Influence of dynamic immunization on epidemic spreading in networks

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HIGHLIGHTS

- A new dynamic immunization strategy in networks is proposed.
- We build a link between dynamic and static immunization.
- This strategy does not affect the epidemic threshold.
- This strategy apparently decreases the final immunization fraction.

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ABSTRACT

We introduce a new dynamic immunization method based on the static immunization algorithm and study the relationship between dynamic and static immunization. By nodes to be immunized according to static immunization strategies, we build a connection between dynamic and static immunization. Using theoretical arguments and computational simulation we show that dynamic immunization (from a finite vaccine reservoir) is not sufficient to prevent epidemic outbreak, nor does it significantly change the asymptotic prevalence. Nonetheless, we do find that less total vaccine is required to implement this strategy. To help understand this better, we examine the extent and distribution of dynamic immunization required to achieve this reduced vaccine demand. Our results suggest that it is not necessary to increase the immunization rate when the infection rate is relatively small.

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1. Introduction

Immunization through vaccination in complex networks has been widely studied in previous work, and many effective immunization schemes have been proposed and investigated, including random immunization \cite{1}, targeted immunization \cite{1}, acquaintance immunization \cite{2}, and other improved immunization strategies \cite{3–6}. Previous work mostly assumes the immunization scheme operates on the network before the epidemic spreading commences. Such immunization strategies \cite{1–6} are called static immunization strategies (SI-Strategy).

In reality, immunization control is always implemented during the epidemic outbreak \cite{7}. This is for two reasons (at least): (1) individual vaccination behavior is generated by the epidemic seriousness \cite{8}; (2) new vaccine is made after the

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beginning of an epidemic. This type of immunization is called a dynamic immunization strategy (DI-Strategy). In general, the DI-Strategy depends on the epidemic information (including the infected density, the infection rate, and so on). Such information changes or evolves [9] during the spreading process. Hence, it is meaningful to study the influence of dynamic immunization on the epidemic dynamics [10–12]. In the disease system with dynamic immunization, it is critical to establish the transition of node \( i \) from a susceptible state (S) to an immunized state (M). There are two approaches to do this:

1. A susceptible node becomes immunized with a constant rate [13] or an information-based rate [14,15,12], and we call this the Poisson approach. Ruan et al. [12] studied the impact of epidemic information on the final vaccination fraction and found that strengthening the information diffusion can reduce the final vaccination fraction. This is helpful when the amount of available vaccine is very small. Jo et al. [14] studied information-based immunization in the susceptible–infected–removed–susceptible model and found that raising the immunization rate can help to unexpectedly promote epidemic outbreak in some cases. Nian and Wang [15] proposed a high-risk immunization strategy which immunizes nodes linked with infected nodes with a certain rate and found that the epidemic can be controlled by raising the immunization rate.

2. A susceptible node gets vaccinated when the epidemic information amount is larger than a threshold value [7,10,16], and we call this the threshold approach. Goldenberg et al. [7] studied the distributed immunization on computer networks where the node informs other nodes in the same immunization cluster to vaccine once a vigilant node contacts one or more infected nodes. Zhang et al. [10] studied the impact of individual decision on the epidemic dynamics by using game theory and found that the heterogeneous network can help to control epidemic spread. In our work [16], when the number of infected neighbors of a susceptible node achieves a threshold value, it will become vaccinated. We focus on the impact of vaccination on the epidemic threshold and found that there exist two kinds of critical values of spreading rate to discriminate between dynamical behaviors.

Inspired by these work, we will combine two approaches together to propose a new DI strategy. In this paper, we mainly consider whether dynamic immunization is better than static immunization. In other words, what is the influence of the dynamic immunization on the epidemic spreading compared to static immunization? To solve this issue, we must build a connection between the two strategies. Therefore, we propose a kind of the DI-Strategy based on the SI-Strategy. Using this framework we would like to: (1) study the impact of the DI-Strategy on epidemic dynamics; (2) investigate the difference between DI-Strategy and SI-Strategy. To our knowledge, there has been no complete work in this area. In this paper, we investigate each of these issues in detail.

