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Global and local targeted immunization in networks with community structure

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Abstract. Immunization plays an important role in the field of epidemic spreading in complex networks. In previous studies, targeted immunization has been proved to be an effective strategy. However, when extended to networks with community structure, it is unknown whether the superior strategy is to vaccinate the nodes who have the most connections in the entire network (global strategy), or those in the original community where epidemic starts to spread (local strategy). In this work, by using both analytic approaches and simulations, we observe that the answer depends on the closeness between communities. If communities are tied closely, the global strategy is superior to the local strategy. Otherwise, the local targeted immunization is advantageous. The existence of a transitional value of closeness implies that we should adopt different strategies. Furthermore, we extend our investigation from two-community networks to multi-community networks. We consider the mode of community connection and the location of community where epidemic starts to spread. Both simulation results and theoretical predictions show that local strategy is a better option for immunization in most cases. But if the epidemic begins from a core community, global strategy is superior in some cases.

Keywords: network dynamics, random graphs, networks
1. Introduction

The social relations in a society can be described as a network in which nodes represent individuals and links stand for social connections [1]. Spreading phenomena on networks emerge as ubiquitous dynamics in an array of applications. For instance, epidemics may spread through physical contact networks, and information can be shared by agents on online social networks.

In the field of epidemic spreading in networks [2–8], immunization plays a significant role [9–15]. An immunized node cannot be infected nor can they spread epidemics to others. But apparently, vaccinating a node generates a cost, leading us to minimize the number of individuals to be immunized. Numerous immunization strategies have been proposed in the last two decades [9–11, 16–22]. Among them, the targeted immunization, where nodes are vaccinated according to their numbers of connections (degrees), has a good performance in epidemic control in general [9]. The highly connected nodes are influential in the network. If they are protected, the epidemic spreading in the network will be hindered substantially.

However, networks in real cases always have prominent community structure where connections are dense within the community, but sparse between communities [23–25]. The community structure can affect epidemic spreading significantly [26–28], thus it is meaningful to consider community structure when studying immunization.

Previous studies of targeted immunization, even taking community structure into account, mainly discussed global strategy that the highly influential nodes in the entire network is vaccinated. Though global targeted immunization can improve the overall prevention effect, it cannot halt the spreading at an early stage. Now, as early-detection methods of epidemic spreading are proposed in recent years [29–31], it is possible to detect the contagious outbreaks within the community where they occur. Therefore, we propose here a local targeted strategy that only highly connected nodes in the
very community where the epidemic originates are vaccinated. This strategy is helpful in reducing infections in an early stage, and may eradicate the epidemic within the community. However, the epidemic may become uncontrollable if it spreads to other communities. So there is still an important question to answer: does this local targeted immunization work better than the global strategy?

In this paper, we use the block model to generate synthetic networks with communities [32], and adopt susceptible-infected-recovered (SIR) model to describe the spreading of epidemics [33–35]. We find that the answer for the above question depends on the closeness between communities. The global strategy is better than the local strategy when the communities are tied closely, but inferior when they are not. The influencing factors of this transition value are also investigated in this paper. It shows that the transition value increases if the epidemic infected rate increases or if the vaccinating proportion decreases. We examine two-community networks in details, and then extend these strategies to multi-community networks. The mode of community connection and the location of community where epidemic outbreak occurs are mainly considered.

The related mathematical models are described in section 2. The analytical framework and simulations are presented in sections 3 and 4, respectively. Multi-community networks are discussed in section 5. Conclusions are drawn in section 6.

2. The model

The block model [32, 36, 37] plays an important role in the field of community detection due to its simplicity. In this section we introduce the two-community block model.

In principle, a two-community block model consists of two communities of equal size. The total $N$ nodes are equally divided into both communities. The number of total links in the network is $M$. In each community, there are $N/2$ nodes and $(1 - \mu)M/2$ links connecting them (inner links). On top of that, there are $\mu M$ links connecting nodes from different communities (outer links). We name $\mu$ the strength of the community structure. It can be easily found that when $\mu$ increases, more inner links change to outer links.

