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A susceptible-infected epidemic model with voluntary vaccinations

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Abstract. An susceptible-infected epidemic model with endogenous behavioral changes is presented to analyze the impact of a prophylactic vaccine on disease prevalence. It is shown that, with voluntary vaccination, whether an endemic equilibrium exists or not does not depend on vaccine efficacy or the distribution of agent-types. Although an endemic equilibrium is unique in the absence of a vaccine, the availability of a vaccine can lead to multiple endemic equilibria that differ in disease prevalence and vaccine coverage. Depending on the distribution of agent-types, the introduction of a vaccine or, if one is available, a subsidy for vaccination can increase disease prevalence by inducing more risky behavior.

1. Introduction

Currently 40 million people worldwide are infected with the human immunodeficiency virus (HIV) [23]. It has already taken the lives of over 20 million people in the two decades since the first reported case of acquired immune deficiency syndrome (AIDS), and an average of 14,000 people are newly infected each day [24]. No effective cure or vaccine for HIV exists, although currently available antiretroviral therapy (ART) can increase the length of time an infected person remains healthy before progressing to full-blown AIDS.

The risk of being infected with HIV can be reduced by engaging in less risky behavior such as using condoms in sexual activities or avoiding the sharing of needles for injection drug users. Indeed, studies have shown that a significant number of adults have adopted various forms of safer sexual behavior in response to the AIDS epidemic [1,6]. Moreover, behavioral changes resulting from government AIDS education campaigns and effective dissemination of information regarding the disease through social networks have significantly reduced HIV prevalence in Uganda [22] located in sub-Saharan Africa, a region of the world that is home to more than 60% of all people currently infected with HIV [23]. However, many

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experts believe that the best hope for containing the epidemic, especially in developing countries where 95% of all new infections occur [23], is a preventive vaccine [5,11,13,21].

The development of an effective HIV vaccine remains at present a monumental scientific challenge, mainly due to the tremendous variability of the virus caused by its high mutation rate [7,13]. Moreover, even if a vaccine is developed eventually, it is extremely unlikely to provide sterilizing immunity, the ability to block infection completely, especially given the mutability of HIV [10,14]. At the same time, there is concern that the availability of an imperfect vaccine that offers only partial or limited immunity can actually cause the prevalence of HIV to rise if vaccinated individuals increase their levels of risky behavior (behavioral disinhibition) [2,17].

Several studies have examined mathematically the effects of behavioral disinhibition on disease incidence and have shown that even modest changes in risk behavior resulting from the presence of a vaccine can increase HIV prevalence if the vaccine is not perfectly effective in preventing infection [2,4,9,15,20]. However, since individual risk behavior is treated as given and exogenous in the mathematical models employed in these studies, the results that are derived from them can be sensitive to model specification and changes in parameter values. For example, whether dissemination of an imperfect vaccine can increase HIV prevalence in these models depends critically on the magnitude of behavioral change and the vaccine coverage. Moreover, without a general theory of how risk behavior is determined with or without a vaccine being available, the conditions under which behavioral disinhibition effects can be sufficiently large to increase HIV prevalence when an imperfect vaccine is available cannot be elucidated or identified using these models.

This paper departs from these earlier studies by considering a simple susceptible-infected (SI) epidemic model in which agents' risk behavior, rather than being exogenously specified, is derived using the utility maximization framework of rational choice theory to examine the population-level impact of a nonsterilizing prophylactic vaccine on the prevalence of a sexually transmitted disease (STD). Previous work on the rational choice epidemiological modeling of STDs have considered the interaction between individuals' incentives to engage in self-protective behavior and the equilibrium disease prevalence [3,8,12,18]. For instance, Chen [3] considers an SI model with utility maximizing agents, establishes the conditions under which an endemic equilibrium exists, and examines how the disease prevalence varies with respect to changes in the reproductive number of the disease as well as the preferences of agents for engaging in risky behavior. The model presented here extends these earlier works in rational choice epidemiology by explicitly incorporating a preventive vaccine into the analysis to assess its effect on the equilibrium prevalence of an STD. The analysis focuses on the case in which vaccination is voluntary, so that vaccine coverage as well as risk behavior are endogenously determined by the model and not exogenously given.

It is shown that the reproductive number of the disease in the model, which determines whether an endemic equilibrium exists or not, does not depend on the efficacy of the vaccine. Although an endemic equilibrium is unique in the absence of a vaccine, the availability of a vaccine can lead to multiple endemic equilibria that differ in disease prevalence and vaccine coverage. Whether the introduction

of an imperfect vaccine can worsen an epidemic depends on the efficacy of the vaccine as well as the costs to agents of taking other preventive actions against the risk of infection, and the conditions under which the availability of a vaccine can result in a higher prevalence are identified.

The exposition is organized as follows. The model is developed in Sect. 2. Results concerning the existence and number of endemic equilibria, as well as the relationship between vaccine availability and disease prevalence, are presented in Sect. 3. Various policy implications of the model are derived in Sect. 4. Lastly, concluding remarks and a summary of results are given in Sect. 5.

2. The model

Consider a population consisting of a continuum of agents, each of whom can be either *susceptible* or otherwise *infected* with an STD. Assume that, once infection occurs, agents remain infected for the remainder of their lives. The utility of being infected is u_i, and the utility of being susceptible and healthy is u_h, where $u_h > u_i \geq 0$. Let the utility at death be 0. Time is discrete, and, in any period, a susceptible agent has the option of taking a costly action to self-protect against the risk of infection. Assume that the self-protective action is perfectly effective at blocking transmission of the disease. The utility cost of self-protection is $c_s > 0$, so that if a susceptible agent chooses to self-protect in some period, then the agent's net utility in that period is $u_h - c_s$.

Suppose that a vaccine for the disease exists and that every susceptible agent can also choose to be vaccinated in any period at a utility cost of $c_v > 0$. Assume that, in any period, the decision to be vaccinated precedes the decision of whether or not to adopt the self-protective action. Vaccination reduces a susceptible agent's chances of acquiring the disease from contacts with infected partners. While an unvaccinated susceptible agent who chooses not to self-protect becomes infected with probability $\beta \in (0, 1]$ after one contact with an infected partner, the corresponding probability for a susceptible agent after vaccination is $\beta_v \in [0, \beta)$. Thus, the vaccine decreases the transmission probability of the disease, and the efficacy of the vaccine can be measured by $(\beta - \beta_v)/\beta$. If $\beta_v > 0$, then the vaccine does not confer sterilizing immunity. Assuming that the vaccine offers lifelong immunity, a susceptible agent will choose to be vaccinated at most once. Given any $x \in \mathbb{R}_+$ and $y \in \mathbb{R}_+$, let $F_t(x, y)$ denote the proportion of agents in period t with vaccination cost $c_v \leq x$ and self-protection cost $c_s \leq y$. Hereafter, an agent with vaccination cost c_v and self-protection cost c_s will be referred to as a type-(c_v, c_s) agent.

