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ORIGINAL ARTICLE

Bistability of Evolutionary Stable Vaccination Strategies in the Reinfection SIRI Model

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Abstract We use the reinfection SIRI epidemiological model to analyze the impact of education programs and vaccine scares on individuals decisions to vaccinate or not. The presence of the reinfection provokes the novelty of the existence of three Nash equilibria for the same level of the morbidity relative risk instead of a single Nash equilibrium as occurs in the SIR model studied by Bauch and Earn (PNAS 101:13391–13394, 2004). The existence of three Nash equilibria, with two of them being evolutionary stable, introduces two scenarios with relevant and opposite features for the same level of the morbidity relative risk: the low-vaccination scenario corresponding to the evolutionary stable vaccination strategy, where individuals will vaccinate with a low probability; and the high-vaccination scenario corresponding to the evolutionary stable vaccination strategy, where individuals will vaccinate with a high probability. We introduce the evolutionary vaccination dynamics for the SIRI model and we prove that it is bistable. The bistability of the evolutionary dynamics indicates that the damage provoked by false scares on the vaccination perceived morbidity risks can be much higher and much more persistent than in the SIR model. Furthermore, the vaccination education programs to be efficient they need to implement a mechanism to suddenly increase the vaccination coverage level.

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1 Introduction

The SIR model is an epidemiological model introduced in the beginning of the 20th century by Kermack and McKendrick (1927) and describes a Susceptible, Infected, Recovered epidemic process. The infection of a susceptible individual can occur at a certain infection rate when he/she contacts with an infectious individual, whereas after some time the infected individual can recover with a certain recovery rate and acquires some immunity, becoming resistant to reinfection. The SIRI epidemiological model extends the SIR model by allowing the possibility of reinfection due to partial immunity. Hence, the recovered individuals in contact with infected individuals can become infected again. The SIRI epidemic model was introduced by Tudor (1990) to model the spread of a herpes-type of infection in either human or animal population. The conditions for asymptotic stability of the disease-free equilibria and the endemic equilibria were derived by Moreira and Wang (1997). The reinfection phenomena present in the SIRI model were also studied by Driessche and Zou (2007) and by van den Driessche et al. (2007). Besides herpes, the authors claim that such a model is appropriate for tuberculosis, including bovine tuberculosis in cattle and wildlife. In a spatial context, the SIRI model and its phase transitions were studied by Stollenwerk et al. (2007, 2010) and by Stollenwerk and Jansen (2010). Based on a cellular automata method, Song et al. (2011) showed that the reinfection can induce the persistence of the disease in the SIRI model with a spatial structure. The phenomenon of partial immune protection present in the SIRI model can also be found in infections caused by Streptococcus pneumoniae (Lipsitch 1997), Neisseria meningitidis (Gupta and Maiden 2001), tuberculosis (Gomes et al. 2004b) and influenza A and B viruses (Andreasen et al. 1997; Hay et al. 2001; Ferguson et al. 2003). Influenza A is an example of a virus that evolves over time resulting in continuous replacement of circulating strains able to reinfect hosts immune to earlier types (Palese and Young 1982; Andreasen et al. 1997; Ferguson et al. 2003). The evolution of influenza A virus is caused by two processes that change the two proteins, hemagglutinin (HA) and neuraminidase (NA), present in the surface of the influenza virus (Andreasen et al. 1997). One process is the antigenic drift, and the other is the genetic shift. The genetic shift occurs as a genome reassortment, resulting in drastic alternation in HA or NA subtypes. The antigenic drift is related to small mutations in HA and NA, which are responsible for annual or biennial influenza epidemics. Cross-immunity is high between similar strains but very low between genetically different strains (Chamchod and Britton 2012). Therefore, reinfection and imperfect vaccination can occur due to the presence of multi-strains. Influenza often requires new vaccines before each annual epidemics (Davies et al. 1983; Sonoguchi et al. 1986; Ferguson et al. 2003). Childhood diseases like measles and rubella also have imperfect vaccines (Moghadas 2004). The SIRI model can be considered a simplified version of multi-strain models with partial cross-immunity and imperfect vaccination (Gomes et al. 2004a, 2005; Aguiar et al. 2008).

The theory of vaccination games probably started with the work of Fine and Clarkson (1986). Bauch and Earn (2004) used the SIR model to study the impact of the changes of the perceived morbidity relative risk on the individuals' decisions between to vaccinate or not. Galvani, Reluga, Chapman and many other authors conducted several studies about how health policies should be designed and implemented to drive the vaccination coverage level to the utilitarian community optimum instead of the level attained according to the individuals' self-interest (Reluga et al. 2006; Galvani et al. 2007; Cojocaru and Bauch 2009; Reluga and Galvani 2011; Liu et al. 2012; Shim et al. 2012; Bauch and Bhattacharyya 2012). Basu et al. (2008) considered a modified version of the SIRS model to simulate transmission of four representative types of the human papilomavirus (HPV). They observed that public perceptions regarding cervical cancer, genital warts, and HPV vaccination generate vaccination levels far lower than those that maximize the overall health-related utility for the population. The SIRI model differs from the SIRS model by allowing the recovered individuals to become directly infected, without previously passing through the susceptible class. Buonomo et al. (2008) considered a nonlinear SIR model with information dependent vaccination, i.e., they introduce a feedback mechanism in the epidemic model which describes the influence of information on the vaccination coverage level obtained by a vaccination campaign. In Bauch and Earn (2004) and in this paper, the available information and the vaccination campaigns are exogenous factors that can modify significantly the vaccination coverage level. Chen (2006) observed that the availability of an imperfect vaccine in a susceptible-infected epidemic model can lead to multiple endemic equilibria and the introduction of a subsidy for vaccination can rise the disease prevalence by increasing the practice of risky behaviors in sexually transmitted diseases. Reluga (2009) found multiple vaccination Nash equilibria resulting from two interacting subpopulations in an SIS model. Liu et al. (2012) studied the effect of the vaccination cost on the multiple Nash and the utilitarian equilibria in a chickenpox vaccination model. Here, we also obtain multiple vaccination Nash equilibria provoked by the possibility of reinfection, that is a characteristic captured by the SIRI model and inexistent in the SIR model.

For diseases in which vaccination is not compulsory, individuals take into account different aspects when deciding between to vaccinate or not, such as the probability of becoming infected and also the adverse consequences that might result from both infection and vaccination. The morbidity relative risk is the ratio between the morbidity risk from vaccination and the morbidity risk from infection. The morbidity relative risk is one of the most relevant information for the susceptible individuals to make their decisions between to vaccinate or not. Susceptible individuals decide to vaccinate or not with some probability that measures their uncertainty. In vaccination models, the probability of vaccination for each susceptible individual determines his/her vaccination strategy. For each susceptible individual, we construct his/her vaccination expected payoff, depending upon his/her vaccination strategy and the population vaccination strategy. The relevant terms that appear in the construction of the susceptible individual vaccination expected payoff are the following: (i) the susceptible individual vaccination strategy; (ii) the population vaccination strategy, that is the mean of all the susceptible individuals vaccination strategies; (iii) the morbidity relative risk; (iv) the probability of infection for a vaccinated individual; and (v) the probability of infec-

tion for a non-vaccinated individual. In this paper, we add to the vaccination expected payoff introduced by Bauch and Earn (2004) a term corresponding to the probability of infection for a vaccinated individual due to imperfect vaccination (see Sect. 3).

A population vaccination strategy is a Nash equilibrium if no single susceptible individual is able to increase his/her expected payoff by changing his/her vaccination strategy from the population strategy (Hofbauer and Sigmund 1998). A population vaccination strategy is an evolutionary stable vaccination (ESV) strategy if no small group of susceptible individuals are able to increase their expected payoff by deviating their vaccination strategy from the population strategy (Maynard-Smith 1982; Hofbauer and Sigmund 1998). An ESV strategy is a strategy that, if adopted by the population, can not be invaded by a competing strategy adopted by a small group of individuals. Hence, the ESV strategies are more robust than the Nash equilibria that are not ESV strategies, and so more likely to be observed in reality (see Sect. 2).

We study the ESV strategies dependence upon the morbidity relative risk and upon the parameters of the SIRI model. Our analysis asks for a detailed bifurcation analysis, differing from the analysis in Bauch and Earn (2004) where bifurcations do not occur. The most relevant quantity for our bifurcation analysis is the basic reproductive number, that is the expected number of new cases of infection produced by a typical infected individual in a susceptible population (Heesterbeek 2002). We introduce the basic reproductive bifurcation threshold that decreases with the reinfection rate. For values of the basic reproductive ratio below the basic reproductive bifurcation threshold, we prove that there is a unique ESV strategy for each morbidity relative risk. Hence, there is a single vaccination scenario corresponding to the unique ESV strategy, and so our result has similar qualitative, but not quantitative, features to the results obtained by Bauch and Earn (2004). However, for values of the basic reproductive ratio above the basic reproductive bifurcation threshold, there are values of the morbidity relative risk, such that there exist three Nash equilibria, with two of them being ESV strategies. This phenomenon is not captured by the SIR model and introduces two new scenarios with relevant and opposite features for the same level of the morbidity relative risk: the low-vaccination scenario where individuals vaccinate with a low probability; and the high-vaccination scenario where individuals vaccinate with a high probability. For the high-vaccination scenario, we prove that the vaccination expected payoff and the probability of vaccination does not increase with the morbidity relative risk. Furthermore, we show that the individuals vaccination expected payoff is higher at the high-vaccination scenario than at the low-vaccination scenario (see Sect. 4).

We introduce the evolutionary vaccination dynamics for an homogeneous population, where the expected probability of vaccination of the population evolves along time such that the individuals payoff increase, based on the replicator or evolutionary dynamics theory (Maynard-Smith 1982; Hofbauer and Sigmund 1998; Nowak 2006). In the vaccination dynamics, the strains evolve along time and the partial cross-immunity is due, mainly, to the new strains. For values of the basic reproductive ratio below the basic reproductive bifurcation threshold, we prove that the unique ESV strategy is a global stable fixed point. However, for values of the basic reproductive ratio above the basic reproductive bifurcation threshold, we prove that the dynamics are bistable for some values of the morbidity relative risk: The two ESV strategies are

stable fixed points of the evolutionary vaccination dynamics; and the Nash equilibrium that is not an ESV is the boundary vaccination threshold between the basin of attractions of the two stable equilibria. Hence, if the individuals adopt vaccination strategies such that the expected probability of vaccination of the population is smaller than the boundary vaccination threshold, then the population vaccination strategy is trapped in the low-vaccination scenario that is undesirable for the population. On the other hand, if individuals adopt vaccination strategies such that the expected probability of vaccination of the population is larger than the boundary vaccination threshold, then the population vaccination strategy is in the high-vaccination scenario (see Sect. 5).

