

A mean-field vaccination game scheme to analyze the effect of a single vaccination strategy on a two-strain epidemic spreading

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Abstract. We propose a mean-field vaccination game framework that combines two distinct processes: the simultaneous spreading of two strains of an influenza-like disease, and the adoption of vaccination based on evolutionary game theory presuming an infinite and well-mixed population. The vaccine is presumed to be imperfect such that it shows better efficacy against the original (resident) strain rather than the new one (mutant). The vaccination-decision takes place at the beginning of an epidemic season and depends upon the vaccine-effectiveness along with the cost. Additionally, we explore a situation if the original strain continuously converts to a new strain through the process of mutation. With the aid of numerical experiments, we explore the impact of vaccinating behavior on a specific strain prevalence. Our results suggest that the emergence of vaccinators can create the possibility of infection-prevalence of the new strain if the vaccine cannot bestow a considerable level of efficiency against that strain. On the other hand, the resident strain can continue to dominate under large-scale vaccine

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A mean-field vaccination game scheme to analyze the effect of a single vaccination strategy avoidance. Moreover, in the case of continuous mutation, the vaccine efficacy against the new strain plays a pivotal role to control the disease prevalence. We successfully obtain phase diagrams, displaying the infected fraction with each strain, final epidemic size, vaccination coverage, and average social payoff considering two-different strategy-update rules and provide a comprehensive discussion to get an encompassing idea, justifying how the vaccinating behavior can affect the spread of a disease having two strains.

Highlights

–We build a mean-field vaccination game scheme to analyze the effect of an imperfect vaccine on a two-strain epidemic spreading taking into account individuals' vaccination behavior.

–En masse vaccine avoidance can enhance the possibility of the original strain prevalence.

–Propensity for vaccination can create the possibility of infection by the new strain if the vaccine is unable to provide a considerable level of efficiency against that strain.

Keywords: evolution models, evolutionary processes, epidemic modeling, evolutionary game theory

Supplementary material for this article is available [online](#)

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1. Introduction

The endemic of infectious diseases is regarded as one of the major menaces to the contemporary human societies as it causes significant damage to humanity regardless of age, sex, lifestyle, ethnic background, and socioeconomic status as well as imposes a financial burden to the society. In modern times, infectious disease outbreaks can spread across the world owing to the prevalence of rapid travel athwart borders [1, 2]. Many of these diseases, such as Pertussis, HIV, Measles, Influenza, etc. have been encumbering us for many years while new diseases, such as Ebola, SARS, and Zika virus, continue to appear from animal to human or spreading to new human populations because of climate change or other anthropogenic disruptions [3]. Pre-emptive vaccination, if available, is considered one of the most preventive measures to evade the spread of infectious disease as well as reduce morbidity and mortality [4–6]. Moreover, vaccination can also diminish the risk of nonvaccinated individuals becoming infected through a voluntary vaccination strategy that depends on human behavior and decisions. Before the outbreak of the epidemic, every individual can choose to take the vaccine or expose herself/himself to the risk of infection. Individual's decision on committing vaccination or not depends on vaccination cost, infection cost, self-interest, the risk of vaccination, and behavior of surroundings [7]. Consequently, a certain percentage of the population goes into the vaccinated class, whereas others remain in the susceptible class [8]. If the herd immunity is achieved, even the non-vaccinators can elude an infection as most of the people surrounding them are immune from infection, cosequently, creating 'free-riders' who pay none of the cost but benefit from the herd immunity as 'public goods'. This situation reveals the so-called 'vaccination dilemma' that can be modeled by mathematical social dilemma games [9]. Modeling the disease dynamics coupling with the complicated natures of human behavior on infection control has therefore become an area of the future complex systems research to better understand the epidemics in modern societies [10–12].

Along with the growing interest in evolutionary game theory and epidemiology, vaccination game [5–17] has been playing an important role to analyze the effect of vaccination on various infectious diseases taking into account the vaccinating behavior of individuals. In addition to vaccination, some studies also focus on analyzing the effect of intermediate defense measures [1, 2, 20, 22] as well as information awareness [7, 23–25] to suppress Influenza-like disease spreading. Most of the studies regarding vaccination game presume perfect immunity from vaccination (for example, [2, 13, 18, 19, 22, 26]), which sounds somewhat idealized. However, in reality, not all vaccines are entirely effective or perfect. Notably, the unavoidable primary vaccine failure rates have been found to range from 2 to 50 percent for licensed vaccines under ideal circumstances in clinical trials [3, 27]. Moreover, imperfect vaccine can trigger pathogen virulence that makes the situation worse. Some of the previous works concerning imperfect vaccination and its consequences on pathogen virulence can be found in [28–32]. These issues have intrigued researchers to incorporate imperfect vaccine provision in disease modeling. However, exploring the imperfect vaccine complication on vaccine uptake behavior via game theoretical perspective has attracted significant attention, because apart from the perceived cost of vaccination with the infection risk, vaccine effectiveness plays an important role towards vaccination decisions. Some of

the contributions relating imperfect vaccination provision in vaccination game can be perceived from the previous literatures such as [20, 33–36], albeit more understandings are due in this context. Recently, Kuga and Tanimoto [20] presented a mean-field approximation (in well-mixed population) as well as multi-agent simulation (MAS) framework of vaccination game dovetailed with the imperfect vaccination provision and intermediate defense measure. The authors mainly adopted the imitation dynamics of vaccination behavior proposed by Fu *et al* [26], and successfully developed an equivalent approach in mean field approximation framework. They also examined the same scenario presuming heterogeneous spatial structures in the population [21]. Another recent work regarding imperfect vaccination together with game theoretical approach has been developed by Chen and Fu [34] concerning the stability and bifurcation analysis taking into account well-mixed as well as spatial populations. Interestingly, the dynamics of epidemic spreading (with imperfect vaccine) in both [20, 34] are almost similar, albeit having difference in the game approach. All these studies are devoted to examining the effect of vaccination on a single infection spreading.

However, there is increasing evidence that many diseases are caused by more than one pathogen strains, such as Influenza, dengue fever, HIV-AIDS, and some sexually transmitted diseases. The multiple variants of the pathogen appear mostly because of the mutation or antigenic drift in the viral genome. Viruses, like HCV (hepatitis C virus), Influenza, are highly mutable, consequently generating multiple strains. Moreover, vaccination against mutable viruses is not very efficient. For example, Influenza A viruses mutate continuously and create new virus strains to which the host has only partial or no immunity at all and can be reinfected with the disease [37]. These factors have impelled researchers to formulate the dynamics of epidemic models with multiple strains.

To date, there are significant studies available, investigating the two-strain epidemic model with single vaccination policy. For instance, Rahman and Zou [38] analyzed the effect of a single vaccination on a two-strain SIR like flu model. This is worth noting that the original model in this regard is proposed by Castillo-Chavez *et al* [39]. Cai *et al* [40], in addition to examining the effect of single vaccination, explored the two-strain epidemic model considering mutation between strains. Nonetheless, these works disregard the vaccinating behavior of individuals rather focus on the stability analysis at equilibrium.

