Chapter 4
Social Dilemma Analysis of the Spread of Infectious Disease

Understanding and controlling the spread of infectious disease is a pressing issue for our society. Contemporary globally connected civilization is more at risk from various modern infectious diseases than classical ones such as pests, cholera, and tuberculosis. Over the last few years, pandemic outbreaks of highly virulent influenza, possibly related to avian flu, severe acute respiratory syndrome (SARS), and middle-eastern respiratory syndrome coronavirus (MARSE) have been a threat. Beyond this, the intentional spread of infectious disease, e.g., "bioterrorism", has come to be recognized as being just as dangerous as nuclear weapons. An infectious disease spreads on human social networks. Each individual can protect himself through several measures. Pre-emptive vaccination is thought to be most effective, although it incurs a partial cost to each individual. This brings about a social dilemma, because an individual may be able to rely on so-called "**herd immunity**" to avoid his own infection without himself being vaccinated. Also, besides vaccination, there may be several practical ways to protect against contagion, such as wearing a mask, keeping away from crowds, and self-isolation by leaving the home less often, which may be less costly and less effective than vaccination. In any case, there is a human-decision-making process regarding what steps should be taken, while the dynamics of infectious-disease spread can themselves be evaluated as a diffusion problem that has been well-studied in physics for many years. Thus, based on the concept of human–environment–social interaction, a basic-physics model for this diffusion problem that considers evolutionary game theory (EGT) may lead us to obtain some meaningful solutions that can be proposed to our society. Following the previous chapter explaining how EGT can be applied to traffic-flow analysis, this chapter describes this practical problem.

Human social networks are a central application of evolutionary game theory because the complexity of the underlying network serves as a key factor determining game equilibrium. The spread of an epidemic throughout such a network is mathematically described by percolation theory (see Fig. 4.1, which provides a schematic image of a 2D percolation model applied to the spreading dynamics of an infectious disease), which is an archetype of the physics of a diffusion processes. Vaccination,

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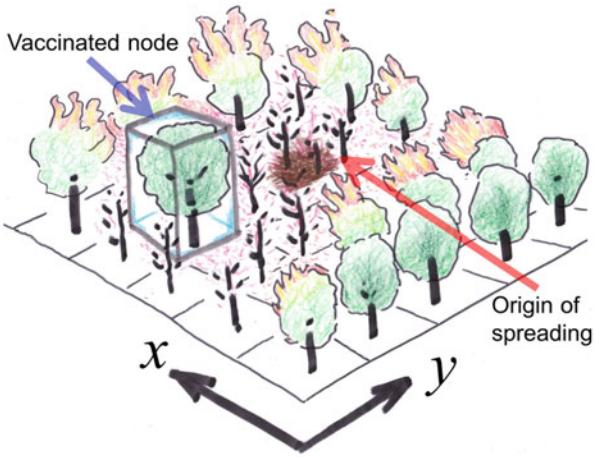

Fig. 4.1 The modeling concept of infectious-disease spread comes from the so-called "2D percolation model", commonly applied to 2D-diffusion problems such as wildfire. In this metaphor, firing is spreading a disease, and a perfectly vaccinated agent is represented by the tree in the box

which is driven by individual decision making, inhibits the spread of infectious diseases. In addition, if so-called herd immunity is established, a free-rider, who pays no cost for vaccination, can escape infection. Here, when we refer to vaccination "cost", we imply not only a direct cost, but also the potential risk of by-effects and psychological negative-costs brought about by vaccination. Obviously, there is a conflict between individual and social benefits; in short, a conflict between individual rational choices: trying to avoid vaccination, or everyone taking the vaccine to achieve the fair Pareto optimum, i.e., the best solution where everyone equally bears the cost to maintain public goods, namely the herd immunity in this context (see Fig. 4.2). This conflict is why we introduce evolutionary game theory into epidemiology; vaccination can be viewed as a game on a complex social network. This specific structure of the social dilemma has been called a **vaccination dilemma** and has been modeled in the framework of the **vaccination game**, explained later. However, vaccination is not an ultimate solution. In some cases, perhaps stochastically, the injection of a vaccine into a human body is not always able to establish immunity because of the imperfectness of the vaccination. Even in such an unwilling case, the vaccine may work to reduce the probability of being infected brought about by physical contact with people around the focal agent in his social network. An expected mathematical model should consider this kind of situation.

4.1 Epidemiological Model and Vaccination Game

Pre-emptive vaccination is one of the best public-health measures for preventing epidemics of infectious diseases and reducing morbidity and mortality.[1] However, most societies entrust vaccination to the autonomy of the individual: vaccination is

[1] Anderson and May (1991).

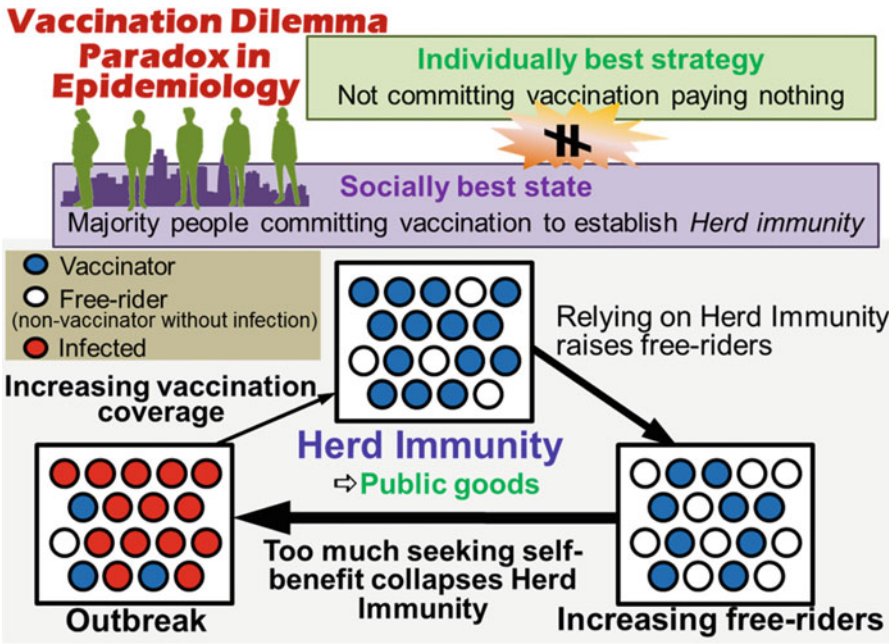

Fig. 4.2 Scheme of the social dilemma working behind the vaccination dilemma

usually voluntary, despite some national or local governments providing subsidies for it. Therefore, decision making at the individual level may be the result of a trade-off between protection and the perceived risks and costs of vaccination and infection. Furthermore, an individual's decision may be influenced by the vaccination behaviors of others.[2] The only example of a vaccination campaign that has completely eradicated a vaccine-preventable disease is smallpox, while cyclic (seasonal) epidemics of other infectious diseases, such as flu-like pathogens and influenza, remain a serious threat to humanity.

One major reason for the difficulty in eradicating vaccine-preventable diseases is related to an inherent vaccination dilemma, sometimes called the "**paradox in epidemiology**". As vaccination coverage increases over a population, the proportion of immunized individuals finally exceeds a critical level above which the disease can no longer persist; this point is called herd immunity, as mentioned above. Once herd immunity is attained, the remaining unvaccinated individuals are quite unlikely to become infected since they are indirectly protected by vaccinated individuals. Thus, unvaccinated individuals obtain benefits from the herd immunity without considering the perceived risks associated with vaccination, such as complications, side effects, and financial costs. There is less incentive for them to get vaccinated, and

[2] As representative works, we cite Chapman and Coups (1999, 2006), Basu et al. (2008).

then, the so-called first-order free-rider problem[3] arises. Some reports suggest that the welfare of a society can be threatened if too many individuals perceive the herd immunity as a public good.[4] As a result, too much self-interest destabilizes the herd-immunity state, and the disease resurges. This paradox makes complete eradication of the disease difficult under a voluntary vaccination policy, and causes a conflict between the optimal vaccination behavior for each individual and the sufficient level of vaccination needed to protect the whole society via the herd immunity.[5] In addition, the number of vaccinated individuals may be reduced by underestimates of infection risk due to lack of knowledge about the disease and/or by overestimation of vaccine risk based on scientifically groundless information.[6]

Interrelations among vaccination coverage, disease prevalence, and the vaccination behaviors of individuals are complicated, and we should duplicate and dynamically as well as quantitatively predict the consequences of these interrelations if we intend to develop effective public-health measures for preventing epidemics of infectious diseases. In this regard, many studies of the vaccination dilemma have applied a game-theoretic framework to a population wherein each individual tries to maximize his or her own payoff. These studies have provided highly fruitful results.[7] Let us call this framework the "**vaccination game**", where both the epidemiological dynamics and the dynamics of the human decision-making process are simultaneously and interdependently considered.

Some of the previous game-theoretic analyses of vaccination behavior have assumed a static game wherein individuals always act with perfect information about their probability of becoming infected. In reality, individuals cannot precisely know this probability. Moreover, the game should allow individuals to update their strategies through learning by imitating others who appear to have adopted more successful strategies. In this context, imitating others means adapting one's strategy based on one's own personal experience and information from media (the former and latter can be called active and passive information, respectively). Also, it is very likely that would be someone who acts opposite to what surrounding people do, because non-vaccination would cost a player nothing if his all neighbors were vaccinating.[8] As those, concerning how an individual updates his own decision,

[3] In 2×2 games, a defector who is harmful to cooperators is called a *first-order free-rider*. When a costly punishment scheme for defectors exists, there can be defined a strategy called the "masked good guy", who cooperates with others but never punishes defectors; such an individual is called a *second-order free-rider*. There is much literature on the second-order free-rider problem. For example, Olson (1965), Axelrod (1986), Yamagishi (1986).

[4] Asch et al. (1994).

[5] Although there are many good references on this issue, several representative ones are cited here. Cullen and West (1979), Fine and Clarkson (1986), Geoffard and Philipson (1997), Bauch et al. (2003), Bauch and Earn (2004).

[6] Jansen et al. (2003).

[7] There are many related references, but due to space limitations, we only cite the most representative here. Bauch (2005).

[8] Such behavior might be meaningful in the context of the minority game. See the following report.

not only imitating others; either his neighbors or media, but also drawing his specific decision based on the observation around him, there might be diverse ways in a real world. To describe this process explicitly, we should construct an appropriate model that combines mathematical epidemiological dynamics with game-theoretic dynamics, taking account of the various rules for strategy adapting. For example, Bauch constructed and analyzed a model that combines epidemiological dynamics with replicator dynamics of evolutionary game theory to capture the imitative behavior of individuals during outbreaks of diseases; he found that imitative behavior provokes periodic outbreaks of such diseases[7]. Vardavas et al. proposed an individual-level adaptive decision-making model that was inspired by a minority-game methodology.[9] By solving the model numerically and analytically, they showed that incentive-based vaccination programs are indispensable for controlling epidemics of infectious disease but that misuse of these programs may lead to a severe epidemic. These studies assumed that the population is homogeneously mixed and that individuals are fully rational in the sense that they make decisions to pursue maximum personal utility based on their perceived risks. Yet, in reality, there are always spatial structures for networks of both disease transmission and an individual's contacts, and any individual's behavior is not completely rational. Accordingly, Fu et al., for example, elevated a model to that of evolutionary game theory to explore the effects of individual adaptation behavior and population structure upon vaccination when a population is faced with an epidemic of an infectious disease.[10]

Let us revisit the term paradox in epidemiology in the context of a game-theoretical application. Any rational individual has a strong incentive to exploit the public good by free-riding on herd immunity. However, this incentive, wherein the individual pays nothing but still obtains a benefit, only works as long as the majority of the community spontaneously receive the vaccination. By contrast, if the majority disregards vaccination, then doing nothing is no longer a better option because infection is likely. In this case, spontaneous vaccination becomes the rational option. This difference implies that the best choice for an individual is to always adopt the strategy of the social minority; either free-ride when the herd immunity is well established or take the vaccination when most people neglect to do so. This situation obviously contains the structure of a minority game, as Vardavas pointed out[8]. A minority game,[11] originally defined as the El Farol Bar problem,[12] is a typical social dilemma that can be observed in many real situations. The most heavily concentrated applications are in financial markets. In a minority game, any individual has an incentive to adopt the strategy of the minority under any circumstance. This duality might be interpreted as a Chicken-type dilemma wherein the fair Pareto optimum is realized when two strategies coexist, as discussed in Chap. 2.

[9] Vardavas et al. (2007).

[10] Fu et al. (2011).

[11] A reader can consult with, for example; Challet et al. (2005).

[12] Brian Arthur (1994).

Based upon the abovementioned review, this section gives the fundamental frameworks of both the epidemiological model and the vaccination game.

4.1.1 SIR/V (SVIR) Model for an Infinite & Well-Mixed Population[13]

We start our discussion with the simple situation, for which dynamics can be formulated by a set of ordinary differential equations (ODEs). Before the discussion, let us introduce one assumption that is substantially important in relation to vaccination. So far, we have implicitly presumed that a vaccination brings an idealized perfect immunity whenever performed. In reality, for infectious diseases such as flu, measles, malaria, and HIV, vaccination does not work perfectly, giving rise to the concept of the "effectiveness" of a vaccination. This presumes a situation in which some vaccinated agents acquire immunity with effectiveness probability[14] e; meanwhile, the remaining agents fail to acquire immunity with probability $1-e$. This can be likened to a lottery that pays out either 100% or 0% of the prize fund according to the probability of winning. Meanwhile, there are protective measures other than vaccination whose efficacy can also be expressed probabilistically. In particular, we are interested in intermediate measures such as wearing masks, gargling, and hand washing that offer partial protection against infection while costing less than vaccination. Some kinds of vaccination rather work to reduce the contagious probability, rather than offering a perfect immunity with a certain probability. Such a mechanism including intermediate measures is called "defense against contagion" in the following text. Some recent studies[15,16,17] have proposed representing a measure of defense against contagion by reducing the risk of infection (denoted as η). Iwamura et al.[16] assumed a lower infection risk in their spatial version of the vaccination game, which they implemented by introducing a reduced infection rate per day per person into the SIR dynamics on an underlying network by means of the Gillespie algorithm.[18] Those two concepts, shown schematically in Fig. 4.3, seem analogous in the sense that one can avoid an infection stochastically, but they definitely differ in how they actually work.

We take the SIR model[19] as the baseline and extend it to consider either SIR/V or SVIR dynamics including vaccinators as SIR variants. We modify the SIR model to reproduce two different scenarios, namely imperfect vaccination (hereinafter the

[13] Kuga and Tanimoto (2018).
[14] Wu et al. (2011).
[15] Iwamura et al. (2016).
[16] Bai (2016).
[17] Cardillo et al. (2013).
[18] Gillespie (1977).
[19] Kermack and McKendrick (1927).

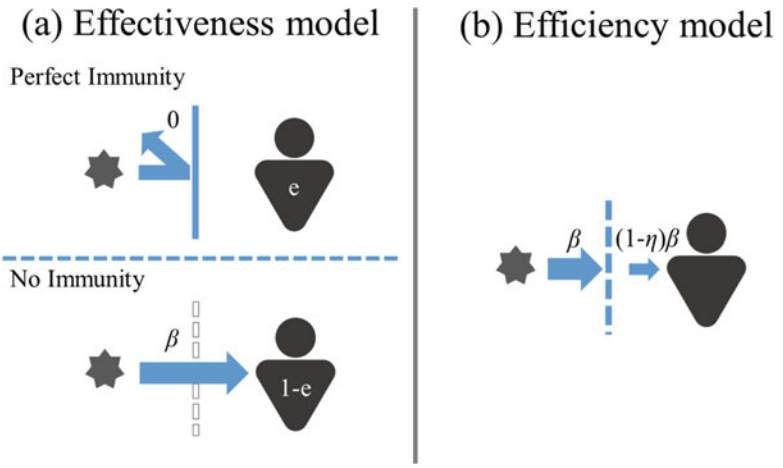

Fig. 4.3 Schematic of two concepts for avoiding infection: (**a**) effectiveness model; (**b**) efficiency model

effectiveness model) and intermediate-defense measures (hereinafter the efficiency model according to Ref [13]). We also assume a population that is infinite and ideally well mixed.

Let us presume that immunity, once acquired, works perfectly. The population is divided into three groups: susceptible individuals (S), who are currently healthy but may or may not be infected with the disease, infectious individuals (I), who are currently infected and will recover, and recovered individuals (R), who are never infected again. Immunity is acquired by either recovering from the disease or by pre-emptive vaccination. The immunity is presumed to be effective over an individual's life span. The SIR model is expressed as

$$\begin{cases} \dfrac{dS(t)}{dt} = -\beta \cdot S(t) \cdot I(t), \\ \dfrac{dI(t)}{dt} = \beta \cdot S(t) \cdot I(t) - \gamma \cdot I(t), \\ \dfrac{dR(t)}{dt} = \gamma \cdot I(t), \end{cases} \tag{4.1}$$

and,

$$S(t) + I(t) + R(t) = 1, \tag{4.2}$$

where β and γ indicate the disease-transmission rate per capita and the rate of recovery, respectively. Obviously, the SIR process always takes place in the unilateral direction, S → I → R, unlike the SIS model[20] wherein immunization efficacy is

[20]Hethcote and van den Driessche (1995).

neglected. Therefore, we can deduce the final epidemic size at the equilibrium of the dynamics. $R(\infty)$ is the fraction of individuals who were once infected with the disease. According to Eqs. (4.1) and (4.2) with initial conditions $S(0) \approx 1$, $R(0) = 0$, $I(\infty) = 0$, and $S(\infty) = 1 - R(\infty)$, we derive

$$R(\infty) = 1 - \exp[-R_0 \cdot R(\infty)]. \tag{4.3}$$

Here, $R_0 \equiv \beta/\gamma$ is called the **basic reproduction ratio**, which is the number of secondary infections caused by a single infected individual. Let x be the fraction of the total population that is vaccinated, such that the remaining fraction $1 - x$ is not. Then, we can rewrite the final epidemic size at the equilibrium of the dynamics when the pre-emptive-vaccination fraction is x, $R(x, \infty)$, by solving the following equation:

$$R(x, \infty) = (1 - x) \cdot (1 - \exp[-R_0 \cdot R(x, \infty)]). \tag{4.4}$$

This equation is obviously nonlinear and transcendental.

4.1.1.1 Effectiveness Model

A vaccinated population is separated into two classes: immune individuals obtaining perfect immunity and non-immune ones who fail to obtain immunity. Let the effectiveness of the vaccine and the vaccination coverage be e ($0 \leq e \leq 1$) and x, respectively. The fraction of vaccinated individuals with immunity must be ex, while the fraction of non-immune individuals is $(1 - ex)$. We can express the final epidemic size R in relation to both x and time t at equilibrium ($t = \infty$) as

$$R(x, \infty) = (1 - ex)(1 - \exp[-R_0 R(x, \infty)]). \tag{4.5}$$

$R(x, \infty)$ gives the respective fractions of four different types of individuals depending on whether they are vaccinated or non-vaccinated and whether they are healthy or infected, as summarized in Table 4.1.

4.1.1.2 Efficiency Model

Let the efficiency of an intermediate defense measure for avoiding infection be η ($0 \leq \eta \leq 1$), describing how the defense measure can decrease the probability of being infected. In the following formulation of the efficiency model, we temporarily regard the vaccinated state as the state prepared with an intermediate defense measure, making it convenient to compare it with the aforementioned effectiveness model. We describe the epidemic-spreading dynamics using the compartment model whereby individuals in a population can be classified into susceptible (S), infected

Table 4.1 Fractions of four types of individual using the effectiveness model

Strategy/state	Healthy	Infected
Vaccinated	$x(e + (1 - e)\exp[-R_0R(x,\infty)])$	$x(1 - e)(1 - \exp[-R_0R(x,\infty)])$
Non-vaccinated	$(1 - x)\exp[-R_0R(x,\infty)]$	$(1 - x)(1 - \exp[-R_0R(x,\infty)])$

(I), recovered (R), and vaccinated (V) states. A non-vaccinated (more precisely, non-prepared with intermediate defense measures) susceptible individual may become infected if they are exposed to infectious individuals at the disease-transmission rate β [per day per person]. An S individual prepared with intermediate defense measures may also become infectious at a rate $(1 - \eta)\beta$. An infected individual recovers at the recovery rate γ [per day].

The SVIR model we use to describe such a condition is

$$\begin{cases} \dfrac{dS(x,t)}{dt} = -\beta S(x,t)I(x,t), \\ \dfrac{dV(x,t)}{dt} = -(1-\eta)\beta V(x,t)I(x,t), \\ \dfrac{dI(x,t)}{dt} = \beta S(x,t)I(x,t) + (1-\eta)\beta V(x,t)I(x,t) - \gamma I(x,t), \\ \dfrac{dR(x,t)}{dt} = \gamma I(x,t), \end{cases} \tag{4.6}$$

with the presumed set of initial values $S(x,0) = 1 - x$, $V(x,0) = x$, and $I(x,0) = 0$. The following constraint is requisite:

$$S(x,t) + V(x,t) + I(x,t) + R(x,t) = 1. \tag{4.7}$$

Because the population is not completely susceptible, it is accurate to use a control reproduction number, $R_{c,}$ instead of the basic reproduction number, R_0. In this case, R_c can be estimated as

$$R_c = \frac{\beta}{\gamma}[S(x,0) + (1-\eta)V(x,0)] = R_0[S(x,0) + (1-\eta)V(x,0)]. \tag{4.8}$$

The final epidemic size and other fractions can be expressed as;

$$S(x,\infty) = (1-x)\exp[-R_0R(x,\infty)], \tag{4.9}$$

$$V(x,\infty) = x\exp[-(1-\eta)R_0R(x,\infty)], \tag{4.10}$$

$$R(x,\infty) = 1 - (1-x)\exp[-R_0R(x,\infty)] - x\exp[-(1-\eta)R_0R(x,\infty)]. \tag{4.11}$$

In the limit of this process, the respective fractions of the four different types of individual at equilibrium are as summarized in Table 4.2.