The existence of the low-vaccination and the high-vaccination scenarios that are stable equilibria of the evolutionary vaccination dynamics contrasts significantly with the case of a single vaccination scenario showed by Bauch and Earn (2004). In the presence of the two possible vaccination scenarios, vaccination scares and vaccination education programs can have a higher impact on the population vaccination strategy than in the case of a single vaccination scenario. For values of the basic reproductive ratio above the basic reproductive bifurcation threshold, we determine a low and a high morbidity threshold for the morbidity relative risk, such that (i) for values of the morbidity relative risk below the low morbidity threshold there is only the high-vaccination scenario; (ii) for values of the morbidity relative risk above the high morbidity threshold there is only the low-vaccination scenario; and (iii) for values of the morbidity relative risk between the low and the high morbidity threshold, the two vaccination scenarios exist. A vaccine scare can wrongly increase the perception of the morbidity relative risk above the high morbidity threshold, such that a catastrophe can occur, and so the population moves abruptly from the high-vaccination scenario to the low-vaccination scenario, provoking a large and quick decrease in the vaccination expected payoff. After a vaccine scare, to drive the population from the low-vaccination scenario to the high-vaccination scenario, the vaccination education programs need two effects to be successful: (i) to decrease the perception of the morbidity relative risk below the high morbidity threshold; and (ii), if the perception of the morbidity relative risk is higher than the low morbidity threshold, to increase the population vaccination strategy above the boundary vaccination threshold. A possible efficient mechanism can consist in offering vaccines and/or make it compulsory, at least to part of the population.

2 Vaccination Nash and ESV Strategies

In this section, we define the Nash and the evolutionary stable vaccination strategies. We classify them in terms of the morbidity relative risk r and of the vaccination-infection risk index π, that we also introduce in this section.

As in Bauch and Earn (2004), we denote by P the probability that a susceptible individual will choose to vaccinate. This probability P is the individual's strategy in the vaccination game. The vaccine uptake level in the population is the proportion of individuals who will be vaccinated and, hence it is the mean of all strategies that will be adopted by the individuals of the population. We denote the vaccine uptake level

or the proportion of the population vaccinated by p, i.e., the population vaccination strategy.

Let r_{v} denote the morbidity risks from vaccination and r_{i} denote the morbidity risks from infection. We define the *morbidity relative risk* r by

$$r = r_{\mathrm{v}}/r_{\mathrm{i}}.$$

Let $\pi_{\overline{\mathrm{v}}}(p)$ denote the probability of a non-vaccinated individual to become infected and $\pi_{\mathrm{v}}(p)$ denote the probability of a vaccinated individual to become infected for a vaccination coverage level p in the population. We define the *vaccination-infection risk* index π by

$$\pi(p) = \pi_{\overline{\mathrm{v}}}(p) - \pi_{\mathrm{v}}(p). \tag{1}$$

The payoff of a non-vaccinated individual is $-r_{\mathrm{i}}\pi_{\overline{\mathrm{v}}}(p)$ and the payoff of a vaccinated individual is $-r_{\mathrm{v}} - r_{\mathrm{i}}\pi_{\mathrm{v}}(p)$.

Definition 1 The *vaccination expected payoff* $E(P, p) \equiv E(P, p; r)$ expressed in r_{i} units is defined by

$$\begin{aligned} E(P, p) &= \frac{(-r_{\mathrm{v}} - r_{\mathrm{i}}\pi_{\mathrm{v}}(p))P + (-r_{\mathrm{i}}\pi_{\overline{\mathrm{v}}}(p))(1 - P)}{r_{\mathrm{i}}} \\ &= -(r + \pi_{\mathrm{v}}(p))P - \pi_{\overline{\mathrm{v}}}(p)(1 - P) \\ &= -\pi_{\overline{\mathrm{v}}}(p) + (\pi(p) - r)P\ . \end{aligned} \tag{2}$$

Using the usual concepts of game theory, we will define the Nash and the evolutionary stable vaccination strategies that are more likely to be adopted by the individuals.

Definition 2 For a given morbidity relative risk $r \geq 0$, the population vaccination strategy P^* is a *vaccination Nash equilibrium*, if

$$E(Q, P^*) - E(P^*, P^*) = (\pi(P^*) - r)(Q - P^*) \leq 0\ , \tag{3}$$

for every strategy $Q \in [0, 1]$.

Hence, if the population vaccination strategy is the Nash equilibrium P^* then no single individual has the incentive to change his/her strategy of vaccination to any other strategy $P \neq P^*$. Since the vaccination-infection risk index π is continuous for the SIRI model [see (8)], we state the following lemma that will be used later.

Lemma 1 (Nash equilibria) *Let us assume that the vaccination-infection risk index π is continuous. The population vaccination strategy P^* is a Nash equilibrium if, and only if, P^* satisfies one of the following conditions:*

(i) $P^* = 0$ *and* $r \geq \pi(0)$*; or*
(ii) $P^* \in (0, 1)$ *and* $r = \pi(P^*)$*; or*
(iii) $P^* = 1$ *and* $r \leq \pi(1)$.

By Lemma 1, for every $P^* \in (0, 1)$, $r = \pi(P^*)$ is the unique morbidity relative risk such that P^* is a Nash equilibrium. Hence, we define the *Nash vaccination expected payoff map* $E : [0, 1] \to [-1, 0]$ by

$$E(p) = E(p, p; \pi(p)),$$

where $p = P^*$ is the Nash equilibrium. By Lemmas 1 (ii) and (2), we have, for $p \in (0, 1)$,

$$E(p) = -\pi_{\overline{\mathrm{v}}}(p).$$

Hence, the Nash vaccination expected payoff map E is minus the probability of infection for a non-vaccinated individual. Again, by Lemma 1 (i), we observe that for $p = 0$, the vaccination expected payoff is constant

$$E(0) = E(0, 0; r) = -\pi_{\overline{\mathrm{v}}}(0),$$

for every $r \geq \pi(0)$. For $p = 1$, by Lemma 1 (iii), we observe that the vaccination expected payoff attains a minimum at $r = \pi(1)$, given by

$$E(1) = E(1, 1; \pi(1)) = -\pi_{\overline{\mathrm{v}}}(1),$$

and a maximum at $r = 0$, given by $E(1, 1; 0) = -\pi_{\mathrm{v}}(1)$. Hence,

$$-\pi_{\overline{\mathrm{v}}}(1) = E(1) \leq E(1, 1; r) = -\pi_{\overline{\mathrm{v}}}(1) + (\pi(1) - r) \leq -\pi_{\mathrm{v}}(1)$$

for every $0 \leq r \leq \pi(1)$.

Now, suppose that all individuals were opting for a vaccination strategy P and a proportion ε of individuals (instead of a single individual) opt for a new vaccination strategy Q. Hence, the new population vaccination strategy is

$$p(\varepsilon) = (1 - \varepsilon)P + \varepsilon Q = P + \varepsilon(Q - P).$$

The vaccination expected payoff of the individuals with vaccination strategy P is

$$E(P, p(\varepsilon)) = -\pi_{\overline{\mathrm{v}}}(p(\varepsilon)) + (\pi(p(\varepsilon)) - r)P\,;$$

and the vaccination expected payoff of the individuals with vaccination strategy Q is

$$E(Q, p(\varepsilon)) = -\pi_{\overline{\mathrm{v}}}(p(\varepsilon)) + (\pi(p(\varepsilon)) - r)Q.$$

We observe that both vaccination expected payoffs $E(P, p)$ and $E(Q, p)$ depend upon the vaccination strategy of the individuals and on the proportion ε of the individuals opting by the new vaccination strategy. The *vaccination expected payoff gain* function

$\Delta E_{P\to Q}(p(\varepsilon)) \equiv \Delta E_{P\to Q}(p(\varepsilon); r)$ of moving from the vaccination strategy P to Q is

$$\Delta E_{P\to Q}(p(\varepsilon)) = E(Q, p(\varepsilon)) - E(P, p(\varepsilon)) = (\pi(p(\varepsilon)) - r)\,(Q - P).$$

Hence, the vaccination expected payoff gain function $\Delta E_{P\to Q}(p(\varepsilon))$ measures the incentive that a group of individuals, of proportion ε, has to change their vaccination strategy from P to Q.

Definition 3 For a given relative morbidity risk $r \geq 0$, the population vaccination strategy P^* is an *evolutionary stable vaccination (ESV)* strategy, if there is a $\varepsilon_0 > 0$, such that for every $\varepsilon \in (0, \varepsilon_0)$ and for every $Q \in [0, 1]$, with $Q \neq P^*$,

$$\Delta E_{P^*\to Q}(p(\varepsilon)) < 0\ .$$

Hence, the population vaccination strategy P^* is an ESV strategy if any small group of individuals that choose a different strategy Q obtain a lower payoff than those choosing P^*.

Lemma 2 (ESV strategies) *Let us assume that the vaccination-infection risk index π is continuous. A population vaccination strategy P^* is an ESV strategy if, and only if, P^* satisfies one of the following conditions:*

(i) $P^* = 0$ *and* $r > \pi(0)$*; or*
(ii) $P^* \in [0, 1]$, $r = \pi(P^*)$ *and π is strictly decreasing at P^*; or*
(iii) $P^* = 1$ *and* $r < \pi(1)$.

Furthermore, a strategy P^* is a Nash equilibrium that is not an ESV strategy if, and only if, P^* satisfies the following condition:

(iv) $P^* \in [0, 1]$, $r = \pi(P^*)$ *and π is not strictly decreasing at P^*.*

Now, we present some extensions of the previous definitions that we will use in the next sections. For a given relative morbidity risk $r \geq 0$, the population vaccination strategy P^* is a *left (resp. right)* ESV strategy if there is a $\varepsilon_0 > 0$, such that for every $\varepsilon \in (0, \varepsilon_0)$ and for every $Q < P^*$ (resp. right $Q > P^*$),

$$\Delta E_{P^*\to Q}(p(\varepsilon)) < 0\ .$$

The population vaccination strategy P^* is a *left (resp. right) weak* ESV strategy if there is an $\varepsilon_0 > 0$, such that for every $\varepsilon \in (0, \varepsilon_0)$ and for every $Q < P^*$ (resp. right $Q > P^*$),

$$\Delta E_{P^*\to Q}(p(\varepsilon)) \leq 0\ .$$

The population vaccination strategy P^* is a *weak* ESV strategy, if P^* is a left and a right weak ESV strategy.

Lemma 3 (weak ESV strategies) *Let us assume that the vaccination-infection risk index π is continuous.*

(i) $P^* \in (0, 1]$ *is a left ESV strategy if, and only if,* $\pi(P^*) \geq r$ *and* π *is left strictly decreasing at* P^*.
(ii) $P^* \in (0, 1]$ *is a weak left ESV strategy if, and only if,* $\pi(P^*) \geq r$ *and* π *is left decreasing at* P^*.
(iii) $P^* \in [0, 1)$ *is a right ESV strategy if, and only if,* $\pi(P^*) \leq r$ *and* π *is right strictly increasing at* P^*.
(iv) $P^* \in [0, 1)$ *is a weak right ESV strategy if, and only if,* $\pi(P^*) \leq r$ *and* π *is right decreasing at* P^*.

