(0) = Q_0 \tag{26}$$

[13] See also Hartl and Feichtinger (1987) for a more general result.

[14] Because each agent is negligible and does not influence the aggregate, any feedback between an individual's action and other individuals' responses can be ignored by the individual. This effectively circumvents a major complication of analyzing continuous time games.

[15] This objective is a simplification similar to that used in the centralized setting.

For an infected individual at instant t, $Q(t) = 1$ and thus there is no decision to be made. For a susceptible individual at instant t, $Q(t) = 0$ and hence the individual trades off costs and benefits of controlling the rate of transition from $\mathcal{S}(t)$ to $\mathcal{I}(t)$. In equilibrium, clearly it must be the case that $Q(t) = I(t)$. I.e. in a symmetric equilibrium, the probability that a given individual is infected must coincide with the fraction of infected individuals in the population.

The above problem is solved for each individual on the background of the aggregate evolution of the infectious disease. This is in turn described by the following modified logistic growth equation:

$$\dot{I}(t) = I(t)\left[\boldsymbol{\varepsilon}(t)\beta(1 - I(t)) - \alpha\right], \quad \boldsymbol{\varepsilon}(t) \equiv \int_{i\in\mathcal{S}(t)} I(t)^{-1}\varepsilon_i(t)di \tag{27}$$

In this equation, $\boldsymbol{\varepsilon}(t)$ denotes the aggregate level of exposure resulting from the susceptible individuals' disaggregate exposure levels. Let $\mu(t)$ be the multiplier on the constraint in the agent's problem.[16] The current-value Hamiltonian for the individual's problem is then given by

$$\begin{aligned} H^D \equiv\ & -Q(t)\pi - (1 - Q(t))(1 - \varepsilon_i(t))c \\ & +\mu(t)\left[\beta I(t)(1 - Q(t))\varepsilon_i(t) - \alpha Q(t)\right] \end{aligned} \tag{28}$$

The evolution of the multiplier is in turn governed by the differential equation

$$\dot{\mu}(t) = \mu(t)\left[\rho + \alpha + \varepsilon_i(t)\beta I(t)\right] + \left[\pi - (1 - \varepsilon_i(t))c\right] \tag{29}$$

Differentiating the current-value Hamiltonian with respect to $\varepsilon_i(t)$ (and supposing that $Q(t) < 1$) yields the following necessary condition for optimality:

$$\mu(t)\beta I(t) + c = 0 \tag{30}$$

The optimal policy can thus be characterized as follows:

$$\varepsilon_i(t) = 0 \quad for \quad -\mu(t)\beta I(t) > c \tag{31}$$

$$\varepsilon_i(t) \in [0, 1] \quad for \quad -\mu(t)\beta I(t) = c \tag{32}$$

$$\varepsilon_i(t) = 1 \quad for \quad -\mu(t)\beta I(t) < c \tag{33}$$

Again, $-\mu(t)\beta I(t)$ is to be interpreted as the marginal benefit of prevention while, c is the corresponding marginal cost. In words, the optimal policy is to fully expose oneself to infection, if the marginal cost of doing so outweighs the marginal benefit and to fully protect, if the marginal cost is outweighed by the marginal benefit. In case of indifference, any level of prevention will do.

[16] An admissible policy for an individual is a pair of functions $(Q(t), \varepsilon_i(t))$ in which for all $t \geq 0$, $Q(t)$ satisfies the differential equation constraint and $\varepsilon_i(t) \in [0, 1]$ and is piecewise continuous. Furthermore, the multiplier $\mu(t)$ is required to be piecewise continuously differentiable.

An individual's steady state conditions imply that

$$Q^* = \frac{\beta I(t)\varepsilon_i(t)}{\beta I(t)\varepsilon_i(t)+\alpha} \tag{34}$$

$$\mu^* = \frac{-\pi+(1-\varepsilon_i(t))c}{\alpha+\rho+\beta I(t)\varepsilon_i(t)} \tag{35}$$

Thus the individual's problem can only be in steady state if the aggregate system is too and vice versa. That is, $\dot{Q}(t)=\dot{\mu}(t)=\dot{I}(t)=0$ in steady state. But the individual's problem can reach a "stationary state" for any number of different (constant) levels of disease prevalence $I(t)$.

To characterize the singular solution, note that $\boldsymbol{\varepsilon}(t)=\varepsilon_i(t)$ for all $i\in\mathcal{S}(t)$ in symmetric equilibrium. Setting $\dot{\mu}(t)=\dot{I}(t)=0$ and using the indifference condition then yields the steady state values:

$$\varepsilon_D^* \equiv \frac{\alpha(\pi-c)}{\beta\pi-c(\alpha+\beta+\rho)} \tag{36}$$

$$I_D^* \equiv \frac{c(\alpha+\rho)}{\beta(\pi-c)} \tag{37}$$

$$\mu_D^* \equiv \frac{-(\pi-c)}{\alpha+\rho} \tag{38}$$

At the steady state level of disease prevalence I_D^*, the agents are willing to use mixed strategies. Furthermore, the strategies ε_D^*, if followed by all, yields the steady state disease prevalence I_D^*. It should be noted that the equilibrium is saddle-path stable.[17] The singular (aggregate) solution just derived fully characterizes the set of steady states under decentralized decision making. That this is the case follows from the next result, which rules out any further steady states:

Proposition 3: The decentralized problem has a unique steady state, which is interior.

Proof: See Appendix A ■

To complete the description of the decentralized equilibrium, the equilibrium path towards the steady state must be characterized. As in the centralized setup, optimal paths take a very simple form, as shown next.