For simplicity, it is assumed that an agent's expected life span is independent of infection status. Specifically, in any period, an agent, whether infected or not, dies at the end of the period with probability $\delta \in (0, 1)$, which is also the mortality rate given a continuum of agents. Note that the expected lifetime utility of an infected agent is therefore u_i/δ. Upon death, an agent, regardless of infection status, is immediately replaced by an unvaccinated susceptible agent with the same costs of vaccination and self-protection. Therefore, the population size is constant over time and $F_t(x, y) = F(x, y)$ for all t, x, and y. Assume that the joint distribution function F is continuous, and denote its density function by f.

All agents, infected or not, acquire one partner in every period. In particular, partner acquisition is characterized by *proportional mixing*, so that the probability of contacting a partner who is infected in any period t is given by the proportion of agents who are infected in that period P_t. Taking current and the future prevalence of the disease as given, the decision problem of a susceptible agent in each period is to maximize expected lifetime utility by choosing whether or not to take the self-protective action and, if the agent has not been vaccinated, whether to do so or not. Given the stated assumptions of the model, consider the following two cases for the optimization problem of a susceptible agent.

Case 1: A vaccinated susceptible agent.

Using dynamic programming [19], the optimization problem of a vaccinated susceptible agent in period t is given by the optimality equation

$$U_v(P_t) = \max\left\{u_h - c_s + (1-\delta)\,U_v(P_{t+1}),\right.$$
$$\left. u_h + (1-\delta)\left[\beta_v P_t \frac{u_i}{\delta} + (1-\beta_v P_t)\,U_v(P_{t+1})\right]\right\}, \quad (1)$$

where $U_v(P_t)$ is the value of the agent in period t. The first expression in the maximand is the value of self-protecting in period t, and the second expression gives the value of risky behavior in that period.

Case 2: An unvaccinated susceptible agent.

Letting $U_n(P_t)$ denote the value of an unvaccinated susceptible agent in period t, $U_n(P_t)$ solves the optimality equation

$$U_n(P_t) = \max\left\{U_v(P_t) - c_v,\ u_h - c_s + (1-\delta)\,U_n(P_{t+1}),\right.$$
$$\left. u_h + (1-\delta)\left[\beta P_t \frac{u_i}{\delta} + (1-\beta P_t)\,U_n(P_{t+1})\right]\right\}. \quad (2)$$

The first expression in the maximand, $U_v(P_t) - c_v$, gives the value of being vaccinated in period t at cost c_v. The second expression is the value of self-protecting in period t without getting vaccinated, while the last expression is the agent's value with no self-protection or vaccination in period t.

Letting $W_v(P) \equiv U_v(P) - u_i/\delta$, $W_n(P) \equiv U_n(P) - u_i/\delta$, and $w \equiv u_h - u_i$, Eqs. (1) and (2), respectively, can be rewritten as

$$W_v(P_t) = \max\{w - c_s + (1-\delta)\,W_v(P_{t+1}),$$
$$w + (1-\delta)(1-\beta_v P_t)\,W_v(P_{t+1})\} \quad (3)$$

and

$$W_n(P_t) = \max\{W_v(P_t) - c_v, w - c_s + (1-\delta)\,W_n(P_{t+1}),$$
$$w + (1-\delta)(1-\beta P_t)\,W_n(P_{t+1})\}. \quad (4)$$

Using the solutions to the optimization problems in (3) and (4), the law of motion governing disease prevalence can be derived. The notations employed in the derivations are introduced below.

- $\sigma_{v,t}(c_v, c_s)$: The proportion of type-(c_v, c_s) unvaccinated susceptible agents in period t who choose to self-protect and be vaccinated in that period.
- $\sigma_{n,t}(c_v, c_s)$: The proportion of type-(c_v, c_s) unvaccinated susceptible agents in period t who choose to self-protect and not to be vaccinated in that period.
- $\rho_{v,t}(c_v, c_s)$: The proportion of type-(c_v, c_s) unvaccinated susceptible agents in period t who choose to be vaccinated but not self-protect in that period.
- $\rho_{n,t}(c_v, c_s)$: The proportion of type-(c_v, c_s) unvaccinated susceptible agents in period t who choose not to self-protect and not to be vaccinated in that period.
- $r_t(c_v, c_s)$: The proportion of type-(c_v, c_s) vaccinated susceptible agents in period t who choose not to self-protect in that period.
- $S_t(c_v, c_s)$: The proportion of type-(c_v, c_s) agents who are unvaccinated and susceptible in period t.
- $I_t(c_v, c_s)$: The proportion of type-(c_v, c_s) agents who are infected in period t.
- $V_t(c_v, c_s)$: The proportion of type-(c_v, c_s) agents who are vaccinated and susceptible in period t.

Note that $\sigma_{v,t}(c_v, c_s) + \sigma_{n,t}(c_v, c_s) + \rho_{v,t}(c_v, c_s) + \rho_{n,t}(c_v, c_s) = 1$ and $S_t(c_v, c_s) + I_t(c_v, c_s) + V_t(c_v, c_s) = 1$ for all t, c_v, and c_s.

With the stated model assumptions, the proportion of type-(c_v, c_s) unvaccinated susceptible agents in period t who survive to period $t+1$ as infected agents is $(1-\delta) P_t \left[\beta_v \rho_{v,t}(c_v, c_s) + \beta \rho_{n,t}(c_v, c_s)\right]$. Analogously, $1-\delta$ is the proportion of type-(c_v, c_s) vaccinated susceptible agents in period t who remain alive in period $t+1$, and, of those, the fraction $P_t \beta_v r_t(c_v, c_s)$ is infected. Therefore, the disease prevalence among type-(c_v, c_s) agents evolves over time according to the following system of equations:

$$S_{t+1}(c_v, c_s) = (1-\delta)\left[\sigma_{n,t}(c_v, c_s) + \rho_{n,t}(c_v, c_s)(1 - \beta P_t)\right] S_t(c_v, c_s) + \delta,$$

$$I_{t+1}(c_v, c_s) = (1-\delta)\left[I_t(c_v, c_s) + \left[S_t(c_v, c_s)\left(\beta_v \rho_{v,t}(c_v, c_s) + \beta \rho_{n,t}(c_v, c_s)\right) + V_t(c_v, c_s)\beta_v r_t(c_v, c_s)\right] P_t\right], \quad (5)$$

$$V_{t+1}(c_v, c_s) = (1-\delta)\left[V_t(c_v, c_s)(1 - \beta_v r_t(c_v, c_s) P_t) + S_t(c_v, c_s) \times \left(\sigma_{v,t}(c_v, c_s) + \rho_{v,t}(c_v, c_s)(1 - \beta_v P_t)\right)\right]. \quad (6)$$

$$(7)$$

The aggregate disease prevalence in period t, P_t, satisfies

$$P_t = \int_0^\infty \int_0^\infty I_t(c_v, c_s) f(c_v, c_s) \, \mathrm{d}c_s \mathrm{d}c_v. \quad (8)$$

In a steady state, $\sigma_{v,t}(c_v, c_s) = \sigma_v(c_v, c_s)$, $\sigma_{n,t}(c_v, c_s) = \sigma_n(c_v, c_s)$, $\rho_{v,t}(c_v, c_s) = \rho_v(c_v, c_s)$, $\rho_{n,t}(c_v, c_s) = \rho_n(c_v, c_s)$, $r_t(c_v, c_s) = r(c_v, c_s)$, $S_t(c_v, c_s) = S(c_v, c_s)$, $I_t(c_v, c_s) = I(c_v, c_s)$, and $V_t(c_v, c_s) = V(c_v, c_s)$ for all t, c_v, and c_s. Additionally, $P_t = P$ for all t, where, using Eq. (8),

$$P = \int_0^\infty \int_0^\infty I(c_v, c_s) f(c_v, c_s) \, \mathrm{d}c_s \mathrm{d}c_v. \quad (9)$$