Assuming the continuity of the vaccination-infection risk index π, we observe that a weak ESV strategy P^* is a vaccination Nash equilibrium, i.e., for all $Q \in [0, 1]$,

$$\Delta E_{P^* \to Q}(P^*) \leq 0.$$

Proof of Lemma 1 *Case (i)* $P^* = 0$. For $Q \geq P^*$, (3) is satisfied if, and only if,

$$\pi(P^*) - r \leq 0.$$

Hence, $P^* = 0$ is a Nash equilibrium if, and only if $\pi(P^*) \leq r$.
Case (ii) $P^* \in (0, 1)$. For $Q \geq P^*$, (3) is satisfied if, and only if,

$$\pi(P^*) - r \leq 0;$$

and for $Q \leq P^*$, (3) is satisfied if, and only if,

$$\pi(P^*) - r \geq 0.$$

Hence, $P^* \in (0, 1)$ is a Nash equilibrium if, and only if $\pi(P^*) = r$.
Case (iii) $P^* = 1$. For $Q \leq P^*$, (3) is satisfied if, and only if,

$$\pi(P^*) - r \geq 0.$$

Hence, $P^* = 1$ is a Nash equilibrium if, and only if $\pi(P^*) \geq r$.

Proof of Lemma 3 For all $P^*, Q, \varepsilon \in [0, 1]$,

$$|p(\varepsilon) - P^*| = |\varepsilon(Q - P^*)| \leq 2\varepsilon. \tag{4}$$

By (4) and continuity of π, there is $\varepsilon_0 > 0$ sufficiently small such that for all $0 < \varepsilon < \varepsilon_0$, π is left strictly decreasing at P^* if, and only if,

$$\pi(p(\varepsilon)) - \pi(P^*) > 0.$$

Hence, $P^* \in (0, 1]$ is a left ESV equilibrium if, and only if, $r \leq \pi(P^*)$ and π is left strictly decreasing at P^*,

$$\pi(p(\varepsilon)) - r = \pi(p(\varepsilon)) - \pi(P^*) + (\pi(P^*) - r) > 0.$$

The other conditions of Lemma 3 follow in a similar way. □

Proof of Lemma 2 The proof follows from Lemma 3 and from the following observations: (i) $P^* = 0$ is a (resp. weak) ESV equilibrium if, and only if, P^* is a (resp. weak) right ESV equilibrium; (ii) $P^* \in (0, 1)$ is a (resp. weak) ESV equilibrium if, and only if, P^* is a (resp. weak) left and right ESV equilibrium; and (iii) $P^* = 1$ is a (resp. weak) ESV equilibrium if, and only if, P^* is a (resp. weak) left ESV equilibrium. □

3 Vaccination Expected Payoff Dependence upon the SIRI Model

In this section, we compute the vaccination expected payoff for the SIRI model. We perform the static analysis of the Nash vaccination expected payoff E, showing that E increases with the vaccination population strategy p and decreases with the basic reproductive number, for a given morbidity relative risk.

3.1 The Reinfection SIRI Model

The SIRI epidemiological model is described by the following ODE system

$$\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= \mu(S + I + R) - \mu p - \beta SI - \mu S \\
\frac{\mathrm{d}I}{\mathrm{d}t} &= \beta SI - \mu I - \gamma I + \tilde{\beta} RI \\
\frac{\mathrm{d}R}{\mathrm{d}t} &= \mu p + \gamma I - \tilde{\beta} RI - \mu R
\end{aligned}$$

where: μ is the per capita mean birth and death rate; β is the mean transmission rate for a first infection; $\tilde{\beta}$ is the mean transmission rate for reinfections; γ is the mean recovery rate and so $1/\gamma$ is the mean infectious period; p is the susceptible vaccine uptake level and so μp susceptible individuals, or newborns, move to the recovered individuals by vaccination. The novelty of the SIRI model is the introduction in the SIR model of a transition from the recovered class to the infected class, determined by the reinfection rate $\tilde{\beta}$. Hence, the recovered individuals are only partially immune and reinfection can occur.

The state variable S corresponds to the density of the susceptible and non-vaccinated individuals, the state variable I corresponds to the density of the infected individuals, and R corresponds to the density of the recovered and vaccinated individuals. We consider a population with fixed size, $S + I + R = 1$.

Let $f = \mu/\gamma$ be the mean birth and death rate in time units given by the infectious mean lifetime period, typically very small; $\sigma = \tilde{\beta}/\beta$ be the ratio between the reinfection and infection rates; let $R_0 = \beta/(\gamma + \mu)$ be the basic reproductive number;

and $\tilde{R}_0 = (1 + f)R_0$ the transformed basic reproductive number. The value of R_0 measures the intensity of the infection and the value of σ measures the force of the reinfection with relation to the first infection. We assume that $\sigma \in (0, 1)$, because after a first infection an individual typically acquires some resistance to the reinfection. In the presence of partial cross-immunity, it is natural to assume that $\tilde{\beta} < \beta$, and so $\sigma \in (0, 1)$, because $\tilde{\beta}$ measures, mainly, the immunity against the new strain and β measures, mainly, the immunity against all the strains. Furthermore, for some sexually transmitted diseases (Tudor 1990; van den Driessche et al. 2007), individuals reconsider some practices after recovering from a first infection, which leads to a smaller interaction with infected individuals and a smaller probability of being reinfected. Normalizing the time scale $\tau = t/\gamma$, by the mean infectious period $1/\gamma$, we obtain the following ODE system

$$\frac{\mathrm{d}S}{\mathrm{d}\tau} = f(1 - p) - (\tilde{R}_0 SI + fS) \tag{5}$$

$$\frac{\mathrm{d}I}{\mathrm{d}\tau} = \tilde{R}_0 SI + \sigma \tilde{R}_0 RI - (1 + f)I \tag{6}$$

$$\frac{\mathrm{d}R}{\mathrm{d}\tau} = fp + I - (\sigma \tilde{R}_0 RI + fR). \tag{7}$$

3.2 The Vaccination Expected Payoff

Now, we will use the values of the state variables described by the ODE system (5–7) to define the probabilities of a non-vaccinated and a vaccinated individual to become infected for a given susceptible vaccine uptake level p. First, we observe that the state variables S, I and R depend, in particular, on p. In (5), we observe that in the susceptible class: (i) f represents the proportion of newborns; (ii) $-fp$ represents the proportion of vaccinated susceptible individuals, or newborns; (iii) $-\tilde{R}_0 SI$ represents the proportion of susceptible individuals that become infected; and (iv) $-fS$ represents the proportion of susceptible individuals that die. Hence, the probability of a non-vaccinated individual become infected $\pi_{\bar{\mathrm{v}}}(p)$ is the ratio between the susceptible individuals that become infected $-\tilde{R}_0 SI$ and all the individuals that leave the susceptible class without vaccination $-(\tilde{R}_0 SI + fS)$, i.e.,

$$\pi_{\bar{\mathrm{v}}}(p) = \frac{\tilde{R}_0 SI}{\tilde{R}_0 SI + fS} = \frac{\tilde{R}_0 I}{\tilde{R}_0 I + f}.$$

In (7), we observe that in the recovered and vaccinated class: (i) fp represents the proportion of vaccinated susceptible individuals; (ii) I represents the proportion of infected that become recovered; (iii) $-\sigma \tilde{R}_0 RI$ represents the proportion of recovered individuals that become infected; and (iv) $-fR$ represents the proportion of recovered individuals that die. Hence, the probability of a recovered or vaccinated individual to become infected $\pi_{\mathrm{v}}(p)$ is the ratio between the recovered or vaccinated individuals that become infected $-\sigma \tilde{R}_0 RI$ and all the individuals that leave the recovered or vaccinated class $-(\sigma \tilde{R}_0 RI + fR)$, i.e.,

$$\pi_{\mathrm{v}}(p) = \frac{\sigma \tilde{R}_0 R I}{\sigma \tilde{R}_0 R I + f R} = \frac{\sigma \tilde{R}_0 I}{\sigma \tilde{R}_0 I + f} .$$

By (1), for the SIRI model, the vaccination-infection risk index is

$$\pi(p) = \pi_{\overline{\mathrm{v}}}(p) - \pi_{\mathrm{v}}(p) = \frac{\tilde{R}_0 I}{\tilde{R}_0 I + f} - \frac{\sigma \tilde{R}_0 I}{\sigma \tilde{R}_0 I + f} = \frac{f}{\sigma \tilde{R}_0 I + f} - \frac{f}{\tilde{R}_0 I + f}. \tag{8}$$

By (2), for the SIRI model, the vaccination expected payoff $E(P, p) \equiv E(P, p; r, R_0)$ is

$$E(P, p) = -\frac{\tilde{R}_0 I}{\tilde{R}_0 I + f} + \left(\frac{f}{\sigma \tilde{R}_0 I + f} - \frac{f}{\tilde{R}_0 I + f} - r \right) P . \tag{9}$$

3.3 SIRI Stationary States

Here, we present the critical vaccine uptake level and the stationary states for the SIRI model.

Definition 4 The *critical vaccine uptake level* $p_c : (0, +\infty) \to [0, 1] \cup \{+\infty\}$ is

$$p_c(R_0) = \begin{cases} 0 & \text{if} \quad R_0 \leq 1 \\ \frac{R_0 - 1}{R_0(1-\sigma)} & \text{if} \quad 1 < R_0 \leq \frac{1}{\sigma} \\ +\infty & \text{if} \quad R_0 > \frac{1}{\sigma} \end{cases} .$$

Let

$$K = f/(f+1) \quad \text{and} \quad A = (\sigma(R_0 - K) - 1)/(2\sigma R_0) ,$$
$$B = A^2 + K(R_0 - 1)/(\sigma R_0^2) \quad \text{and} \quad C = K(1-\sigma)/(\sigma R_0).$$

Lemma 4 (SIRI stationary states) *The stationary stable value of the infected individuals* $I^*(p) \equiv I^*(p; R_0)$ *for the SIRI model is*

$$I^*(p) = \begin{cases} A + \sqrt{B - Cp} > 0 & \text{if} \quad 0 \leq p < p_c \\ 0 & \text{if} \quad p_c \leq p \leq 1 \end{cases} .$$

The value $p_c \equiv p_c(R_0) \leq 1$ is called the critical vaccine uptake level because, as shown in Lemma 4, p_c is the vaccination boundary between the disease-free stationary state $I^*(p) = 0$ and the endemic stationary state $I^*(p) = A + \sqrt{B - Cp}$. We note that the notation $p_c \equiv p_c(R_0) = +\infty$ means that, for this value of R_0, there is only an endemic stationary state, and there is no disease-free stationary state for any vaccine uptake level $p \in [0, 1]$. The basic reproductive ratio $R_0 = 1/\sigma$, above which we have $p_c = +\infty$, is the *reinfection threshold* (Gomes et al. 2004a, b; Stollenwerk et al.