Proposition 4: In the unique decentralized equilibrium, for all times $t\geq 0$ and individuals $i\in\mathcal{S}(t)$, equilibrium strategies are given by

$$\varepsilon_i^* = 0 \quad for \quad I(t)>I_D^* \tag{39}$$

$$\varepsilon_i^* = \frac{\alpha(\pi-c)}{\beta\pi-c(\alpha+\beta+\rho)} \quad for \quad I(t)=I_D^* \tag{40}$$

$$\varepsilon_i^* = 1 \quad for \quad I(t)<I_D^* \tag{41}$$

Proof: See Appendix B ■

[17] The eigenvalues of the Jacobian are given by $\alpha+\rho+\beta I(t)\varepsilon(t)>0$ and $-\alpha-\beta I(t)\varepsilon(t)<0$ respectively.

Akin to the optimal policy in the centralized setup, in the decentralized equilibrium individuals play pure strategies outside of steady state in order to approach the singular solution as rapidly as possible. Once there, the individuals switch to mixed strategies to maintain the steady state level of disease prevalence. The proof follows similar steps as those in the centralized setup and is thus omitted.

For reasons that parallel those in the centralized setup, the steady state is necessarily interior under decentralization. But because individuals do not internalize the effects that their preventive behavior has on other individuals, the centralized and aggregate decentralized steady state policies and disease prevalences differ.

4.1. Externalities on the Equilibrium Path. Before further characterizing the implications of the model, I will briefly discuss the issue of strategic externalities. As shown above, the only thing that determines an agent's equilibrium exposure at a given point in time, is the probability rate of infection associated with exposure at that given moment. Other agents' past, present or future behavior is entirely irrelevant. The future plays no role because agents reoptimize at each instant and have no ability to commit to future preventive behavior.[18]

Next, note that past behavior of other agents only feeds through to the present decisions through their effect on the present level of disease prevalence. The exact history is immaterial and all that matters is the present probability rate of infection. Last, present behavior by other agents is irrelevant because from the perspective of an individual agent, all that matters is the fraction of the population that is infected, not how the fraction of susceptible individuals is distributed between protected and non-protected individuals. This is because at a given point in time, no susceptible individual can transmit the disease, whatever his level of exposure.[19] Of course, other agents' past and present decisions do influence a given individual's *future* choices.

In contrast to the lack of externalities on the transition path, in steady state the game is one of strategic substitutes in the sense that the more others protect themselves against infection, the lower is the incentive to protect oneself. As a consequence, the players necessarily use mixed strategies in steady state, i.e. they randomize between full prevention and full exposure. To see this, consider an individual agent and denote by $\boldsymbol{\varepsilon}(t)$ a measure of aggregate exposure, such as the average across the susceptible population at time t. From the indifference condition of the agent, I will determine how the quantity

$$-\beta I(t)\mu(t) \tag{42}$$

varies with $\boldsymbol{\varepsilon}(t)$, where

$$I(t) = \frac{\beta\boldsymbol{\varepsilon}(t) - \alpha}{\beta\boldsymbol{\varepsilon}(t)}, \quad \mu(t) = \frac{-\pi + (1 - \varepsilon_i(t))c}{\alpha + \rho + \beta I(t)\varepsilon_i(t)} \tag{43}$$

These are the relevant state and costate values in a symmetric steady state equilibrium. Direct inspection of the derivative of the benefit from prevention with respect to $\boldsymbol{\varepsilon}(t)$ shows that the sign is positive under assumption (6). In other words, the more others expose themselves, the less inclined will the remaining agent be to expose him or herself.

[18] This is in sharp contrast with vaccination, which will be discussed in further detail below.

[19] This discussion is similar to that found in Francis' (1997) analysis of the work by Brito et al. (1991).

5. Welfare, Policy and Rational Disinhibition

In this section, I call on the results of the previous sections to draw implications about welfare and public policy towards preventive measures. The relative simplicity of the model makes it straightforward to derive comparative statics results and to conduct welfare analysis and arrive at some interesting and surprising conclusions. The first important result follows directly from the steady state level of disease prevalence of the planner's problem:

Result (a) For any recovery rate $\alpha \geq 0$, eradication through continual prevention is suboptimal.

This result should not be taken as a statement about the undesirability of eradicating infectious diseases, but rather as a statement about the suitability of this particular means of obtaining it. If eradication is found to be desirable, then alternative means such as vaccination and/or treatment may be more suitable for achieving this goal.

Next, I turn attention to a comparison of the centralized outcome and the decentralized equilibrium. It follows from inspection that for $\alpha > 0$, it is the case that $\varepsilon_D^* = \boldsymbol{\varepsilon}^* > \varphi_C^*$ so the central planner chooses lower exposure than do individuals in the decentralized equilibrium. As a consequence, $I_D^* > I_C^*$ so steady state disease prevalence is lower under centralized decision making than under decentralized decision making. These observations follow from the fact that the steady state multipliers are ranked so that $\lambda_C^* < \mu_D^*$ for $\alpha > 0$. This means that the shadow cost of infection is smaller from the perspective of the individual than it is from that of the population as a whole. This stems from the fact that the individuals do not internalize the positive external effects that their preventive behavior has on other individuals. It is instructive to be explicit about the nature of these externalities. When an individual decides to prevent infection, this has a direct positive externality on future susceptible individuals (which are either remaining members of the current pool of susceptibles or infected individuals who recover).

Note that for all levels of disease prevalence $I(t) \notin [I_C^*, I_D^*]$, equilibrium exposure levels are socially optimal and hence active public intervention is undesirable. This is because for prevalence levels outside of this interval, decentralized equilibrium levels are already moving prevalence in the right direction (from a social perspective) as fast as possible.