So far, there are a few studies available, investigating the influence of vaccinating behavior on two-strain disease spreading. A recent work in this avenue is presented by Pharaon and Bauch [41], where they explore the influence of social behavior on pathogen virulence. The authors consider the perceived severity of pathogen strains and the efficacy of infection control to affect the transmission rate of the disease strains. However, they avoid the long-term evolutionary process with repeated epidemic seasons. In our current study, we intend to include the vaccine efficiency and the relative cost of vaccination with respect to infection cost to affect an individual's vaccinating behavior. Unlike [41], we incorporate an evolutionary game theoretic framework that considers repeated epidemic seasons to observe an overall scenario of infection spreading and the extent of vaccination coverage required to control a disease spreading.

To this aim, we investigate the effect of a single vaccination policy on a two-strain SIR like epidemic model considering a mean-field approximation framework of

vaccination game presuming an infinite and well-mixed population. We mainly adopt the scheme developed by Kuga and Tanimoto [20], where individuals are allowed to change their strategies (vaccinate or not) at the beginning of an epidemic season based on last season's experience. Furthermore, taking into account the factors regarding vaccine effectiveness against disease strains mentioned earlier (as in [37]), we incorporate the notion of imperfect vaccination in epidemic dynamics, where vaccine bestows better effectiveness against the original or resident strain (strain 1) than the new or mutant (strain 2) strain. Moreover, we inspect our results considering the absence and presence of mutation. The absence of mutation means both strains are not directly connected but they compete through susceptible people, whereas in the presence of mutation, genetic changes give a competitive advantage to the mutant strain [37]. In our game approach, we integrate this epidemiological process into the imitation dynamics [13, 26] of vaccination behavior where individuals use anecdotal information to estimate costs and benefits of vaccination.

The rest of this article is organized as follows: section 2 presents a detailed description of our methodology together with a SI_1I_2R/V epidemic model; section 3 provides an inclusive discussion supported by a series of phase diagrams generated from the numerical simulation, and finally, section 4 concludes the findings of this paper.

2. Methods and model description

As discussed above, here we combine two dynamical processes, namely the disease spreading via a two-strain epidemic model in a local time scale, and the evolution of vaccination decision-making process in a global time scale (figure 1). Usually, an epidemic season ends when the number of infected individuals becomes zero. At the end of each epidemic season, we estimate six fractions of individuals comprising vaccinated and healthy, vaccinated but infected with either strain, non-vaccinated but healthy, and non-vaccinated and infected with either strain. As we are considering influenza-like disease, the number of vaccinees remains fixed during an epidemic season, and the vaccination decision takes place at the beginning of each epidemic season by comparing payoffs (obtained in the last season) among the relevant groups of individuals. The imitation process is being undertaken with the help of pairwise Fermi functions. Finally, the fraction of vaccinators is updated using the evolutionary dynamics given in section 2.4. This process is repeated for several seasons until we reach a steady state.

2.1. Disease spreading

The dynamics of a two-strain epidemic model coupled with the imperfect vaccination policy is explored where the individuals in a well-mixed and infinite population are classified into five compartments: susceptible— S , vaccinated— V , infected with original strain (strain one)— I_1 , infected with new strain (strain two)— I_2 , and recovered— R (figure 1). The model has the following parameters: β_1, β_2 —transmission rate of infection with strain one and strain two, respectively; ρ —mutation rate from strain 1 to strain 2; γ_1, γ_2 —recovery rate for infected individuals with strain one and strain two,

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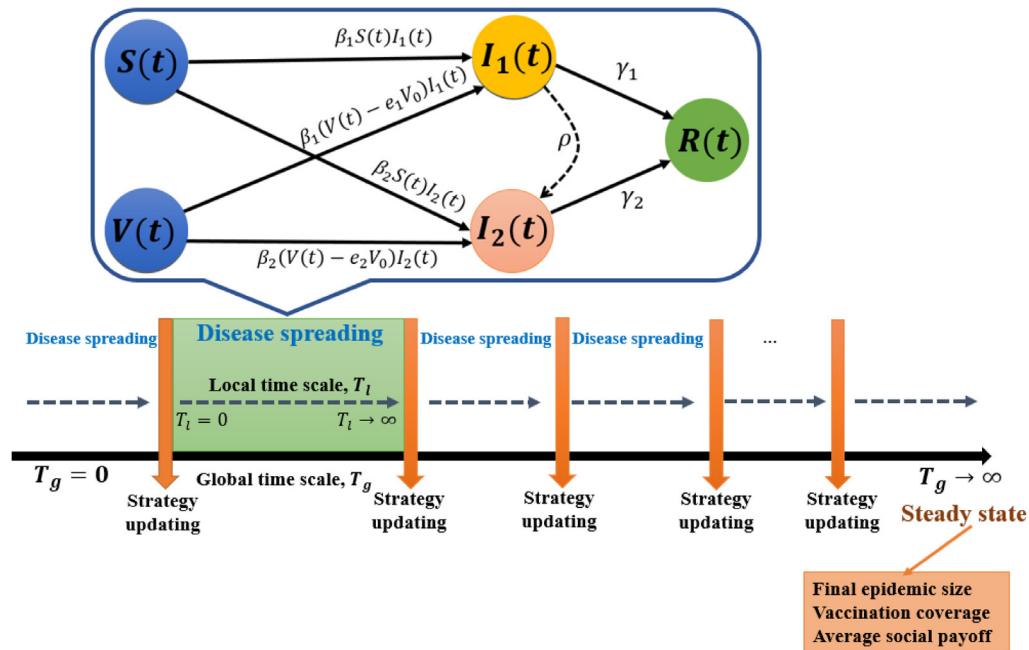


Figure 1. The schematic of the whole dynamic setup. The disease spreading process takes place at each local time scale (T_l) which presumes an *SIR*-like model having two strains and a single vaccination provision. The dashed arrow denotes the mutation from strain 1 to strain 2 with the rate ρ . The parameters e_1 ($0 \leq e_1 \leq 1$) and e_2 ($0 \leq e_2 \leq e_1$) represent the vaccine effectiveness against strain 1 and strain 2, respectively. $V(t) - e_i V_0$, $i = 1, 2$ (V_0 is the fraction of vaccinators at the beginning of an epidemic season) denotes the fraction of vaccinators susceptible to strain i at time t , where $V(t)$ being the vaccinators present in the V state at time t . Notably, the proportion of individuals remain in the S and V compartments at the end of a local time scale are ‘successful free riders’ and ‘healthy-vaccinated people’, respectively. The change of strategies takes place at the end of each local time scale. The process is repeated until the system reaches a steady state on a global time scale (T_g).

