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# A new perspective of epidemic controlling: immunization therapy strategy in weighted complex network

Yu-Ying Huang[1], Yu-Xin Ma[2],Yi-Xian Chen[2],Lin-Zi Zhang[2] and Han-Li[3]
*1 2 3 School of Management Science and Engineering, Central University of Finance and Economics*
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**Abstract.** The immunization strategy towards the epidemic spreading problem has attracted widespread attention of scientists from many different fields. However, the traditional immune behavior is achieved by deleting the edges in the network, which can lead to variations in the network structure and furthermore, seriously damage the efficiency of the network. In this paper, we studied a new type of immune strategy applied to weighted networks– immunization therapy strategy, which is to maintain the necessary network efficiency by lowering the weight of edges to suppress the spread of epidemic. It is similar to the inflammation around the infected parts of our body, which can either prevent epidemic from further spreading or do no harm to the function of the body. Thus, we name this novel strategy as "immunization therapy strategy". We first let the rate of transmission be proportional to the edge weight according to the $S-I$ epidemic spreading model. In addition, we give specific dynamic evolution for infected nodes that boosts efficient epidemic control. Theoretical analysis and simulation outputs indicate that the immunization therapy strategy can efficaciously prevent the spread of the epidemic, while maintaining the high efficiency of the network.

**Keywords:** complex network; $S-I$ model; epidemic controlling; immunization therapy.

## 1. Introduction

As the theories of complex networks developed [1], researchers have been paying more and more attention to epidemic spreading in complex networks. In the real world, usually there are only a few infection sources at the very beginning. However, without effective control and management, the epidemic spread will boom in a large-scale society. The immunization strategies in previous studies concentrated on selecting nodes according to their statues among the whole network, for instance, in terms of their nodes degrees. There are mainly 3 immunization that have been studied most up to now: random immunization[2]-[3],target immunization[4] and the distance-based local immunization[5].

Random immunization means the nodes are randomly chosen during the implement period. Without considering whether the node degrees are heavy or tiny, we choose all the nodes with the same probability. Target immunization takes the heterogeneity characteristic of the network into consideration and pick out nodes with larger degree for immunization. All of the edges connected to certain nodes will be eliminated from the network if these nodes are immunized. Eliminating such edges will change the structure of the whole network, which means some spreading paths of the epidemic will disappear. However, it is necessary to acquire the global information of the network in implementation of target immunization. To put things into a further step, the local immunization does not require us to get the global information. It just makes use of the nearby local information of infected nodes while controlling epidemic spread. Local immunization achieves the goal of controlling epidemic spreading in large-scale society by isolating infected nodes as well as nodes within a certain distance d of them.

The main idea of traditional immunization is eliminating the interconnections of some chosen nodes and the rest parts of the network. All the connections that link a node to others will be cut off immediately as long as it is immunized. Thought the epidemic spreading can be controlled by this way, the problem is the network is always destructed [6]- [7]. When the proportion of immunized nodes heat a certain point, the whole network will become invalid. As a result, the traditional immunization methods are not proper for comprehensive implement.

Actually, the epidemic spreading chances can be reduced by receiving prophylactic vaccination in the real stage. In weighted networks, the weight of each edge is defined as the tightness among nodes, and different degrees of tightness have different influence on epidemic spreading rate. Thus, we can control epidemic outbreak not by absolutely eliminating some edges but by just reducing the weights (degrees of tightness) of some edges, just like what we do in the real world. This method can either provide us with the same epi-

demic-control effect as tradition immunizations do or maintain the connectivity and the desirable efficiency of the whole network.

Having recognized the drawbacks of traditional immunizations [8]-[9], in this essay, we propose a concise immunization strategy with high efficiency on the perspective of weighted network – immunization therapy strategy. We achieve it by decreasing the weights of certain edges, i.e. reducing the weights of those edges that are connected with specific nodes. We derive the term "therapy" from a kind of physiological phenomenon: In order to prevent epidemic from spreading, whenever certain tissues are infected, there will be self-repair and inflammatory activities nearby these tissues. In such condition, self-repair therapy takes effect without absolutely destroy body functions. In the respect of epidemic-controlling efficiency and maintaining of the efficiency of the whole network, we make some contrasts between our new immunization therapy strategy and traditional immunization strategies. Both simulation and theoretical analysis tell us that immunization therapy strategy can either effectively control epidemic diffusion or keep the network efficiency high. What is more, we introduce mechanism of self-repair therapy into the target immunization, i.e. decrease weights of the edges connected to nodes with high degrees. In this paper, an accurate mathematical model is constructed and some applicable knowledge is deduced. The immunization, whose usefulness has been proved by experiments, is significant in guiding immune behaviors in the real stage.