For the special case $\alpha = 0$, i.e. when there is no possible recovery from the disease, inspection of the different steady state values shows that $I_D^* = I_C^*$, $\varepsilon_D^* = \boldsymbol{\varepsilon}^* = \varphi_C^*$ and $\lambda_C^* = \mu_D^*$. In other words, the outcomes under centralized and decentralized decision making coincide. In this case, the equilibrium takes a particularly simple form. For disease prevalence levels below the steady state level, the optimal policy to to fully expose to infection, i.e. not to prevent at all. Once the critical level of infection is reached, the optimal policy switches to full prevention from then onwards. With this policy in place, infection stays constant at the critical level in perpetuity. The outcome of this special case of the model thus mirrors that obtained by Geoffard and Philipson (1996). The reason that the decentralized equilibrium outcome is socially optimal can be understood in terms of the externalities that flow from individual's decentralized decisions. Ceteris paribus, even when recovery is not possible, susceptible individuals' decisions have external effects on other susceptible individuals. But in a large homogeneous population like the one considered here, the only possible equilibria are symmetric equilibria. In particular, this means that each individual decides to switch from no prevention to full

prevention at exactly the same critical level of disease prevalence. As a consequence, when one individual decides to start preventing infection, so does everyone else and there are therefore no positive externalities on them. To sum up, only individuals that are exposed to infection can benefit from others' preventive efforts, but in a symmetric equilibrium everyone starts protecting themselves at the same time, thereby ruling out external effects in equilibrium.

The above results are summarized as follows:

Result (b) If $\alpha > 0$, then in the decentralized equilibrium, both exposure and steady state disease prevalence are strictly higher than in the centralized solution. If $\alpha = 0$, then the decentralized equilibrium coincides with the solution of the centralized problem.

Turning to welfare, in the centralized setting, the steady state flow welfare is given by

$$W_C \equiv \frac{-c(\beta + \rho - \alpha)}{\beta} \tag{44}$$

while in the decentralized solution, it is given by

$$W_D \equiv \frac{-c(\beta + \rho)}{\beta} = W_C - \frac{\alpha}{\beta} \tag{45}$$

The former is obtained by evaluating the planner's Hamiltonian at the centralized solution while the latter is obtained by evaluating the planner's Hamiltonian at the aggregate decentralized exposure level and the corresponding disease prevalence. These values lead to the following straightforward but important result:

Result (c) If $\alpha > 0$, then in the decentralized equilibrium, steady state flow utility is lower than in the solution of the centralized setting, while if $\alpha = 0$, then they coincide.

It follows immediately from inspection of W_C and W_D that the steady state welfare loss due to non-cooperative behavior is increasing in the recovery rate and decreasing in the infectivity of the disease. Interestingly, neither W_C nor W_D depends on the magnitude of the health premium π (although the first-best optimal and the decentralized equilibrium exposure levels do). Last, steady state welfare is decreasing in the prevention cost c in both settings.

5.1. Comparative Statics and Policy Implications. Turning to policy implications of the present analysis, I start by outlining some interesting comparative statics results (which all follow from direct inspection of the relevant derivatives). I will then use these for a discussion of the relevant policy implications of these. Recall for comparison that in the classical model, increased infectivity β or decreased recovery rate α lead to higher (endemic) steady state disease prevalence.

I start by stating comparative statics results that have no counterpart in the classical analysis, namely for the "economic" parameters c and π:

Result (d) In both the centralized and decentralized settings, steady state exposure and disease prevalence are increasing in prevention cost c and decreasing in the health premium π. Furthermore, steady state welfare is decreasing in c and independent of π.

I next turn to the basic epidemiological parameters α and β. The comparative statics results for infectivity and the recovery rate are as follows:

Result (e) In the centralized setting, steady state exposure and disease prevalence are decreasing in infectivity β. Furthermore, steady state prevalence is independent of the recovery rate α while steady state exposure is increasing in α.

Interestingly, as α is increased, the optimal centralized policy is modified exactly to keep prevalence at an unchanged level. Next, I turn to the decentralized setting.

Result (f) In the decentralized setting, steady state equilibrium exposure and disease prevalence are increasing in the recovery rate α and decreasing in infectivity β.

The interpretation of these results is straightforward. Increasing the infectivity or decreasing the recovery rate effectively makes exposure more unappealing, which is in turn reflected in decreased steady state prevalence. Interestingly, the comparative statics results of the decentralized equilibrium are in fact the *exact opposite* of those of classical model. Before discussing the significance of these results, I will outline the effects of such changes on steady state welfare.

Result (g) In the decentralized setting, steady state flow welfare W_D is increasing in β and constant in α while in the centralized setting, W_C is increasing in α and increasing in β when $\rho > \alpha$.

Having states the comparative statics results, I now discuss some of the most important implications of these to optimal public policy. The first immediate observation is that the classical comparative statics results that steady state disease prevalence is increasing in β and decreasing in α may lead to the premature conclusion that a worthy policy aim is to seek to decrease β and increase α with a view to decrease disease incidence and prevalence. A possible rationale is as follows. Given a fixed number of unprotected contacts, reducing infectivity β must decrease the number of new infections (i.e. incidence). Furthermore, since infection is undesirable, decreasing β must increase overall welfare.

There are several examples of how drugs can achieve decreased infectivity. For example, pre-exposure prophylaxis (PrEP) has been shown to reduce the probability of contracting HIV from non-protected sex with an infected individual.[20] Similar effects have recently been documented for vaginal gel containing the AIDS drug tenofovir (see Karim et al., 2010).

Such policies could be misguided on two separate counts. First, it is not necessarily the case that a benevolent planner would always wish to reduce steady state prevalence, as evidenced by the fact that the first-best steady state level of infection is interior. Second, and more importantly, the comparative statics of the classical model on which such policies are based are inadequate as they take individuals' behavior as given and fixed. As is clear from the present analysis, equilibrium exposure levels (and hence equilibrium infection rates) are very much influenced by the magnitudes of α and β and the mechanistic disease accounting inherent in the classical approach fails to account for the behavioral consequences of changing the parameters of the model. In short, there are two countervailing effects of changing these parameters. For example, while decreasing infectivity β reduces overall infection, given the exposure levels, these in turn increase in response to the decrease in infectivity, a result also found by Kremer (1996) in a model

[20] Post-exposure prophylaxis (PEP) may have similar incentive effects as PrEP, as discussed in Szekeres et al. (2004).

of abstinence without prevention such as prophylaxis.[21] It must therefore be determined which and when one effect dominates the other.