By Eqs. (5)–(7), $S(c_v, c_s)$, $I(c_v, c_s)$, and $V(c_v, c_s)$ solve

$$S(c_v, c_s) = (1-\delta)\left[\sigma_n(c_v, c_s) + \rho_n(c_v, c_s)(1-\beta P)\right] S(c_v, c_s) + \delta, \tag{10}$$

$$I(c_v, c_s) = (1-\delta)\left[I(c_v, c_s) + \left[S(c_v, c_s)\left(\beta_v \rho_v(c_v, c_s) + \beta \rho_n(c_v, c_s)\right) + V(c_v, c_s)\beta_v r(c_v, c_s)\right] P\right], \tag{11}$$

$$V(c_v, c_s) = (1-\delta)\left[V(c_v, c_s)(1-\beta_v r(c_v, c_s) P) + S(c_v, c_s)\left(\sigma_v(c_v, c_s) + \rho_v(c_v, c_s)(1-\beta_v P)\right)\right]. \tag{12}$$

Equations (3) and (4), respectively, yield

$$\begin{aligned} W_v(P) &= \max\left\{w - c_s + (1-\delta) W_v(P), w + (1-\delta)(1-\beta_v P) W_v(P)\right\} \\ &= \max\left\{\frac{w - c_s}{\delta}, \frac{w}{\delta + (1-\delta)\beta_v P}\right\} \end{aligned} \tag{13}$$

and

$$\begin{aligned} W_n(P) &= \max\left\{W_v(P) - c_v, w - c_s + (1-\delta) W_n(P), w + (1-\delta)(1-\beta P) W_n(P)\right\} \\ &= \max\left\{W_v(P) - c_v, \frac{w - c_s}{\delta}, \frac{w}{\delta + (1-\delta)\beta P}\right\}. \end{aligned} \tag{14}$$

Note that, since $W_n(P) \geq (w - c_s)/\delta$ for all P, $W_n(P) > (w - c_s)/\delta - c_v$ for all P. Therefore, in a steady state, no susceptible agent would choose to adopt the self-protective action after being vaccinated, i.e. $\sigma_v(c_v, c_s) = 0$ for all c_v and c_s. Consequently, Eq. (14) reduces to

$$W_n(P) = \max\left\{\frac{w}{\delta + (1-\delta)\beta_v P} - c_v, \frac{w - c_s}{\delta}, \frac{w}{\delta + (1-\delta)\beta P}\right\}. \tag{15}$$

Given P, define the functions $\gamma_n(P)$ and $\gamma_v(P)$, respectively, as follows:

$$\frac{w - \gamma_n(P)}{\delta} = \frac{w}{\delta + (1-\delta)\beta P},$$

$$\frac{w - \gamma_v(P)}{\delta} = \frac{w}{\delta + (1-\delta)\beta_v P}.$$

It is easy to see that both $\gamma_n(P)$ and $\gamma_v(P)$ are increasing in P. The benefit to a susceptible agent of vaccinating without self-protection given steady state prevalence P is

$$B(P) \equiv \frac{w}{\delta + (1-\delta)\beta_v P} - \frac{w}{\delta + (1-\delta)\beta P}.$$

Using Eqs. (13) and (15), the optimal behavior for type-(c_v, c_s) susceptible agents in a steady state given prevalence P can be characterized as follows, assuming that,

in the case of indifference, agents choose the action that carries the lowest risk of infection:

$$r(c_v, c_s) = \begin{cases} 1 & \text{if } c_s > \gamma_v(P) \\ 0 & \text{otherwise,} \end{cases} \tag{16}$$

$$\sigma_n(c_v, c_s) = \begin{cases} 1 & \text{if } c_s \leq \gamma_n(P) \text{ and } c_s \leq \gamma_v(P) + \delta c_v \\ 0 & \text{otherwise,} \end{cases} \tag{17}$$

$$\rho_n(c_v, c_s) = \begin{cases} 1 & \text{if } c_s > \gamma_n(P) \text{ and } c_v > B(P) \\ 0 & \text{otherwise,} \end{cases} \tag{18}$$

$$\rho_v(c_v, c_s) = \begin{cases} 1 & \text{if } c_s > \gamma_v(P) + \delta c_v \text{ and } c_v \leq B(P) \\ 0 & \text{otherwise.} \end{cases} \tag{19}$$

Figure 1 depicts the relationship between the optimal behavior of unvaccinated susceptible agents and their costs of vaccination and self-protection.

Definition 1. *A* steady state equilibrium *is given by functions* σ_n, ρ_v, ρ_n, r, S, I, V, *and aggregate prevalence* P *satisfying Eqs.* (9)–(12) *and* (16)–(19). *If* $P > 0$, *then the equilibrium is* endemic.

Remark 2. Note that a no-disease steady state equilibrium in which $P = 0$ always exists.

3. The results

The set of endemic equilibria is now characterized. The analysis begins by establishing the conditions under which an endemic equilibrium exists.

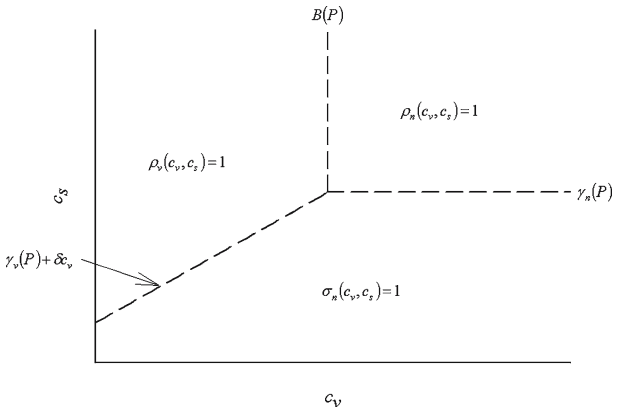

Fig. 1. The relationship between the optimal behavior of unvaccinated susceptible agents and their costs of vaccination and self-protection given steady state prevalence P

3.1. Existence of an endemic equilibrium

Using Eqs. (9)–(12) and (16)–(19), it is straightforward to show that, given steady state prevalence P,

$$I(c_v, c_s) = \begin{cases} 0 & \text{if } c_s \leq \gamma_n(P) \text{ and } c_s \leq \gamma_v(P) + \delta c_v \\ \frac{(1-\delta)\beta_v P}{\delta + (1-\delta)\beta_v P} & \text{if } c_s > \gamma_v(P) + \delta c_v \text{ and } c_v \leq B(P) \\ \frac{(1-\delta)\beta P}{\delta + (1-\delta)\beta P} & \text{if } c_s > \gamma_n(P) \text{ and } c_v > B(P). \end{cases} \tag{20}$$

