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Human disease-behavior interactions on complex networks models: incorporating evolutionary game into epidemiology

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Human Disease-behavior Interactions on Complex Networks Models: Incorporating Evolutionary Game into Epidemiology

WANG Zhen

A thesis submitted in partial fulfillment of the requirements for the degree of Doctor of Philosophy

Principal Supervisor: Prof. ZHOU Changsong

Hong Kong Baptist University

August 2014
Declaration

I hereby declare that this thesis represents my own work which has been done after registration for the degree of PhD at Hong Kong Baptist University, and has not been previously included in a thesis, dissertation submitted to this or other institution for a degree, diploma or other qualification.

Signature: ____________________________

Date: August 2014
Abstract

In the past decade, the study of disease dynamics on complex networks has attracted great attention from both theoretical and empirical viewpoints. Under such a framework, people try to predict the outbreak of disease and propose immunization mechanisms. However, this framework possesses a limitation, which makes it inconsistent with realistic cases. That is, this framework does not consider the impact of human behavior or decision-making progress on disease dynamic characters and prevention measures. To further resolve this problem, we in this thesis propose behavioral epidemiology based on game theory, which involves the interactions between disease dynamics and human behavior in complex networks. Motivated by realistic cases, we proceed with the research from theoretical models and consider the following aspects. We first re-construct a scheme of risk perception incorporating local and global information and show that this new evaluation scenario not only promotes vaccination uptake, but also eliminates the disease spreading. This interesting finding could be attributed to the positive feedback mechanism between vaccination uptake and disease spreading. Then, we introduce a self-protection measure, which, due to low cost, can only provide temporary protection. By simulations and analysis we show that this measure leads to multiple effects: contrary with cases of low (high) efficiency and cost of the self-protection measure, middle values drive more infection and larger cost, which is related to the loss of positive feedback between prevention measures and disease propagation. Subsequently, another scheme of adaptive protection is proposed, where a healthy agent can cut the connection with infected ones. We find that adaptive protection can effectively eradicate the disease and result in an optimal level of pruning infected links. Different from these proposals focusing on individual interest, we lastly study a subsidy policy from the viewpoint of population benefit. We find that disease can be well controlled with an increase of the vaccination level, while the total expense reduces. Taken together, these findings of the thesis further demonstrate that the interplay between disease dynamics and human behavior plays an important role in the control of diseases. The models presented in this thesis, especially combining with empirical data, may serve as a foundation for further investigation of the subject in the future.
Acknowledgements

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<th>Full Form</th>
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<tbody>
<tr>
<td>SIS</td>
<td>susceptible-infected-susceptible</td>
</tr>
<tr>
<td>SIR</td>
<td>susceptible-infected-recovered</td>
</tr>
<tr>
<td>HIV</td>
<td>human immunodeficiency virus</td>
</tr>
<tr>
<td>AIDS</td>
<td>acquired immune deficiency syndrome</td>
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<tr>
<td>SARS</td>
<td>severe acute respiratory syndrome</td>
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<tr>
<td>FMD</td>
<td>foot-and-mouth disease</td>
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<tr>
<td>NE</td>
<td>Nash Equilibrium</td>
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<tr>
<td>PD</td>
<td>prisoner’s dilemma</td>
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<tr>
<td>PGG</td>
<td>public goods game</td>
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<tr>
<td>SF network</td>
<td>scale-free network</td>
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<tr>
<td>ER network</td>
<td>Erdős-Rényi network</td>
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<tr>
<td>BA network</td>
<td>Barabási-Albert network</td>
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<td>SW network</td>
<td>small-world network</td>
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Chapter 1

Introduction

1.1 Overview

Human society has been attacked by various kinds of infectious disease. The early record can date back to Black Death in medieval Europe (Dols et al., 1977; Ziegler, 2013; Twigg et al., 1984; Gottfried, 2010), when millions of people lost their family (see Fig. 1.1 for illustration). While in the recent decades, many types of newly emerging diseases, such as, severe acute respiratory syndrome (SARS) (Consortium et al., 2004; Fouchier et al., 2003; Osterhaus et al., 2004; Small and Tse, 2005), the H5N1 strain of avian influenza (Small et al., 2007; Chen et al., 2005; Claas et al., 1998; Khazen et al., 2009) and human immunodeficiency virus (HIV) (on HIV/AIDS. and Organization, 2006; Organization et al., 2011; HIV and LONG, 2012; Morris and Kretzschmar, 1997), frequently take place and propagate all over the world, which not only bring heavy threat for public health, but also induce the economic loss for agents and countries. For example, in the early stages of SARS outbreak, 8089 persons were infected and 774 of them did not survive in mainland China. But after just several weeks, this disease quickly spread to 37 countries and cause more deaths (Smith, 2006). Another typical example is the
outbreak of the H1N1 influenza in 2009. This disease brought huge challenge to many socio-economic systems and even forced the governments and public health organizations to consider the necessary prevention measures for controlling its propagation and reducing the economic expense (Fraser et al., 2009). In this sense, it becomes of particular importance to have a better understanding of propagation process, and then to design effective prevention measures.

Based on this consideration, various types of mathematical models were proposed since the 18th century (Bernoulli, 1760; Anderson et al., 1992; Keeling and Rohani, 2008; Diekmann and Heesterbeek, 2000; Hethcote, 2000). With these compartment models, the whole population is divided into several parts according to the transmission and infection status (Anderson et al., 1992; Blower, 2008; Anderson et al., 1979; Pastor-Satorras and Vespignani, 2007; Klein et al., 2007; Boccaletti et al., 2006). At the same time, more complex evolutionary dynamics are considered to incorporate the realistic events like birth and death (Anderson et al., 1979). These mathematical models can provide prediction for the outbreak of many diseases (Fenner et al., 1988; Cozzo et al., 2013; Newman, 2003; Dorogovtsev et al.,
2008), enable researchers to understand more characteristic details of some diseases (Bailey et al., 1975; Anderson and May, 1982; Anderson et al., 1992) and calculate the infection risk (Liu et al., 2009; Yang et al., 2007; Kitchovitch and Liò, 2010). Even though these dynamics models help to explain some phenomena and seem logistic in highly transmissible disease, there still exists a serious limitation: because of the lack of enough understanding of the spreading mechanism of disease, the early dynamics model just assumed that the whole population is fully well-mixed or has a homogeneously mixing pattern (Bailey et al., 1975; Hethcote, 2000; Ewald, 1994) (namely, one healthy person can have contact with any infected agent). This simple assumption is very un-realistic for diseases spreading through close contact. Therefore, the development pace of mathematical epidemiology slowed down until the largely rising of complex network research in the middle of the 20th century (Albert and Barabási, 2002; Dorogovtsev and Mendes, 2002; Newman, 2003; Pastor-Satorras and Vespignani, 2001).

To mimic the realistic contact process, spatial complex network is introduced into the epidemiology, namely, the so-called spatial epidemiology (Gómez et al., 2010; Pastor-Satorras and Vespignani, 2001; Eguiluz and Klemm, 2002; Gang et al., 2005; Bogúná et al., 2003a; Pastor-Satorras and Vespignani, 2002a). Along this research line, many infectious diseases can be modeled, where agents are denoted by the nodes of networks and their direct contacts are described in the form of edges (Pastor-Satorras and Vespignani, 2001; Lloyd and May, 2001; Bauch, 2002; KRETZSCHMAR, 1995; Kretzschmar and Morris, 1996). Correspondingly, the number of edges connected to the nodes is the degree, while the probability distribution of all the degrees over the network is the degree distribution. When implemented to describe contact infection disease, these network models can easily obtain more details about the interaction of individuals and their environment. For example, using the stochastic network models, we can know the relationship
between network structure and propagation pattern of sexually transmitted epi-
demic (KRETZSCHMAR, 1995; Kretzschmar and Morris, 1996; Gómez-Gardeñes et al., 2008; Eames and Keeling, 2002). More specially, with the introduction of many physics concepts (such as, phase transition and percolation) recently, spatial epidemiology models are also used to predict the outbreak threshold of some diseases or computational virus on the community networks and technology networks (Boguná and Pastor-Satorras, 2002; Boguná et al., 2003b; Wang et al., 2003; Moreno et al., 2003; Barthélemy et al., 2005, 2004). The research shows that this threshold is closely related with details of network topology, which can be further validated by statistical physics methods, such as mean-field analysis (Kleczkowski and Grenfell, 1999; Pastor-Satorras and Vespignani, 2002a; Liu et al., 2009; Heesterbeek, 2000; Kermark and Mckendrick, 1927; Gómez et al., 2011), percolation theory (Newman, 2002b; Meyers et al., 2005), pair approximation (Meyers et al., 2005; Van Baalen and Rand, 1998; Keeling and Grenfell, 1997; Altmann, 1995; Morris, 1997; Filipe and Gibson, 1998), Markovian chain (Wang et al., 2003; Kostova, 2009; Gómez et al., 2010; Granell et al., 2013) and reaction diffusion (Colizza et al., 2007; Colizza and Vespignani, 2007; Vespignani, 2008; Meloni et al., 2009; Saldaña, 2008; Lund et al., 2013).

Besides the prediction of threshold, another role of networked epidemiology models is to discover the potential measures preventing the diffusion of infection, which has widespread applications. One of the most renowned examples is the acquaintance immunization (Cohen et al., 2003; Madar et al., 2004; Gallos et al., 2007; Fu et al., 2008), which requires the immunization of random acquaintances of random nodes and can dramatically reduce the threshold of infection. Turning to highly heterogeneous networks, targeted immunization, which needs the knowledge of node location and global knowledge, will be another effective way to eradicate the infection sources (Pastor-Satorras and Vespignani, 2002b; Salathé and Jones, 2010; Wang et al., 2009). When applied to the outbreak of foot-and-mouth disease
(FMD), the mass vaccination and ring vaccination, in which cases the nodes having close contact with infected neighbors are protected, can be used to control the diffusion of disease (Keeling et al., 2003; Müller et al., 2000). Based on these early outcomes, more immunization scenarios were subsequently proposed to prevent the fast propagation of infection (De Wals et al., 2001; Pastor-Satorras and Vespignani, 2005; Liu et al., 2003; Chen et al., 2008b; Zanette and Kuperman, 2002; Gómez-Gardenes et al., 2006; Greenhalgh, 1986).

In spite of great achievements of the recent years, there still exists one shortcoming of particular relevance that limits the development of spatial epidemiology. This is that human behavior is almost neglected, which in fact plays a crucial role in the decision-making process of prevention strategy through a disease-behavior feedback mechanism (Funk et al., 2010; Perisic and Bauch, 2009a; Zhang et al., 2010; Araz et al., 2012; Funk et al., 2009; Galvani et al., 2007; Vardavas et al., 2007; Reluga, 2010). The most obvious example is that in the above mentioned prevention issues, vaccination or immunization is completely compulsory, but does not involve the consideration of human willingness, religious belief, individual cost (Fu et al., 2011; Reluga and Galvani, 2011; Shim et al., 2012b; Perisic and Bauch, 2009b; Liu et al., 2012a; Shim et al., 2009; Salathé and Bonhoeffer, 2008). In this sense, we need to look for new theoretical framework, which integrates spatial network models of infectious disease with tools from behavior science (Rat-Aspert and Fourichon, 2010; Wells and Bauch, 2012; Morsky and Bauch, 2012; Wu et al., 2011; Zhang et al., 2013a). This is also the main research target of this thesis.

For the study of human behavior, game theory is a very valuable tool, which has two basic elements: payoff functions and corresponding strategies (Zhang et al., 2012; Xia and Liu, 2014; Massad et al., 2005; Yaqub et al., 2014). By evaluating the payoffs of the strategies, we can predict individual behavior and population trait (Bauch and Earn, 2004; Xia and Liu, 2013; Bauch, 2005). In the following
chapter, we will also show more details of the development of evolutionary game
theory, especially for the spatial extension (Nash et al., 1950; Hofbauer and Sigmund, 2003, 1998; Gintis, 2000; Smith, 1982). Thus, if game theory can be mapped
into the spatial epidemiology models, where we need to define the detailed strategies and payoff functions, a new framework can be established: spatial behavior epidemiology. For example, during the vaccination campaign of seasonal influenza, if vaccination or non-vaccination can be regarded as two opponent strategies, we can predict how the vaccination behavior and disease infection change through combining the payoff functions of different states (Bauch et al., 2003; D’Onofrio et al., 2007; Chen, 2006; Arinaminpathy et al., 2012; Wu and Zhang, 2013). Similar to the behavior research, individual decision of vaccination also depends on the environment factor: perceived infection risk and vaccination status of its neighbors. When infection risk is high, more people are more likely to take vaccination. In this case, enough protection behavior forms herd immunity, which nourishes the emergence of non-contribution agents as the free rider problem (Bauch et al., 2010; Montopoli et al., 2009; Bauch et al., 2010). On the contrary, as the disease prevalence declines, people will not take the vaccination, which in turn leads to large infection.

Under this new framework of spatial behavior epidemiology, we can qualitatively predict the spreading situation of disease and advise appropriate protection policies (Galvani et al., 2007; Vardavas et al., 2007; van Boven et al., 2008). Of course, during the decision-making process, agents also face many uncertain factors. One typical example was the outbreak of the influenza A (H1N1) in 2009. The national diffusion of infection forced public health officials to decide to close school closure to reduce the possible contact with infection sources and mitigate the economic pressure (Lempel et al., 2009; Sander et al., 2009). The disease was, to large extent, controlled with school closure (Halder et al., 2010; Lee et al., 2010), but further complexity of virus made the efficiency of such prevention measure to be
limited in some regions.

Since the first proposal, spatial behavior epidemiology has proceeded, as a major landscape of disease control, for more than 10 years (Funk et al., 2010; Galvani et al., 2007; Bauch and Earn, 2004). People mainly focus on this framework from two aspects. One aspect is that most of existing works are from the theoretical analysis. This is actually not difficult to be understood. Given the novelty of this framework, related data are usually absent. In this sense, the most effective and available method is referring the individual behavior response according to their trajectories of infection and recovery. Thus, drawing a model, which incorporates behavior into epidemiology, becomes the popular approach for studying and quantifying these feedback mechanisms. Another aspect is the experimental viewpoint, which just started to attract researchers’ attention three years ago (Araz and Jehn, 2013; Shim et al., 2012a; Ibuka et al., 2014; The, 2014). With the experimental circumstance, the scenario of disease season is designed to evaluate the perceived values of epidemiological parameters. Based on these perceived values, people try to understand and explain the potential mechanisms for the realistic disease, and finally look for the possible policy of preventing disease spreading. One typical example is that, in a recent study (Shim et al., 2012a), the authors implemented a survey about the infection risk of influenza; they unveiled that the altruism could promote vaccination.

Aiming to enrich the content of spatial epidemiology, I try to study the behavior-disease feedback questions theoretically in this thesis. On the one hand, this is due to the difficulty of obtaining empirical data. On the other hand, it is still lacking to have a comprehensive exploration for the influence of behavior dynamics on the control of epidemic. As mentioned above, I will refer to game theory to study the evolution of individual behavior. While for the disease models, I still use the traditional epidemiological setups: susceptible-infected-susceptible (SIS)
and susceptible-infected-recovered (SIR) models. Combining these two fields and employing complex networks as the contact networks, the theme of this thesis is the interplay of disease-behavior dynamics on complex network models: incorporating evolutionary game into epidemiology. In this thesis, I will consider this new framework from several aspects, as I will describe in the following subsection. These aspects are very close to our life: some of them are based on the individual consideration; the remaining are according to the population benefit.

1.2 Thesis organization

In this thesis, I mainly consider the spatial behavior epidemiology from theoretical viewpoints, which are inspired by the realistic issues in our life. The thesis is divided into seven chapters and its organization is as follows.

Chapter 1 gives an overview of the thesis.

In Chapter 2, I will briefly introduce the backgrounds of related fields: traditional epidemic dynamics, complex networks, spatial epidemiology, game theory for evolutionary behavior study and spatial disease-behavior feedback dynamics. By introduction to the emerging new field, the shortcomings of previous framework can be better resolved, which finally leads to the theme of this thesis.

In Chapter 3, I will first focus on the influence of risk perception on vaccination and spreading of disease. Different from the simple assumption that agent can only obtain the risk information from the local connection in previous studies, I suggest that the infection information of risk perception comes from two cases: local neighborhood and whole system. This is also consistent with realistic case. Then, I explore the effect of risk perception on two cases of permanent and temporary vaccination.
Now that disease brings great threat to human society, in Chapter 4 I propose to model the effects of temporary self-protection measure, such as, wearing the facemask, frequently washing hands. This strategy seems to be very valuable in some particular cases, where the cost of vaccine is too high or the supply of vaccination is not sufficient. This new strategy is low expense, but its efficiency is also limited. I study how the cost and efficiency of such a strategy affect the propagation of disease.

Then in Chapter 5, I consider another scenario: adaptive protection strategy, which is of particular importance for the case that there are neither vaccination nor other protection measures. In this case, cutting the link with the infection sources is the most effective method. In this chapter, I will show the effect of cost and efficiency of adaptive protection strategy on the disease control and topology evolution of contact network.

To promote vaccination uptake, subsidy is usually a valuable policy for the public health official. However, whether subsidy really promotes the vaccination level is still unclear. Motivated by this question, I will explore the impact of subsidy on vaccination campaign in Chapter 6, where, except for controlling the disease diffusion, reducing the total expense for the whole society is another target.

Finally in Chapter 7, we conclude the thesis and provide outlook for the future work.
Chapter 2

Background of disease and behavior study in spatial complex networks

As described in the first chapter, this thesis mainly focuses on the impact of voluntary prevention measures on spatial disease spreading models with game theory framework. In this chapter, I will respectively review the related background from five aspects of disease dynamics.

2.1 Traditional epidemic dynamics

Human being has been haunted by various kinds of infectious diseases for long time. The early record of conflict between human being and infectious disease is the Black Death bubonic plague in 14th century, which caused a great number of people to be infected and to die in Europe (Dols et al., 1977; Ziegler, 2013; Twigg et al., 1984; Gottfried, 2010; Campbell, 1932). From that time, numerous
novel types of disease subsequently took place, brought threat to human society and caused enormous morbidity and mortality (Organization et al., 2008; Blum and Nelson-Mmari, 2004). The recent outbursts of the severe acute respiratory syndrome (SARS) (Consortium et al., 2004; Fouchier et al., 2003; Osterhaus et al., 2004; Small and Tse, 2005), avian influenza and swine influenza (H1N1) also re-triggered great attention, where controlling the spreading range and reducing the economic loss were the main targets (Fraser et al., 2009; Mashonganyika et al., 2009; Real-Time, 2009). Thus, how to design effective protection policy has become extremely important and has captured renewed interest in the scientific community (Liu et al., 2012a; for Disease Control et al., 2008; Klein et al., 2007; Keeling and Grenfell, 1997; Henderson et al., 1999; Basu et al., 2008). In this subsection, I will briefly recall the traditional development of epidemiology and the previous control measures, which mainly come from the viewpoints of mathematics and dynamics evolution. At the same time, the outcomes of traditional theory are also discussed, based on which I can proceed with this thesis study.

Up to now, a great number of dynamical scenarios relevant to disease spreading have been proposed, where the common character is that the whole population is divided into several compartments, each of them denotes one state of epidemic dynamics processes (such as, I denoting infected agent, while S representing susceptible or healthy individual) (Bailey et al., 1975; Anderson and May, 1982; Keeling and Rohani, 2008; Wang et al., 2011). To more vividly mimic the realistic situations in the populations, other characters are also introduced in the subsequent literatures, such as, the birth rate, death rate, mutation rate, and so on (Anderson et al., 1979; Lopez and Murray, 1998). Among all these mathematical epidemiological scenarios, two typical models, the susceptible-infected-susceptible (SIS) and susceptible-infected-recovered (SIR) models, have attracted remarkable attention in both theoretical as well as experimental analysis (Keeling and Ross, 2008; Pastor-Satorras and Vespignani, 2002a; Keeling et al., 2001; BJøRNSTAD
et al., 2002). In these compartmental models, individuals are usually divided into several states: susceptible (S), infected (I), and recovered (R). In particular, recovered (R) means individuals who are either recovered from the disease and immune to further infection, or dead. In both models, the susceptible (S) agent can be infected with the probability $\beta$ (namely, the infection rate) when it has the contact with one infected individual. As for the infected (I) agent, he can recover and acquire immunity at an average rate of recovery from infection $\mu$ (namely, the recovery rate). One key quantity to reflect the severity of the disease is $R_0$, the so-called basic reproductive number (Heffernan et al., 2005), which is the average number of secondary infections introduced by one single infected agent, during one infectious period, into the susceptible population. This parameter can indicate the epidemic threshold for the well-mixed populations, where $R_0 = \beta/\mu$. If $R_0 < 1$, the disease vanishes at last, while $R_0 > 1$ may introduce more phases except for disease-free state.

Figure 2.1 shows the schematic representation of both models, where there exists obvious difference. In the SIS model, the recovered agent becomes susceptible again, i.e., the recovered person can not be endowed with a long lasting immunity after the attack of infection. This model is mainly suitable for the diseases like seasonal flu, gonorrhoea and other sexually transmitted diseases. At variance, if the recovered agent acquires long-term immunity and cannot be further infected, SIR model is the appropriate candidate, which is suitable to model diseases, such as smallpox, pandemic influenza, pertussis and so on. While for the steady regime, the difference is also distinct. In SIS model, dynamics can sustain since the recovered agents can be re-infected over time. While for SIR model, the dynamics stops if there is no new infected agent coming into the population, namely, only susceptible and recovered agents finally exist in the population.

Moreover, the evolution process of both models can be described using the ordinary
Figure 2.1: [Adapted from Lin and Li (2014)]. Schematic illustration of SIS (a) and SIR (b) models, where $\beta$ and $\mu$ denote the transmission rate and recovery rate, respectively.

differential equations (Anderson et al., 1992). Take SIR model as the example, if we use $s(t)$, $i(t)$ and $r(t)$ to denote the respective fraction of three compartments or states, the time evolution of the whole population can be expressed as follows

$$\frac{ds}{dt} = -\beta si, \quad \frac{di}{dt} = \beta si - \mu i, \quad \frac{dr}{dt} = \mu i,$$

(2.1)

where the last equation is redundant, because the variables satisfy $s(t) + i(t) + r(t) = 1$ at all the time steps. By solving these equations, it is easy to get the conditions where the disease can spread or vanish.