The rest of this paper is organized as follows: In the next section, we propose an approach to model one relationship between SI-Strategy and DI-Strategy; then in Section 3, we investigate the DI-strategy based on the targeted immunization (that is, the TI-based immunization for short); in Section 4, we study the DI-strategy based on the random immunization (that is, the RI-based immunization); in Section 5, a theoretical model is presented to explain the simulation results; then in Section 6, we discuss the impact of initial infection conditions; finally, in Section 7 we conclude the paper and give some discussions.

2. An SIS model with the contact immunization

Let us consider a given static network with size \( N \), denoted by \( G = (V,E) \)—a graph \( G \) of nodes \( V \) connected by links \( E \). The nodes of \( G \) can be enumerated with index \( i = 1, 2, \ldots, N \). \( A = (a_{ij}) \) denotes the adjacency matrix of \( G \), where if node \( i \) links to node \( j \) in \( G \), then \( a_{ij} = 1 \), otherwise \( a_{ij} = 0 \). The maximal eigenvalue of the adjacency matrix \( A \) is denoted by \( \lambda_{\text{max}}(A) \).

As we know, a node that is in contact with one or more infected nodes should be preferred to immunize since it is likely to get infected at the next time step. Such nodes are called high-risk nodes [15,17]. Intuitively, an infectious disease can be controlled or suppressed by immunizing those high-risk nodes. Distinct from the literature [15], we only consider the high-risk nodes among a certain set \( \Omega \). Clearly, \( \emptyset \subset \Omega \subset V \).

We further assume that a high-risk node \( i \) in \( \Omega \) gets vaccinated with an immunization rate \( \delta \), which reflects the immunization level and can be easily revised for more realistic cases. For example, we can assume that \( \delta_i \), as a function of node \( i \), may be for each \( i \), or each node can choose its immunization rate [18].

Let us consider some special cases for \( \delta \). When \( \delta = 0 \), no node can be immunized and the epidemic model does not include an immunization term. When \( \delta = 1 \), all high-risk nodes are vaccinated. Therefore, the transition from the S state to the M state is regarded as a combination of the threshold approach (becoming a high-risk node) and the Poisson approach (with \( \delta \)). For convenience, we call such dynamic immunization as contact immunization. The contact immunization is similar to the contact tracing where the neighbor of an infected node is traced [17]. So contact immunization is not only considered to be the voluntary vaccination (e.g., the information-driven vaccination [12] or the information dependent vaccination [19]), but also a program vaccination.

In this model, each node lies in one of three states: S-susceptible, I-infected and M-immunized. During a time step, an infected node may recover and become susceptible again with rate \( \gamma \). For a susceptible node in the normal case, if it does not belong to \( \Omega \), then the node can be infected by one of its infected neighbors and each neighbor with rate \( \beta \). In the special case that the node belongs to \( \Omega \), if it is a high-risk node, then it will be vaccinated with rate \( \delta \), otherwise it can be infected by one of its infected neighbors as in the normal case. The spreading and immunization process compared to the static immunization is illustrated in Fig. 1. Similar to the previous work, we define the effective spreading rate \( \lambda = \beta / \gamma \).
Fig. 1. Illustration of the epidemic spreading and immunization process for both the static immunization and dynamic immunization on a network with seven nodes. The static immunization—(a) At $t = 0$, node 5 and 6 are immunized in advance and other nodes are in the susceptible state. (b) At $t = 1$, node 2 and 4 are randomly chosen as the initial infected nodes. (c) At $t = 2$, node 3 can be infected by node 2 and 4 with rate $1 - (1 - \beta)^2$; node 2 and 4 recover with rate $\gamma = 1$. The dynamic immunization—(d) At $t = 0$, all nodes are susceptible, where only node 5 and 6 in set $\Omega$ will be immunized. (e) At $t = 1$, node 2 and 4 are randomly selected as the initial infected seeds. (f) At $t = 2$, node 3 is infected with rate $1 - (1 - \beta)^2$. At the same time, node 5 and 6 can be immunized with rate $\delta$ since they belong to $\Omega$ and are connected to node 2 or 4. If node 6 is not vaccinated, then it can be infected by node 4 with rate $\beta$. That is, node 6 becomes infected with rate $(1 - \delta)\beta$ under the condition that the number of its infected neighbor is not zero. All infected nodes recover from the infection with rate $\gamma = 1$.