Table 4.2 Fractions of four types of individual using the efficiency model

Strategy/state	Healthy	Infected
Vaccinated	$x \exp[-(1-\eta)R_0R(x,\infty)]$	$x(1-\exp[-(1-\eta)R_0R(x,\infty)])$
Non-vaccinate	$(1-x)\exp[-R_0R(x,\infty)]$	$(1-x)(1-\exp[-R_0R(x,\infty)])$

Comparing Tables 4.1 and 4.2, it is worth noting that the "success probability of free-riding" is always given by $\exp[-R_0R(x,\infty)]$, regardless of whether we presume perfect or imperfect vaccination, or even intermediate defense measures.

Figure 4.4 shows the final epidemic size (FES) for different levels of vaccination coverage using the effectiveness and efficiency models. From Fig. 4.4, the so-called critical-vaccination coverage that eradicates an epidemic spread can be read from the border of the extinct phase at which FES $= 0$. This border suggests the critical vaccination coverage for suppressing the spread of an infection, which can be determined analytically as $x_c = (1 - 1/R_0)/(1 - \varepsilon)$ for the efficiency model and $x_c = (1 - 1/R_0)/e$ for the effectiveness model. Clearly, as long as a less-reliable defense measure is provided, say $\eta < 0.6$, we cannot prevent an epidemic from breaking out, even if all individuals use that particular defense measure.

4.1.1.3 Relationship Between Effectiveness and Efficiency Models

Let us establish an explicit relationship between the effectiveness and efficiency models. Eqs. (4.3) and (4.11) give

$$\begin{aligned} R(x,\infty) &= (1-ex)(1-\exp[-R_0R(x,\infty)]) \\ &= 1-(1-x)\exp[-R_0R(x,\infty)] - x\exp[-(1-\eta)R_0R(x,\infty)]. \end{aligned} \tag{4.12}$$

Equation (4.8) gives the relationship between e and η as

$$e = \frac{\exp[-(1-\eta)R_0R(x,\infty)] - \exp[-R_0R(x,\infty)]}{1-\exp[-R_0R(x,\infty)]}. \tag{4.13}$$

Figure 4.5 shows the relationship between e and η indirectly. The vacant region for $x \approx 1$ and $\eta \approx 1$ is due to the fact that a multivalued e–η relationship is inevitable. It is worth noting that, for smaller values of η, e appears insensitive to x, implying that e constantly relates to η irrespective of x. By contrast, for larger η (i.e., $\eta > 0.6$), the colored contours appear slanted, suggesting that the e–η relationship becomes fully dependent on x. In other words, epidemic dynamics that assume a higher effectiveness of vaccination (close to perfect immunity) work differently from those that assume an equivalent efficiency of the intermediate defense measure, which is influenced strongly by how many individuals use that particular vaccination or defense measure.

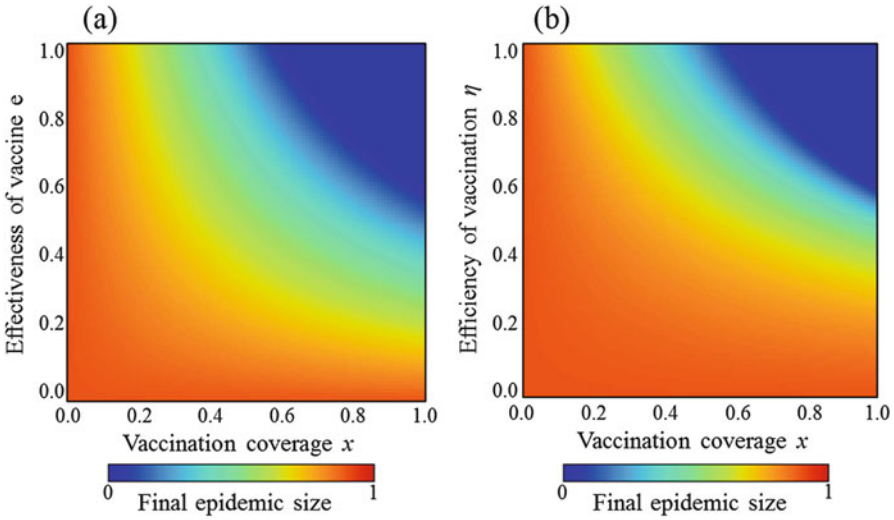

Fig. 4.4 Final epidemic size according to vaccination coverage and (**a**) effectiveness e (effectiveness of a vaccination) or (**b**) efficiency η (efficiency of an intermediate defense measure). We presume that $R_0 = 2.5$, which is applied consistently in this study

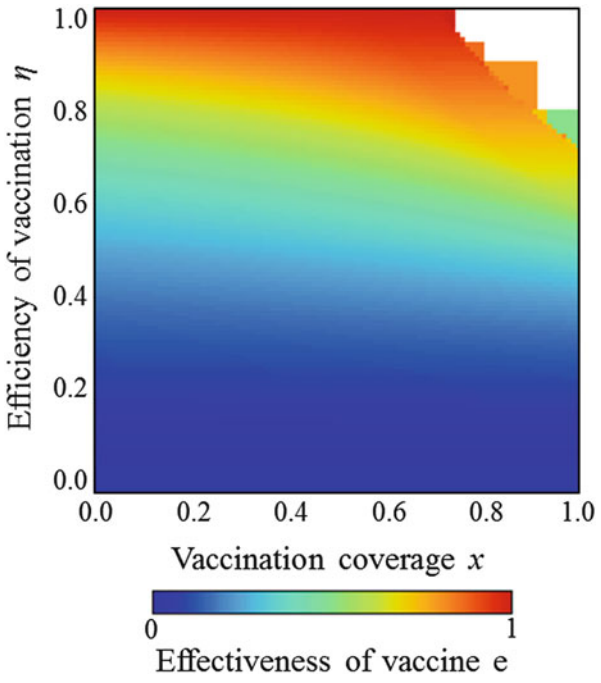

Fig. 4.5 Effectiveness e plotted on (x, η) plane

4.1.2 *Vaccination Game*

In this subsection, let us define the vaccination game. Without loss of the generality, let us presume a certain spatial structure among agents, defining a social network. A well-mixed population, presumed in the previous subsection, should be recognized as a specific situation wherein a complete graph is presumed as an underlying social network.

As Fig. 4.6 suggests, in a world, the vaccination game is a model that integrates the mathematical framework of epidemiology supported by SIR/V with the evolutionary game. The former part predicts how an infectious disease spreads on a complex human social network, while the latter emulates people's decision-making process for whether to commit to a costly provision or to try to free-ride on the public good that is herd immunity.

Consider a population in which each individual on a social network decides whether to be vaccinated. Seasonal and periodic infectious diseases, such as flu, are assumed to spread through such a population. For example, the protective efficacy of a flu vaccine persists for less than a year because of waning antibodies and year-to-year changes in the circulating virus. Therefore, under a voluntary vaccination program, individuals must decide every year whether to be vaccinated. Thus, the dynamics of our model consists of two stages: the first stage is a vaccination campaign and the second is an epidemic season.

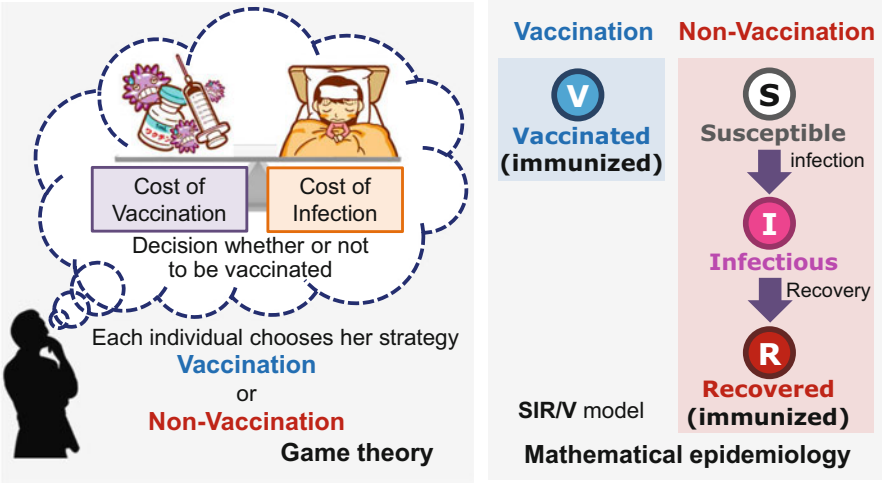

Fig. 4.6 Vaccination game

4.1.2.1 The First Stage: The Vaccination Campaign

Here, in this stage, each individual makes a decision whether to get vaccinated before the beginning of the seasonal epidemic, i.e., before any individuals are exposed to the epidemic strain. Vaccination imposes a cost, C_v, on each individual who decides to be vaccinated. The cost of vaccination includes the monetary cost and other perceived risks, such as adverse side effects. If an individual is infected, he incurs the cost, C_i, of infection.

In real, as we discussed already, the vaccination does not provide perfect immunity. Individuals who unfortunately are infected despite taking either the vaccination or the intermediate defense measure against contagion are assigned the cost $C_v + C_i$ of vaccination and infection. Needless to say, an individual neither vaccinated nor taking any intermediate defense measure against contagion faces the risk of being exposed to infection during a season.

To simplify the evaluation of each individual's payoff, without loss of generality, we rescale the cost by defining a relative cost of vaccination, namely $C_r = C_v/C_i$ ($0 \leq C_r \leq 1$; $C_i = 1$). Consequently, the payoff of each individual at the end of an epidemic season depends on his/her final state. Table 4.3 summarizes the payoff whether committing to a provision (either vaccination or intermediate defense measure against contagion) or not and whether having been healthy or infected.

4.1.2.2 The Second Stage: The Epidemic Season

Here, at the beginning of this stage, the epidemic strain enters the population, and a number I_0 (sufficiently small compared with the total population) of randomly selected susceptible individuals are identified as the initially infected ones. Then, the epidemic spreads according to SIR/V dynamics. At the end of one epidemic season, we can observe the final epidemic size, previously discussed in Eq. (4.4).

It is likely that, after one epidemic season, an individual would reevaluate their decision of whether or not to commit to a provision based on whether or not they were infected during the season. For instance, they may shift to non-vaccination if quite a few of their neighbors successfully avoided infection during the season, or if media claims that the current infection rate is not as serious as expected. Depending on how each individual adjusts their strategy, as defined in the later sub-section, the fractions committing or not committing to a provision in the next season evolve. Although what we seek is not the annual dynamics in terms of the final epidemic size and vaccination coverage over long epidemic seasons, we repeat the set of one vaccination campaign and one epidemic season many times until its time evolution

Table 4.3 Payoff structure determined at the end of an epidemic season

Strategy/state	Healthy	Infected
Vaccinated	$-C_r$	$-C_r - 1$
Non-vaccinated	0	-1

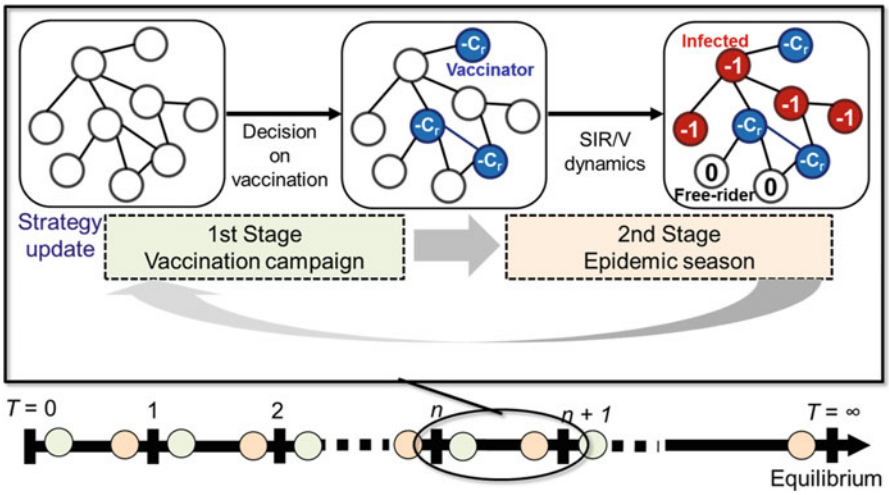

Fig. 4.7 Time sequence in a vaccination game. T denotes the number of epidemic seasons

reaches a certain equilibrium. We then evaluate the social equilibrium where the final epidemic size, vaccination coverage, and social-average payoff are measured. The vaccination-game concept is explained in Fig. 4.7.

4.1.3 Multiagent Simulation (MAS) Approach

The classic SIR or SIR/V model, discussed in Sect. 4.1.1, is given by coupled (integro-) differential equations and does not assume any spatial structure for the population. Such an analytical approach can be applied to cases presuming spatial structure amid individuals. However, a more convenient and powerful multiagent simulation (MAS) approach can be applied to various cases presuming a spatial structure of finite population size.

Suppose that the whole population has a spatial structure, represented by a network consisting of nodes and links. The dynamics of SIR/V on a spatially structured population are not captured by a system of differential equations; thus, we numerically simulate an epidemic spreading on a network using the Gillespie algorithm[18] in the extended SIR/V model. For social networks, we can account for any topology in the MAS model. Many interesting toplogies have been discussed; rings (representing 1D regular graphs), lattices (representing 2D regular graphs), random regular networks (RRGs), Barabási-Albert scale-free (BA-SF) networks,[21]

[21] Barabási and Albert (1999).

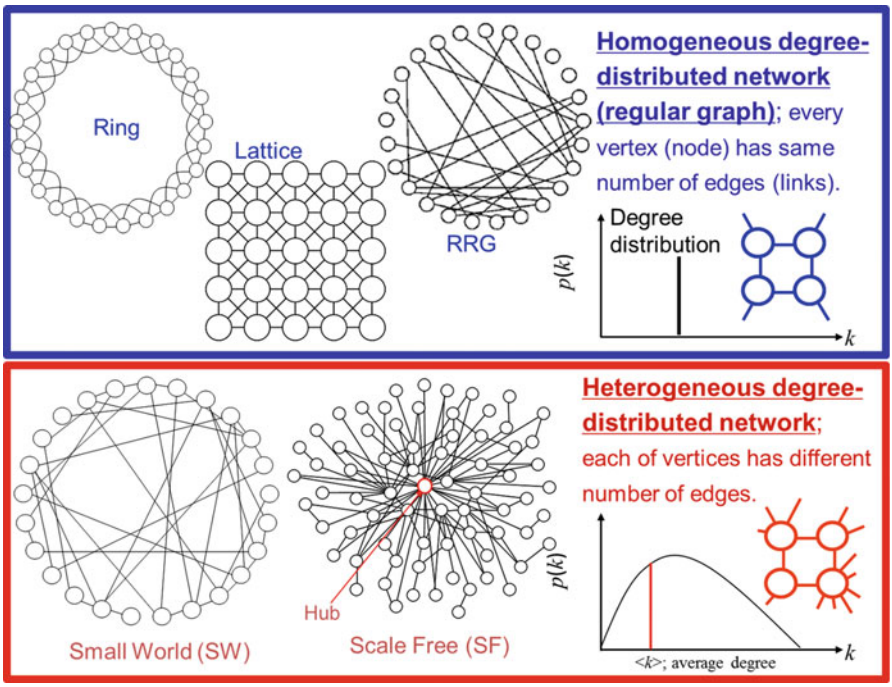

Fig. 4.8 Representative topologies

Erdős–Rényi random (E-R random) graphs,[22] and small-world (SW) network.[23] Figure 4.8 gives some such models. Meanwhile, a couple of network properties have been defined to evaluate its topology and complexity; such as average degree, $<k>$, degree distribution, $P(k)$, average path length, average cluster coefficient, and assortative coefficient. In rings, lattices, and RRGs, each vertex has the same degree k, i.e., the same number of links, and so these are called homogenous degree-distributed networks. The degree distribution of a scale-free graph obeys a scale-free distribution, and that of the E-R random graph obeys a Poisson distribution. These are classified as heterogeneous degree-distributed networks. A scale-free network has a small number of agents (called "hubs") with a huge number of links, while the vast majority of agents have a small number of links. It is why its degree-distribution is scale-free. And it is the substance of scale-free network. A small-world network can be constructed from a regular graph such as a ring or a lattice. Starting from such a regular graph, severing links with "short-cut" probabilities (usually presumed to be a small value), and re-connecting them randomly, the graph would become a small-world. One of the most important characteristics is that an SW network has a quite small average path length compared with the original

[22] Bollobás (1985).

[23] Watts and Strogatz (1998).

regular graph. Some complex networks observed in real human social systems can feature scale-free and small-world characteristics.

In the model, the whole population N is divided S, I, R, and V individuals. The disease parameters are β, which is the transmission rate per day per person, and γ, which is the recovery rate per day (i.e., the inverse of the mean number of days required to recover from the infection).

For the sake of explanation, let us be concerned with the square lattice, RRG, and B-A SF network. An epidemic spreads much more easily on the RRG and the BA-SF network, even when the transmission rate is lower than that on the square lattice.[24] In the following text, we set the disease-transmission rate β to ensure that the risk of infection in a population with only the unvaccinated individuals is equivalent for all different network structures. This requires us to calibrate the value of β such that the final proportion of infected individuals across the respective networks will be 0.9 as a reference value. We should set $\beta = 0.46$ day^{-1} person^{-1} for the square lattice, $\beta = 0.37$ day^{-1} person^{-1} for the RRG, and $\beta = 0.55$ day^{-1} person^{-1} for the BA-SF network (see Fig. 4.9).[25] Also, we should necessarily set the recovery rate. Throughout the following text, we presume $\gamma = 1/3$ day^{-1}. A typical flu is assumed to determine these disease parameters.

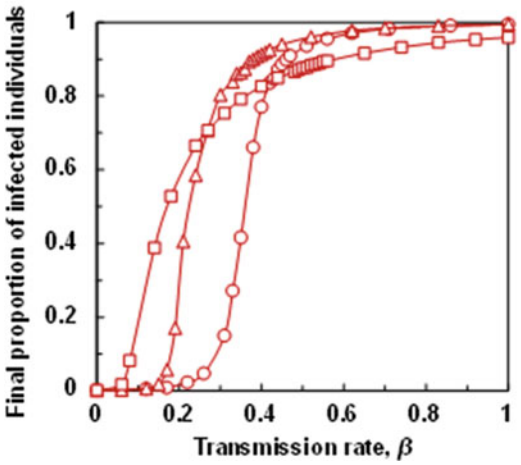

Fig. 4.9 Final proportion of infected individuals as a function of transmission rate β when no individuals are vaccinated on each network: square lattice (circles), random regular graph (RRG) (triangles), Barabási-Albert scale-free (BA-SF) network (squares). For the lattice (circles): population size $N = 70 \times 70$ with von a Neumann neighborhood, recovery rate $\gamma = 1/3$ day^{-1}, seeds of epidemic spread $I_0 = 5$. For RRG (triangles): population size $N = 4900$, degree $k = 4$, recovery rate $\gamma = 1/3$ day^{-1}, seeds of epidemic spread $I_0 = 5$. For BA-SF network (squares): population size $N = 4900$, average degree $<k> = 4$, recovery rate $\gamma = 1/3$ day^{-1}, seeds of epidemic spread $I_0 = 5$. Each plotted point represents an average over 100 runs

[24] Keeling and Eames (2005), Pastor-Satorras and Vespignani (2001).

[25] Fukuda et al. (2014).

At the beginning of each epidemic season, we randomly place a number I_0 of initially infected agents. According to the Gillespie algorithm,[19] we exactly follow the epidemiological dynamics of the SIR/V model. This is analogous to a simulation based on the percolation theory.[26] The epidemic season lasts until no infection exists in the population. Each individual who gets infected during the epidemic incurs the cost of infection, C_i. However, the cost paid by a "free-rider" who does not vaccinate and still is free from infection is zero.

As shown in Fig. 4.7, right after one epidemic season, another vaccination campaign begins, during which each agent in the network refreshes their strategy. We repeat the set of one vaccination campaign and one epidemic season many times until its time evolution reaches a certain equilibrium to obtain the final epidemic size. The vaccination coverage and the social average payoff are measured.

4.1.4 Decision-Making Process Concerning Vaccination

In this sub-section, we describe how each agent adapts their strategy, regardless of whether they commit to a provision in a vaccination campaign; this is the first stage in the vaccination game, schematically shown in Fig. 4.7. One's motivation in adjusting their strategy over time is to maximize their own payoff as long as each individual is presumed to behave in a rational way. According to what has been suggested by evolutionary game theory, even if one behaves in an altruistic manner, there must be compensation in the long run. If not, such cooperative behavior cannot be evolutionary stable. Roughly speaking, one can update their strategy through social imitation or self-estimation.