Under decentralized decision-making, individuals exhibit *rational disinhibition*, also known as *behavioral disinhibition* or *risk compensation*. This is the observed phenomenon that individuals, when facing reduced risks from engaging in risky activities, may compensate by increasing the level of such activities. In the present context, rational disinhibition is simply the notion that by decreasing the risks associated with unprotected contacts, individuals may rationally increase their level of risky exposure.[22]
These results mean that PrEP style interventions unambiguously lead to lower steady state welfare in the decentralized setting and decreases steady state welfare in the centralized setting when the future is sufficiently unimportant (i.e. when ρ is high enough) or when today's actions have a low impact on future welfare (i.e. when α is low enough). Importantly, in the case of an HIV/AIDS type disease from which recovery is not possible, first-best steady state welfare would be reduced.

To appreciate the full importance of this result, denote the value function of the social planner, i.e. the overall level of discounted social welfare, by $V(\beta)$. For any fixed policy, from revealed preferences it must be the case that $V(\beta) \geq V(\beta')$ for $\beta' \geq \beta$. In other words, if infectivity decreases from β' to β, the planner could choose the exact same path for disease prevalence with infectivity β as he could have with infectivity β', but at a lower cost. In fact, he can do even better by reoptimizing and choosing a better path (given infectivity β). The implication of this insight is that while a decrease in infectivity indeed increases overall welfare, the increase is derived from the fact that on the path towards the new (and higher) level of steady state disease prevalence, the planner induces agents to fully expose themselves to infection (i.e. to not engage in any preventive behavior at all). It is precisely the immediate welfare gains associated with higher exposure levels on the equilibrium path that accounts for the overall increase in welfare. In short, welfare is increased *despite* the fact that it leads to higher levels of infection.

Turning to the decentralized setting, the welfare implications are less clear cut, as there is no revealed preference argument equivalent to that used to analyze the planner's problem. Controlling for the behavior of other individuals, an individual cannot be harmed by lower levels of infectivity, for reasons mirroring those pertaining to the planner's response. However, in equilibrium, all individuals increase their exposure in response to decreased infectivity, thereby leading to suboptimally high levels of disease incidence and prevalence. It is therefore not clear what the net effect on social welfare of infectivity reducing measures is under decentralized decision making. In order to make

[21] Similar results have been obtained independently by Gersovitz (2010) in the framework of Gersovitz and Hammer (2004). As that model contains both treatment and prevention and the existence of a unique steady state is assumed, his results are not easily comparable to those presented here.

[22] Cohen et al. (2009) find evidence to support the existence of risk compensation in a sample of young men in Kenya. Abbas et al. (2007) simulate a model in which different levels of disinhibition are postulated and find that the introduction of PrEP have a beneficial effect (but this study confounds disease prevalence and welfare and the authors are lead to their conclusion by observing that their simulations show that prevalence decreases as PrEP is introduced). Similar results are found in Paltiel et al. (2009). Crepaz et al. (2004) conduct a meta-analytic review and find that while HIV infected individuals' exposure level is unaffected by the availability of PrEP, as would be expected from rational self-interested decision makers. More importanltly, they find that the exposure level of uninfected individuals may increase. See also discussions in Philipson and Posner (1993), Szekeres et al. (2004), Eaton and Kalichman (2007) and Blower et al. (2000).

	Steady State Exposure		**Steady State Prevalence**		**Steady State Welfare**	
	Centralized	Decentralized	Centralized	Decentralized	Centralized	Decentralized
	$\frac{\alpha(\pi-c)}{\beta\pi-(\beta+\rho)c}$	$\frac{\alpha(\pi-c)}{\beta\pi-c(\alpha+\beta+\rho)}$	$\frac{c\rho}{\beta(\pi-c)}$	$\frac{c(\alpha+\rho)}{\beta(\pi-c)}$	$\frac{-c(\beta+\rho-\alpha)}{\beta}$	$\frac{-c(\beta+\rho)}{\beta}$
α	$+$	$+$	0	$+$	$+$	0
β	$-$	$-$	$-$	$-$	$+$ for $\rho > \alpha$	$+$
π	$-$	$-$	$-$	$-$	0	0
c	$+$	$+$	$+$	$+$	$-$	$-$

Table 1: Comparative Statics.

progress on this front, a careful calibration of the model should be performed. This seems a worthwhile project for future exploration.

It should be pointed out that if steady state welfare is to be increased, then infectivity-reducing policies should be accompanied by measures that induce behavioral changes that counterweight the tendency towards rational disinhibition. One possible avenue is to couple the administration of such drugs with risk reduction counselling, as described in Martin et al. (2004). The overall welfare effect of this combination of policies is, however, not clear. This is because the increase in welfare due to a reduction in β stems from the increase in exposure to infection along the equilibrium path to the new steady state. Therefore it is quite possible that such "risk-reduction" measures decrease welfare on the equilibrium path, thereby making the overall effect ambiguous. Last, it should also be noted that while both prophylaxis and PrEP work by reducing the rate at which exposure leads to infection, there is an important difference in the timing of these two measure. PrEP works for an extended period of time and therefore is not subject to the same continual decision making that prophylaxis is. In short, the choice to use prophylaxis is made "in the heat of the moment" whereas the use of PrEP is not.

The comparative statics results are summarized in Table 1.

5.2. Social and Private Usefulness of Prevention. To complete this section, note that the eradication steady state, which is obtained through sustained abstinence, is ruled out under both centralized and decentralized decision making, regardless of parameter values. This means that in steady state, prevention effort must necessarily be less than 100%. In contrast, the endemic steady state with full exposure (i.e. with no preventive effort at all) may be viable, if prevention costs are large enough. It turns out that optimal full exposure (i.e. no prevention at all) and the resulting endemic steady state is viable for a wider range of parameters in the decentralized setting than in the centralized setting. Specifically, in the decentralized setting, full exposure is optimal if

$$c > \pi \left(\frac{\beta - \alpha}{\rho + \beta} \right) \tag{46}$$

which is ruled out by assumption (6).[23] In the centralized setting, full exposure is optimal if

$$c > \pi \left(\frac{\beta - \alpha}{\rho + \beta - \alpha} \right) > \left(\frac{\beta - \alpha}{\rho + \beta} \right) \tag{47}$$

[23] See Appendix C for details of the parameter restrictions.