We will determine the existence of the critical value of $\lambda$ (i.e., the epidemic threshold, denoted by $\lambda_c$) above which an epidemic will prevail and persist in a population. It is interesting for us to study the impact of $\delta$ on $\lambda_c$. Although the implementation of contact immunization strategy is related to the network topology, we consider Barabási–Albert (BA) scale-free networks [20] as an instance, which allows us to consider several kinds of immunization strategies.

In this paper, we performed Monte Carlo simulations over BA scale-free networks with the mean degree $\langle k \rangle \approx 6$ and $N = 2000$. Initially, 1% of nodes are infected and others are susceptible. All simulations use a parallel updating strategy in which the disease dynamics are applied to each node by considering the actual state of the node and its neighbors at each time step. In order to output our results, we evolve the disease dynamics for 10,000 time steps in total and make time average to reduce the fluctuation of the infection density $I(t)$ and the immunization fraction $M(t)$. Specifically, we let $I = \frac{1}{T} \sum_{t=t_0}^{t_0+T} I(t)$ and $M = \frac{1}{T} \sum_{t=t_0}^{t_0+T} M(t)$ (that is, $t_0 = 9901$ and $T = 100$). To minimize the random fluctuation caused by initial conditions, we make average of $I$ and $M$ over 100 realizations of different randomly chosen initial infectious nodes. Throughout, we assume $\gamma = 1$.

3. The targeted immunization-based contact immunization

It is well known that the targeted immunization (TI) scheme applied on scale-free networks is very effective in controlling epidemic outbreak [1]. Hence, we firstly consider the contact immunization strategy corresponding to the targeted immunization. According to the mechanism of contact immunization, we have a subset $\Omega = \{v \in V : \deg(v) \geq k_t\}$ where $\deg(v)$ denotes the degree of node $v$ and $k_t$ is a control parameter.

By using the numerical simulations, we investigate the influence of $\delta$ on the disease dynamics from two perspectives: (i) the epidemic threshold $\lambda_c$; (ii) the immunization fraction at the steady state.

In Fig. 2(a) and (c), we illustrate the change of the infection density at the steady state $I$ with respect to the infection rate $\beta$ when $k_t = 30$ and $k_t = 40$. For a standard networked-SIS epidemic model with targeted immunization, there exists a threshold value of degree $k_t^*$ related to the infection rate $\beta$ for a given recovery rate [1,16]. Accordingly, we can derive an inverse relation—the epidemic threshold $\beta_c = \varphi(k_t)$ (where $\varphi(\cdot)$ is a function). As shown in Fig. 2(a) and (c), we find
that $\beta_c = 0.15$ for $k_t = 30$ and $\beta_c = 0.13$ for $k_t = 40$. These values are identical to the epidemic thresholds for contact immunization. Therefore, the dynamic immunization has no influence on the epidemic threshold.

In Fig. 2(b) and (d), one can see other important information. Although the parameter $\delta$ has no effect on the epidemic threshold (even the epidemic prevalence), it can affect the size of the final immunization fraction. Let us denote $M_d$ and $M_s$ be the final immunization fraction of the dynamic immunization and the static immunization, respectively. From Fig. 2(b) and (d), there exists another critical value $\beta_c^*$: when $\beta > \beta_c^*$ one can see a “convergence” between $M_d$ and $M_s$, i.e., $M_d = M_s$. For convenience, we call this critical value the convergence threshold. When $M_d = M_s$, the dynamic behavior is identical with that for the static immunization. In this case, the dynamic immunization model is reduced to the static immunization model. Otherwise, when $\beta < \beta_c^*$, $M_d$ is smaller than its upper value $M_s$, which shows that a dynamic immunization strongly can reduce the amount of vaccine required.

We notice the presence of the convergence threshold. Intuitively, when $\beta > \beta_c$, the epidemic will always exist in the networks. Therefore, all nodes in the set $\Omega$ will finally get vaccinated in the steady state no matter how small the value of $\delta$. However, it is not the case for the contact immunization. In fact, based on our simulation observations, we argue that in the set $\Omega$ there exists some nodes that are surrounded by the immunized nodes and that therefore remain susceptible all the time.