Social imitation is "copying" from others. The information source, from which a focal agent copies, might be an acquaintance, whether a neighbor, relative, friend, or someone sharing common benefits with the focal agent; or they might be on the media. In the latter case, the information is global, unlike in the former case relying on local information.

The second idea, self-estimation, differs from copying. In many real situations concerning decision making, it is conceivable that an agent will engage not in social imitation but drawing a personality-independent decision that directly suggests that one of the alternative strategies to be taken in the next time-step is conceivable. In this case, a focal agent quantitatively estimates the given situation based on the observation of what currently happens to his neighbors including himself. That quantitative estimation teaches him which strategy is stochastically best for the next time-step. One commonly shared idea concerning the strategy-update rule in evolutionary game theory, the "aspiration model",[27] belongs to this second idea.

[26] Sahimi (1994).

[27] There have been many previous studies on the aspirational model; let us suggest some representative literature below; Macy and Flache (2002), Chen and Wang (2008).

4.1.4.1 Social Imitation

As mentioned above, this concept comes from how one copies others. We note two ideas.

Individual-Based Risk Assessment (IB-RA)
Fu et al. pioneered a model[10] in which agents, spatially distributed on an underlying network and exposed to infectious risk, learn whether or not to vaccinate from one of their neighbors. This idea exactly reflects the assumption of the vast majority of studies dealing with spatial prisoner's dilemma (SPD) games, namely pairwise comparison based on a Fermi function (as described by PW-Fermi). Agent i randomly selects agent j from his neighbors. Let us assume that their payoffs are π_i and π_j, respectively. The probability of agent i copying agent j's strategy, s_j, either vaccination or non-vaccination, instead of his own strategy, s_i, is $P(s_i \leftarrow s_j)$, which is defined as

$$P(s_i \leftarrow s_j) = \frac{1}{1 + \exp\left[\frac{\pi_i - \pi_j}{\kappa}\right]}, \tag{4.14}$$

where κ indicates the sensitivity to the gain difference. For $\kappa \to \infty$ (weak-selection pressure), an individual i is insensitive to the payoff difference $\pi_i - \pi_j$ against another individual j and the probability $P(s_i \leftarrow s_j)$ approaches 1/2 asymptotically, regardless of the payoff difference. For $\kappa \to 0$ (strong selection pressure), individuals are sensitive to the payoff difference, and they definitely copy the successful strategy that earns the higher payoff, even if the difference in the payoff is very small. We assume that $\kappa = 0.1$.

Strategy-Based Risk Assessment (SB-RA)[28]
Equation (4.14) indicates that as the negative-payoff difference increases, the probability that an individual will change their strategy to that of their successful neighbor increases. Observing Eq. (4.14) from a different viewpoint, this rule of strategy adaptation can be interpreted as follows: each individual evaluates both the risk of maintaining their own strategy and imitating that of their opponent and then selects the one with the smaller risk. In this method, each individual i assesses the risk based only on one certain individual j because Eq. (4.14) uses only the payoff of i's opponent (individual j). Thus, we call the updating rule, described by Eq. (4.14) an individual-based risk-assessment updating rule (IB-RA).

However, when we assume that the information regarding the consequences of adopting a certain strategy are disclosed to the society and everyone in the population has access to this information, then individuals no longer rely heavily on the payoff of any one neighbor. Instead, in adapting their strategy, they tend to assess the risk based on a socially averaged payoff that results from adopting a certain strategy.

To reflect the above situation, we propose the following modified imitation probability:

$$P(s_i \leftarrow s_j) = \frac{1}{1+\exp\left[\frac{\pi_i - <\pi_{s_j}>}{\kappa}\right]}, \tag{4.15}$$

where $<\pi_{s_j}>$ is an average payoff obtained from a collective payoff over individuals who adopt the same strategy as that of a randomly selected neighbor j of the individual i. The sampling number is a control parameter that ranges from only one individual (i.e., only one of i's neighbors, j) to all individuals among the whole population who adopt the same strategy as j. That is, if s_j is the strategy of vaccination (cooperation, C), then $<\pi_{s_j}>=-C_r$ (since the payoff of a vaccinated individual is uniquely determined[28]); whereas, if s_j is the strategy of no-vaccination (defection, D), then $<\pi_{s_j}>$ takes a value between 0 and 1, depending on the fractions of infected and healthy individuals (free-riders) with the strategy s_j in the population at the end of the epidemic. Moreover, if sampling is impossible because the population size of individuals with the strategy s_j is too small, the individual i uses the payoff of one randomly selected neighbor instead of $<\pi_{s_j}>$ in Eq. (4.15), leading to an expression that is the same as Eq. (4.14). Thus, when the sampling rate is set to zero, Eq. (4.15) reduces to Eq. (4.14).

Equation (4.15) implies that an individual i assesses the risk of changing their strategy based on the payoff attained by adopting a certain strategy, and not the payoff attained by a certain other individual. Thus, we call the updating rule (4.15) a "strategy-based risk assessment updating rule" (SB-RA). Note that, risk assessment based on the consequences of a vaccination strategy is the same as that based on a unique individual because the immune effect of vaccination is perfect during an epidemic season. However, for the no-vaccination strategy, the risk may differ from season to season because the degree of the epidemic may differ.

Figure 4.10 schematically summarizes IB-RA and SB-RA.

4.1.4.2 Self-Estimation[15]

Instead of "copying probability from one of the neighbors," we directly assign an agent a "probability of vaccinating," P_C, triggered by his consciousness of how dangerous it is to ignore vaccination. Namely,

$$P_C = \frac{1}{1+\exp[(C_r - <C_D>)/\kappa]}, \tag{4.16}$$

[28]This is true only when a vaccination brings perfect immunity. When we presume a general provision, we have either imperfect vaccination dealt by the effectiveness model or an intermediate protection measure for contagion by the efficiency model (see Fig. 4.3). The cooperator's payoff is stochastically variable. In this case, we must evaluate $<\pi_{s_j}>$ fairly, as in the defector's case.

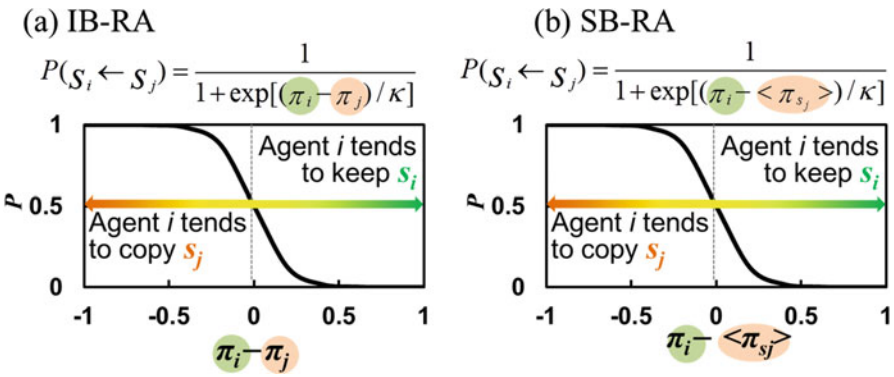

Fig. 4.10 Strategy update based on the concept of social imitation; (**a**) IB-RA, (**b**) SB-RA

$$\frac{< C_D >= C_i \cdot n_i + {}^{i}C_f \cdot n_f}{n_D,} \tag{4.17}$$

where $C_i(= 1)$is the cost of being infected, C_f is the cost of free riding, which is zero, and n_D, n_i, and n_f are the numbers of non-vaccinators, infected agents, and free riders, respectively, in the agent's neighborhood. Therefore, $n_D = n_i + n_f$. $<C_D>$ indicates the average payoff of non-vaccinators in agent i's neighborhood. In the following text, let us call this model "direct commitment (DC)".

One problem that arises is how to evaluate Eq. (4.17) if there are no non-vaccinators in agent i's neighborhood. We establish the following four cases as our sub-model.

Case 1: Agent i retains their strategy.
Case 2: As a substitute, we assume that $P_C = 1\text{-}C_r$.
Case 3: Agent i switches to the strategy opposite theirs.
Case 4: Substituting $<C_D> = 0$, we continue to rely on Eq. (4.16).

Case 1 expresses the fact that people tend to maintain the status quo. Case 2 assumes that an agent relies on the vaccination cost as alternative information. Case 3 assumes that an agent tends to take an inverse strategy if they are stalemated due to lack of information. Case 4 assumes that an agent behaves in an optimistic manner by assuming that free riders can be successful.

In the following text, we show the result of comparing Cases 1 and 4 of the DC model by means of MAS.

We assume that $N = 4900$ and $I_0 = 5$. We also assume that $\beta = 0.46$ and $\gamma = 1/3$ (the flu is assumed). In a simulation episode, one time-step consisting of the first stage, a vaccination campaign and the second stage, and an epidemic season continues until 3000 time-steps have passed. In a simulation study with varying relative vaccination cost, C_r, we observe the average vaccination coverage and

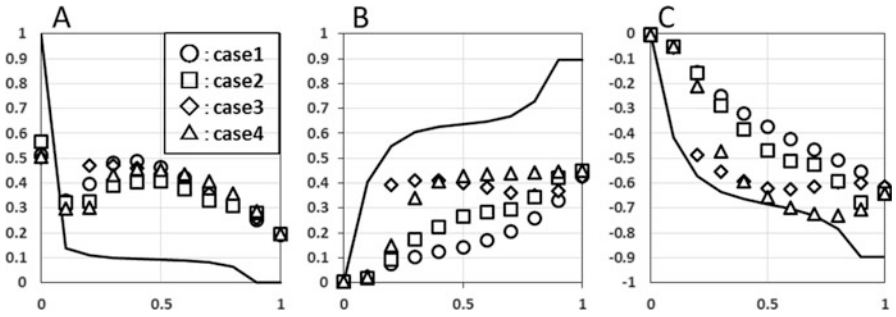

Fig. 4.11 Relationship between the vaccination cost and (**a**) the vaccination coverage, (**b**) the final epidemic size, and (**c**) the average social payoff for each of the four cases. Different symbols indicate the four cases, while the solid line indicates the result of the default setting according to Fu et al. (2011)

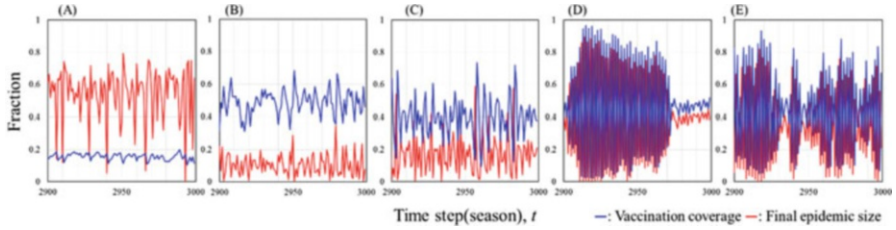

Fig. 4.12 Time evolution of the vaccination coverage (blue) and the final epidemic size (red) assuming $C_r = 0.3$: (**a**) default case, (**b**) Case 1, (**c**) Case 2, (**d**) Case 3, and (**e**) Case 4

final epidemic size in the last 1000 time-steps. The statistics shown below are based on 100 independent simulation episodes.

In Fig. 4.11, we show (a) the vaccination coverage, (b) the final epidemic size, and (c) the average social payoff versus the vaccination cost, C_r. Except at $C_r = 0$, our new adaptation model shows higher vaccination coverage, therefore leading to smaller final epidemic sizes than seen in the default model. However, note that, as far as the social payoff is concerned, Cases 3 and 4 show worse performance than either Cases 1 or 2 for the range of reasonable vaccination cosst, although they seem better than the default case.

To further examine the results, Fig. 4.12 shows a typical time evolution of 100 realizations for the last 100 time-steps prior to quasi-equilibrium in each case (Fig. 4.12b–e) with the default model (Fig. 4.12a) assuming $C_r = 0.3$. In the default case, as a general tendency, we see that larger final epidemic sizes with small time-fluctuations (compare Case 1 (Fig. 4.12b) and Case 2 (Fig. 4.12c)) result from lower but more stable vaccination coverage. Conversely, in Case 2 and more clearly in Case 1, relatively higher and stable vaccination coverage successfully results in stably lower final epidemic sizes. Interestingly, the situation we observe in Cases

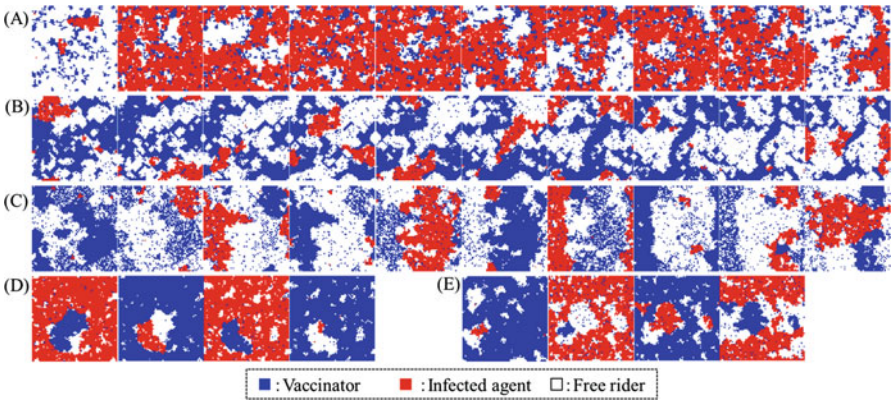

Fig. 4.13 Snapshots after 2940 time-steps assuming $C_r = 0.3$: (**a**) Default case, (**b**) Case 1, (**c**) Case 2, (**d**) Case 3, and (**e**) Case 4

3 (Fig. 4.12d) and 4 (Fig. 4.12e) is quite different. Significant time-fluctuations in both the vaccination coverage and the final epidemic size occur. This time-fluctuation seems to have two time-step-periodic dynamics, as confirmed below. This fluctuation may unwillingly cause the "vaccination-effectiveness" result mentioned above, because it brings a lower social payoff than the default model despite higher vaccination coverage and lower average final epidemic size for $0.3 \leq C_r \leq 0.6$.

Figure 4.13 offers further insight by showing continuous snapshots after 2940 time-steps for each of the four cases. Obviously, Cases 3 (Fig. 4.13d) and 4 (Fig. 4.13e) show two-time-step-periodic flipping in which a situation with vaccinators with a small number of infected agents who failed to free ride follows a situation with infected agents with a small number of vaccinators. This inevitably results in a pandemic-like situation in Cases 1 (Fig. 4.13b) and 2 (Fig. 4.13c) every other time-step, because the majority of vaccinators form small vaccinator clusters and are less spatially spread-out over the entire domain.

One plausible reason why these settings, especially those of Case 4, bring about such acute two-time-step flipping can be formulated as follows. From the inherent nature of its definition, Case 4 sees the non-vaccination strategy as being more advantageous than the vaccination strategy if there are no infected neighbors. Our model framework in not only Case 4 but also in other cases urges infected agents to adopt a vaccination strategy in the next time-step. This implies that agents in Case 4 tend to adopt the defective strategy (non-vaccination) if the outcome of the current time-step is good, whereas they adopt a cooperative strategy (vaccination) if the outcome is bad. This specific feature consequently results in the time flipping that was also observed in Win-Stay and Lose-Shift (WSLS)[29] of PD games and is a

[29]Imhof et al. (2007).

typical self-reflecting strategy (taking either the same offer to the current one or its opposite), unlike the copying-from-others' strategy seen in tit-for-tat games. As confirmed above, Cases 3 and 4 contain some fragments of a "self-reflecting strategy." One recent study[30] reports that some strategic adaptations based on the concept of WSLS can enhance cooperation for spatial-PD games. This is because sparsely located cooperative agents showing time-flipping manners with time-alternating defection are able to realize a reasonable amount of mutual reciprocity. This is interesting because the mechanism appears to be very different from the usual network reciprocity as previously understood, in which a situation of compactly clustered cooperators would be more likely to result in efficient network reciprocity. However, in the vaccination game, which has a different game structure than PD games, Cases 3 and 4 somehow contain a "self-reflecting" feature that does not result in any preferable consequences.

Unlike Cases 3 and 4, Cases 1 and 2 are able to achieve a preferable consequence, whereby a higher social payoff is established than that in the case of the default model. Figure 4.13b, c indicate that, in both cases, quite a few vaccinators are sparsely located and, consequently, vaccinators can ubiquitously exist in any corner of the domain. This is crucially important for suppressing the spread of epidemics.[31]

As discussed, we have established a new strategy-adaptation idea in the vaccination game. Our update rule does not provide a "copying probability" from a focal agent's neighbor, as in conventional models; rather, it directly gives a "vaccination probability" derived from a stochastic comparison between the vaccination cost commonly disclosed in public and the expected benefit resulting from adopting the non-vaccination strategy observed in an agent's neighborhood. We further define four subordinate models depending on how an agent behaves if they do not encounter a non-vaccinator among their neighbors.

The simulation results show that our new adaptation model generally results in higher vaccination coverage and smaller final epidemic sizes than those in the conventional model, which assumes social imitation of one of the neighbors.

However, depending on the subordinate models, there were two final consequences that either efficiently suppress epidemic spreading or do not. Specifically, the case assuming that an agent that retains their strategy even if there are no neighboring defectors (non-vaccinators) (Case 1) allows vaccinators to be sparsely located in the domain, successfully hampering the spread of an epidemic in this domain. Conversely, the case assuming that an agent takes the reverse strategy if there are no defectors (Case 3) or assumes that a free ride will be successful if they have no defectors among their neighbors (Case 4) results in an acute time-flipping behavior, which allows huge pandemics every two time-steps.

[30] Amaral et al. (2016).

[31] Fukuda et al. (2015).

4.1.4.3 Mean-Field Approximation (MFA) for the Three Updating Rules; IB-RA, SB-RA, and DC

In Sect. 4.1.1, we discussed the analytic model of SIR/V presuming an infinite and well-mixed population. Here, no spatial structure—i.e., no network that connects all of the individuals—is considered. Hence, when we apply the vaccination game to the analytic model, the strategy-update rule should be introduced. Since any spatial structure is considered, we must rely on the so-called mean-field approximation to evaluate a neighbor's payoff.

Individual-Based Risk Assessment (IB-RA)
In the present framework, there are four classes of individual in relation to cost burden: (i) a successful free-rider (SFR) who pays nothing; (ii) a failed free-rider (FFR) who pays -1; (iii) an infected vaccinator (IV) who pays $-C_r - 1$; and (iv) a healthy vaccinator (HV) who pays $-C_r$. Each individual can choose from one of two strategies: vaccination (hereinafter V) or non-vaccination (hereinafter NV). Thus, the transition probability that affects the time transition of x, which should be considered in the IB-RA rule, is covered by one of the following eight cases:

$$P(HV \leftarrow SFR) = \frac{1}{1+\exp[-(0-(-C_r))/\kappa]}, \tag{4.18a}$$

$$P(HV \leftarrow FFR) = \frac{1}{1+\exp[-(-1-(-C_r))/\kappa]}, \tag{4.18b}$$

$$P(IV \leftarrow SFR) = \frac{1}{1+\exp[-(0-(-C_r-1))/\kappa]}, \tag{4.18c}$$

$$P(IV \leftarrow FFR) = \frac{1}{1+\exp[-(-1-(-C_r-1))/\kappa]}, \tag{4.18d}$$

$$P(SFR \leftarrow HV) = \frac{1}{1+\exp[-(-C_r-0)/\kappa]}, \tag{4.18e}$$

$$P(SFR \leftarrow IV) = \frac{1}{1+\exp[-(-C_r-1-0)/\kappa]}, \tag{4.18f}$$

$$P(FFR \leftarrow HV) = \frac{1}{1+\exp[-(-C_r-(-1))/\kappa]}, \tag{4.18g}$$

$$P(FFR \leftarrow IV) = \frac{1}{1+\exp[-(-C_r-1-(-1))/\kappa]}. \tag{4.18h}$$

Strategy-Based Risk Assessment (SB-RA)
As discussed above, SB-RA modifies the imitation probability to reflect the situation in which an individual tends to assess the risk based on a socially averaged payoff because of the prevalence of information about epidemics (probably given by the media). In the analytic framework, the transition probability that we must consider now is one of the following:

$$P(HV \leftarrow NV) = \frac{1}{1 + \exp[-(\langle \pi_D \rangle - (-C_r))/\kappa]}, \tag{4.19a}$$

$$P(IV \leftarrow NV) = \frac{1}{1 + \exp[-(\langle \pi_D \rangle - (-C_r - 1))/\kappa]}, \tag{4.19b}$$

$$P(SFR \leftarrow V) = \frac{1}{1 + \exp[-(\langle \pi_C \rangle - 0)/\kappa]}, \tag{4.19c}$$

$$P(FFR \leftarrow V) = \frac{1}{1 + \exp[-(\langle \pi_C \rangle - (-1))/\kappa]}. \tag{4.19d}$$

Direct Commitment (DC)
Direct commitment is the representative framework of "self-estimation", which differs from "social-imitation" methods such as IB-RA and SB-RA. Applying MFA, the transition probability that we must consider now is one of the following:

$$P(V \leftarrow NV) = \frac{1}{1 + \exp[-(\langle \pi_D \rangle - \langle \pi_C \rangle)/\kappa]}, \tag{4.20a}$$

$$P(NV \leftarrow V) = \frac{1}{1 + \exp[-(\langle \pi_C \rangle - \langle \pi_D \rangle)/\kappa]}. \tag{4.20b}$$