respectively. We assume that strain 1 can be converted to strain 2 within host through an antigenic shift. The process is known as mutation [42]. Usually, the mutant strain has stronger virulence to transmit disease, therefore, a vaccinated individual conceivably has more possibility of becoming infected after being in contact with an individual infected with strain two [40]. This is why we presume that the vaccine provides better effectiveness against the original strain (strain 1) than that of the new strain (strain 2). We further assume that an individual cannot be infected by both strains at once and that if anyone infected with either strain, she/he can never be infected with another strain in the same season, that is, there is no co-infection and super-infection. The susceptible people are divided into two subclasses namely, nonvaccinated and vaccinated. As vaccine is presumed imperfect, some vaccinators still can be infected by either strain. Hence, we include the vaccine effectiveness in our disease modeling. The vaccinated individuals are separated into two subclasses: perfect immune individuals and non-immune individuals; non-immune individuals become susceptible to either strain. Here we choose two different effectiveness parameters, e_1 and e_2 ($0 \leq e_1, e_2 \leq 1$) against the original and new strain, respectively but with the condition, $e_1 \geq e_2$. At any time

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t , five fractions of individuals are denoted as, $S(t)$, $V(t)$, $I_1(t)$, $I_2(t)$, and $R(t)$. As the vaccine is assumed pre-emptive, the number of vaccinees remains same during an epidemic season. If $V(0) = V_0$ is the fraction of vaccinators at the start of an epidemic season, then a proportion e_i ($i = 1, 2$) of V_0 , that is, $e_i V_0$ is the fraction of vaccinators who get immunity from strain i ($i = 1, 2$) but the proportion $(V(t) - e_i V(0))$ still remain susceptible to strain i , where $V(t)$ is the fraction of vaccinators in the V compartment at time t . Figure 1 illustrates the layout of the SI_1I_2R/V epidemic model along with the whole dynamical system. The dynamics of the epidemic model integrated with the imperfect vaccine policy can be presented by a system of five ordinary differential equations (ODEs),

$$\frac{dS(t)}{dt} = -\beta_1 S(t) I_1(t) - \beta_2 S(t) I_2(t), \quad (1)$$

$$\frac{dV(t)}{dt} = -\beta_1 (V(t) - e_1 V(0)) I_1(t) - \beta_2 (V(t) - e_2 V(0)) I_2(t), \quad (2)$$

$$\frac{dI_1(t)}{dt} = \beta_1 (S(t) + V(t) - e_1 V(0)) I_1(t) - \gamma_1 I_1(t) - \rho I_1(t), \quad (3)$$

$$\frac{dI_2(t)}{dt} = \beta_2 (S(t) + V(t) - e_2 V(0)) I_2(t) - \gamma_2 I_2(t) + \rho I_1(t), \quad (4)$$

$$\frac{dR(t)}{dt} = \gamma_1 I_1(t) + \gamma_2 I_2(t), \quad (5)$$

where ρ is a non-negative parameter depicting the mutation rate from strain 1 to strain 2. The parameter, $\rho = 0$ means the absence of mutation. All the parameters: $\beta_1, \beta_2, \gamma_1$, and γ_2 , are assumed as positive constants. As the state space variables in our epidemic model (equations (1)–(5)) represent the fractions of the total population at each state, we do not consider a population of any particular size, i.e. $S(t) + V(t) + I_1(t) + I_2(t) + R(t) = 1$. The initial conditions are: $V(x, 0) = x$, and $S(x, 0) = 1 - x$, where x denotes the fraction of vaccinators at the beginning of a season.

Basic reproduction ratio: Many epidemiological models possess a threshold parameter known as the basic reproduction ratio (or number) that yields the average number of secondary infections caused by a single infection in a completely susceptible population [5]. Following the [43], the vaccine-dependent basic reproduction ratio corresponding to each strain of the epidemic model (1)–(5) can easily be derived as,

$$\left. \begin{aligned} R_0^1 &= \frac{\beta_1}{\gamma_1 + \rho} (S(0) + (1 - e_1)V(0)) \\ R_0^2 &= \frac{\beta_2}{\gamma_2} (S(0) + (1 - e_2)V(0)) \end{aligned} \right\} \quad (6)$$

where $R_0^i, i = 1, 2$, denotes the basic reproduction number of the strain i . Notably, if $R_0^1 < 1$ and $R_0^2 < 1$, then both strains die out, that is, a stable disease free equilibrium (DFE) exists, however, if any of these parameters possesses a value more than one, the DFE is no longer stable. The case when $R_0^1 > 1$, $R_0^2 > 1$ but $R_0^1 > R_0^2$ (or $R_0^1 < R_0^2$), strain 1 (strain 2) dominates strain 2 (strain 1), and accordingly stronger strain outperforms

Table 1. Six fractions of individuals at the end of an epidemic season with their respective payoffs (within brackets).

Strategy/state	Healthy	Infected with strain 1	Infected with strain 2
Vaccinated (V)	HV($-C_r$)	$I_1V(-C_r - 1)$	$I_2V(-C_r - 1)$
Non-vaccinated (NV)	SFR(0)	FFR ₁ (-1)	FFR ₂ (-1)

the weaker one and thereby weaker one dies out eventually. If both parameters are equal but greater than one, then both strains coexist in the population [5]. We will examine the effects of these vaccine-dependent basic reproduction ratios in our outlined evolutionary process.

2.2. Payoff structure

The non-vaccinated and infected (with either strain) individuals, called ‘failed free riders’, are supposed to carry out an infection cost C_i . However, non-vaccinated individuals who remain healthy during the epidemic season, that is, avoiding both infections, are termed as, ‘successful free rider’ incurring no cost at all. To evaluate each individual’s payoff, the cost is rescaled by defining a relative cost of vaccination as, $C_r = C_v/C_i$; ($0 \leq C_v \leq 1$; $C_i = 1$) without loss of generality. Therefore, a vaccinator who remains healthy during the epidemic incurs a relative cost $-C_r$, while a vaccinated but infected individual carrying out a cost of $-C_r - 1$.

Because of analytical complexity in solving the model (equations (1)–(5)), we estimate several fraction of individuals by considering the flux $\varphi_{A \rightarrow B}$, which indicates the total fraction of individuals transferring from state A to state B . In our case, $\varphi_{S \rightarrow I_1}(x, \infty)$, $\varphi_{S \rightarrow I_2}(x, \infty)$, $\varphi_{V \rightarrow I_1}(x, \infty)$, and $\varphi_{V \rightarrow I_2}(x, \infty)$ are representing the flux from non-vaccinated to infected with strain 1, non-vaccinated to infected with strain 2, vaccinated to infected with strain 1 (I_1V), and vaccinated to infected with strain 2 (I_2V), respectively at equilibrium. However, the successful free riders (SFR), denoted by $S(x, \infty)$, are the fraction who remain in the S state at equilibrium ($t \rightarrow \infty$), and the failed free riders, denoted by FFR _{i} , $i = 1, 2$, are the fractions $\varphi_{S \rightarrow I_1}(x, \infty)$, and $\varphi_{S \rightarrow I_2}(x, \infty)$, that is, the non-vaccinators who get infected by a strain i ($i = 1, 2$). The individuals remaining in the V state at equilibrium ($V(x, \infty)$) are the healthy vaccinated people (perfectly immune) denoted by HV. All these fractions with their respective payoffs are summarized in table 1.