By Eqs. (9) and (20), an equilibrium prevalence P must satisfy

$$P = \frac{(1-\delta)\beta P}{\delta + (1-\delta)\beta P}\lambda_n(P) + \frac{(1-\delta)\beta_v P}{\delta + (1-\delta)\beta_v P}\lambda_v(P), \tag{21}$$

where $\lambda_n(P) \equiv \int_{B(P)}^{\infty}\int_{\gamma_n(P)}^{\infty} f(c_v, c_s)\,\mathrm{d}c_s\mathrm{d}c_v$ and $\lambda_v(P) \equiv \int_0^{B(P)}\int_{\gamma_v(P)+\delta c_v}^{\infty} f(c_v, c_s)\,\mathrm{d}c_s\mathrm{d}c_v$. In particular, Eq. (21) implies that an endemic equilibrium exists if there is an equilibrium prevalence $P > 0$ such that

$$1 = \frac{(1-\delta)\beta}{\delta + (1-\delta)\beta P}\lambda_n(P) + \frac{(1-\delta)\beta_v}{\delta + (1-\delta)\beta_v P}\lambda_v(P). \tag{22}$$

For convenience, let $g(P)$ denote the function on the right-hand side of Eq. (22). Proposition 3 below gives the necessary and sufficient condition for the existence of an endemic equilibrium.

Proposition 3. *An endemic equilibrium exists if and only if* $(1-\delta)\beta/\delta > 1$.

Proof. Note that $g(1) < 1$ and that $g(0) > 1$ if $(1-\delta)\beta/\delta > 1$. Therefore, by continuity, there exists $P \in (0, 1)$ such that $g(P) = 1$ if $(1-\delta)\beta/\delta > 1$. Now, $g(P) < (1-\delta)\beta/\delta$ for all $P > 0$. Consequently, no endemic equilibrium can exist if $(1-\delta)\beta/\delta \leq 1$. □

The quantity $(1-\delta)\beta/\delta$ is the *reproductive number* of the disease in the model, and it measures the expected number of secondary infections that can be caused by an infected agent over the agent's lifetime without the vaccine if no susceptible agent adopts self-protective behavior. Notice that the reproductive number does not depend on β_v, so that, with voluntary vaccinations, whether an endemic equilibrium exists or not is entirely independent of vaccine efficacy.

3.2. Number of endemic equilibria

An endemic equilibrium of this model is unique in the absence of a vaccine. To see this, note that, when a vaccine is not available, a susceptible agent of type-(c_v, c_s) would choose to adopt the self-protective action in a steady state with prevalence P if $c_s \leq \gamma_n(P)$. Therefore, $P > 0$ is an endemic equilibrium prevalence in the absence of a vaccine if it satisfies

$$1 = \frac{(1-\delta)\beta}{\delta + (1-\delta)\beta P}\int_0^{\infty}\int_{\gamma_n(P)}^{\infty} f(c_v, c_s)\,\mathrm{d}c_s\mathrm{d}c_v. \tag{23}$$

Since the right-hand side of Eq. (23) is decreasing in P, uniqueness obtains. However, the availability of a vaccine in general can lead to multiple endemic equilibria which differ in disease prevalence and vaccine coverage.

Example 4. Suppose $w = 10$, $\delta = 1/10$, $\beta = 2/3$, $\beta_v = 1/3$, and

$$f(c_v, c_s) = \begin{cases} 1 & \text{if } c_v \in [12, 13] \text{ and } c_s \in [9, 10] \\ 0 & \text{otherwise} \end{cases}.$$

The reproductive number of the disease is 6. With the given model specification, the following endemic equilibria exist (see the Appendix for details).

- **Low vaccine coverage equilibrium:** In this equilibrium, no agent chooses to adopt the self-protective action or to be vaccinated. The disease prevalence is 0.833, which is also the unique endemic equilibrium prevalence when the vaccine is not available.
- **Medium vaccine coverage equilibrium:** In this equilibrium, no agent adopts the self-protective action, and, in each period, 42.3% of all unvaccinated susceptible agents choose to be vaccinated. The disease prevalence is 0.769.
- **High vaccine coverage equilibrium:** In this equilibrium, no agent adopts the self-protective action, and, in each period, all unvaccinated susceptible agents choose to be vaccinated. The disease prevalence is 0.667.

To understand how and when multiple endemic equilibria can arise, notice that, when the efficacy of the vaccine is sufficiently low, the benefit of vaccination as measured by $B(P)$ is small when the disease prevalence is either very low or very high. To see this, differentiate $B(P)$:

$$B'(P) = \frac{w(1-\delta)(\beta - \beta_v)\left(\delta^2 - (1-\delta)^2 \beta\beta_v P^2\right)}{(\delta + (1-\delta)\beta P)^2 (\delta + (1-\delta)\beta_v P)^2}.$$

This derivative is positive for small P, while it is negative for sufficiently high prevalence if $\delta^2 < (1-\delta)^2 \beta\beta_v$. Therefore, when this inequality holds, the benefit of vaccination is higher for "intermediate" levels of prevalence, and $B(P)$ reaches its maximum at prevalence level $\delta/(1-\delta)\sqrt{\beta\beta_v}$. This implies that when the disease prevalence is high, the benefit of vaccination may be sufficiently low relative to its cost c_v that susceptible agents would choose not to be vaccinated even if vaccination is chosen at lower prevalence levels. This property of the benefit function B creates a positive feedback loop which can generate multiple endemic equilibria given a low efficacy vaccine. To see why, suppose, for instance, that agents expect the disease prevalence P to be so high that the cost of vaccination exceeds its benefit $B(P)$. If, in addition, the cost of self-protection is sufficiently high, then the proportion of susceptible agents who engage in risky behavior without prior vaccination will be sizable. With a suitably large reproductive number $(1-\delta)\beta/\delta$, the disease prevalence will be high enough to render the option of vaccination suboptimal, thus confirming agents' beliefs. On the other hand, if agents expect the disease prevalence to be lower so that obtaining a vaccination is optimal, then, by their actions, the disease prevalence can be driven down to a level at which the

benefit of being vaccinated does exceed its cost, thereby establishing this outcome as an equilibrium. Notice that, through the positive feedback mechanism inherent in the system given a low efficacy vaccine, agents' beliefs concerning the disease prevalence can be self-fulfilling, and this is the source for the existence of multiple endemic equilibria. The self-confirmatory nature of agents' beliefs implies that, in general, the impact of a vaccine on disease prevalence depends not only on vaccine efficacy but also on how the availability of the vaccine affects agents' expectations concerning future prevalence. As Example 4 shows, the introduction of a vaccine may have little effect if agents believe that to be the case, or it can lower prevalence significantly if agents are sufficiently optimistic about the impact of the vaccine.

Proposition 6 below gives a condition on the model parameters that guarantees the uniqueness of an endemic equilibrium for any distribution function F. More specifically, Proposition 6 shows that an endemic equilibrium is unique if the efficacy of the vaccine is sufficiently high. Before stating this uniqueness result, the following lemma is first established.

Lemma 5. *If* $P_2 > P_1$ *and* $B(P_2) > B(P_1)$, *then* $\lambda_n(P_1) \geq \lambda_n(P_2)$ *and* $\lambda_n(P_1) - \lambda_n(P_2) \geq \lambda_v(P_2) - \lambda_v(P_1)$.