4.1.5 Vaccination Game Through Analytic Approach

Here, let us revisit the analytical approach. Recall that x means the fraction of the total population that either vaccinates or takes intermediate defensive measures. Looking back at Fig. 4.7, strategy updating takes place after each epidemic season, as defined above, thereby inevitably changing x. We have two different epidemic models, namely the effectiveness model and the efficiency model, and three different updating rules, namely IB-RA, SB-RA, and DC. Hence, we establish the following six different types of dynamics:

Effectiveness model + IB-RA:

$$\begin{aligned}\frac{dx}{dt} = \; & x(1-x)(e+(1-e)\exp[-R_0R(x,\infty)])\exp[-R_0R(x,\infty)] \\ & \quad (P(SFR \leftarrow HV) - P(HV \leftarrow SFR)) \\ & + x(1-x)(e+(1-e)\exp[-R_0R(x,\infty)]) \\ & \quad (1-\exp[-R_0R(x,\infty)])(P(FFR \leftarrow HV) - P(HV \leftarrow FFR)) \\ & + x(1-x)(1-e)(1-\exp[-R_0R(x,\infty)])\exp[-R_0R(x,\infty)] \\ & \quad (P(SFR \leftarrow IV) - P(IV \leftarrow SFR)) \\ & + x(1-x)(1-e) \\ & \quad (1-\exp[-R_0R(x,\infty)])^2(P(FFR \leftarrow IV) - P(IV \leftarrow FFR)),\end{aligned} \tag{4.21}$$

Efficiency model + IB-RA:

$$\begin{aligned}\frac{dx}{dt} = \; & x(1-x)\exp[-(1-\eta)R_0R(x,\infty)]\exp[-R_0R(x,\infty)] \\ & \quad (P(SFR \leftarrow HV) - P(HV \leftarrow SFR)) \\ & + x(1-x)\exp[-(1-\eta)R_0R(x,\infty)] \\ & \quad (1-\exp[-R_0R(x,\infty)])(P(FFR \leftarrow HV) - P(HV \leftarrow FFR)) \\ & + x(1-x)(1-\exp[-(1-\eta)R_0R(x,\infty)])\exp[-R_0R(x,\infty)] \\ & \quad (P(SFR \leftarrow IV) - P(IV \leftarrow SFR)) \\ & + x(1-x)(1-\exp[-(1-\eta)R_0R(x,\infty)]) \\ & \quad (1-\exp[-R_0R(x,\infty)])(P(FFR \leftarrow IV) - P(IV \leftarrow FFR)),\end{aligned} \tag{4.22}$$

Effectiveness model + SB-RA:

$$\begin{aligned}\frac{dx}{dt} = \; & -x(1-x)(e+(1-e)\exp[-R_0R(x,\infty)])P(HV \leftarrow NV) \\ & - x(1-x)(1-e)(1-\exp[-R_0R(x,\infty)])P(IV \leftarrow NV) \\ & + x(1-x)\exp[-R_0R(x,\infty)]P(SFR \leftarrow V) \\ & + x(1-x)(1-\exp[-R_0R(x,\infty)])P(FFR \leftarrow V),\end{aligned} \tag{4.23}$$

Efficiency model + SB-RA:

$$\begin{aligned}\frac{dx}{dt} = \; & -x(1-x)\exp[-(1-\eta)R_0R(x,\infty)]P(HV \leftarrow NV) \\ & - x(1-x)(1-\exp[-(1-\eta)R_0R(x,\infty)])P(IV \leftarrow NV) \\ & + x(1-x)\exp[-R_0R(x,\infty)]P(SFR \leftarrow V) \\ & + x(1-x)(1-\exp[-R_0R(x,\infty)])P(FFR \leftarrow NV),\end{aligned} \tag{4.24}$$

Effectiveness or efficiency model + DC:

$$\frac{dx}{dt} = -xP(V \leftarrow NV) + (1-x)P(NV \leftarrow V). \tag{4.25}$$

It is worthwhile to note that Eq. (4.25) is qualitatively consistent with what are called replicator dynamics (see; Eq. (2.6) in Sect. 2.1.1), one of the most common

concepts in evolutionary game theory for expressing a system's dynamics. All of the above dynamical ODEs can be solved numerically. We introduce a so-called explicit scheme[32] for the time-varying terms to obtain a numerical solution; namely, vaccination coverage at equilibrium.

Figures 4.14 and 4.15 relating to the effectiveness and efficiency models, respectively, give the final epidemic size (left-hand panels), vaccination coverage (central panels), and average social payoff (right-hand panels) for various strategy-updating rules, namely IB-RA (upper panels), SB-RA (middle panels), and DC (lower panels).

The regions colored uniformly in light red (final epidemic size), dark blue (vaccination coverage), and light blue (average social payoff) indicate a pandemic taking place, where most individuals do not use vaccination (precisely speaking, not using either imperfect vaccination or an intermediate defense measure); thus, an almost-full-scale spread of infection occurs. Roughly speaking, these regions emerge when a smaller effectiveness (efficiency) is presumed or a larger cost is imposed. This seems quite natural because most individuals tend to shy away from vaccination if it is less reliable and/or too expensive. The border between each of these monotone regions and the remaining region implies a combination of critical effectiveness (efficiency) and critical vaccination cost to control the spread of an epidemic, causing an obvious change between the pandemic and controlled phases. As far as the controlled phase is concerned, interestingly, lower effectiveness (efficiency) can realize a higher vaccination coverage, which is also helped by the effect of lower cost. Even if a large fraction of individuals use vaccination, the epidemic cannot be eradicated because of the lower reliability of vaccination.

The detailed tendencies of the three updating rules differ, although the overall tendency is the same to some extent. Comparing the effectiveness model and the efficiency model, the latter has a wider pandemic phase at first glance. This implies that an intermediate defense measure with a certain η is less effective at suppressing the spread of an epidemic than imperfect vaccination, with e being defined as having the same numerical value as η.

To validate this theoretical framework, we conducted a series of numerical simulations based on the MAS approach discussed previously. Because we assumed a well-mixed population, we presumed a complete graph as the underlying network connecting the agents. Following previous studies, we set $\beta = 0.00088$, which was determined as the minimum transition rate β for which the final epidemic size exceeds the predefined threshold of 0.9 without vaccination. The results are shown in Figs. 4.16 and 4.17. Generally, all those results are respectively consistent with Figs. 4.14 and 4.15, although subtle discrepancies arise from the fact that the simulation presumed a finite population size of $N = 1000$.

[32] A reader can consult any standard textbooks on applied mathematics with numerical approaches. One example is provided below: Tanimoto (2014).

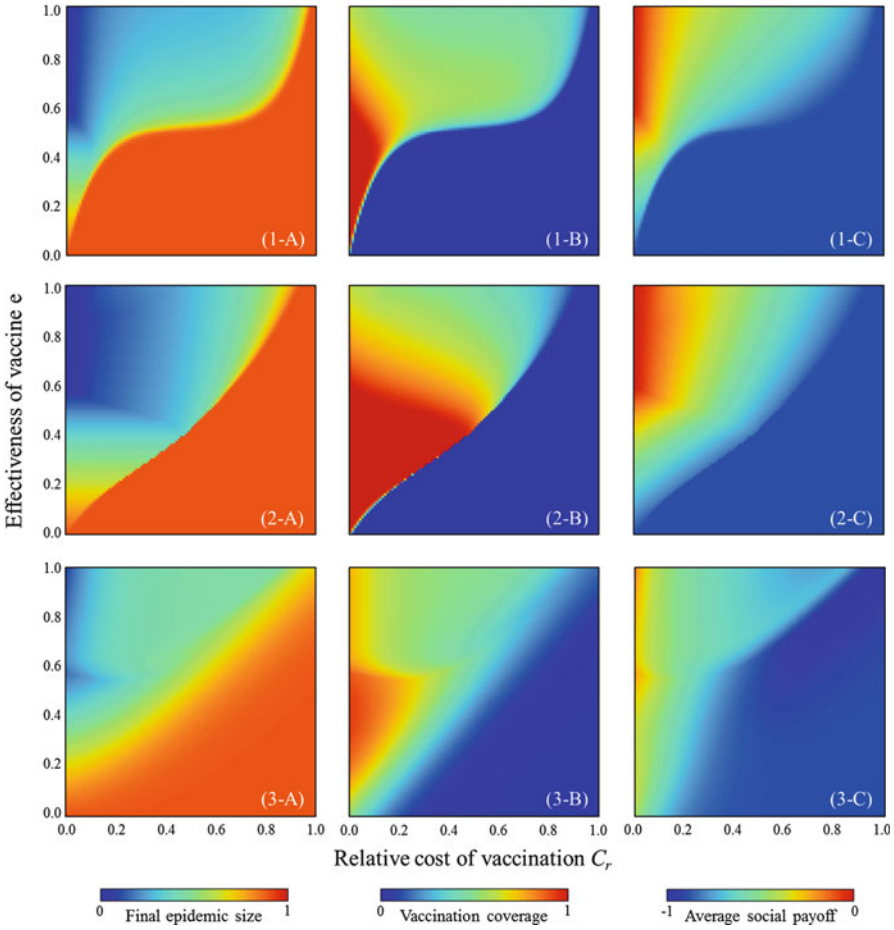

Fig. 4.14 Final epidemic size (left-hand panels; *-A), vaccination coverage (central panels; *-B), and average social payoff (right-hand panels; *-C) for strategy-updating rule IB-RA (upper panels; 1-*), SB-RA (middle panels; 2-*), and DC (lower panels; 3-*). The effectiveness model is applied

4.1.5.1 Summary

Presuming an infinite and well-mixed population, we established an analytical framework for a vaccination game in which three different strategy-updating rules were separately implemented. Our main concern was the extent to which the evolutionary picture differed when either imperfect vaccination or an intermediate defense measure was introduced to suppress the spread of an epidemic. We

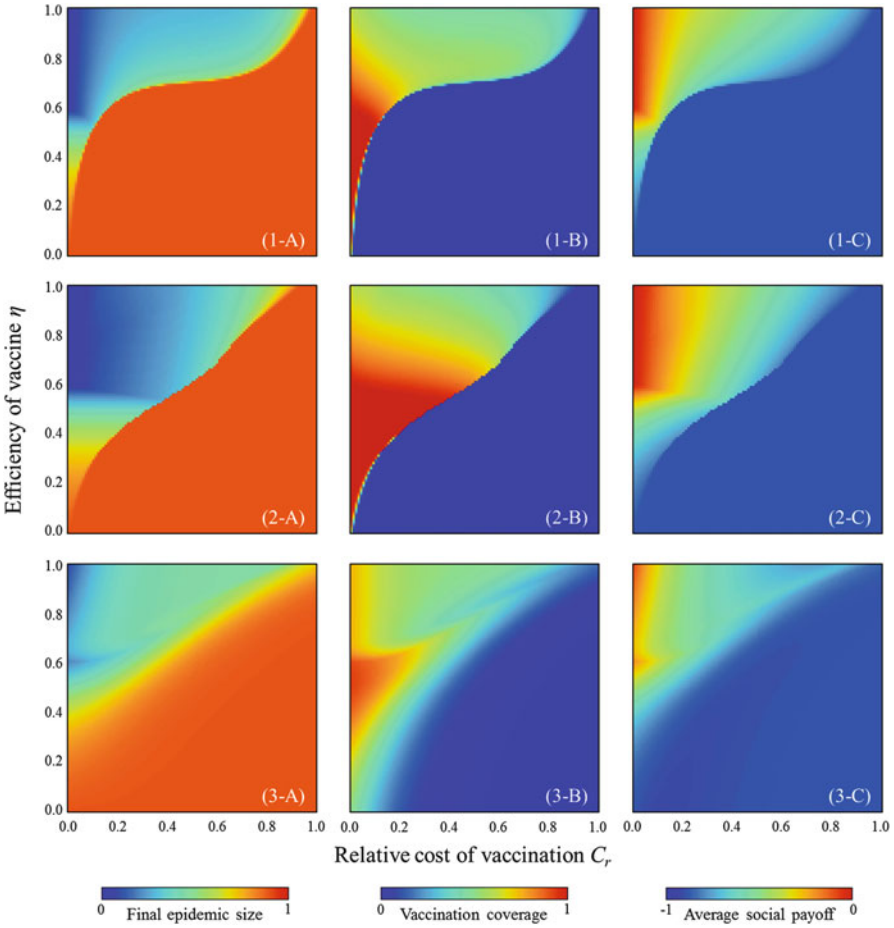

Fig. 4.15 Final epidemic size (left-hand panels; *-A), vaccination coverage (central panels; *-B), and average social payoff (right-hand panels; *-C) for strategy-updating rules IB-RA (upper panels; 1-*), SB-RA (middle panels; 2-*), and DC (bottom panels; 3-*). The efficiency model is applied

successfully established the respective evolutionary formulas, showing numerical results. We validated our framework by comparing its predictions with simulation results. As long as the same coefficient values for effectiveness and efficiency are presumed, an intermediate defense measure is marginally inferior to an imperfect vaccination.

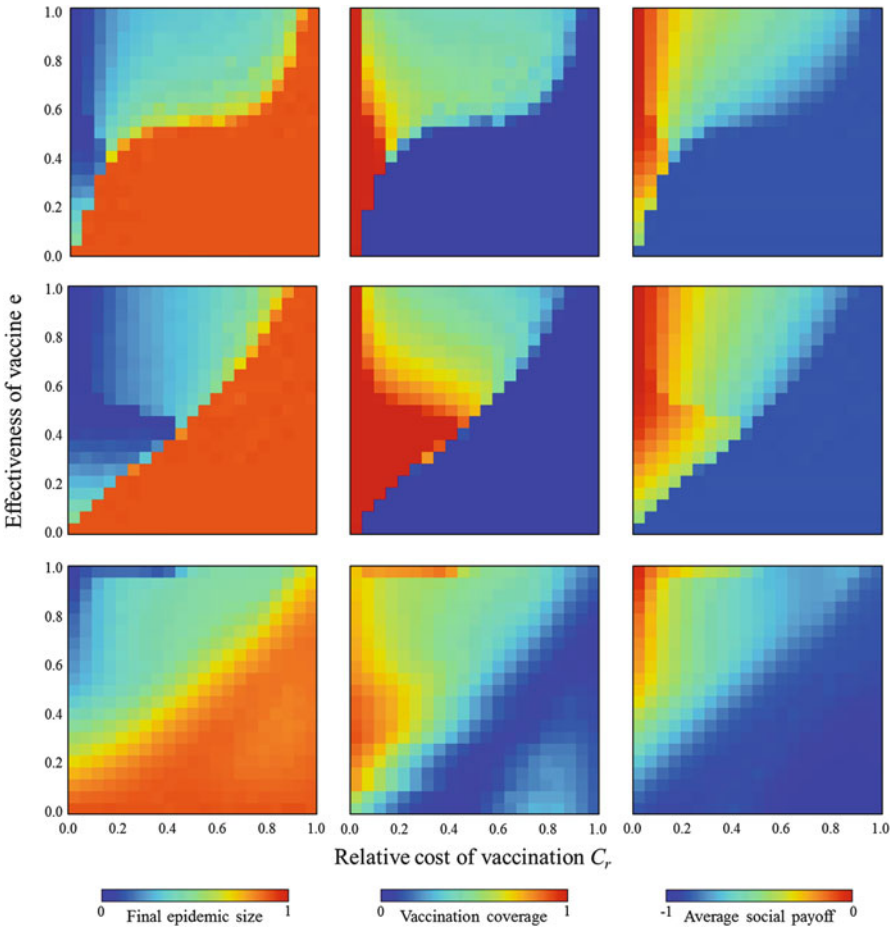

Fig. 4.16 MAS result for the final epidemic size (left-hand panels; *-A), vaccination coverage (central panels; *-B), and average social payoff (right-hand panels; *-C) for strategy-updating rules IB-RA (upper panels; 1-*), SB-RA (middle panels; 2-*), and DC (lower panels; 3-*). Effectiveness model is applied. We presumed a complete graph with $N = 1000$, $\beta = 0.00088$, and $\gamma = 1/3$

4.2 Optimal Subsidy-Policy Design for Vaccination

Many studies (called "vaccination games") have been reported on reproducing the vaccination dilemma. Meanwhile, returning to the social aspect of a voluntary vaccination policy, it may be possible to either solve or relax the vaccination dilemma. One provision is financial support from the central (or municipal) governments in the form of a subsidy. In fact, aged individuals and other people needing support are subsidized for the vaccination of seasonal influenza in Japan. On the other hand, the Japanese government is suffering from a huge budget deficit resulting from the stably increasing cost of medical care as well as the welfare cost

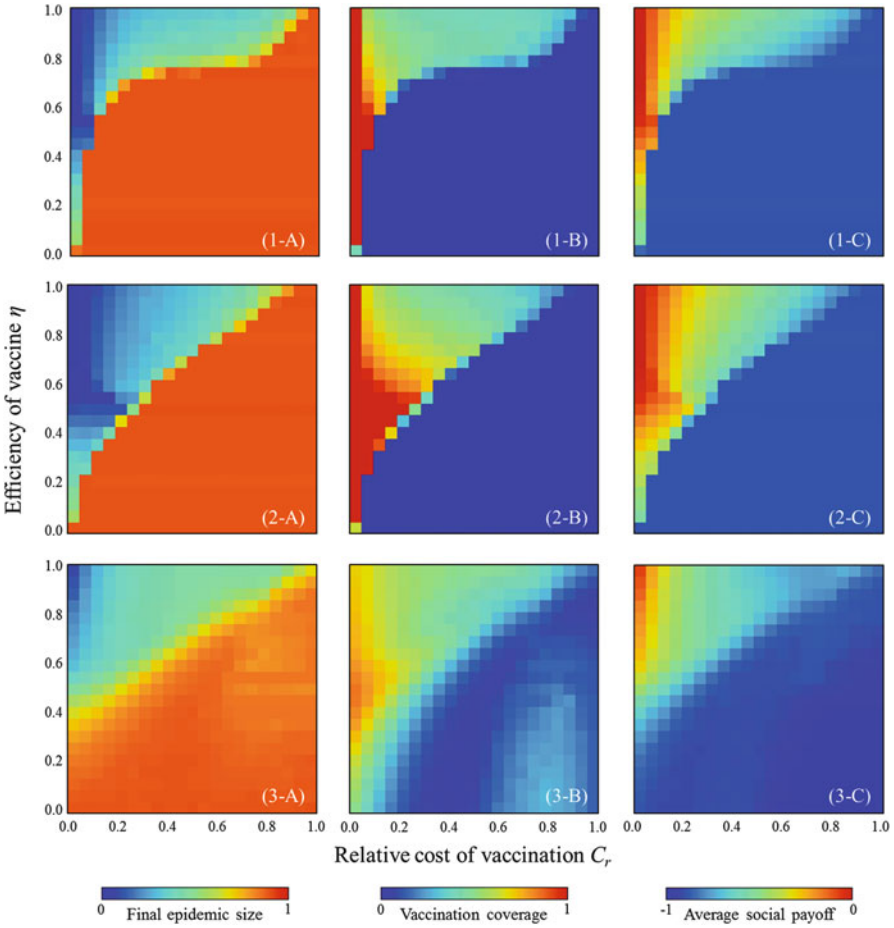

Fig. 4.17 MAS results for final epidemic size (left-hand panels; *-A), vaccination coverage (central panels; *-B), and average social payoff (right-hand panels; *-C) for strategy-updating rules IB-RA (upper panels; 1-*), SB-RA (middle panels; 2-*), and DC (lower panels; 3-*). The efficiency model is applied. We presumed a complete graph with $N = 1000$, $\beta = 0.00088$, and $\gamma = 1/3$

for an aged population. As long as a subsidy effectively reduces the entire social cost including the additional tax burden, such a subsidy policy for vaccination can be fully justified. Therefore, the question of what subsidy policy meeting this criterion can minimize the social cost is quite an important subject.

There have been several previous studies approaching this subject from different viewpoints. Gavious & Yamin[33] pioneered this field. Based on a macro model referring to SIR dynamics validated by field observations, they insisted that a

[33]Gavious and Yamin (2013).

sufficiently large subsidy is needed when an epidemic period becomes long, and middle- and younger-aged people should be subsidized rather than the elderly. Zhang et al.[34] gave a holistic report by means of a multiagent-simulation (MAS) approach. Their model presumed that epidemic dynamics and strategy dynamics share with a same time scale unlike most of the previous vaccination games (e.g., Fu et al. [10]) presuming. They compared two policies; distributing free-tickets as long as the budget allows (hereafter, called free-ticket policy) and distributing a discount ticket to all individuals (they called this a partial-offset policy; hereafter we call it a discount policy). They defined the total social cost as the sum of the infection and vaccination costs paid by each individual. In this sense, their subsidy was externalized as a "gift-of-nature". As we discuss later, externalizing the effect from a subsidy is justified if one evaluates the total social cost. Zhang et al.,[35] relying on Fu's vaccination-game model as well as an analytic approach [10], comparing a free-ticket policy with a discount policy for various cases. Zhang et al.[36] also investigated how random and targeted subsidization of individuals differently suppress disease spread. Tang et al.[37] explored an interesting subsidy system. They introduced a new specific system in which only neighboring individuals are able to mutually support each other, unlike the usual subsidy system in which the central government comprehensively levies an additional tax upon all individuals. They concluded that their system is more efficient than the conventional one in terms of increasing vaccination coverage as a whole. Li et al.[38] reported a well-designed theoretical model considering a subsidy system assuming an SIRS model (which allows an individual to be infected twice in a single season), rather than an SIR model. Ding et al.[39] highlighted how effectively a subsidy policy works to minimize disease spread on a scale-free network when a free-ticket subsidy is preferentially distributed to higher-degree agents (hub agents), rather than using random allocation.