Now, we would like to investigate the effect of the contact immunization on $M_d$. Since $M_d < M_s$ for a given $\beta$ only if it satisfies $\beta < \beta_c^*$, we define a new quantity $Q = 1 - \frac{M_d}{M_s}$. This characterizes the level of reduction of vaccine usage. The larger the value of $Q$, the more obvious the difference induced by the dynamic immunization. For convenience, we call this quantity the immunization efficiency. As we know, $M_s = |\Omega|/N$ which is a constant for a given immunization strategy. While, $M_d$ is related to the system parameter $\beta$ and $\delta$. Therefore, $Q = f(\beta, \delta, \ldots)$ where the ellipsis symbol denotes other factors, e.g., $k_t$. Our objective is to study the impact of $\delta$ on the disease dynamics, so other parameters are given and we concentrate on how $\delta$ affects $Q$.

Let us only consider the case $k_t = 30$. Fig. 3 shows that $Q$ changes as a function of $\delta$. Intuitively, when the immunization rate $\delta$ increases, the contact immunization becomes stronger and then $Q$ becomes larger. However, the relation is not valid for a small infection rate. In Fig. 3, we observe an inverse case for $\beta = 0.1$. In this case, $Q$ decreases when $\delta$ increases from 0.1 to 1. We argue that this is due to the interaction between vaccination and infection. Although increasing $\delta$ can favor vaccination, it also hals the spreading of an epidemic with a small infection rate across hub nodes and hence decreases propensity for vaccination. In addition, the error bar in this figure is relatively large, showing that the immunization efficiency is highly dependent of the initial infection distribution and the simulation randomness.
Fig. 3. The immunization efficiency $Q$ as a function of the immunization rate $\delta$. We demonstrate the case for the TI-based contact immunization when $k_t = 30$ and $\beta = 0.1, 0.15 (\approx \beta_c)$ (a); and the case for the RI-based contact immunization when $f = 0, 0.06, 0.13 (\approx \beta_c)$ (b). The error bars show the variation due to different initial infection.

Fig. 4. The infection density at the steady state as a function of the infection rate for (a) $f = 0.5$ and (c) $f = 0.3$. The immunization fraction at the steady state as a function of the infection rate for (b) $f = 0.5$ and (d) $f = 0.3$.

4. The random immunization-based contact immunization

Another widely-used immunization strategy is the so-called random immunization (RI) [1]. This means that a fraction $f$ of all nodes is randomly selected to be immunized. Random immunization is not a good immunization strategy in scale-free networks since it requires a large number of nodes to immunize. However, when other immunization strategies cannot be completely and effectively implemented, the random immunization is still a useful immunization scheme in reality.

We investigate the case of contact immunization corresponding to random immunization. Similar to the section above, we firstly consider the impact of $\delta$ on the epidemic threshold. In Fig. 4, we can see that parameter $\delta$ cannot affect the epidemic threshold—while parameter $f$ can.

In addition, when $\beta < \beta_c$, the value of $M_d$ presented in Fig. 4(b), (d) is less than the corresponding $M_t$. Moreover, for each $\beta \in [0, 0.3]$ we have $M_d < M_t$ holds. This indicates that the convergence threshold is absent in the observation windows $[0, 0.3]$. This is somewhat different from the targeted immunization-based contact immunization, where $M_t = M_d$ for a large $\beta$ (see Fig. 2(b) and (d)).

Compared to the TI-based contact immunization, one can see that the curvature of $M_d - \beta$ curves in Fig. 4(b) and (d) is larger than that of curves in Fig. 2(b) and (d) when $\beta < \beta_c$. This also reflects the difference between the two immunization
strategies. However, we also obtain a similar observation in Fig. 3. That is, raising the value of $\delta$ leads to an increase in $Q$ for a large $\beta$ and a decrease in $Q$ for a small $\beta$.