Proof. Now, given $P_2 > P_1$ and $B(P_2) > B(P_1)$,

$$\begin{aligned}
\lambda_n(P_2) &= \int_{B(P_2)}^{\infty} \int_{\gamma_n(P_2)}^{\infty} f(c_v, c_s)\, \mathrm{d}c_s \mathrm{d}c_v \\
&\leq \int_{B(P_2)}^{\infty} \int_{\gamma_n(P_1)}^{\infty} f(c_v, c_s)\, \mathrm{d}c_s \mathrm{d}c_v \\
&\leq \int_{B(P_1)}^{\infty} \int_{\gamma_n(P_1)}^{\infty} f(c_v, c_s)\, \mathrm{d}c_s \mathrm{d}c_v \\
&= \lambda_n(P_1).
\end{aligned}$$

Therefore,

$$\lambda_n(P_1) - \lambda_n(P_2) \geq \int_{B(P_1)}^{B(P_2)} \int_{\gamma_n(P_1)}^{\infty} f(c_v, c_s)\, \mathrm{d}c_s \mathrm{d}c_v \geq 0. \tag{24}$$

In addition, since

$$\int_{0}^{B(P_1)} \int_{\gamma_v(P_2)+\delta c_v}^{\infty} f(c_v, c_s)\, \mathrm{d}c_s \mathrm{d}c_v \leq \int_{0}^{B(P_1)} \int_{\gamma_v(P_1)+\delta c_v}^{\infty} f(c_v, c_s)\, \mathrm{d}c_s \mathrm{d}c_v = \lambda_v(P_1),$$

it is the case that

$$\lambda_v(P_2) \leq \lambda_v(P_1) + \int_{B(P_1)}^{B(P_2)} \int_{\gamma_v(P_2)+\delta c_v}^{\infty} f(c_v, c_s)\, \mathrm{d}c_s \mathrm{d}c_v. \tag{25}$$

It is straightforward to verify that $\gamma_v(P_2) + \delta c_v > \gamma_n(P_1)$ for all $c_v \in [B(P_1), B(P_2)]$. Therefore, using Eq. (25),

$$\lambda_v(P_2) - \lambda_v(P_1) \leq \int_{B(P_1)}^{B(P_2)} \int_{\gamma_n(P_1)}^{\infty} f(c_v, c_s)\, \mathrm{d}c_s \mathrm{d}c_v. \tag{26}$$

Together, Eqs. (24) and (26) give $\lambda_n(P_1) - \lambda_n(P_2) \geq \lambda_v(P_2) - \lambda_v(P_1)$. □

Assume henceforth that $(1-\delta)\beta/\delta > 1$, so that an endemic equilibrium exists.

Proposition 6. *If* $(1-\delta)^2 \beta\beta_v/\delta^2 \leq 1$*, then an endemic equilibrium is unique.*

Proof. The restriction $(1-\delta)^2 \beta\beta_v/\delta^2 \leq 1$ implies that $B(P)$ is strictly increasing in P over the interval $[0, 1)$. By Lemma 5, this gives $\lambda_n(P_1) \geq \lambda_n(P_2)$ and $\lambda_n(P_1) - \lambda_n(P_2) \geq \lambda_v(P_2) - \lambda_v(P_1)$ for all $P_2 > P_1$. Now, $(1-\delta)\beta/[\delta + (1-\delta)\beta P]$ is strictly decreasing in P, and $(1-\delta)\beta_v/[\delta + (1-\delta)\beta_v P]$ is weakly decreasing in P. Therefore, if P_2 is an endemic equilibrium prevalence, i.e. $g(P_2) = 1$, then $g(P_1) > g(P_2)$ for all $P_1 < P_2$ and $g(P_3) < g(P_2)$ for all $P_3 > P_2$, so that any solution to Eq. (22) must be unique. □

3.3. Vaccine availability and disease prevalence

As shown in Example 4, although the availability of a vaccine has the potential to reduce disease prevalence, depending on parameter values and the distribution function F, the introduction of a vaccine can also lead to a perverse outcome in which the prevalence is higher than that in the absence of a vaccine.

Example 7. Suppose $w = 4$, $\delta = 1/10$, $\beta = 2/3$, $\beta_v = 1/2$, and

$$f(c_v, c_s) = \begin{cases} \frac{1}{3} & \text{if } c_v \in [0, 1] \text{ and } c_s \in [0, 3] \\ 0 & \text{otherwise.} \end{cases}$$

Without the vaccine, the endemic equilibrium prevalence, which can be found using Eq. (23), is 0.167, and the proportion of susceptible agents who choose to self-protect in each period is 2/3. When the vaccine is available, the disease prevalence in the unique endemic equilibrium is 0.175 (see the Appendix for details).

To gain some intuition for the conditions under which an endemic equilibrium prevalence with the vaccine being available can exceed the disease prevalence in the absence of the vaccine, consider Fig. 2. The unique endemic equilibrium prevalence without the vaccine is denoted by P_n. In the absence of the vaccine, a susceptible agent of type-(c_v, c_s) will choose the risky action if (c_v, c_s) falls in region I or II, and the agent will adopt the self-protective action otherwise. If the vaccine is made available, then, given disease prevalence P_n, a type-(c_v, c_s) unvaccinated susceptible agent will choose the risky action without vaccination if (c_v, c_s) is in region I, and the agent will choose to be vaccinated (without subsequently taking the self-protective action) if (c_v, c_s) is in regions II and III. Notice that if the proportion of

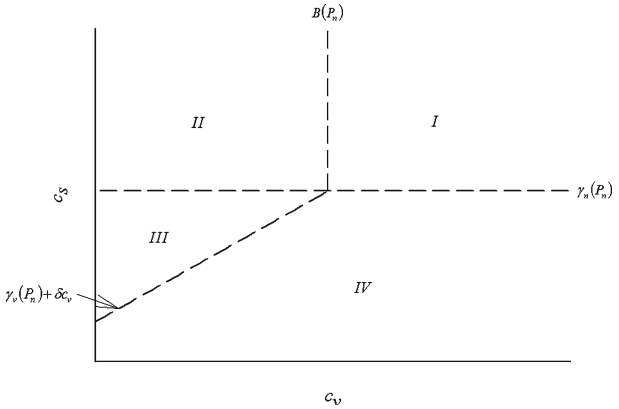

Fig. 2. Given steady state prevalence P_n, susceptible agents with (c_v, c_s) in regions I or II engage in risky behavior whether or not the vaccine is available. All agents in region II and none from region I choose to be vaccinated when the vaccine is introduced. Susceptible agents in regions III and IV adopt self-protective behavior in the absence of the vaccine. When the vaccine is available, agents in region III choose to be vaccinated and engage in risky behavior

susceptible agents in region III is sufficiently large and if the proportion in region II is sufficiently small, then, as long as $\beta_v \neq 0$,

$$g(P_n) > \frac{(1-\delta)\beta}{\delta + (1-\delta)\beta P_n} \int_0^{\infty} \int_{\gamma_n(P_n)}^{\infty} f(c_v, c_s)\, \mathrm{d}c_s \mathrm{d}c_v = 1, \tag{27}$$

where the equality follows from the definition of P_n. Since $g(1) < 1$, Eq. (27) implies by continuity that there exists $P_v \in (P_n, 1)$ such that $g(P_v) = 1$, i.e. given Eq. (27), there is an endemic equilibrium when the vaccine is available in which the disease prevalence exceeds P_n.