Returning to the concept of a subsidy, we should note that a vaccinator given a free ticket may induce some neighboring agents to become voluntary vaccinators. Because of this positive effect, an appropriate subsidy policy may reduce the total social payoff as a whole. This seems analogous to the effect of a "stubborn vaccinator" investigated by Fukuda et al.[40] or a "zealot cooperator" in a spatial version of a 2×2 game, investigated Matsuzawa et al.,[41] meaning an agent insensitive to strategy updating. One important difference between a subsidized vaccinator and a stubborn vaccinator is whether or not their spatial location in a domain is frozen. Different people are given free tickets from one season to another.

[34]Zhang et al. (2013).
[35]Zhang et al. (2014).
[36]Zhang et al. (2017).
[37]Tang et al. (2017).
[38]Li et al. (2017).
[39]Ding et al. (2018).
[40]Fukuda and Tanimoto (2016).
[41]Matsuzawa et al. (2016).

Motivated by all of the aforementioned background, this section quantitatively answers the question of whether or not a vaccination-subsidy policy can really reduce the total social cost, comprising vaccination cost, diseases cost, and the tax burden needed to implement the subsidy. If this is possible, the question becomes what subsidy policy (e.g., distributing free or discount tickets) can minimize the total social cost. To do that, we, first off, establish a comprehensive vaccination-game model taking account of various subsidy mechanisms, and shed some light on what subsidy policy realizes the socially best solution. We use MAS as well as analytical approaches.

4.2.1 How We Model Subsidy Policy

Regardless of whether we take an MAS approach (see Sect. 4.1.3) or an analytic approach (See; Sect. 4.1.5), we follow the vaccination game depicted in the previous Section.

The key point is how we model various vaccination policies taken by the government.

From a policy-design point of view, we build four different subsidization procedures, which depend on whether a free-ticket or discount policy is presumed, and what target individuals are presumed.

Model A-1: Free-ticket policy. The target is as many randomly selected individuals as the subsidy budget will allow.

Model A-2: Free-ticket policy. The target is randomly selected individuals who did not vaccinate in the previous time step. As many tickets are distributed as the subsidy budget allows. Unlike Model A-1, this model targets potential non-vaccinators with the intent of increasing social-vaccination coverage.

Model A-3: Free-ticket policy. The target is randomly selected individuals who vaccinated in the previous time step. As many tickets are distributed as the subsidy budget allows. Unlike the previous two models, Model A-3 only gives the privilege of a free ticket to a cooperator (vaccinator) in the previous time step.

Model B: Discount policy. All individuals are given a discounting ticket to reduce the individual cost burden, C_r, by as much as possible. But this premier makes sense only when he decides committing vaccination.

Figure 4.18 illustrates the subsidy models above, where f_C is the cooperation rate (rate of vaccination) after each individual decides whether to vaccinate.

Let σ be the fraction of vaccinators to be subsidized out of the total population, N. Hence, the additional tax burden per capita for subsidies, TAX, is given as

$$TAX = \frac{C_r \cdot \sigma \cdot N}{N} = C_r \cdot \sigma. \tag{4.26}$$

Here, we define the total social payoff (TSP) as below:

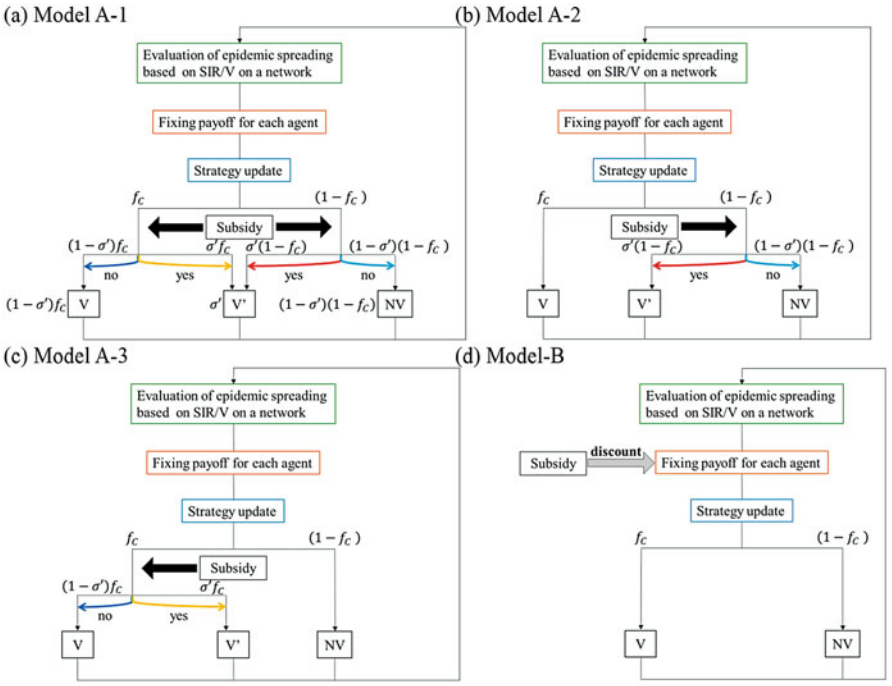

Fig. 4.18 Four models for subsidy policies; (**a**) Model A-1, (**b**) Model A-2, (**c**) Model A-3, and (**d**) Model-B

$$TSP = -C_r \cdot (VC - \sigma) - 1 \cdot FES - TAX = -C_r \cdot VC - 1 \cdot FES. \tag{4.27}$$

TSP must be negative, hence, |*TSP*| means the total social cost taking account of all aspects including the budget for a subsidy policy. It is worthwhile to note that *TSP* is independent of the size of a subsidy. This is quite natural, because governmental expenditure should be consistent with revenue as a whole. Eq. (4.27) revels that the subsidy works, in a sense, as a social "catalyst", allowing us to evaluate how much a subsidy (of a certain amount based on a certain policy) is able to enhance the number of voluntary vaccinators (or vaccinators in general). Thus, *TSP* can be thought of as an appropriate index for evaluating the social efficiency of a subsidy policy in the present study. We do not consider the additional cost burden levied on each individual, *TAX*, when evaluating each individual's payoff, because, in the real world, we cannot recognize exactly how much tax each individual has paid specifically for subsidies, as tax is generally levied. Thus, the breakdown of expenditure is not visible to us. However, a subsidy given as a free ticket or a discounting coupon is, of course, manifestly visible to the subsidized individual. This is why such a subsidy policy is generally favored and enthusiastically accepted by typical people in the street, on which the politics of "populism" tends to ride.

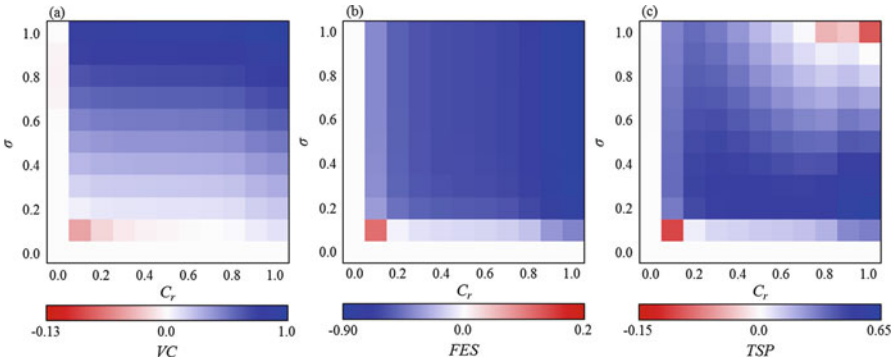

Fig. 4.19 Color indicates the difference between subsidy ($\sigma > 0$) and non-subsidy ($\sigma = 0$) cases. Panel (**a**) vaccination coverage (VC), (**b**) final epidemic size (FES), (**c**) total social payoff (TSP). Models A-1, lattice, and IB-RA are presumed

4.2.2 Results and Discussion; MAS Approach

We measure the vaccination coverage (VC), final epidemic size (FES), and total social payoff (TSP) with varying vaccination cost (C_r) and subsidy size (σ). To highlight how the introduction of a subsidy affects a social equilibrium, we consider the difference between cases with and without a subsidy. In the following visual results throughout this section, we are concerned on those differences for VC, FES and TSP. This, the three evaluation values respectively range from negative to positive. Hereafter, let us call a case without subsidy "default".

Figure 4.19 respectively shows the results for VC, FES, and TSP when presuming Model A-1 as a subsidy setting, where a free ticket is distributed to randomly selected individuals regardless of V or NV, using a lattice for an underlying network and IB-RA for a strategy-updating rule. Red indicates a region where a subsidy devastates social efficiency, leading to a smaller fraction of vaccinators, larger final epidemic size, and smaller total social payoff than in the default case.

Let us consider the total social payoff (Fig. 4.19c), where there are two negative regions. One occurs when a smaller vaccination cost and subsidy budget are presumed. Another takes place when a larger vaccination cost and subsidy budget are presumed. The former accords with regions of negative-VC difference (Fig. 4.19a) and positive-FES difference (Fig. 4.19b); in this region, subsidies going to defective individuals who have no intention of vaccinating eventually reduce the number of voluntary vaccinators (e.g., self-financed vaccinators), leading to a wider spread of disease and pushing up the total social cost due to a larger number of infectious individuals. This is quite ironic from the viewpoint of social context. The implication drawn from this particular case is that too small of a budget for the subsidy policy in the case of a relatively smaller vaccination cost fails to increase the number of voluntary vaccinators, but rather causes individuals to refrain from cooperating in establishing a herd immunity. Unlike the first negative region

with a smaller budget, the second negative region with too much of a subsidy in the case of a relatively larger vaccination cost (Fig. 4.27c) successfully realizes a larger VC (Fig. 4.19a) and smaller FES (Fig. 4.19b) vis-à-vis the default case. This fact proves that overspending on the financial support brings too much vaccinators including forcefully committing individuals. It is obviously over the socially approved level whereat the cost of infection of society as a whole is balanced by the cost of vaccination including a subsidy policy (which may be the social optimum). Therefore, to ensure an optimal subsidy-policy design to pre-empt vaccination, an appropriate level (neither too large nor too small) of subsidy expenditure is quite important. Hereafter, let us call the former region the "first negative region" and the latter the "second negative region".

Figures 4.20 and 4.21 illustrate how targeting subsidies at different groups affects the total social cost in comparison to Fig. 4.19. Interestingly, Model A-3 (Fig. 4.21), where financial support is given only to cooperative individuals, does not incur the first negative region of TSP difference. This suggests that, to increase the number of voluntary vaccinators, helping potential vaccinators rather than helping potential social-defectors aiming to free-ride on herd immunity would be more beneficial. Relating to this, comparison of Fig. 4.19 with Fig. 4.20 implies that the result of helping potential social defectors brings almost no difference from that of targeting any arbitrary people.

Figure 4.22 shows the result when the Model-B discount policy is presumed. This should be compared with Fig. 4.21 on the grounds that target individuals are potential cooperators (the discount becomes meaningful only when an individual voluntarily vaccinates). The result shows no specific difference between those two policies. As long as the intent is to increase the number of voluntary vaccinators in a society, a "priority system" distributing free tickets to some potential vaccinators does not differ in effect from an "egalitarian system" allowing a certain discount (distributing discount coupons) to all potential vaccinators.

Figure 4.23 highlights the difference in strategy updating. This should be compared with Fig. 4.19. With presuming SB-RA relying on global information when updating strategy, the first negative region with a small budget is slightly extended compared with IB-RA relying on local information (only referring to the neighbors' payoff), whereas the second negative region with a too-high budget shrinks slightly vis-à-vis IB-RA. Yet, as a whole, the difference between those two strategy-updating rules looks small, which indicates whether an individual decision referring to global or only local information makes a less-significant difference in the social cost.

Figures 4.24 and 4.25 compare the topological contributions. Figure 4.24 presumes RRG while Fig. 4.25 show the case of BA-SF, which should be also compared with Fig. 4.19. The comparison between Fig. 4.19 and Fig. 4.24 confirms that the effect of random links results in a quite large difference from the case of a regular & homogeneous network (lattice; Fig. 4.19). The existence of random links devastates the efficiency of subsidy policy, resulting from the significantly wider negative regions compared with those observed in Fig. 4.19. In particular, the first negative region with a smaller budget spreads to much larger C_r as well as larger σ. Moreover, comparing RRG (Fig. 4.24) with BA-SF (Fig. 4.25), we should note that

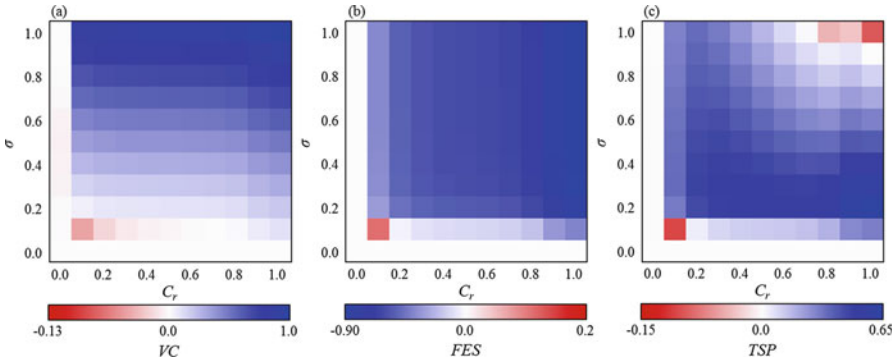

Fig. 4.20 Color indicates the difference between subsidy ($\sigma > 0$) and non-subsidy ($\sigma = 0$) cases. Panel (**a**) vaccination coverage (VC), (**b**) final epidemic size (FES), (**c**) total social payoff (TSP). Models A-2, lattice, and IB-RA are presumed

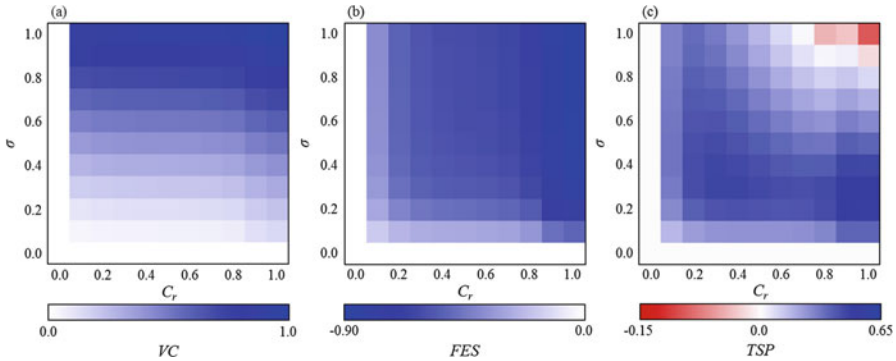

Fig. 4.21 Color indicates the difference between subsidy ($\sigma > 0$) and non-subsidy ($\sigma = 0$) cases. Panel (**a**) vaccination coverage (VC), (**b**) final epidemic size (FES), (**c**) total social payoff (TSP). Models A-3, lattice, and IB-RA are presumed

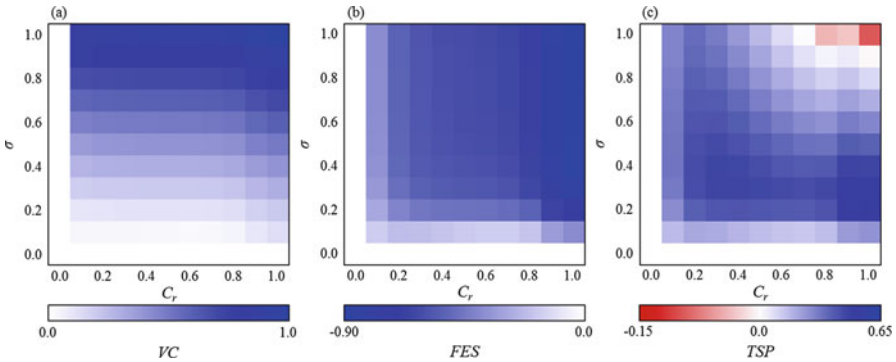

Fig. 4.22 Color indicates the difference between subsidy ($\sigma > 0$) and non-subsidy ($\sigma = 0$) cases. Panel (**a**) vaccination coverage (VC), (**b**) final epidemic size (FES), (**c**) total social payoff (TSP). Models B, lattice, and IB-RA are presumed

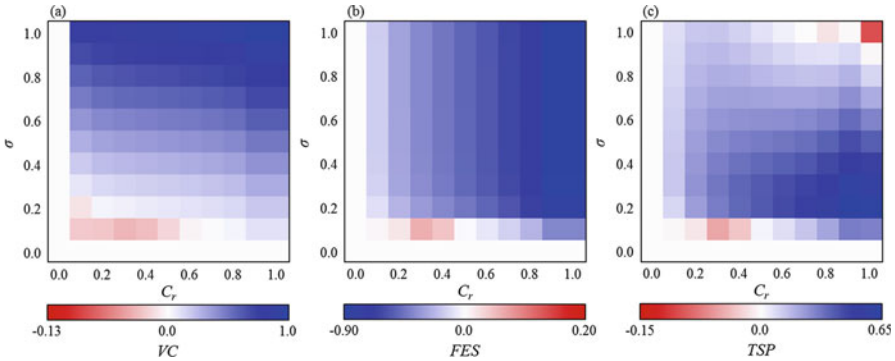

Fig. 4.23 Color indicates the difference between subsidy ($\sigma > 0$) and non-subsidy ($\sigma = 0$) cases. Panel (**a**) vaccination coverage (VC), (**b**) final epidemic size (FES), (**c**) total social payoff (TSP). Models A-1, lattice, and SB-RA are presumed

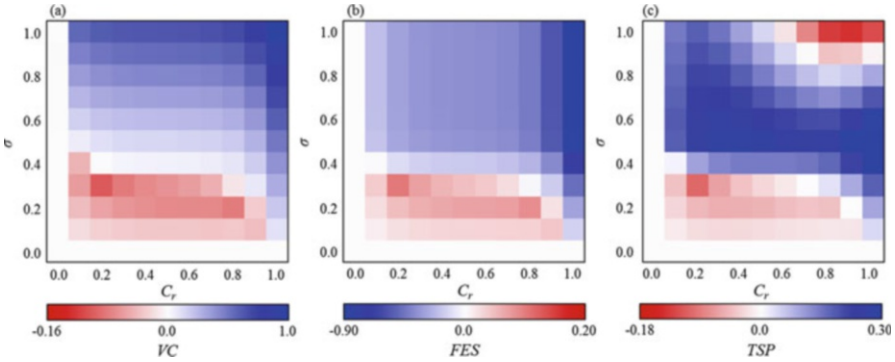

Fig. 4.24 Color indicates the difference between subsidy ($\sigma > 0$) and non-subsidy ($\sigma = 0$) cases. Panel (**a**) vaccination coverage (VC), (**b**) final epidemic size (FES), (**c**) total social payoff (TSP). Models A-1, RRG, and IB-RA are presumed

the latter shows worse performance than the former. Thus, not only the existence of random links but also the non-uniform degree distribution undermines to the subsidy system. As is commonly recognized, human social networks can feature scale-free degree distributions, unlike small-world characteristics.[42] Recalling this fact, we should note the important result observed in Fig. 4.25c that the subsidy works inversely to reduce the total social cost when almost any vaccination-cost range ($C_r < 0.9$) and feasibly realistic budget size ($\sigma < 0.5$) are presumed, which may be meaningful from the standpoint of social applications.

Figure 4.26 provides the results when BA-SF and SB-RA are presumed. Comparing with Fig. 4.25, we should note that, unlike the lattice case (comparison

[42]Masuda (2017).