5. Analytic explanations

We make use of the microscopic Markov-chain approximation (MMA) approach [21–23] to build a mathematical model. This allows us to obtain the conditions for epidemic extinction. In order to do this, we denote the probability of each node $i$ to be infected and to be immunized at time $t$ as $p_{i,t}$ and $q_{i,t}$, respectively. During the time interval $[t, t + 1)$, the change of $p_{i,t}$ depends on two properties: the recovery event and the infection events during this interval, assuming that these events are independent.

Since the state transition of a susceptible node $i$ is related to the connection between node $i$ and $\Omega$, we consider the following two cases:

Case I: $i \in V \setminus \Omega$. In this case, for a susceptible node $i$, there exist two exclusive events: (i) be infected with rate $\pi_{\text{inf}}^{(1)}(i)$; (ii) remain susceptible with rate $1 - \pi_{\text{inf}}^{(1)}(i)$.

Case II: $i \in \Omega$. This case is coupled with the contact immunization and then becomes more complicated. For a susceptible node $i$, there are three exclusive events: (i) be immunized with rate $\pi_{\text{vac}}(i)$; (ii) be infected with rate $\pi_{\text{inf}}^{(2)}(i)$; (iii) be still susceptible with rate $1 - \pi_{\text{vac}}(i) - \pi_{\text{inf}}^{(2)}(i)$.

Hence, the network epidemic model is described by

\begin{align}
 p_{i,t+1} &= (1 - \gamma)p_{i,t} + (1 - p_{i,t})\pi_{\text{inf}}^{(1)}(i), \quad i \in V \setminus \Omega \tag{1a} \\
 p_{i,t+1} &= (1 - \gamma)p_{i,t} + (1 - p_{i,t} - q_{i,t})\pi_{\text{inf}}^{(2)}(i), \quad i \in \Omega \tag{1b} \\
 q_{i,t+1} &= q_{i,t} + (1 - p_{i,t} - q_{i,t})\pi_{\text{vac}}(i), \quad i \in \Omega. \tag{1c}
\end{align}

Next, we would like to explore the specific forms of $\pi_{\text{inf}}^{(1)}(i)$, $\pi_{\text{inf}}^{(2)}(i)$ and $\pi_{\text{vac}}(i)$. Considering that node $i$ may be infected by multiple connections simultaneously, the probability that a node is not infected by any of its neighbors (denoted by $\zeta_{i,t}$) is given by

$$\zeta_{i,t} = \prod_{j \in \mathcal{N}(i)} (1 - \beta p_{j,t}). \tag{2}$$

Here, $\mathcal{N}(i)$ denotes the neighborhood of node $i$ in the network $G$. So we have

$$\pi_{\text{inf}}^{(1)}(i) = 1 - \zeta_{i,t} = 1 - \prod_{j \in \mathcal{N}(i)} (1 - \beta p_{j,t}). \tag{3}$$

When node $i$ belongs to the set $\Omega$, we develop expressions for both $\pi_{\text{inf}}^{(2)}(i)$ and $\pi_{\text{vac}}(i)$. As stated in our model, when at least one of node $i$’s neighbors is infected, node $i$ gets vaccinated with rate $\delta$ and becomes infected with rate $(1 - \delta)\pi_{\text{inf}}^{(1)}(i)$. Therefore, we have

$$\pi_{\text{vac}}(i) = \delta \times \text{Prob(at least one of the neighbors of } i \text{ is infected)}$$

$$\quad = \delta \times [1 - \text{Prob(all neighbors of } i \text{ are healthy})]$$

$$\quad = \delta \times \left[1 - \prod_{j \in \mathcal{N}(i)} (1 - p_{j,t})\right],$$

and

$$\pi_{\text{inf}}^{(2)}(i) = (1 - \delta)\pi_{\text{inf}}^{(1)}(i) \left[1 - \prod_{j \in \mathcal{N}(i)} (1 - p_{j,t})\right].$$

On substituting all these expressions into Eqs. (1), we obtain a closed equation.