# 2. Methods

## 2.1. Our definition of immunization therapy

In complex networks, the common immunization [10]-[11] way cuts the spreading path of epidemic by deleting nodes, such as deleting the edges around given nodes. The result is identical to the errors and attacks rules in the reality. Concretely, only less than half of the efficiency of the original network will be achieved while 15% of the maximum nodes are attacked. Moreover, when this proportion comes to 30%, the overall network will be collapsed and may finally be desperate to turn out to be unconnected pieces. As a result, in the real world, the conventional immunization methods are not possible to be carried out. Thus, we attempt to lower the edges weight associated with the immunized nodes, but we do not lower the weight down to 0 to prevent from damaging the entire network. This implementation will assure the appropriate immune effects while maintaining the essential information transmission, and meanwhile, the overall network can preserve the normal operation in a certain range.

To make things more simple, we make an assumption when we apply the immunization method in complex network: The edges connected to immunized nodes will be reduced by $q$ times, where $q$>1. If $q$ tends toward infinity, our immunization therapy strategy equals to traditional strategies. If $q$ equals to 1, the immunize effect will become extremely weak, which means we let the epidemic spread without any immunize intervene. We consider the weights of classical binary $BA$ network [12]-[13] as the tightness between nodes and add weighted mechanism to it, in which various degrees of tightness have different impacts on epidemic spreading efficiency. Especially, we use $w_{kk'} = w_0(kk')^{\beta}$ to describe the proportional relation between epidemic spreading speed and edge weights, where $w_{kk'}$ is weight of the edge connecting a node with degree $k$ and another node with degree $k'$. $w_0$ is invariant and different types of network have different $\beta$. Then, the strength of $k$ degree node can be calculated according to the edge weight:

$$N_k = k\Sigma_{k'} \, Pro(k'/k) w_{kk'} \tag{1}$$

Here, we only take non-associative network whose degree correlation probability can be described as $Pro(k'/k) = k' \, Pro(k')/\langle k \rangle$ into consideration. From what has been analyzed above, we can draw a function:

$$N_k = w_0 \langle k^{1+\beta} \rangle k^{1+\beta} / \langle k \rangle \tag{2}$$

## 2.2. Our epidemic spreading model

We implement the well-known Susceptible-Infected ($S-I$) model to conduct our research about the dynamic spreading behavior of epidemic in the weighted networks. In this model, the node has two statuses only: susceptible state ($S$) and infected state ($I$), where infected nodes cannot be recovered. During the be-

ginning burst phase of the spreading, because of the lack of deep understanding to the epidemic which also has the characteristics of sudden outbreak, we often have no proper control measures timely, resulting in the rapid spreading of the epidemic and the great destruction to the society. That is why we simulate the spreading of epidemic with $S-I$ model. Then we just transform the $S-I$ model a bit and extend the original model to the Susceptible-Infected-Susceptible ($SIS$) model or Susceptible-Infected-Recovered ($SIR$) model[14]-[15]. Therefore, the $S-I$ model is adequate for our research in weighted networks. In our study, we set the aggregate transmission rate among nodes by degree $k$ as $\eta_k$. Thus, the rate of transmission between degree $k$ node and degree $k'$ node is:

$$\eta_{kk'} = \eta k \frac{w_{kk'}}{N_k} \tag{3}$$

where $w_{kk'}$ means the weight of the edge, $N_k$ means node strength, and $\eta$ is a constant.

We can figure out that from Eq.(3), the greater the edge weight, the higher the epidemic spreading rate $\eta_{kk'}$, which is consistent with the fact. When we take $Internet$ network[16]-[17] into consideration, the frequency of knowledge flow can be depicted by the edge weights; in the U.S. airport network ($USAN$) the traffic frequency can be described by the edge weights as well. Greater the weight, higher the passenger flow. In the non-associated networks, Eq.(3) can be shown as the following form: $\eta_{kk'} = \eta k'^{\beta}\langle k\rangle/\langle k^{1+\beta}\rangle$.

In addition, we are able to obtain a susceptible node's infection probability at time $t$ with degree $k$ is $1-\Pi_{\forall k' \epsilon \Psi(t)}(1-\eta_{kk'})$, in which $\Psi(t)$ means the degree sequence of infected neighbors adjoining to a $k$-degree susceptible node. In order to study the spreading characteristics effectively, we firstly get the evolution equation from the $S-I$ model by means of the mean field theory in weighted networks:

$$\partial i_k(t)/\partial t = k(1-i_k(t))\Sigma_{k'} P(k'/k) i_{k'}(t)\eta_{kk'}$$

Where the right item is positively related to the degree $k$, and $1-i_k(t)$ indicates the probability of a $k$ degree susceptible node. As for the last item $\Sigma_{k'} P(k'/k) i_{k'}(t)\eta_{kk'}$, the summation indicates the average probability that an infected neighbor infects a susceptible node at time $t$ with degree $k$. The rate of transmission from degree $k'$ node to degree $k$ node is represented by $\eta_{kk'}$, which is the main distinction between the novel one and the common $S-I$ model.

