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A dynamic vaccination strategy to suppress the recurrent epidemic outbreaks

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ABSTRACT

Efficient vaccination strategy is crucial for controlling recurrent epidemic spreading on networks. In this paper, based on the analysis of real epidemic data and simulations, it’s found that the risk indicator of recurrent epidemic outbreaks could be determined by the ratio of the epidemic infection rate of the year to the average infected density of the former year. According to the risk indicator, the dynamic vaccination probability of each year can be designed to suppress the epidemic outbreaks. Our simulation results show that the dynamic vaccination strategy could effectively decrease the maximal and average infected density, and meanwhile increase the time intervals of epidemic outbreaks and individuals attacked by epidemic. In addition, our results indicate that to depress the influenza outbreaks, it is not necessary to keep the vaccination probability high every year; and adjusting the vaccination probability at right time could decrease the outbreak risks with lower costs. Our findings may present a theoretical guidance for the government and the public to control the recurrent epidemic outbreaks.

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1. Introduction

A challenging problem in epidemiology is how to understand epidemic spreading dynamics [1–6] and design informing prevention strategies so that the outbreaks of epidemics could be suppressed or reduced effectively. This problem has got great attention in statistical physics and many other disciplines [2,7,8]. According to the percolation theory, the simplest strategy to suppress the outbreaks is to immunize individuals uniformly or randomly, which the implementation do not require preparation or information at all [9,10]. However, it was revealed that random immunization is inefficient for heterogeneous networks. And then, many more effective immunization strategies were developed, ranging from global strategies such as targeted immunization based on node degree [10] or betweenness centrality [11], to local strategies such as acquaintance immunization [12] and (bias) random-walk immunization [13,14], and to some others in between [15]. Later, the graph partitioning [16] and optimization of the susceptible size [17] became the new improvements. Besides the degree heterogeneity, community structure has a major influence on disease immunity [18,19]. Recently, based on the message-passing approach, an optimal set of nodes for immunization [20] was found, and the immunization was mapped onto the optimal percolation problem [21]. Inspired by the idea of explosive percolation, an “explosive immunization” method has been proposed [22]. In contrast to the above approaches, recent research increasingly explored the pivotal implications of individual behavior in populations, such as reducing the risk of infection by adaptive rewiring the links incident to infected individuals [23], information-driven vaccination [24], and the dynamical interplay between awareness and epidemic spreading [25]. These strategies can theoretically prevent the prevalence of an epidemic and thus bring us a huge hope of controlling an epidemic.

All the above studies are focused on the prevention of a single outbreak of epidemic. However, in realistic situations, the empirical data shows that some epidemics are recurrent, i.e., it will outbreak from time to time [26–29], such as SARS (Severe Acute Respiratory Syndrome), H1N1 (Swine Influenza), H5N1 (Avian Influenza), Ebola, and MERS (Middle East Respiratory Syndrome) etc. If these diseases do not confer immunity, individuals would be infected over and over again. Therefore, an effective dynamic vaccination strategy to suppress the outbreak of recurrent epidemic is called for. As we all know, a large vaccine coverage can greatly reduce the risk of epidemic outbreak, but it increases the health-
care costs and the financial burden of the public health authorities greatly. In fact, the outbreak of recurrent epidemic is not always seasonal and there is even no outbreak in some years [26]. Therefore, we are wondering whether the immune coverage can be adjusted according to the outbreak risk every year so that the recurrent epidemic outbreak can be suppressed to the greatest extent at lowest cost. More precisely, is it possible to improve the immune coverage before a higher risk of epidemic outbreak and decrease the immune probability in lower risk case?

To achieve the above goal, in this paper, we firstly introduce a network model of SIRS to reproduce the spreading pattern of recurrent epidemic. Then, we try to find out the risk indicator of epidemic outbreak based on our proposed model. Lastly, according to the risk indicator, a dynamic vaccination probability is designed year by year to suppress the epidemic outbreak. Our simulation results show that the dynamic vaccination strategy is much more effective than the other vaccination ones. Particularly, the dynamic vaccination could decrease the maximal and average infected density but increase the time intervals of epidemic outbreak and individuals attacked by epidemic at very low cost. These findings are very useful for public health authorities to optimize vaccination and drug delivery plans.