4. Policy implications

Using the results presented in Sect. 3, some policy implications of the model can be derived.

4.1. Promoting behavioral changes

To analyze the effect of intervention programs aimed at promoting safer forms of behavior, consider public health policies that lower the cost of self-protection so that the distribution function of agent-types is shifted from $F = F_1$ with density function f_1 to $F = F_2$ with density function f_2, where

$$\int_0^x f_2(c_v, c_s)\, \mathrm{d}c_s \geq \int_0^x f_1(c_v, c_s)\, \mathrm{d}c_s \tag{28}$$

for all $c_v > 0$ and $x > 0$. Let P_i denote an endemic equilibrium prevalence given the distribution function F_i, $i = 1, 2$. It can be shown that if P_1 or P_2 is unique, then a shift of the distribution function from F_1 to F_2 cannot result in a higher equilibrium prevalence.

Proposition 8. *If P_1 or P_2 is unique, then $P_2 \leq P_1$.*

Proof. Let $g_i(P)$ denote the function $g(P)$ given $F = F_i$, $i = 1, 2$. If P_1 is the unique endemic equilibrium prevalence given the distribution function F_1, then $g_1(P) \gtreqless 1$ as $P \lesseqgtr P_1$. Condition (28) implies that $g_2(P) \leq g_1(P)$ for all $P \in [0, 1]$. Therefore, $g_2(P) < 1$ for all $P \in (P_1, 1]$. Since $g_2(P_2) = 1$, this implies that $P_2 \leq P_1$. An analogous argument can be used when P_2 is unique. □

When multiple endemic equilibria exist given F_1 and F_2, then, as Fig. 3 shows, shifting the distribution function from F_1 to F_2 can, depending on which equilibrium is reached, increase disease prevalence.

4.2. Subsidizing vaccinations

The effect of public policies that reduce the cost of vaccination is now considered. Analogous to the analysis of the impact of promoting behavioral changes, suppose the distribution function of agent-types shifts from $F = F_1$ with density function f_1 to $F = F_3$ with density function f_3, where

$$\int_0^x f_3(c_v, c_s)\,\mathrm{d}c_v \geq \int_0^x f_1(c_v, c_s)\,\mathrm{d}c_v$$

for all $c_s > 0$ and $x > 0$. As Example 9 below shows, a subsidy for vaccination, by increasing the proportion of agents who choose to be vaccinated and by decreasing the level of safe behavior, can increase disease prevalence.

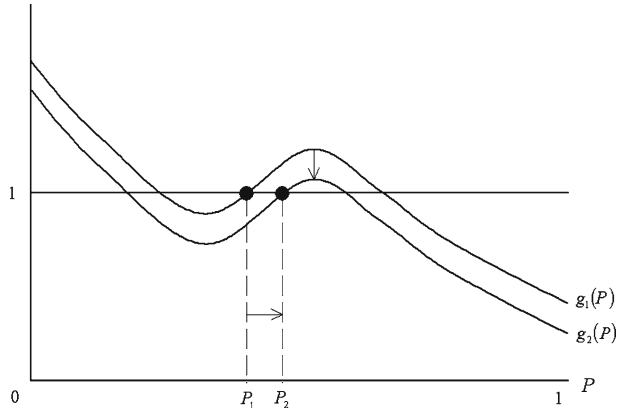

Fig. 3. When multiple endemic equilibria coexist given F_1 and F_2, public health policies that lower the cost of safe behavior and shift the distribution function from F_1 to F_2 can worsen the epidemic

Example 9. Suppose $w = 4$, $\delta = 1/10$, $\beta = 2/3$, $\beta_v = 1/2$,

$$f_1(c_v, c_s) = \begin{cases} \frac{1}{3} & \text{if } c_v \in \left[\frac{1}{2}, \frac{3}{2}\right] \text{ and } c_s \in [0, 3] \\ 0 & \text{otherwise,} \end{cases}$$

and

$$f_3(c_v, c_s) = \begin{cases} \frac{1}{3} & \text{if } c_v \in [0, 1] \text{ and } c_s \in [0, 3] \\ 0 & \text{otherwise.} \end{cases}$$

The unique endemic equilibrium prevalence given the density function f_1 is 0.169. With the density function f_3, the unique endemic equilibrium prevalence is 0.175 (see the Appendix for details).

Although a subsidy, all else being equal, gives agents a greater incentive to be vaccinated, its impact on the equilibrium disease prevalence depends crucially on agents' costs of adopting the self-protective action. Among agents who have a high cost of self-protection and who therefore engage in risky behavior, a subsidy for vaccination, by increasing the proportion of susceptible agents who are vaccinated and less likely to become infected, can lower the prevalence of the disease. On the other hand, a subsidy for vaccination gives agents with a low cost of self-protection less incentive to adopt the safe behavior and, by making the vaccine more accessible, encourages risky behavior. Therefore, all else being the same, a subsidy tends to increase the prevalence level among agents with a low cost of self-protection. Consequently, depending on the distribution of agent-types, a subsidy that makes vaccines more accessible does not necessarily result in a lower prevalence of the disease.

4.3. Mass vaccination programs

Suppose now that, instead of vaccinations being voluntary, a mandatory vaccination program is implemented whereby a proportion $\mu \in (0, 1]$ of the new susceptible agents in each period is vaccinated. As before, every susceptible agent, whether vaccinated or not, has the option of adopting the self-protective action in each period. The decision problem of a vaccinated susceptible agent is still given by Eq. (3), while, for an unvaccinated susceptible agent, the optimality equation (4) reduces to

$$\begin{aligned} W_n(P_t) = \max\{&w - c_s + (1-\delta) W_n(P_{t+1}), \\ &w + (1-\delta)(1-\beta P_t) W_n(P_{t+1})\}. \end{aligned} \tag{29}$$

The disease prevalence among type-(c_v, c_s) agents evolves according to the system of equations

$$S_{t+1}(c_v, c_s) = (1-\delta)[S_t(c_v, c_s)(1 - \beta\rho_t(c_v, c_s) P_t)] + \delta(1-\mu), \tag{30}$$

$$\begin{aligned} I_{t+1}(c_v, c_s) = (1-\delta)[&I_t(c_v, c_s) + [S_t(c_v, c_s)\beta\rho_t(c_v, c_s) \\ &+ V_t(c_v, c_s)\beta_v r_t(c_v, c_s)] P_t], \end{aligned} \tag{31}$$

$$V_{t+1}(c_v, c_s) = (1-\delta)[V_t(c_v, c_s)(1 - \beta_v r_t(c_v, c_s) P_t)] + \delta\mu, \tag{32}$$

where $\rho_t(c_v, c_s)$ is the proportion of type-(c_v, c_s) unvaccinated susceptible agents who choose the risky behavior, and $r_t(c_v, c_s)$ denotes the proportion of type-(c_v, c_s) vaccinated susceptible agents who choose the risky behavior.