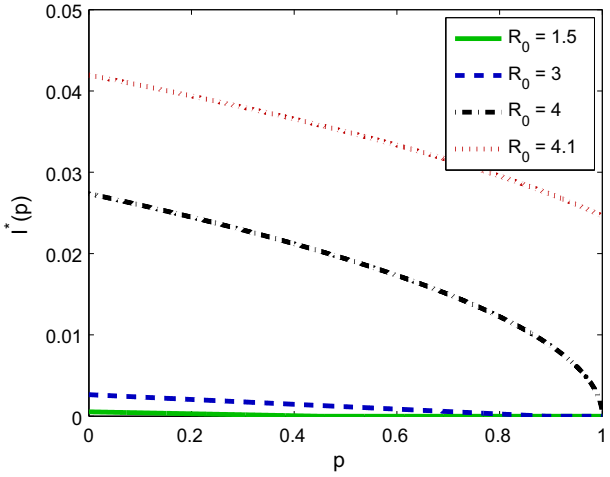

Fig. 1 The stationary value of infected individuals $I^*(p)$ for the basic reproduction numbers $R_0 = 1.5$, $R_0 = 3$, $R_0 = 4$ and $R_0 = 4.1$. The other parameters are $f = 0.01$ and $\sigma = 0.25$, and so, $1/\sigma = 4$ (Color figure online)

2010). We also note that the endemic state $I^*(p) = A + \sqrt{B - Cp}$ decreases with $0 < p < p_c$. (see Fig. 1). By (10) below, we observe that $0 \leq p \leq 1$.

Proof of Lemma 4 The stationary equation given by (5) is

$$S^*(p) = \frac{f(1-p)}{R_0(1+f)I^*(p) + f}\,. \tag{10}$$

The stationary equation given by (6) is the disease-free stationary state $I^*(p) = 0$ or the endemic stationary state

$$\tilde{R}_0 S^*(p) + \sigma \tilde{R}_0(1 - S^*(p) - I^*(p)) - (1+f) = 0\,.$$

Hence, applying (10), we obtain

$$C_2 I^{*2}(p) + C_1 I^*(p) + C_0 = 0,$$

where $B_0 = f(R_0 - 1)$, $B_1 = (1-\sigma) f R_0$ and

$$\begin{aligned} C_0 &= B_0 - B_1 p, \\ C_1 &= \sigma R_0 \tilde{R}_0 - \tilde{R}_0 - \sigma f R_0, \\ C_2 &= -\sigma R_0 \tilde{R}_0. \end{aligned}$$

The positive solution is

$$\begin{aligned} I^*(p) &= -C_1/(2C_2) + \sqrt{(C_1^2 - 4C_0C_2)/(2C_2)^2} \\ &= -C_1/(2C_2) + \sqrt{(C_1^2 - 4B_0C_2 + 4B_1C_2p)/(4C_2^2)}. \end{aligned}$$

Hence, $I^*(p) = A + \sqrt{B - Cp}$ with $A = -C_1/(2C_2)$, $B = (C_1^2 - 4B_0C_2)/(4C_2^2)$ and $C = -4B_1C_2/(4C_2^2) = -B_1/C_2$. Now, we observe that the endemic stationary value vanishes, $I^*(p) = 0$, for the critical vaccine uptake level $p_c \in (0, 1)$ presented in Definition 4. □

3.4 The Stationary Vaccination Expected Payoff

Here, we derive the stationary vaccination-infection risk index and the stationary vaccination expected payoff for the SIRI model. We prove that the stationary vaccination expected payoff increases as the vaccination probability increases.

Theorem 1 (Stationary vaccination-infection risk index) *For the SIRI model, if* $0 \leq p < p_c$, *the stationary probability of a vaccinated individual to become infected is given by*

$$\pi_{\mathrm{v}}(p) = \frac{\sigma \tilde{R}_0(A + \sqrt{B - Cp})}{\sigma \tilde{R}_0(A + \sqrt{B - Cp}) + f}$$

and the stationary probability of a non-vaccinated individual to become infected is given by

$$\pi_{\overline{\mathrm{v}}}(p) = \frac{\tilde{R}_0(A + \sqrt{B - Cp})}{\tilde{R}_0(A + \sqrt{B - Cp}) + f}.$$

Furthermore, the stationary vaccination-infection risk index $\pi(p) \equiv \pi(p; R_0)$ *is*

$$\pi(p) = \begin{cases} \frac{f}{f + \sigma \tilde{R}_0(A + \sqrt{B - Cp})} - \frac{f}{f + \tilde{R}_0(A + \sqrt{B - Cp})} & \text{if } \ 0 \leq p < p_c \\ 0 & \text{if } \ p_c \leq p \leq 1 \end{cases}.$$

Proof of Theorem 1 The proof follows from applying the formulas presented in Lemma 4 to (8). □

By Lemma 1, (i) for $p = 0$, the 0-*probability vaccination Nash level region* is implicitly given by $r \geq \pi(0, R_0)$, (ii) for every $p \in (0, 1)$, the *p-probability vaccination Nash level curve* is implicitly given by $r = \pi(p, R_0)$, and (iii) for $p = 1$, the 1-*probability vaccination Nash level region* is implicitly given by $r \leq \pi(1, R_0)$ (see Fig. 2).

For every $p \in [0, 1]$, the *left level point* $R_0^L(p)$ is defined implicitly by $p_c(R_0^L(p)) = p$, and the *critical level point* $R_0^M(p)$ by $d\pi(p, R_0^M(p))/dR_0 = 0$, i.e., $R_0^M(p)$ is the

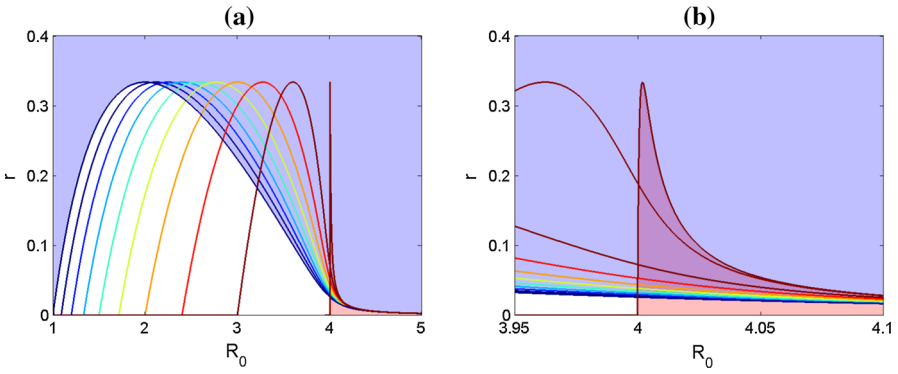

Fig. 2 The p-probability vaccination Nash level curves. In blue it is denoted the 0-probability vaccination Nash level region, and in red the 1-probability vaccination Nash level region. In **b**, we show the blowup of **a** around $R_0 = 4$, to exhibit the 1-probability vaccination Nash level region. We observe that: for $p = 0$, the left level point is $R_0^L(0) = 1$ and the critical level point is $R_0^M(0) \approx 2.001$; for $p = 1$, the left level point is $R_0^L(1) = 4$ and the critical level point is $R_0^M(1) \approx 4.002$; the maximum morbidity relative risk is $r^M(p) = 1/3$ for every p. The other parameters are $f = 0.001$ and $\sigma = 0.25$ (Color figure online)

maximum point of π with respect to R_0. Let the maximum morbidity relative risk $r^M(p)$ of the p-probability level curve be $r^M(p) = \pi(p, R_0^M(p))$ (see Fig. 2).

We observe that, (i) the left level points at $p = 0$ and $p = 1$ are $R_0^L(0) = 0$ and $R_0^L(1) = 1/\sigma = 4$, (ii) $R_0^L(p) < R_0^M(p)$, (iii) both $R_0^L(p)$ and $R_0^M(p)$ are strictly increasing with p, (iv) $r^M(p) = (1-\sqrt{\sigma})/(1+\sqrt{\sigma})$ does not depend upon p, as we will show in (11) (see Fig. 2).

By (9) and Lemma 4, the *stationary vaccination expected payoff* $E(P, p) \equiv E(P, p; r, R_0)$ for the stationary SIRI model is

$$E(P,p) = \begin{cases} -\frac{\tilde{R}_0(A+\sqrt{B-Cp})}{\tilde{R}_0(A+\sqrt{B-Cp})+f} + (\pi(p) - r)\,P & \text{if} \quad 0 \le p < p_c \\ -r\,P & \text{if} \quad p_c \le p \le 1 \end{cases}.$$

Theorem 2 (SIRI vaccination expected payoff) *The stationary Nash vaccination expected payoff* $E : [0,1] \to [-1,0]$ *for the SIRI model is*

$$E(p; R_0) = \begin{cases} \frac{f}{\tilde{R}_0 I^*(p)+f} - 1 & \text{if} \quad 0 \le p < p_c \\ 0 & \text{if} \quad p_c \le p \le 1 \end{cases}.$$

Furthermore, for $0 \le p < p_c$,

$$\frac{\partial E(p; R_0)}{\partial p} = -\frac{\partial I^*}{\partial p}\frac{f\tilde{R}_0}{(\tilde{R}_0 I^*(p) + f)^2} > 0$$
$$\frac{\partial E(p; R_0)}{\partial R_0} = -f\frac{(1+f)I^*(p) + \tilde{R}_0 \partial I^*/\partial R_0}{(\tilde{R}_0 I^*(p) + f)^2} < 0$$

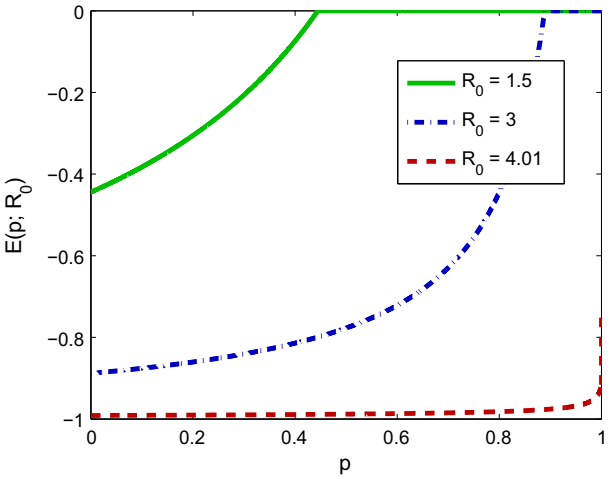

Fig. 3 The Nash vaccination expected payoff $E(p; R_0)$ for $R_0 = 1.5$, $R_0 = 3$ and $R_0 = 4.01$. The other parameters are $f = 0.001$ and $\sigma = 0.25$ (Color figure online)

where the stationary value of infected individuals $I^*(p)$ is given by Lemma 4. Furthermore, $E(p) = 0$ is a global maximum, for every $p \geq p_c$.

Recall from Sect. 2 that the Nash vaccination expected payoff is

$$E(p) = E(p; r) = E(p, p; \pi(p)),$$

for all $0 \leq p \leq 1$. For $p = 0$, $E(0) = -\pi_{\bar{v}}(0)$, and for $p = 1$,

$$-\pi_{\bar{v}}(1) \leq E(1, 1; r) \leq -\pi_v(1),$$

for all $0 \leq r \leq \pi(1)$. Hence, (i) for every $p \geq p_c$, $E(p) = 0$ is a global maximum; (ii) for every $p < p_c$, $E(p)$ increases with p and so the worst case appear when the morbidity relative risk r is above the vaccination-infection risk $\pi(0)$ and the ESV strategy is not to vaccinate $p = 0$ (see Fig. 3). Furthermore, $E(p)$ decreases with the basic reproductive number R_0.