The paper is organized as follows. In Section 2 the network model and the epidemic spreading model are introduced. Next in Section 3 the risk indicator of epidemic outbreak based on our proposed model are determined. Based on the risk indicator, a strategy with dynamic immune probability is proposed in Section 4. Then, a theoretical analysis based on Markov dynamics is presented to explain the numerical results in Section 5. Finally the main conclusions and discussion are addressed in Section 6.

2. The recurrent epidemic model

In this section, the recurrent epidemic spreading model on complex network is produced. In a network, each node represents an individual, and the epidemic spreads among them along the connections. Here, we take the uncorrelated configuration model (UCM) as an example. By following Ref. [30], we construct the UCM network with size $N = 1000$ and the degree distribution $p(k) \sim k^3$, where the degree $k$ is limited in the range $k \in (4, \sqrt{N})$.

In the study of epidemic spreading, scientists design many infectious disease model [2], and the most widely used models are SIS model (Susceptible – Infected – Susceptible) and SIR model (Susceptible – Infected – Recovered). These two models in the process of transmission system may eventually reach a steady state [31], namely the number of susceptible, infected and refractory individuals are (almost) constant. But in reality it’s well known that some diseases exist for a long time and fluctuate occasionally, such as the Influenza. Influenza virus persists in the population and causes outbreaks of disease once in a while. Therefore, the two classic epidemic models are not suitable for the transmission of recurrent epidemic.

Recently, Zheng et al. [26] have designed a model of recurrent epidemic spreading, which takes into account environmental factors and seasonal variations in the infected rate. In particular, as is shown in Fig. 1, susceptible individuals may be infected with the probability $\beta$ by the infected neighbors, and it may also be infected by the virus in the surrounding environment at a certain probability $\mu$. Then the infected individuals are recovered to refractory state with the probability $\mu$. And then the refractory individuals will change into susceptible state with the probability of $\delta$. In our simulation, $\mu$ and $\delta$ are fixed at 0.20 and 0.02. In this model, when $p_0 = 0.00$, the infected density will decay to zero, and when $p_0 = 0.01$ the infected density can be sustained and can reproduce the recurrent behaviors of non-periodic epidemic patterns, thus $p_0$ is set as 0.01. For the infection rate $\beta$, it will change every 52 time step (corresponding to one year, and one time step represents one week), since the strain of influenza virus change every year. The value of the infection rate in each year is randomly chosen from the truncated Gaussian distribution with average $\langle \beta \rangle$ and standard deviation $\sigma$. In this paper, $\langle \beta \rangle = 0.1$, $\sigma = 0.1$ are fixed and only positive $\beta$ is reserved.

3. The risk indicator of the recurrent epidemic outbreak

The ultimate aim of the study on epidemic spreading is to control it. To avoid infecting from some epidemics, such as influenza, vaccination is advised every year. It’s believed that to prevent the influenza outbreaks the best way is to keep the immune probability as high as possible. However, high probability of immunization will cost greatly and the risk of influenza outbreaks may not be high every year. Then a question is addressed: Is it possible to regulate the immune probability of each year with the outbreak risk? That is to say, when the outbreak risk is high, the immune probability is large, and vice versa. If this strategy is effective, outbreak could be controlled at a lower cost. To realize this idea, first of all, the risk indicator of recurrent epidemic outbreak should be identified.

In our former work [26], it has been found that the recurrent outbreaks of epidemic depend not only on the infection rate but also on the density of susceptible individuals. Each year, the World Health Organization (WHO) [32] provides formal recommendation for the composition of influenza vaccines based on the information provided by the WHO Global Influenza Surveillance and Response System, which makes the estimation of the basic reproduction number $R_0$ [33] possible to some extent. It may reflect the infected rate in an indirect way. But in reality, it’s hard to count the number (or density) of the susceptible individuals. We believe there must be something else that could indicate the high density of susceptible individuals. For this purpose, we turn to study the time series of the weekly consultation rates of influenza-like illness (per 1000 consultations) from 1999 to 2013 in Hong Kong for the General Out-Patient Clinics (GOPC) and the General Practitioners (GP) [34], which are shown in Fig. 2. From the figures we noticed an interesting phenomenon: in a period before most outbreaks (not all) the average consultation rate is lower than the average one, which are marked by the light blue ellipses. The consultation rate could be regarded as the density of infected individuals, and the low infected density in a period means less individuals to be in refractory state in the next period, as a result more individuals are in the susceptible state, i.e., low infected density in a period will make large susceptible density in the next period. This may be the reason for the above phenomenon.