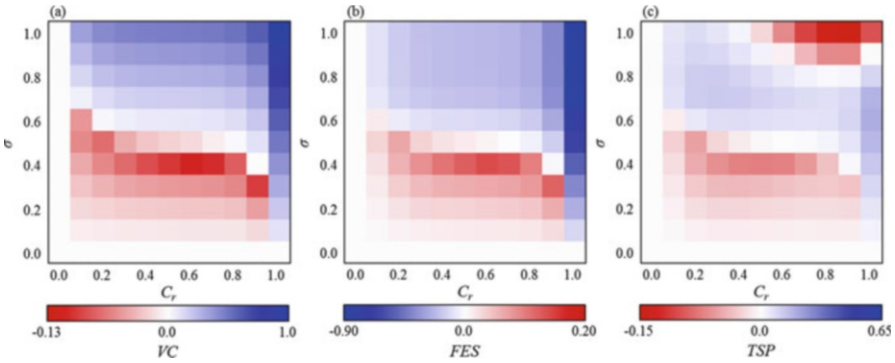

Fig. 4.25 Color indicates the difference between subsidy ($\sigma > 0$) and non-subsidy ($\sigma = 0$) cases. Panel (**a**) vaccination coverage (VC), (**b**) final epidemic size (FES), (**c**) total social payoff (TSP). Models A-1, BA-SF, and IB-RA are presumed

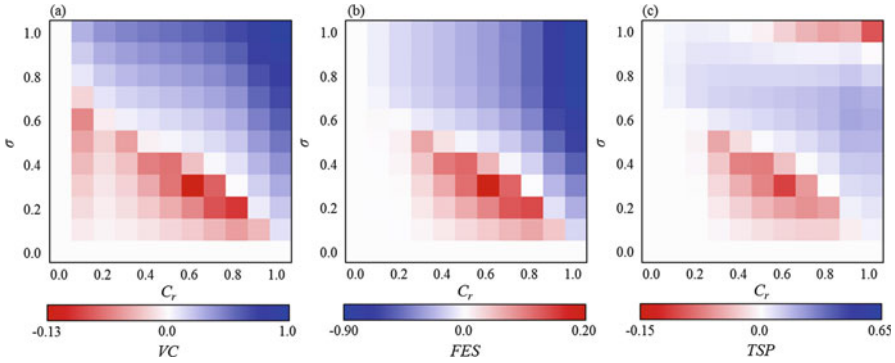

Fig. 4.26 Color indicates the difference between subsidy ($\sigma > 0$) and non-subsidy ($\sigma = 0$) cases. Panel (**a**) vaccination coverage (VC), (**b**) final epidemic size (FES), (**c**) total social payoff (TSP). Models A-1, BA-SF, and SB-RA are presumed

between Figs. 4.19 and 4.23), BA-SF causes a relatively significant difference between the two different update rules. Contrasting with the lattice case, the introduction of global information (Fig. 4.26) somewhat relaxes the drawbacks of subsidy introduction. In particular, the first negative region with a relatively smaller budget is shrunken compared with the counterpart region in Fig. 4.25.

We show the free-ticket and discount-coupon cases with BA-SF being presumed as an underlying network, as shown in Figs. 4.27 and 4.28 (where IB-RA is imposed). Between those two, likewise the lattice case does (comparison between Figs. 4.21 and 4.22), there is no phenomenal difference. But more importantly, as the lattice showing as well, the first negative region, where a relatively smaller budget size rather devastating the social efficiency than the default case, does disappear. Although, as mentioned above, a subsidy policy presuming a BA-SF network may work badly in terms of social efficiency vis-a-vis the lattice case, it would be significantly improved

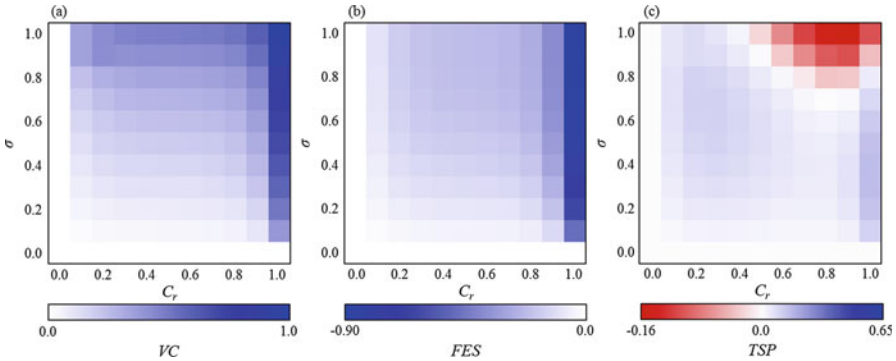

Fig. 4.27 Color indicates the difference between subsidy ($\sigma > 0$) and non-subsidy ($\sigma = 0$) cases. Panel (**a**) vaccination coverage (VC), (**b**) final epidemic size (FES), (**c**) total social payoff (TSP). Models A-3, BA-SF, and IB-RA are presumed

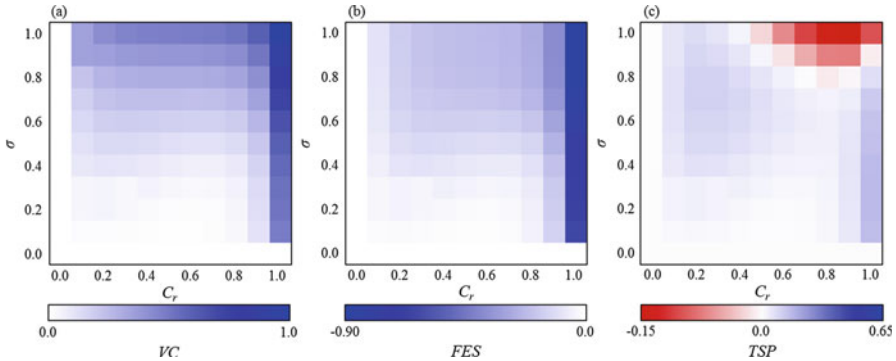

Fig. 4.28 Color indicates the difference between subsidy ($\sigma > 0$) and non-subsidy ($\sigma = 0$) cases. Panel (**a**) vaccination coverage (VC), (**b**) final epidemic size (FES), (**c**) total social payoff (TSP). Models B, BA-SF, and IB-RA are presumed

if the government were to rely on the principle of "heaven helps those who help themselves"; e.g., a policy distributing free tickets only to potential vaccinators or distributing discount coupons that are only valid to those who actually vaccinate. This seems quite important from a social-application point of view.

4.2.3 Results and Discussion: Analytic Approach

We utilize the modified SIR model, which can reproduce the epidemic dynamics arising from imperfect vaccination, namely the effectiveness model introduced by our previous work Ref [13]. In the present study, we further modify for the

vaccination game under a subsidy policy. We assume a population that is infinite and ideally well mixed, implying that the total population of N in the MAS model is normalized as $N = 1$. This suggests that the individual tax burden for a subsidy denoted by TAX in Eq. (4.26) is equivalent to the exact size of the subsidy budget (hereafter denoted by SB) in the analytical model. Namely, to connect the MAS model with the analytical model, we should note

$$TAX = \frac{C_r \cdot \sigma \cdot N}{N} = C_r \cdot \sigma = C_r \cdot \sigma \cdot 1 = SB. \tag{4.28}$$

4.2.3.1 Epidemic Dynamics

A vaccinated population is separated into two classes: immune individuals obtaining perfect immunity and non-immune ones failing to obtain immunity. Let the effectiveness of the vaccine and the vaccination coverage be e ($0 \leq e \leq 1$) and x, respectively. The effectiveness refers to a situation in which some vaccinated agents acquire immunity with effectiveness probability e; meanwhile, the remaining agents fail to acquire immunity with probability $1 - e$. The fraction of vaccinated individuals with immunity must be ex, while that of non-immune individuals is $(1 - ex)$. We can express the final epidemic size, R, in relation to both x and time t at equilibrium ($t = \infty$) as

$$R(x, \infty) = (1 - ex)(1 - \exp[-R_0 R(x, \infty)]), \tag{4.29}$$

where R_0 is the basic reproduction number. $R(x, \infty)$ gives the respective fractions of four different types of individual depending on whether they are vaccinated or non-vaccinated and healthy or infected, as summarized in Table 4.4.

4.2.3.2 Payoff Structure

Again, an epidemic season continues until all infected individuals recover, meaning that the number of infected individuals is zero. As discussed in Sect. 4.1., we put the individual cost burden to support a subsidy system aside. If non-vaccinated individuals are infected, they incur an infection cost of 1. By contrast, non-vaccinated individuals who fortunately remain healthy can avoid the cost burden. Moreover, individuals who unfortunately are infected despite committing vaccination must pay the cost $C_r + 1$. Consequently, the payoff of each individual at the end of an epidemic season depends upon their final state. Table 4.5 summarizes the payoff whether committing to a provision (either vaccination or defense against contagion) or not and whether remaining healthy or becoming infected.

Table 4.4 Fractions of four types of individual using the effectiveness model without subsidy policy

Strategy/state	Healthy	Infected
Vaccinated	$x(e + (1 - e) \exp[-R_0R(x, \infty)])$	$x(1 - e)(1 - \exp[-R_0R(x, \infty)])$
Non-vaccinated	$(1 - x) \exp[-R_0R(x, \infty)]$	$(1 - x)(1 - \exp[-R_0R(x, \infty)])$

Table 4.5 Payoff structure determined at the end of an epidemic season

Strategy/state	Healthy	Infected
Vaccinated	$-C_r$	$-C_r - 1$
Non-vaccinated	0	-1

4.2.3.3 Subsidy Policies

As depicted in Sect. 4.2.1, we presume four different subsidy policies; Models A-1, A-2, A-3, and B. In the former three models, the limited subsidy is randomly distributed after each individual decides whether to vaccinate (see Fig. 4.18). In Model A-2, the defectors who decided not to vaccinate randomly receive the subsidy and vaccinate without vaccination cost. By contrast, in Model A-3, the cooperators who decide to vaccinate with their own expense can cover the vaccination cost by a subsidy. In Model A-1, the subsidy is randomly distributed to the population irrespective of whether individuals are defectors or cooperators. In these frameworks, a subsidized individual definitely vaccinates without a vaccination cost irrespective of his decision because he is given a free ticket. Hence, the subsidy refunds the voluntary vaccination cost for a cooperator, and a free ticket literally works for a defector. In the discount policy, Model B, all vaccinators can receive a certain amount of a subsidy, which reduces the vaccination cost. One point to note is that the acting discount rate by the subsidy depends on the vaccination coverage at a certain time-step.

Model A-1

In this scenario, the total subsidy is distributed to a certain fraction of individuals. Let this fraction be σ'. In Model A-1, $\sigma' = SB/C_r$ indicates that σ' happens to be consistent with σ if one refers to Eq. (4.27); $\sigma = SB/C_r$. Let the cooperation (defection) rate be f_C ($f_D = 1 - f_C$). All subsidized cooperators $\sigma' f_C$ and all subsidized defectors $\sigma'(1 - f_C)$ take vaccination without personal cost. Therefore, the vaccination coverage is $x = f_C + \sigma'(1 - f_C)$. We can find the respective fractions of eight different types of individual depending on whether they are vaccinated, non-vaccinated, or subsidized and whether they are healthy or infected, as summarized in Table 4.6. We also can present the payoff structure in Table 4.7 instead of Table 4.5.

Table 4.6 Fractions of eight types of individual using effectiveness model A-1

		Healthy	Infected
C	Vaccinated	$(1-\sigma^{'})f_C(e+(1-e)\exp[-R_0R(x,\infty)])$	$(1-\sigma^{'})f_C(1-e)(1-\exp[-R_0R(x,\infty)])$
	Subsidized	$\sigma' f_C(e+(1-e)\exp[-R_0R(x,\infty)])$	$\sigma' f_C(1-e)(1-\exp[-R_0R(x,\infty)])$
D	Non-vaccinated	$(1-\sigma^{'})(1-f_C)\exp[-R_0R(x,\infty)]$	$(1-\sigma^{'})(1-f_C)(1-\exp[-R_0R(x,\infty)])$
	Subsidized	$\sigma^{'}(1-f_C)(e+(1-e)\exp[-R_0R(x,\infty)])$	$\sigma^{'}(1-f_C)(1-e)(1-\exp[-R_0R(x,\infty)])$

Table 4.7 Payoff structure presuming Model A-1

		Healthy	Infected
C	Vaccinated	$-C_r$	$-C_r-1$
	Subsidized	0	-1
D	Non-vaccinated	0	-1
	Subsidized	0	-1

Subsequently, we can evaluate the expected payoffs in the form of the average social payoff $\langle\pi\rangle$, the average corporative payoff $\langle\pi_C\rangle$, and the average defective payoff $\langle\pi_D\rangle$ for the imperfect vaccination:

$$\begin{aligned}\langle\pi\rangle = \; & -C_r(1-\sigma')f_C(e+(1-e)\exp[-R_0R(x,\infty)]) \\ & -(C_r+1)(1-\sigma')f_C(1-e)(1-\exp[-R_0R(x,\infty)]) \\ & -\sigma' f_C(1-e)(1-\exp[-R_0R(x,\infty)]) - (1-\sigma')(1-f_C)(1-\exp[-R_0R(x,\infty)]) \\ & -\sigma'(1-f_C)(1-e)(1-\exp[-R_0R(x,\infty)]) - SB,\end{aligned} \tag{4.30}$$

$$\begin{aligned}\langle\pi_C\rangle = \; & -C_r(1-\sigma')(e+(1-e)\exp[-R_0R(x,\infty)]) \\ & -(C_r+1)(1-\sigma')(1-e)(1-\exp[-R_0R(x,\infty)]) \\ & -\sigma'(1-e)(1-\exp[-R_0R(x,\infty)]),\end{aligned} \tag{4.31}$$

$$\begin{aligned}\langle\pi_D\rangle = \; & -(1-\sigma')(1-\exp[-R_0R(x,\infty)]) \\ & -\sigma'(1-e)(1-\exp[-R_0R(x,\infty)]).\end{aligned} \tag{4.32}$$

It is worth noting that $\langle\pi\rangle$ is not consistent with the fraction weighted sum of $\langle\pi_C\rangle$ and $\langle\pi_D\rangle$, because $\langle\pi\rangle$ accounts $-SB$. As mentioned in Sect. 4.2.1, the total social payoff, *TSP,* which is consistent with $\langle\pi\rangle$ must consider the tax burden to support the subsidy. Unlike Eq. (4.27), Eq. (4.30) explicitly contains the tax term $-SB$, which is attributed to the fact that we did not account for the cost burden at an individual level, as we assumed in Sect. 4.2.1.

Model A-2

In this scenario, the total amount of a subsidy is distributed to a certain fraction of defectors. Let this fraction be $\sigma^{'}$. In Model A-2, note that $\sigma^{'} = SB/C_r(1-f_C)$. All subsidized defectors $\sigma^{'}(1-f_C)$ take vaccination without personal cost. Therefore, the vaccination coverage is $x = f_C + \sigma^{'}(1-f_C)$. We can obtain the respective fractions of

Table 4.8 Fractions of six types of individual using the effectiveness model A-2

		Healthy	Infected
C	Vaccinated	$f_C(e + (1 - e) \exp[-R_0R(x, \infty)])$	$f_C(1 - e)(1 - \exp[-R_0R(x, \infty)])$
D	Non-vaccinated	$(1 - \sigma')(1 - f_C) \exp[-R_0R(x, \infty)]$	$(1 - \sigma')(1 - f_C)(1 - \exp[-R_0R(x, \infty)])$
	Subsidized	$\sigma'(1 - f_C)(e + (1 - e) \exp[-R_0R(x, \infty)])$	$\sigma'(1 - f_C)(1 - e)(1 - \exp[-R_0R(x, \infty)])$

Table 4.9 Payoff structure with presuming Model A-2

		Healthy	Infected
C	Vaccinated	$-C_r$	$-C_r - 1$
D	Non-vaccinated	0	-1
	Subsidized	0	-1

Table 4.10 Fractions of six types of individual using effectiveness model A-3

		Healthy	Infected
C	Vaccinated	$(1 - \sigma')f_C(e + (1 - e) \exp[-R_0R(x, \infty)])$	$(1 - \sigma')f_C(1 - e)(1 - \exp[-R_0R(x, \infty)])$
	Subsidized	$\sigma' f_C(e + (1 - e) \exp[-R_0R(x, \infty)])$	$\sigma' f_C(1 - e)(1 - \exp[-R_0R(x, \infty)])$
D	Non-vaccinated	$(1 - f_C) \exp[-R_0R(x, \infty)]$	$(1 - f_C)(1 - \exp[-R_0R(x, \infty)])$

six different types of individual depending on whether they are vaccinated, non-vaccinated, or subsidized and whether they are healthy or infected, as summarized in Table 4.8. We also can present the payoff structure in Table 4.9.

We can evaluate the expected payoffs in the form of the average social payoff $\langle \pi \rangle$, the average cooperative payoff $\langle \pi_C \rangle$, and the average defective payoff $\langle \pi_D \rangle$ for imperfect vaccination as

$$\begin{aligned} \langle \pi \rangle = \; & -C_r f_C(e + (1 - e)\exp[-R_0R(x, \infty)]) \\ & -(C_r + 1)f_C(1 - e)(1 - \exp[-R_0R(x, \infty)]) \\ & -(1 - \sigma')(1 - f_C)(1 - \exp[-R_0R(x, \infty)]) \\ & -\sigma'(1 - f_C)(1 - e)(1 - \exp[-R_0R(x, \infty)]) \\ & -SB, \end{aligned} \tag{4.33}$$

$$\begin{aligned} \langle \pi_C \rangle = & -C_r(e + (1 - e)\exp[-R_0R(x, \infty)]) \\ & -(C_r + 1)(1 - e)(1 - \exp[-R_0R(x, \infty)]), \end{aligned} \tag{4.34}$$

$$\begin{aligned} \langle \pi_D \rangle = & -(1 - \sigma')(1 - \exp[-R_0R(x, \infty)]) \\ & -\sigma'(1 - e)(1 - \exp[-R_0R(x, \infty)]). \end{aligned} \tag{4.35}$$

Model A-3

In this scenario, the total subsidy amount is distributed to a certain fraction of cooperators. Let this fraction be σ'. In Model A-3, note $\sigma' = SB/C_r f_C$. All subsidized

Table 4.11 Payoff structure presuming Model A-3

		Healthy	Infected
C	Vaccinated	$-C_r$	$-C_r-1$
	Subsidized	0	-1
D	Non-vaccinated	0	-1

cooperators $\sigma' f_C$ take vaccination without personal cost. We can obtain the fractions of six different types of individual depending on whether they are vaccinated, non-vaccinated, or subsidized and whether they are healthy or infected, as summarized in Table 4.10. We also can present the payoff structure in Table 4.11.

We can evaluate the expected payoffs in the form of the average social payoff $\langle \pi \rangle$, the average cooperative payoff $\langle \pi_C \rangle$, and the average defective payoff $\langle \pi_D \rangle$ for imperfect vaccination as

$$\begin{aligned}\langle \pi \rangle = \; & -C_r(1-\sigma')f_C(e+(1-e)\exp[-R_0R(x,\infty)]) \\ & -(C_r+1)(1-\sigma')f_C(1-e)(1-\exp[-R_0R(x,\infty)]) \\ & -\sigma' f_C(1-e)(1-\exp[-R_0R(x,\infty)]) \\ & -(1-f_C)(1-\exp[-R_0R(x,\infty)])-SB,\end{aligned} \tag{4.36}$$

$$\begin{aligned}\langle \pi_C \rangle = \; & -C_r(1-\sigma')(e+(1-e)\exp[-R_0R(x,\infty)]) \\ & -(C_r+1)(1-\sigma')(1-e)(1-\exp[-R_0R(x,\infty)]) \\ & -\sigma'(1-e)(1-\exp[-R_0R(x,\infty)]),\end{aligned} \tag{4.37}$$

$$\langle \pi_D \rangle = -(1-\exp[-R_0R(x,\infty)]). \tag{4.38}$$

Model B

Under the discount-subsidy policy, vaccination coverage is equal to the cooperation rate because the subsidy is distributed to all vaccinated individuals. Hence, the fractions of four types of individual are the same as in Table 4.4. The total amount of subsidy is equally distributed to all vaccinators, and a vaccinator can reduce the vaccination cost by SB/f_C. Thus, we obtain Table 4.12 for the modified payoff structure.

We can evaluate the expected payoffs in the form of the average social payoff $\langle \pi \rangle$, the average cooperative payoff $\langle \pi_C \rangle$, and the average defective payoff $\langle \pi_D \rangle$ for imperfect vaccination as

$$\begin{aligned}\langle \pi \rangle = \; & \left(-C_r+\frac{SB}{f_C}\right)f_C(e+(1-e)\exp[-R_0R(x,\infty)]) \\ & +\left(-C_r-1+\frac{SB}{f_C}\right)f_C(1-e)(1-\exp[-R_0R(x,\infty)]) \\ & -(1-f_CS)(1-\exp[-R_0R(x,\infty)])-SB,\end{aligned} \tag{4.39}$$

Table 4.12 Payoff structure under the cooperator-preferential subsidy policy

		Healthy	Infected
C	Subsidized	$-C_r + \frac{SB}{f_C}$	$-C_r - 1 + \frac{SB}{f_C}$
D	Non-vaccinated	0	-1

$$\langle \pi_C \rangle = \left(-C_r + \frac{SB}{f_C}\right)(e + (1-e)\exp[-R_0 R(x,\infty)]) + \left(-C_r - 1 + \frac{SB}{f_C}\right)(1-e)(1-\exp[-R_0 R(x,\infty)]), \tag{4.40}$$

$$\langle \pi_D \rangle = -(1-\exp[-R_0 R(x,\infty)]). \tag{4.41}$$

4.2.3.4 Strategy Adaptation

Following Sect. 4.1.4, we specify how IB-RA and SB-RA are embedded in our theoretical framework.