In a steady state, $\rho_t(c_v, c_s) = \rho(c_v, c_s)$, $r_t(c_v, c_s) = r(c_v, c_s)$, $S_t(c_v, c_s) = S(c_v, c_s)$, $I_t(c_v, c_s) = I(c_v, c_s)$, $V_t(c_v, c_s) = V(c_v, c_s)$, and $P_t = P$ for all t, c_v, and c_s. Using Eqs. (30)–(32), $S(c_v, c_s)$, $I(c_v, c_s)$, and $V(c_v, c_s)$ satisfy

$$S(c_v, c_s) = (1-\delta)[S(c_v, c_s)(1-\beta\rho(c_v, c_s)P)] + \delta(1-\mu), \tag{33}$$

$$I(c_v, c_s) = (1-\delta)[I(c_v, c_s) + [S(c_v, c_s)\beta\rho(c_v, c_s) + V(c_v, c_s)\beta_v r(c_v, c_s)]P], \tag{34}$$

$$V(c_v, c_s) = (1-\delta)[V(c_v, c_s)(1-\beta_v r(c_v, c_s)P)] + \delta\mu, \tag{35}$$

and the aggregate steady state prevalence P solves Eq. (9). The optimization problem of a vaccinated susceptible agent in a steady state is given by the optimality equation (13), while, for an unvaccinated susceptible agent, Eq. (29) yields

$$\begin{aligned} W_n(P) &= \max\{w - c_s + (1-\delta)W_n(P), w + (1-\delta)(1-\beta P)W_n(P)\} \\ &= \max\left\{\frac{w-c_s}{\delta}, \frac{w}{\delta + (1-\delta)\beta P}\right\}. \end{aligned} \tag{36}$$

Given P, Eqs. (13) and (36), respectively, imply that $r(c_v, c_s)$ satisfies Eq. (16) and

$$\rho(c_v, c_s) = \begin{cases} 1 & \text{if } c_s > \gamma_n(P) \\ 0 & \text{otherwise,} \end{cases} \tag{37}$$

where it is assumed in Eq. (37) that agents choose the action with the lower probability of infection in the case of indifference.

Definition 10. *A steady state equilibrium with mandatory vaccine coverage μ is given by functions ρ, r, S, I, V and aggregate prevalence P satisfying Eqs.* (9), (33)–(35), (37), *and* (16).

Using Eqs. (33)–(35), (37), and (16), it is straightforward to show that, given steady state prevalence P,

$$I(c_v, c_s) = \begin{cases} \frac{\mu(1-\delta)\beta_v P}{\delta+(1-\delta)\beta_v P} + \frac{(1-\mu)(1-\delta)\beta P}{\delta+(1-\delta)\beta P} & \text{if } c_s > \gamma_n(P) \\ \frac{\mu(1-\delta)\beta_v P}{\delta+(1-\delta)\beta_v P} & \text{if } \gamma_v(P) < c_s \le \gamma_n(P) \\ 0 & \text{if } c_s \le \gamma_v(P) \end{cases}. \tag{38}$$

Equations (9) and (38) imply that, in equilibrium, the steady state prevalence P solves

$$\begin{aligned} P = {} & \frac{\mu(1-\delta)\beta_v P}{\delta + (1-\delta)\beta_v P} \int_0^\infty \int_{\gamma_v(P)}^{\gamma_n(P)} f(c_v, c_s)\, \mathrm{d}c_s \mathrm{d}c_v \\ & + \left(\frac{\mu(1-\delta)\beta_v P}{\delta + (1-\delta)\beta_v P} + \frac{(1-\mu)(1-\delta)\beta P}{\delta + (1-\delta)\beta P}\right) \int_0^\infty \int_{\gamma_n(P)}^\infty f(c_v, c_s)\, \mathrm{d}c_s \mathrm{d}c_v. \end{aligned} \tag{39}$$

The following result shows that if vaccine efficacy and coverage are sufficiently high, then the disease can be eradicated in equilibrium.

Proposition 11. *Given mandatory vaccine coverage μ, a unique endemic equilibrium exists if and only if* $(1-\delta)(\mu\beta_v + (1-\mu)\beta)/\delta > 1$.

Proof. Using Eq. (39), an endemic equilibrium prevalence $P > 0$ solves

$$1 = \frac{\mu(1-\delta)\beta_v}{\delta + (1-\delta)\beta_v P} \int_0^\infty \int_{\gamma_v(P)}^{\gamma_n(P)} f(c_v, c_s)\, \mathrm{d}c_s \mathrm{d}c_v + \left(\frac{\mu(1-\delta)\beta_v}{\delta + (1-\delta)\beta_v P} + \frac{(1-\mu)(1-\delta)\beta}{\delta + (1-\delta)\beta P} \right) \int_0^\infty \int_{\gamma_n(P)}^\infty f(c_v, c_s)\, \mathrm{d}c_s \mathrm{d}c_v. \quad (40)$$

Let $h(P)$ denote the function on the right-hand side of Eq. (40). Now, $h(0) = (1-\delta)(\mu\beta_v + (1-\mu)\beta)/\delta$ and $h(1) < 1$ for all $\mu \in (0, 1]$. Therefore, if $(1-\delta)(\mu\beta_v + (1-\mu)\beta)/\delta > 1$, then, by the continuity of h, there must exist $P \in (0, 1)$ such that Eq. (40) holds. Note that $h(P) < 1$ for all $P > 0$ if $(1-\delta)(\mu\beta_v + (1-\mu)\beta)/\delta \leq 1$. Therefore, an endemic equilibrium does not exist if $(1-\delta)(\mu\beta_v + (1-\mu)\beta)/\delta \leq 1$.

To establish uniqueness, suppose $P_1 \in (0, 1)$ and $P_2 \in (P_1, 1)$ both solve Eq. (40). Since $\gamma_v(P)$ and $\gamma_n(P)$ are increasing in P , it follows that

$$\begin{aligned} h(P_1) &\geq \frac{\mu(1-\delta)\beta_v}{\delta + (1-\delta)\beta_v P_1} \int_0^\infty \int_{\gamma_v(P_1)}^{\gamma_n(P_2)} f(c_v, c_s)\, \mathrm{d}c_s \mathrm{d}c_v \\ &\quad + \left(\frac{\mu(1-\delta)\beta_v}{\delta + (1-\delta)\beta_v P_1} + \frac{(1-\mu)(1-\delta)\beta}{\delta + (1-\delta)\beta P_1} \right) \int_0^\infty \int_{\gamma_n(P_2)}^\infty f(c_v, c_s)\, \mathrm{d}c_s \mathrm{d}c_v \\ &\geq \frac{\mu(1-\delta)\beta_v}{\delta + (1-\delta)\beta_v P_1} \int_0^\infty \int_{\gamma_v(P_2)}^{\gamma_n(P_2)} f(c_v, c_s)\, \mathrm{d}c_s \mathrm{d}c_v \\ &\quad + \left(\frac{\mu(1-\delta)\beta_v}{\delta + (1-\delta)\beta_v P_1} + \frac{(1-\mu)(1-\delta)\beta}{\delta + (1-\delta)\beta P_1} \right) \int_0^\infty \int_{\gamma_n(P_2)}^\infty f(c_v, c_s)\, \mathrm{d}c_s \mathrm{d}c_v \\ &\geq h(P_2). \end{aligned}$$

It is straightforward to show that the last inequality must be strict. Therefore, $h(P_1) \neq h(P_2)$, so that P_1 and P_2 cannot both satisfy Eq. (40). □