In Ref. [26], we have presented a model to simulate non-periodic outbreaks of recurrent epidemics, and it has been proved that the model could well simulate the influenza outbreak process. Inspired by this model, we are wondering whether the phenomenon that lower infected density before outbreak could also be detected in this model. Fig. 3(a) and (b) show the evolution of infection rate $\beta$ and the infected density $\rho_1$ in our model, respectively. The horizontal black line represents the average infection rate $\langle \beta \rangle$. Arrows 1, 3 and 4 indicate the typical outbreaks and the light blue ellipses indicate the infected density before the outbreaks. The simulation time series can well reflect the non-periodic and persistently outbreak features (see Fig. 2). What’s more, it’s also found that in a period before most outbreaks (not all) the infection density is relatively lower (marked by light blue ellipses), which is similar to the phenomenon in the real data (see Fig. 2). However, both the simulations and real data indicate that not all the lower infection density will indicate the emergence of the outbreaks in the coming period. Thus, lower infected density in the
previous year do have some relations to the outbreaks in the coming year, but it's not the sufficient condition for the outbreaks.

It is noting that the infected rate $\beta$ is controllable in the model, which makes the investigation of the relations between $\beta$ and the epidemic outbreaks possible. After carefully studied, it's found that a very large $\beta = 0.296$ does not always induce the epidemic outbreak (example indicated by arrow 2). But sometimes epidemic may outbreak even $\beta$ is rather smaller than 0.296 (see arrow 1, 3 and 4 in Fig. 3). Therefore a large $\beta$ is not the sufficient condition for the outbreak, either.

Neither the lower infected density in the former year nor the larger infection rate is the sufficient condition for the outbreaks, then what on earth are the relations between epidemic outbreaks and the infected density in the former year $I_{t-1}$ and the infection rate $\beta$? To answer this question, Fig. 4(a) and (b) plot the maximal infected density $I_{t,max}$ vs. the infection rates $\beta$ and the densities of infectious individuals $I_{t-1}$ in the former year before epidemic outbreaks. From Fig. 4(a), it can be seen when influenza outbreaks, most infection ratios are larger and shows weakly positive correlation. However, most densities of infectious individuals in the former year are smaller and shows weakly negatively correlation. Therefore, epidemic outbreak may appear at large $\beta$ and small $I_{t-1}$. From the above analysis we naturally guess that the maximal infected density is mainly determined by the ratio $\frac{I_{t-1}}{I_{t}}$, and the larger this ratio is the larger the maximal infected density could be. To test our assumption, the maximal infected density vs. the ratio is

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**Fig. 1.** Schematic figure of the extended SIRS model. The symbols $S$, $I$ and $R$ represent the susceptible, infectious, and refractory states, respectively, and the parameters $\beta$, $\mu$ and $\delta$ represent the infectious, refractory and recovery rates, respectively. $p_0$ represents the probability that a susceptible node will be infected by environment or other factors.

**Fig. 2.** The time series of the weekly consultation rates $C$ of influenza-like illness (per 1000 consultations) from 1999 to 2013 in Hong Kong for the General Out-Patient Clinics (GOPC) and the General Practitioners (GP), respectively, where the data from 2009/6/13 to 2010/5/23 in (a) are not available. The light blue ellipse indicates the low infected density before a outbreak period. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)
plotted in Fig. 4(c). It is easily to observe that the maximal infected density is strongly correlated to the ratio $p_i \tau_i$ (correlation coefficient $\tau = 0.77$). From the above investigation, it concludes that the combination of the large infection rates and low infected density in the former year is the necessary conditions for the recurrent epidemic outbreaks. Thus, the risk indicator of the recurrent epidemic outbreak could be predicted by the ratio $p_i \tau_i$.