Though the above mentioned framework has obtained a great number of achievements from the viewpoint of mathematical epidemiology, there are two serious shortcomings restricting its further development, especially when compared with realistic cases. In previous framework, vast majority of research works simply assume that, based on the compartments models, the whole population is well-mixed, namely, one infected (susceptible) person can equally contact with any other member(s) of the population. Obviously, this assumption is not consistent with realistic situations. In our life, the transmission of most diseases is mediated through direct contact progress in the local interaction rather than the un-realistic spreading between arbitrary agents (sexually transmitted diseases are typical examples) (Cojocaru, 2008; Perisic and Bauch, 2009b; Moreno et al., 2003). Another limitation in the classical epidemiological theory is that the above models only consider the question from the viewpoint of disease evolution dynamics, while fully neglect how to reduce the risk of infection and control its propagation.
To overcome the first shortcoming (namely, the neglect of contact pattern), the introduction of the spatial contact network between interaction agents becomes very meaningful. In this sense, individuals have disease-causing contacts only along the links in the spatial network (namely, each person has different contact patterns). Moreover, to mimic the realistic cases of heterogeneous contact, we can also vary the number of connections for each person. Based on the consideration of connection networks, the more realistic case that each individual poses different probability of disease-causing contact with other individuals can be implemented as well. In section 2.2, I will briefly review the development of spatial network; then try to show the spatial epidemiological models in section 2.3, where the spatial network is used to mimic the social contact network. To conquer the second shortcoming of traditional epidemiology study, it becomes necessary to introduce prevention measures. This is also valuable from the viewpoint of public health and economic loss. In section 2.4, I will review the suggested control measures in the research of spatial disease dynamics.

2.2 Development of spatial complex networks

In this section, I will briefly review the development of complex network. Based on the framework of spatial network, the traditional epidemiology dynamics is introduced into the spatial contact network in section 2.3.

Complex network, as a widely discussed concept in the last decades, is closely related with our life, ranging from the chemical reaction network in our body to a variety of communication networks in realist society (Boccaletti et al., 2006; Strogatz, 2001; Albert and Barabási, 2002; Newman, 2003; Watts, 1999; Dorogovtsev and Mendes, 2002). For instance, many social, physical, biological and technological systems can be mimicked via network framework, where individuals
or organizations are denoted by nodes while connections or links stand for the interactions among them. Moreover, with high development of economy and education, more and more people have also taken up this discipline, which furthers the application of complex networks (Pastor-Satorras et al., 2003; Pastor-Satorras and Vespignani, 2007; Uzzi et al., 2007; Barabasi and Crandall, 2010; Knoke and Yang, 2008). In this sense, it is very necessary to review the development of complex network, especially the recent achievements by borrowing the method of statistical physics and nonlinear dynamics since the middle of the 20th century.

In the early stage, the graph in mathematics was the tool used to describe networks, which could be regarded as a system constructed by some nodes according to a certain rule (Bower, 2005; Erdös and Rényi, 1959; Bollobás, 1998b; Newman et al., 2001; Bollobás, 1998a). Within this system, the connection relationship between the nodes could be denoted by edges. Since the initial basic concept and method, the subsequent progress was very slow and limited till Erdös and Rényi proposed random graph theory Erdös and Rényi (1960), which is usually regarded as the first systematic work of complex network. After this seminal idea, three more basic types of graphs were suggested (Grab et al., 2007). The first is the simple graph, where the nodes are connected by links without any direction and weight information. The second is the directed graph, where the links between nodes have particular directions denoted by the arrows. The last is the weighted graph, whose links possess the particular weight magnitude. The two latter graphs are also ubiquitous in our life. For instance, the links in social collaboration networks are usually directional (Newman, 2001a; Abbasi and Altmann, 2011; Tomassini and Luthi, 2007), while weights denoting the synaptic strengths also exist in brain neural network (Zemanová et al., 2008).

Although there has been great accumulations, the really explosive progress, which
changed the research development in history of complex network, is the suggestion of topological properties: the small-world feature characterized by short path length and scale-free character unveiling the presence of hub nodes with large number of connections than the average degree (Strogatz, 2001; Albert and Barabási, 2002; Newman, 2003; Watts, 1999; Barabási and Albert, 1999). Along this line, the study of complex networks enters into a new era: network topology is proposed from the early homogeneous to heterogeneous form (Almaas et al., 2004; Jeong et al., 2000; Barabási, 2002); various forms of two-dimension and three-dimension networks appear (Barabási et al., 2000; Ripeanu, 2001; Albert and Barabási, 2000); and even the community and modular networks have been found in many biological and social organizations (Sporns et al., 2004; Ravasz et al., 2002; Radicchi et al., 2004). For example, biological and technological networks, such as protein-protein interaction network, neural networks, metabolic networks, world wide web (WWW) and Internet, usually belong to the disassortative scale-free networks, where the large-degree nodes are inclined to connect with small-degree nodes (Newman, 2002a; Medina et al., 2000; Jeong et al., 2001; Martinez, 1991). However, the vast majority of social networks, such as scientist collaboration and citation networks, sexual contact networks and company directors networks, fall into the range of assortative mixing patterns, where large-degree (small-degree) nodes connecting to large-degree (small-degree) nodes build the scale-free topology (Newman, 2002a; Grossman and Ion, 1995; Watts and Strogatz, 1998; Newman, 2001b). As for community or modular networks, there usually exist loose connections between these communities or modules (Haykin, 1994; Dagan et al., 2008). Moreover, in recent research (De Domenico et al., 2013; Battiston et al., 2014), the previous single network was even extended to multi-layer networks, where the status of nodes not only depends on the internal connection circumstance, but also relies on the situations on other layers or networks. Similarly, the robustness or frangibility of network also brings another research tide, and even combines with
the empirical cases (Albert et al., 2000).

Besides the research progress of network property, the dynamics happening on these topologies has also attracted great attentions for long time (Boccaletti et al., 2006; Castellano et al., 2009), which shows that network structure plays a significant role in determining the states and characters of the system activity. For example, in recent reports (Santos and Pacheco, 2005; Gómez-Gardeñes et al., 2007), where evolutionary game models were implemented on heterogeneous scale-free networks, the evolution of cooperation dynamics was promoted to a extremely high level. If the voter model is introduced into the small-world networks, the plateau formed by active interfaces hinders the diffusion of opinions so that the consensus eventually relies on the linear system size (Castellano et al., 2003; Vilone and Castellano, 2004). By means of complex network, we can better understand the bridge between structure and function in the study of neural science (Hall et al., 1992; Amit, 1992). Moreover, some other models, such as food web line (Martinez, 1991), percolation (Parshani et al., 2010a; Leicht and D’Souza, 2009), synchronization (Arenas et al., 2008; Boccaletti et al., 2002), routing traffic (Echenique et al., 2004), traffic flow (Mukherjee and Manna, 2005; Coclite et al., 2005), flocking behavior (Kattas et al., 2012; Xu et al., 2012), particle diffusion and individual mobility (Colizza et al., 2007; Belik et al., 2011; Gómez-Gardeñes and Latora, 2008; Reichenbach et al., 2007) are also introduced into the spatial interaction topology. In virtue of new mathematical and statistical methods, these evolutionary dynamics in turn promote the development of complex network. A typical example is that based on the situation of information flow, engineers try to design more effective networks to decrease the blocking of information transmission and enhance the robustness of networks (Demetrius and Manke, 2005; Callaway et al., 2000; Ahlswede et al., 2000).

Now that network topology and dynamics model have mutual impact, adaptive
network recently becomes a new candidate to describe their dynamical effect (Gross and Blasius, 2008; Gross et al., 2006). There are usually two aspects for the adaptive network. On the one hand, the nodes are individual dynamical systems which are coupled through static links (Bornholdt and Rohlf, 2000). On the other hand, the adaptive network not only involves the evolving of dynamics models over time, but also characterizes the joint development of the network topologies, i.e., the so-called co-evolution (Poncela et al., 2008; Snijders et al., 2007). The latter has recently attracted great attention, where a feedback loop between the state and network topology takes place, as schematically featured in Figure 2.2.

We can mention some examples. In a recent work (Zhang et al., 2011b), where an aspiration-induced reconnection scenario was introduced into a public goods game, the authors unveiled that a middle aspiration not only guaranteed the maximal cooperation level, but also excited a negative feedback effect, where the weak peak of reconnection made the initial homogeneous network posses the strong heterogeneous pattern. It is also worth mentioning how the dynamical topology affects the diffusion of opinion dynamics. In (Vazquez et al., 2008), Federico et al. assumed that node can adjust its connection with a certain probability (or otherwise update...
its opinion) in the voter model, where the state of system on adaptive networks could be depicted by the interface density $\rho$ quantifying the percentage of edges connecting nodes with different opinion states. Correspondingly, $\rho = 0$ ($\rho \neq 0$) means active (frozen) phase. They showed that the competition between opinion imitation and the rewiring links impelled a fragmentation transition from active to the frozen phases. Similarly, more dynamics models, such as, the game theory, synchronization, disease spreading, were extensively examined as well during the past years (Perc and Szolnoki, 2010; Boccaletti et al., 2006).

Moreover, some researchers also start to focus on introducing adaptive networks into application (Gross and Blasius, 2008; Ganguly et al., 2009; Lee, 1995). The adaptive Boolean network can be used to elucidate how the networks can self-organize towards dynamical criticality (Bornholdt and Rohlf, 2000). When the joint propagation of both disease and information opinions were incorporated into the social adaptive network, it was shown that this framework enables us to observe the emergence of new phase transitions (Ehrhardt et al., 2006; Gross et al., 2006). While for the technical distribution networks, such as power network and traffic network, the component failure, such as, the traffic jams or electrical line failure would bring an incentive for the improvement of additional management or connections (Wang et al., 2005).

Beyond the traditional fashion of single network layer, more complex forms of network were proposed recently, where the system is usually composed of more than two related networks, namely, the so-called ”multilayer network” (Cardillo et al., 2013b). Under such a framework, the state of one node is not only affected by its own network, but also relies on the situation of other networks (Battiston et al., 2014; Buldyrev et al., 2010; Parshani et al., 2010a). However, because this form has gone beyond the scope of this thesis, I do not give more details herein and in the following chapters.
2.3 Spatial epidemiological study and compulsive protection measures

To mimic more realistic contact pattern, spatial contact network should be introduced into the traditional epidemiological study. In this section, I will briefly review the development of spatial epidemic study and control measures. Besides, the shortcoming of compulsive protection measures in spatial contact network is also discussed.

In virtue of network viewpoint, disease dynamics is integrated into the framework of spatial contact network, where nodes present component units or agents and the contact can only take place through links between nodes. When the empirical features of social networks, such as, small-world, scale-free and community, are considered, various phenomena of disease dynamics, especially the threshold values of disease, are investigated. For example, on the scale-free network, it was shown that the threshold of disease outbreak was absent (namely, it is difficult to fully eradicate the source of infection, see Figure 2.3), which could get further validation from the mean-field analysis (Pastor-Satorras and Vespignani, 2001). This explains the fact why there always exist computer viruses and these viruses have long lifetime on Internet and World Wide Web (WWW), because both of them fall into the range of scale-free networks. While for the small-world network, the theoretical threshold of disease outbreak is closely related to average degree of the network (Moore and Newman, 2000; Da Gama and Nunes, 2006; Newman et al., 2002). In the line with these discoveries, a series of works are motivated on the different social networks, where the disease threshold heavily relies on the topological properties, such as, degree correlation (Boguná et al., 2003a; Moreno et al., 2003), community structure (Wu and Liu, 2008; Liu and Hu, 2005), link length (Newman, 2002b), and the k-core (Castellano and Pastor-Satorras, 2012), to
Figure 2.3: [Adapted from Pastor-Satorras and Vespignani (2001)]. Surviving probability $P_s$ of infected nodes on scale-free networks for different network size. From top to bottom, the sizes of network are $5 \times 10^5$, $2.5 \times 10^4$, $1.25 \times 10^4$ and $6.25 \times 10^3$, respectively.

name but a few. Looking at some examples more specifically, in recent researches (Parshani et al., 2010b; Castellano and Pastor-Satorras, 2010), the authors showed that the vanishing threshold of spatial disease dynamics results from the active behavior of the largest hub node, which usually acts as a self-sustained infection source. In (Li and Wang, 2006), it showed that the relaxation behavior of diseases could affect the final phase transition of system (from disease-free phase to endemic phase).

Similar to the development of complex network, the co-evolution of disease dynamics on spatial population also attracts much attention. Under this scenario, many novel phenomena have been examined. For example, recently Thilo et al. considered the rewiring process in the SIS model, where, besides infection and recovery, the susceptible (S) agent can cut the infected link and re-connect with one new susceptible agent with a certain probability (Gross et al., 2006). They found that such adaptive rewired events not only introduce new phases (such as oscillation and bistable phase) and reduce the infection risk under certain conditions, but also make the contact network possess the assortative mixing pattern.
Figure 2.4: [Adapted from Colizza et al. (2007)]. Schematic illustration of networked metapopulations, where each node denotes one metapopulation. Within each node, there are many particles or agents. Then these particles or agents can diffuse among different metapopulations as shown by the arrows.

i.e., large-degree nodes are inclined to contact large-degree nodes. In (Zhou et al., 2012), where susceptible nodes can cut the connection with infected ones yet avoid getting isolated in the growing networks, it was found that a novel phenomenon, the so-called "epidemic reemergence" takes place: a few infected agents could survive for long time, but after long time they burst and hold the prevalence for a short time. This process repeated for several times before the final vanishing of all the infection sources. Within the framework of co-evolution, not only disease dynamics evolves, but the topology properties of contact network also change with time, which is much closer to some social phenomena. Recently, the influence of other characteristics, such as temporary link deactivation (Tunc et al., 2013), different time scales (Riley, 2007) and fluctuation (Palop et al., 2006), was also unveiled.

Besides the simple assumption of spatial network, the networked metapopulation becomes a hot topic during the past decade, which is particularly suitable to describe the disease of simultaneous outbreak in several countries or states. Under this framework, the total population is still denoted by one contact network, but each node dose not denote one agent. Conversely, in each node, there are many agents, namely, a small population or metapopulation (see Figure 2.4 for schematic
illustration). Then, agent can spread the disease within the same metapopulation or node, at the same time disease can also diffuse through the link between these nodes or metapopulations. This model can be mapped to the realistic case that one individual can carry the disease to its own or other countries via traffic tools, such as, airplane. In this sense, people mainly consider the problem from two aspects. One aspect is the diffusion of agents between different nodes. For example, in one early work (Colizza et al., 2007), the authors showed that by using the basic reaction diffusion process, the topological properties of systems had a crucial impact on phase diagram: the network heterogeneity could support the infection diffusion even for limited infected agents, but suppressed the critical point in density-driven phase transitions. The other aspect in the research of metapopulation is community, where agent can leave its own spot with a certain probability, but he returns to this spot with another probability. In recent research (Wang et al., 2013), where two types of location-specific heterogeneous commuting patterns were considered, the authors showed that under different scenarios, the contact rate could lead to various phases for disease prevalence.

In the networked metapopulation case, though there are topological links between each metapopulation, the contact still keep the well-mixed pattern. To depict the realistic case more vividly, the multilayer networks were suggested recently (Min and Goh, 2013). In this framework, the spatial contact pattern within each layer is depicted via the traditional complex network, and then the disease can also diffuse between network layers. We can examine some recent achievements. For example, the partial overlapping of network changed the threshold of disease outbreak between two networks (Buono et al., 2014); multilayer structure promoted the propagation of disease located on different layers (Zhao et al., 2014); competing disease dynamics affected the respective threshold of each layer (Karrer and Newman, 2011); interconnection between different systems decrease the defense with infection (Sanz et al., 2014). In addition, the incorporation of other dynamics on
multilayer networks is also worth particular attention. In (Granell et al., 2013), where the social awareness dynamics and disease dynamics separately spread on each layer, the authors found that the threshold of disease heavily depended on the propagation of both activities.

Now that the disease brings huge risk to human society, especially remarkable for worldwide pandemics, it becomes of utmost importance to propose the preventive measures. Till now, there have been a great number of immunity policies and methods proposed in the spatial population (Madar et al., 2004; Cohen et al., 2003). For example, compared with the random immunization, Pastor et al. showed that targeted immunization, as the primary measure of preventing disease, could successfully eradicate the infection sources on complex contact networks (Pastor-Satorras and Vespignani, 2002b). With the targeted immunization, requiring the immunization of random selected acquaintances of random selected agents, was used as the main measure, the infection threshold could be dramatically reduced (Wang et al., 2009; Pastor-Satorras and Vespignani, 2002b). Another interesting measure is mass and ring vaccination (Müller et al., 2000; Greenhalgh, 1986), where the spreading of infection could be controlled fast. However, among all these existing researches, there is a serious question (which is one shortcoming of the traditional spatial epidemic study) that majority of the above-mentioned prevention measures are based on the premise of compulsory requirements, and completely neglect the individuals’ willingness and desire, which disagrees with many social factors including religious beliefs, the human right and social total expense as well. That is, the previous studies only considered the question from the viewpoint of mathematical evolution dynamics, but individual behavior traits were fully neglected. While in the realistic world, how people take prevention measures depends on many social factors around them and is closely related with human behavior. Thus to overcome this limitation, we need to find new methods of incorporating human behavior into disease prevention. Through development
of recent years, game theory has been proved to be a useful way of predicting individual behavior or population traits based on evaluating utility/payoff function of different strategies. In section 2.4, I will briefly review the development of evolutionary game theory, and then in section 2.5 I will try to incorporate game theory into spatial epidemiology study, which produces a new field: disease-behavior feedback dynamics study, where the prevention measures are mainly voluntary.

2.4 Evolutionary game theory

In this section, I will briefly show the development of evolutionary game theory, especially for spatial game, and then exhibit its application in related issues. This thesis will focus on the applications of game theory to spatial epidemiology, where there exist corresponding strategies and payoff or utility functions.

The game theory can be dated back to the early work "Theory of games and economic behavior" (Neumann and Morgenstern, 1944), whose function is to predict individual behavior and population traits by evaluating the payoff/cost functions of different strategies. So, in the framework of game theory, there are two basic factors: strategies and payoff or utility functions of these strategies; while the core question is how to maximize utility function by comparing different strategies. After this pioneering work, various mathematical definitions, analysis methods and a key assumption that the players are rational players were introduced (Weibull, 1997; Nowak, 2006a; Axelrod, 2006). During this process, the most remarkable achievements were the suggestions of Replicator Dynamics and Nash Equilibrium (NE) in different types of games, such as prisoner’s dilemma, hawk-dove game, etc. (Hofbauer and Sigmund, 1998; Smith, 1982; Hofbauer and Sigmund, 1998). Under the Replicator Dynamics, the strategies can evolve in the populations based on
their payoff or utility. While for Nash Equilibrium, it is just one profile of strategies such that no player has a unilateral incentive to deviate from this by choosing another strategy. That is to say, in one Nash equilibrium, the strategies form best responses to each other. However, all these assumptions still possess obvious difference from realistic life, where the agents are not completely rational and follow certain evolutionary rules. With the development of nonlinear dynamics and statistical physics, many new spatial and temporal methods and notions were introduced into game theory, leading to the so-called evolutionary game theory.

During the development of evolutionary game theory, a great number of games have been proposed, while the most interesting question is how to produce the cooperative behavior, which is helpful for explaining the evolution of human society (Szabó and Fath, 2007; Ohtsuki and Nowak, 2006; Nowak et al., 1994; Szabó and Tőke, 1998). Up to now, the prisoner’s dilemma (PD) and public goods game (PGG) have attracted the greatest attention, where the conflict between individual benefit and collective benefit drives the individual strategy choice. In previous theory (Boyd and Lorberbaum, 1987; Rapoport, 1965), the survival of cooperation is very difficult because non-cooperation behavior can get the higher short-term benefit, but this is not consistent with the fact of ubiquitous cooperation existing in our life. To overcome this unrealistic result, many scenarios have been proposed for supporting the evolution of cooperation, such as, tit-for-tat strategy (Nowak et al., 1993; Nowak and Sigmund, 1992; Imhof et al., 2007), voluntary participation (Szabó and Hauert, 2002; Hauert et al., 2002), win-stay-lose-shift (Nowak et al., 1993; Chen et al., 2008a; Imhof et al., 2007), spatially lose-shift (Nowak and May, 1992; Santos and Pacheco, 2005; Huberman and Glance, 1993), mobility of agents (Vainstein et al., 2007; Helbing and Yu, 2009) and so on. Recently Nowak attributed all these existing scenarios to five mechanisms: kin selection, direct reciprocity, indirect reciprocity, network reciprocity and group selection (Nowak, 2006b) (see Figure 2.5 for schematic illustration). While among
Figure 2.5: [Adapted from Nowak (2006b)]. Schematic illustration of five mechanisms for the evolution of cooperation.

five mechanisms, network reciprocity, where agents are located on the spatially structured topology and have the interaction only with their neighbors, has attracted the most notable attention. Under the network reciprocity, cooperation behavior can survive through forming the compact clusters on complex networks, which help to resist the exploitation of non-cooperation behaviors (Nowak and May, 1992). After this seminal discovery, the role of spatial interaction networks and various mechanisms have been abundantly suggested (for comprehensive reviews see Refs. (Szabó and Fath, 2007; Roca et al., 2009)).

Similar to the development of complex network and spatial epidemiology, the co-evolution of evolutionary game has also attracted great attention recently (Perc and Szolnoki, 2010). This framework not only involves the evolution of strategy, but also characterizes the adaptive development of interaction topology or evolution dynamics. We can mention some examples. In (Zimmermann and Eguiluz, 2005; Ebel and Bornholdt, 2002), if agents could adjust their linked neighbors according to their performance or strategies, this scenario would be very beneficial for promoting the evolution of cooperation. The growth of network, based on the payoff of game rather than the degree of nodes, played a positive role in guaranteeing high cooperation level (Poncela et al., 2009a,b). Particularly, if the individual ability for updating its strategy was a function of the performance of itself or its
neighbors, the promotion of cooperation was also an unavoidable result (Szolnoki and Perc, 2008).

Besides the evolutionary game models on single network, recently interdependent network reciprocity also becomes a hot topic. Here, it is worth mentioning that interdependent system is usually composed of more than 2 networks, while the evolutionary status of nodes not only depend on the circumstance of the local connections, but also rely on the status of other network. In this sense, how to build the interdependency seems to be of most importance. Because strategy and utility function are two basic factors of game theory, the interdependency of different systems is mainly built through above factors. We can look at some examples. In a recent work (Wang et al., 2012), an asymmetric utility function was assumed; it was unveiled that the stronger bias of utility function boosted the cooperation level. Similarly, an asymmetric imitation dynamics was introduced into interdependent networks (Santos et al., 2014), where there were different games on each network layer; the authors found that the decline of cooperation behavior on one layer necessarily led to the outbreak of cooperation on another layer. While for the co-evolution mechanisms on interdependent network, recent works showed that the partial overlapping between layers usually resulted in optimal situation of cooperation.

In additional to the theoretical development, the application of game theory is also useful. But how to introduce game framework into other topic becomes the first question. As above mentioned, there are two basic factors in game theory: strategies and payoff functions. So, if we can construct the strategies and the corresponding payoff functions, mapping game theory into these topics seems to be natural. For example, if the lane-change behaviors of drivers can be regarded as two strategies with respective payoffs, then traditional traffic flow could be studied within the framework of game theory (Yamauchi et al., 2009; Nakata
et al., 2010). For preventing the dangerous climate change, collective-risk social dilemma was used to study the investment behaviors of agents: if the individual investment could reach a climate target, the dangerous climate change could be avoided (Milinski et al., 2008). Moreover, other social dilemma topics, such as, environmental issues, the protection of species diversity and alarm calls, also get the help from the method of game theory. While for the topic of this thesis, I till try to introduce game theory into spatial prevention measures in the epidemiology, namely, to perform the disease-behavior feedback dynamics study. The most important question is to find corresponding strategies and their cost functions and then try to map epidemiology study into the framework of game theory. Under this framework, individual decisions of whether taking prevention behavior (such as, vaccination or not) can be regarded as the strategies, while the cost of these behaviors (such as, the cost of vaccination or infection) denotes the payoff functions of these strategies. When individuals update their behaviors (or make decisions), game theory is naturally incorporated into spatial epidemiology study, where the behavior and disease construct a feedback. In section 2.5, I will briefly show the existing achievements.