Using the estimated fractions together with their payoffs depicted in table 1, we can evaluate the average social payoff, π , the average payoff for vaccinators (cooperators), $\langle \pi_c \rangle$, and the average payoff for non-vaccinators (defectors), $\langle \pi_D \rangle$ as follows:

$$\pi = -C_r HV - (C_r + 1)(I_1V + I_2V) - (\text{FFR}_1 + \text{FFR}_2), \quad (7)$$

$$\langle \pi_c \rangle = (-C_r HV - (C_r + 1)(I_1V + I_2V))/x, \quad (8)$$

$$\langle \pi_D \rangle = -(\text{FFR}_1 + \text{FFR}_2)/(1 - x). \quad (9)$$

2.3. Strategy update

As mentioned before, the strategy update takes place at the beginning of each epidemic season depending upon the estimated payoff attained in the last season. Here, we consider two different strategy update rules proposed by Fu *et al* [26] and Fukuda *et al* [13]. Although these rules were mainly proposed for the agent-based modeling, the present study, however, does not consider any spatial structure in the population rather relying on the so-called mean-field approximation, where agents are indistinguishable, and all players in the same state adopt the same strategy [44].

2.3.1. Individual-based risk assessment (IB-RA). In agent-based modeling, an agent, i (say), mimics one of its randomly chosen neighbor, j 's (say) strategy by comparing their payoffs π_i and π_j , respectively. The probability that player i (having strategy S_i) imitates j 's strategy, S_j , can be estimated by the well-known pair-wise Fermi function [45, 46] as follows:

$$P(S_i \leftarrow S_j) = \frac{1}{1 + \exp[-(\pi_j - \pi_i)/k]}, \quad (10)$$

where the parameter $k > 0$ signifies the strength of selection; smaller k characterizes more sensitivity to the payoff difference. An archetypal choice of k is 0.1 that has been used in many previous studies (such as, [1, 2, 7, 20]). Although Fu *et al* [26] originally adopted this Fermi function for a structured population, in a later work, Fukuda *et al* [13] named this rule as individual-based risk assessment (IB-RA) as the interaction is one to one. Following [20], we adopt this rule in our mean-field game approach, wherein, instead of agent to agent interaction, every fraction (depicted in table 1) assesses its payoff with the other relevant fractions according to the formula (10). For instance, vaccinated and healthy (HV) individuals compare their payoff with the successful free riders (SFR) by the probability,

$$P(\text{HV} \leftarrow \text{SFR}) = \frac{1}{1 + \exp[-(0 - (-C_r))/k]}.$$

These transition probabilities affect the evolution of the fraction of vaccinators.

2.3.2. Strategy-based risk assessment (SB-RA). This rule was proposed by Fukuda *et al* [13] for the agent-based modeling, where a focal player compares its payoff with the socially averaged payoff of all the agents surrounding it having a strategy different from the focal player. Unlike IB-RA, this rule permits an individual to compare her/his strategy with the society rather than a single individual. Therefore, the formula for SB-RA becomes,

$$P(S_i \leftarrow S_j) = \frac{1}{1 + \exp[-(\langle \pi_j \rangle - \pi_i)/k]}, \quad (11)$$

where $\langle \pi_j \rangle$ is the average payoff attained by averaging payoffs of all neighbors who adopt the same strategy S_j , of a randomly selected neighbor j of an individual i . Nevertheless, in our mean-field game, we adopt this rule by comparing a particular fraction's (say, HV, belonging to vaccinators group) payoff with the average payoff of other fractions that are belonging to a separate or mutually exclusive group (say, NV-non-vaccinators

group). For example, HV fraction compares its payoff ($-C_r$) with the average payoff of non-vaccinators group ($\langle \pi_D \rangle$) defined in (9) by the formula,

$$P(HV \leftarrow NV) = \frac{1}{1 + \exp[-(\langle \pi_D \rangle - (-C_r))/k]}.$$

Accordingly, all fractions assess their payoff with the average payoff of their corresponding mutually exclusive group. All the relevant transition probabilities regarding IB-RA and SB-RA can be found in the supplementary material (available online at stacks.iop.org/JSTAT/2020/033501/mmedia).

2.4. Evolutionary dynamics

The evolutionary dynamics yield the quantitative rate of change of vaccinators for the next generation by incorporating all possible transition probabilities defined in (10)–(11). Equations (12) and (13), respectively, represent the dynamical equations corresponding to IB-RA and SB-RA case. Notably, these equations have been extended from the master equation of the mean-field approach used previously, for example in [47], to estimate the temporal evolution of the cooperators' (vaccinators') density.

IB-RA case:

$$\begin{aligned} \frac{dx}{dt} = & HV \cdot SFR (P(SFR \leftarrow HV) - P(HV \leftarrow SFR)) + HV \cdot FFR_1 (P(FFR_1 \leftarrow HV) - P(HV \leftarrow FFR_1)) \\ & + HV \cdot FFR_2 (P(FFR_2 \leftarrow HV) - P(HV \leftarrow FFR_2)) + I_1V \cdot SFR (P(SFR \leftarrow I_1V) - P(I_1V \leftarrow SFR)) \\ & + I_1V \cdot FFR_1 (P(FFR_1 \leftarrow I_1V) - P(I_1V \leftarrow FFR_1)) + I_1V \cdot FFR_2 (P(FFR_2 \leftarrow I_1V) - P(I_1V \leftarrow FFR_2)) \\ & + I_2V \cdot SFR (P(SFR \leftarrow I_2V) - P(I_2V \leftarrow SFR)) + I_2V \cdot FFR_1 (P(FFR_1 \leftarrow I_2V) - P(I_2V \leftarrow FFR_1)) \\ & + I_2V \cdot FFR_2 (P(FFR_2 \leftarrow I_2V) - P(I_2V \leftarrow FFR_2)). \end{aligned} \quad (12)$$

SB-RA case:

$$\begin{aligned} \frac{dx}{dt} = & -HV \cdot NV \cdot P(HV \leftarrow NV) - I_1V \cdot NV \cdot P(I_1V \leftarrow NV) - I_2V \\ & \cdot NV \cdot P(I_2V \leftarrow NV) + SFR \cdot V \cdot P(SFR \leftarrow V) \\ & + FFR_1 \cdot V \cdot P(FFR_1 \leftarrow V) + FFR_2 \cdot V \cdot P(FFR_2 \leftarrow V). \end{aligned} \quad (13)$$

3. Result and discussion

The current framework combines two dynamical processes, the disease spreading process demonstrated by equations (1)–(5) and the evolution of the vaccination decision making process supported by equations (12) and (13). That is, the whole dynamical setup is governed by equations (1)–(5) along with equation (12) or (13). The disease spreading process provides the outcome of the epidemic that affects the evolution of vaccination choice. Due to the complexity of evolutionary equations (equations (12) and (13)), here we rely on numerical simulations to analyze our results. The baseline parameters (summarized in table 2) of this study are assumed to represent an influenza-like disease. The transmission rates and recovery rates are chosen to have the basic reproduction number corresponding to either strain as 2.5 (used in several previous studies such as [1, 7, 20, 25]) in the absence of vaccination strategy (or quantitatively when $e_1 = e_2 = 0$).