IB-RA

We refer to Eq. (4.14). In the present framework, there are eight possible classes of individual state in relation to the cost burden: (i) a healthy defector (HD), who pays nothing, (ii) an infected defector (ID), who pays -1, (iii) an infected cooperator (IC), who pays $-C_r - 1$, (iv) a healthy cooperator (HC), who pays $-C_r$, (v) and (vi) a healthy subsidized defector and cooperator (HSD, HSC), respectively, who pay nothing due to a subsidy covering vaccination cost, and (vii) and (viii) an infected subsidized defector and cooperator (ISD, ISC, respectively) who pay -1 due to infection but pay nothing due to subsidy covering the vaccination cost. Each individual has two strategies: vaccination (V) (i.e., cooperation, C) and non-vaccination (NV) (i.e., defection, D). Thus, the transition probability that affects the time transition of f_C, which should be considered in the IB-RA rule, is covered by one of the following 32 cases:

$$P(HC \leftarrow HD) = P(HC \leftarrow HSD) = \frac{1}{1+\exp[-(0-(-C_r))/\kappa]}, \tag{4.42a}$$

$$P(HC \leftarrow ID) = P(HC \leftarrow ISD) = \frac{1}{1+\exp[-(-1-(-C_r))/\kappa]}, \tag{4.42b}$$

$$P(IC \leftarrow HD) = P(IC \leftarrow HSD) = \frac{1}{1+\exp[-(0-(-C_r-1))/\kappa]}, \tag{4.42c}$$

$$P(IC \leftarrow ID) = P(IC \leftarrow ISD) = \frac{1}{1+\exp[-(-1-(-C_r-1))/\kappa]}, \tag{4.42d}$$

$$P(HCS \leftarrow HD) = P(HCS \leftarrow HSD) = \frac{1}{1+\exp[-(0-0)/\kappa]}, \tag{4.42e}$$

$$P(HCS \leftarrow ID) = P(HCS \leftarrow ISD) = \frac{1}{1+\exp[-(-1-0)/\kappa]}, \tag{4.42f}$$

$$P(ICS \leftarrow HD) = P(ICS \leftarrow HSD) = \frac{1}{1+\exp[-(0-(-1))/\kappa]}, \tag{4.42g}$$

$$P(ICS \leftarrow ID) = P(ICS \leftarrow ISD) = \frac{1}{1+\exp[-(-1-(-1))/\kappa]}, \tag{4.42h}$$

$$P(HD \leftarrow HC) = P(HSD \leftarrow HC) = \frac{1}{1+\exp[-(-C_r-0)/\kappa]}, \tag{4.42i}$$

$$P(HD \leftarrow IC) = P(HSD \leftarrow IC) = \frac{1}{1+\exp[-((-C_r-1)-0)/\kappa]}, \tag{4.42j}$$

$$P(HD \leftarrow HCS) = P(HSD \leftarrow HCS) = \frac{1}{1+\exp[-(0-0)/\kappa]}, \tag{4.42k}$$

$$P(HD \leftarrow ICS) = P(HSD \leftarrow ICS) = \frac{1}{1+\exp[-((-1)-0)/\kappa]}, \tag{4.42l}$$

$$P(ID \leftarrow HC) = P(ISD \leftarrow HC) = \frac{1}{1+\exp[-(-C_r-(-1))/\kappa]}, \tag{4.42m}$$

$$P(ID \leftarrow IC) = P(ISD \leftarrow IC) = \frac{1}{1+\exp[-((-C_r-1)-(-1))/\kappa]}, \tag{4.42n}$$

$$P(ID \leftarrow HCS) = P(ISD \leftarrow HCS) = \frac{1}{1+\exp[-(0-(-1))/\kappa]}, \tag{4.42o}$$

$$P(ID \leftarrow ICS) = P(ISD \leftarrow ICS) = \frac{1}{1+\exp[-((-1)-(-1))/\kappa]}. \tag{4.42p}$$

When Model B is presumed, only the following probabilities are possible;

$$P(HSC \leftarrow HD) = \frac{1}{1+\exp[-(0-(-C_r+SB/f_C))/\kappa]}, \tag{4.43a}$$

$$P(HSC \leftarrow ID) = \frac{1}{1+\exp[-(-1-(-C_r+SB/f_C))/\kappa]}, \tag{4.43b}$$

$$P(ISC \leftarrow HD) = \frac{1}{1+\exp[-(0-(-C_r-1+SB/f_C))/\kappa]}, \tag{4.43c}$$

$$P(ISC \leftarrow ID) = \frac{1}{1+\exp[-(-1-(-C_r-1+SB/f_C))/\kappa]}, \tag{4.43d}$$

$$P(HD \leftarrow HSC) = \frac{1}{1+\exp[-(-C_r+SB/f_C-0)/\kappa]}, \tag{4.43e}$$

$$P(HD \leftarrow ISC) = \frac{1}{1+\exp[-(-C_r-1+SB/f_C-0)/\kappa]}, \tag{4.43f}$$

$$P(ID \leftarrow HSC) = \frac{1}{1+\exp[-(-C_r+SB/f_C-(-1))/\kappa]}, \tag{4.43g}$$

$$P(ID \leftarrow ISC) = \frac{1}{1+\exp[-(-C_r-1+SB/f_C-(-1))/\kappa]}. \tag{4.43h}$$

SB-RA

We refer to Eq. (4.15). The transition probabilities that we must consider are as follows:

$$P(HC \leftarrow D) = \frac{1}{1+\exp[-(\langle\pi_D\rangle-(-C_r))/\kappa]}, \tag{4.44a}$$

$$P(IC \leftarrow D) = \frac{1}{1+\exp[-(\langle\pi_D\rangle-(-C_r-1))/\kappa]}, \tag{4.44b}$$

$$P(HSC \leftarrow D) = \frac{1}{1+\exp[-(\langle\pi_D\rangle-0)/\kappa]}, \tag{4.44c}$$

$$P(ISC \leftarrow D) = \frac{1}{1+\exp[-(\langle\pi_D\rangle-(-1))/\kappa]}. \tag{4.44d}$$

$$P(HD \leftarrow C) = \frac{1}{1+\exp[-(\langle \pi_C \rangle - 0)/\kappa]}, \tag{4.44e}$$

$$P(ID \leftarrow C) = \frac{1}{1+\exp[-(\langle \pi_C \rangle - (-1))/\kappa]}, \tag{4.44f}$$

$$P(HSD \leftarrow C) = \frac{1}{1+\exp[-(\langle \pi_C \rangle - 0)/\kappa]}, \tag{4.44g}$$

$$P(ISD \leftarrow C) = \frac{1}{1+\exp[-(\langle \pi_C \rangle - (-1))/\kappa]}. \tag{4.44h}$$

For Model B, we should consider

$$P(HSC \leftarrow D) = \frac{1}{1+\exp[-(\langle \pi_D \rangle - (-C_r + SB/f_C))/\kappa]}, \tag{4.45a}$$

$$P(ISC \leftarrow D) = \frac{1}{1+\exp[-(\langle \pi_D \rangle - (-C_r - 1 + SB/f_C))/\kappa]}, \tag{4.45b}$$

$$P(HD \leftarrow C) = \frac{1}{1+\exp[-(\langle \pi_C \rangle - 0)/\kappa]}, \tag{4.45c}$$

$$P(ID \leftarrow C) = \frac{1}{1+\exp[-(\langle \pi_C \rangle - (-1))/\kappa]}. \tag{4.45d}$$

4.2.3.5 Global Time Evolution

Strategy updating takes place after each epidemic season as defined above (see Fig. 4.18). This inevitably brings increasing or decreasing x via increasing or decreasing f_C. Since there are four subsidy models and two strategy-updating rules, we can deduce eight different dynamical equations for predicting global FES as below.

Model A-1 + IB-RA

$$
\begin{aligned}
\frac{df_C}{dt} = {} & f_C(1-f_C)(1-\sigma')^2(e+(1-e)\exp[-R_0R(x,\infty)])\exp[-R_0R(x,\infty)] \\
& (P(HD \leftarrow HC) - P(HC \leftarrow HD)) \\
& + f_C(1-f_C)(1-\sigma')^2(e+(1-e)\exp[-R_0R(x,\infty)])(1-\exp[-R_0R(x,\infty)]) \\
& (P(ID \leftarrow HC) - P(HC \leftarrow ID)) \\
& + f_C(1-f_C)\sigma'(1-\sigma')(e+(1-e)\exp[-R_0R(x,\infty)])^2 \\
& (P(HSD \leftarrow HC) - P(HC \leftarrow HSD)) \\
& + f_C(1-f_C)\sigma'(1-\sigma')(1-e)(e+(1-e)\exp[-R_0R(x,\infty)]) \\
& (1-\exp[-R_0R(x,\infty)])(P(ISD \leftarrow HC) - P(HC \leftarrow ISD)) \\
& + f_C(1-f_C)(1-\sigma')^2(1-e)(1-\exp[-R_0R(x,\infty)])\exp[-R_0R(x,\infty)] \\
& (P(HD \leftarrow IC) - P(IC \leftarrow HD)) \\
& + f_C(1-f_C)(1-\sigma')^2(1-e)(1-\exp[-R_0R(x,\infty)])^2 \\
& (P(ID \leftarrow IC) - P(IC \leftarrow ID)) \\
& + f_C(1-f_C)\sigma'(1-\sigma')(1-e)(e+(1-e)\exp[-R_0R(x,\infty)]) \\
& (1-\exp[-R_0R(x,\infty)])(P(HSD \leftarrow IC) - P(IC \leftarrow HSD)) \\
& + f_C(1-f_C)\sigma'(1-\sigma')(1-e)^2(1-\exp[-R_0R(x,\infty)])^2 \\
& (P(ISD \leftarrow IC) - P(IC \leftarrow ISD)) \\
& + f_C(1-f_C)\sigma'(1-\sigma')(e+(1-e)\exp[-R_0R(x,\infty)])\exp[-R_0R(x,\infty)] \\
& (P(HD \leftarrow HSC) - P(HSC \leftarrow HD)) \\
& + f_C(1-f_C)\sigma'(1-\sigma')(e+(1-e)\exp[-R_0R(x,\infty)])(1-\exp[-R_0R(x,\infty)]) \\
& (P(ID \leftarrow HSC) - P(HSC \leftarrow ID)) \\
& + f_C(1-f_C)\sigma'^2(e+(1-e)\exp[-R_0R(x,\infty)])^2 \\
& (P(HSD \leftarrow HSC) - P(HCS \leftarrow HSD)) \\
& + f_C(1-f_C)\sigma'^2(1-e)(e+(1-e)\exp[-R_0R(x,\infty)])(1-\exp[-R_0R(x,\infty)]) \\
& (P(IDS \leftarrow HSC) - P(HCS \leftarrow ISD)) \\
& + f_C(1-f_C)\sigma'(1-\sigma')(1-e)(1-\exp[-R_0R(x,\infty)])\exp[-R_0R(x,\infty)] \\
& (P(HD \leftarrow ISC) - P(ISC \leftarrow HD)) \\
& + f_C(1-f_C)\sigma'(1-\sigma')(1-e)(1-\exp[-R_0R(x,\infty)])^2 \\
& (P(ID \leftarrow ISC) - P(ISC \leftarrow ID)) \\
& + f_C(1-f_C)\sigma'^2(1-e)(e+(1-e)\exp[-R_0R(x,\infty)])(1-\exp[-R_0R(x,\infty)]) \\
& (P(HDS \leftarrow ISC) - P(ICS \leftarrow HSD)) \\
& + f_C(1-f_C)\sigma'^2(1-e)^2(1-\exp[-R_0R(x,\infty)])^2 \\
& (P(ISD \leftarrow ISC) - P(ISC \leftarrow ISD)).
\end{aligned}
\tag{4.46}
$$

Model A-1 + SB-RA

$$\begin{aligned}\frac{df_C}{dt} = &-f_C(1-f_C)(1-\sigma')(e+(1-e)\exp[-R_0R(x,\infty)])P(HC \leftarrow D)\\ &-f_C(1-f_C)(1-\sigma')(1-e)(1-\exp[-R_0R(x,\infty)])P(IC \leftarrow D)\\ &-f_C(1-f_C)\sigma'(1-e)\exp[-R_0R(x,\infty)]P(HSC \leftarrow D)\\ &-f_C(1-f_C)\sigma'(1-e)(1-\exp[-R_0R(x,\infty)])P(ISC \leftarrow D)\\ &+f_C(1-f_C)(1-\sigma')\exp[-R_0R(x,\infty)]P(HD \leftarrow C)\\ &+f_C(1-f_C)(1-\sigma')(1-\exp[-R_0R(x,\infty)])P(ID \leftarrow C)\\ &+f_C(1-f_C)\sigma'(e+(1-e)\exp[-R_0R(x,\infty)])P(HSD \leftarrow C)\\ &+f_C(1-f_C)\sigma'(1-e)(1-\exp[-R_0R(x,\infty)])P(ISD \leftarrow C).\end{aligned} \tag{4.47}$$

Model A-2 + IB-RA

$$\begin{aligned}\frac{df_C}{dt} = &f_C(1-f_C)(1-\sigma')(e+(1-e)\exp[-R_0R(x,\infty)])\exp[-R_0R(x,\infty)]\\ &(P(HD \leftarrow HC) - P(HC \leftarrow HD))\\ &+f_C(1-f_C)(1-\sigma')(e+(1-e)\exp[-R_0R(x,\infty)])(1-\exp[-R_0R(x,\infty)])\\ &(P(ID \leftarrow HC) - P(HC \leftarrow ID))\\ &+f_C(1-f_C)\sigma'(e+(1-e)\exp[-R_0R(x,\infty)])^2\\ &(P(HSD \leftarrow HC) - P(HC \leftarrow HSD))\\ &+f_C(1-f_C)\sigma'(e+(1-e)\exp[-R_0R(x,\infty)])(1-e)(1-\exp[-R_0R(x,\infty)])\\ &(P(ISD \leftarrow HC) - P(HC \leftarrow ISD))\\ &+f_C(1-f_C)(1-\sigma')(1-e)(1-\exp[-R_0R(x,\infty)])\exp[-R_0R(x,\infty)]\\ &(P(HD \leftarrow IC) - P(IC \leftarrow HD))\\ &+f_C(1-f_C)(1-\sigma')(1-e)(1-\exp[-R_0R(x,\infty)])(1-\exp[-R_0R(x,\infty)])\\ &(P(ID \leftarrow IC) - P(IC \leftarrow ID))\\ &+f_C(1-f_C)\sigma'(1-e)(1-\exp[-R_0R(x,\infty)])(e+(1-e)\exp[-R_0R(x,\infty)])\\ &(P(ID \leftarrow IC) - P(IC \leftarrow ID))\\ &+f_C(1-f_C)\sigma'(1-e)^2(1-\exp[-R_0R(x,\infty)])^2(P(ID \leftarrow IC) - P(IC \leftarrow ID)).\end{aligned} \tag{4.48}$$

Model A-2 + SB-RA

$$\begin{aligned}\frac{df_C}{dt} = &-f_C(1-f_C)(e+(1-e)\exp[-R_0R(x,\infty)])P(HC \leftarrow D)\\ &-f_C(1-f_C)(1-e)(1-\exp[-R_0R(x,\infty)])P(IC \leftarrow D)\\ &+f_C(1-f_C)(1-\sigma')\exp[-R_0R(x,\infty)]P(HD \leftarrow C)\\ &+f_C(1-f_C)(1-\sigma')(1-\exp[-R_0R(x,\infty)])P(ID \leftarrow C)\\ &+f_C(1-f_C)\sigma'(e+(1-e)\exp[-R_0R(x,\infty)])P(HSD \leftarrow C)\\ &+f_C(1-f_C)\sigma'(1-e)(1-\exp[-R_0R(x,\infty)])P(ISD \leftarrow C).\end{aligned} \tag{4.49}$$

Model A-3 + IB-RA

$$\begin{aligned}\frac{df_C}{dt} = &f_C(1-f_C)(1-\sigma')(e+(1-e)\exp[-R_0R(x,\infty)])\exp[-R_0R(x,\infty)]\\ &(P(HD \leftarrow HC) - P(HC \leftarrow HD))\\ &+f_C(1-f_C)(1-\sigma')(e+(1-e)\exp[-R_0R(x,\infty)])(1-\exp[-R_0R(x,\infty)])\\ &(P(ID \leftarrow HC) - P(HC \leftarrow ID))\\ &+f_C(1-f_C)(1-\sigma')(1-e)(1-\exp[-R_0R(x,\infty)])\exp[-R_0R(x,\infty)]\\ &(P(HD \leftarrow IC) - P(IC \leftarrow HD))\\ &+f_C(1-f_C)(1-\sigma')(1-e)(1-\exp[-R_0R(x,\infty)])(1-\exp[-R_0R(x,\infty)])\\ &(P(ID \leftarrow IC) - P(IC \leftarrow ID))\\ &+f_C(1-f_C)\sigma'(e+(1-e)\exp[-R_0R(x,\infty)])\exp[-R_0R(x,\infty)]\\ &(P(HD \leftarrow HSC) - P(HSC \leftarrow HD))\\ &+f_C(1-f_C)\sigma'(e+(1-e)\exp[-R_0R(x,\infty)])(1-\exp[-R_0R(x,\infty)])\\ &(P(ID \leftarrow HSC) - P(HSC \leftarrow ID))\\ &+f_C(1-f_C)\sigma'(1-e)(1-\exp[-R_0R(x,\infty)])\exp[-R_0R(x,\infty)]\\ &(P(HD \leftarrow ISC) - P(ISC \leftarrow HD))\\ &+f_C(1-f_C)\sigma'(1-e)(1-\exp[-R_0R(x,\infty)])(1-\exp[-R_0R(x,\infty)])\\ &(P(ID \leftarrow ISC) - P(ISC \leftarrow ID)).\end{aligned} \tag{4.50}$$

Model A-3 + SB-RA

$$\begin{aligned}\frac{df_C}{dt} = &-f_C(1-f_C)(1-\sigma')(e+(1-e)\exp[-R_0R(x,\infty)])P(HC \leftarrow D)\\ &-f_C(1-f_C)(1-\sigma')(1-e)(1-\exp[-R_0R(x,\infty)])P(IC \leftarrow D)\\ &-f_C(1-f_C)\sigma'(e+(1-e)\exp[-R_0R(x,\infty)])P(HSC \leftarrow D)\\ &-f_C(1-f_C)\sigma'(1-e)(1-\exp[-R_0R(x,\infty)])P(ISC \leftarrow D)\\ &+f_C(1-f_C)\exp[-R_0R(x,\infty)]P(HD \leftarrow C)\\ &+f_C(1-f_C)(1-\exp[-R_0R(x,\infty)])P(ID \leftarrow C).\end{aligned} \tag{4.51}$$

Model B + IB-RA

$$\begin{aligned}\frac{df_C}{dt} = &f_C(1-f_C)(e+(1-e)\exp[-R_0R(x,\infty)])\exp[-R_0R(x,\infty)]\\ &(P(HD \leftarrow HSC) - P(HSC \leftarrow HD))\\ &+f_C(1-f_C)(e+(1-e)\exp[-R_0R(x,\infty)])(1-\exp[-R_0R(x,\infty)])\\ &(P(ID \leftarrow HSC) - P(HSC \leftarrow ID))\\ &+f_C(1-f_C)(1-e)(1-\exp[-R_0R(x,\infty)])\exp[-R_0R(x,\infty)]\\ &(P(DH \leftarrow ISC) - P(ISC \leftarrow HD))\\ &+f_C(1-f_C)(1-e)(1-\exp[-R_0R(x,\infty)])^2\\ &(P(ID \leftarrow ISC) - P(ISC \leftarrow ID)).\end{aligned} \tag{4.52}$$

Model B + SB-RA

$$\begin{aligned}\frac{df_C}{dt} = &-f_C(1-f_C)(e+(1-e)\exp[-R_0R(x,\infty)])P(HSC \leftarrow D)\\ &-f_C(1-f_C)(1-e)(1-\exp[-R_0R(x,\infty)])P(ISC \leftarrow D)\\ &+f_C(1-f_C)\exp[-R_0R(x,\infty)]P(HD \leftarrow C)\\ &+f_C(1-f_C)(1-\exp[-R_0R(x,\infty)])P(ID \leftarrow VC).\end{aligned} \tag{4.53}$$

All dynamical equations above can be solved numerically. We introduce a so-called explicit scheme for the time-varying terms to obtain a numerical solution for vaccination coverage at equilibrium.