In Fig. 2, we observe some regions where the vaccination Nash level curves intercept. Hence, there might exist more than one vaccination Nash equilibrium for the same relative morbidity risk. By Theorem 2, the vaccination expected payoff is higher at the Nash equilibrium with higher vaccination probability. The existence of these multiple vaccination equilibria will be studied in the next section.

Proof of Theorem 2 The stationary Nash vaccination expected payoff is

$$E(p; R_0) = -\pi_{\bar{v}}(p) = \frac{f}{\tilde{R}_0 I^*(p) + f} - 1\,.$$

Case $p_c \leq p \leq 1$ $E(p; R_0) = 0$ and so p is a global maximum.

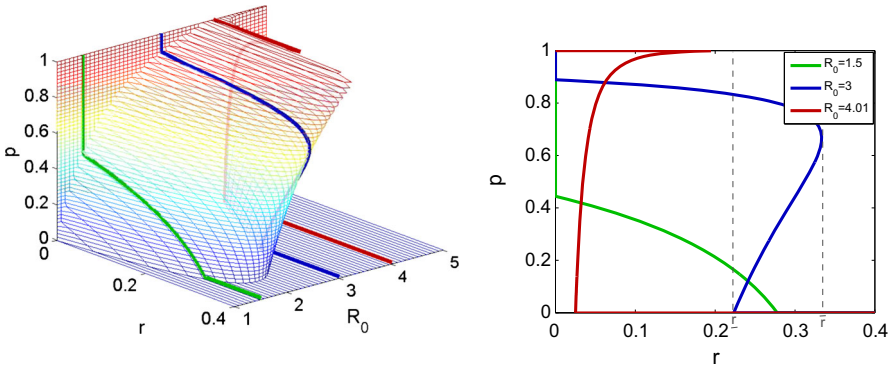

Fig. 4 The vaccination Nash equilibria strategies for different morbidity relative risks r and different basic reproductive ratios R_0. The strategies for the small basic reproductive ratio $R_0 = 1.5 < R_B \approx 2.001$, for the large basic reproductive ratio $R_0 = 3 < 1/\sigma = 4$ and for $R_0 = 4.01 > R_C \approx 4.002$ are highlighted on the left and plotted on the right. For $R_0 = 3$, the low morbidity threshold $\underline{r} = \underline{r}(3)$ and the high morbidity threshold $\bar{r} = \bar{r}(3)$ are also marked with dashed lines. The other parameters are $f = 0.001$ and $\sigma = 0.25$ (Color figure online)

Case $0 < p < p_c$: Since $-1 \leq E(p; R_0) < 0$ and $\partial I^*/\partial p < 0$, we get

$$\partial E(p; R_0)/\partial p = -\frac{\partial I^*}{\partial p}\frac{f\tilde{R}_0}{(\tilde{R}_0 I^*(p) + f)^2} > 0.$$

Since $\partial I^*/\partial R_0 > 0$ and $I^*(p) > 0$, we get

$$\frac{\partial E(p; R_0)}{\partial R_0} = -f\frac{(1+f)I^*(p) + \tilde{R}_0 \partial I^*/\partial R_0}{(\tilde{R}_0 I^*(p) + f)^2} < 0.$$

4 Vaccination Scenarios

In this section, we study the Nash and the ESV strategies effects on the vaccination population strategy depending upon the morbidity relative risk and upon the basic reproductive number.

4.1 Basic Reproductive Bifurcation Thresholds

We will introduce the basic reproductive bifurcation and critical thresholds and the low and the high morbidity relative risk thresholds 4.

Definition 5 The *basic reproductive bifurcation threshold* is

$$R_B = \frac{1}{\sqrt{\sigma}} + \frac{f}{1+f}.$$

Furthermore, a basic reproductive ratio R_0 is *small*, if $R_0 \leq R_B$, and *large*, if $R_0 > R_B$.

As usual, we consider the hypotheses that f is sufficiently small such that $1/\sqrt{\sigma} < R_B < 1/\sigma$. We observe that when $\sigma = \tilde{\beta}/\beta$ tends to zero, R_B tends to $+\infty$.

Definition 6 The *basic reproductive critical threshold* is

$$R_C = \frac{1}{\sigma} + \frac{f}{1+f}\frac{1}{\sqrt{\sigma}}\,.$$

Hence, $R_C = R_B/\sqrt{\sigma}$ and $1 < R_B < 1/\sigma < R_C$.

We observe that the critical level points at $p = 0$ and $p = 1$ are $R_0^M(0) = R_B \approx 2.001$ and $R_0^M(1) = R_C \approx 4.002$ (see Fig. 2).

Lemma 5 *The vaccination-infection risk index π attains its unique local (and global) maximum at the point*

$$p_M : (R_B, +\infty) \to (0, 1]$$

given by

$$p_M \equiv p_M(R_0) = \begin{cases} \frac{B}{C} - \frac{1}{C}\left(A - \frac{f}{\sqrt{\sigma}\tilde{R}_0}\right)^2 & \text{if} \quad R_0 < R_C \\ 1 & \text{if} \quad R_0 \geq R_C \end{cases}.$$

Hence, $p_M(R_0^M(p)) = p$, i.e., p_M is the inverse function of R_0^M. Furthermore, the maximum morbidity relative risk $r^M(p)$ of the p-probability level curve is

$$r^M(p) = \pi\left(p, R_0^M(p)\right) = \pi\left(p_M(R_0), R_0\right),$$

where $R_0 = R_0^M(p)$. By Lemma 5 and Theorem 1, we obtain

$$r^M(p) = \frac{1-\sqrt{\sigma}}{1+\sqrt{\sigma}}. \tag{11}$$

We also observe that $p_M(R_B) = 0$, $p_M(R_C) = 1$, and $p_M(R_0)$ is an increasing function for $R_0 \in [R_B, R_C]$.

Proof of Lemma 5 By (8), we obtain

$$\frac{\partial \pi}{\partial p} = -\frac{f\sigma\tilde{R}_0(\partial I^*/\partial p)}{(\sigma\tilde{R}_0 I^*(p) + f)^2} + \frac{f\tilde{R}_0\,\partial I^*/\partial p}{(\tilde{R}_0 I^*(p) + f)^2}$$

Hence, $\partial\pi/\partial p = 0$ if, and only if, (i) $\partial I^*/\partial p = 0$ or (ii)

$$(\sigma\tilde{R}_0 I^*(p) + f)^2 - \sigma(\tilde{R}_0 I^*(p) + f)^2 = 0\,. \tag{12}$$

Case (i): $\partial I^*/\partial p = 0$. Since $I^*(p) = A + \sqrt{B - Cp}$ and $C > 0$ does not have any solution.

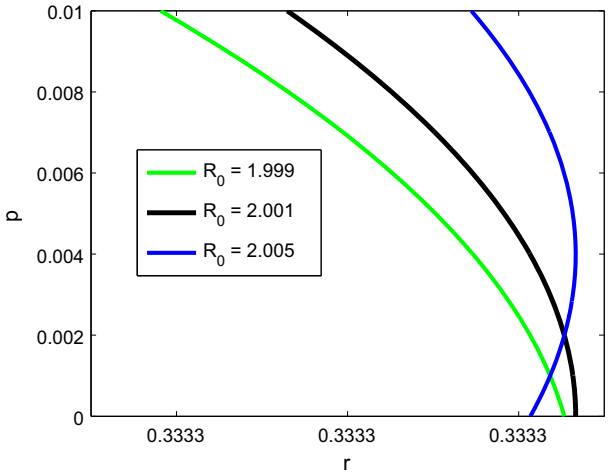

Fig. 5 A blowup close to $p = 0$ of the vaccination Nash strategies for basic reproductive ratios $R_0 = 1.999$, $R_0 = R_B \approx 2.001$ and $R_0 = 2.005$. The other parameters are $f = 0.001$ and $\sigma = 0.25$ (Color figure online)

Case (ii): (12) is equivalent to $I^*(p) = f/(\sqrt{\sigma}\tilde{R}_0)$. Hence, p_M is a candidate to be maximum at

$$p_M \equiv p_M(R_0) = \frac{B}{C} - \frac{1}{C}\left(A - \frac{f}{\sqrt{\sigma}\tilde{R}_0}\right)^2 . \tag{13}$$

Now, $p_M(R_B) = 0$ if

$$R_B = \frac{1}{\sqrt{\sigma}} + \frac{f}{1+f}$$

and, $p_M(R_C) = 1$ if

$$R_C = \frac{1}{\sigma} + \frac{f}{1+f}\frac{1}{\sqrt{\sigma}} .$$

Hence, for $R_0 \in (R_B, R_C)$ the vaccination-infection risk map $\pi(p)$ attains its maximum at $p_M \in (0, 1)$ given by (13). For $R_0 \geq R_C$, the vaccination-infection risk map $\pi(p)$ attains its maximum at $p_M = 1$. □

We observe that the derivative of the vaccination-infection risk map at $p = 0$ satisfies the following properties (see Fig. 5):

(i) for $R_0 = R_B$, $\partial\pi(0, R_B)/\partial p = 0$;
(ii) for $R_0 < R_B$, $\partial\pi(0, R_0)/\partial p < 0$;
(iii) for $R_0 > R_B$, $\partial\pi(0, R_0)/\partial p > 0$.

The vaccination-infection risk map has the following properties:

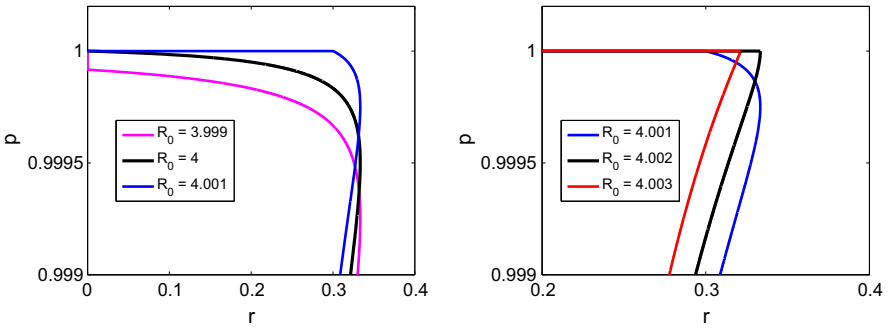

Fig. 6 A blowup close to $p = 1$ of the vaccination Nash strategies for the basic reproductive ratios around $R_0 = 1/\sigma = 4$ (*left*) and $R_0 = R_C \approx 4.002$ (*right*). The other parameters are $f = 0.001$ and $\sigma = 0.25$ (Color figure online)

(i) for $1 < R_0 \leq R_B$,

$$0 < p_c(R_0) \leq p_c(R_B) < 1,$$

and so $\pi(p_c(R_0)) = 0$, $\pi(p)$ is strictly decreasing for $p \in [0, p_c(R_0)]$ and $\pi(p) = 0$ for $p \in [p_c(R_0), 1]$ (see Fig. 4 for $R_0 = 1.5$);

(ii) for $R_B < R_0 \leq 1/\sigma$,

$$p_c(R_B) < p_c(R_0) \leq p_c(1/\sigma) = 1, \quad \text{and} \quad 0 < p_M(R_0) < p_c(R_0),$$

and so $\pi(p)$ is strictly increasing for $p \in [0, p_M(R_0)]$, $\pi(p)$ is strictly decreasing for $p \in [p_M(R_0), p_c(R_0)]$, and $\pi(p) = 0$ for $p \in [p_c(R_0), 1]$ (see Fig. 6 (left) for $R_0 = 3.999$);

(iii) for $1/\sigma < R_0 \leq R_C$,

$$p_c(R_0) = +\infty, \quad \text{and} \quad p_M(1/\sigma) < p_M(R_0) \leq 1,$$

and so $\pi(p)$ is strictly increasing for $p \in [0, p_M(R_0)]$, $\pi(p)$ is strictly decreasing for $p \in [p_M(R_0), 1]$, and $\pi(1) > 0$ (see Fig. 6 for $R_0 = 4.001$);

(iv) for $R_0 > R_C$, $\pi(p)$ is strictly increasing for $p \in [0, 1]$ (see Fig. 6 (right) for $R_0 = 4.003$).