2.5 Introduction of spatial disease-behavior feedback dynamics

Combining all the content in the above sections, the limitations (lacking the spatial contact model and neglecting the impact of human behavior) can be resolved well. In this section, I can briefly show the development of behavior epidemiology, where the voluntary prevention measures are considered. Then, I exhibit the future challenge in this new discipline.
In the behavioral epidemiology, voluntary prevention measures, incorporating both dynamics of disease propagation and individual human behavior traits, are the primary control methods. Up to now, several potential measures, such as, vaccination, reducing the contact with infected agents, wearing mask, frequently washing hands and taking pharmaceutical drugs, have been proposed, where the adoption or not of prevention measures usually have their own cost. Among the existing measures, vaccination uptake attracts most attention from theoretical and practical viewpoints (Basu et al., 2008; Bauch and Earn, 2004; Shim et al., 2012a; Bauch et al., 2003; Ibuka et al., 2014; Bauch, 2005). With the outbreak of disease, people are more likely to taking vaccination to prevent the further spreading of infection in the epidemic season. However, when the vaccination reaches a certain level, the enthusiasm of vaccination starts to decline, which in turn amplifies the infection risk. Thus, agents face the new dilemma: to vaccinate or not. In this sense, game theory is introduced into the traditional spatial epidemiology for the decision-making process. We can look at some typical achievements.

In one recent work (Zhang et al., 2010), if agents only made the rational decision based on the lowest cost (i.e., if the cost of vaccination was lower, they only took vaccination; and vice versa), the authors found that disease could be effectively controlled on the heterogeneous scale-free networks, especially compared with the traditional case without considering human behavior. This phenomenon was attributed to a positive feedback mechanism motivated by the vaccination behavior of hub nodes. More specially, if some agents always held the selection of vaccination during the flu-like disease, these people could prevent the formation of susceptible clusters and inspired the more agents to take vaccination (Liu et al., 2012b). Besides, the influence of free-riders (Ibuka et al., 2014), imitation dynamics (Cardillo et al., 2013a), social contact network (Mbah et al., 2012), opinion clustering of taking vaccination (Salathé and Bonhoeffer, 2008) are also examined in the frame of game theory.
In addition to the behavior epidemiology on single network, its fast development with the multilayer networks also attracts great attention. Distinguishing from the above-mentioned cases, where the disease and prevention information simultaneously propagate in the same network, behavioral epidemiology usually assumes that these diffusion processes take place on different layers. The information update on one layer not only depends on its own layer, but also is affected by the information of other layers, which together decide the final results. In one recent work (Mao and Yang, 2012), the prevention information and infection information were diffused on two networks with full or partial overlapping, the authors found that, compared with the case of single network, the coupled diffusion dynamics could reduce the frequency and threshold of infection. Similarly, if the triple diffusion dynamics, which included disease diffusion, information flow regarding disease and preventive behaviors against disease, were incorporated into the multilayer viewpoint, the authors could explain the empirical trends of influenza infection and online query frequency within the empirical population (Mao, 2014). Aside from these discoveries, one recent report showed that the multilayer networks possessed two-side effects on the vaccination uptake (namely, accelerating or decelerating disease elimination) (Bauch and Galvani, 2013). These influence also changed with the change of social factors, which could be used to predict the optimal scenario of guaranteeing public health.

Though there have been several achievements in the disease-behavior feedback dynamics study, we still face many new challenges in this field. The typical challenge is how to collect the empirical data and combine with the theoretical models, in order to make more effective policies. This may be a long-term task. Due to the lack of data, I will mainly focus on the theoretical models in this thesis (namely, consider the problems from theoretical viewpoints). Another challenge is how to build proper coevolution models, where the prevention measures and topology connections (updating rule) together change over time.
In the following chapters, I will show several disease-behavior interaction models on complex networks in details. Inspired by realistic case that agents can obtain the infection information from both local and global environment, I will firstly examine the impact of risk perception on vaccination level; then turn to the temporary self-protection scheme, which is ubiquitous in our life; next, I explore the influence of adaptive protection scenario, which is particularly useful in the case of vaccine absence; and finally I consider the impact of subsidy policy, which may provide new sight for protecting public health.
Chapter 3

Impact of risk perception on vaccination behavior

As described in last chapter, introducing behavior into spatial epidemiology is a useful and new field. Under this framework, agents can decide whether or not to take the prevention measures. Before introducing more complex protection measures, we will first examine the impact of risk perception on the vaccination campaign in this chapter, where the evaluation of infection risk mainly depends on local information (such as the general contact of family members) and global information (such as daily TV news and media report).

3.1 Introduction

How to resist the spreading of disease and protect public health is one of major challenges in human society. To elucidate this puzzle, various mathematical dynamical processes, which classify the whole population into several compartments,
have been proposed to mimic the transmission pathway (Bailey et al., 1975; Anderson and May, 1982; Ewald, 1994; Moreno et al., 2003; Barthélémy et al., 2005). Recently, combining with the fast development of complex network theory (Boccaletti et al., 2006; Newman, 2003), spatial contact network is also used to investigate the spatial patterns of infectious disease spread from person to person (Bogúná and Pastor-Satorras, 2002; Wang et al., 2003; Gang et al., 2005; Bogúná et al., 2003a). A great number of literature has explored the impact of contact network properties, such as degree distribution (Pastor-Satorras and Vespignani, 2001) and cluster coefficient (Small and Tse, 2005; Bogúná et al., 2003a), on disease dynamics. However, these achievements are still not sufficient for eradicating the threat of disease, we still need to look for the protection measures.

During the past decades, a great number of protection scenarios (based on spatial populations) have been identified that can reduce the possibility of disease outbreak and its potential threat (Keeling et al., 2003; Chen et al., 2008b; Madar et al., 2004). Examples include targeted immunization (Wang et al., 2009; Pastor-Satorras and Vespignani, 2002b), acquaintance immunization (Cohen et al., 2003), ring immunization (Müller et al., 2000; Greenhalgh, 1986), meta-population diffusion and commuting (Colizza et al., 2007; Wang et al., 2013), and of course vaccination campaign (Perisic and Bauch, 2009b; Wu et al., 2011). Among these mechanisms, vaccine uptake, which maps the interplay between human behavior and epidemic spreading to the framework of game theory, has attracted the greatest attention (Perisic and Bauch, 2009b; Wu and Zhang, 2013; Fu et al., 2011). When a formal game theoretical analysis is incorporated into the conflict between group interest and self-interest, voluntary vaccination level is unlikely to exceed the group-optimal state, which thus results in a substantial increase of mortality and morbidity after the attack of disease (Bauch et al., 2003; Shim et al., 2009). More recently, provided that individual decision-making is related with the belief systems, it is found that the clusters of un-vaccination usually lead to a dramatic
increase in disease outbreak probability even if facing a rising vaccine rate (Ibuka et al., 2014; Bauch and Earn, 2004). Furthermore, when self-protection policy is considered as a feasible strategy within vaccination game, a counter-intuitive phenomenon takes place: the increasing of effectiveness of self-protection may decrease the system payoff (Zhang et al., 2013b). Other traits, which underlie behavior-disease feedback vaccine study and play a significant role in controlling the infection, involve the impact of zealots (Liu et al., 2012b), peer pressure (An, 2011), policy resistance (Poland et al., 2009), social learning (Bauch and Battacharyya, 2012; Mbah et al., 2012), social influence (Xia and Liu, 2013) and individual attitude (Yaqub et al., 2014; Kahn et al., 2003) (for comprehensive review refer to Ref. (Funk et al., 2010)).

While behavior response to vaccination campaign has been extensively studied, there has been relatively little systematic investigation into the impact of risk perception on the vaccination behavior and the spread of disease (Zhang et al., 2010; Kitchovitch and Liò, 2010). In previous reports, individual perceived infection risk equals to that of actual infection risk, where the information comes from the social or spatial neighborhood only (i.e., the so-called locally available information) (Funk et al., 2010; D’Onofrio et al., 2007; Fu et al., 2011; Coelho and Codeço, 2009). Examples are the spread of information via word of mouth, the evaluation of disease prevalence among acquaintances, daily contact of family members. However, except for local information, a neglected fact is that globally available information also plays a vital role in the assessment of perceived infection risk. This is particular evident for the outbreak of large-scale infection, where the information about the spread of disease is available to everyone (Buonomo et al., 2008; Min and Goh, 2013). Typical examples are any sort of news published by newspaper, TV stations, websites and other media channels. Thus, based on such a viewpoint, a natural question arises as to whether the vaccine coverage level and the eradication of infection becomes more beneficial if individuals base their
behavior on both locally available information and globally available information (i.e., the perception of infection risk is jointly affected by local social neighborhood and public information).

Aiming to systematically explore the impact of perceived risk on vaccination uptake, in this chapter we propose a new risk perception approach, which involves both locally and globally available infection information. Under such a scenario, the relationship between both resources of information is adjusted through a so-called precaution level $J$. $J = 0$ returns the traditional version, where just neighbors’ infection information is notified to agents, while $J > 0$ includes the information of both population and neighborhood. Compared with the traditional case (Zhang et al., 2010; Perisic and Bauch, 2009b,a), this simple mechanism (i.e., increasing $J$) can actually promote vaccination and inhibit the expansion of epidemics significantly, regardless of the contact networks and efficiency period of vaccination. We can see more details in the following sections.

### 3.2 Model and method of risk perception

For simplicity, we consider susceptible-infected-susceptible (SIS) epidemiological model to investigate the impact of risk perception on vaccination coverage level and epidemic size. Through out this work, agents are located on the nodes of the interaction networks and have contact only with their direct neighbors. In its basic version, the susceptible $S$ agent can be infected with probability $\beta$ if he contacts one infected ($I$) neighbor at each time step. Meanwhile, the infected individual can recover and return to the susceptible state again with the probability $\mu$. To assess the effect of risk perception conveniently, we fix $\mu = 1$ in this chapter. Moreover, we also define $\beta_{\text{perc}}$ the perceived infection rate to distinguish from the actual infection probability $\beta$. During the transmission process of epidemic, if one
susceptible agent has \( k_{inf} \) infected neighbors, then its total probability \( \lambda \) of being infected is

\[
\lambda = 1 - (1 - \beta)^{k_{inf}}.
\]  

(3.1)

However, this simple expression of the actual infection risk, as an increasing function of the fraction of infected people in the neighborhood, is not consistent with the individual perceived infection risk. Except for the neighbors’ information (i.e., locally available information), public social information also plays a crucial role in the evaluation of infectivity. Along this line, susceptible agent’s perceived risk of infection during the contact with infected individual could be written in the following format

\[
\beta_{perc} = \beta \times R(k_{inf}, N_I),
\]  

(3.2)

where \( R(k_{inf}, N_I) \) is the so-called risk perception or alertness of individual (Bagnoli et al., 2003; Kitchovitch and Liò, 2010), which mainly comes from two aspects: (i) global awareness of risk (such as, broadcasting media news and reports), as shown by the total number \( N_I \) of infected agents in the population; (ii) local perception based on the information of infected neighbors. This is actually consistent with the realistic examples in our life. For example, during the outbreak of epidemic, people in general obtain the infection information through public media report and local mouth-to-mouth communication. Through integrating all the information, they can give an estimation for the potential risk and then take the action of decreasing economic cost. Based on these facts, we thus suppose

\[
R(k_{inf}, N_{inf}) = \exp[J \left( \frac{k_{inf}}{k} \right)^\alpha H],
\]  

(3.3)

\[
= \exp[J \left( \frac{k_{inf}}{k} \right)^\alpha \left( \frac{N_I}{N^*} \right)],
\]  

(3.4)

where the infection risk is evaluated in the exponential form, because the realistic disease risk exponentially decays. In addition, since local information belongs
to one part of global information, we write the risk evaluation in the proportion form. With respect to parameters, $J$ (a non-negative parameter) denotes the level of precaution, $\alpha$ is private perception alarm based on the infected neighbors, $k$ represents the number of neighbors of that given agent (i.e., the degree of the given node), and $H$ corresponds to the fraction between total infected number $N_I$ and the size of population $N$. It is worth pointing out that due to more uncertain factors among neighborhood, the local information is expressed in the power form. And the precaution level $J$ makes the infection risk more sensitive to change of local information which is consistent with realistic case: local information usually changes due to re-infection and recovery of neighbor nodes. Evidently, $J = 0$ returns the previous reports (Zhang et al., 2010; Perisic and Bauch, 2009b,a), where the perceived infection risk equals to the actual case, namely, neglecting the influence of population information. For $J > 0$ (namely, increasing population’s precaution level), agents becomes more sensitive for the outbreak of any infection, which may not takes places among his neighborhood. After clarifying the infection rate $\beta_{perc}$, it is easy to obtain the total perceived probability of being infected

$$\lambda_{perc} = 1 - (1 - \beta_{perc})^{k_{inf}}. \tag{3.5}$$

Based on this perceived risk, agent can decide whether he needs to take vaccination or not before any infection. To get vaccinated will incur a cost $C_V$ to individual, which may correlate to the immediate expenditure and other related health side-effects. To be simple, we assume that the vaccine is risk-free and provides full protection against infection. On the contrary, if agent does not take vaccination, he can perceive his infection probability as described in Eq. (3.5). While in reality, once one individual caught the disease, he needs to pay an infection cost $C_{I-N}$, which usually involves the disease complications, the expenses for treatment and the economic loss due to the absence of work. In this sense, we can get the expected
costs of vaccination \( P_V \) and non-vaccination \( P_N \) as follows:

\[
P_V = C_V, \quad (3.6)
\]

\[
P_N = C_{I-N} \times \lambda_{perc}. \quad (3.7)
\]

Without loss of generality, we choose \( C_{I-N} = 1 \) and let \( c = C_V/C_{I-N} \) denote the relative cost of vaccination, which is restricted into the region \([0,1]\) by assuming \( 0 \leq C_V \leq C_{I-N} \). Thus, the above cost functions can be revised as

\[
P_V = c, \quad (3.8)
\]

\[
P_N = \lambda_{perc}. \quad (3.9)
\]

From the perspective of population interest, the optimal state is maximizing the vaccination coverage level, which can eradicate the threat of disease. On the other hand, individual always faces the temptation of low cost (namely, elevate personal interest), similar to the setup of prisoner’s dilemma (Zimmermann and Eguíluz, 2005; Szabó and Hauert, 2002). To implement individual decision-making process, we assume that agent will choose the strategy with lower cost. Obviously, if

\[
P_N > P_V, \quad (3.10)
\]

vaccination is personal choice; otherwise agent does not take vaccination. In each time step (corresponding to one disease season), each node has one chance to synchronously update the strategy. Moreover, in realistic life, the disease contact networks are usually heterogeneous (namely, different person or node has different number of neighbors) (Salathé et al., 2010). To mimic this character, we consider Barabási-Albert (BA) scale-free and Erdős-Rényi (ER) networks with average degree \( < k > = 6 \) and the size \( N = 2000 \) (Barabási and Albert, 1999; Erdős and
Rényi, 1959). Since the heterogeneity of contact networks may introduce additional disturbances, final results are averaged over up to 50 independent runs for each set of parameter values in order to assure suitable accuracy. Before each realization, 10 infected agents, as the seeds, are randomly assigned to the whole population.

According to the efficiency of the vaccination, vaccination can usually be divided into permanent case and temporary case. For example, the vaccines of these following diseases (smallpox, measles, parotitis and varicella) are always effective to resist the invasion of infection. However, with respect to the diseases like seasonal influenza and hepatitis B, the vaccine can only play a role in reducing the infection for a limited seasons. Based on these facts, we will consider the impact of risk perception on permanent vaccination and temporary vaccination in what follows.

3.3 Results and analysis

3.3.1 Permanent vaccination campaign

To begin with, we explore the impact of risk perception on permanent vaccination case. Figure 3.1 features how the number of infected and vaccinated agents (i.e., $N_I$ and $N_V$) vary as a function of time for different values of precaution level $J$. Obviously, $J = 0$ recovers the previous setup (Zhang et al., 2010; Perisic and Bauch, 2009b,a), where the information of perceived infection risk only comes form the local neighborhood. We can observe that, after a peak of infection outbreak, the disease still prevails for long time. However, with the increment of $J$ (namely, individual decision-making or perceived infection risk involves both local and global information), individual enthusiasm of vaccination is greatly enhanced.
Figure 3.1: The time courses of number of infected agents $N_I$ (a), number of vaccinated agents $N_V$ (b) and the average perceived risk $\langle \beta_{perc} \rangle$ (c) for different precaution level $J$ on the BA networks. It is obvious that the increment of $J$ is beneficial for preventing the spreading of disease. Depicted results are obtained for $c = 0.8$, $\beta = 0.2$ and $\alpha = 1.0$.

The larger the value of $J$, more evident the peak of the number of vaccinated agents $N_V$. As a result, the overall number of infection will be effectively reduced: the peak of infection shrinks and disease is completely eradicated in the early steps.

In addition to $N_I$ and $N_V$, it is interesting to examine how the average perceived infection risk $\langle \beta_{perc} \rangle$ changes. To answer this question, Fig. 3.1(c) shows the time course of the average perceived infection risk of the whole population. Compared with the horizontal line of $J = 0$, we can see that the larger value of $J$ can lead to higher peak of average perceived risk $\langle \beta_{perc} \rangle$ at earlier step, which means agents’ decision about vaccination becomes more sensitive for any infection in the population. Thus, it is now easy to understand the fact that the
fast rise of average perceived risk inspires the high tide of vaccination, which in turn suppresses the spreading of infection.

Now that risk perception possess an active effect on reducing infection, it is instructive to examine whether this scenario is general on different contact networks. Figure 3.2 shows the time courses of infected number $N_I$, vaccinated number $N_V$ and the average perceived risk $<\beta_{perc}>$ for different $J$ on ER network. Importantly, qualitatively identical results can be obtained. It is shown that larger values of $J$ inspire individuals becoming more sensitive for the existence of any infection, which can be revealed by the faster increase of $<\beta_{perc}>$. As the response to high
perceived risk, the likelihood of choosing vaccination sharply increases, which in turn restrains and even eradicates the initially fast outspread of disease. With further elimination of infected agents, average infection risk becomes small again, which induces the decline of vaccination. Together with observation in Fig. 3.1, these results suggest that risk perception motivates a positive feedback mechanism (irrespective of contact networks), which is beneficial for promoting vaccination and guaranteeing public health.

Based on these observations, it becomes of utmost significance to elucidate more detailed promotive effect of precaution level $J$. For this purpose, we mainly focus on the case of BA networks. Before the formal analysis, we define a new parameter, vaccination inclination $\rho_k$, for nodes with degree $k$ (Zhang et al., 2010) (since the decision of an individual is influenced by its $k$ neighbors),

$$\rho_k = \frac{V_k}{N_k},$$

where $N_k$ denotes the number of nodes with degree $k$ and $V_k$ represents the number of vaccinated individuals with degree $k$ over the whole time range. Figure 3.3 features the dependence of the vaccination inclination $\rho_k$ on degree $k$ for different values of $J$. For the traditional version (i.e., $J = 0$), the vaccination inclination is zero for small-degree nodes, then it climbs nearly linearly with $k$, and only saturates at the large-degree nodes, since the infection probability increases with $k$. As is well-known, small-degree nodes usually hold majority percentage within the framework of BA networks, thus these unvaccinated nodes are more vulnerable to the attacks of infection. However, with enhancing $J$, the changes take place. One can see that, except for large-degree nodes, the agents holding small-degree nodes become more inclined to take vaccination. In fact, the larger the value of $J$, the higher the vaccination inclination for small-degree nodes. This is actually in agreement with what we would expect, given that individuals become more
sensitive for any infection. Moreover, we can also get the quantitative support for the elevation of vaccination inclination on small-degree nodes. Initially, a limited number of infected agents are located in the population randomly. If there exist(s) infected agent(s) among the neighborhood of small-degree node, its infection density (i.e., $\frac{k_{inf}}{k}$) will be a very large value, compared to the infection density of whole population (i.e., $\frac{N}{N_I}$). According to Eq. (3.3), the risk perception $R(k_{inf}, N_I)$ obtains a value much larger than 1 (i.e., $R \gg 1.0$). Substituting this value into Eq. (3.2), the perceived infection risk $< \beta_{perc} >$ remarkably enlarges, which naturally excites the vaccination inclination for small-degree nodes. Thus, self-vaccination enthusiasm of small-degree nodes play a crucial role in avoiding the spreading of infection.

In order to further validate the role of small-degree nodes, it is next instructive to monitor how the vaccination dynamics varies under specially prepared situation. Motivated by the observation of Fig. 3.3, we divide individuals into two types based on a threshold $k_{th}$, which herein is a small value. If the degree of nodes is less than the threshold, then agents can still obtain the infection information through
both local and global interaction (namely, for small-degree nodes the perceived infection risk $\beta_{\text{perc}}$ holds as Eq. (3.2)). On the contrary, the individuals with degree above the threshold can only get the information from their neighborhood, which recovers the traditional version for this type of agents (i.e., $\beta_{\text{perc}} = \beta$). Figure 3.4 exhibits the evolution of the number of infected agents $N_I$, the number of vaccinated agents $N_V$ and average perceived infection risk $< \beta_{\text{perc}} >$. Similarly as in Fig. 3.1, it can be observed that even if only small-degree agents can receive the infection information from population, $\beta_{\text{perc}}$ will fast reach a peak with the increment of $J$. In this sense, these small-degree nodes are more inclined to taking vaccination, which is reflected by the enhanced vaccination number $N_V$. With higher value of $J$, the spreading of epidemic can be controlled more effectively. As such, the promotion of vaccination uptake level can be attributed to positive feedback effect excited by the small-degree nodes. Along this discovery, it is also easy to understand why the elimination of disease is more evident on BA network than ER network. This is because BA network possesses more small-degree nodes, which naturally leads to smaller $N_I$, larger $N_V$ and larger $< \beta_{\text{perc}} >$.