Table 2. Baseline parameters and their assumed values.

Parameter	Definition	Value
β_1	Transmission rate for strain 1 (per day per person)	0.8333 (assumed)
β_2	Transmission rate for strain 2 (per day per person)	0.75 (assumed)
ρ	Mutation rate (if considered)	0.1 (assumed)
γ_1	Recovery rate from strain 1 (per day)	0.3333 (assumed)
γ_2	Recovery rate from strain 2 (per day)	0.3 (assumed)
e_1	Vaccine effectiveness against strain 1	[0, 1]
e_2	Vaccine effectiveness against strain 2	[0, e_1] (assumed)

3.1. Time series

Before analyzing the impact of vaccinating behavior on strain prevalence, let us first explore the initial strain prevalence on disease dynamics in a single season time series (figure 2), disregarding the vaccination cost ($C_r = 0$) and mutation ($\rho = 0$). It is clear that the higher initial infection by a strain, the higher the risk of predominance of that strain (figures 2(a) and (b)). If we presume same level of initial infection for both strains ($I_1(0) = I_2(0) = 0.0001$), strain 2 appears to dominate on the ground of having significant difference between e_1 and e_2 (figure 2(c)). However, decreasing the vaccine efficacy against strain 1 (say, $e_1 = 0.4$, and $e_2 = 0.3$) reduces the invasion of the second strain (figure 2(d)) rather confirms almost similar risk by each strain, because this will increase the pool of susceptible people available for infection by the resident strain (strain 1) on the ground of $\beta_1 > \beta_2$. It is worth noting that all of our phase plane analyses are based on the assumption, $I_1(0) = I_2(0) = 0.0001$.

3.2. Phase plane analyses

Now we examine how the cost and vaccine efficacy affect individuals' vaccinating behavior, and how that vaccinating behavior influences the strain prevalence. All phase planes (e_2 versus e_1) in figures 3–6 suggest that the sensitivity is mostly coming along the direction of vaccine efficacy against strain 2 (e_2). We note that the transition from blue to red color presumes a transition from the good/better (less infection or disease-free) state to a bad/worse (endemic) situation and vice versa. In general, the lower cost boost up vaccination coverage that helps suppressing the disease and results in a better societal payoff, whereas the higher cost does not enhance vaccine uptake even if the vaccine is highly effective, as a result, brings lower payoff in the social perspective ($(^*-v)$ heat maps in figures 3–6). We mainly split our discussion into two parts according to the absence ($\rho = 0$) and the presence ($\rho \neq 0$) of mutation.

3.2.1. Case I ($\rho = 0$). In this case, both strains are not directly connected but can compete through susceptible individuals. Figure 3 (IB-RA update rule) and figure 5 (SB-RA update rule) display instances for the absence of mutation, where we investigate the infection by each strain, the final epidemic size, the vaccination coverage, and the average societal payoff attained from vaccination as a function of (e_2, e_1) with various cost levels. Here we perceive the influence of cost and vaccine efficacy in

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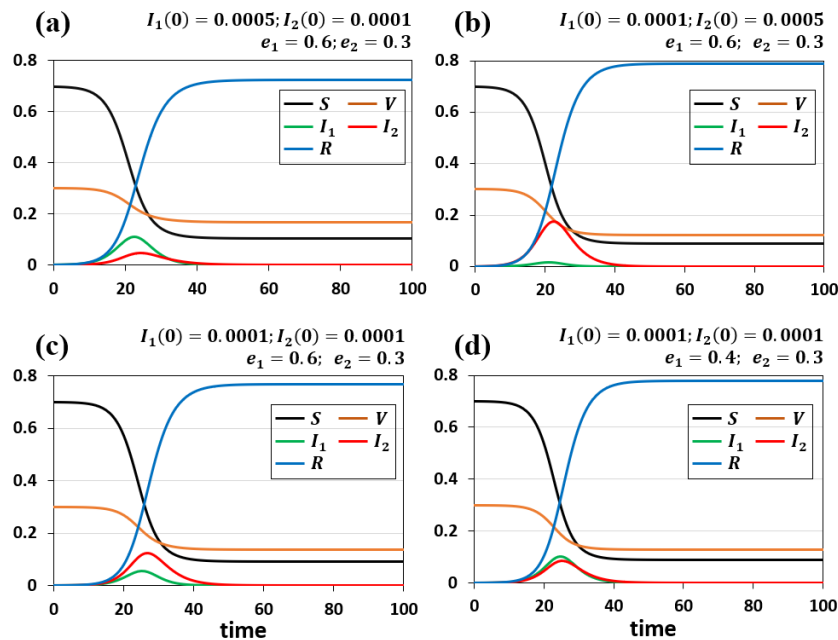


Figure 2. The variation of the dynamics of several fractions of individuals for different initial conditions of $I_1(t)$ and $I_2(t)$ when $V(0) = 0.3$ and $\rho = 0$. (a)–(c) Show the time series with $(e_1, e_2) = (0.6, 0.3)$, whereas (d) displays that for $(e_1, e_2) = (0.4, 0.3)$. Other relevant parameters are considered as in table 2. Clearly the initial infected fraction has an impact on the risk of infection for each strain. Especially, the condition $I_1(0) = I_2(0) = 0.0001$ implies (c) the higher risk of infection with strain 2 than that of strain 1 on the ground of $(e_1, e_2) = (0.6, 0.3)$, however, (d) infection risks of both strains are almost similar whenever $(e_1, e_2) = (0.4, 0.3)$.

every heat map, especially, the vaccine efficacy, e_2 seems to affect more on vaccination coverage (*-iv)) as well as disease prevalence (*-iii)), which is quite conceivable because along with e_1 a satisfactory level of effectiveness against the second strain is necessary to inspire vaccination although this level varies with the cost. With a lower vaccination cost ($C_r = 0.1$), the vaccination coverage seems very low (red-colored) as long as e_2 is below a threshold level. Afterwards it shows a non monotonic tendency with e_2 , i.e. vaccination coverage reaches its maximum with the increase of e_2 and then decreases with the further upsurge of e_2 as some people might want to free ride on herd immunity (triangular regime enclosed by black dotted lines in figure 3(a-iv)) for a more effective vaccine. However, an expensive vaccine ($C_r = 0.8$) does not entail the same tendency as that of the former case because the vaccine uptake for the latter case does not reach to a considerable level that ensures the herd immunity. It is also evident that the threshold level of e_2 to confirm an upsurge of the vaccine uptake is proportional to the cost level (see (*-iii) heat maps in figures 3 and 5). The vaccinated individuals who miss out on immunity are more likely to be infected with strain 2 rather than strain 1 on the ground of $e_1 \geq e_2$. This is why infected fraction with strain 2 contributes more in the final epidemic size (see phase diagrams (a-i)–(a-iii) of figures 3 and 5) in case of lower cost. However, this dominance (strain 2) declines with the increase of cost level (yellowish region enclosed by black dotted lines in (*-ii) heat maps of figures 3 and 5). Because the increase of cost level causes the lower vaccination coverage, consequently,