Definition 7 For large basic reproductive ratios $R_0 > R_B$, the *low morbidity threshold* is $\underline{r} \equiv \underline{r}(R_0) = \pi(0)$; and the *high morbidity threshold* is $\overline{r} \equiv \overline{r}(R_0) = \pi(p_M)$.

4.2 Vaccination Scenarios for Free Morbidity Relative Risk

For free morbidity relative risk $r = 0$ diseases, we will show that the reinfection threshold $R_0 = 1/\sigma$ plays the following role, (i) for $R_0 \leq 1/\sigma$, the left ESV strategy p_c eradicates the disease; and (ii) for $R_0 > 1/\sigma$, the ESV strategy corresponding to full vaccination coverage 1 does not eradicate the disease.

Theorem 3 (Free relative morbidity risks) *Suppose that the disease is free of morbidity relative risk $r = 0$. The following strategies are the Nash equilibria:*
For $R_0 < 1/\sigma$:

(i) p_c is a left ESV strategy and a right weak ESV strategy; and
(ii) $p^* \in (p_c, 1]$ are weak ESV strategies.

For $R_0 \geq 1/\sigma$:

(iii) $P^* = 1$ is an ESV strategy.

Proof of Theorem 3 *Case* $R_0 < 1/\sigma$ By Lemma 4, $\pi|(0, p_c)) > 0$ is decreasing and $\pi([p_c, 1]) = 0$. Hence, by Lemma 3 (i) and (iv), we get that $P^* = p_c$ is a left ESV strategy and a weak right ESV strategy. By Lemma 3 (ii) and (iv), we get that $P^* \in (p_c, 1]$ are weak ESV strategies. Furthermore, there is no other Nash equilibrium. *Case* $R_0 \geq 1/\sigma$: By Lemma 4, $\pi|[0, 1]) > 0$. Hence, by Lemma 2 (iii), $P^* = 1$ is an ESV strategy. Furthermore, there is no other Nash equilibrium. □

For a free morbidity risk disease, this theorem reinforces the relevance of the reinfection threshold $R_0 = 1/\sigma$:

(i) For $R_0 < 1/\sigma$, the left ESV strategy $P^* = p_c$ gives the lower bound for the Nash equilibria of vaccination uptake level of the population. Hence, the vaccine uptake level of the population $P^* \geq p_c$ guarantees that the population is at a disease-free stationary state.
(ii) For $R_0 = 1/\sigma$, the ESV strategy $P^* = 1$ gives the vaccine uptake level of the population that still reaches a disease-free stationary state.
(iii) For $R_0 > 1/\sigma$, the ESV strategy $P^* = 1$ gives the vaccine uptake level of the population. However, even a full vaccination level of susceptible individuals, or newborns, is not enough to eradicate the disease (i.e., to attain a disease-free stationary state). Hence, further action is needed like, for instance, to vaccine the recovery individuals as well (e.g., influenza).

4.3 Vaccination Scenarios for Positive Morbidity Relative Risks

In Theorem 4 below, we will show that for small basic reproductive ratios $R_0 \leq R_B$, there is a single ESV strategy (see also Fig. 4, for $R_0 = 1.5$). In Theorem 5, we will show that for large basic reproductive ratios $R_0 > R_B$ and for morbidity relative risks between the low and the high morbidity thresholds $\underline{r}(R_0) < r < \overline{r}(R_0)$, there are a low and a high ESV strategies P_L and P_H, with $P_L < P_H$ (see also Fig. 4, for $R_0 = 3$ and $R_0 = 4.01$). By Theorem 2, the expected payoff is larger for the high ESV strategy P_H than for the low ESV strategy P_L.

4.3.1 Vaccination Scenarios for Small Basic Reproductive Ratios

Here, we characterize the Nash and the ESV strategies for small basic reproductive ratios $R_0 \leq R_B$ and positive morbidity relative risks.

Theorem 4 (Small basic reproductive ratios) *For small basic reproductive ratios* $R_0 \in (1, R_B]$ *and morbidity relative risks* $r > 0$*, the Nash equilibria are the following:*

$$p_E \equiv p_E(r; R_0) = \begin{cases} 0 & \text{if} \quad r \geq \pi(0) \\ \pi^{-1}(r) & \text{if} \quad 0 < r < \pi(0) \end{cases} .$$

Furthermore, $P_E(r; R_0)$ *are ESV strategies.*

Proof of Theorem 4 By Lemma 5, $\pi|[0, p_c)$ is strictly decreasing and $\pi(p_c) = 0$. Hence, by Lemma 2 (ii), $P^* \in [0, p_c)$ is an ESV strategy with $0 = \pi(p_c) < r = \pi(P^*) \leq \pi(0)$. Hence, in particular, 0 is an ESV strategy for $r = \pi(0)$. By Lemma 2 (i), 0 is an ESV strategy for $r > \pi(0)$. Furthermore, there is no other Nash equilibrium. □

For small values of the basic reproductive ratio, there is a unique ESV strategy for each morbidity relative risk. Hence, there is a single vaccination scenario corresponding to the unique ESV strategy, and so our result has similar qualitative features, but not the same quantitative features, to the results obtained by Bauch and Earn (2004).

4.3.2 Vaccination Scenarios for Large Basic Reproductive Ratios

Here, we characterize the Nash and the ESV strategies for large basic reproductive ratios $R_0 > R_B$ and positive morbidity relative risks.

By Lemma 5, for large basic reproductive ratios $R_0 > R_B$, we observe that $0 < \underline{r} < \overline{r}$. Using Lemma 2, for every $0 < r < \overline{r}$, we will construct below the *high* vaccination strategies $p_H \equiv p_H(r; R_0)$ that are going to be ESV strategies.

For $R_0 \in (R_B, 1/\sigma]$, (i) $0 < p_M < p_c \leq 1$, and so (ii) $\pi|(p_M, p_c)$ is strictly decreasing, and (iii) $\pi|[p_c, 1] = 0$. Let $\pi_D : (p_M, p_c) \to \mathbb{R}^+$ be the strictly decreasing branch of the vaccination-infection risk map $\pi_D = \pi$. For every $0 < r < \overline{r}$, let

$$p_H(r; R_0) = \pi_D^{-1}(r).$$

For $R_0 \in (1/\sigma, R_C)$, (i) $0 < p_M < 1 < p_c$, and so (ii) $\pi|(p_M, 1]$ is strictly decreasing. Let $\pi_D : (p_M, 1] \to \mathbb{R}^+$ be the strictly decreasing branch of the vaccination-infection risk map $\pi_D = \pi$. Observing that $\pi(1) > 0$, let

$$p_H(r; R_0) = \begin{cases} \pi_D^{-1}(r) & \text{if} \quad \pi(1) \leq r < \overline{r} \\ 1 & \text{if} \quad r < \pi(1) \end{cases} .$$

Hence, $p_H(r)$ is a continuous piecewise smooth map with a non-smooth point at $r = \pi(1)$.

For every $R_0 \geq R_C$, the vaccination-infection risk index π is increasing. For every $0 < r < \overline{r}$, let

$$p_H(r; R_0) = 1 .$$

For $R_0 > R_B$, let the *low* vaccination strategy be $p_L \equiv p_L(r; R_0) = 0$, for $r > \underline{r}$. Furthermore, (i) $p_M > 0$, and so (ii) $\pi|[0, p_M]$ is increasing. Let $\pi_N : [0, p_M] \to \mathbb{R}^+$ be the increasing branch of the vaccination-infection risk map $\pi_N = \pi$. Let

$$p_N \equiv p_N(r; R_0) = \pi_N^{-1}(r).$$

Theorem 5 (Large basic reproductive ratios) *For large basic reproductive ratios $R_0 > R_B$ and morbidity relative risks $r > 0$, the Nash equilibria are the following:*

(i) (low-vaccination scenario) *the low ESV strategy $p_L = 0$, for $r > \underline{r}$;*
(ii) (high-vaccination scenario) *the high ESV strategy p_H, for $r < \overline{r}$; and*
(iii) the Nash equilibrium p_N that are not weak ESV strategy, for $\underline{r} \leq r \leq \overline{r}$.

Furthermore, for $R_0 \in (R_B, R_C)$ and $r = \overline{r}$,

(iv) the Nash equilibrium $p_N = p_M$ is a right ESV strategy.

Proof of Theorem 5 By Lemma 2 (i), 0 is an ESV strategy for $r > \pi(0) = \underline{r}$.
By construction, (i) π is strictly decreasing at $P_H(r; R_0)$ or (ii) $P_H(r; R_0) = 1 < \pi(1)$. Hence, by Lemma 2 (ii) and (iii), $P_H(r; R_0)$ are ESV strategies.
By construction, π is increasing at $\pi_N^{-1}(r)$. Hence, by Lemma 2 (iv), $\pi_N^{-1}(r)$ are Nash equilibria that are not ESV strategies.
π is left increasing and right decreasing at p_M because p_M is a o local maximum. Hence, by Lemma 3 (ii) and (iii), p_M is a right ESV strategy but p_M is a weak left ESV strategy.
Furthermore, there is no other Nash equilibrium. □

For large values of the basic reproductive ratio $R_0 > R_B$, we have three cases to consider (i) $r < \underline{r}$; (ii) $\underline{r} < r < \overline{r}$; and (iii) $r > \overline{r}$:

(i) for morbidity relative risks below the low morbidity threshold $r < \underline{r}$, there is only one ESV strategy that is the high-vaccination scenario, but for some diseases, like influenza, there might be the need to take another actions, like to vaccine the recovered individuals, because there might not be a critical probability $p_c \leq 1$ that will eradicate the disease.
(ii) for morbidity relative risks between the low and high morbidity thresholds $\underline{r} < r < \overline{r}$, there are three Nash equilibria with two of them being ESV strategies. This phenomenon is not captured by the SIR model, studied in Bauch and Earn (2004), and introduces two scenarios with relevant and opposite features: the low-vaccination scenario where individuals will vaccinate with a low probability; and the high-vaccination scenario where individuals will vaccinate with a high probability.
(iii) for morbidity relative risks above the high morbidity threshold $r > \overline{r}$, there is only one ESV strategy that is the low-vaccination scenario, and so there is the urge to do a vaccination program that reduces the morbidity relative risk, to promote vaccination for part of the population and, if possible, to introduce a new vaccine.