By definition, we can obtain that any given link points to a node in the same community with the probability $(1 - \mu)/(1 + \mu)$, and to a node in the different community with the probability $2\mu/(1 + \mu)$. Therefore, if $(1 - \mu)/(1 + \mu) < 2\mu/(1 + \mu)$ (i.e. $\mu > 1/3$), the number of outer links is more than the number of inner links, which is not in accordance with the normal definition of community. Thus, we focus on networks with $\mu$ lower than 1/3.

Next, we present a simple description of the SIR epidemic model [33, 34] with immunization. Nodes, or individuals, are divided into four groups: susceptible (S), infected (I), recovered (R), and immunized (V). At the beginning, $vN$ nodes are assigned as immunized nodes and kept unchanged, and the other nodes are all susceptible. Here $v$ is the vaccinating proportion. The epidemic starts to spread from community 1 at $t = 0$, i.e. one randomly chosen node in community 1 turns into infected nodes. At every time step, susceptible nodes may become infected from the contact with infected nodes at a rate $\beta$. Each infected node turns to be recovered with the rate $\gamma$, and will never be infected again. Without loss of generality, we set $\gamma = 1$ henceforth. Vaccinated nodes cannot be infected in our assumption. After a long period of time, there will be no
infected node in the system. The proportion of recovered nodes \( r_\infty \) measures the severity of the disease.

As introduced in section 1, we consider two immunizing strategies in this paper: the global targeted immunization and the local targeted immunization. For the global case, we vaccinate the most highly connected nodes in the entire network, while for the local case we merely vaccinate the most highly connected nodes in community 1. In both cases, we vaccinated the same proportion of nodes \( v < 1 \).

### 3. Theoretical analysis

In this section, we set up the theoretical framework of the analysis. Here, the method used is enlightened by [38].

The aim of our calculation is to obtain the expressions of \( r_\infty \) in both global targeted immunization case \( (r_\infty^{\text{Global}}) \) and local targeted immunization case \( (r_\infty^{\text{Local}}) \). Before the calculating, we first introduce some symbols used in this section. The degree distribution of the community 1, the community 2, and the entire network is \( P^1(k) \), \( P^2(k) \) and \( P(k) \), respectively. The proportion of susceptible, infected, and recovered nodes at time \( t \) in community \( g \) \((g = 1, 2)\) is \( s^g(t) \), \( i^g(t) \) and \( r^g(t) \). Specially, we denote the density of susceptible, infected, and recovered nodes in community \( g \) in the degree class \( k \) by \( s^g_k(t) \), \( i^g_k(t) \), \( r^g_k(t) \), respectively. That is, \( i^g_k(t) \) equals to the number of infected nodes of degree \( k \) in community \( g \) divided by the number of nodes in the community \((N/2)\). Thus, the total proportion of recovered nodes in community \( g \) is

\[
  r^g_\infty = \sum_k P^g(k) r^g_k(\infty). \tag{1}
\]

Then the total proportion of recovered nodes in the entire network is \( r_\infty = (r_\infty^1 + r_\infty^2)/2 \). If we denote the vaccinated nodes of degree \( k \) in community \( g \) by \( v^g_k \), naturally we have \( i^g_k(t) + s^g_k(t) + r^g_k(t) + v^g_k = 1 \) for any time step \( t \). The community \( \bar{g} \) refers the community that is different from community \( g \), i.e.

\[
  \bar{g} = \begin{cases} 
    1 & g = 2, \\
    2 & g = 1.
  \end{cases} \tag{2}
\]

To start with, we establish the SIR model evolution equations \([6, 39]\)

\[
  \frac{ds^g_k(t)}{dt} = -\beta s^g_k(t) \Theta^g(t), \tag{3}
\]

\[
  \frac{di^g_k(t)}{dt} = -i^g_k(t) + \beta s^g_k(t) \Theta^g(t), \tag{4}
\]

\[
  \frac{dr^g_k(t)}{dt} = i^g_k(t). \tag{5}
\]

Here \( \Theta^g(t) \) is the average density of infected nodes pointed by any given link in community \( g \). It can be calculated by