4. Varying vaccination probability to suppress the epidemic outbreak

Vaccination is the most effective way to prevent influenza and reduce its impact. In consideration of the virus infection rate changes every year, people are advised to vaccinate before influenza season of the year. Large vaccination probability is an effective way to suppress the epidemic outbreaks, but it will cost much. It’s known that large infection rate and small infected density in the former year may induce the epidemic outbreaks, thus the risk indicator of epidemic outbreaks could be estimated by the ratio $p_i \tau_i$, which make it possible to regulate the vaccination probability accordingly. Therefore, with the risk indicator on hand, we will perform the varying vaccination in the following work. Apparently, the vaccination probability $p_i(\tau)$ in the yth year should be positively correlated to the ratio $p_i \tau_i$, and the simplest form is

$$p_i(\tau) = \frac{\beta}{\rho_i \rho_f}.$$ 

Unfortunately, it’s hard to determine the parameter $\alpha$, if $\alpha$ is small the epidemic outbreaks can’t be depressed and if $\alpha$ is large, $p_i(\tau)$ will increase beyond 1. Thus, to make $p_i(\tau)$ positively correlated with $p_i \tau_i$ and meanwhile make $p_i(\tau)$ changes in the range $[0.0, 1.0]$, $p_i(\tau)$ in the yth year is defined as following:

$$p_i(\tau) = 1 - e^{-\alpha p_i \tau_i}$$

(1)

where $\alpha$ is a tunable parameter. In our simulation, $\beta$ in a year is obtained at the end of the former year and $\rho_i \rho_f$ is obtained by averaging the infected density of the former year. Thus, the average vaccination probability ($p_i$) in a long time can be tuned by $\alpha$. The changes of $p_i(\tau)$ with $p_i \tau_i$ can be seen in Fig. 4(d), obviously, $p_i(\tau)$ is positive to $p_i \tau_i$ and changes between 0.0 and 1.0.

To investigate how the vaccination probability affects the influenza spreading, firstly the epidemic spreading model introduced above evolves freely for 20,800 weeks (400 years), and then with probability $p_i(\tau)$ those individuals who are not in infected state are randomly selected to take vaccination at the beginning of each year. In our simulation, the epidemic outbreak is defined as $m + 3s$ with $m$ and $s$ being the mean and standard deviation of the infected density without any vaccination, respectively [27]. The change of infected density $\rho_i$ with time $t$ before and after the vaccination is plotted in Fig. 5(a)–(d). The yellow symbols and lines represent the simulation and theoretical results, respectively. The black vertical line marks the starting time of the vaccination. The simulation results show that with the increasing of parameter $\alpha$ (i.e., $(p_i)$), the outbreak amplitude decreases greatly and the average infected density decreases clearly. When the average vaccination probability is large enough, the epidemic is under control with a lower infected density. Our theoretical analyses also confirm the simulation results very well (see the next section for detail). To investigate the effect of varying vaccination probability strategy on recurrent epidemic spreading, the parameter $\alpha$ is tuned to regulate the average vaccination probability. It’s worth noting that the average vaccination probability multiplied by the total individual number is the vaccination number, thus when the total individual number is almost unchanged, the average vaccination probability can be used to represent the vaccination cost to some extent. To quantify the change of epidemic spreading with average vaccination probability $\langle p \rangle$, maximal infected density $\rho_i \rho_f \rho_m$, average infected density $\rho_i \rho_f$, outbreak interval $\tau$ and average time interval $T_n$ of an individual attacked by the epidemic are calculated in Fig. 6. It’s worth noting that the smaller the $\rho_i \rho_f \rho_m$ and $\langle \rho_i \rangle$ are, the better the effect of controlling the recurrent epidemic is. As to $\tau$ and $T_n$, the larger values indicate the better controlling effect. For comparison, the strategies that the vaccination probability is constant for each year and the vaccination probabilities are determined only by infection rate $\beta$ or by densities of infectious individuals in the former year $I_i$ (i.e., the $p_i(\tau)$ is replaced by $1 - e^{-\alpha p_i} = 1 - e^{-\alpha \beta}$) are also studied. From the Fig. 6(a) and (c), it can be seen that with the increasing of average vaccination probability, the maximal infected density $\rho_i \rho_f \rho_m$ and average infected density $\rho_i \rho_f$ decrease greatly in all strategies, but it decreases much sharper when the vaccination probability varies year by year with risk indicator. Furthermore, the varying vaccination probability strategy makes the average outbreak interval $\tau$ and average time interval of individual attacked by influenza $T_n$ much larger than the case of other vaccination strategies Fig. 6(b) and (d). These simulation results show that at the same cost, the varying vaccination probability with risk indicator $(\frac{p_i \tau_i}{\rho_i \rho_f})$ could depress the recurrent epidemic outbreaks much more effectively.