In other words, for prevention to be privately optimal with decentralized decision making, the costs of prevention must be lower than the level that would make prevention socially optimal with centralized decision making.

6. Continual Prevention, Permanent Prevention and Abstinence

In this section, I offer a detailed discussion of the relation between different ways that preventive behavior has been modeled in the literature. In particular, I will juxtapose moment-by-moment measures that only yield short-term protection with long-term measures such as vaccination. Last, I will discuss the relation to abstinence as a protective measure.

In the present model, preventive behavior is modeled as an ongoing activity that must be sustained through time in order to remain effective against infection. In other words, the protective effects are modeled as being entirely transitory. The upshot is that prevention decisions are particularly simple, depending only on the current population-wide infection level. By contrast, vaccinations provide a more prolonged (or even permanent) protection. Vaccination decisions are therefore necessarily forward-looking in nature.

Note that if decision makers can perfectly commit to full (continual) prevention from some point in time onwards, then the model with no spontaneous recovery reduces to a model with vaccination. To be more precise, at a given moment in time, agents that are protected at that moment cannot be infected, whereas in the vaccination model, no agent that has been vaccinated in the past can be infected. Thus the temporal extent of the effects of preventive behavior are a key ingredient in understanding the relation between the different models and their predictions.

Most dynamic models of vaccination following Anderson and May (1991), such as Geoffard and Philipson (1997), Francis (1997) and Francis (2004), are essentially modified *susceptible-infected-recovered* models (also known as SIR models). In the classical SIR model, infected individuals recover spontaneously and thereby acquire permanent immunity to further infection. In vaccination type models, vaccination takes susceptible individuals directly to the class of recovered (and thus immune) individuals, thereby bypassing a spell of infection. It is well known that disease prevalence is non-monotonic in the classical SIR model; it first increasing and then decreases due to the effects of herd immunity. In the model of Francis (1997), agents are homogeneous and as a result, equilibrium (centralized as well as decentralized) involves all agents vaccinating at exactly the same point in time, namely when a critical level of disease prevalence is reached. Specifically, this means that until that level is reached, there is no vaccination at all whereas after that point in time, all remaining susceptible individuals vaccinate. The equilibrium path of the model is therefore quite simple to describe. Until the critical threshold is reached, the equilibrium path of disease prevalence of the homogeneous model with vaccination is identical to that of the simple SI model, i.e. it is increasing over time. Once the threshold is reached, all uninfected individuals vaccinate and therefore disease prevalence remains constant thereafter.[24]

Turning to continual prevention, the decision to protect one self at a point in time rests solely on disease prevalence at that point in time. Specifically, full prevention is optimal for prevalence levels above a critical threshold and zero prevention is optimal for prevalence levels below it. But as long as no individual engages in preventive behavior,

[24]This discussion is confined to models of closed populations.

the equilibrium path of disease prevalence coincides with that in the SI model, i.e. it is increasing over time. Therefore, if prevention is optimal at any point in time it remains optimal in perpetuity. In other words, a "commitment" to maintain full preventive effort is credible because the incentive to prevent infection becomes stronger as time passes.

In short, in the special case $\alpha = 0$, the equilibrium paths of the two models coincide.[25] Despite the discussion above, this close correspondence between the two models is rather striking. The reason is that while in the continual prevention framework individuals' optimal behavior is necessarily myopic, in the vaccination model agents must solve a potentially complicated continuation game at each point in time. It is not therefore a priori clear why the conditions that prompt agents top vaccinate are exactly the conditions prompting them to engage in preventive behavior.

Although this discussion has mainly focused on the special case of the SIS model in which $\alpha = 0$, it should be emphasized that the vaccination model to which this setting is compared is also a special case of a more general model, namely that studied in Francis (2004), in which immunity can be achieved both through vaccination and through spontaneous recovery at exogenous rate $\gamma > 0$. The steady state of that model is very similar to that of the classical SIR model. Specifically, the disease eventually dies out and individuals are either immune (through recovery) or susceptible in perpetuity (but protected through herd immunity). In other words, if there is a positive rate of recover and associated acquired immunity, then the vaccination model's predictions differ radically from those of the continual prevention model.

A more direct and important distinction between the two modes of prevention is that while continual prevention can sensibly be modeled within an SIR or and SIS framework, vaccination makes sense only within an SIR setting (and not within an SIS setting). In other words, received wisdom from research on vaccination models is of little use in a framework with recovery like the one studied here.

For completeness, it should be noted that the reason that the Francis (1997) model, although formally SIR type model, has a monotonic prevalence path in equilibrium, is that once infected, individuals cannot recover. They therefore constitute a perpetual source of infection to the susceptible population. In contrast, in the classical SIR model, recovered (and hence immune) individuals serve to stifle the propagation of the infection in the population, eventually halting it altogether.

To sum up this discussion, there is not in general a direct correspondence between the models of continual and permanent protection, i.e. of prophylaxis and vaccination. In the special case of the former model in which $\alpha = 0$, the steady state outcome resembles that in the special case of the latter model in which $\gamma = 0$. If either $\alpha > 0$ or $\gamma > 0$ (or both), then the predictions of the two models are incomparable.

Next, I turn to the model of Kremer (1996).[26] To understand the model and it's conclusions, it is useful to consider the following analogy. The underlying model is of the susceptible-infected (or SI) variety, but with the following addition. Before contact between susceptible and infected individuals takes place, each decision maker decides

[25] Indeed, the correspondence is exact. Letting $\pi = (\overline{u} - \underline{u})$, $N = 1$, $\rho = \delta$ and $c/\rho = \theta$, equation (14) in Francis (1997) is identical to equation (18) in the present paper, showing that the threshold prevalence above which individuals choose to vaccinate is the same as the level above which they engage in preventive behavior.