5. Summary and concluding remarks

In this paper, a rational choice SI epidemic model is presented to analyze the impact of a preventive vaccine on disease prevalence. It is assumed in the model that agents

choose their behavior by comparing the cost and benefit of different actions and picking the one that yields the highest net benefit. It is shown that, with voluntary vaccination, whether an endemic equilibrium exists or not does not depend on vaccine efficacy or the distribution of agent-types (Proposition 3). By comparison, in epidemic models of vaccines which treat agents' behavior as exogenous and therefore make no distinction between voluntary and mandatory vaccination, the condition for disease eradication depends critically on vaccine coverage, vaccine efficacy, and risk behavior [see, for example, 16]. Moreover, in the model presented here, multiple endemic equilibria that differ in disease prevalence and vaccine coverage can coexist (Example 4), unless the vaccine is sufficiently efficacious (Proposition 6). This implies that individuals' expectations concerning the impact of a vaccine can be self-fulfilling and determine which outcome results from the introduction of a vaccine. Note that this multiplicity result concerning the potential impact of a low efficacy vaccine cannot be derived from epidemic models which treat agents' behavior as exogenous since these models take vaccine coverage as given and fixed. In the rational choice SI model, the introduction of a vaccine (Example 7) or, if one is available, a subsidy for vaccination (Example 9) can increase disease prevalence by inducing more risky behavior. On the other hand, unless multiple equilibria coexist, public health policies that promote safer forms of behavior and lower the cost of self-protective activities have the effect of decreasing disease prevalence (Proposition 8), although such behavioral intervention programs cannot result in the eradication of the disease. The analysis here also shows that if a vaccine is sufficiently efficacious and if the vaccine coverage is sufficiently high, then the disease can be eradicated using a mandatory vaccination program. Furthermore, the condition for eradication is independent of agents' preferences for risky behavior (Proposition 11). Taken together with Proposition 3, this result implies that whether eradication can be achieved depends critically on the type of vaccination policy that is implemented.

While epidemic models which treat agents' behavior as exogenous also predict that the introduction of an imperfect vaccine accompanied by increases in risky behavior can lead to higher disease prevalence [2,4,9,15], the rational choice model presented here, by focusing explicitly on individuals' incentives to engage in risky behavior or to be vaccinated, provides more specific predictions of the conditions under which behavioral disinhibition caused by the availability of a vaccine can increase prevalence [condition (27)]. In particular, the analysis here indicates that the introduction of a vaccine is likely to increase prevalence if there is a large proportion of people with low vaccination cost and moderate cost of adopting the self-protective action, since they are the people who are most likely to be vaccinated and to increase their risk behavior when a vaccine is available. Therefore, the model here identifies the segment of the population that can be targeted by policy-makers when designing vaccination policies and prevention programs so as to minimize the likelihood that the availability of a vaccine will lead to a perverse outcome in which prevalence is higher.

A key topic for future research is to determine how robust these results and policy implications are with respect to the model assumptions. In particular, future work can consider alternative specifications of self-protective behavior and patterns

of mixing. There are a number of means by which individuals can reduce the risk of infection, including altering the pattern or rate of partner acquisition. Encompassing these choices into the model allows for a richer analysis of the trade-offs between different forms of self-protective behavior and how these are affected by the availability of a prophylactic vaccine.

Appendix

Example 4. With the specified parameter values and distribution of agent-types, the functions g and B, respectively, are given by

$$g(P)=\begin{cases}\frac{6}{6P+1} & \text{if } P\in\left[0,\frac{8-\sqrt{46}}{18}\right)\cup\left[\frac{8+\sqrt{46}}{18},1\right]\\ \frac{6\left(54P^3+153P^2-84P+7\right)}{(6P+1)^2(3P+1)^2} & \text{if } P\in\left[\frac{8-\sqrt{46}}{18},\frac{61-\sqrt{2369}}{156}\right)\cup\left[\frac{61+\sqrt{2369}}{156},\frac{8+\sqrt{46}}{18}\right)\\ \frac{3}{3P+1} & \text{if } P\in\left[\frac{61-\sqrt{2369}}{156},\frac{61+\sqrt{2369}}{156}\right)\end{cases}$$

and $B(P)=300P/(6P+1)(3P+1)$. The equation $g(P)=1$ has three solutions in $[0,1]$: 0.667, 0.769, and 0.833. In the medium vaccine coverage equilibrium, the proportion of unvaccinated susceptible agents who choose to be vaccinated in each period is given by $\int_{12}^{B(0.769)}\int_{9}^{10}\mathrm{d}c_s\mathrm{d}c_v=0.423$.

Example 7. With the specified parameter values and distribution of agent-types, Eq. (23) is

$$1=\begin{cases}\frac{6(1-2P)}{(6P+1)^2} & \text{if } P\in\left[0,\frac{1}{2}\right)\\ 0 & \text{if } P\in\left[\frac{1}{2},1\right],\end{cases}$$

which yields the solution $P=1/6$. The proportion of susceptible agents who choose to self-protect in each period is $\int_0^1\int_0^{\gamma_n\left(\frac{1}{6}\right)}\frac{1}{3}\mathrm{d}c_s\mathrm{d}c_v=2/3$, where $\gamma_n(P)=24P/(6P+1)$. The function g is given by

$$g(P)=\begin{cases}\frac{6\left(-8748P^5-2916P^4+4617P^3+882P^2+20P+8\right)}{(6P+1)^3(9P+2)^3} & \text{if } P\in\left[0,\frac{33-\sqrt{1041}}{36}\right)\\ \frac{3(118-189P)}{20(9P+2)^2} & \text{if } P\in\left[\frac{33-\sqrt{1041}}{36},\frac{58}{99}\right)\\ \frac{135(2-3P)^2}{(9P+2)^3} & \text{if } P\in\left[\frac{58}{99},\frac{2}{3}\right)\\ 0 & \text{if } P\in\left[\frac{2}{3},1\right].\end{cases}\tag{41}$$

The equation $g(P)=1$ has one solution in $[0,1]$: 0.175.

Example 9. With the specified parameter values, the function g given the density function f_1 is

$$g_1(P) = \begin{cases} \frac{6(1-2P)}{(6P+1)^2} & \text{if } P \in \left[0, \frac{73-\sqrt{5281}}{36}\right) \\ \frac{3\left(-1382184P^5-644436P^4+654210P^3+133521P^2+4428P+1444\right)}{80(6P+1)^3(9P+2)^3} & \\ & \text{if } P \in \left[\frac{73-\sqrt{5281}}{36}, \frac{59-\sqrt{3049}}{108}\right) \\ \frac{3(58-99P)}{10(9P+2)^2} & \text{if } P \in \left[\frac{59-\sqrt{3049}}{108}, \frac{38}{69}\right) \\ \frac{3(118-189P)^2}{80(9P+2)^3} & \text{if } P \in \left[\frac{38}{69}, \frac{118}{189}\right) \\ 0 & \text{if } P \in \left[\frac{118}{189}, 1\right]. \end{cases}$$

The equation $g_1(P) = 1$ has a unique solution in $[0, 1]$: 0.169. With the density function f_3, the function g is given by Eq. (41), and the unique endemic prevalence in this case is 0.175.

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