5. Theoretical analysis based on Markov dynamics

Let $P_i(t)$, $P_i(t)$, and $P_i(t)$ be the probability for individual $i$ to be in the state of $S$, $I$ and $R$ at time $t$, respectively. Then we have $P_i(t) = \frac{1}{N} \sum_{i=1}^{N} P_i(t)$, $P_i(t) = \frac{1}{N} \sum_{i=1}^{N} P_i(t)$, $P_i(t) = \frac{1}{N} \sum_{i=1}^{N} P_i(t)$, where $P_i(t)$, $P_i(t)$ and $P_i(t)$ represent the densities of susceptible, infected, and refractory individuals at time $t$, respectively. Let $q_i^{S,R}(t)$, $q_i^{S,R}(t)$, and $q_i^{S,R}(t)$ be the transition probability from the state $S$ to $I$, $I$ to $R$ and $R$ to $S$, respectively. By the Markov chain approach [35,36] we have

$$q_i^{S,R}(t) = 1 - (1 - \rho_0) \prod_{l \in \Lambda_i} [1 - \beta(t) \rho_i(t)],$$

$$q_i^{S,R}(t) = \mu,$$

$$q_i^{S,R}(t) = \delta,$$

(2)

where $\Lambda_i$ represents the neighbors of individual $i$, the term $(1 - \rho_0)$ represents the probability that individual $i$ is not infected by the environment, while the term $\prod_{l \in \Lambda_i} [1 - \beta(t) \rho_i(t)]$ is the probability that individual $i$ is not infected by the infected neighbors, thus, $(1 - \rho_0) \prod_{l \in \Lambda_i} [1 - \beta(t) \rho_i(t)]$ is the probability for individual $i$ to be in susceptible state.

Based on this analysis, we formulate the following difference equations to describe the evolution of individual’s states probability without the immunization

$P_i(t + 1) = P_i(t) (1 - q_i^{S,R}(t)) + P_i(t) q_i^{S,R}(t),$

$P_i(t + 1) = P_i(t) (1 - q_i^{S,R}(t)) + P_i(t) q_i^{S,R}(t),$

$P_i(t + 1) = P_i(t) (1 - q_i^{S,R}(t)) + P_i(t) q_i^{S,R}(t).$

(3)

The first term on the right-hand side of the first equation is the probability that individual $i$ is remained as susceptible state, and the second term stands for the probability that individual $i$ is changed from the refractory to susceptible state. Similarly, we have the same explanation for the second and third equations of Eq. (3).

Substituting Eq. (2) into Eq. (3) we obtain

$$P_i(t + 1) = P_i(t) (1 - \rho_0) \prod_{l \in \Lambda_i} [1 - \beta(t) \rho_i(t)] + P_i(t) \delta,$$

$$P_i(t + 1) = P_i(t) (1 - \delta) + P_i(t) \mu,$$

$$P_i(t + 1) = 1 - P_i(t) - P_i(t).$$

(4)
Fig. 3. (a) The change of infection rate $\beta$ and (b) the infected density $\rho_I$ with time $t$. The black horizontal solid line represent average infection rate $\langle \beta \rangle$. The size of the networks is $N = 10000$, and the average degree $\langle k \rangle = 6.95$. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Fig. 4. The dependence of the maximal infected density $\rho_{I,\text{max}}$ on (a) infection rate $\beta$, (b) densities of infectious individuals in the former year $I_{t-1}$ and (c) the ratio $\beta/I_{t-1}$. (d) The change of the vaccination probability $p(y)$ with the ratio $\beta/I_{t-1}$.