[26] A similar model is that of Auld (2003).

between staying at home (the safe option) and exposing oneself to infection (the risky option). Only those susceptibles that choose the risky option can get infected. Were it not for the risk of infection, the risky option dominates the safe option and so a susceptible individual faces a tradeoff between desirable exposure and the risk of welfare reducing infection. It is immediately clear that all infected individuals will choose the risky option since they have nothing to loose from doing so. The decision a the typical susceptible individual is more complicated, depending on the assessed probability of infection. It is precisely the determination of this probability that makes the Kremer (1996) different from other SI type models like the one of Geoffard and Philipson (1996). Kremer assumes that the probability of infection is proportional to the number of infected individuals *as a fraction of people choosing the risky option.* In particular, this means that the decision to stay at home (i.e. to choose the safe option) changes the rates of contact between susceptible and infected individuals that choose risky the risky option.[27] Rather than being a model of preventive measures (such as condom use), the model is more like a model of abstinence. Since the probability of getting infected is a decreasing function of the fraction of susceptibles that chooses the risky option, the game has strategic complementarities in the sense that the more susceptibles choose to expose themselves, the more attractive does exposure become to other susceptible individuals. In turn, this means that there can be multiple (self-confirming) equilibria. In one equilibrium, all individuals in the population expose themselves to infection. In another equilibrium, only infected individuals expose themselves whereas all susceptible individuals choose the safe option.

It is interesting to note that with a slight addition to the Kremer (1996) model, its predictions can be reconciled with those of the present model. Suppose that those individuals who choose the risky option, have the additional possibility of engaging in costly preventive behavior as modeled in the present paper. Furthermore, assume that prevention is not too costly, so that for sufficiently high probability of infection, it is still desirable to expose oneself but to do so with preventive measures. In this extended model, choosing the risky option at the first stage becomes a dominant strategy. At the second stage, prevention is chosen only if disease prevalence is sufficiently high and not otherwise. Thus the individuals never make use of the safe option and both optimal decisions and the evolution of the infection exactly mirror those derived in the present paper.

7. Conclusion

In the present paper, I have analyzed a simple model of disease propagation in which individuals may engage in privately costly preventive behavior. I find that in the decentralized equilibrium in which agents act non-cooperatively, individuals over-expose themselves to infection compared to the socially optimal level chosen by a central planner. This leads to socially suboptimal steady state disease prevalence and welfare. In the special case where recovery is not possible, as with HIV/AIDS, the decentralized equilibrium outcome is shown to be first best optimal. This suggests that a key source of inefficiency is continued interaction.

The comparative statics of the model are used to consider the desirability of different policy measure aimed at reducing disease prevalence. I argue that subsidies may be cost ineffective because preventive measures are strategic substitutes. This means that

[27] In this respect, the framework of Kremer (1996) resembles a model of quarantine.

subsidies to some individuals may crowd out the preventive efforts of other individuals.

Last, I argue that the introduction of PrEP style measures that are aimed at reducing the infectiousness of the disease and thereby reduce disease incidence and prevalence may bring about increased levels of infection. This is because of rational disinhibition, which makes individuals more prone to risky behavior as a reaction to reduced rates of disease infectivity.

While economic epidemiology takes the sensible step of analyzing models of infection with tools of rational decision making, the natural next step is to analyze decentralized models with non-cooperative/strategically sophisticated agents. Once this is done, it becomes clear that equilibrium outcomes and policy conclusions can change radically and thus one must be careful in interpreting results from representative-agent setups. While the model presented here is admittedly a simplification of reality, it still has some striking implications that merit further analysis.

APPENDIX

A. PROOFS THAT STEADY STATES ARE INTERIOR

A.1. The Centralized Setting. Because of the bang-bang nature of the optimal policy, there are only three policies to consider, namely the singular policy and the two corner policies.[28] For $\varphi(t) = 1$ to be optimal in perpetuity, it must be that $\lambda(t)\beta I(t) + c > 0$ for all t. In this case, the laws of motion become

$$\dot{\lambda}(t) = \lambda(t)\left[\rho + \alpha + \beta\left(2I(t) - 1\right)\right] + \pi \tag{48}$$

$$\dot{I}(t) = I(t)\left[\beta(1 - I(t)) - \alpha\right] \tag{49}$$

Next, consider the rate of change (over time) in the optimality condition. It is given by

$$\begin{aligned} \frac{d}{dt}\left[\lambda(t)\beta I(t) + c\right] &= \beta\left[\dot{\lambda}(t)I(t) + \lambda(t)\dot{I}(t)\right] \\ &= \beta I(t)\left[\lambda(t) + \frac{\pi}{\rho + \beta I(t)}\right] \end{aligned} \tag{50}$$

[28] Note also that because of monotonicity of the optimal path $I(t)$, there can be at most one switch between these policies.

The bracketed expression is positive if and only if

$$\lambda(t) \geq \frac{-\pi}{\rho + \beta I(t)} \tag{51}$$

This condition is implied by the inequality $\lambda(t) \geq \lambda_C^*$ whenever disease prevalence is above the singular solution, i.e. when $I(t) \geq I_C^*$. When disease prevalence is above this level, setting $\varphi(t) = 1$ perpetually is optimal. From full exposure, it then follows that in steady state $I(t) = (\beta - \alpha)/\beta$ and thus the multiplier is given by

$$\lambda(t) = \frac{-\pi}{\rho + \beta - \alpha} \tag{52}$$

But then

$$\lambda(t)\beta I(t) + c = \frac{-\pi(\beta - \alpha) + c\,(\rho + \beta - \alpha)}{\rho + \beta - \alpha} \tag{53}$$

This expression is positive if and only if

$$\pi < c\left(\frac{\beta + \rho - \alpha}{\beta - \alpha}\right) < c\left(\frac{\beta + \rho}{\beta - \alpha}\right) \tag{54}$$