Then, we replace the Eq.(3) by Eq.(4) with $Pro(k'/k) = k' Pro(k')/\langle k\rangle$ and overlook the terms with higher order. Then we are able to obtain the following equation:

$$\begin{aligned}\partial i_k(t)/\partial t &= \frac{\lambda k^{1+\beta}\langle k\rangle}{\langle k^{1+\beta}\rangle}\sum_{k'}\frac{k' Pro(k')}{\langle k\rangle} i_{k'}(t) \\ &= \frac{\lambda k^{1+\beta}\langle k\rangle}{\langle k^{1+\beta}\rangle}\theta_k(t)\end{aligned} \tag{5}$$

We can see in the equation that no terms are associated with degree $k$, which means $\theta_k(t)$ is independent of $k$ in the non-associative network. Consequently, we have:

$$\theta_k(t) = \theta(t) = \Sigma_{k'} \frac{k' Pro(k')}{\langle k\rangle} i_{k'}(t) \tag{6}$$

We deduce the partial deviation guidance for the formula above with respect to variable $x$ and we get following functions:

$$\begin{aligned}\partial\theta(t)/\partial t &= \sum_{k'}\frac{k' Pro(k')}{\langle k\rangle}\frac{\partial i_{k'}(t)}{\partial t} \\ &= \Sigma_{k'}\frac{k' Pro(k')}{\langle k\rangle}\frac{\eta k^{1+\beta}\langle k\rangle}{\langle k^{1+\beta}\rangle}\theta(t) \\ &= \frac{\eta\langle k^{2+\beta}\rangle}{\langle k^{1+\beta}\rangle}\theta(t)\end{aligned} \tag{7}$$

Consolidating Eq.(5) to Eq.(7) under the uniform initiate condition $i_k(t=0)=i_0$, we have:

$$i_k(t)=i_0+i_0\frac{\eta k^{1+\beta}\langle k\rangle}{\langle k^{1+\beta}\rangle}[e^{t/\tau}-1] \tag{8}$$

$i(t)$, which means the average density of infected nodes, can be calculated by the following equation:

$$i(t)=\sum_k P\,ro(k)i_k(t)=i_0+i_0\frac{\eta\langle k^{1+\beta}\rangle\langle k\rangle}{\langle k^{1+\beta}\rangle}[e^{t/\tau}-1] \tag{9}$$

There are

$$\tau=\frac{\langle k^{1+\beta}\rangle}{\eta\langle k^{2+\beta}\rangle} \tag{10}$$

Eq.(10) tells us that when epidemic outbreaks, the growth of time scale has a close relationship with the network's heterogeneity, i.e. if some nodes have unequal degrees, they will have different impact on the epidemic spreading. As *N* (the network's node number) tends to infinity, $\tau$ convergent to a constant. According to the construction of transmission rate, we can see that $\beta$ have a big influence on the epidemic spreading speed on the complex network. Fig 1 displays how steady-state infected nodes density changes with different $\beta$. It is easy to find out that a larger $\beta$ means a lower epidemic spreading speed. We can find out from the Fig.1 that if $\beta$ is 1, the value of $\tau$(time scale) fit perfectly with the theoretical value. Fig.2 makes comparison of the early-stage infected nodes density and the theoretical values. The $\beta$ values of the three lines from top to bottom is 1, 1.5 and 2. If $\beta$ is 0, the network becomes the classic unweighted *BA* model [18]-[19]. Consequently, it is necessary for us to concentrate on the important nodes and protect them with more cautiousness.