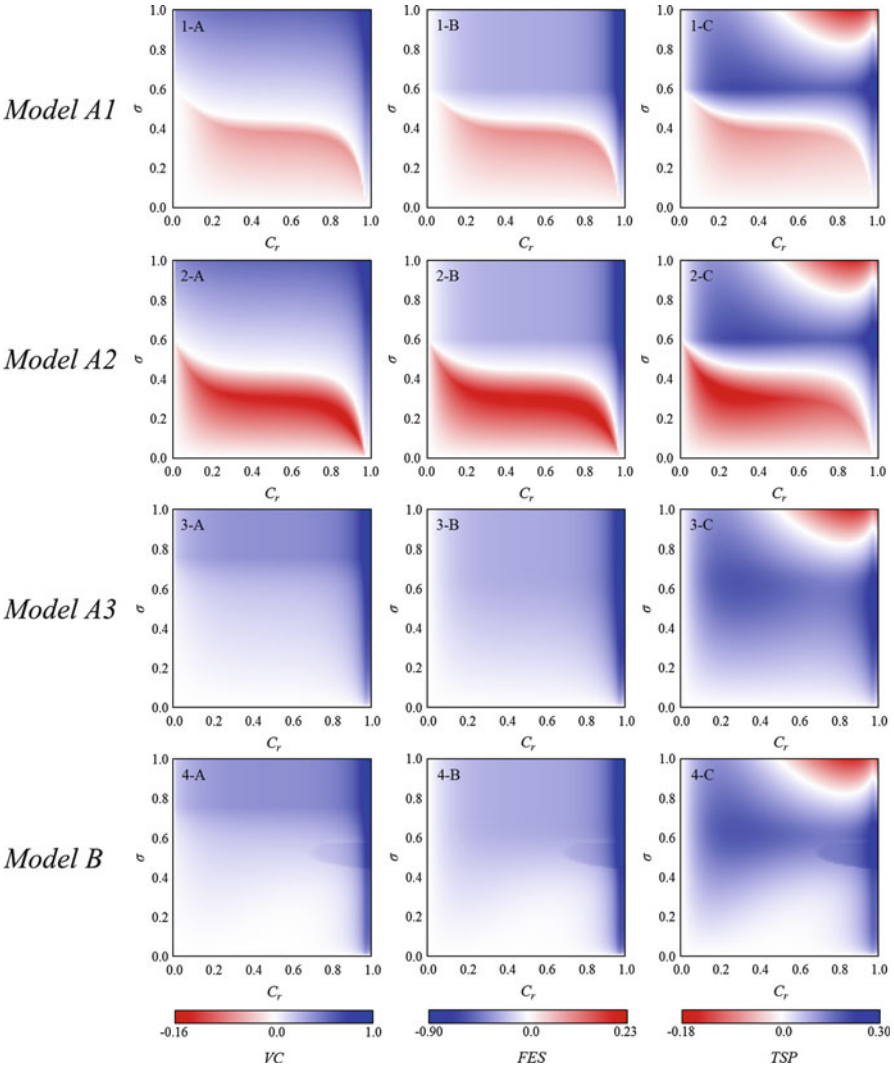

Fig. 4.29 Analytical result: Color indicates difference between subsidy ($\sigma > 0$) and non-subsidy ($\sigma = 0$) cases. Panel (*-A) Vaccination coverage (VC), (*-B) final epidemic size (FES), (*-C) total social payoff (TSP). Panel (1-*) Model A1, (2-*) Model A2, (3-*) Model A3, (4-*) Model B. $e = 1.0$ and IB-RA are presumed

4.2.3.6 Discussion

Figures 4.29, 4.30, 4.31, and 4.32 present our analytical results. Figures 4.29 and 4.30 provide the results under the four types of subsidy policies when respectively presuming $e = 1.0$ (perfect vaccination) and $e = 0.4$ (low reliable vaccination) using IB-RA as a strategy-updating rule. Figures 4.31 and 4.32 present the same presuming SB-RA.

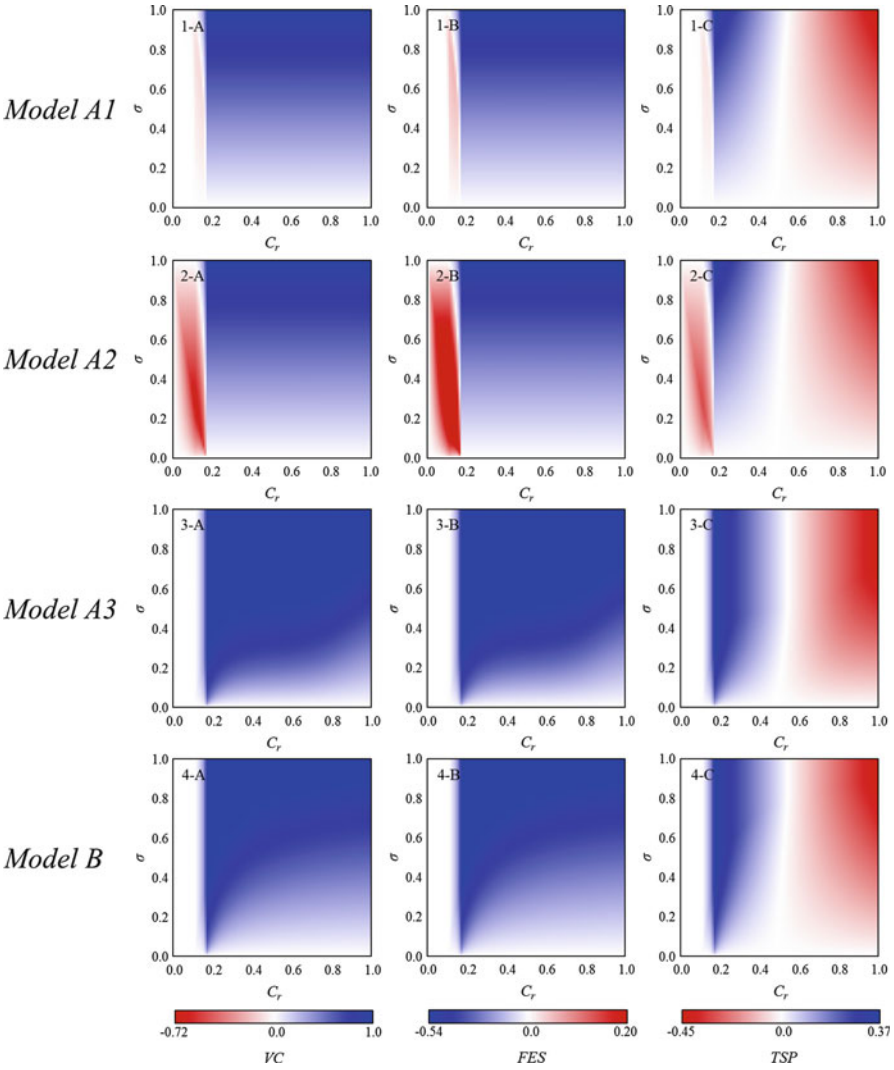

Fig. 4.30 Analytical result: Color indicates difference between subsidy ($\sigma > 0$) and non-subsidy ($\sigma = 0$) cases. Panel (*-A) Vaccination coverage (VC), (*-B) final epidemic size (FES), (*-C) total social payoff (TSP). Panel (1-*) Model A1, (2-*) Model A2, (3-*) Model A3, (4-*) Model B. $e = 0.4$ and IB-RA are presumed

Because of the mean-field approximation, panels (VC, FES and TSP) in the top row (assuming Model A1) in Fig. 4.29 should be compared with Fig. 4.24 presuming RRG. Qualitatively, both show the same tendency. Differences in detail level come from finite resolution and insufficient population, as well as insufficient average degree in the simulation, which seems to some extent inevitable. Thus, we conclude that our theoretical model well-reproduces the MAS-simulation result.

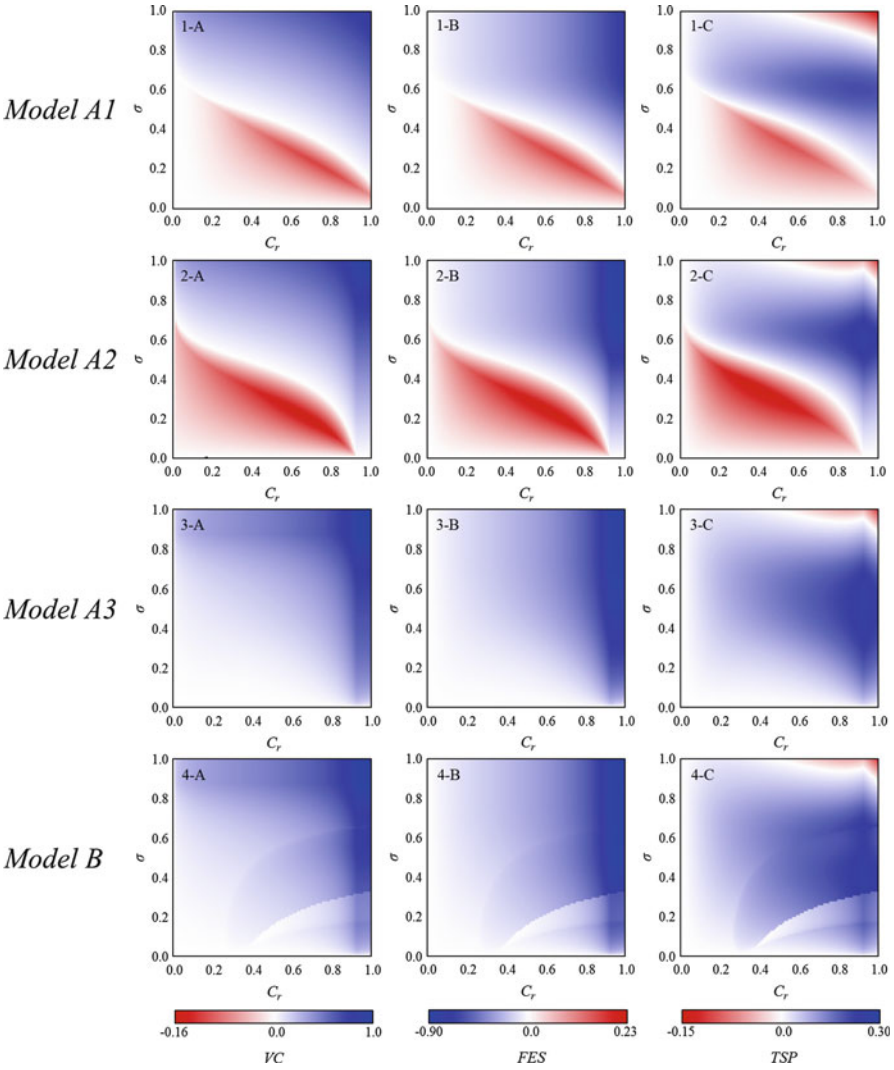

Fig. 4.31 Analytical result: Color indicates difference between subsidy ($\sigma > 0$) and non-subsidy ($\sigma = 0$) cases. Panel (*-A) Vaccination coverage (VC), (*-B) final epidemic size (FES), (*-C) total social payoff (TSP). Panel (1-*) Model A1, (2-*) Model A2, (3-*) Model A3, (4-*) Model B. $e = 1.0$ and SB-RA are presumed

Let us compare the subsidy systems assuming perfect vaccination ($e = 1.0$) and IB-RA in Fig. 4.29. Focusing on TSP under Models A-1 and A-2, the negative region exists in a relatively smaller subsidy size of σ(hereafter, the first negative region) and a larger subsidy size of σ, dovetailed with a higher vaccination cost (hereafter, second negative region). In the first negative region, a subsidy going to

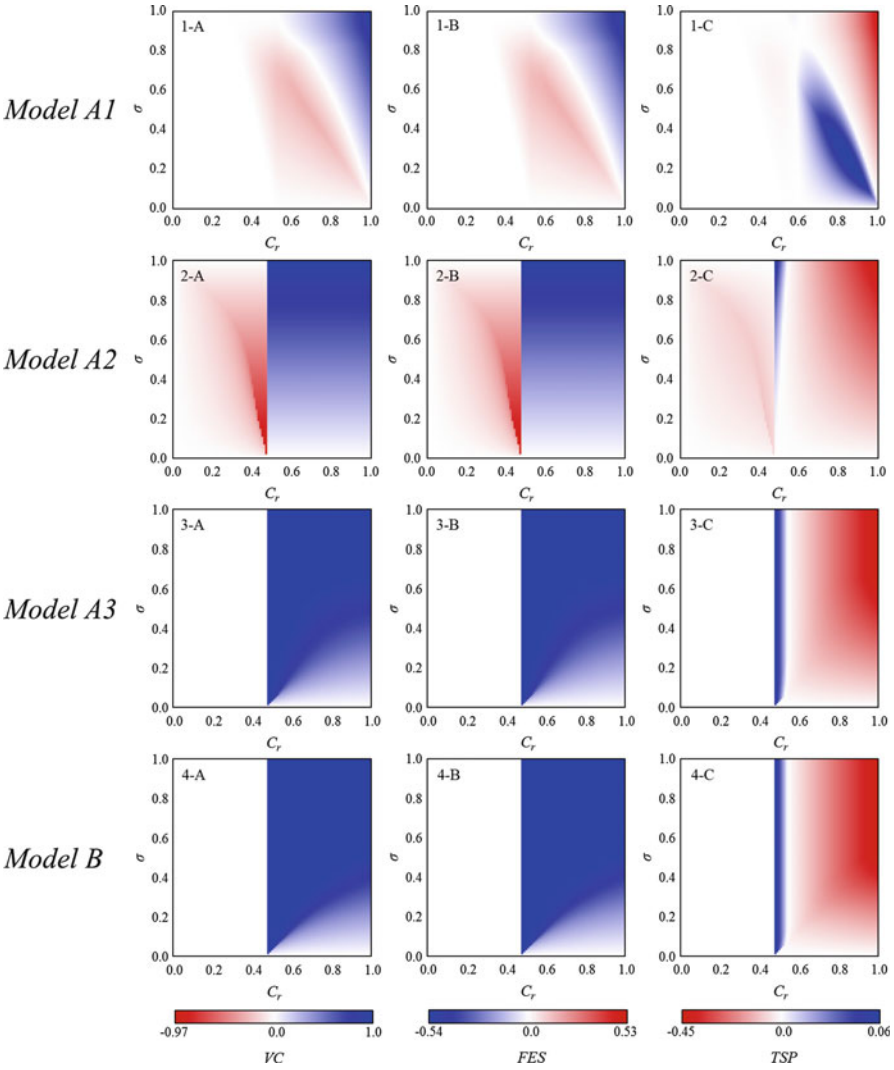

Fig. 4.32 Analytical result: Color indicates difference between subsidy ($\sigma > 0$) and non-subsidy ($\sigma = 0$) cases. Panel (*-A) Vaccination coverage (VC), (*-B) final epidemic size (FES), (*-C) total social payoff (TSP). Panel (1-*) Model A1, (2-*) Model A2, (3-*) Model A3, (4-*) Model B. $e = 0.4$ and SB-RA are presumed

non-vaccinators (i.e., defectors) counterproductively works to suppress the social cost because being a defector becomes cost-advantageous (since they may avoid infection by being given free-tickets), reducing the fraction of cooperators (i.e., vaccinators) as compared with the default case. As a consequence, the number of self-financed vaccinators comes down, worsening the social efficiency. On the other

hand, in the second negative region, due to a relatively higher vaccination cost compared to disease cost, some reasonable number of people getting infected is rather beneficial to the entire society, rather than spending too much on vaccination. Furthermore, although the general tendencies of Models A-1 and A-2 are the same, more deliberate comparison reveals that Model A-2 more badly works so that makes the first negative region more red than Model A-1 does. Namely, Model A-2 delivers all free-tickets to non-vaccinators, which consequently reduces self-financed vaccinators more significantly than does Model A-1. Therefore, one important social implication that can be noted is that a subsidy helping people who potentially aim to free-ride on the public good devastates social efficiently. A subsidy system should be based on the principle that "heaven helps those who help themselves". As we confirm latter in the discussion on Models A-3 and B, a subsidy system focused only on potential vaccinators more efficiently suppresses the total social cost vis-a-vis the default case.

Recalling what happens when only non-vaccinators are subsidized, let us move on to Models A-3 and B, where only vaccinators are subsidized. The first negative region does not occur under any settings, whether relying on global information (SB-RA) or not (IB-RA). As a whole, those two subsidy models outperform other models (Models A-1 and A-2). Hence, we would say that a subsidy policy focused only on potential vaccinators should be adopted. Again, this is because subsidies going to potential non-vaccinators eventually impede the increase of self-financed vaccinators due to misled people who aim either to free-ride or to be given a free ticket despite not cooperating.

Comparing Model A-3 with Model B, we find that they show analogous tendencies. This is consistent with what we observed in the MAS result (Figs. 4.21 and 4.22). However, on the whole. Model A-3 seems better than Model B in terms of social efficiency. Pairs of the broken-line boxes in Models A-3 and B prove this, where the region in Model A-3 looks bluer than that by Model B. One plausible cause for this tendency is that distributing free tickets only to limited eligible individuals drives people to vaccinate (increasing self-financed vaccinators) more significantly than offering a discount coupon to all eligible individuals, due to the non-linearity of the Fermi function considered when updating a strategy. This can explained in detail below.

Recall the two arguments (i.e., payoffs) in the Fermi function. Let us suppose a non-vaccinator (defector) copies V from a vaccinator (cooperator) who is given either a free-ticket in the case of Model A-3 or a discount coupon in the case if Model B, and compare those two models. Note that the state-transition probability of Model A-3 is larger than that of Model B, which is attributed to the reduction in price by a free-ticket or discount-coupon. Since the same σ is presumed for this comparison, the fraction (number) of vaccinators given free-tickets in Model A-3 is less than that given discount-coupons (that is consistent with the entire number of vaccinators) in Model B, which is considered in the final dynamical equations (Eq. (4.50) or Eq. (4.51) for Model A-3 and Eq. (4.52) or Eq. (4.53) for Model B). But, again,

because of non-linearity of the Fermi function (state-transition probability), the attractive force causing NV to become V in Model A-3 is greater than that in Model B.

Comparing Fig. 4.29 with Fig. 4.30, or Fig. 4.31 with Fig. 4.32, with decrease of vaccine reliability (decrease of e) the first negative region becomes smaller whereas the second negative region is larger. Interestingly, in the case assuming $e = 0.4$ and IB-RA, both regions in which introducing a subsidy is not justified are insensitive to σ. In fact, if C_r becomes more than 0.6, the subsidy obviously deteriorates the social efficiency regardless of the size of subsidy. For C_r below 0.2, this deterioration effect becomes more slight.

Figures 4.31 and 4.32, presuming SB-RA, present a quite different picture than Figs. 4.29 and 4.30, resulting from the difference in the strategy-update rules. Let us compare our respective TSPs with $e = 0.4$. The black and gray boxes indicate the first and second negative regions. As discussed above, the first negative region results from the situation whereby subsidizing non-vaccinators hampers the increase of self-financed vaccinators, while the second region is brought about by the fact that spending too much on vaccination becomes less beneficial on the whole than allowing a reasonable level of infectious individuals. Although the first negative regions (black boxes) are at comparable levels, the second negative region (gray box) of Fig. 4.32 is less than that of Fig. 4.30, only appearing at larger $\sigma$and larger C_r. Thus, sharing global information during strategy-updating events (SB-RA) helps to justify a subsidy system. Observing carefully, we can note that the range of vaccination costs justifying subsidies (colored blue) is insensitive to σ; however, although it clearly appears around $0.2 \leq C_r \leq 0.5$ in Fig. 4.30, it almost disappears in Fig. 4.32. If we make the same comparison for Model A-2 in Figs. 4.30 and 4.32 (see black and gray boxes in respective right panels), we find the same behavior. There is none of any range of vaccination cost justifying subsidy insensitive to σ in Model A-2 of Figs. 4.31 and 4.32. Moreover, remarkably, in the right-hand panel of Model A-2 in Fig. 4.32, there is almost no parameter region in which a subsidy is justified. Subsidizing only non-vaccinators (Model A-2) in the case presuming a strategy-updating rule relying on global information (SB-RA) and a unreliable vaccination ($e = 0.4$) is not justified at all.

4.2.4 Summary and Social Implications

In order to help society establish an effective subsidy policy to combat the spread of infectious disease and mitigate the risk of pandemics, this study proposed a comprehensive "vaccination game", wherein a subsidy system is considered in the context of both the dynamics of individual decision-making based on evolutionary game theory and the spread of disease using the SIR/V model through a social network with consideration of a subsidy system.

For our analysis, we performed not only multi-agent simulation (MAS) considering how the underlying topology of the social network affected equilibrium, but also a theoretical approach presuming a mean-field approximation. In particular, our analytic model deals with imperfectly working vaccination parameterized by "effectiveness", which does not always bring a perfect immunity by taking a vaccine.

We presume four types of subsidy systems depending on whether a free-ticket or a discount-coupon is given, as well as individual attributes, such as being a potential vaccinator or a defector trying to free-ride on herd immunity.

We mainly observed our results in terms of vaccination coverage (VC), final epidemic size (FES), and total social payoff (TSP) (or, looking negatively, total social cost), using these to indicate social efficiency.

First of all, we confirmed that our analytical approach is capable of reproducing the result obtained by the MAS approach.

Our result suggests that spending too little on subsidy or too much for a relatively higher vaccination cost results in an ironic situation where introducing a subsidy incurs a higher social cost than the default case. Little spending on the subsidy results in making self-financed vaccinators decrease as a fraction of society (hereafter; let us call this the "first regime"). Overspending on a subsidy when the vaccination cost is high brings the situation that rather some people being infected becomes rather socially efficient than too much vaccinators due to the relation of vaccination cost to infection cost; this devastates the social efficiency compared to the default case realizing (which we hereafter call the "second regime").

The MAS result shows that the underlying social network significantly influences equilibrium. In particular, a scale-free network rather than a lattice expands the parameter region in which a subsidy system deteriorates the social efficiency.

If a vaccine's reliability degrades (presuming low effectiveness), the parameter region in which a subsidy is counterproductive due to the second regime grows and becomes less sensitive to the subsidy size.

A subsidy that applies only to potential cooperators is quite important for the optimal social design of a subsidy system. Although a subsidy applying to people who have no intention of vaccinating unless given either a free-ticket or a discount-coupon might be thought efficient, or at least socially favored or accepted in the context of a high-welfare society, such a scheme could reduce the number of inherently cooperative vaccinators (self-financed vaccinators), owing to disregard for the principle of "heaven helping those who help themselves".

Although the difference between free-ticket policy and discount-coupon policy was observed to be small (so long as tickets were only given to potential vaccinators), this theoretical approach shows that the free-ticket slightly outperforms the discount-coupon policy. This is because a larger payoff difference is brought by a free-ticket instead of a discount-coupon, triggering an increase in self-financed vaccinators.

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