But this inequality is inconsistent with (6) and thus $\varphi(t) = 0$ is optimal. To rule out that setting $\varphi(t) = 0$ perpetually can be optimal, a slightly more delicate argument is needed. This is because as $I(t) \to 0$, the multiplier $\lambda(t) \to -\infty$ and hence it is not necessarily the case that $(\lambda(t)\beta I(t) + c) \to c > 0$, which would render $\varphi(t) = 1$ optimal. In other words, there is a discontinuity at zero prevalence.[29]

Instead, suppose that $\lambda(t)\beta I(t) + c < 0$, which makes $\varphi(t) = 0$ optimal. In this scenario, the laws of motion reduce to

$$\dot{\lambda}(t) = \lambda(t)\,[\rho + \alpha] + [\pi - c] \tag{55}$$

$$\dot{I}(t) = -\alpha I(t) \tag{56}$$

The rate of change (over time) in the optimality condition is given by

$$\begin{aligned} \frac{d}{dt}\left[\lambda(t)\beta I(t) + c\right] &= \beta\left[\dot{\lambda}(t)I(t) + \lambda(t)\dot{I}(t)\right] \\ &= \beta\rho\left[\lambda(t) + \frac{\pi - c}{\rho}\right] \\ &= \beta\rho\left[\lambda(t) - \lambda_C^*\right] \end{aligned} \tag{57}$$

Note that if the bracketed expression is negative and the supposition that $\lambda(t)\beta I(t) + c < 0$ holds, then indeed setting $\varphi(t) = 0$ will remain optimal in perpetuity. When $\varphi(t) = 0$ for all t, the growth rate of the multiplier is given by $\dot{\lambda}(t)/\lambda(t) = \alpha + \rho$. As a result, when $I(t) \to 0$ it follows that $\lambda(t) \to -\infty$. Next, recall that $\lambda(t)$ is the current value multiplier and hence the present value multiplier is given by $\eta(t) \equiv e^{-\rho t}\lambda(t)$. But $\eta(t)$ grows at rate α which implies that $\lim_{t\to\infty} \eta(t) = -\infty$, thus violating the transversality condition

[29] I am grateful to Robert Rowthorn for pointing this out.

$\lim_{t\to\infty}\eta(t)=0$.[30] In conclusion, the steady state must necessarily be interior (in both policy and state variable) ■

A.2. The Decentralized Setting. With some modifications, the proof follows similar steps as those in the centralized setting. For it to be optimal to set $\varepsilon_i(t)=1$ perpetually for all $i\in\mathcal{S}(t)$, it must be that $\mu(t)\beta I(t)+c>0$. In symmetric equilibrium, the laws of motion become

$$\begin{aligned}\dot{\mu}(t) &= \mu(t)\left[\rho+\alpha+\beta I(t)\right]+\pi &(58)\\ \dot{I}(t) &= I(t)\left[\beta(1-I(t))-\alpha\right] &(59)\end{aligned}$$

The rate of change in the optimality condition is given by

$$\begin{aligned}\frac{d}{dt}\left[\lambda(t)\beta I(t)+c\right] &= \beta\left[\dot{\mu}(t)I(t)+\mu(t)\dot{I}(t)\right]\\ &= \beta I(t)\left[\mu(t)+\frac{\pi}{\rho+\beta+\beta(1-I(t))}\right] &(60)\end{aligned}$$

The bracketed expression is positive whenever

$$\mu(t)\geq\frac{-\pi}{\rho+\beta+\beta(1-I(t))}\equiv\widehat{\mu}(t) \tag{61}$$

Note that $\widehat{\mu}(t)\geq\mu_D^*$ if

$$c\leq\pi\left(\frac{2\beta-\alpha-\beta I(t)}{2\beta+\rho-\beta I(t)}\right) \tag{62}$$

which is implied by (6). Next, for $\mu(t)\geq\widehat{\mu}(t)$ setting $\varepsilon_i(t)=1$ perpetually is optimal. Therefore, in symmetric equilibrium full exposure implies that in steady state $I(t)=(\beta-\alpha)/\beta$. Then the multiplier is finite and given by

$$\mu(t)=\frac{-\pi}{\rho+\beta} \tag{63}$$

Using this in the optimality condition gives

$$\mu(t)\beta I(t)+c=\frac{-\pi(\beta-\alpha)+c\left(\rho+\beta\right)}{\rho+\beta} \tag{64}$$

This expression is negative under (6) and therefore $\mu(t)\beta I(t)+c<0$, rendering $\varepsilon_i(t)=0$ as the optimal policy. For $\mu(t)<\widehat{\mu}(t)$, setting $\varepsilon_i(t)=1$ for all t cannot be an optimal path and the system returns to the singular solution.

For $\varepsilon_i(t)=0$ for all t to be optimal, it must be that $\mu(t)\beta I(t)+c<0$. In this case, the laws of motion in symmetric equilibrium are given by

$$\begin{aligned}\dot{\mu}(t) &= \mu(t)\left[\rho+\alpha\right]+\left[\pi-c\right] &(65)\\ \dot{I}(t) &= -\alpha I(t) &(66)\end{aligned}$$

[30] See Seierstad and Sydsaeter (1987, pp. 244-245). For any candidate endemic steady state, the multiplier is finite and hence the transversality condition is satisfied.