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\[ \Theta^g(t) = \frac{1}{\langle k \rangle} \left[ 1 - \frac{1}{1 + \mu} \left( \sum_k (k - 1) P^g(k) i^g(t) \right) \right] + \frac{2\mu}{1 + \mu} \left( \sum_k (k - 1) P^g(k) i^g(t) \right), \]  

(6)

where \( \langle k \rangle \) stands for the average degree of the network. Because we assume the epidemic starts to spread from community 1, the initial conditions of the equations are:

\[ s^1_k(0) = 1 - \frac{1}{N}, \quad s^2_k(0) = 1 - v^1_k, \quad i^1_k(0) = 2/N, \quad i^2_k(0) = 0, \quad r^1_k(0) = 0 \text{ and } r^2_k(0) = 0. \]

We find that the ordinary differential equations (3) and (5) can be directly integrated as

\[ r^g_k(t) = \int_0^t i^g_k(\tau) d\tau. \]  

(7)

and

\[ s^g_k(t) = s^g_k(0)e^{-\beta\phi^g(t)}, \]  

(8)

where the auxiliary function

\[ \phi^g(t) = \int_0^t \Theta^g(\tau) d\tau. \]  

(9)

To calculate \( r^g_k(\infty) \) (or \( s^g_k(\infty) \), equivalently, as \( r^g_k(\infty) = 1 - s^g_k(\infty) - v^g_k \)), we need to obtain the value of \( \phi^g(t) \). In order to obtain a closed relation for \( \phi^g(t) \), it is more convenient to focus on the time evolution of \( \phi^g(t) \). For this purpose, we obtain

\[ \frac{d\phi^g}{dt} = \Theta^g(t) \]

\[ = \frac{1}{\langle k \rangle} \left[ 1 - \frac{1}{1 + \mu} \left( \sum_k (k - 1) P^g(k) i^g_k \right) \right] + \frac{2\mu}{1 + \mu} \left( \sum_k (k - 1) P^g(k) i^g_k \right) \]

\[ = \frac{1}{\langle k \rangle} \left[ 1 - \frac{1}{1 + \mu} \left( \langle (k - 1)(1 - r^g_k - s^g_k - v^g_k) \rangle \right) \right] + \frac{2\mu}{1 + \mu} \left( \langle (k - 1)(1 - r^g_k - s^g_k - v^g_k) \rangle \right) \]

\[ = 1 - \frac{1}{\langle k \rangle} - \phi^g \]

\[ - \frac{1}{\langle k \rangle} \left[ 1 - \frac{1}{1 + \mu} \left( \langle (k - 1)s^g_k(0)e^{-\beta\phi^g} \rangle \right) + \langle (k - 1)v^g_k \rangle \right] \]

\[ + \frac{2\mu}{1 + \mu} \left( \langle (k - 1)s^g_k(0)e^{-\beta\phi^g} \rangle + \langle (k - 1)v^g_k \rangle \right). \]  

(10)
Here, \( \langle f(k) \rangle = \sum_k f(k) P(k) \) and \( \langle f(k) \rangle_g = \sum_k f(k) P^g(k) \). Generally, the above equation cannot be solved in a closed form, but it is possible to obtain information on the infinite time limit, i.e. at the end of the epidemics.

When \( t \to \infty \), it has \( i_\infty = 0 \) and \( \Theta^g = 0 \). Therefore \( d\phi^g / dt = 0 \) and we have the self-consistent equation

\[
1 - \frac{1}{\langle k \rangle} - \phi^g_\infty
- \frac{1}{\langle k \rangle} \left| \frac{1 - \mu}{1 + \mu} \left( \langle (k - 1) s_k^g(0)e^{-\beta k \phi^g_k} \rangle_g + \langle (k - 1) v_k^g \rangle_g \right) \right|
+ \frac{2\mu}{1 + \mu} \left( \langle (k - 1) s_k^g(0)e^{-\beta k \phi^g_k} \rangle_g + \langle (k - 1) v_k^g \rangle_g \right) = 0. \tag{11}
\]