Subsequently, we proceed with examining how actual infection risk $\beta$ affects the update of vaccination. It is usually expected that the elevation of $\beta$ can monotonously activate individual vaccination inclination, which, similar to previous treatment Zhang et al. (2010), can be represented by the total vaccination within the time span $t \in [0, 200]$. Different from the intuition, Fig. 3.5 shows a nonlinear relationship between the vaccination sum and actual infection risk $\beta$. It is clear that vaccination uptake has an optimal level but at the same $\beta$ drops to a low value, irrespective of the values of $J$. While for the enhanced $J$, it not only elevates the maximal number of vaccination, but also makes the peak occur at smaller $\beta$. For these observations, we can provide the mathematical explanation with $c = 0.8$ as an example. We first repeat the traditional case ($J = 0$). For small $\beta$, few people choose vaccination due to high cost $c$ and low infection risk. As $\beta$ increases to the
Figure 3.4: The time courses of number of infected agents $N_I$ (a), number of vaccinated agents $N_V$ (b) and the average perceived risk $< \beta_{perc} >$ (c) for different precaution level $J$ on the BA networks. To reveal the role of small-degree nodes, the total population is divided into types of agents according to a threshold $k_{th}$. Obvious that the increment of $J$ is still beneficial for reducing the spreading of disease. Depicted results are obtained for $k_{th} = 10$, $c = 0.8$, $\beta = 0.2$ and $\alpha = 1.0$.

threshold $\beta_{th}$ (herein $\beta_{th} = 0.8$), the vaccination inclination quickly increases, the infection is effectively controlled and there remain a limited number of infected agents. Particularly, when $\beta$ reaches the threshold, the cost of non-vaccination

$$P_N = 1 - (1 - \beta_{perc})^{k_{inf}} = 1 - (1 - \beta_{perc}) = \beta_{perc} = \beta$$  \hspace{1cm} (3.12)

for agents having only one infected neighbor is not larger than $c = 0.8$, which naturally causes vaccination dropping to a very low level. However, when the precaution level $J$ is considered, the perceived infection risk $\beta_{perc}$ increases faster than the actual infection risk (i.e., $\beta_{perc} > \beta$), which in turn accelerates vaccination
enthusiasm and the emergence of peak. As observed, the larger the value of \( J \), the earlier the arising of vaccination uptake peak. This point can also be proved through Eqs. (3.2) and (3.3): \( \beta_{\text{perc}} \) is enhanced with larger \( J \), thus the cost of non-vaccination \( P_N \) becomes larger, which is beneficial for promoting vaccination according to Eq. (3.10). However, we notice that though the enhanced \( J \) inspires larger vaccination peak, it also makes the incline of vaccination take place at earlier stage. This is because, after the peak, majority of agents have eliminated the infected neighbor(s) (i.e., \( k_{\text{inf}} = 0 \) making \( P_N = 0 \)). In this case, the cost of non-vaccination is smaller for them (i.e., \( P_N < P_V \)), which leads to the stimulation of vaccination descending. Besides, there exists small percentage of agents who still have the infected neighbor(s) (namely, the earliest infection seeds). With further increment of \( \beta \), these remaining infected individuals will be isolated by the vaccinated neighbors. Under this case, the average perceived infection risk \( < \beta_{\text{perc}} > \) of the whole population will approach to actual value \( \beta \) more closely. Finally, at the threshold \( \beta_{\text{th}} \), only few individuals possess one infected neighbor.
Figure 3.6: The time courses of number of infected agents $N_I$ (a), number of vaccinated agents $N_V$ (b) and the average perceived risk $<\beta_{perc}>$ (c) for different perception alarm $\alpha$ on the BA networks. It is obvious that the increment of $\alpha$ decrease the vaccination enthusiasm and is not beneficial for the control of disease. Depicted results are obtained for $c=0.8$, $\beta=0.2$ and $J=1.0$.

and at the same time have perceived infection risk larger than actual infection value ($\beta_{perc} > \beta$ for most remaining individuals, who do not need to take any action). For those people possessing one infected neighbor, their non-vaccination cost always satisfies

$$P_N = 1 - (1 - \beta_{perc})^k_{inf} = 1 - (1 - \beta_{perc}) = \beta_{perc} > c,$$

therefore only these limited people certainly choose vaccination. Actually, even for the traditional version ($J=0$), these agents having one infected neighbor are also apt to vaccination due to $\beta > c$ after $\beta_{th}$. This is the reason why the number of agents choosing vaccination is identical after the threshold in Fig. 3.5.
Finally, an interesting remaining question concerns how the private perception alarm $\alpha$ impacts the vaccination level. According to Eq. (3.3), one can see that enhanced $\alpha$ will make $R(k_{inf}, N_{inf})$ and $\beta_{perc}$ sharply decrease, which at last reduce the cost of non-vaccination $P_N$. In this way, individual desire of vaccination is rebated. Figure 3.6 shows the time courses of number of infected, vaccinated agents ($N_I, N_V$) and the average perceived infection risk $< \beta_{perc} >$ for different $\alpha$, which is identical with our expectation. Compared to the case of $\alpha = 0$, the trend of average risk $< \beta_{perc} >$ differs and it even has a valley, which means that agents become less sensitive to infection even located in their neighborhoods. Naturally, the peak of vaccination is inhibited and epidemic can last for longer time.

### 3.3.2 Temporary vaccination campaign

In this subsection, we mainly turn our attention to the impact of risk perception on temporary vaccination uptake. We will consider two types of vaccination efficiency period. The first case is homogeneous efficiency period (namely, vaccination will lose its validity after a fixed time $\delta$). In this case, agents need to decide whether to take vaccination after this time interval. Figure 3.7 shows its impact on the number of infected, vaccinated agents ($N_I, N_V$) and the average perceived infection risk $< \beta_{perc} >$. Similar to observation of permanent vaccination, we can see that enhanced $J$ is beneficial for the vaccination uptake and control of infection, which means that the risk perception mechanism is universally effective in eradicating the spreading of epidemics. But, except for these, we can see that there exist oscillations. Obviously, the larger the value of $J$, the larger the amplitude of the first vaccination peak. After this largest peak, the trend changes: enhanced $J$ makes amplitude of the oscillations become smaller and smaller and finally enables reaching the stationary state faster.
Figure 3.7: The time courses of number of infected agents $N_I$ (a), number of vaccinated agents $N_V$ (b) and the average perceived risk $\langle \beta_{perc} \rangle$ (c) for different perception alarm $\alpha$ under homogeneous efficacy period $\delta$. Similar to Fig. 3.1, the increment of $J$ is beneficial for the control of disease. Besides, larger $J$ induces larger amplitude of first vaccination peak, but makes the subsequent oscillations smaller and reaches the stable state earlier. Depicted results are obtained for $c = 0.7$, $\beta = 0.2$, $\delta = 20$ and $\alpha = 1.0$ on the BA networks.

For these oscillations (which is caused by the efficiency period), we can provide the following explanation. At the beginning, larger $J$ will greatly magnify perceived infection risk, which can also be validated by first peak of $\beta_{perc}$. Correspondingly, the positive feedback mechanism leads to higher vaccination enthusiasm and less infection. However, after the efficiency period $\delta$, vaccine loses its function for certain individuals. Subsequently, a new round of disease outbreak starts, which results in next peak of vaccination. Because some people still possess the efficient vaccination, the infection becomes less severe. In this line, the amplitude of oscillations thus becomes smaller and smaller. Moreover, since the accumulative number of vaccinated agents is larger for enhanced $J$, it is easy to understand why
Figure 3.8: The time courses of number of infected agents $N_I$ (a), number of vaccinated agents $N_V$ (b) and the average perceived risk $<\beta_{\text{perc}}>$ (c) for different perception alarm $\alpha$ under heterogeneous efficacy period $\delta_i$ (herein $\delta_i = k_i$). The evolution trend is qualitatively similar to the homogeneous case. Depicted results are obtained for $c = 0.7$, $\beta = 0.2$ and $\alpha = 1.0$ on the BA networks.

the amplitude of oscillations decrease and reach the stationary state faster.

Then, we focus on the second case: heterogeneous efficiency period (namely, the effective period of vaccination is different for each agent). To avoid new parameter, we model that individual vaccination time period equals to its degree (i.e., $\delta_i = k_i$), which agree with the empirical observation that strongly resistant agents usually have more connection and thus longer vaccination time intervals (Zhang et al., 2010). Interestingly, quantitatively identical result can be obtained from Figure 3.8. As the case of homogeneous period, it can be observed that larger $J$ induces larger peak of vaccination at first, but the amplitude of oscillations trends to be smaller and they reach the stable state earlier. Thus, regardless of the efficiency period of vaccination, risk perception setup is beneficial for promoting
vaccination.

### 3.4 Summary and discussion

During the vaccination campaign, individual vaccination decision is closely related with perceived infection risk. Different from traditional setup, we incorporate both locally and globally available information into the risk perception, which is reflected via the so-called precaution level $J$. We have found that the enhanced precaution level is greatly beneficial for promoting vaccination level of population, irrespective of contact networks and efficiency period of vaccination. The essence of this comforting discovery can be attributed to a positive feedback mechanism motivated by small-degree nodes. Since these nodes are more sensitive to the infection among their neighborhoods, their vaccination inclination is larger. Importantly, this point can be validated via simple mathematic analysis. Moreover, we have also unveiled that a medium actual infection risk can guarantee the optimal vaccination enthusiasm. The larger the value of $J$, more obvious this trend. This is because, for majority of nodes, elevated $J$ enables the cost of non-vaccination to exceed the requirement of vaccination quickly. With respect to the influence of private perception alarm $\alpha$, because it makes the agents become less sensitive to the change of local information, it is not beneficial for promoting vaccination and preventing disease spreading.

Notably, the theoretical scenario of risk perception we have considered here seem to be reasonable and are in fact easily justifiable with realistic examples. We hope that this work will inspire future studies, especially in terms of understanding more realistic evaluation framework based on the empirical data and some co-evolution processes (Perc and Szolnoki, 2010).
Chapter 4

Impact of temporary self-protection measure on disease spreading

Infectious disease can usually bring tremendous harm to human beings, irrespective of physical hurt or psychological injury. Thus, prevention measures seem necessary to reduce and even eradicate the harm. In the last chapter, I have showed the impact of risk perception on the vaccination. However, in some emergency situations, effective vaccines may be not sufficient or people do not want to spend too much monetary cost for the prevention of disease. Under this case, temporary self-protection measures, such as wearing facemask and frequently washing hands, can mitigate the propagation of disease. Inspired by this fact, I will explore the impact of self-protection measure on the eradication of disease in the framework of game theory in this chapter.
4.1 Importance of self-protection measure for the defence of disease

Historically, diseases propagation has been a great threat to human society, leading to enormous morbidity and mortality. According to the statistical outcome, thousands of people have died annually, and in the future many persons still continue to suffer the pain and economic expense caused by the infectious disease (Dols et al., 1977; Organization et al., 2008). Based on these facts, researchers try to understand the intrinsic transmission and control traits of the infectious diseases. Recent progress showed that the compartment model (such as, SIS, SIR) in spatial populations is very useful framework, namely, the so-called spatial epidemiology (Boccaletti et al., 2006; Anderson et al., 1992; Anderson and May, 1991; Blower, 2008; Anderson et al., 1979). Under this framework, the voluntary vaccination has been shown to be a very useful measure of guaranteeing the public health (Bauch and Earn, 2004; Funk et al., 2010; Fu et al., 2011; Bauch et al., 2003).

With the voluntary vaccination program, vaccination level can get fast increase to form the herd immunity, which prevents the further propagation of disease (Wu et al., 2011; Cardillo et al., 2013a; Liu et al., 2012a). However, the high immunity level subsequently makes some people abandon the vaccination selection due to the temptation of non-cost free rider behavior (Bauch et al., 2010; Ibuka et al., 2014); and in turn amplifies the infection risk of disease. In this sense, it forms a long-standing dilemma. While during the vaccination uptake, the individual decisions with respect to vaccination are usually affected by many factors, such as, the perceived infection risk, cost of infection and vaccination, the vaccination situation around yourself, and even the religion belief. So, by introducing some special mechanisms, we can get some new understanding for the role of vaccination. We can examine some typical examples. In a recent research (Mbah et al., 2012),
if individuals decide the vaccination uptake based on the self-interest principle, the impact of vaccination on superspreaders could be depressed or counteracted. Borrowing the social impact theory, the authors tried to characterize the impact of social influence on individual interaction relationship, they unveiled that individual high conformity to social influence could enhance vaccination coverage (Xia and Liu, 2013). In a recent survey study (Shim et al., 2012a), the authors showed that altruism played a crucial role in the influenza vaccination decisions, and it could shift vaccination decision from individual self-interest to collective optimum, which is helpful for reducing the infection risk. More specially, if a tiny fraction of agents always held the vaccination selection, these committed ones would lead to the promotion of vaccination uptake (Liu et al., 2012b).

Although a great number of reports about the effect of vaccination campaign have been accumulated, vast majority of them simply assume that individuals can take always two alternative actions: taking vaccination or taking risks (Bauch and Galvani, 2013; Fu et al., 2011; Zhang et al., 2011a; Xia and Liu, 2013). This means that completely effective vaccine is sufficient to the population at any time, which is obviously inconsistent with the realistic cases of our life, especially for the severe diseases. In this sense, a practically relevant strategy is completely neglected, this is the individual self-protection measures. In reality, once there exists the mass outbreak of dangerous disease (such as, H1N1, SARS), the supply of vaccine usually can not satisfy the demand of cure and the side effect of the vaccines can not be unverified in time. In this case, individual self-protection strategy (such as reducing exposure and contact, wearing face masks, washing hands) becomes of utmost significance, which can provide the temporary prevention from infection and lower cost than the vaccine. However, the efficiency of the self-protection strategy is also limited. If individual can escape from the infection under this policy, his cost will be very small. At variance, provided that this action fails to protect the agent (namely, agent still gets the infection after self-protection action),
his cost will be a bit more. Thus, this new policy composes a new dilemma by itself. Motivated by these interesting facts, one question naturally poses itself, which we would study in this chapter. That is, if we can consider the temporary self-protection strategy during the disease season (when the vaccine is not sufficient), is it really beneficial for the prevention of disease?

Aiming to answer the above question, we consider an evolutionary epidemiological framework to explore the impact of temporary self-protection measure on the system cost and epidemic size. Different from the previous chapter where vaccination is the principal measure on the network, temporary self-protection measures, such as, wearing facemasks, washing hands frequently, taking pharmaceutical drugs, and avoiding contact with sick people, are considered as the main strategies, which seems particular reasonable before obtaining enough vaccine (Stöhr and Esveld, 2004). On the other hand, self-protection measure is also suitable for another case that people initially do not want to spend too much monetary cost for the prevention of disease infection. Thus, there are three usable strategies: temporary self-protection, taking vaccination and taking risk, and there also exist competitions between these strategies. Before the spreading of disease, agent can estimate the expected cost of three different strategies; and then chooses the strategy that makes his own loss minimal (similar to Chapter 3). For this proposal, there exist two new parameters: cost of self-protection strategy and its efficiency. Subsequently, we will mainly examine how these two parameters affect the control of disease.
4.2 Model and method of self-protection measure

Following the foregoing chapters, we still consider the susceptible-infected-susceptible (SIS) epidemiological model to depict the spreading of dynamics of disease. Agents are divided into classes: susceptible and infected. When susceptible agent has the touch or connection with one infected individual, he will be infected with the probability $\beta$. An infected agent can also come back to the susceptible state with the recovery rate $\mu$. Without loss of generality, we fix $\mu = 0.25$ in this chapter (for other values of $\mu$, the qualitative results are also observed). Thus, when agents are located on the spatial contact networks, the total probability of being infected for one susceptible agent is

$$\lambda = 1 - (1 - \beta)^{k_{inf}}. \quad (4.1)$$

where $k_{inf}$ denotes the number of infected neighbors of the given agent. To decrease the number of related parameters, we assume that each individual has complete recognition for the risk of infection, i.e., $\beta_{perc} = \beta$ and $\lambda_{perc} = \lambda$. Here it is worth mentioning that if we evaluate the perceived risk $\beta_{perc}$ as the form of last chapter, the qualitative results do not change. Moreover, another reason of selecting the above simple assumption is that we mainly focus on the impact of self-protection measure (rather than risk perception) in this chapter. Similar to the treatment of last chapter, if we assume that $C_r$ is the cost of taking risk due to the infection, the expected cost for taking risk strategy could be written as

$$P_r = C_r \times \lambda = C_r \times [1 - (1 - \beta)^{k_{inf}}]. \quad (4.2)$$
Correspondingly, if the agent plans to take vaccination, he needs to spend the cost of vaccine

\[ P_v = C_v, \]  

(4.3)

which is assumed to incorporate the time needed to be vaccinated and the abrupt accident of vaccine. Except for the above setups, another new measure under consideration is self-protection strategy, which needs a relatively low cost but provides only limited protection from infection. The cost function of temporary self-protection policy is described as

\[ P_s = C_s + (1 - \delta) \times C_r \times [1 - (1 - \beta)^{k_{inf}}]. \]  

(4.4)

where \( C_s \) is the cost of temporary self-protection strategy \((C_s \leq C_v)\), \( \delta \) is the efficiency of such an action and the second term of this equation corresponds to the cost of infection once the self-protection measure fails. Obviously, \( \delta = 1 \) means that the low-cost protection strategy is completely effective; \( \delta = 0 \) denotes the case of complete inefficiency; \( 0 < \delta < 1 \) is an in-between condition.

To further reduce the number of independent parameters, we fix \( C_r = 1 \) and use \( c = C_v/C_r \) and \( g = C_s/C_r \) to denote the relative cost of vaccination and temporary self-protection \((g \leq c)\). In this way, the cost function for the three strategies can be translated into

\[ P_r = 1 - (1 - \beta)^{k_{inf}}, \]  

(4.5)

\[ P_v = c, \]  

(4.6)

and

\[ P_s = g + (1 - \delta) \times [1 - (1 - \beta)^{k_{inf}}]. \]  

(4.7)

After estimating the expected cost of various strategies, individuals need to make decisions about the possible strategy at each step. Following the previous chapter,
we still assume that agents are perfect rational and they only choose the strategy that make their own cost minimal. That means, individual $i$ will choose the strategy $s_i$ as
\[
s_i = \min\{P_r, P_v, P_s\}.
\] (4.8)

In each time step (corresponding to one disease season), each node has one chance to synchronously update the strategy.

As for the interaction contact networks, we use either a regular $L \times L$ square lattice with periodic boundary condition, the Erdős-Rényi random network (ER) (Erdős and Rényi, 1959) or the scale-free network with $N$ nodes and an average degree of six generated via the Barabási-Albert (BA) algorithm (Barabási and Albert, 1999). Before the infection, we randomly put 0.4% agents of the total population as the infection seeds. Because the efficiency of self-protection policy and heterogeneous contact networks may introduce more stochastic factors into the system, our final results are averaged over 100 independent realizations for each set of parameter values. In this chapter, we focus on two key quantities. One is disease size $f_I$ equalling the fraction of infected agents; the other is individual average cost $< C >$, which can be regarded as the economic indication brought by the prevention measures.

### 4.3 Results and analysis

#### 4.3.1 Impact of self-protection efficiency

To begin with, we explore the impact of the temporary self-protection policy on the eradication of disease. Figure 4.1 shows the disease size $f_I$ (or the fraction of infected individuals) and the individual average cost $< C >$ as a function of the efficiency parameter $\delta$. From Eq. (4.7), it is obvious that $\delta = 0$ (namely, the
Figure 4.1: (a) The disease size or the fraction of infected individuals $f_I$; (b) individual average cost $< C >$ as functions of the efficiency $\delta$ of self-protection policy for different values of self-protection cost $g$ on BA network. It is seen that, too low or too large $\delta$ induces fewer agents to be infected, and the individual average cost is also low. However, for intermediate efficiency, the epidemic size is enlarged, which results in larger mean cost. Depicted results are obtained for $c = 0.5$, $\beta = 0.18$ and $N = 2000$.

complete inefficiency) makes the cost of self-protection strategy highest. Namely, nobody chooses this strategy but epidemic size can be controlled mainly due to the protection of vaccination. At the same time, the individual average cost will be maintained on a low level. However, one can see that, with the increment of self-protection efficiency $\delta$, the outbreak of epidemic will become severe. At middle value of $\delta$, the infected number reaches a peak. In this regime, self-protection strategy holds the dominant position within the population. However, this policy can not provide the absolute guarantee to escape from the infection risk. Even if one person takes the temporary protection policy, he could still be infected, which can be further validated by the enhanced average cost in Fig. 4.1(b). Finally, when the self-protection is sufficiently effective (i.e., large $\delta$), the infection risk will decline again. Here, self-protection becomes the primary option due to high efficiency. While for the individual average cost $< C >$, it drops to a low level as well. Thus, the self-protection measure leads to a multiple effect, middle efficiency makes the competition of strategies be more beneficial for self-protection strategy, which but conversely induces more infection and higher total expense for disease. Moreover, it is also worth mentioning that this phenomenon, is robust against
Figure 4.2: (a) The disease size or the fraction of infected individuals $f_I$; (b) individual average cost $\langle C \rangle$ as a function of the efficiency $\delta$ of self-protection policy for different values of self-protection cost $g$ on ER network. Interestingly, qualitatively identical trend with Fig. 4.1 can be observed: self-protection policy induces a multiple effect. Contrary with the sufficient protection at low or high efficiency, the intermediate efficiency leads to the great expansion of infection. Depicted results are obtained for $c = 0.5$, $\beta = 0.18$ and $N = 2000$.

the cost of self-protection. With increment of self-protection cost $g$, the existence of infection and cost peak usually needs higher efficiency value. For all these observations, we will provide detailed explanations in what follows.

Along this line, it is meaningful to check how these observations vary under different contact networks. Importantly, qualitatively identical results are unveiled on ER network and square lattice. Fig. 4.2 and Fig. 4.3 depict the epidemic size $f_I$ and individual average cost $\langle C \rangle$ on these topologies for different values of efficiency $\delta$. Similar to Fig. 4.1, it can be observed that the intermediate efficacy of self-protection policy causes the outbreak of disease and leads to higher cost. While too low or too high efficiency is beneficial for the elimination of infection. Moreover, larger cost of self-protection makes the peak of disease outbreak at larger efficiency, which is in agreement with the phenomenon of BA network as well. Thus, all these observations validate the robustness of the multiple effects of self-protection measure: due to competition with vaccination, middle efficiency of self-protection measure can not provide enough protection for public health, which results in higher social expense. While low or high efficiency drives people to take
vaccination or self-protection as main choice, which eliminates the spreading of epidemic.

To better understand this multiple effect, we feature the characteristic spatial distributions of various states for different values of efficiency $\delta$ in Fig. 4.4. For small $\delta$ value, it is obvious that the sufficient prevalence of vaccination uptake (green nodes) can produce herd immunity and provide sufficient guarantee for public health. At the same time, we also observe that just limited agents choose self-protection policy and finally these agents are still infected due to low efficiency. However, with the increment of $\delta$, the fraction of choosing temporary self-protection policy greatly increases due to the temptation of possible low cost. Correspondingly, the number of individuals choosing vaccination sharply decline. In such a circumstance, the agents of taking risk can not get enough protection and isolation from the infected agents. As a result, an outbreak of disease takes place: many agents of taking risk and self-protection strategies are infected. It is worth mentioning that these infected agents are inclined to form large clusters. With further increasing $\delta$, one can see that, because of low cost and high efficiency, the enthusiasm of self-protection is elevated to large extent. Though small fraction of agents are still infected, the high efficiency of self-protection still guarantee the

![Figure 4.3](image_url)
Figure 4.4: Characteristic distribution of different states for different values of efficiency $\delta$ on square lattice. The color codes are: vaccination (green), taking risk and uninfected (light gray), taking risk and infected (blue), self-protection and uninfected (red), self-protection and infected (black). From (a) to (c), the values of $\delta$ are 0.3, 0.6, and 0.9, respectively. Compared with the low and high efficiency, intermediate $\delta$ induce the infected agents forming larger clusters. Depicted results are obtained for $c = 0.5$, $g = 0.2$, $\beta = 0.18$ and $L = 100$.