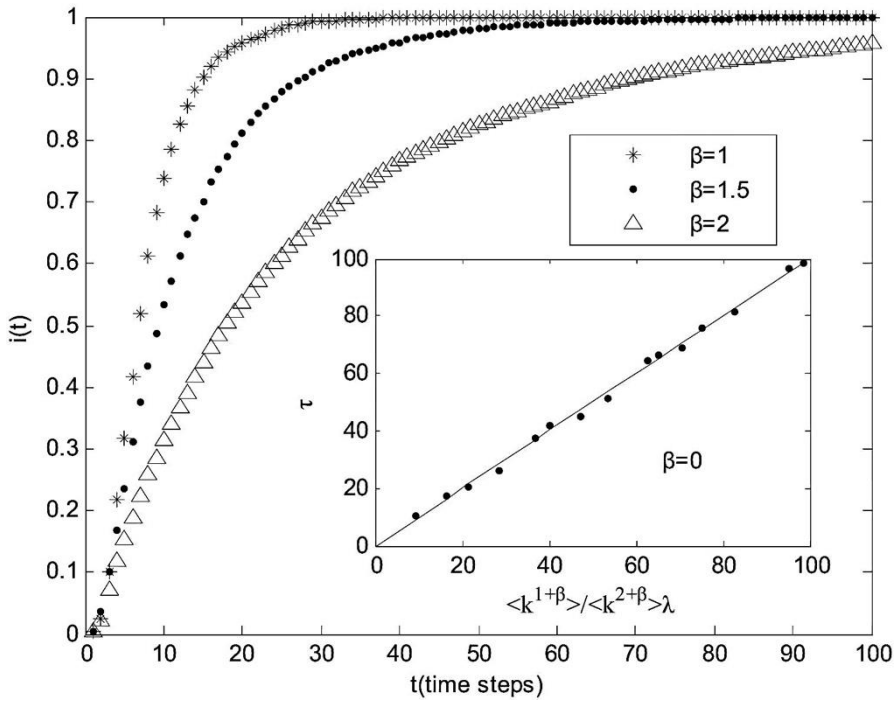


Fig. 1: With different $\beta$, the infected nodes proportion varies, which can be defined as a function of time: i(t). The inner graph makes contrasts of different $\boldsymbol{\tau}$ when $\beta\beta$ is 1.The results are drawn from 50 independent experiments.

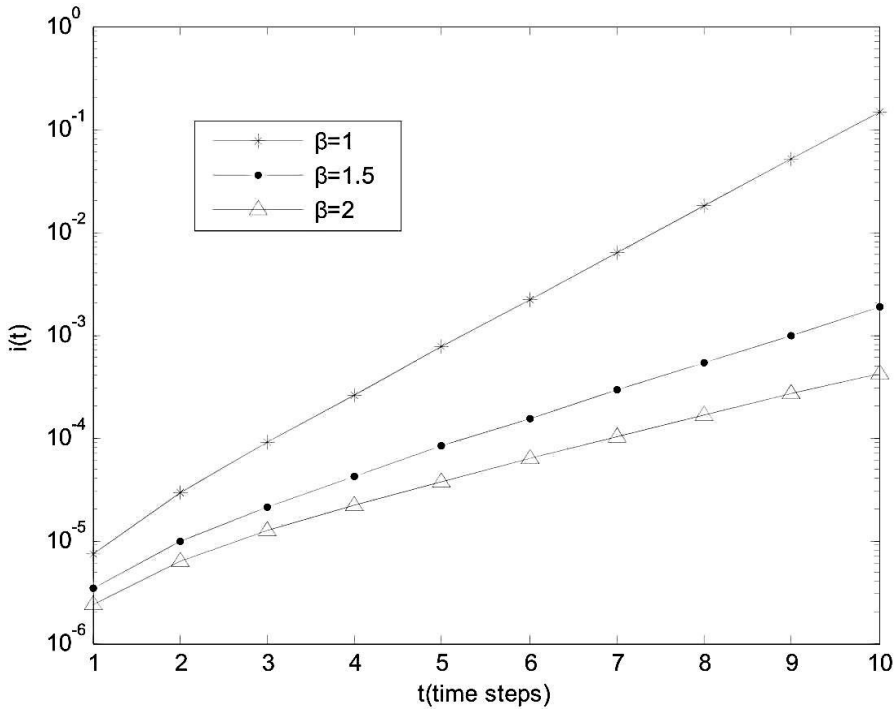


Fig. 2: Fig.2 shows the contradistinction between theoretical value and $\boldsymbol{\tau}$ under different $\beta$, in which the dash line means the theoretical value and we put the results into semi-log coordinate. The results are drawn from 50 independent experiments.

## 2.3. Network Efficiency Analysis

Network efficiency is applied to depict the diffusion and transportation capability in the dynamic network. High efficiency indicates close interaction between nodes, and information or goods can diffuse quickly in the network. The definition of network efficiency in unweighted network is contrary to the summation of the shortest path lengths between two nodes:

$$E = \frac{1}{N(N-1)} \sum_{i \neq i'} \frac{1}{d_{ii'}} \tag{11}$$

where $d_{i,i'}$ is the shortest path length connecting node $i$ and node i'. It is easy to find out that in the network, $E = 1$.

In the weighted network, the epidemic-spreading ability from one node to another is proportional to the edges weights and the shortest path length between them. The following equation describes the length of the shortest path in a weighted network:

$$d_{ii'}^{w} = min \sum_{m,n \in P_{i,i'}} \frac{1}{w_{mn}} \tag{12}$$

where $P_{i,i'}$ is a path connecting nodes i and $i'$. In this way, the efficiency of the weighted network E can be defined as:

$$E = \frac{1}{N(N-1)} \sum_{i \neq i'} \frac{1}{d_{ii'}^{w}} \tag{13}$$

If all of the edge weights are decreased by q times, the original edge-deleted network efficiency will decrease by $1/q$ times, while the network efficiency shown in Eq.(13) is obviously greater than $1/q$. Consequently, network in Eq.(13) can be more efficient than the original one.

