



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 [1], targeted immunization [1], acquaintance immunization [2], and other improved immunization strategies [3–6]. Previous work mostly assumes the immunization scheme operates on the network before the epidemic spreading commences. Such immunization strategies [1–6] are called *static immunization strategies* (SI-Strategy).

In reality, immunization control is always implemented during the epidemic outbreak [7]. This is for two reasons (at least): (1) individual vaccination behavior is generated by the epidemic seriousness [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 of 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 $i = 1, 2, \dots, N$. $A = (a_{ij})$ 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 $\Lambda_{\max}(A)$.

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 Ω . Clearly, $\emptyset \subset \Omega \subset V$.

We further assume that a high-risk node i in Ω gets vaccinated with an immunization rate δ , which reflects the immunization level and can be easily revised for more realistic cases. For example, we can assume that δ_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 δ . 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 δ). 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 γ . For a susceptible node in the normal case, if it does not belong to Ω , then the node can be infected by one of its infected neighbors and each neighbor with rate β . In the special case that the node belongs to Ω , if it is a high-risk node, then it will be vaccinated with rate δ , 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 $\lambda = \beta/\gamma$.

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