Taking the initial conditions into equation (11), we obtain

\[
1 - \frac{1}{\langle k \rangle} - \phi^g_\infty
- \frac{1}{\langle k \rangle} \left| \frac{1 - \mu}{1 + \mu} \left( \langle (k - 1) s_k^g(0)e^{-\beta k \phi^g_k} \rangle_g + \langle (k - 1) v_k^g \rangle_g \right) \right|
+ \left( \langle (k - 1) v_k^g \rangle_g + \langle (k - 1) v_k^g \rangle_g \right) = 0. \tag{12}
\]

By solving equation (12) (set \( g = 1 \) and 2, respectively), we can obtain \( \phi^g_\infty \) and \( \phi^g_\infty \). Once the value of \( \phi^g_\infty \) and \( \phi^g_\infty \) are obtained, it is possible to compute \( r^g_\infty \). Considering \( r^g_\infty = 1 - s_k^g(\infty) - v_k^g \) and equation (1), we have

\[
r^g_\infty = 1 - \langle s_k^g(0)e^{-\beta k \phi^g_k} \rangle_g - \langle v_k^g \rangle_g. \tag{13}
\]

Finally, we can calculate the total infected proportion \( r_\infty \) by \( r_\infty = (r^1_\infty + r^2_\infty) / 2. \)

Clearly, different immunizing strategies depend on different expressions of \( v_k^g \) and \( v_k^g \). We set \( v \) as the total vaccinating proportion. Thus, for the global targeted immunization, we have

\[
v_k^1 = \begin{cases} 1 & k \geq k_g \\ 0 & k < k_g \end{cases} \tag{14}
\]

and

\[
v_k^2 = v_k^1. \tag{15}
\]

For the local targeted immunization, we have

\[
v_k^1 = \begin{cases} 1 & k \geq k_l \\ 0 & k < k_l \end{cases} \tag{16}
\]
Here \( k_g \) and \( k_l \) satisfy
\[
\sum_{i=1}^{k_g} P(i) = 1 - v, \tag{18}
\]
and
\[
\sum_{i=1}^{k_l} P(i) = 1 - 2v. \tag{19}
\]

To sum up, we present a method to calculate the theoretical value of \( r_\infty \) without solving the ordinary differential equations, for both the global case and the local case in this section. However, the expressions of \( r_\infty^{\text{Global}} \) and \( r_\infty^{\text{Local}} \) are not straightforward. Therefore, we use numerical methods to solve equation (11), substituting corresponding results into equation (13) to obtain \( r_\infty^1 \) and \( r_\infty^2 \). Related discussion of these theoretical predictions will be presented in the next section.

4. Simulation results

In this section, we investigate the immunization strategies by Monte Carlo simulations, and compare simulation results with theoretical predictions. To start with, we introduce the simulation settings.

For a given value of \( \mu \), the networks are generated by the following method. First, we built two isomorphic Barabási–Albert (BA) networks [40] to simulate two communities. The BA network is built from a network with 40 nodes and 20 random-wired links. Every time a new node is introduced into the network, it will attach to 20 existing nodes. The probability that an existing node is chosen to be connected by the new node is proportional to the degree of the existing node. This procedure is repeated for 4960 times. Thus, each BA network has 5000 nodes and the average degree is \( \langle k \rangle = 39.76 \).

Then, we randomly choose \( [\mu M/2] \) links in community 1, rewiring one random end of the links to randomly chosen nodes in community 2. Next, we randomly choose \( [\mu M/2] \) inner links in community 2, and rewire one random end of the links to randomly chosen suitable nodes in community 1, which have no connection with the original nodes to avoid two links connecting the same pair of nodes. Thus, we obtain a network with \( N = 10,000 \) nodes, \( M \approx 2 \times 10^5 \) links, and two communities. Ten different rewiring processes are performed for each \( \mu \), and all results below are averaged by these ten topologies. The degree distributions of two typical networks are shown in figure 1. During the SIR simulation, one randomly chosen node in community 1 is determined to be the epidemic seed. And 1000 independent SIR experiments are repeated in each case.