Then, to represent the immunization effect before and after change, we set the related network efficiency $\varepsilon$ as:

$$\varepsilon = E/E_0 \tag{14}$$

In this equation, $E_0$ is the pre-immunization efficiency while $E$ is the after-immunization efficiency. It can be found out from Eq.(14) that $\varepsilon$ represents both the efficiency and the changes of network. We will apply $\varepsilon$ to weighted network in the following statement.

## 3. Results

Previous studies have shown that large degree nodes have the most important impact on the spread of epidemic in scale-free networks. As a traditional immune strategy, target immunization is regarded as the most efficient method due to its ability to immunize the minimum proportion of nodes to achieve the desired results. Nevertheless, it will greatly lower the network efficiency as well. In order to ensure the effective operation of the whole network, we will bring in the immunization therapy strategy as we have defined before, and have a look on the target immunization therapy.

The immunization therapy looks for the way to repress the epidemic by lowering the weight of edge. In our model, the weight of edge is computed by $w_{kk'} = w_0(kk')^\beta$. Thus, after reducing $q^\beta$ times, $w_{kk'}$ will be

$$w'_{kk'} = w_{kk'}/q^\beta = w_0(kk'/q)^\beta \tag{15}$$

Here, the transmission rate $\eta'_{kk'}$ is

$$\eta'_{kk'} = \eta(k'/q)^\beta \langle k\rangle/\langle k^{1+\beta}\rangle = \eta_{kk'}/q^\beta \tag{16}$$

We can draw a conclusion from Eq.(15) and Eq.(16) that if edge weight is reduced by $q^\beta$ times, the transmission rate $\eta'_{kk'}$ will also be reduced by $q^\beta$ times. This is because the destination node degree is reduced by $q^\beta$ times. In our model, $k'/q$ is defined as a key factor. In order to get the average nodes density of all k-degree nodes after immunization, the $k$ in the Eq.(8) is replaced by a key factor $k/q$ and then the following equation can be deduced.

$$i'_k(t) = i_0 + i_0 \frac{\eta\langle(k/q)^{1+\beta}\rangle\langle k\rangle}{\langle k^{1+\beta}\rangle}\left[E^{t/\tau} - 1\right] \tag{17}$$

When all the nodes with degrees bigger than $k_t$ are immunized, i$(t)$ becomes

$$\begin{aligned} i'_k(t) &= \textstyle\sum_{k\geq k_t} p(k)\, i'_k(t) + \sum_{k<k_t} p(k)\, i_k(t) \\ &= i_0 + i_0 \frac{\langle k\rangle}{\langle k^{2+\beta}\rangle}\left(E^{t/\tau} - 1\right) \\ &\cdot \left[\textstyle\sum_{k\geq k_t} p(k)(k/q)^{1+\beta} + \sum_{k<k_t} p(k)k^{1+\beta}\right] \end{aligned} \tag{18}$$

Eq.(18) tell us that if the topological parameters in a network are fixed to certain values, which means $\beta$ can be generally fixed, the infected nodes density can only be influenced by the value of $k_t$ and $q$ after immunization therapy. If we take the extreme condition into consideration, where $q$ tends toward infinity and $k_t = \min\{k\}$, the epidemic cannot diffuse and $i'(t) = i_0$;When merely $q$ tends toward infinity, the immunization therapy becomes the traditional one. When $k_t \geq \max\{k\}$ or $q = 1$, immunization does not work and $i'(t) = i(t)$.

We use function $p(k) = 2m^2k^3$ to describe the degree distribution in a weighted BA network, in which m means the number of edges connected to the original network when we add a new point to it. If we apply $p(k)$ to Eq.(16) and make k a continuous one, we can get:

$$\begin{aligned} i'(t) &= i_0 + i_0 \frac{\langle k\rangle}{\langle k^{2+\beta}\rangle}\left(e^{t/\tau} - 1\right) \\ &\cdot \left[\textstyle\sum_{k>k_t} 2m^2k^3(k/q)^{1+\beta} + \sum_{k<k_t} 2m^2k^3k^{1+\beta}\right] \\ &= i_0 + i_0 \frac{\langle k\rangle}{\langle k^{2+\beta}\rangle}\left(e^{t/\tau} - 1\right)\cdot 2m^2 \\ &\cdot \left(\int_{k_t}^{max\{k\}} k^{\beta-2}/q^{\beta+1} + \int_{min\{k\}}^{k_t} k^{\beta-2}\right) \\ &= i_0 + i_0 \frac{\langle k\rangle}{\langle k^{2+\beta}\rangle}\left(e^{t/\tau} - 1\right)\frac{2m^2}{\beta-1} \\ &\cdot \left(\frac{k^{\beta-1}}{q^{\beta+1}}\Big|_{k_t}^{max\{k\}} + k^{\beta-1}\Big|_{min\{k\}}^{k_t}\right) \end{aligned} \tag{19}$$