Formation of herd immunity. Thus, even if more free-riders exist, the infection risk is controlled. These visual observations provide insight into the multiple effect of self-protection policy.

Next, we provide quantitative characterization to understand why an intermediate efficiency value causes a larger infection. For this purpose, we mainly consider BA network as the contact network in the following. Figure 4.5 shows the evolution courses for the fraction of taking vaccination, self-protection measure, individual average cost and epidemic size with different values of $\delta$. For low efficiency ($\delta = 0.3$), one can see that nobody chooses the self-protection policy, and the system returns back to the traditional two-strategy case (vaccination or not) (Zhang et al., 2010; Bauch and Earn, 2004; Bauch et al., 2003). To restrain the outbreak of disease, vaccination becomes the first option and infection is finally controlled because of the existing positive feedback mechanism. For the intermediate value $\delta = 0.6$, the fraction of self-protection is greatly improved and reaches a high level. At the same time, the vaccination level is remarkably reduced. However, due to the limited effectiveness of self-protection, the epidemic size and individual average cost are elevated to high level. Here it is specially evident that the positive feedback mechanism between individual strategies and disease size vanishes (namely, the
Figure 4.5: The time courses for the fraction of self-protection (a); the fraction of taking vaccination (b); the disease size or the faction of infected agents $f_I$ (c); and average cost $< C >$ (d) with different values of $\delta$ on the BA networks. Compared with the case of too low or too high $\delta$, intermediate efficiency induces more people to take the strategy of self-protection, but also destroys the feedback mechanism of vaccination, which brings high risk of infection. Depicted results are obtained for $c = 0.7$, $g = 0.3$, $\beta = 0.18$ and $N = 2000$. (To show the evolution trend more obviously, we just record $10^3$ time steps, which do not affect the final results.)

peak of self-protection or vaccination can not lead to the reduce of infection).

Finally, with high efficiency of self-protection of measure, people lose interest for vaccination due to high cost. On the contrary, good effectiveness and lower cost induce the fraction of self-protection to reach a peak, which in turn decreases the infection risk. Thus, we argue that too low or too high efficiency can guarantee a positive feedback mechanism, which is beneficial for the decline of disease. Middle efficiency can make this key mechanism die out.

To further elucidate the multiple effect, we monitor how strategies distribute by degree classes. Figure 4.6 presents the distribution of three strategies at the steady state for different degree classes on BA networks for different values of $\delta$. For low
efficiency (e.g., $\delta = 0.3$), it is clear that nobody takes the self-protection strategy, as shown in the above figures, because the low efficiency may bring higher expense in case the person is infected after the self-protection measure. Interestingly, in this case, the middle- and large-degree nodes are more likely to take vaccination (as shown in Fig. 4.6(a)). Due to the formation of herd immunity, more small-degree nodes choose the option of taking risk. Correspondingly, only small fraction of small-degree nodes is infected. However, with the middle efficiency, the most evident change takes place: nobody chooses vaccination; small- and middle-degree nodes take the self-protection strategy. In this case, the partial efficiency of self-protection and complete disappearance of vaccination can not form the herd immunity, which thus causes more nodes be infected, especially for middle- and large-degree nodes. With further increase of $\delta$, middle- and large-degree are obviously inclined to take the option of self-protection strategy due to
Figure 4.7: (a) The disease size or the fraction of infected individuals $f_I$; (b) individual average cost $< C >$ as functions of the cost $g$ of self-protection policy for different values of efficiency $\delta$ on BA network. It is obvious that for low $\delta$, the epidemic size and average cost monotonously decreases with the increment of $g$. While for larger $\delta$, the multiple effect occurs again: middle $g$ induces the peak of disease infection and mean cost. Depicted results are obtained for $c = 0.7$, $\beta = 0.18$ and $N = 2000$.

the high efficiency. Compared with the case of middle efficiency, infection turns to small- and middle-degree nodes. Based on these behaviors, we find that if prevention measures hold for the middle- and large-degree nodes (compare $\delta = 0.3$ and $\delta = 0.9$ in Fig. 4.6(d)), the disease spreading can be controlled well. This multiple effect of self-protection may shed light into the realistic case, where people are facing the decision to make self-protection or vaccination.

4.3.2 Impact of self-protection cost

Except for the effect of efficiency of self-protection policy, the impact of cost for self-protection remains of utmost importance. In what fellows, we will turn our attention to this point. Figure 4.7 features the fraction $f_I$ of infected agents (or the disease size) and the individual average cost $< c >$ as a function of the cost $g$ of self-protection for different values of $\delta$. It is evident that when efficiency is low (e.g., $\delta = 0.1$ and 0.3), the epidemic size and individual mean cost monotonously decline with the increment of cost parameter $g$. In this case, the low cost of self-protection drives more people to choose the self-protection rather than vaccination uptake.
However, low efficiency of self-protection can not provide complete protection and a high infection risk still exists (namely, more people are still infected even if they choose vaccination), which conversely leads to higher expense (see Fig. 4.7(b)). With the cost $g$ increases, low efficiency makes people give up the self-protection option. That is to say, the epidemic size becomes independent of the cost of $g$. Then the herd immunity provided by the vaccination suppresses the infection risk.

More interestingly, as $\delta$ becomes larger, an counter-intuitive observation appears: the epidemic size and individual average cost reach the peak at mediate cost $g$. The reason is similar to the analysis of impact of efficiency of self-protection. When $g$ is very small, the self-protection policy possesses the advantage of low cost and good efficiency, many agents will choose this strategy, which eradicates the high infection risk. Once the cost becomes very large, this advantage will vanish and the epidemic size returns to fixed level due to vaccination. If the cost $g$ is middle, some agents are still apt to self-protection policy. However, due to lower efficiency than vaccination and the decline of vaccination, the disease outbreaks. Thus, these results suggest the multiple effect is also prevalent with respect to the cost of temporary self-protection policy.

Now that the multiple effect exists against the cost of temporary self-protection, it is instructive to explore the mechanisms more quantitatively. Figure 4.8 shows the evolution courses for the fraction of taking vaccination, fraction of self-production, individual average cost and epidemic size for different values of $g$. When the small cost is considered ($g = 0.1$), the fraction of self-protection is very high due to high efficiency. Though the number of agents choosing vaccination is 0, the epidemic can be greatly controlled and the mean cost is low. However, with middle cost ($g = 0.4$), the motivation of choosing self-protection dramatically decreases, at the same time the enthusiasm of taking vaccination is not very high. The lack of efficient protection measures makes the outbreak of disease. Correspondingly, the
Figure 4.8: The time courses for the fraction of self-protection (a); the fraction of taking vaccination (b); the disease size or the fraction of infected agents \( f_I \) (c) and average cost \( < C > \) (d) with different values of \( g \) on the BA networks. For low cost, self-protection is promoted and can provide the safety guarantee due to high efficiency. While for high cost, high vaccination inclination can form the herd immunity to prevent more infection. However, intermediate \( g \) will face an embarrassed circumstances: neither vaccination nor self-protection holds the absolute advantage to protect public health. Depicted results are obtained for \( c = 0.7, \delta = 0.7, \beta = 0.18 \) and \( N = 2000 \). (To show the evolution trend more obviously, we just record \( 10^3 \) time steps, which do not affect the final results.)

Average cost is maximal as well. As the cost \( g \) is large, the choice of self-protection is fully neglected. The campaign of eliminating epidemic returns to the traditional two-strategy model, where more people are inclined to take vaccination to suppress the spreading of infection.

Another interesting observation in Fig. 4.7 is that there exists the step-like phenomenon for some curves. This means that at the same step, the faction of vaccination and fraction of self-protection are the same, which thus causes the identical epidemic size. To understand this phenomenon better, we can give some quantitative analysis. According to the requirement of choosing strategy, the threshold
Figure 4.9: $\delta - g$ phase diagrams for (a) the disease size or the fraction of infected individuals $f_I$; (b) individual average cost $< C >$ on BA network. Depicted results are obtained for $c = 0.7$, $\beta = 0.18$ and $N = 2000$.

Condition $g_{th}$ for one person taking self-protection with $k_{inf}$ infected neighbor(s) is

$$P_s < P_r,$$  \hspace{1cm} (4.9)

$$g_{th} + (1 - \delta) \times [1 - (1 - \beta)^{k_{inf}}] < 1 - (1 - \beta)^{k_{inf}},$$  \hspace{1cm} (4.10)

$$g_{th} < \delta \times [1 - (1 - \beta)^{k_{inf}}].$$  \hspace{1cm} (4.11)

Substituting $\delta = 0.9$, $\beta = 0.18$ and $c = 0.7$ into the above equation, we can obtain $g_{th}$ = 0.162, 0.29484, 0.40376, 0.49310 and 0.56633 for $k_{inf}$ = 1, 2, 3, 4 and 5, respectively. Importantly, these threshold values are in good agreement with the simulation points with steps changing.

Finally, to give a holistic profile, we show the $\delta - g$ phase diagrams about disease size $f_I$ and individual average cost $< C >$ in Fig. 4.9. The multiple effect of self-protection strategy is clear: the medium values of efficiency and cost can amplify the infection risk and overall expense. While for low (high) efficiency and cost of self-protection, people can get the sufficient protection with vaccination uptake (self-protection measure). This framework may provide new indication into disease prevention.
4.4 Conclusion

Different from the traditional two-strategy setup, we have introduced a new strategy, temporary self-protection into the epidemiological model in this chapter. Via numerous simulation, we find that this strategy produces a multiplex effect on the eradication of disease. If the efficiency or cost of self-protection is very low or high, the infection risk can be removed effectively. However, the intermediate efficiency or cost elevates the infection risk and enhances the social cost. While for this multiple effect, it is related with the loss of positive feedback mechanism between vaccination or self-protection and disease size, as shown in the last chapter. If either vaccination or self-protection takes the dominated strategy, herd immunity can be guaranteed. But once there is drastic competition between these two strategies, epidemic can diffuse fast. Moreover, we also show that this multiple effect is general for different interaction topology. For homogeneous network, infected agents are apt to forming clusters; while for heterogeneous network, vaccination or self-protection holding middle- and large-degree nodes is beneficial for the eradication of disease.

Although this research is just from theoretical viewpoint, the results can shed light on realistic cases in our life. In empirical circumstance, there may be more factors that can affect the efficiency and cost of self-protection policy. Incorporating these factors will make our investigation closer to life and provide meaningful instruction for the control of disease.
Chapter 5

Adaptive protection in epidemiology

As described in Chapter 4, self-protection strategy could be an effective method to control the propagation of disease. However, in some cases, such as the national severe contagion and the complete absence of vaccination, this measure usually loses its function. Then, cutting the link with infected sources becomes better method. Once the infected connection is pruned, the agent also need to connect to new health individual to maintain his contact with the environment. In this chapter, I will explore the influence of such adaptive protection strategy for the epidemiology dynamics within the coevolution framework of disease-behavior interplay.

5.1 Importance of adaptive protection strategy

The issue about how disease spreads and how it can be eradicated has been studied for long time (Bailey et al., 1975; Anderson and May, 1982; Yang et al., 2007). During the last decades, due to the increasing availability of analysis tools and
data information about contact networks, transmission patterns, control and immunity policy, it is good time to explore theoretical and computational scenarios for possible forecasting the outbreak and elimination of disease (Ewald, 1994; Moreno et al., 2003; Barthélemy et al., 2005). Up to now, several frameworks have been proposed, where the agents are distributed on the same static contact network (Pastor-Satorras and Vespignani, 2001; Boguná and Pastor-Satorras, 2002; Wang et al., 2003). Under this scenario, the epidemic outbreak threshold, similar to critical phase transmission, can be well predicted for different contact patterns (Pastor-Satorras and Vespignani, 2001; Gang et al., 2005; Boguná et al., 2003a). Recently, this mathematical analysis method is introduced into more complex case of interacting populations with the help of multiplex networks (Granell et al., 2013; Buono et al., 2014; Saumell-Mendiola et al., 2012). However, all these achievements do not involve human behavior traits.

To this regard, combining the human behavior trait, especially the process of making decision, into spatial epidemic study has become a novel framework, namely, the so-called "behavior-disease feedback dynamics study" (Bauch et al., 2003; Fu et al., 2011; Bauch and Earn, 2004). Along this line, several scenarios have been proposed, including the voluntary vaccination (Perisic and Bauch, 2009b; Wu et al., 2011; Vardavas et al., 2007), the competition of individual and collective benefit (Bauch et al., 2003; Zhang, 2013), and zealot preference (Liu et al., 2012b). Recently, Shang et al. reported that if individual awareness of infection risk was incorporated into vaccination campaign, the ratio of vaccinated agents and the time of vaccination decision were enormously affected (Xia and Liu, 2014). When the imitation dynamics of vaccination behavior was implemented in spatial populations, a double-edged sword effect took place, where vaccine uptake was promoted just for the low cost (Fu et al., 2011). Similarly, in Chapter 3, we have examined the impact of risk perception, which is beneficial for decreasing infection. In Chapter 4, we have further studied the effect of temporary self-protection
strategy, which can lead to the multiple effect.

In spite of these recent development in this line, there is a situation of particular relevance and prevalence that has received relatively little attention till now. This is the case of individual adaptive protection, where the susceptible agent can try to cut down the link with the infected neighbors and rewire to healthy agents. Moreover, the similar treatments can also be found in realistic life. For example, when the severe diseases take outbreak, part of people preferentially choose to cut the existing connections with those infected agents though there is some economic expense. In case these connections are not pruned successfully due to necessary collaborations, they still face the possible infection risk. In this sense, adaptive protection become a mixed strategy: agent can successfully remove the infection risk with certain probability, otherwise the threat of being infected still exists. However, some other persons (such as, people selling public insurance) do not like to take this action because they have to maintain their present connections to maintain individual benefit, in spite of the existence of infection risk. It is worth mentioning that in one early work (Gross et al., 2006), where it is assumed that the susceptible agent can take this type of rewiring action, the authors have considered similar dynamics but neglect individual desire. In virtue of evolutionary game theory, we here regard adaptive protection and taking risk as two parallel strategies with their respective cost functions, and allow irrational selection during decision-making process. That is, compared with the early work (Gross et al., 2006), susceptible agent does not necessarily choose adaptive protection (but just as one option related with its cost). Moreover, compared with the temporary self-protection setup which mainly competes with vaccination strategy in Chapter 4, adaptive protection can change the interaction topology forever. In reality, if the effective vaccine can not be immediately produced with some severe diseases, the setup of adaptive protection becomes of particular importance.
In this chapter, we consider the adaptive protection policy under the framework of spatial disease-behavior dynamics study. Different from the temporary self-protection strategy in Chapter 4, the agent choosing adaptive protection can cut the links to infected neighbors with a probability $Q$ (namely, the efficiency of adaptive protection), otherwise he still faces the infection risk with probability $1 - Q$. The opposite treatment is the strategy of taking risk (namely, nothing to do). For these two strategies, they usually need to spend the corresponding costs. To decrease the number of parameter, we use $C$ to denote the relative cost of rewiring links. Our aim is to study how this mechanism affects the spreading of disease on spatial populations. Via numerous simulations, we find that the infection risk could be eradicated, at the same time the topology properties also change.

### 5.2 Model and method of adaptive protection

In this chapter, we consider the effect of adaptive protection strategy on the eradication of disease. Under this strategy, each susceptible individual has probability $Q$ to cut down the link with an infected neighbor and reconnect to one susceptible agent (namely, preventing the propagation of disease), as similar to previous treatment (Gross et al., 2006). On the other hand, with the remaining probability $1 - Q$ the agent fails to protect himself, which means that he may still be infected. As an alternatively comparative setup, we also introduce the strategy of taking risk, namely, agent does not take any protection measure for potential infection risk.

As for the epidemiology model, we adopt the susceptible-infected-susceptible (SIS) model to investigate the role of adaptive protection strategy during the spreading of epidemic. In this model, people are divided into two compartments: susceptible
and infected. Infected individual transmits the disease to each susceptible neighbor with a probability $\beta$. At the same time, the infected agent can return to the susceptible state with the recovery probability $\mu$. In the spatial contact network, if one susceptible agent has $k_{inf}$ infectious neighbors, the total probability of being infected is

$$\lambda = 1 - (1 - \beta)^{k_{inf}}. \quad (5.1)$$

When the epidemic takes place, individuals need to make decision about their strategies. For simplicity, we here assume that the perceived infection probability $\lambda_{perc}$ equals to the actual probability $\lambda$, namely, $\lambda_{perc} = \lambda$ (herein we have proved that if we use the form of the perceived risk in Chapter 3, the qualitative result does not change. To decrease the number of parameter, we select the above formula). If agent choose the strategy of taking risk, his expected cost of this action can be described by the following function

$$P_{risk} = C_1 \times \lambda = C_1 \times [1 - (1 - \beta)^{k_{inf}}]. \quad (5.2)$$

where $C_1$ denotes the cost of infection, incorporating the expense and time for health and mental care as well as mortality. However, when an agent takes the adaptive protection strategy, there are two possibilities: he can successfully cut each link with the infected neighbors with probability $Q$ (the "the efficiency of adaptive protection strategy"); otherwise he still needs to face the potential infection risk from these infected neighbors. In particular, provided that the agent successfully cuts one connection with infected neighbor, he needs to build a new link with one randomly chosen susceptible agent from the whole population to keep the total number of edges unchanged (repeated connection and self-loops are not allowed in the present framework). With such an action, the agent spends the cost $C_2$, involving the expense of cutting a infected link and re-constituting a new link (as illustrated in Fig. 5.1). In this way, we can easily obtain the expected cost
function for adaptive protection strategy

\[ P_{prot} = Q \times k_{in,f} \times C_2 + C_1 \times [1 - (1 - \beta)^{(1-Q)k_{in,f}}]. \quad (5.3) \]

In this formula, the first term denotes the expense of successfully rewiring the links, while the second term represents the infection cost when cutting infected links fails. To be simple, we fix \( C_1 = 1 \) and denote \( C = C_2/C_1 \) as the relative cost of cutting infected link (herein it is worth noting that re-scaling cost does not change the dynamics and results). Then, we get the new forms for Eqs. (5.2) and (5.3) as follows:

\[ P_{risk} = 1 - (1 - \beta)^{k_{in,f}}, \quad (5.4) \]
\[ P_{prot} = Q \times k_{in,f} \times C + 1 - (1 - \beta)^{(1-Q)k_{in,f}}. \quad (5.5) \]

Obviously, \( Q = 0 \) comes to the case that agent can only take the risk when facing the infection. \( Q = 1 \) represents switching connection with infected neighbors is a certain event. While \( 0 < Q < 1 \) will make the adaptive protection become a mixed strategy. Different from the flexibility of cutting some links, it is interesting that an agent will cut all the infected links once adaptive process takes place. Moreover, it is also meaningful to mention that, compared with fixed interaction topology under temporary self-protection policy (previous chapter), co-evolution of strategy and structure adjustment is considered here.

From the viewpoint of collective benefit, the optimal case is to successfully cut the sources of infection. While in the opinion of agent, the best choice is to reduce his own cost. However, in reality, people are generally not perfect rational, and their decisions are usually affected by the information fluctuation or irrational factors. To describe this situation, we consider one stochastic decision-making process. At each time step, the probability of selecting adaptive protection for each agent is
Figure 5.1: Schematic figure for the adaptive protection scenario. Each susceptible agent faces two options: one is the adaptive protection strategy and another is the strategy of taking risk. For the adaptive protection, the agent can successfully prune the infected link with probability $Q$; otherwise he will still be infected. While for the strategy of taking risk, agents need to face the possible infection risk. For different strategies, we also show the details of cost function in the figure.

(Fu et al., 2011),

$$W = \frac{1}{1 + \exp[(P_{prot} - P_{risk})/K]},$$  \hspace{1cm} (5.6)

where $K$ denotes the noise level or the strength of selection (Szabó and Tóke, 1998). $K \to \infty$ means totally random selection (the process of throwing a coin), while $K = 0$ corresponds to the local best-response selection. As previous treatment (Liu et al., 2012b; Wu and Zhang, 2013), here we fix $K = 0.1$ which implies that the better strategy will be immediately adopted, yet it is possible that individuals will occasionally take a worse choice. In each time step (corresponding to one disease season), each node has one chance to adjust its choice and synchronously updates the strategy.

Due to the heterogeneity of social network, we choose the Barabási-Albert (BA) network as the initial contact network with size $N = 5000$ (Barabási and Albert, 1999). But with the evolution of disease-behavior dynamics, the network topology
and degree of nodes change. To explore the effect of adaptive strategy on the eradication of disease, we use the BA networks with different average degree $<k>$. Before the infection, we distribute 20 infected agents to the population as the infection seeds. Since rewiring of edge of networks may introduce additional disturbances, final results are averaged over up to 200 independent realizations for each set of parameter values.

### 5.3 Results and analysis

#### 5.3.1 Effect of efficiency of adaptive-protection strategy

To begin with, we first examine how the disease spreading varies under the consideration of adaptive protection strategy. Figure 5.2 features the color map encoding the final fraction $f_I$ of infected agents and the frequency $f_P$ of successfully cutting the infection link on the $C - Q$ parameter plane. It is clear that when the strategy
Figure 5.3: Color map depicting (a) fraction $f_I$ of infected individuals, (b) the fraction $f_P$ of successfully cutting infection link on the $C - Q$ parameter plane for larger average degree $<k> = 10$. The other conditions are the same as Figure 5.2.

of taking risk is the mere selection (i.e., $Q = 0$), the disease completely dominates the system (i.e., nobody takes the adaptive protection strategy), irrespective of the values of cost $C$. However, with the increment of $Q$ (i.e., elevating the efficiency of adaptive protection), this tide sharply changes: more and more people are likely to take the adaptive protection strategy due to the temptation of possibly low cost, which reaches a peak at the moderate $Q$ value. At the same time, one can also see that the size of epidemic will be eliminated quickly. As $Q$ further increases, the epidemic is completely eradicated. Because of the removal of infection sources, the enthusiasm of selecting adaptive protection strategy recovers 0 again. That is to say, there is a maximal level of choosing adaptive protection and successfully cutting infection link at medium $Q$. The smaller the cost of cutting link, more evident this trend. These results thus suggest that adaptive protective policy could play a significant role in controlling the spreading of epidemic.

To validate the effect of adaptive protection policy, we also explore the fraction of infection and cutting infection link for larger average degree in Fig. 5.3. It can be observed that the increment of $Q$ monotonously diminishes the outbreak of epidemic. Moreover, a medium efficiency of adaptive protection guarantees an
maximal level of cutting the infection link. This is agreement with the observations made by small number of connections, thus introducing adaptive protection is in general effective in eradicating the risk of infection. In addition, it is worth mentioning that, compared with case of small average degree, the optimal level of choosing adaptive protection is more obvious for small cost. At variance, with the increment of $C$, this trend fast dies out, since the potential number of infected agents greatly increases, which further elevates the expense of adaptive protection.