In what follows, we derive an approximate condition for epidemic outbreak from (1). To this end, we consider the equilibrium state. Note that both Eqs. (1b) and (1c) describe the dynamics of node $i \in \Omega$. We first consider the equilibrium phase of $\Omega$. Let $q_{i,t} = q_i$ in Eq. (1c), we have $(1 - p_i - q_i)\pi_{\text{vac}}(i) = 0$. This indicates that $1 - p_i - q_i = 0$ or $\pi_{\text{vac}}(i) = 0$. By substituting $1 - p_i - q_i = 0$ into Eq. (1b) and letting $p_{i}^1 = p_i$, we have $p_i = 0$, while $\pi_{\text{vac}}(i) = 0$ can imply $\pi_{\text{inf}}^{(2)}(i) = 0$. Therefore, we have $p_i = 0$ for each $i \in \Omega$, which means that there is no infection in the set $\Omega$ at the steady state.

Next we consider the existence of a positive solution of Eq. (1a) when $p_i = 0$ for each $i \in \Omega$. At this time, Eq. (1a) is a closed equation since

$$\pi_{\text{inf}}^{(1)}(i) = 1 - \prod_{j \in \mathcal{N}(i) \setminus \Omega} (1 - \beta p_{j,t}). \tag{4}$$
Recently, Darabi Sahneh and Scoglio applied the Taylor series expansion at the epidemic threshold to establish the existence condition for a positive solution [24]. Unfortunately, this approach appears to be not suitable for our model because the adjacency matrix $\hat{A} = (\hat{a}_{ij})$ of a subgraph $\hat{G}$ where all nodes in set $\Omega$ are removed from $G$ is not necessarily irreducible. Hence, we make use of another approach, i.e., to determine the local stability of the infection-free equilibrium ($p_i = 0$ for each $i \in V \setminus \Omega$).

Near the infection free equilibrium, Eq. (1a) can be simplified to

$$p_{t+1} = (1 - \gamma)p_t^i + \beta \sum_{j=1}^{N} \hat{a}_{ij} p_{j,t},$$

where $\hat{N} = |V| - |\Omega| = N - |\Omega|$. This formulation uses the approximation $(1 - a)(1 - b) \simeq 1 - a - b$ when $a \ll 1, b \ll 1$.

We introduce a vector function $p_t = (p_{1,t}, p_{2,t}, \ldots, p_{N,t})^T \in \mathbb{R}^\hat{N}$ (the state vector of the subnetwork $\hat{G}$). With this notion, the model is given by a collective form

$$p_{t+1} = (1 - \gamma)p_t + \beta \hat{A} p_t = [\beta \hat{A} + (1 - \gamma)I]p_t.$$

Here $I$ denotes an $\hat{N}$-dimensional unit matrix. From above, one can get that the local stability of the zero solution of system (5) can be established by

$$\Lambda_{\max}[\beta \hat{A} + (1 - \gamma)I] < 1$$

where $\Lambda_{\max}(\hat{A})$ is the maximum eigenvalue of matrix $\hat{A}$. So the epidemic threshold $\lambda_c$ for any undirected network with contact immunization obeying

$$\lambda_c = \frac{1}{\Lambda_{\max}(\hat{A})}.$$  

When $\Omega = \emptyset$ or $\delta = 0$, the epidemic model is just the standard SIS model in networks [25,21]. In that case, the epidemic threshold $\lambda_c = 1/\Lambda_{\max}(A)$ [21,26], which is less than $1/\Lambda_{\max}(\hat{A})$ since matrix $\hat{A}$ is a sub-matrix of $A$. Hence, only if $\delta > 0$, by Eq. (5) one can obtain that the immunization rate $\delta$ has no impact on the epidemic threshold.

It is worthwhile to remark that when $\lambda > \lambda_c$, the zero solution of model (1) is unstable and the steady state $p_i$ is not necessarily positive for all $i \in V \setminus \Omega$ because the subgraph $\hat{G}$ may not be connected even when the graph $G$ is connected. In addition, all $p_i$ values are determined by (1a) and the infection density at the steady state is not related to the immunization rate $\delta$. These theoretical results are in good agreement with the above results obtained by Monte Carlo simulations.