Fig. 5. (a)–(d) represent the time evolutions of the infected density $\rho_I$ with different vaccination strength $a$ (corresponding to different $\langle p \rangle$). The black vertical line indicates the time that the vaccination begins. The horizontal dash line in each figure indicates the influenza outbreak. The yellow symbols and solid lines represent the simulation and theoretical results, respectively. Other parameters are the same as Fig. 3. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)
With Eq. (4) on hands, however, it is still very hard to identify the epidemic threshold as the effects of time-dependent $\beta(t)$ and $p_0$. Specifically, we consider a case of single epidemic outbreak with $p_0 = 0$ and constant $\beta$ with a SIS model. According to Ref. [35,36], we write down the Markov chain approach as

$$P_i^F(t+1) = 1 - [1 - (1 - \mu)P_i^F(t)] \prod_{j \neq i} (1 - \beta P_j^F(t))$$

For the consider networks, the study of the asymptotic state yields the derivation of the epidemic threshold $\tilde{\beta}_\mu = \frac{1}{\Lambda_{max}}$, where $\Lambda_{max}$ is the largest eigenvalue of the adjacency matrix $A$ [35,36]. For the uncorrelated SF networks, the largest eigenvalue is $\Lambda_{max} = \langle k^2 \rangle$ [37,38]. In our case, the epidemic threshold for the case of constant $\beta$ is in between $[0.3,0.5]$, i.e. $0.1 < \beta_c < 0.2$ (see Ref. [26] in details). When the $\beta$ is time-dependent at range $[0,1]$, for $\beta < \beta_c$, epidemic can not outbreak. However, for $\beta > \beta_c$ epidemic may outbreak. Thus, a fluctuant $\beta(t)$ around the epidemic threshold is a necessary condition to guarantee the recurrent outbreaks.

Next, we introduce the addition of immunization. At the beginning of each year, $p(y) \times N$ individuals who are in susceptible and refractory states are randomly selected to take vaccination with probability $q_{SR,R} = \frac{p(y) \times N}{1 \times P(t) + q_{SR,R} N} = \frac{p(y)}{1 + q_{SR,R} P(t)}$. Thus, after immunization, the probability of being susceptible state for each individual is immediately decreased to $(1 - q_{SR,R}) \times P(t)$, and the probability of being refractory state is immediately increased to $P(t) + q_{SR,R} \times P(t)$. And then substitute the above two new susceptible and refractory probabilities into Eq. (4) and continue to iterate the equation till the next immunization.

6. Discussion and conclusion

The suppression of recurrent epidemic outbreaks on the network is a challenging problem in recent years. Under the framework of complex network, this paper studies the dynamic vaccination strategy on recurrent epidemic spreading. Based on the real influenza data and simulation results, we found that the outbreak of influenza is related to the epidemic infection rate $\beta$ of the year and the average infected density of the former year $I_{-1}$. According to the study of these two quantities, we found out the risk indicator and designed a dynamic vaccination strategy. Taking into account the risk indicator, the varying vaccination strategy could depress the recurrent epidemic outbreaks effectively. In particular, the outbreak peaks will be lowered obviously even at very small average vaccination probability, and the outbreak interval will be prolonged notably.

The results in this paper tell us that to depress the influenza outbreaks it is not necessary to keep the vaccination probability high every year. Adjusting the vaccination probability at right time also could decrease the outbreak risks to some extent. In our work we only consider the uncorrelated scale free networks and the vaccinated individuals are selected randomly, but in reality, there are often groups of peoples that connect firmly. Thus to study the vaccination strategy in community networks and investigate whether selecting individuals with large connections or in high connected communities to vaccinate is better is needed. In addition, in the disease spread process, both time and space play an important role [39–43], and many significant results have been achieved to understand the traveling wave and pattern formation [2,44]. Therefore, to incorporate the spatial details or realistic population mixing structures to epidemic spreading process and assess the potential impact of different intervention strategies are our future works.

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