It is next of interest to explore the microscopic effect of adaptive protection on the evolution of topology of contact networks. As the treatment of previous work (Gross et al., 2006), the most effective quantity to reflect the property of network is degree distribution. Besides, another key parameter is the average degree of the next neighbors of a given node $< k_{nn} >$, which not only reveals the dynamical evolution of interaction topology, but also depicts the evolution character of disease-behavior dynamics (namely, how the self-protection scheme drives individual strategy choice). Figure 5.4 shows the degree distribution of different states and the whole network for different efficiency. It is clear that for small $Q$ the nodes of susceptible and infected states have a Poisson degree distribution, which means that the initial scale-free network has changed into a homogeneous topology. Because both large-degree and small-degree susceptible nodes tend to cut the infected links, nearly all agents will keep the same number of neighbors at last. However, with the increment of $Q$ ($Q = 0.6$), small-degree nodes are more inclined to prune the connection with hub nodes, which results in limited links left for these small-degree nodes. Under such a case, contact network becomes a heterogeneous pattern. When $Q$ is enough large (though the infected nodes keep homogeneous links), the heterogeneity of degree distribution for susceptible agents further enhances. This is because the links between large-degree and small-degree nodes are not pruned. In this sense, the total property of degree distribution unveils strong heterogeneity.
Figure 5.4: The degree distribution $\phi_k$ for susceptible, infected individuals (left) and the whole network (right) under different values of efficiency $Q$. From (a) to (c), the values of $Q$ are 0.025, 0.6 and 0.9, respectively. For small $Q$, both susceptible and infected nodes show the Poisson distribution, which means the total network becomes a homogeneous topology. As for intermediate $Q$, though the distribution is nearly similar for susceptible and infected nodes and has a broader range, the heterogeneity is obviously enhanced. When sufficient large $Q$ is considered, the further strengthened heterogeneity of susceptible agents makes the whole network show stronger heterogeneity and much broader degree rank, in spite of homogeneous distribution for infected individuals. Depicted results are obtained for $C = 0.1$, $\beta = 0.35$, $\mu = 0.2$ and $< k >= 4$ for initial networks.
Then, we turn to the analysis of average degree of the next neighbors of given nodes. Figure 5.5 shows how \(< k_{nn} >\) varies as a function of the node degree \(k\) for susceptible, infected agents and the whole network under different values of \(Q\). Obviously, if \(< k_{nn} >\) increases with the evaluation of degree \(k\), it means that network possesses an assortative mixing pattern, i.e., large-degree (small-degree) nodes tend to connect with large-degree (small-degree) nodes. At variance, once \(< k_{nn} >\) monotonously declines, a disassortative mixing pattern shows itself, i.e., large-degree (small-degree) nodes are apt to connecting with small-degree (large-degree) nodes. In particular, un-assortative network means that the value of \(< k_{nn} >\) becomes independent of the degree \(k\).

From Fig. 5.5, it is clear that, for small \(Q\) (low efficiency of adaptive protection policy), \(< k_{nn} >\) is independent of degree \(k\), irrespective of susceptible and infected individuals. In this sense, vast majority of nodes have the identical number of neighbors, namely, homogeneous network is the consequence of strategy and topology co-evolution. Compared with initial scale-free network, it is clear that the range of degree sharply shrinks, which means though the ratio of successfully cutting down the infected links is low, both large-degree and low-degree nodes trend to decrease connection and reduce the potential risk because of the tiny expense difference between adaptive protection and taking risk (see Eqs. (5.4) and (5.5) for more details). However, as the efficiency of adaptive protection strategy enhances, (in spite of slim separation between susceptible and infected agents), both values of \(< k_{nn} >\) gradually increase with the degree \(k\); namely, assortative mixing pattern appears. The range of degree is extended as well. In such a case, once small-degree susceptible nodes have infected neighbor(s), larger \(Q\) will make the expense difference between adaptive protection and taking risk become more obvious, which means that adaptive protection possesses more advantage. In this case, small-degree nodes are more inclined to cut the links with large-degree nodes. Due to pruning connection with small-degree nodes, larger-degree nodes
Figure 5.5: The mean nearest-neighbor degree $< k_{nn} >$ depending on the node degree $k$ for susceptible, infected individuals (left) and the whole network (right) under different values of efficiency $Q$. From (a) to (c), the values of $Q$ are 0.025, 0.6 and 0.9, respectively. For small $Q$, $< k_{nn} >$ becomes independent of degree $k$, which means that contact network becomes uncorrelated and homogeneous. For intermediate $Q$, the values $k_{nn}$ for susceptible and infected agents increase with degree $k$, which leads to an assortative mixing pattern. When $Q$ is enough large, infected agents reveal slight assortative property, the disassortative pattern of susceptible individuals is very strong, which makes the whole network possess a disassortative character. Depicted results are obtained for $C = 0.1$, $\beta = 0.35$, $\mu = 0.2$ and $< k > = 4$ for initial networks. (Here it is worth pointing out that due to potential heterogeneity of cutting links, the degree distribution rang may be identical or differential for susceptible and infected agents.)
Figure 5.6: (a) Number of clusters of susceptible, infected clusters $N_S, N_I$ and (b) number of agents in the respective largest cluster $N_{SS}, N_{II}$ as a function of efficiency $Q$. For small $Q$, we can see that infected agents form the giant cluster and a great number of susceptible agents are isolated. However, with the increment of $Q$, the giant infected cluster start to collapse gradually and susceptible expand even though the size of largest susceptible cluster is still smaller than that of largest infected cluster. When $Q$ is sufficiently large, the less infected agents will be fast eliminated from the system and the remainder susceptible agents form a giant cluster. Depicted results are obtained for $C = 0.1$, $\beta = 0.35$, $\mu = 0.2$ and $< k >= 4$ for initial networks.

are only left the connections with other larger-degree nodes, which naturally leads to the assortative mixing pattern. Interestingly, when the value of $Q$ is sufficiently large ($Q = 0.9$), all these trends will change. Owning to the lower cost of adaptive protection for large-degree nodes, their enthusiasm of cutting the infection sources becomes extremely high. These large-degree nodes thus cut the links with other large-degree nodes (whose risk property is usually high) and turn to reserve the connections with small-degree nodes. While for the infected nodes, they are quickly removed from the susceptible agents and form small clusters, where the link number is basically equal. Combining these two observations, the whole network shows strong character of disassortative mixing. As such, the consideration of adaptive protection policy results in different mixing pattern of contact network under different efficiency $Q$.

Along this line, it is also meaning to investigate the spatial configuration characters of susceptible and infected nodes. Due to the events of cutting the infected links and re-constructing new healthy connections, agents holding the same states
Figure 5.7: Examples of the topology variation under the adaptive protection policy. From left to right, the topology changes with the increment of $Q$. The color code is susceptible cluster (red) and infected cluster (blue). For small $Q$, one giant infected cluster is surrounded by many susceptible agents. With increment of $Q$, this giant infected cluster collapses and one giant susceptible cluster starts to form. Finally, the system is composed of one giant susceptible cluster and several infected agents at large $Q$.

are more inclined to form clusters. Figure 5.6 features the number $N_S$, $N_I$ of susceptible, infected clusters and the number $N_{SS}$, $N_{II}$ of individuals in the respective largest cluster (herein the largest cluster denotes the cluster possessing the maximal number of nodes of the same states) as a function of $Q$. If taking risk is the only choice ($Q = 0$) or the efficiency of adaptive protection is very low ($Q$ close to 0), agents can not protect themselves and avoid the infection very well. In this case, the limited successful removal of infected links makes susceptible agents fall into small and isolated clusters. While for infected individuals, they can fast propagate the disease to their neighbors so that they finally form the giant clusters. To provide a more intuitive comprehension, we can observe the spatial network in Fig. 5.7(a), where there is a giant infected cluster and many small and isolated susceptible clusters are located around this giant infected cluster. With the increment of $Q$, the elevation of successfully pruning the infected links makes the number of isolated infected clusters climb faster, but the disaggregation of giant infected cluster and the expansion of giant susceptible cluster are still slow. In particular, though the size of giant infected cluster is still larger than that of giant susceptible cluster for $Q = 0.6$, the infection sources can be effectively isolated, which makes the number of infected clusters exceed that of susceptible clusters.
From Fig. 5.7(b) we can also see that except for the large susceptible and infected clusters, a great number of isolated susceptible and infected agents are around these two clusters. Interestingly, when the efficiency of adaptive protection is sufficiently high \((Q > 0.7)\), the infection sources will be isolated quickly even before a few people obtain infection. Then the remainder susceptible agents form giant cluster(s) via adjusting the connection. The larger the value of \(Q\), more obvious this trend. This observation can be clearly validated by the spatial pattern of contact topology in Fig. 5.7(c), where giant susceptible cluster is surrounded by several infected infected nodes.

To vividly unveil the impact of efficiency \(Q\), we also provide a schematic explanation of the topology change in Fig. 5.8. As \(Q\) is low, a minority of susceptible agents cut the links with the infection sources, and they are usually located around a giant infected cluster in the form of isolate agents. With the increment of \(Q\), adaptive protection strategy can prune the infection sources more effectively, which split the system into two giant clusters and several small clusters. Under the extremely high efficiency, the invasion of infection is even prohibited in the early stage so that the remainder agents form a large susceptible alliance.
5.3.2 Effect of transmission rate of disease

Except for the inherent factors of adaptive protection, the transmission rate $\beta$ also plays an important role in the spreading and eradication of disease. In previous research, where the adjustment of link is just based on a probability rather than the introduction of game framework (Gross et al., 2006), one can see that the change of transmission rate will lead to different phases: endemic, health, bistable and even oscillatory phases. It is fascinating to explore the impact of transmission rate within the game-epidemic feedback framework. Fig. 5.9 shows the color map encoding the final fraction $f_I$ of infected agents and the frequency $f_P$ of successfully cutting the infection link on the $\beta - Q$ parameter plane. Look at the entire phase diagram, it is clear that disease decreases and there exist an optimal fraction of successfully cutting the infected links with the increment of $Q$. But under different transmission rate, the system property will be different. For low transmission rate ($\beta < 0.3$), the number of infected agents is less at low $Q$ and this number gradually decrease with $Q$. At the same time, the range of optimal fraction of successfully cutting infected links is largest. When $\beta$ gets a middle value in the interval $[0.3, 0.8]$, the fraction of infected individuals sharply increases. More interesting is that the optimal observation of successfully cutting infected links is more evident, though its range shrinks. Finally, if the infection becomes an assured event during the contact with infected agent (namely, $\beta$ approaching to 1), nobody will take the adaptive protection strategy even if its efficiency is high. This is to say, the optimal observation of cutting infected links dies out and disease propagates freely in the system. Thus, these results suggest that the combination of efficiency $Q$ and transmission rate $\beta$ can leads to double optimal choice of adaptive protection.

To explain the above impact of transmission rate, it is important to check how the adaptive protection strategy changes microscopic dynamics of agents. Figure 5.10 features the number $N_S$, $N_I$ of susceptible, infected cluster and the number $N_{SS}$,
Figure 5.9: Color map depicting (a) fraction $f_I$ of infected individuals, (b) the fraction $f_P$ of successfully cutting infection link on the $\beta - Q$ parameter plane. For different values of $\beta$, the cases are different. Though the infection is mild at low $\beta$, the optimal observation of successfully cutting the infected links is not very obvious. Then, at middle $\beta$, this optimal trend becomes evident along with the reduction of its range. For high infection risk, this trend will vanish and disease holds the whole system. Depicted results are obtained for $C = 0.2$, $\mu = 0.2$ and $< k > = 4$ for initial networks.

$N_{II}$ of individuals in the respective largest cluster as a function of $Q$ for different values of infection rate $\beta$. What we firstly notice is the fact that at $Q = 0$ (namely, the strategy of adaptive protection has no effect) the number $N_S$ of susceptible clusters is always higher than that of infected clusters and the giant susceptible cluster is always smaller, irrespective of the transmission rate. But, as $Q$ increase, this trend changes a lot. For small $\beta$, where infection is mild and the decline of infection is slow, the initial giant infected cluster gradually collapses and the largest susceptible cluster also expand as the efficiency enhances. At the same time, it is clear that except for small $Q$, the number of both infected and susceptible clusters is nearly identical. If the infection risk becomes slightly large (namely, middle $\beta$), we can see that the collapse speed of giant infected cluster becomes slow (though its initial size increases). That is to say, till middle efficiency ($Q$ approaching to 0.6) the infection links can be effectively removed and many small infected clusters or agents are isolated, which is proved by the tremendous number of infected clusters (close to 2500, which, in fact, is the largest number). After this point, the giant cluster of susceptible fast expands till holding
the whole system. Once the infection risk is sufficient large, one can see though the number $N_I$ of infected clusters increases, the largest cluster of infected agents is still maintained till very large efficiency (namely, $Q$ is about 0.7). After this value, we can see the collapse speed of giant infected cluster is relatively fast. It is also worth mentioning that the maximal value of both infected and susceptible clusters decreases compared with the case of middle efficiency (namely, $Q = 0.5$). Last, provided that infection via contact with infected neighbor(s) becomes an inevitable event (namely, $\beta = 1.0$), the size of giant infected and susceptible clusters almost keep constant for any efficiency of adaptive protection strategy, This is because that the expense of adaptive protection strategy exceeds that of taking risk in most cases, the effect of adaptive protection strategy becomes limited. This can also get the support from the slight change of number of respective clusters. Thus, from these microscopic analysis, we get that middle transmission rate and middle efficiency jointly accelerate the collapse of giant infected cluster, which results in the most effective protection.

Finally, an remaining important question is how the epidemic size is affected by cost $C$ for the fixed efficiency $Q$. To answer this question, Fig. 5.11 features the color map encoding the final fraction $f_I$ of infected agents and the frequency $f_P$ of successfully cutting the infection link on the $\beta - C$ parameter plane for different values of $Q$. As efficiency $Q$ increases, the overall observation is similar with the previously obtained results: infection is eliminated and middle efficiency generates optimal level of adaptive protection. However, these phase diagrams are fully different at each efficiency. For low efficiency of adaptive protection (namely, small $Q$, top panel), we can see that the larger the value of $\beta$, more severe the infection (less the faction of successful cutting the infected links). This is actually consistent with our expectation, the larger infection risk make the advantage of adaptive protection strategy become less and less for same cost, then the decline of successfully cutting infected link and the outbreak of disease are the
Figure 5.10: Number of clusters of susceptible, infected clusters $N_S, N_I$ (left) and number of agents in the respective largest cluster $N_{SS}, N_{II}$ (right) as a function of efficiency $Q$ for different values of $\beta$. From top panel to bottom panel, the values of $\beta$ are 0.2, 0.5, 0.9 and 1.0, respectively. Depicted results are obtained for $C = 0.2$, $\mu = 0.2$ and $<k> = 4$ for initial networks.
inevitable outcomes. While for the same infection rate, it is also evident that with the increment of cost $C$ of adaptive protection strategy, the fraction of infected individuals grows and the rate of successfully cutting infected links monotonously drops. If turning to the case of middle efficiency (middle panel), we can see that except for small $\beta$, the above described trend is further strengthened: the number of infected agents and proportion of effectively cutting infected links fast decrease with the increment of $\beta$ and $C$. While for small $\beta$, though the infection risk is better controlled, an intermediate cost leads to a highest level of cutting infected links. This is because for small cost, no protection action is better option due to low cost. With the amplification of infected agents, adaptive protection becomes very necessary to decrease the possibility of infection, which results in the increment of cutting infected links. Since the vast majority of persons have been infected, the choice of adaptive protection strategy loses its merit in this case of large cost.

Different from the above cases, if the high efficiency $Q$ is considered, a counter-intuition phenomenon takes place on the $\beta - C$ parameter plane (the bottom panel). It is clear that for large infection rate (namely, large $\beta$) nearly nobody (in fact very small fraction of population) is infected and chooses adaptive protection strategy. This is because with the high efficiency of cutting the infection sources, the initial infected seeds are isolated fast, which enables most of persons keep susceptible state. Thus, the fraction $f_P$ of successfully cutting the infection links seems particularly low. Due to effectively isolation, the fraction $f_I$ of infected agents is extremely low as well. When the infection rate $\beta$ is small, this will be completely different. Susceptible agents unhesitatingly select the cutting the connection with the infected neighbors at the small expense, which eradicates the infection sources. However, with the increment of cost, infection monotonously increases and there exists an optimal level of choosing adaptive protection. This is because that the increment of expense makes people produce mind with good luck: if other susceptible agents can cut the links with infected neighbor(s), he does not
Figure 5.11: Color map depicting fraction $f_I$ of infected individuals (left) and the fraction $f_P$ of successfully cutting infection link (right) on the $\beta - C$ parameter plane for different values of $Q$. From (a) to (c), the values of $Q$ are 0.2, 0.65 and 0.9, respectively. For small $Q$, it is clear that the epidemic size monotonously increases with the increment of $\beta$ and $C$ (the fraction of cutting infected edges decreases), which is consistent with the expectation. This trend is strengthened with middle $Q$, except for the optimal ratio of successfully cutting infected links at small $\beta$. While for large $Q$, infection just takes place at extremely large cost and small infection risk, at the same time there is also a slight optimal observation of cutting the infected links. Depicted results are obtained for $\mu = 0.2$ and $< k > = 4$ for initial networks.
need any expense again (which is similar to the free-rider phenomenon in game theory and vaccination campaign (Montopoli et al., 2009; Bauch et al., 2010)). In the beginning, the infected sources can not be separated so that many susceptible agents are infected. In this case, some people just are aware of the importance of adaptive protection and turn to cutting the connection with more infected agents, which results in the increment of successfully cutting infected links. But if the expense of cutting infected links is further amplified, the advantage of adaptive strategy will fully vanish. So, more individuals are finally infected and the ratio of successfully cutting infected links declines.

5.4 Conclusion

The eradication and control of infection disease is one key question to guarantee the public health. To reach this aim, many immunity method and policies have proved their effect, such as voluntary vaccination and the temporal self-protection strategy described in last chapter. Different from these setups, this chapter examines a new individual decision process in the context of adaptive protection policy. When agent takes this strategy, he can cut the links with infected neighbors and rewires to new susceptible agents with the probability $Q$ (namely, the efficiency of adaptive protection); otherwise he still faces the infection risk with the probability $1 - Q$. In this sense, this novel setup is a mixed strategy. For $Q = 0$, agent can just take risk and has no any action. When $Q = 1$, the action of cutting infected link is absolutely effective. $0 < Q < 1$ involves these two cases. Moreover, it is also worth mentioning that, compared with the previous treatment (Gross et al., 2006) where susceptible just simply cutting the infected links, the cost function for each action is considered. If agent chooses the strategy of adaptive protection, he needs to spend the cost of rewiring links $C$ and facing possible risk, while agents taking risk just needs to support the expense of possibly high infection
risk. Then, incorporating these two strategies and their respective cost functions into the evolutionary game framework, we can let agents make decision of which is better.

By means of numerous simulations, we find that the increment of efficiency of adaptive protection strategy can decrease the infection risk, while middle efficiency supports an optimal level of cutting infected links. As the cost enhances, this trend becomes less and less conspicuous, this is because the advantage of adaptive protection is declining. Then, we explore how this efficiency affects the interaction topology. We find that with the increment of efficiency of adaptive protection, the heterogeneity of contact topology becomes stronger and the range of degree distribution becomes broader. At low efficiency, the network becomes homogeneous and has no any assortative property. While for middle efficiency, both susceptible and infected networks shows the assortative mixing pattern, which naturally lead to the assortative character of the whole network. When the large efficiency is considered, though susceptible network shows a homogeneous property and slightly assortative mixing pattern, the whole network unveils a strong heterogeneity and disassortative property due to the larger infected cluster. When turn to microscopic distribution of clusters, we find that at small efficiency the giant cluster of infected agents will hold the system, which are surround by a great number of small susceptible clusters. With the increment of efficiency, the giant infected cluster gradually collapse and the giant susceptible cluster forms, both of which are encircle by their respective small clusters. When the number of infected cluster reaches the maximal value, the growth speed of giant susceptible cluster become faster till holding the whole system at high efficiency.

Moreover, we have also explored the effect of transmission rate on the epidemic size. We see an optimal observation of successful cutting infected links, which is most obvious in the middle risk $\beta$. Through the analysis of clusters, we find that
the collapse of giant infected cluster is most effective under this middle risk, which supports the better protection. At last, we also examine the effect of transmission rate with different cost, and find different phases diagrams, which can be attributed to the increase or decrease of expense advantage of adaptive protection strategy.

Notably, the theoretical setup of adaptive protection strategy we have considered here seems to be reasonable in some realistic cases. When agents make their decisions whether to cut the link with infected person, they need to consider the possible expense. While in reality, even if some people know the possible risk and economic expense, they still insist maintaining the previous connection, such as, the persons selling the insurance. In the similar case, we may need to adjust the present model involving more evaluation factors. But before any realistic and systematic proof, this proposal still offers fascinating new insights into the research of epidemic prevention. We hope that this work will inspire future studies, especially in terms of combing with empirical data in societies via a coevolutionary approach.
Chapter 6

Impact of subsidy policy on voluntary vaccination dilemma

In foregoing chapters (Chapters 3,4,5), we have shown the impact of perceived risk and effective protection measures. But these proposals are mainly based on the consideration of agents (namely, from the viewpoints of individual benefit), while in some cases of our life, we need to consider the questions from the angle of population benefit. In this sense, it becomes very valuable to propose possible policies suitable for the population, which, to large extent, may get the attention of the policy markers. Inspired by the ubiquitous subsidy policy in our life, we propose a theoretical framework in this chapter to examine the impact of such a policy. Though there may exist the difference with empirical case, we believe that this scenario can shed new light into the prevention of disease.
6.1 Subsidy policy under the framework of vaccination campaign

The advent of complex networks is beneficial for the understanding of the influence of contact topology on the propagation of disease on top of them. Up to now, a great number of research achievements have been accumulated, which include the prediction of critical point of disease outbreak (combining with physics definition), and the effect of information diffusion on the eradication of infection. For example, in recent reports (Pastor-Satorras and Vespignani, 2001; Boguña et al., 2003a; May and Lloyd, 2001), where the epidemic was allowed to spread on scale-free network, the threshold of epidemic outbreak was absent for the exponent $\gamma$ of power-law distribution belonging to the range $(2, 3]$ (Pastor-Satorras and Vespignani, 2001); while it appeared in an anomalous way if the value of $\gamma$ was located in the range $(3, 4)$ (Pastor-Satorras and Vespignani, 2002a; Cohen et al., 2002). In addition, it has been well-known, AIDS can spread simultaneously through three types of pathways: sexual activity, blood and breast milk; rumor or propagation information can diffuse based on online social network and traditional physical network (namely, the spreading routes are multiple at present). It thus becomes of utmost of importance to propose more effective methods of eliminating, at least reducing the infection risk. Many compulsive scenarios, such as, ring immunization (Müller et al., 2000; Greenhalgh, 1986), targeted immunization (Pastor-Satorras and Vespignani, 2002b; Wang et al., 2009), acquaintance immunization (Cohen et al., 2003; De Wals et al., 2001), have been taken into account.