Also, we make simulations by iterating Eqs. (1) on a BA scale free network with $N = 2000$ and take the case $k_i = 30$ for the TI-based immunization and the case $f = 0.5$ for the RI-based immunization as illustrated in Fig. 5. Fig. 5(a) and (c) show that raising the immunization rate cannot change the epidemic threshold and the final infection fraction. We also find the epidemic threshold predicted by model (1) is smaller than that obtained by Monte Carlo simulations (Figs. 2(a) and 4(a)). This is mainly due to the first order approximation of the mathematical model which neglects the dynamical correlation between connected nodes [27].

In stochastic simulations, we have seen that the curvature of the $M_d - \beta$ curves for the RI-based contact immunization is clearly larger than that of curves for the TI-based contact immunization when $\beta < \beta_i$. The difference between the two kinds of immunization strategies in the shape of the immunization curve also can be predicted by the theoretical model (1) (Fig. 5(b) and (d)).

6. Influence of initial infection condition

From Eq. (1c), we notice that the value of $Q$ or $M_d$ is dependent of the initial (infection) condition, such as the number or location of infected nodes. So it is interesting to explore the influence of the initial infection condition on $Q$. In the above simulations, it is assumed that the initial infection density $I(0)$ is 0.01. To have a better comparison, we mainly examine the change of $Q$ with respect to $I(0)$ from 0.005 to 0.015.

Simulations (Fig. 6) tell us that $Q$ decreases when $I(0)$ increases irrespective of whether it is the RI-based or the TI-based contact immunization, although these curves are not entirely smooth. This demonstrates that the dynamic immunization displays advantages in the case of small initial infection conditions. Hence, when we take the dynamic immunization strategies early, the amount of vaccine required is small.

7. Conclusion and discussion

To sum up, an SIS epidemic model with contact immunization is proposed and analyzed. Contact immunization based on the static immunization is a particular kind of dynamic immunization. We have studied two kinds of contact immunization: the TI-based and RI-based immunization. Our results suggest that these two types of contact immunizations do not affect the epidemic threshold and the epidemic prevalence (the infection density at the steady state). This is a little surprising since
Fig. 5. Simulations obtained by Eqs. (1). The infection density at the steady state as a function of the infection rate for (a) $k_t = 30$ and (c) $f = 0.3$. The immunization fraction at the steady state as a function of the infection rate for (b) $k_t = 30$ and (d) $f = 0.3$.

Fig. 6. (a) The strength of dynamic immunization $Q$ as a function of the initial infection density for the targeted immunization-based contact immunization ($k_t = 30, 40$) and (b) for the random immunization-based contact immunization ($f = 0.3, 0.5$). The immunization rate $\delta = 0.5$, $\gamma = 1$ and $\beta = \beta_c$. All simulations are averaged over 100 epidemic simulations.

the contact immunization can interrupt the spreading path of an infectious disease. We also give an analytic explanation by using the microscopic Markov-chain analysis. In the theoretical analysis, we derive the epidemic threshold of the model and find that it is completely dependent on the subnetwork by removing potentially immunized nodes and hence, indeed, not related to the contact immunization.

Hence in a sense, the dynamic immunization is the same as static immunization. However, we further observe that the contact immunization apparently decreases the final immunization fraction when $\beta \leq \beta_c$. A potential application is to implement the dynamic immunization in epidemic control. When $\beta \leq \beta_c$, $M_c$% of the nodes are required to vaccinate for static immunization while $M_d$% of the nodes are required for dynamic immunization. In this case, we can use $Q$% less vaccine. This point is helpful in reality as previously emphasized by Ruan et al. [12].

Finally, we explored the relation between the immunization rate and the immunization efficiency $Q$ for the different infection rates and find that $Q$ is not positively correlated to the immunization rate $\delta$ for a small $\beta$ value. Hence, our results suggest that it is not necessarily a good choice to enlarge the value of $\delta$ in controlling a weak infectious disease.

In the present work, we only use SIS dynamics to study the impact of the contact immunization and it is interesting to consider other epidemic models, e.g., the SIRS model [14,15]. Additionally, our strategy can be easily extended to other network models, including the weighted network and time-varying network. Especially for the dynamic network based on
the mobile agents [28–30], the mobile parameter may have an important influence on the immunization effect and our analysis method may not be adoptable for this case.

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References