We should treat the optimization of Eq.(14) along with Eq.(19) as a combined optimization issue to reach immunization goal, and enlarge the whole network efficiency as much as possible at the same time. Since the number of unknowns equals to the number of equations, the best results of $k_t$ and $q$ can be always found in order to get the expected results. Moreover, we can find from Eq.(18) that the density of infected node is decided by the degree distribution $p(k)$ of a particular network as well. Therefore, the network topology will also influence the epidemic spreading and the resulting immunization strategy.

The immunized nodes density $i(t)$ changing with time $t$ are depicted in Fig.3 with different $\beta$ and q. It can be observed that the greater $\beta$ and q, the better the result of the inflammation immunization. Fig.4 depicts the variations of transmission velocity with time t and different $\beta$ and q, where the velocity of transmission[20]-[21] can be defined as follow:

$$V_{inf}(t) = \frac{di(t)}{dt} \approx \frac{I(t) - I(t-1)}{N(t)} \tag{20}$$

where i(t) $= I(t)/N(t)$, N(t) is the amount of nodes with time t.

From Figure 4 we can find that the epidemic spreading velocity can be reduced by decreasing $\beta$ and q. Actually, we can observe from Eq.(16) that as the value of q grows larger, the immunization effect becomes better. And meanwhile, the value of $\beta$ is inversely proportional to i(t). Therefore, we need to work harder to repress the epidemic spreading efficiently in the network with small $\beta$.

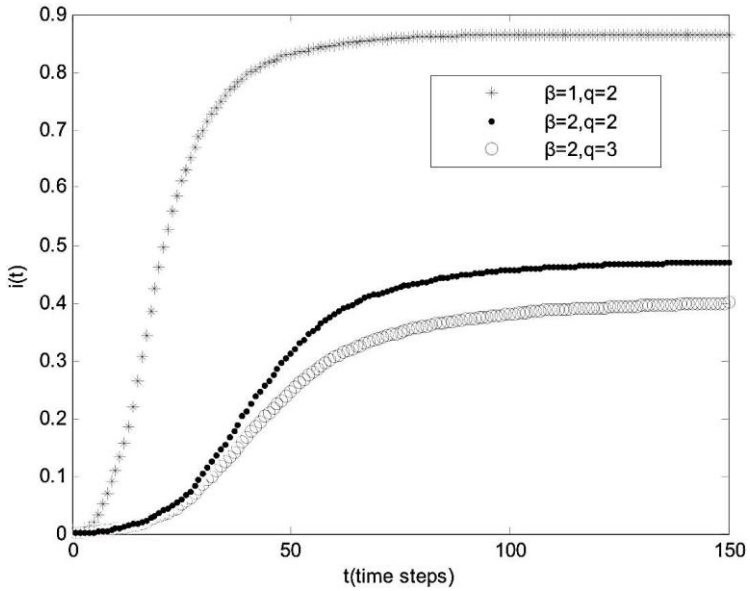


Fig. 3: Fig.3 shows how steady-state infected nodes proportion i(t) changes with time t. Different lines show the results under different $\beta\beta$ and q. These results are drawn from 50 independent experiments.

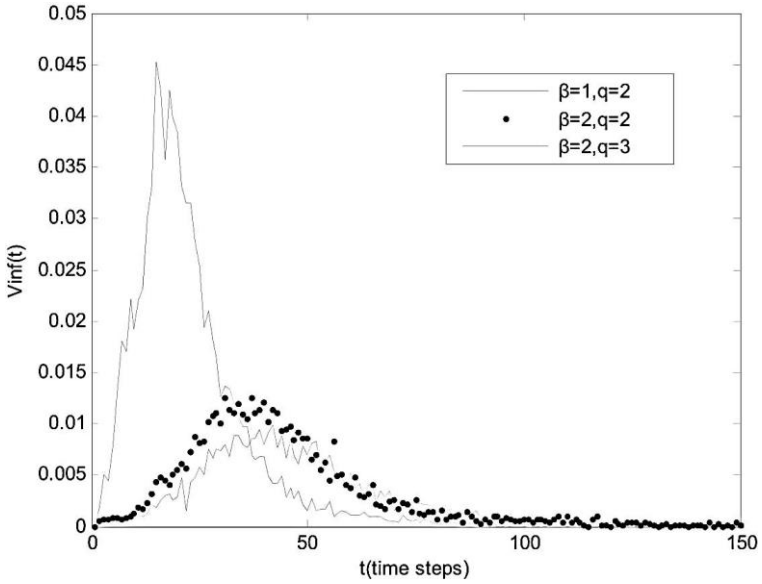


Fig. 4: Fig.4 shows how velocity $V_{inf}$ (t) changes with time t. Different lines show the results under different $\beta$ and q. These results are drawn from 50 independent experiments.