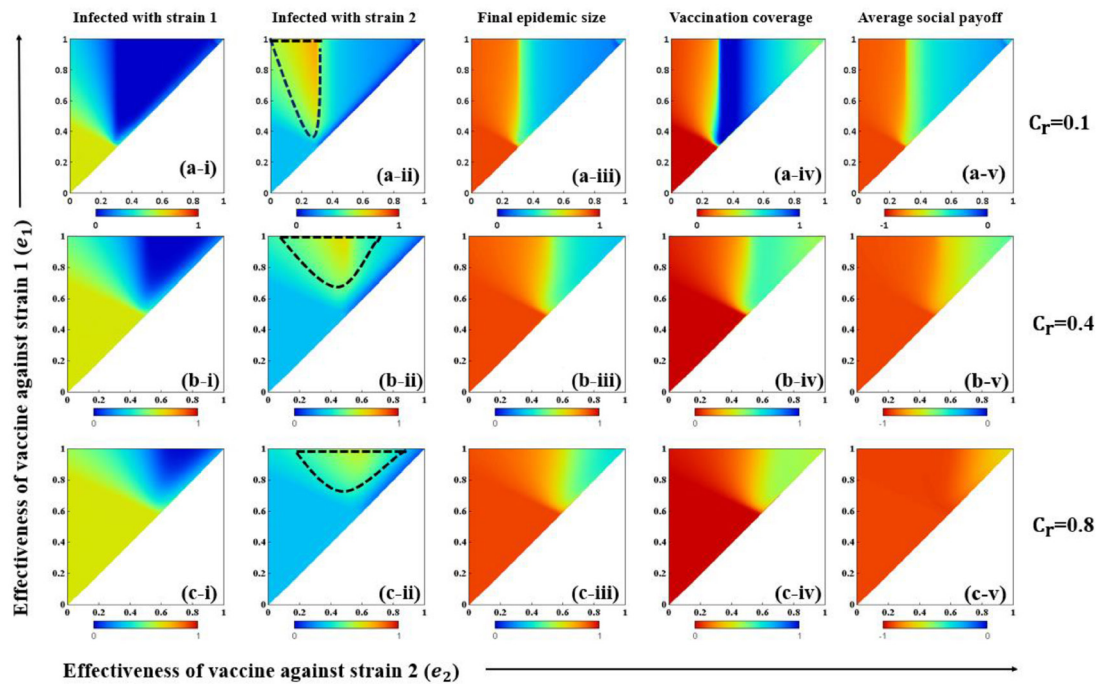


Figure 3. e_2 versus e_1 heat maps with strategy-updating rule IB-RA considering absence of mutation; first two columns represent fraction of infected people with each strain, third column as final epidemic size, fourth and fifth column represent vaccination coverage and average social payoff, respectively. On the other hand, first, second, and third row depict three relative costs as, $C_r = 0.1, 0.4, 0.8$, respectively. Here $\beta_1 = 0.8333, \beta_2 = 0.75, \gamma_1 = 0.3333, \gamma_2 = 0.3$. The regions enclosed by black dashed lines indicate more infections taking place with strain 2 whenever e_1 reaches a satisfactory level (varies with C_r) and e_2 is yet to reach its threshold level (varies with C_r). Each heat map in third, fourth and fifth columns depicts a threshold level of e_2 where the situation gets better. Each heat map assumes $I_1(0) = I_2(0) = 0.0001$.

the fraction of free riders emerge in the population and consequently, the infection with strain 1 becomes more likely than that of strain 2 as $\beta_1 > \beta_2$. This is why the fraction of infection with strain 1 is showing a proportional relationship with the extent of non-vaccinators and the cost level C_r (see the yellowish region in phase diagrams (*-i) of figures 3 and 5). Notably, the average social gain is seen to be better for the low priced vaccine than that of the high priced vaccine that is quite imaginable.

3.2.2. Case II ($\rho = 0.1$). In this case, we assume that strain 1 mutates to strain 2 with a rate $\rho = 0.1$. Figure 4 (IB-RA) and figure 6 (SB-RA) represent several phase planes concerning the infection with each strain, total infection, vaccination coverage and overall societal gain when a strain continuously converts to another type through the process of mutation. The existence of such mutation implies strain 2 to be the sole contributor to the disease prevalence (see (*-ii) heat maps in figures 4 and 6). This is why all phase diagrams are showing the sensitivity along the direction of vaccine efficacy against the mutant strain (e_2). This case also shows a non-monotonic tendency of

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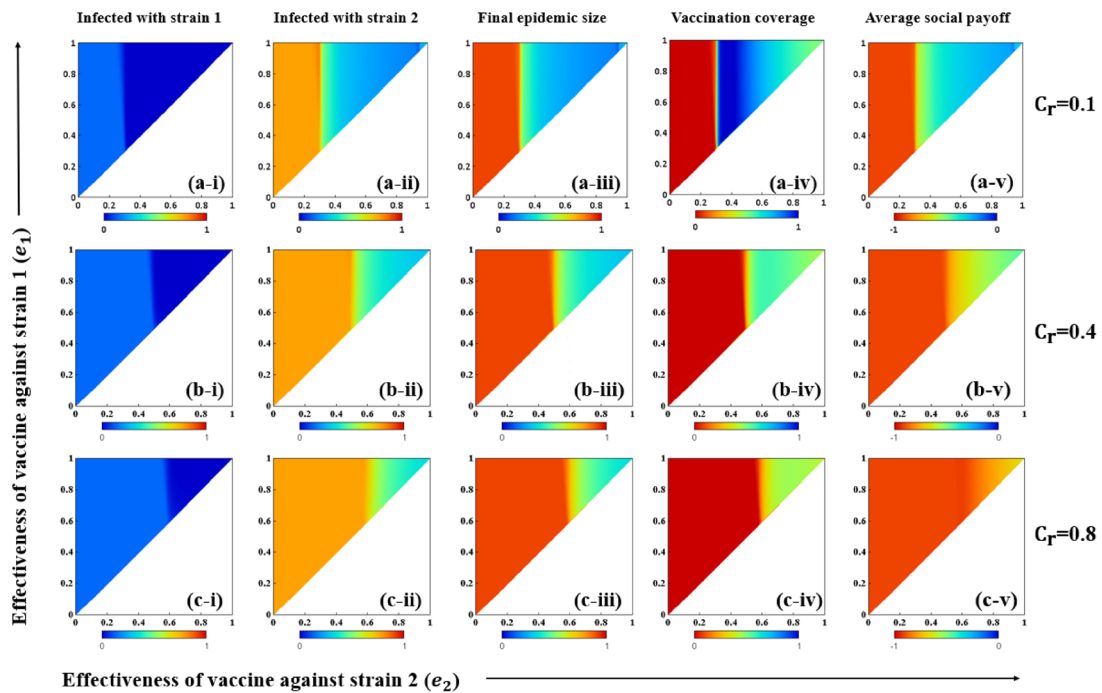