5 Evolutionary Vaccination Dynamics

Based on the replicator dynamics theory, we introduce on the SIRI model the evolutionary vaccination dynamics for an homogeneous vaccination strategy p of the population, where the individuals change their strategies along time, such that their payoffs increase (Maynard-Smith 1982; Hofbauer and Sigmund 1998; Bauch 2005; Nowak 2006; Cojocaru et al. 2007).

For the evolutionary vaccination dynamics, we prove that: (i) the ESV strategies are attractors of the dynamics; and (ii) the Nash equilibria that are not ESV strategies are boundaries of the basin of attractions of the ESV strategies.

Consider that a small group, of size ε, opts to change its vaccination strategy from the population vaccination strategy P to $P + \Delta P$. The payoff gain function satisfies

$$\frac{\Delta E_{P\to(P+\Delta P)}}{\Delta P} = \frac{E(P+\Delta P, p(\varepsilon)) - E(P, p(\varepsilon))}{\Delta P} = \pi(p(\varepsilon)) - r \ , \quad (14)$$

where

$$p(\varepsilon) = (1-\varepsilon)P + \varepsilon(P+\Delta P) = P + \varepsilon\Delta P.$$

The evolutionary vaccination dynamics is given by

$$\frac{\mathrm{d}p}{\mathrm{d}\tau} = \alpha(p) \lim_{\Delta P\to 0} \frac{\Delta E_{P\to(P+\Delta P)}}{\Delta P} = \alpha(p)(\pi(p) - r) \ , \quad (15)$$

where $\alpha(p) \geq 0$ is a smooth map that measures the *vaccination strategy adaptation speed* of the population and might depend upon the parameters of the SIRI model and of the relative morbidity risk r.

A point p is a *dynamic equilibrium* if, and only if, $\mathrm{d}p/\mathrm{d}\tau = 0$. Hence, a point p is a dynamic equilibrium if, and only if,

$$\text{(i)} \quad \alpha(p) = 0 \quad \text{or} \quad \text{(ii)} \quad \pi(p) = r \ .$$

As usual, we assume the following on the vaccination strategy adaptation speed α: (i) $\alpha(p) > 0$, for all $0 < p < 1$, and so α does not generate any interior dynamic equilibria; (ii) if $\pi(0) < r$ then $\alpha(0) = 0$ and $\alpha'(0) > 0$, and so p is bounded below by 0; (iii) if $\pi(1) > r$ then $\alpha(1) = 0$ and $\alpha'(1) < 0$, and so p is bounded above 1; (iv) if $\pi(0) > r$ then $\alpha(0) > 0$, and so α does not generate any extra dynamic equilibrium at 0; and (v) if $\pi(1) < r$ then $\alpha(1) > 0$, and so α does not generate any extra dynamic equilibrium at 1.

A dynamic equilibrium p is a *left (resp. right) attractor* with basin of attraction $B = (q_l, p]$ (resp. $B = [p, q_r)$), if, for all $q \in B$,

$$\lim_{t\to+\infty} p(t; q) = p.$$

A dynamic equilibrium p is an *attractor*, if p is a left attractor and a right attractor. A dynamic equilibrium p is a global attractor, if its basin of attraction is $B = [0, 1]$.

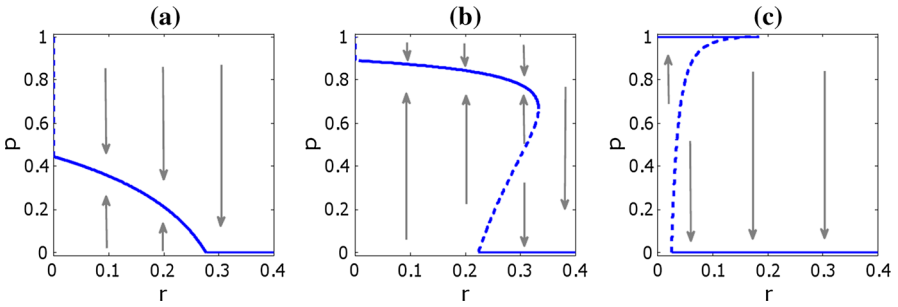

Fig. 7 The stable (*solid line*) and the unstable (*dashed line*) equilibria of ODE (15) for the basic reproductive numbers $R_0 = 1.5$ (a), $R_0 = 3$ (b), and $R_0 = 4.01$ (b). The other parameters are $f = 0.001$ and $\sigma = 0.25$ (Color figure online)

In Fig. 7, we show the evolutionary vaccination dynamics for different vaccination scenarios.

Theorem 6 (Free of morbidity relative risks) *Suppose that the disease is free of morbidity relative risks $r = 0$. The dynamic equilibria are the following:*
For $R_0 < 1/\sigma$:

(i) the Nash equilibria $p^* \in [p_c, 1]$ are equilibria points; and
(ii) the left ESV p_c is a left attractor, whose basin of attraction is $[0, p_c]$.

For $R_0 \geq 1/\sigma$:

(iii) 1 is a global attractor.

In the case of an ideal situation, where the morbidity relative risks $r = 0$, Theorem 6 indicates that, for $R_0 < 1/\sigma$, the vaccination population strategy tends to p_c and so the disease is eradicated. For $R_0 \geq 1/\sigma$, the vaccination population strategy tends to 1, meaning that all susceptible, or newborns, get vaccinated but new measures, like to vaccine the recovered should be taken to eradicate the disease.

Let us introduce the function

$$F(p) = \alpha(p)(\pi(p) - r)$$

to simplify the presentation of the proofs of Theorems 6, 7 and 8.

Proof of Theorem 6 *Case (i)* $R_0 < 1/\sigma$. By Lemma 4, for every $p^* \in [p_c, 1]$, $\pi(p^*) = 0 = r$. Hence, $F(p^*) = 0$, and so p^* is an equilibrium point.
Case (ii) $R_0 < 1/\sigma$. By Lemma 5, for all $q \in [0, p_c)$, $\pi(q) > 0 = r$, and so $F(q) > 0$. Hence,

$$\lim_{t \to +\infty} p(t; q) = p_c,$$

and so p_c is a left attractor with basin of attraction $[0, p_c]$.

Case (iii) $R_0 \geq 1/\sigma$. Either $\pi(1) = 0$ or $\pi(1) > 0$. If $\pi(1) = 0$ then $F(1) = 0$, and so 1 is an equilibrium point. If $\pi(1) > 0$ then $\alpha(1) = 0$, and so 1 is an equilibrium point.
By Lemma 5, for all $q \in [0, 1)$, $\pi(q) > 0$, and so $F(q) > 0$. Hence,

$$\lim_{t \to +\infty} p(t; q) = 1,$$

and so 1 is a global attractor. □

Recall the ESV strategy p_E presented in Theorem 4.

Theorem 7 (Small basic reproductive ratios) *For small basic reproductive ratios* $R_0 \in (1, R_B]$ *and morbidity relative risks* $r > 0$*, the ESV strategy* p_E *is a global attractor.*

For small basic reproductive ratios, the vaccination population strategy tends to p_E. If the morbidity relative risks are small, p_E is close to p_c (the case $r = 0$) and so the disease is close to eradication. However, if the morbidity relative risks are large, then p_E is close to 0 and so the vaccination is not effective. Hence, the vaccination programs have the goal to keep the morbidity relative risks small.

Proof of Theorem 7 *Case (i)* $r > \pi(0)$. Since $p_E = 0$ and $\pi(0) < r$, we get that $\alpha(0) = 0$, and so 0 is a Nash equilibrium. For every $q \in [0, 1]$, $\pi(q) \leq \pi(0) < r$, and so $F(q) < 0$. Hence,

$$\lim_{t \to +\infty} p(t; q) = 0,$$

and so 0 is a global attractor.
Case (ii) $0 < r \leq \pi(0)$. Hence, $\pi(P_E) = r$ and so p_E is a Nash equilibrium. By Lemma 5, for every $q \in [0, p_E)$, $\pi(q) > r$, and so $F(q) > 0$. Hence,

$$\lim_{t \to +\infty} p(t; q) = p_E,$$

and so p_E is a left attractor in $[0, p_E)$. By Lemma 5, for every $Q \in (p_E, 1]$, $\pi(q) < r$, and so $F(q) < 0$. Hence,

$$\lim_{t \to +\infty} p(t; q) = p_E,$$

and so p_E is a right attractor in $[0, p_E]$. Therefore, p_E is a global attractor.

□

Recall the low and high ESV strategies p_L and p_H presented in Theorem 5.

Theorem 8 (Large basic reproductive ratios) *For large basic reproductive ratios* $R_0 > R_B$ *and morbidity relative risks* $r > 0$*, the dynamic equilibria are the following:*

(i) for $r > \overline{r}$*, the low ESV strategy 0 is a global attractor;*
(ii) for $r < \underline{r}$*, the high ESV strategy* p_H *is a global attractor;*

(iii) for $\underline{r} < r \leq \overline{r}$, *the low ESV strategy* $p_L = 0$ *is an attractor, whose basin of attraction is*

$$[0, p_N);$$

(iv) for $\underline{r} \leq r < \overline{r}$, *the high ESV strategy* p_H *is an attractor whose basin of attraction is*

$$(p_N, 1].$$

Furthermore, for $R_0 \in (R_B, R_C)$ *and* $r = \overline{r}$,

(v) the Nash equilibrium p_H *is a right attractor.*

Proof of Theorem 8 *Case (i)* $r = \underline{r}$. Hence, $\pi(0) = \underline{r} = r$, and so 0 is a dynamic equilibrium.
Case (ii) $r > \underline{r}$. Hence, $\pi(0) = \underline{r} < r$, and so $\alpha(0) = 0$. Thus, 0 is a dynamic equilibrium.
Case (iia) $r > \overline{r}$. For all $q \in [0, 1]$, $\pi(q) \leq \overline{r} < r$, and so $F(q) < 0$. Hence,

$$\lim_{t \to +\infty} p(t; q) = 0,$$

and so 0 is a global attractor.
Case (iib) $\underline{r} < r \leq \overline{r}$. By Lemma 5, for all $q \in [0, p_N)$, $\pi(q) < r$, and so $F(q) < 0$. Hence,

$$\lim_{t \to +\infty} p(t; q) = 0,$$

and so 0 is an attractor with basin of attraction $[0, p_N)$.
Case (iii) $\underline{r} \leq r \leq \overline{r}$. $\pi(p_H) = r$, and so p_H is a dynamic equilibrium.
Case (iiia) If $p_H < 1$, by Lemma 5, for all $q \in (p_H, 1]$, $\pi(q) < r$, and so $F(q) < 0$. Hence,

$$\lim_{t \to +\infty} p(t; Q) = p_H,$$

and so p_H is a right attractor with basin of attraction $(p_H, 1]$. In particular, $p_H(\overline{r}; R_0)$ is a right attractor.
Case (iiib) $\underline{r} \leq r < \overline{r}$. By Lemma 5, for all $q \in (p_N, p_H)$, $\pi(q) > r$, and so $F(q) > 0$. Hence,

$$\lim_{t \to +\infty} p(t; q) = p_H,$$

and so p_H is a left attractor with basin of attraction $(p_N, p_H]$. Therefore, p_H is an attractor with basin of attraction $(p_N, 1]$

Case (iv) $r < \underline{r}$. For all $q \in [0, 1]$, $\pi(q) \geq \overline{r} > r$, and so $F(q) > 0$. Hence,

$$\lim_{t \to +\infty} p(t; q) = p_H,$$

and so p_H is a global attractor with basin of attraction $[0, 1]$.