We fix β to 1, which means there's no inflammation effect, and make a contrast of immunization therapy. With various q, we use immunized nodes proportion to illustrate how steady-state infected nodes density i(t)(Fig.5) and efficiency of the network $\varepsilon$ (Fig.6) changes. We can figure out from Fig.5 that if the immunized nodes proportion near 0.25, the epidemic spreading can be absolutely offset by traditional target immunization; If q equals 10, which is relevant larger, the target immunization therapy is similar to the traditional target immunization; If q ranges between 5 and 10, steady-state infected nodes density i(t) change mildly with tiny differences between them. Consequently, the target immunization therapy is preferred when q ranges from 5 to 10. Fig.6 tells us that if q equals 10,the network is much more efficient after the target immunization therapy compared with the traditional strategy immunization; If q ranges from 5 to 10,the efficiency change mildly. When the proportion of immunized nodes is 0.25, if q equals 10,the network after target immunization therapy is 7-8 times more efficient than the traditional one and if q equals to 5,the network after target immunization therapy is approximately 20 times more efficient than the traditional one! From what has been mentioned above, we can find out that the target immunization therapy can either control epidemic spreading or protect the efficiency of the network.

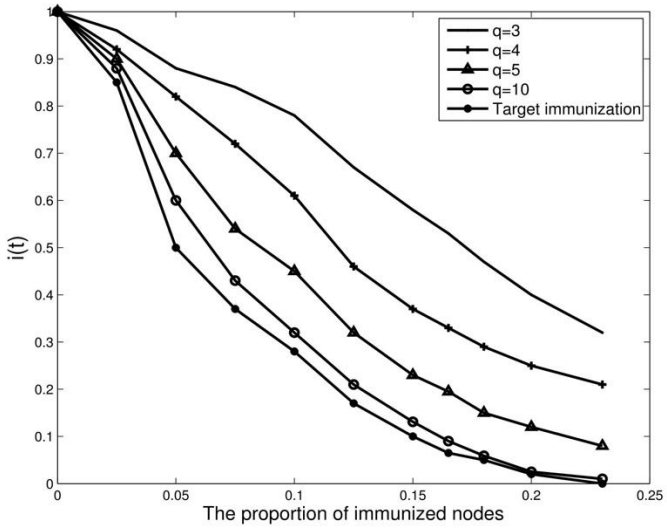


Fig. 5: $\beta$ is fixed to 1.With various q, we use immunized node proportion to illustrate how steady-state infected nodes density i(t) changes. We draw our result from the average solutions of 50 independent experiments.

## 4. Results

The theorem of common immunization strategy is to cut off the links between nodes and other networks. The connections of the immunized node with other nodes will be deleted out of the network. Although it cuts down the spreading paths, it destroys the network. In our study, in weighted network, a simple but effective immunization strategy – immunization therapy strategy is studied in view of the shortcomings of the traditional immunization method to reduce the network efficiency greatly. By decreasing the edge weights, it performs immunization therapy, like reducing a specific portion of weight of all edges linked to a particular node. This new immunization mechanism is studied by comparing the immunization performances with common immunization strategy and retaining the network efficiency. Theoretical analysis and simulation results reveal that the immunization strategy can efficiently suppress the epidemic spreading while maintaining a high-efficiency network.