We first verify that different strengths of the community \( \mu \) affect the spreading behaviors. In figure 2, the curve of epidemic prevalence \( r_\infty \) versus vaccinating proportion \( v \) shows that \( r_\infty \) decreases as \( v \) increases. Because \( r_\infty \) indicates the proportion of
individuals who have been infected, lower $r_\infty$ means better immunity effect. Compared with random immunization and no immunizations cases, targeted immunizations (both global and local) have better performances. For the case of $\mu = 0.10$, the local targeted immunization performs superior to global targeted immunization, while for $\mu = 0.20$ the global targeted immunization has a better effect.

This result can be explained as follows. When $\mu$ is small, there are fewer connections between communities. If we vaccinate highly influential nodes in community 1, it is difficult for the epidemic to spread. However, when $\mu$ is relatively large, controlling

Figure 1. The degree distributions of two typical networks. Panel (a) stands for one network with $\mu = 0.01$, and panel (b) stands for one network with $\mu = 0.20$. We adopt log–log coordinates in each panel.

Figure 2. The relationship between the epidemic prevalence $r_\infty$ and the vaccinating proportion $v$ (simulation results). Panel (a) stands for the case of $\mu = 0.10$ and panel (b) stands for the case of $\mu = 0.20$. Lines with blue circles, red squares, yellow triangles, and black stars stand for cases of global targeted immunization, local targeted immunization, random immunization and no immunization, respectively. Parameters are shown in each panel. Network topologies are presented in the main text.
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Epidemics in one community is impossible, and the global immunizing is helpful to prevent the spread of the epidemic in the entire network.

By figure 2, we assume that there exists a critical community strength $\mu_c$ that if $\mu < \mu_c$ the local strategy is more effective, while if $\mu > \mu_c$ the global strategy works better. Whether this phenomenon is special or general is worth studying. Therefore, we perform numerous simulations, varying vaccinating proportion $v$ and infected rate $\beta$, to verify this hypothesis. Some results are presented in figure 3.

In each simulation of figure 3, we find that $r^{\infty}_{\text{Local}}$ is outstripping $r^{\infty}_{\text{Global}}$ as $\mu$ increases. The existence of $\mu_c$ is also confirmed by the theoretical predictions that obtained by the method presented in section 2, which are shown in figure 4. Theoretical predictions are relatively consistent with simulation results. The discrepancy between theoretical predictions and simulation results may due to many reasons. The main cause of the discrepancy is the systematic error of equation (3) to equation (5). These equations are mean-field approximations for the dynamical process, and cannot describe the simulation precisely in any finite-size network topologies. Besides, the number of simulation runs may also play a role.

By figures 3 and 4, it seems that a higher $\beta$ or lower $v$ leads to a higher $\mu_c$. We set $v$ and $\beta$ as variables, and obtain figure 5. In this figure, theoretical values and simulation values are also compared, and their differences are not large. We calculate $\mu_c$ (both theoretical and simulation results) by the following method. First, we pick up $\mu_1$ and $\mu_2$ that satisfy when $\mu = \mu_1$ we have $r^L < r^G$ and when $\mu = \mu_2$ we have $r^L > r^G$. Here $r^L$ and $r'^L$ stand for the epidemic proportion for the local targeted immunization and $r^G, r'^G$ stand for the corresponding global cases. We then calculate the intersection point of line $(\mu_1, r^G)(\mu_2, r'^G)$ and line $(\mu_1, r^L)(\mu_2, r'^L)$. In practice, we calculate $\mu_c$ when $\mu_2 - \mu_1 \leq 0.005$.

It can be seen from figure 5(a) that $\mu_c$ increases and reaches a steady point as $\beta$ rises. It implies that if the network has an apparent community structure, we should adjust the immunizing strategy from local to global, when the epidemic is severe. Figure 5(b)
shows that $\mu_c$ gradually decreases while $v$ increases, because a higher $v$ also hinders the spreading of the epidemic.

5. Extention to multi-community networks

Apparently, a real-world network is composed of more than two communities. In this section, we extend the two-community block model to multi-community networks, and mainly discuss the effect of community connection modes. Four-community networks
are taken as examples and some typical cases of community connection modes are
selected (seen in figure 6).