Again, consider the rate of change of the optimality condition:

$$\begin{aligned}\frac{d}{dt}\left[\lambda(t)\beta I(t)+c\right] &= \beta\left[\dot{\mu}(t)I(t)+\mu(t)\dot{I}(t)\right] \\ &= \beta I(t)\left[\mu(t)-\lambda_C^*\right] \end{aligned} \tag{67}$$

For $\mu(t) \leq \lambda_C^*$, an analogous argument to the one in the centralized case shows that an optimal path with perpetual full prevention cannot satisfy the transversality condition.[31] Last, for $\mu(t) > \lambda_C^*$, setting $\varepsilon_i(t) = 0$ for all t cannot be an optimal path and the system returns to the singular solution. This completes the proof ■

B. PROOFS THAT OPTIMAL POLICIES ARE OF THE MRAP TYPE

B.1. The Centralized Setting. This appendix confirms that the conditions of Sethi (1977), Theorem 3.1 (i)-(vi) are satisfied for the centralized and decentralized problems respectively. I start with the former of these. From the logistic growth equation, solve for the exposure rate to get the expression

$$\varphi(t) = \frac{\dot{I}(t)+\alpha I(t)}{\beta I(t)(1-I(t))} \tag{68}$$

Substituting this into the objective function and rearranging yields the following modified objective function:

$$\int_0^\infty e^{-\rho t}\left[-I(t)[\pi-c]-c\left(\frac{\beta-\alpha}{\beta}\right)+\left(\frac{c}{\beta I(t)}\right)\dot{I}(t)\right]dt = \int_0^\infty e^{-\rho t}\left[M(I(t))+N(I(t))\dot{I}(t)\right]dt \tag{69}$$

where

$$M(I(t)) \equiv -c\left(\frac{\beta-\alpha}{\beta}\right)-I(t)(\pi-c) \tag{70}$$

$$N(I(t)) \equiv \frac{c}{\beta I(t)} \tag{71}$$

Next, define

$$\begin{aligned}\Delta^C(I(t)) &\equiv -\left(\rho N(I(t))+M'(I(t))\right) \\ &= \frac{-\rho c}{\beta I(t)}+(\pi-c)\end{aligned} \tag{72}$$

First, note that $I(t) = I_C^*$ is the unique solution to the equation

$$\Delta^C(I(t)) = 0 \tag{73}$$

Next, given $I(t) = I_C^*$, $\varphi(t) = \varphi_C^*$ is the unique solution to the equation

$$\dot{I}(t) = 0 \tag{74}$$

[31] This is because for $\varepsilon_i(t) = 0$, the growth rate of $\mu(t)$ coincides with that of $\lambda(t)$ when $\varphi(t) = 0$, i.e. it is given by $\alpha + \rho$.

Last, it is easy to verify that $\Delta^C(I(t)) > 0$ for $I(t) > I_C^*$ while $\Delta^C(I(t)) < 0$ for $I(t) < I_C^*$. This proves that conditions (i)-(iii) are satisfied. Conditions (iv)-(vi) hold trivially ■

B.2. The Decentralized Setting. Turning to the decentralized problem, solve the differential equation governing the probability of being infected for the individual exposure rate to obtain

$$\varepsilon(t) = \frac{\dot{Q}(t) + \alpha Q(t)}{\beta I(t)(1 - Q(t))} \tag{75}$$

Substituting into the individual's objective function and rearrange to get

$$\int_0^\infty e^{-\rho t}\left[-Q(t)\left(\pi - c - \frac{\alpha c}{\beta I(t)}\right) - c + \left(\frac{c}{\beta I(t)}\right)\dot{Q}(t)\right] dt = \int_0^\infty e^{-\rho t}\left[\widehat{M}(I(t)) + \widehat{N}(I(t))\dot{Q}(t)\right] dt \tag{76}$$

where

$$\widehat{M}(I(t)) \equiv -Q(t)\left(\pi - c - \frac{\alpha c}{\beta I(t)}\right) - c \tag{77}$$

$$\widehat{N}(I(t)) \equiv \frac{c}{\beta I(t)} \tag{78}$$

Next, define

$$\begin{aligned}\Delta^D(I(t)) &\equiv -\left(\rho\widehat{N}(I(t)) + \widehat{M}'(I(t))\right) \\ &= \frac{-(\rho+\alpha)c}{\beta I(t)} + (\pi - c)\end{aligned} \tag{79}$$

Here, I have made the substitution $Q(t) = I(t)$. It is easily verified that $I(t) = I_D^*$ is the unique solution to the equation

$$\Delta^D(I(t)) = 0 \tag{80}$$

Given $Q(t) = I(t) = I_D^*$, $\varepsilon_i(t) = \varepsilon_D^*$ is the unique solution to the equation

$$\dot{Q}(t) = 0 \tag{81}$$

The last step is to note that $\Delta^D(I(t)) > 0$ for $I(t) > I_D^*$ while $\Delta^D(I(t)) < 0$ for $I(t) < I_D^*$. Conditions (iv)-(vi) hold trivially. This completes the proof ■

C. Parameter Restrictions

The following inequalities ensure that the endemic steady state solutions are indeed interior in the centralized and decentralized settings respectively:

$$\begin{aligned}
I_C^* &\leq 1 \quad if \quad c \leq \pi\left(\frac{\beta}{\beta+\rho}\right) \geq \pi\left(\frac{\beta-\alpha}{\beta+\rho}\right) \\
I_C^* &\leq \frac{\beta-\alpha}{\beta} \quad if \quad c \leq \pi\left(\frac{\beta-\alpha}{\beta-\alpha+\rho}\right) \geq \pi\left(\frac{\beta-\alpha}{\beta+\rho}\right) \\
\varphi_C^* &\leq 1 \quad if \quad c \leq \pi\left(\frac{\beta-\alpha}{\beta-\alpha+\rho}\right) \\
I_D^* &\leq 1 \quad if \quad c \leq \pi\left(\frac{\beta}{\alpha+\beta+\rho}\right) \leq \pi\left(\frac{\beta-\alpha}{\beta+\rho}\right) \\
I_D^* &\leq \frac{\beta-\alpha}{\beta} \quad if \quad c \leq \pi\left(\frac{\beta-\alpha}{\beta+\rho}\right) \\
\varepsilon_i^* &\leq 1 \quad if \quad c \leq \pi\left(\frac{\beta-\alpha}{\beta+\rho}\right)
\end{aligned}$$

All these conditions are simultaneously satisfied under the assumption in (6).

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