Figure 4. e_2 versus e_1 heat maps with strategy-updating rule IB-RA considering mutation with the rate $\rho = 0.1$; first two columns represent fraction of infected people with each strain, third column as final epidemic size, fourth and fifth column represent vaccination coverage and average social payoff, respectively. On the otherhand, first, second, and third row depict three relative costs as, $C_r = 0.1, 0.4, 0.8$, respectively. Here $\beta_1 = 0.8333, \beta_2 = 0.75, \gamma_1 = 0.3333, \gamma_2 = 0.3$. Each heat map in third, fourth and fifth columns depicts a threshold level of e_2 where the situation gets better. Each heat map assumes $I_1(0) = I_2(0) = 0.0001$.

vaccination coverage with e_2 , especially when the cost is low. Moreover, the level of e_2 to upsurge the vaccination coverage is showing a positive correlation with the cost, C_r .

3.2.3. β_1 versus β_2 phase diagram. We consider an instance with a low-priced vaccine ($C_r = 0.1$), where other relevant parameters are chosen as, $\rho = 0, e_1 = 0.7, e_2 = 0.4$, and explore the impact of transmission rates, (β_1, β_2) on the vaccine dependent basic reproduction number for each strain (R_0^1, R_0^2 defined in equation (6)), the final epidemic size, and the vaccination coverage presuming IB-RA update rule (figure 7). Clearly lower transmission rates possess a disease-free equilibrium (DFE) (region enclosed by black dotted lines in panel (c)), where R_0^1 and R_0^2 are below one (panels (a) and (b)). Clearly this scenario does not inspire vaccination (panel (c)). However, the upsurge of $\beta_1(\beta_2)$ leads to the predominance of strain 1(2) that also increases vaccination coverage. As we presume $e_1 > e_2$, the intensity of infection due to strain 1 seems less than that of strain 2. Panels (c) and (d) in figure 7 suggest that strain 2 can predominate even if the vaccine uptake reaches the peak level (deep blue regime in panel (d)).

3.2.4. IB-RA versus SB-RA. Apparently, from a global viewpoint, when comparing the two different strategy updating rules (IB-RA and SB-RA), we can observe an

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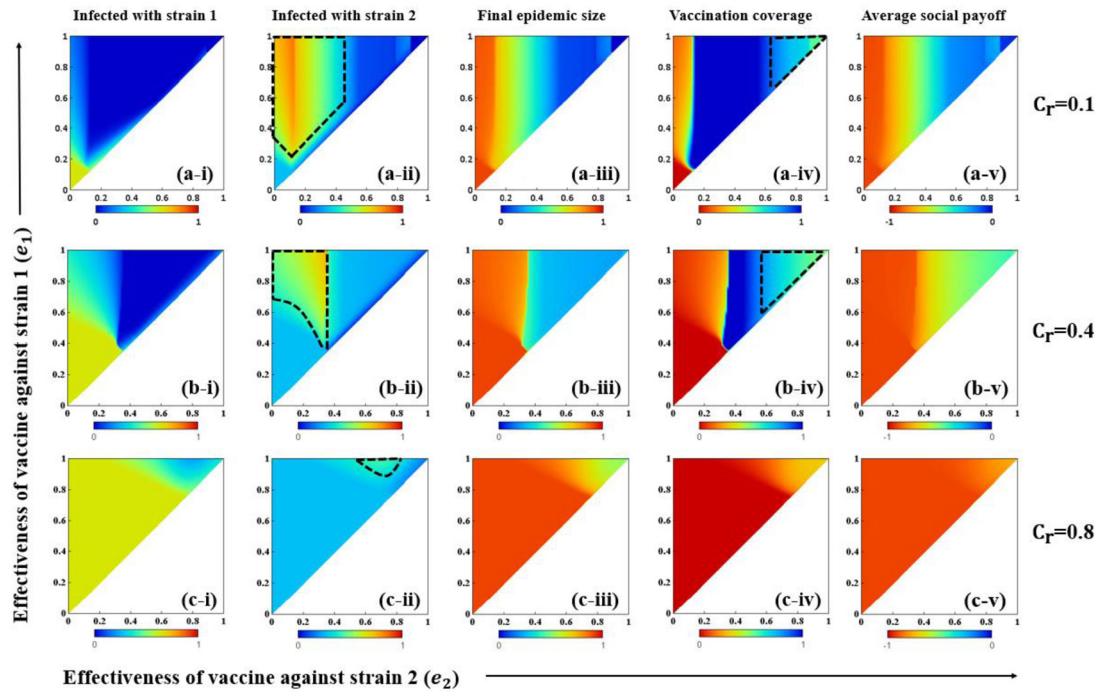


Figure 5. e_2 versus e_1 heat maps with strategy-updating rule SB-RA considering absence of mutation; first two columns represent fraction of infected people with each strain, third column as final epidemic size, fourth and fifth column represent vaccination coverage and average social payoff, respectively. On the other hand, first, second, and third row depict three relative costs as, $C_r = 0.1, 0.4, 0.8$, respectively. Here $\beta_1 = 0.8333, \beta_2 = 0.75, \gamma_1 = 0.3333, \gamma_2 = 0.3$. The regions enclosed by black dashed lines (in second column) indicate more infections taking place with strain 2 whenever e_1 reaches a satisfactory level (varies with C_r) and e_2 is yet reach its threshold level (also varies with C_r). However, another two regions enclosed by black dashed lines in vaccination coverage heat maps indicating a little decline of vaccine uptake due to the emergence of free riders for higher effective (against both strains) vaccine. Each heat map in third, fourth and fifth columns depicts a threshold level of e_2 where the situation gets better. Each heat map assumes $I_1(0) = I_2(0) = 0.0001$.

overall same tendency with relatively better average social payoff, higher vaccination coverage, and lower final epidemic size by SB-RA whenever cost is 0.0 and 0.4 but the opposite scenario happens for a higher cost ($C_r = 0.8$), which is plausible because SB-RA uses global knowledge rather than local knowledge [7] and consequently, brings more effective force to suppress the disease spreading as long as the vaccination cost is of a considerable level, but afterward IB-RA seems to perform better than SB-RA. In this regard, we claim that higher vaccination cost possesses a more negative impression on global scale rather than local viewpoint. This typical phenomenon was observed in the original study regarding SB-RA by Fukuda *et al* (see figure 4 of [13]). Although the authors presumed spatial networks in their study, still agreeing with the well-mixed situation.