Under these scenarios, though the performance of public health care services has been improved prominently, it is still extremely difficult to eradicate infectious diseases to entail a world free of pathogens (Organization et al., 2008; Brown et al., 1998). This is particular evident for the diseases caused by a virus with
a high mutant capacity. The seasonal influenza, as a typical example, induces annual epidemics circulating worldwide. Since seasonal flu spreads easily from person to person and can generate severe symptoms, this disease results in a serious public health burden, with three to five million cases of severe infections and about 250,000 to 500,000 deaths yearly (Organization et al., 2008). To prevent the infections, pre-vaccination before the beginning of a flu season becomes one important strategy, which is generally implemented through a voluntary tactic, different from the traditional consideration of compulsive setup. We can look at some examples more specifically. In a recent work (Fu et al., 2011), the authors explored the influence of imitation dynamics on vaccination campaign and found that the vaccination level was promoted under the game framework. In spite of the existence of free-riding behavior in vaccination experiments, the vaccination uptake was till enhanced when facing severe influenza. However, all these works just simply assumed that the vaccine was completely effective, i.e., the vaccinated agents got perfect immunity against any infection. This is obviously inconsistent with many empirical cases: some severe disease, such as, HIV and measles, are difficult to get perfect vaccine because of the frequent mutation of infection virus (Wu et al., 2011). Current questionnaire also shows that the perceived effectiveness of vaccine is often lower than its actual efficiency (Wu et al., 2011). Therefore, different from the previous Chapters (Chapter 3 and Chapter 4), consideration of imperfect vaccine in the theoretical analysis of vaccination game seems more reasonable.

Turning to the seasonal influenza, when the influenza takes place, the forward preparation of vaccination is usually hurried. In this case, the temporary uptake of residual vaccine in previous seasons or hasty vaccine without massive test becomes a feasible option (Davey, 2007). However, as we know, the infection virus of seasonal influenza usually has mutation from one season to another due to the resistance to drugs. This means that the vaccine of seasonal influenza is usually
imperfect effective. To offset this fault, various policies have been deployed to promote the general public to participate in vaccination campaigns. However, one particularly relevant case, the effectiveness of subsidy policy, is still unclear. Thus, it is urgent to study the effect of subside policies on the vaccination performance.

In this chapter, we consider the imperfect vaccine, whose efficiency is denoted by one parameter $\epsilon \in [0, 1]$, in the vaccination campaign of diseases like seasonal influenza. When one agent takes vaccination, he may get the protection but may also be infected due to the limited effectiveness of vaccine. If this agent is further infected, he still spends the expense of the infection. In this sense, it is not beneficial for the popularization of vaccination, which is contrary to the collective benefit. To promote the vaccination, we introduce the subsidy policy into the vaccination framework, where the infected vaccinators will get the subsidy for their contribution. In the collective or government’s viewpoint, it is more expected that suitable subsidy will promote vaccination enthusiasm and control the spreading of disease. Our aim is to study how this policy affects the promotion of vaccination and the eradication of disease on spatial populations. By means of numerical computer simulations, we find that this simple mechanism can promote the vaccination level, irrespective of the potential interaction topology. Besides, more interesting phenomena are explored. This study may shed light to the role of subsidy in the epidemic control of realistic society.
6.2 Subsidy model and detailed algorithm

6.2.1 Mathematical model of subsidy policy

**Basic Model.** To study the impact of pre-vaccination option on the flu transmission in human social networks, as in (Fu et al., 2011), we consider an iterated two-stage disease-vaccination feedback processes, which is composed of the decision-making dynamics and the infection dynamics. At the beginning of each flu season, each person decides whether or not to get vaccinated, based on his tradeoff between the benefit and cost. To clarify the analysis, we assume that the vaccination incurs a marginal cost $C_V$ to each vaccinated person, which is relevant to the immediate expenditure, time spent in taking the vaccination, and health-related side-effects, e.g., fever, arm swelling, and nasal congestion. Vaccine-induced death is excluded here, since present techniques of flu vaccine production ensures the basic safety. Unvaccinated individuals pay nothing at this stage. Besides, define $\varepsilon$ as the vaccine efficacy, i.e., each vaccinated individual becomes immune to the disease with the rate $\varepsilon$, and remains susceptible at the rate $1 - \varepsilon$. This decision-making dynamics mediates the vaccination opinion formation of individuals in each flu season, after which individuals fall in three specific status: vaccinated and immune, vaccinated and invalid, and unvaccinated. The latter two kinds of people are both susceptible to the disease.

In the second stage of each flu season, infectious carriers are introduced by randomly choosing $I_0$ susceptible individuals, with their health status changed to be infectious. The infection dynamics among individuals is characterized by the standard susceptible-infectious-recovered (SIR) compartment model. Per unit time, each infectious person can infect any susceptible neighbor with transmission rate $\beta$, and is recovered to become immune to the disease with recovery rate $\mu$. The epidemic spreading is simulated by using the stochastic Gillespie Algorithm (see
the following section for more details), which proceeds until no more appearance of newly infected individuals. Due to the expenses of medical care or services, or the absence from routine work, the infection results in a cost $C_I$. Without loss of generality, by assigning $C_I = 1$, the vaccination cost is rescaled to be $C = C_V/C_I$, which does not affect the final results. Generally, the vaccination cost is lower than the infection cost for the season flu, thus the relative cost of vaccination is confined to the range $C \in [0, 1]$.

Table 6.1: Payoffs obtained by the four types of individuals at the end of each flu season, without the implementation of subsidy policy. Here, Vac-Heal, Vac-Inf, Unv-Heal and Unv-Inf denoted the status of Vaccinated and Healthy, Vaccinated and Infected, Unvaccinated and Healthy, Unvaccinated and Infected, respectively.

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According to the vaccination decisions and the infection history, there exist four types of individuals at the end of each flu season, i.e., vaccinated and healthy, vaccinated and infected, unvaccinated and healthy, unvaccinated and infected. When subsidy policies are excluded, as illustrated in Table 6.1 their payoffs obtained in that flu season are $-C, -C - 1, 0, -1$, respectively. Among these people, those who refuse the vaccination and also successfully avoid the infection receive the highest payoff (namely, the so-called ”free-rider”), benefiting from the high-level vaccination coverage in the neighborhood. The presence of such free-riders leads to a dilemma in the voluntary vaccination, since they exploit the vaccination efforts of the other people (Montopoli et al., 2009; Bauch et al., 2010; Ibuka et al., 2014). Observing the unfair case, vaccinated individuals might follow the strategy adopted by free-riders. Thus, it is important to consider the update of vaccination decisions of individuals in each flu season.
Collecting the payoff and comparing with that of the neighbors at the end of each flu season, each individual decides whether to change his strategy in the next season. Specifically, each individual $i$ randomly selects one neighboring individual $j$ as the estimator. The higher payoff the individual $j$ acquires, the more probable his strategy will be imitated by the individual $i$. According to the Fermi updating rule (Szabó and Tóke, 1998), which has been extensively applied in the studies of imitation dynamics, individual $i$ adopts individual $j$’s vaccination decision with a probability

$$W(P_j - P_i) = \frac{1}{1 + \exp[-\alpha(P_j - P_i)]},$$

(6.1)

where $\alpha$ represents the noise level or the strength of selection (Szabó and Tóke, 1998; Fu et al., 2011), and $P_i, P_j$ represent the payoffs earned by $i, j$, respectively. When $\alpha = 0$, the imitation is totally random, while for $\alpha \to \infty$, it returns to the local best-response scenario (Nowak and May, 1992). As previous treatment (Liu et al., 2012b; Wu and Zhang, 2013), $\alpha = 5$ is considered throughout this chapter, which implies that better strategy is adopted with a high probability, yet it is still possible that individuals make a worse choice occasionally.

**Subside Policy.** To promote individual aspiration of taking vaccination, various subsidy policies are usually introduced in the real-world. Here, we focus on an intuitive strategy that vaccinated individuals who get infected will be compensated with a monetary subsidy $S$, which means that their payoff becomes $-C - 1 + S$. Table 6.2 summarizes the payoffs obtained by different types of individuals, under

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<td>Payoffs</td>
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the deployment of the subsidy policy. To better show the full procedure of subsidy considered here, Fig. 6.1 gives the schematic presentation, where agents make decision of vaccination or not in the first stage; then when the vaccinated agents are infected, they will get a subsidy $S$ for their overfull expense in the second stage.

To model human social networks characterized by local connections among individuals, the population infrastructure is simulated with a regular $L \times L$ square lattice, or Erdős-Rényi random graphs (Erdős and Rényi, 1959), and Barabási-Albert scale-free networks (Barabási and Albert, 1999) with the average degree $\langle k \rangle = 4$ and size $N$. The key quantities, the fraction of vaccination and fraction of infection, are determined within last $10^3$ full steps of overall $10^4$ time steps. Moreover, since the consideration of subsidy and interaction topology may introduce additional disturbances, final results are averaged over up to 100 independent runs for each set of parameter values.

### 6.2.2 Gillespie algorithm

Different from the traditional Monte Carlo simulations, here we use the Gillespie Algorithm to model the epidemiological evolution in the vaccination seasons (Gillespie, 1977). Before the epidemic reason, $I_0 = 10$ infected agents are randomly distributed into the whole population as the infection seeds. Each time step is regarded as one epidemic season, which is composed of the following elementary steps.

Firstly, at time step $t$, we need to calculate the respective transmission rate $g_i(t)$ for each susceptible and infected individual. For the susceptible agent, his total ability of being infected is $g_i(t) = \beta \times$ number of infected neighbors. While for infected
Figure 6.1: Schematic illustration of the voluntary vaccination under the subsidy policy. We model the vaccination campaign as a two-stage game. In the first stage, individuals make independent decisions about vaccination. If they choose the vaccination (blue node), they will spend a cost $C$. However, the effectiveness of vaccine is limited. In the following second stage (flu season), once a person gets immunity or is not protected but luckily escapes from further infection (blue node), there is no more cost. At variance, some vaccinators fail to obtain immunity and become infected (red node), they still need to spend the infection cost 1. But due to the existence of subsidy to these guys, their final cost becomes $−C−1+S$. While for non-vaccinators, they will be infected with the cost 1 (orange node) or escape from any infection and any cost as a free-rider (gray node). Totally, there will be four statuses: vaccinated and healthy, vaccinated and infected, unvaccinated and healthy, unvaccinated and infected.

A person, the rate of recovering from infection is still $g_i(t) = \mu$. The transmission rate for the whole population is thus $\xi(t) = \sum_i g_i(t)$.

Then, we define the time $t' = t + \Delta t$, when the next transition event takes place, which is supported to be a Poisson process. We let $\Delta t$ sampled from an exponential distribution with mean $\frac{1}{\xi(t)}$. If we choose a uniform random number $\omega \in [0, 1)$, the time interval will be $\Delta t = -\frac{\ln(1-\omega)}{\xi(t)}$.

Thirdly, with a probability proportional to $g_i(t)$, we choose the agent whose state
(susceptible or infected) changes at time $t'$. Generate a uniform random $p$ belong to the interval $[0,1]$. If the condition satisfies the inequation $\sum_{j=1}^{\zeta-1} g_j(t)/\xi(t) \leq p < \sum_{j=1}^{\zeta} g_j(t)/\xi(t)$, then this agent $\zeta$ is the chosen individual of changing state.

Forth, repeating the above steps 1-3 till all the infected individuals vanish in the system.

After all these steps are finished, the system enters into next season. Here, it is worth mentioning that the length of each time or flu season is closely related with the parameters of $\beta$ and $\mu$. To have a comparison with the previous results (Fu et al., 2011; Liu et al., 2012b; Wu and Zhang, 2013), we select $\beta = 0.46$ and $\mu = 1/3$ for all the figures.

### 6.3 Results and analysis

#### 6.3.1 Effect of subsidy on eradication of disease

To begin with, we first inspect how the vaccination coverage and epidemic size vary as the function of relative vaccination cost $C$ within the framework of subsidy.
Figure 6.3: The phase diagrams of fraction of vaccination (a), the total subsidy amount (b), the fraction of infected vaccinators, (c) and individual average cost (d) on the $C - S$ parameter plane on the scale-free networks. The parameter is $\epsilon = 0.7$.

Policy. Figures 6.2 presents the results generated by simulated epidemic in scale-free networks, with a typical vaccine efficacy $\epsilon = 0.7$ (Galvani et al., 2007; Luce et al., 2008). It is evident, that without subsidy (i.e., $S = 0$), the vaccination coverage quickly declines monotonically with the increment of the relative cost $C$, which correspondingly leads to the continuous increase of the epidemic size captured by the fraction of infected persons at the long-term equilibrium. Notably, there exist two special threshold values $C_H, C_L$ characterizing the crossover from a global vaccination coverage that eliminates the presence of infected individuals to the appearance of unvaccinated people, and the crossover from the mixture of vaccinated and unvaccinated individuals to the disappearance of vaccinated persons, respectively. Interestingly, when the subsidy policy is introduced, the vaccination performance can be greatly improved. One can clearly observe that both $C_H$ and $C_L$ increase with the augmentation of the subsidy level $S$, which
Figure 6.4: Total subsidy amount and fraction of infected vaccinators in dependence on the cost of vaccination $C$ for different values of subsidy $S$ on scale-free network. The trend is the same with Fig. 6.3. The parameter is $\epsilon = 0.7$.

implies that the parametric range of the disappearance of vaccinated people as well as the global invasion of the disease can be suppressed. In particular, for $S = 1.5$, even when the relative cost is increased to $C = 1.0$, there are still agents taking vaccination. If the relative cost $C$ has a moderate value ranging from $C_L$ to $C_H$, one can see, with the same relative cost $C$, that the vaccination coverage is increased dramatically as the subsidy level increases, which leads to a suppressed epidemic size. These results suggest that the introduction of subsidy to infected vaccinators is beneficial for elevating the total vaccination level and eliminating the spreading of disease.

Along this line, it is interesting to explore the variance of total subsidy and individual cost, which is specially instructive from the viewpoint of collective benefit. To answer this question, we present the color map encoding the fraction of vaccination ($a$), the total subsidy amount ($b$), the faction of infected vaccinators ($c$) and individual average cost ($d$) on the $C - S$ parameter plane in Figure 6.3, respectively.

From Fig. 6.3(a), we can clearly see that the trend is consistent with Figure 6.2: the improvement of subsidy level monotonically increases the vaccination coverage. Let us turn to the total amount of subsidies offered to infected persons who
get vaccinated. Fig. 6.3(b) displays a nonlinear phenomenon: intermediate price $C$ of vaccine leads to highest subsidy. For small relative cost $C$, ($C < 0.2$), all the individuals choose the vaccination as the best strategy irrespective of the subsidy amount (see Fig. 6.3(a)), which thus causes the same number of (on average $(1 - \varepsilon) \times N$) ineffectively vaccinated individuals susceptible to the disease (this point is supported in Fig. 6.3(c)). In this parametric region, the total subsidy amount naturally increases with the improvement of individual subsidy level. Interesting, for the moderate value of the relative cost $C$ and fixed subsidy level $S$, the total subsidy amount can reach the maximum value. Such phenomenon becomes much more salient as the subsidy level $S$ increases. Compared with the case of low price of vaccine, the enthusiasm of individual vaccination markedly declines (see Fig. 6.3(a)). Then, the initial herd immunity is also destroyed, which further enlarges the potential infection risk of un-effectively vaccinators (namely, the fraction of infected vaccinators reaches the maximum value, see Fig. 6.3(c)). This maximum fraction finally results in highest subsidy amount. If the relative cost $C$ is increased to a high level ($C > 0.85$), one can see that only a few or even none individuals choose vaccinate. In this case, the subsidy and faction of infected vaccinators become meaningless and return to 0. To show this non-linear relationship more evident, we select several typical values of subsidy $S$ in Fig. 6.4 as well.

Moreover, it is interesting to examine the phase diagram of the average individual cost in Fig. 6.3(d). One can see that, with the increment of $S$, the average individual cost monotonously decrease (namely, the total social expense declines as well). This trait is particularly evident for the middle cost of vaccine (i.e., $C \approx 0.6$), because the change of vaccination level is most acute in this range. Combining with the decreased social cost and increased total vaccination level, the meaning of this proposed framework becomes very obvious (especially from the economic viewpoint for collective benefit). Although there is certain cost of
subsidy (for government or other public organizations), the total social expense decreases with large extent. More importantly is that elevated vaccination level controls the spreading of disease.

To explain the aforementioned results, we monitor how four types of agents (namely, vaccinated and healthy, vaccinated and infected, unvaccinated and healthy, unvaccinated and infected) distribute by degree classes. Figure 6.5 shows the distribution of individuals at the steady state for different degree classes and subsidy values on scale-free networks. As it can be seen, the fraction of unvaccinated and infected individuals is continuously decreased as the subsidy level increases, regardless of the interaction degree. But more important is the fact that subsidy policy promotes the uptake of vaccination on large-degree nodes. For small subsidy, the disease holds the system, and the large-degree and middle-degree nodes

**Figure 6.5:** Distribution of individual states in dependence on degree for different values of subsidy $S$ on scale-free networks. From (a) to (c), the values of $S$ are 0, 0.5 and 1.0, respectively. The parameters are $\epsilon = 0.7$ and $C = 0.6$. 
are infected with high probability (even though vaccination action also takes place on these nodes). With the increment of subsidy $S$ (Fig. 6.5(b)), one can see that fraction of infection obviously declines, this is mainly because that large-degree and middle-degree nodes are more inclined to take vaccination. Of particular evidence, provided that the subsidy is sufficient large, it becomes extremely difficult for unvaccinated agents to occupy any node. Based on these analysis, we can conclude the competition of vaccination behavior on large-degree and middle-degree nodes is crucial for the elimination of disease. The subsidy policy considered here brings great benefit for large-degree and middle-degree vaccinators.

Aside from the effect of subsidy policy on the eradication of disease, another important question is whether the present observations are robust against the connectivity structure of human social networks. Interestingly, qualitatively identical results can be observed on other interaction networks. Results featured in Figure 6.6 and Figure 6.7 show how the faction of vaccination and the fraction of infection change as a function of the price $C$ of vaccine on Erdös-Rényi (ER) random network and square lattice for different subsidy $S$. Similarly to Fig. 6.2, it can be observed that the introduction and increment of subsidy $S$ monotonously elevate the vaccination level and reduce the infection risk. Simultaneously, the
critical threshold values $C_H$ and $C_L$ also keep the same trend with the case of scale-free network. Since the effect of homogeneous network on vaccination has been inherently identified as the worse case (Fu et al., 2011; Liu et al., 2012b; Wu and Zhang, 2013), the change of critical points is not sharp on square lattice. This point, however, does not affect the consequence that subsidy policy can be regarded as one universally effective method in promoting the vaccination level.

To give a more comprehensive understanding of the effect of subsidy policy on vaccination level and epidemic size, we visually inspect characteristic spatial distributions of four types of agents (namely, vaccinated and healthy, vaccinated and infected, unvaccinated and healthy, unvaccinated and infected) for different values of subsidy $S$. Figure 6.8 features the results obtained for $\epsilon = 0.7$ and $C = 0.5$, whereas for $S = 0$ (namely, no consideration of any subsidy) nobody chooses taking vaccination and the disease holds the whole system. This is consistent with the quantitative prediction of Fig. 6.7, where $C = 0.5$ is the threshold $C_L$ of vaccination vanishing out. While among these non-vaccinators, we can also see that the uninfected non-vaccinators (namely, the free-riders, denoted by yellow) are scattered and at most form small clusters, this is because there is no herd immunity contributed by immune agents. Interestingly, with the increment of subsidy...
Figure 6.8: Spatial distribution of different states under the subsidy policy on the square lattice. From (a) to (c), the values of $S$ are 0, 0.5 and 1.5, respectively. The color code is vaccinated and infected (green), vaccinated and uninfected (red), unvaccinated and infected (blue), unvaccinated and uninfected (yellow). The parameter are $\epsilon = 0.7$ and $C = 0.5$.

$S$, some people are inclined to taking vaccination (red and green), though part of them are still infected. In this case, we can see that due to the appearance of collective immunity protection for some agents, the free-riders extend more effectively, whose clusters become larger. It is natural that the dominance of epidemic (infected agents) declines. For very large value of $S$, even if the vaccinators fail to obtain immunity, they can also get enough monetary subsidy. In this case, more and more agents take the strategy of vaccination and even form clusters, which guarantee the herd immunity. As a result, the domain of free-riders expands to several giant clusters and the risk of infection is controlled greatly. Thus, these visual results suggest that with the increment of subsidy, successful vaccinators tend to form compact and large clusters, which induces larger domain of uninfected agents and reduce the survival space of disease.

6.3.2 Multiple impacts of vaccine efficacy on vaccination level

Different from the simple assumption of fixed efficiency of vaccination in the above section, another aspect that deserves our attention is the impact of vaccination efficiency. In realistic life, even if the same individual takes the identical vaccine,
the efficiency usually varies in different seasons due to the change of external environment. On the other hand, assuming the same vaccine is chosen by different people, the case will be more complex because of the difference of immunity system. Along this line, if the subsidy is taken into account, the effect of vaccine efficiency is more interesting.

6.3.2.1 Fixed subsidy

We first examine the impact of vaccine efficacy $\varepsilon$ on the vaccination level in the case of no implementation of subsidy policy. Fig. 6.9(a) shows how vaccination

![Figure 6.9: Fraction of vaccination in dependence on the efficiency of vaccination $\epsilon$ in the cases of non-subsidy, i.e., $S = 0$, (a); subsidy, i.e., $S = 1$ (b) and the difference of infected agents under these two cases (c) for different values of $C$ on the scale-free network.](image-url)
coverage varies as a function of the vaccination efficacy \( \epsilon \) for different values of the vaccine price \( C \) and without the subsidies (i.e., \( S = 0 \)). It is of particular evidence that middle efficiency \( \epsilon \) can guarantee an optimal vaccination level for low price \( C \) of vaccine. When the parameter \( \epsilon \) is small, no one chooses the strategy of vaccination, due to the low protection effect (namely, this is equal to the case of nonprofit expense). However, as the vaccine possesses a certain efficacy (namely, middle efficiency), to reduce the spread of epidemic people zealously choose vaccination, which makes the uptake reach the optimal level. With the continuous increment of vaccine efficiency, the dilemma starts to pose itself: (due to high efficiency of vaccine and temptation of high benefit) as long as most neighbors of individuals choose vaccination, the herd immunity is easily formed. In this case, the enthusiasm of few agents declines and total vaccination level decreases. Based on this point, it is predictive that, with the increment of vaccination cost, the vaccination coverage will reduce due to the further suppressed vaccination incentive for individuals. This is the reason why the optimal vaccination coverage level needs high efficiency as vaccine price increases (namely, with large \( C \) the peak of vaccination needs high efficiency and becomes less and less prominent).