In this figure, balls stand for communities and connections between balls stand for
links between communities (outer links). Communities are labeled as core communities or peripheral
communities.

Topologies of these four models are generated by the following method. We first
build four isomorphic isolated BA networks (each has $N_0 = 5000$ nodes and $M_0 \approx 1 \times 10^5$
links, as we adopted in the above section). Then, if community $P$ and community $Q$
are connected in the model, we add $\sigma M_0$ links to connect randomly chosen nodes in
community $P$ and community $Q$. We also avoid two links connecting the same pair of
nodes. In this section we use $\sigma$ to quantify the community closeness. Hence, 4B model
or 4C model respectively has $4N_0$ nodes and $(1 + 3\sigma)M_0$ links, while 4A model or 4D
model respectively has $4N_0$ nodes and $(1 + 4\sigma)M_0$ links.

We should note that the positions of communities in these models are usually not
equitable. There are core communities that have more groups of outer links and periph-
eral communities that have fewer groups of outer links. Epidemics starts so spread
from different communities may produce different results. So we need to take it into
consideration. Specially, there is only one type of community in 4A model, and three
types of communities in 4D model.

Besides, we can directly extend our theoretical framework to networks with multi-
community by modify equation (6) and some other relevant equations. Thus, we can
calculate $r_\infty$ by both simulation methods and theoretical methods. The results are
shown in figure 7. The community connection modes surely affect the epidemic spreading
behaviors. Some interesting phenomena emerge in the multi-community networks.

Firstly, in 4A model where the status of communities are equivalent, global immuni-
ization and local immunization have slight differences (see figure 7(a)). While for other
models, there are relative obvious differences between these two strategies. Secondly,
for the case of epidemic starting from a peripheral community (see figures 7(b), (d), (f)

Figure 6. An illustration of four typical four-community models. Balls stand for
communities and connections between balls stand for links between communities
(outer links). Communities are labeled as core communities or peripheral communities.
and (g)), local immunization has an advantage for all the value range of $\sigma$. It means that if epidemic starts from a peripheral region of the social network, local protection is recommended. Thirdly, in 4B and 4C model, if epidemic starts from a core community (see figures 7(c) and (e)), global strategy is in an advantageous position in the middle or high value range of $\sigma$. Fourthly, the theoretical predictions have similar trend with simulation results. In 4D model when $\sigma > 0.4$, the local strategy has a large superiority in simulation results, but theoretical predictions do not show this phenomenon. Fifthly,
epidemic will be more prevalent if it starts from a core community. It is proved by the comparison between figures 7(b) and (c), and between figures 7(d) and (e).

6. Conclusion and outlook

In previous studies, the targeted immunization is an effective strategy because the most influential nodes are protected. However, social networks have significant community structures in the real world. Thus, we question whether the better strategy is to vaccinate nodes who have the most number of connections in the entire network (global), or those in the community where the epidemic begins to spread (local). Based on the two-community block model, we investigate this question and observe the following results. In general, if the network has apparent community structure (the community strength $\mu$ is small), the local targeted immunization performs better than the global targeted immunization. Otherwise, the global targeted immunization performs better than its counterpart. It implies that there is a critical $\mu_c$ that we should adopt different strategies in the case of $\mu < \mu_c$ or $\mu > \mu_c$. The influential factors of $\mu_c$ are investigated, and it shows that an increasing infected rate $\beta$ or a decreasing vaccinating proportion $v$ leads to a higher $\mu_c$. We also provide explanations for the above phenomenon. Besides, we establish a theoretical framework and find that the theoretical predictions are relatively consistent with the simulations. In addition, we extend the two-community block model to multi-community block model. If the community connection modes are considered, conclusions are complicated. In most cases, the local strategy is still a better option, but if the epidemic starts from a core community, the global strategy may be superior in some cases.

In future study, we will explore whether there is an immunization strategy better than these existing ones. In the two-community block model, the simple weighted strategy seems not working well. Thus, we need to consider the micro-structure of the network, such as the k-shell number [41, 42], the betweeness centrality, the clustering coefficient, or the assortativity of the networks [43].

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