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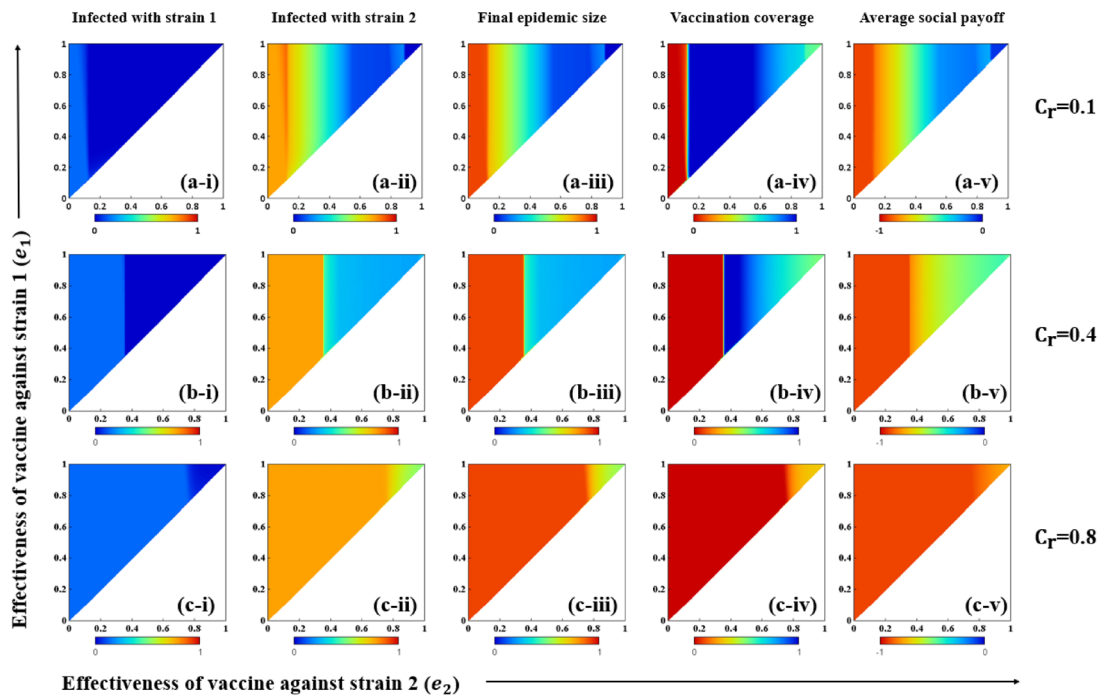


Figure 6. e_2 versus e_1 heat maps with strategy-updating rule SB-RA considering mutation with the rate $\rho = 0.1$; first two columns represent fraction of infected people with each strain, third column as final epidemic size, fourth and fifth column represent vaccination coverage and average social payoff, respectively. On the otherhand, first, second, and third row depict three relative costs as, $C_r = 0.1, 0.4, 0.8$, respectively. Here $\beta_1 = 0.8333, \beta_2 = 0.75, \gamma_1 = 0.3333, \gamma_2 = 0.3$. Each heat map in third, fourth and fifth columns depicts a threshold level of e_2 where the situation gets better. Each heat map assumes $I_1(0) = I_2(0) = 0.0001$.

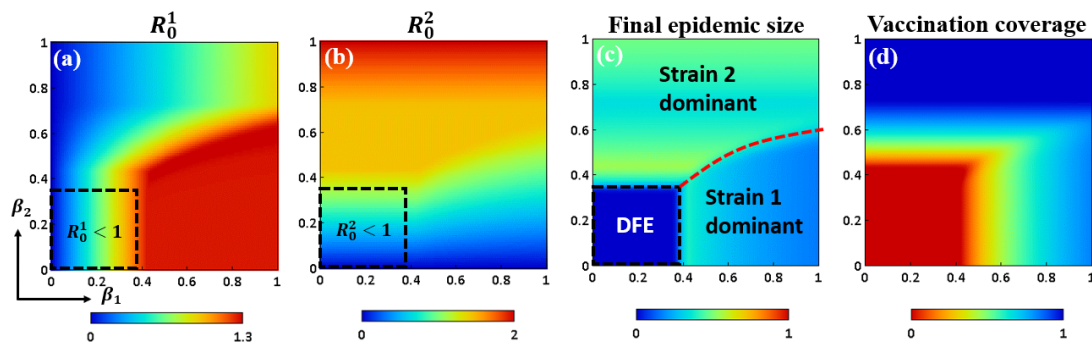


Figure 7. The representation of the basic reproduction number for each strain (panel (a, b)), the final epidemic size (panel (c)), and the vaccination coverage (panel (d)) as a function of transmission rates, (β_1, β_2) . The corresponding parameters are set as, $C_r = 0.1, \rho = 0, e_1 = 0.7, e_2 = 0.4, \gamma_1 = 0.3333, \gamma_2 = 0.3$, with IB-RA update rule. The lower transmission rates yield a disease-free equilibrium (DFE) (panel (c)), where the corresponding basic reproduction numbers (R_0^1 and R_0^2) are below one (panel (a) and (b)). No one opts vaccination for a DFE. However, the upsurge of $\beta_1(\beta_2)$ depicts the dominance of strain 1(2) and boosts up vaccination coverage. Clearly, the large-scale propensity for vaccination (deep blue regime in panel (d)) favors the strain 2-dominant situation as $e_1 > e_2$.

4. Conclusion

This study aimed to develop a mean-field approximation framework for a vaccination game, depicting the effect of a single vaccination strategy when there are two strains of a disease prevailing in an infinite and well-mixed population. Since vaccines may not be always perfect, we adopt the imperfect vaccination provision in disease modeling. Furthermore, it is observed that in conjunction with the vaccine-effectiveness, the vaccination cost can also play an important role to oppress disease spreading. Under the baseline parameters (depicted in table 2), our numerical experiments confirm that vaccine effectiveness against new strain (mutant) consumes more impact on disease eradication, and provide an apparent estimation of vaccination coverage as well as threshold levels of vaccine efficiency (especially for e_2) needed to control a disease spreading. It has been observed that in the absence of mutation, strain 1 dominates when a vast majority of the population are non-vaccinators. This dominance increases with the degree of non-vaccinators. However, strain 2 can emerge among the vaccinators if the vaccine efficacy against that strain is low. Moreover, the presence of continuous mutation makes strain 2 as the only contributor to the disease dynamics. In general, two different strategy update rules- IB-RA and SB-RA- possess the same tendency. However, SB-RA yields better results whenever the cost is low but IB-RA has been found to work well in case of higher cost.

The concept of a well-mixed situation is somewhat idealized. Indeed, individuals in a society are connected by some social networks [19, 21, 26, 34, 36]. Therefore, exploring the outlined evolutionary process under a structured population would be exciting to investigate. Moreover, it would be also interesting to examine the current setting with short-term cross-immunity, isolation, late vaccination (if applicable), superinfection, etc. to get a more general impression.

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