According to the main aim of this chapter, it is subsequently interesting to study how the impact of vaccine efficiency changes if we introduce the subsidy policy. Fig. 6.9(b) shows the the fraction of vaccination in dependence on the efficiency \( \epsilon \) for different vaccine cost \( C \) at subsidy \( S = 1 \). Different scenarios are exhibited: all the individuals are more prone to taking the vaccination no matter how small the vaccine efficacy and what is the vaccine price. This is mainly because the subsidy amount is larger than the price of vaccine (namely, the provided subsidy can compensate the vaccination cost). In this case, even if the vaccinator is infected, he just need less cost (namely, vaccination is a rational option). However, when the vaccine efficiency becomes large, (due to the formation of herd immunity) the temptation as a free-rider appears as well, which leads to the decline of vaccination
Figure 6.10: Distribution of individual states in dependence on degree for different values of vaccine efficiency $\epsilon$ in the case of non-subsidy, i.e., $S = 0$, on scale-free networks. From (a) to (c), the values of $\epsilon$ are 0, 0.5 and 0.8, respectively. The parameters is $C = 0.2$.

level. In fact, the larger the cost of vaccination, the earlier the emergence of such a temptation. This explains why such decline of vaccination coverage becomes much evident for the relatively large vaccination cost.

Besides the impact for the vaccination uptake, it is crucial to investigate what effect vaccine efficiency does bring to the epidemic size. To answer this question, we display the difference of infection fraction between the non-subsidy case (namely, Fig. 6.9(a)) and subsidy case (namely, Fig. 6.9(b)) versus the efficiency $\epsilon$ in Fig. 6.9(c). One can clearly find that, irrespective of the values of cost $C$ and efficiency $\epsilon$, the non-subsidy case always incurs larger attacks than that of the subsidy case, which again validates the conclusion of above section that the
consideration of subsidy is beneficial for eliminating the infection risk. Moreover, it is more interesting that, regardless of the value $C$, there always exists a moderate efficiency $\epsilon$, which can maximally reduce individual infection under the framework of subsidy policy. Such a phenomenon can easily get the understanding from the vaccination level in Figs. 6.9(a) and (b). Before the difference of infection fraction reaching the peak, nobody (nearly each agent) prefers to choose the non-vaccination (vaccination) behavior in the case of non-subsidy (subsidy) case, which thus induces that the difference of infection fraction become larger with the increment of efficiency $\epsilon$. But after these critical values of $\epsilon$, the trend completely differs: agents start to choose vaccination in the non-subsidy case (Fig. 6.9(a)) and the coverage level of vaccination declines in the subsidy case (Fig. 6.9(b)) which induces the difference of vaccination level decrease. Correspondingly, the

Figure 6.11: Distribution of individual states in dependence on degree for different values of vaccine efficiency $\epsilon$ in the case of subsidy, i.e., $S = 1.0$, on scale-free networks. From (a) to (c), the values of $\epsilon$ are 0, 0.5 and 0.8, respectively. The parameters is $C = 0.2$. 
difference of infection fraction also declines.

To further explain the impact of efficiency on vaccination campaign, we investigate how the four types of agents (namely, vaccinated and healthy, vaccinated and infected, unvaccinated and healthy, unvaccinated and infected) distribute by degree classes. Figure 6.10 shows the distribution of individuals at the steady state for different degree classes and vaccine efficiency $\epsilon$ in the non-subsidy case ($S = 0$). For small $\epsilon$, it is clear that nobody choose the vaccination (green and yellow) on each degree node. At variance, if the middle efficiency $\epsilon$ is introduced, one can see that the enthusiasm of vaccination is greatly promoted, especially for large-degree and middle-degree nodes where vaccination behavior becomes absolutely
dominated. With sufficient large efficiency, though there is obvious elevation for successful vaccinators (green), the total vaccination level slightly declines on large-degree and middle-degree nodes. In addition, we can also see that, irrespective of the degree of nodes, epidemic size (yellow and blue) reduces with the increment of vaccine efficiency $\epsilon$.

But when we turn to the case of subsidy case, the distribution of four states becomes completely different. As shown in Fig. 6.11, for small efficiency $\epsilon$, all the nodes will unconditionally choose the vaccination option, due to fact high subsidy can compensate the cost of vaccination. Among these vaccinators, the unsuccessful agents (yellow) accounts for the large proportion on large-degree and middle-degree nodes. With the increment of $\epsilon$, total vaccination still keeps the same level, but successful vaccination becomes the main trait, especially for small-degree nodes (green). Once sufficient high efficiency is considered, we can see that though the total vaccination coverage decreases, the successful rate of vaccination is greatly elevated on large-degree and middle-degree nodes. This guarantees the formation of herd immunity and there is slight drop for the fraction of successful vaccinators on small-degree nodes. Of course, with the increment of efficiency, disease (yellow and blue) are further controlled.
Figure 6.14: Spatial distribution of different states under the subsidy case, i.e., $S = 1.0$, for different values of $\epsilon$ on the square lattice. From (a) to (c), the values of $\epsilon$ are 0.1, 0.35 and 0.8, respectively. The color code is vaccinated and infected (green), vaccinated and uninfected (red), unvaccinated and infected (blue), unvaccinated and uninfected (yellow). The parameter is $C = 0.1$.

Similar to previous treatment, we also inspect how the faction of vaccination and epidemic size change on the square lattice. Figure 6.12 features vaccination coverage varying as a function of the vaccination efficacy $\epsilon$ for different values of the vaccine price $C$ without and with subsidy in (a) and (b). Importantly, qualitatively similar observation is obtained. If there is no any subsidy, middle vaccine efficiency can guarantee an optimal vaccination level for small cost $C$, but this trend converts into one monotonously increasing curve with the increment of efficiency for large cost. On the contrary, provided that the subsidy policy is taken into account, vaccination enthusiasm is sharply driven to an extremely high level but declines with the elevation of efficiency. This is because high efficiency boosts the temptation of free-rider due to the formation of herd immunity. The larger the value of $C$, more obvious the downfall. Finally, when we explore the epidemic size (see Fig. 6.12(c)), we also find that the case with subsidy can notably reduces the infection risk compared as the case without any subsidy, while this effect is of particular evidence at middle efficiency, irrespective of the value of cost.

It remains instructive to visually examine the spatial distributions of four types of individuals (namely, vaccinated and healthy, vaccinated and infected, unvaccinated and healthy, unvaccinated and infected) for different efficiency $\epsilon$ without and with subsidy. Figure 6.13 features the results for $S = 0$ and $C = 0.1$, whereat for small
Figure 6.15: Fraction of vaccination and infection in dependence on the efficiency of vaccination $\epsilon$ for different values of subsidy $S$ on the scale-free network. The parameter is $C = 0.5$.

If nobody chooses vaccination but very minority luckily escape from the infection as the free-riders (yellow). With middle $\epsilon$, more people are inclined to taking vaccination and form vaccination clusters (red and green), but due to limited efficiency, some of them still fail to get the immunity. In this case, the herd immunity is not complete (namely, the expansion of free-riders is limited), the epidemic just decreases to a slightly low level. However, if the vaccine possesses high efficiency, though the coverage of vaccination reduces again, high successful rate guarantees the formation of complete herd immunity, which induces better control of epidemic and larger space for free-riders.

We now turn to the case of subsidy with different vaccine efficiency, and Fig. 6.14 shows the spatial distributions of four types of agents. Obviously, low efficiency can greatly promote the enthusiasm of vaccination (red and green). As the efficiency $\epsilon$ increases, the giant cluster of vaccinators start to collapse, and even becomes very small segments. But at the same time, one can see that herd immunity becomes more and more notable, since the free-riders around the successful vaccinators fast expand and the number of infected agents (green and blue) declines.
6.3.2.2 Fixed cost

Except for the case of fixed subsidy, in what follows we will explore the effect of vaccine efficiency on vaccination coverage and epidemic size for different subsidy yet fixed cost. In this case, we can predict if the subsidy exceeds the cost of vaccine, people may be inclined to taking vaccination, while the epidemic size should be still closely related with the concrete efficiency. We will first explore these cases on scale-free network and square lattice and then give the microscopic analysis and visual observation.

Figure 6.15 features how the fraction of vaccination and infection vary as a function of efficiency $\epsilon$ for different values of subsidy $S$ on scale-free network. It is clear that, with respect to fixed cost, vaccine efficiency brings multiple effect as well, which is closely related with the subsidy. For small subsidy ($S = 0.4 < C$), no vaccination becomes a popular and rational option at low efficiency. In this case, even if an agent chooses vaccination, he still may be infected with high probability, then the total cost ($C + 1$) enormously exceeds the subsidy. Thus the infection risk is maintained at a high level till the vaccine efficiency can sufficiently provide protection for individual safety. When elevation of subsidy is considered ($S = 0.8$),
Figure 6.17: Distribution of individual states in dependence on degree for different values of vaccine efficiency $\epsilon$ in the case of middle subsidy, i.e., $S = 0.8$, on scale-free networks. From (a) to (c), the values of $\epsilon$ are 0.1, 0.4 and 0.9, respectively. The parameters is $C = 0.5$.

one can see that the enthusiasm of vaccination is fast inspired to a relatively large level at the middle efficiency. Due to broad existence of vaccination behavior, herd immunity is formed in the system. But at the same time, some people start to face the temptation as free-riders: no-cost (no-vaccination) yet enjoying the protection from other vaccinators. In fact, the higher the efficiency of vaccine, more obvious this trend. Under the impulse of this temptation, though infection risk is better controlled because of higher efficiency, the vaccination coverage gradually declines (namely, there is a slight peak of vaccination). Different from these cases, if we introduce large enough subsidy, more interesting observation takes place. Under this scenario, even if the efficiency is very low, the desire of vaccination is completely motivated, which indicates that all the agents choose the strategy of
vaccination. Simultaneously the infection risk quickly decreases with the increment of efficiency. However, when the efficiency reaches an intermediate value, a counter-intuitive phenomenon appears: part of individuals face the temptation as free-riders in virtue of the formation of herd immunity, the uptake of vaccination start to decline, which in turn cause more people to be infected (namely, there exist a minimal point for the infection). This phenomenon is further amplified by the increment of efficiency, at which case, the temptation of free-riders becomes larger, vaccination uptake declines faster and infection risk is more prominent.

Interestingly, if we turn to the homogeneous network, the similar phenomenon is still maintained. Figure 6.16 shows the faction of vaccination and infection independence on the efficiency for different subsidy on square lattice. For small

\( \text{Figure 6.18: Distribution of individual states in dependence on degree for different values of vaccine efficiency } \epsilon \text{ in the case of high subsidy, i.e., } S = 1.0, \text{ on scale-free networks. From (a) to (c), the values of } \epsilon \text{ are 0.1, 0.4 and 0.9, respectively. The parameters is } C = 0.5. \)
subsidy \((S = 0.25)\), one can see that vaccination campaign is just elevated to a certain level at large efficiency, which but is less than the case of scale-free network due to the inherent property of network. At middle subsidy, though there is better improvement of vaccination level and epidemic risk control compared with the case of small subsidy, the optimal phenomenon of vaccination become slightly unconspicuous. But if the large subsidy is taken into account, the counter-intuitive observation occurs again: the vaccination level decreases with the increment of efficiency, while an intermediate efficiency value can guarantee an minimal infection risk. As such, combining with all the observations on scale-free network, random network and square lattice, we can conclude designating subsidy policy as being universally effective in the management of epidemic control.

Moreover, it is worth mentioning that, in Fig. 6.15 and Fig. 6.16, all the results converge to the same value at \(\epsilon = 1\), irrespective of the values of subsidy \(S\). This is actually easy to be understood. Because at this point any vaccinator can successfully resist the infection of disease, subsidy has lost its initial function.

To have a better understanding about the impact of efficiency on vaccination level and epidemic size, we explore how four types of agents (namely, vaccinated and healthy, vaccinated and infected, unvaccinated and healthy, unvaccinated and
Figure 6.20: Spatial distribution of different states in case $S = 0.8$ for different values of $\epsilon$ on the square lattice. From (a) to (c), the values of $\epsilon$ are 0.05, 0.25 and 0.8, respectively. The color code is vaccinated and infected (green), vaccinated and uninfected (red), unvaccinated and infected (blue), unvaccinated and uninfected (yellow). The parameter is $C = 0.5$.

infected) distribute by degree classes in scale-free networks. Figure 6.17 exhibits the distribution of individuals at the steady state for vaccine efficiency and subsidy values on scale-free networks. Because the trend of small $S$ and middle $S$ is similar (see Fig. 6.15), we mainly focus on middle and large $S$ here. For low efficiency, all the nodes choose the non-vaccination behavior, irrespective of the degree. Among them, just minority of small-degree nodes luckily escape from the infection. While for the middle efficiency, one can see that infection risk obviously declines to a low level. Though vaccination level is elevated to a relative high level (yellow and green), but failing vaccinators still hold the majority. However, at high efficiency, the adverse case takes place: successful vaccinators can provide sufficient protection to form herd immunity, which leads to smaller epidemic.

Interesting, if we add the compensation for failing vaccinators (namely, large subsidy $S$), the counter-intuitive phenomenon takes place in Fig. 6.18. Due to the guarantee of high subsidy, all the people unconditionally choose vaccination behavior to depress the infection risk at low vaccine efficiency, though most of them do not escape from the fate of being infected. With the further increment of vaccine efficiency, in view of maintaining the initial high vaccination coverage, the infection risk (yellow and blue) further decreases. Distinguishing from the case of low efficiency, we can see that successful vaccinators hold high proportion, which
is specially evident for small-degree nodes. However, provided that the sufficiently high efficiency is considered, the total trend changes: due to the temptation of having no contribution but hitchhiking others’ immunity achievement as the free rider, the enthusiasm of vaccination and the successful vaccination rate decline for the nodes of all the degrees. In turn, epidemic starts unboundedly outbreak, which holds all the nodes as the dominated state.

Finally, a remaining question of importance is to visually examine the spatial distribution of four types of agents on spatial network. Figure 6.19 unveils the spatial patterns of individuals for different values of $\epsilon$ at $C = 0.5$ and low subsidy $S = 0.5$. Since the absence of stimulation of high efficiency and large compensation, nobody is likely to taking vaccination. The epidemic holds the whole system except for the existence of few scattered free-riders (yellow). With the increment of efficiency ($\epsilon = 0.25$), a part of population start vaccination to decrease the infection, but many of them still fail to get the immunity and epidemic just get suitable control. When the high efficiency is considered, we can see though the total vaccination level has a slight decline, the successful vaccinators increase and they are always surrounded by the large clusters of free-riders. In this sense, the infection extent further shrinks. However, if the large subsidy is introduced, the impact of efficiency on spatial patterns is completely different. From Figure 6.20, all the agents choose the option of vaccination at low efficiency, in spite of more infections among them. This is because more economic compensation motivates unconditionally vaccinating. With middle efficiency, it is clear that total vaccination level declines but the epidemic (green and blue) gets control to very large extent. At the same time, we see that the successful vaccinators (red) tend to form compact clusters and are located in the center of giant free-riders clusters, which is the indication of herd immunity. For sufficiently large subsidy, the interestingly counter-intuitive phenomenon appears: infection risk does not decline but rather enhances, and successful vaccinators split into small slices. Under this case, each
agent dreams to get protection from herd immunity, which results in the fact that just few people invest to vaccination behavior and risk is elevated.

6.4 Conclusion

Different from the simple assumption that the vaccination is fully effective in previous chapters, the incomplete vaccine is considered here, which is specially suitable for the disease whose infection virus has high mutation rate, such as, seasonal influenza. In the vaccination campaign, even if agent chooses vaccination, he may still be infected due to limited protection, whose efficiency is denoted by the parameter $\epsilon$. To control the spreading of disease and promote the enthusiasm of vaccination, we introduce the subsidy policy, which exists in realistic life. Under such a case, the vaccinators failing to get immunity can get the compensation even larger than the cost of vaccination.

Through a great number of computer simulations, we have shown that the introduction of subsidy can promote the vaccination level in the population, which is further elevated with the increment of subsidy. Along with the enhancement of vaccination coverage, the infection risk and epidemic size decline and vanish. On the other hand, it is also interesting that though there is some cost for subsidy (or even maximal cost), the total social expense drops to a relatively low level, which is consistent with the desire of society. In this sense, this theoretical research outcome may be helpful for governments or communities to design effective policy during the epidemic management. We show that this mechanism is robust or universally effective on different networks. For heterogeneous scale-free network, this aim is mainly realized by promoting large-degree and middle-degree nodes vaccination with the increment of subsidy. While on homogeneous square lattice,
the emergence of herd immunity is based on the formation of compact clusters of successful vaccinators.

Then, fixing the amount of subsidy or cost of vaccine, we explore the impact of vaccination efficiency and find the multiple effect. First fixing the amount of subsidy, we unveil that for traditional case (namely, no subsidy) intermediate efficiency can lead to an optimal vaccination level for small cost of vaccine, but this trend change into the monotonous increase with efficiency at high vaccine price. Interestingly, when subsidy is introduced, the vaccination passion monotonously decreases with the efficiency, irrespective of the expense of vaccination. Comparing the infection between these two cases, we find that the middle efficiency can, to large extent, decrease the infection risk under the case of subsidy policy. Turn to case of fixed cost of vaccine, we find that for low and middle subsidy, the increment of efficiency enhances the vaccination and enables the infection risk monotonously decline, which is consistent with our expectation. However, if the subsidy is sufficient large, the efficiency produces one counter-intuitive observation: though vaccination coverage decrease with efficiency, but the intermediate rather than high efficiency leads to a minimal risk. This is because that the herd immunity declines under the temptation of free-riders.

Though our research just considers the theoretical viewpoint, it is very inspiring to help improving and constituting related policy. In realistic life, the subsidy event is also popular, but not necessary to failing vaccinators, which means that there may be many types of subsidy. We hope that this modeling work can shed light into subsidy policy in more details and even combines with the empirical data in future.
Chapter 7

Conclusions and research outlook

7.1 Contributions of the thesis

Combining behavioral dynamics into spatial epidemiology, this thesis further develops the new framework for the disease research: behavior-disease interactions on complex networks. Under this framework, individual behaviors decision for the protection measures (such as, taking vaccination, reducing the contact with infected agents) can be pictured by game theory. Similarly, each selection possesses its own payoff function. Through modeling the realistic scenarios and computational study, two nontrivial and related issues about the control of disease spreading are addressed:

(i) How does the interplay between behavior and disease affect the disease dynamics and infection risk, especially compared with the result of traditional epidemiology research?

(ii) How does this framework shed new light into the social management with disease outbreak, such as constituting public health policy?
The main contents and contributions of this thesis can be summarized as follows:

(1) Risk perception plays a crucial role in the infection dynamics and disease control. Different from previous setup (Zhang et al., 2010; Perisic and Bauch, 2009b,a), we have proposed to study the risk perception involving both the local and global infection information via the so-called precaution level in Chapter 3. Interestingly, the precaution level can greatly promote vaccination uptake and eliminate the disease, irrespective of efficiency period of vaccination. This newly finding is caused by the positive feedback mechanism motivated by small-degree nodes. To large extent, this study enriches the content of disease prevention, which is justifiable with realistic cases.

(2) Though vaccination is regarded as the most popular prevention measure for disease, it usually needs to spend relatively high cost. Inspired by this fact, we have explored the effect of temporary self-protection scheme in Chapter 4, which possesses the advantage of low cost yet partial protection effect. Through numerical computations, we have unveiled that this new strategy can produce a multiple effect on the eradication of disease, namely, middle efficiency or cost of self-protection strategy enlarges the infection risk. While for this multiple effect, it is mainly induced by the loss of positive feedback mechanism between prevention measure and infection. Since the temporary self-protection measures are ubiquitous in our life, this proposal can provide new sight for the emergency cases, such as, vaccination being not enough.

(3) In our life, we can observe the case that the vaccination is not immediately available during the outbreak of some severe diseases. Motivated by this point, we have explored a new strategy, adaptive protection, in Chapter 5. Under such a scheme, health agents can cut the connections with infected neighbors. We have found that the increment of adaptive protection efficiency depresses the infection risk, and intermediate efficiency can support an optimal level of pruning
the infected links. With the increment of adaptive-protection efficiency, the giant infection cluster gradually collapses and turns to the expansion of giant cluster of health agents. While for the influence of transmission rate, middle value can make the giant infected cluster collapse most effectively. Obviously, this setup of adaptive protection belongs to the co-evolution scenario, which further extends the scope of spatial behavior-disease research.

(4) Besides the above achievements which focus on the individual benefit, it is also constructive to consider the problem from the viewpoint of benefit of the whole population. In this sense, we have proposed the subsidy policy in Chapter 6, where the vaccine is not completely effective. If the vaccinated agent is infected, he can get the subsidy because of his contribution to public health. Interestingly, we have exhibited that the increment of subsidy can greatly promote the vaccination level, which eventually controls the infection. Except for this finding, the total social expense also falls to a relatively low level, which is consistent with our desire. While for the vaccination efficiency, it will lead to a multiple effect, which is related the balance between vaccination and subsidy. Totally, this research idea may shed light to the establishment of real policy.

7.2 Research outlook

In order to explore the impact of behavior on disease dynamics, we have incorporated game theory into epidemiology on complex networks. As described above, we have unveiled that the disease-behavior interactions can greatly affect the final state of infection. In spite of these achievements, there are still many unexplored problems related to behavior-disease feedback problems that deserve further attention. We will discuss the potential directions for future exploration.
This thesis is mainly from the theoretical viewpoints, so the first and most obvious challenge for further research is whether we can collect empirical data and combine the models with these data. Using the appropriate data, we can validate the accuracy of theoretical models and disease parameter selection. If these models are reasonable, we can further predict the behavioral parameters of realistic cases. Then, the behavior-disease models can become more useful policy supporting tools.

Secondly, as is well-known, social dynamics, including information propagation and opinion formation, plays a crucial role in the perception of risk and the safety of prevention measures (such as, vaccination), but there exists no uniform frame to incorporate more details into a new framework. Under the new framework, it will be hopeful to get uniform criterion which can guarantee the information safety of more agents and to prevent the infection more effectively. On the other hand, when facing the disease outbreak, it may be beneficial for immediately designing effective control measures with opinion formation networks.

Thirdly, it may be interesting and constructive to introduce the behavior-disease framework into multilayer networks. Different from the traditional assumption of single network, many realistic systems are usually composed of several layered networks. In the multilayer viewpoints, we may consider that each layer will have its own function. For example, some layers are used for the disease transmission, some others may be used for the propagation of vaccination information, while the others may be employed for information flow regarding disease. By assuming each layer has its own dynamics for one process, we can more clearly explore how two or more dynamics processes located at different layers affect the prevention measures and disease spreading. In this sense, we can also research the problems from both theoretical and empirical aspects.
Along this research line, another non-negligible problem is considering the co-evolutionary scenarios on the multilayer networks. Under these cases, how the disease spreading threshold varies becomes of particular interest, because the topology dynamics and disease dynamics jointly affect individual behavior selection. Moreover, if the co-evolution models can combine with empirical data, we may get better prediction and control schemes for some severe diseases.

Finally, the remaining problem needing our particular attention is that, in the present thesis, we mainly focus on computational simulations yet ignore the statistical analysis in more details. While in reality, many analysis is very important to validate the computational results. This is one point that we need to adjust in future.

If we can further develop our theory and get the validation of empirical data, the behavior network epidemiology will a useful tool for the evaluation of realistic disease spreading and the constitution of prevention measures of governments.
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