□

Let us consider large basic reproductive ratios $R_0 > R_B$ and so $\underline{r} < \overline{r}$. *First period:* Suppose that the true relative morbidity risk $r_T \in (\underline{r}, \overline{r})$ and the population vaccination strategy is in the high-vaccination scenario with P_T. *Second period:* A false vaccine scare occurs and the relative morbidity risk rises above the high morbidity threshold $r_F > \overline{r}$. Hence the vaccination dynamics will lead the population vaccination strategy to the only stable equilibria that is $P_F = 0$. Hence, the individuals get totally unprotected. *Third period:* A vaccination program is implemented to restore the true relative morbidity risk $r_T \in (\underline{r}, \overline{r})$. Now, the population vaccination strategy keeps being trapped in the low-vaccination scenario $P_F = 0$, that is an attractor, and it is not able to move to the high-vaccination scenario. Hence, the vaccination program to be efficient has to offer vaccines or to make the vaccine compulsory to part of the population to increase, in this way, the population vaccination strategy above the probability of the Nash vaccination equilibrium, that is not a weak ESV strategy, P_N. Only now the vaccination dynamics can start helping the population to evolve and reach the initial equilibrium P_T.

We observe that if the true relative morbidity risk is below the low morbidity threshold $r_T < \underline{r}$ then the vaccination program to be efficient might have not to offer vaccines or to make the vaccine compulsory because the vaccination dynamics will drive the population strategy to the high-vaccination scenario. However, it might take too long to achieve the ESV strategy equilibrium and, again, an extra incentive like to offer vaccines or to make the vaccine compulsory for part of the population might be needed.

6 Conclusions

We made an analyzes of the SIRI susceptible, or newborns, vaccination model. For high values of the large basic reproductive ratios R_0, there are the low morbidity threshold $\underline{r}$ and the high morbidity threshold $\overline{r}$ with the following property: For every relative morbidity risk $\underline{r} < r < \overline{r}$ there are two ESV strategies: the low-vaccination scenario where individuals will vaccinate with a low probability; and the high-vaccination scenario where individuals will vaccinate with a high probability. We introduce the evolutionary vaccination dynamics and prove that these two ESV are attractors and have a common boundary point, in their basins of attraction, consisting of a Nash equilibrium that is not an ESV strategy. Hence, the evolutionary vaccination dynamics is bistable. The existence of these two vaccination scenarios contrasts with the previous studies for the SIR model, where there is only a single vaccination scenario for the same level of the morbidity relative risk (Bauch and Earn 2004). The appearance of different vaccination scenarios is due to the partial immunity in the SIRI epidemic

model. We show that the vaccination expected payoff is smaller at the low-vaccination scenario than at the high-vaccination scenario. We study the effect of vaccine scares and the effect of vaccination education programs. A vaccine scare can wrongly increase the perception of the morbidity relative risk above the high morbidity threshold $\overline{r}$ and so the population will change its vaccination strategy moving from the high-vaccination scenario to the low-vaccination scenario. Hence, when a vaccine scare emerges it is very important to have, immediately, an effective vaccination education program to decrease the value of the perceived morbidity relative risk. To drive the population from a low-vaccination scenario to a high-vaccination scenario, we observe that a vaccination education program will have not only to advertise the advantages of the vaccine but will also have to give an incentive to increase the probability of vaccination, like to offer the vaccines or, even, to make it compulsory for part of the population. Future work can consist in analyzing other epidemiological models and do a comparative study with the results obtained for the SIR model and for the reinfection SIRI model. In particular, (i) to consider distinct compartments for recovered and vaccinated individuals to study different levels of immunity after recovery from natural infections and after vaccination (Alexander et al. 2004); (ii) to consider different strains in the epidemic model and to study the effects of imperfect vaccines (Nuño et al. 2005); and (iii) to endogenize the side effects of vaccination or the available information of education campaigns and to introduce higher death rates for infected individuals.

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References

Aguiar M, Kooi B, Stollenwerk N (2008) Epidemiology of dengue fever: a model with temporary cross-immunity and possible secondary infection shows bifurcations and chaotic behaviour in wide parameter regions. Math Model Nat Phenom 3:48–70

Alexander ME, Bowman C, Moghadas SM, Summers R, Gumel AB, Sahai BM (2004) A vaccination model for transmission dynamics of influenza. SIAM J Appl Dyn Syst 3:503–524

Andreasen V, Lin J, Levin SA (1997) The dynamics of cocirculating influenza strains conferring partial cross-immunity. J Math Biol 35:825–842

Basu S, Chapman GB, Galvani AP (2008) Integrating epidemiology, psychology, and economics to achieve HPV vaccination targets. PNAS 105:19018–19023

Bauch CT, Earn DJD (2004) Vaccination and the theory of games. PNAS 101:13391–13394

Bauch CT (2005) Imitation dynamics predict vaccinating behaviour. Proc R Soc Lond B 272:1669–1675

Bauch CT, Bhattacharyya S (2012) Evolutionary game theory and social learning can determine how vaccine scares unfold. PLoS Comput Biol 8(4):e1002452

Buonomo B, d'Onofrio A, Lacitignola D (2008) Global stability of an SIR epidemic model with information dependent vaccination. Math Biosci 216:9–16

Chamchod F, Britton NF (2012) On the dynamics of a two-strain influenza model with isolation. Math Model Nat Phenom 7:49–61

Chen FH (2006) A susceptible-infected epidemic model with voluntary vaccinations. J Math Biol 53:253–272
Cojocaru MG, Bauch CT, Johnston MD (2007) Dynamics of vaccination strategies via projected dynamical systems. Bull Math Biol 69(5):1453–1476
Cojocaru M, Bauch CT (2009) Vaccination strategy dynamics of population groups with distinct perceived probability of infection. J Inequal Pure Appl Math 10(1):1–16
Davies J, Grilli E, Smith A (1983) Influenza A: infection and reinfection. J Hyg (Cambridge) 92:125–127
Ferguson NM, Galvani AP, Bush RM (2003) Ecological and immunological determinants of influenza evolution. Nature 422:428–433
Fine PEM, Clarkson JA (1986) Individual versus public priorities in the determination of optimal vaccination policies. Am J Epidemiol 124(6):1012–1020
Galvani AP, Reluga TC, Chapman GB (2007) Long-standing influenza vaccination policy is in accord with individual self-interest but not with the utilitarian optimum. Proc Natl Acad Sci 104:5692–5697
Gomes MG, White LJ, Medley GF (2004) Infection, reinfection, and vaccination under suboptimal immune protection: epidemiological perspectives. J Theor Biol 228:539–549
Gomes MG, Franco AO, Gomes MC, Medley GF (2004) The reinfection threshold promotes variability in tuberculosis epidemiology and vaccine efficacy. Proc R Soc Lond B 271:617–623
Gomes MG, White LJ, Medley GF (2005) The reinfection threshold. J Theor Biol 236:111–113
Gupta S, Maiden MCJ (2001) Exploring the evolution of diversity in pathogen populations. Trends Microbiol 9:181–185
Hay AJ, Gregory V, Douglas AR, Lin YP (2001) The evolution of human influenza viruses. Philos Trans R Soc London B 356:1861–1870
Heesterbeek JAP (2002) A brief history of R0 and a recipe for its calculation. Acta Biotheor 50:189–204
Hofbauer J, Sigmund K (1998) Evolutionary games and population dynamics. Cambridge University Press, Cambridge
Kermack WO, McKendrick AG (1927) A contribution to the mathematical theory of epidemics. Proc Roy Soc Lond 115:700–721
Lipsitch M (1997) Vaccination against colonizing bacteria with multiple serotypes. Proc Natl Acad Sci USA 94:6571–6576
Liu J, Kochin BF, Tekle YI, Galvani AP (2012) Epidemiological game-theory dynamics of chickenpox vaccination in the USA and Israel. J R Soc Interface 9:68–76
Maynard-Smith J (1982) Evolution and the theory of games. Cambridge University Press, Cambridge
Moghadas SM (2004) Modelling the effect of imperfect vaccines on disease epidemiology. Discrete Contin Dyn Syst Ser B 4:999–1012
Moreira HN, Wang Y (1997) Global stability in an $S \to I \to R \to I$ model. SIAM Rev 39:496–502
Nowak M (2006) Evolutionary dynamics: exploring the equations of life. Belknap Press, Cambridge
Nuño N, Feng Z, Martcheva M, Castillo-Chavez C (2005) Dynamics of two-strain influenza with isolation and partial cross-immunity. SIAM J Appl Math 65:964–982
Palese P, Young JF (1982) Variation of influenza A, B, and C viruses. Science 215:1486–1474
Reluga TC, Bauch CT, Galvani AP (2006) Evolving public perceptions and stability in vaccine uptake. Math Biosci 204(2):185–198
Reluga TC (2009) An SIS epidemiology game with two subpopulations. J Biol Dyn 3:515–531
Reluga TC, Galvani AP (2011) A general approach for population games with application to vaccination. Math Biosci 230(2):67–78
Shim E, Chapman GB, Townsend JP, Galvani AP (2012) The influence of altruism on influenza vaccination decisions. J R Soc Interface 9:2234–2243
Song L-P, Jin Z, Sun G-Q (2011) Reinfection induced disease in a spatial SIRI model. J Biol Phys 37:133–140
Sonoguchi T, Sakoh M, Kunita N, Satsuta K, Noriki H, Fukumi H (1986) Reinfection with influenza A (H2N2, H3N2, and H1N1) viruses in soldiers and students in Japan. J Infect Dis 153:33–40
Stollenwerk N, Jansen V (2010) Population biology and criticality: from critical birth-death processes to self-organized criticality in mutation pathogen systems. World Scientific, Sigapore
Stollenwerk N, Martins J, Pinto A (2007) The phase transition lines in pair approximation for the basic reinfection model SIRI. Phys Lett A 371:379–388
Stollenwerk N, van Noort S, Martins J, Aguiar M, Hilker F, Pinto A, Gomes MG (2010) A spatially stochastic epidemic model with partial immunization shows in mean field approximation the reinfection threshold. J Biol Dyn 4:634–649

Tudor D (1990) A deterministic model for herpes infections in human and animal populations. SIAM Rev 32:136–139
van den Driessche P, Wang L, Zou X (2007) Modeling diseases with latency and relapse. Math Biosci Eng 4:205–219
van den Driessche P, Zou X (2007) Modeling relapse in infectious diseases. Math Biosci 207:89–103