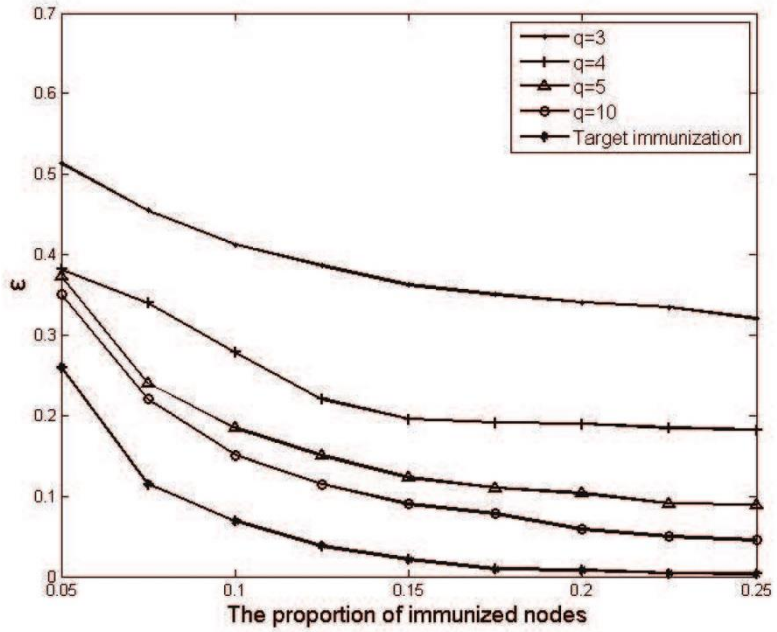


Fig. 6: $\beta$ is fixed to 1.With various q, we use immunized nodes proportion to illustrate how network efficiency $\boldsymbol{\varepsilon}$ changes. We draw our result from the average solutions of 50 independent experiments.

What is more, by introducing therapy mechanism into target immunization, i.e. decrease weights of the edges connected to nodes with high degree, and by constructing an accurate mathematical model, we deduce some applicable knowledge. The usefulness of the immunization therapy strategy has been proved by experiments and it is significant in guiding some real-world immunization performances. Besides, except for normal network structure, our model is also fit for some specific network structures like community structure.


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## 6. References

[1] C. Nowzari, V. M. Preciado, and G. J. Pappas, “Analysis and control of epidemics: A survey of spreading processes on complex networks,” IEEE Control Systems, vol. 36, no. 1, pp. 26–46, 2016.

[2] L. G. A. Zuzek, C. Buono, and L. A. Braunstein, “Epidemic spreading and immunization strategy in multiplex networks,” 2015, pp. 49–56.

[3] G. Andrea and B. W. Rogers, “Diffusion and pro-tection across a random graph,” Network Science, vol. 3, no. 3, pp. 361–376, 2015.

[4] A. Gola, A. A. Walters, S. Uderhardt, A. M. Salman, B. R. Halbroth, S. M. Khan, C. J. Janse, R. N. Germain, A. J. Spencer, and A. V. Hill, “Prime and target immunization protects against liver-stage malaria,” 2017.

[5] Q. Wu and Y. Lou, “Local immunization pro-gram for susceptible-infected-recovered network epidemic model,” Chaos, vol. 26, no. 2, p. 67, 2016.

[6] W. Huang, T. Ho, M. Langberg, and J. Kliewer, “Single-source/sink network error correction is as hard as multi-ple-unicast,” in Communication, Control, and Computing, 2015, pp. 423–430.

[7] P. W. L. Fong, “Preventing sybil attacks by privi-lege attenuation: A design principle for social net-work systems,” in IEEE Symposium on Security and Privacy, 2011, pp. 263–278.

[8] R. Zahedi and M. Khansari, “Network immu-nization under limited budget using graph spec-tra,” International Journal of Modern Physics C, vol. 27, no. 08, 2016.

[9] C. Gao, L. Zhong, X. Li, Z. Zhang, and N. Shi, “Combination methods for identifying influential nodes in net-works,” International Journal of Mod-ern Physics C, vol. 26, no. 06, pp. 042 805–22, 2015.

[10] S. Goyal, S. Jabbari, M. Kearns, S. Khanna, and J. Morgenstern, “Strategic network forma-tion with attack and immunization,” in Inter-national Conference on Web and Internet Eco-nomics, 2016, pp. 429–443.

[11] T. Yoshida and Y. Yamada, “A community structure-based approach for network immuniza-tion,” Computational Intelligence, 2015.

[12] R. C. Mucic, J. J. Storhoff, C. A. M. And, and L. Letsinger, “Dna-directed synthesis of binary nanoparticle net-work materials,” J.am.chem.soc, vol. 120, no. 48, 2016.

[13] J. Manni, A. Aziz, F. Gong, U. Loganathan, and Amin, “Network-based binary file extraction and analysis for malware detection,” 2014.

[14] M. Altmann, “Susceptible-infected-removed epi-demic models with dynamic partnerships,” Jour-nal of Mathemat-ical Biology, vol. 33, no. 6, p. 661, 1995.

[15] E. Purwati,“Model matematika sird (susceptibles-infected-recovery-deaths) pada penyebaran virus ebola,” 2016.

[16] J. Hein-Magnussen, P. Kuhlmann, and L. Sten-feldt Hansen, “Communications terminal, a sys-tem and a method for internet/network tele-phony,” 2015.

[17] R. J. Bies, “Internet-based submission of cable network content,” 2015.

[18] S. Abdallah, “A new methodology for generalizing unweighted network measures,” Physics, 2016.

[19] L. O. Prokhorenkova, “Global clustering coeffi-cient in scale-free weighted and unweighted net-works,” Internet Mathematics, vol. 12, no. 1, pp. 47–58, 2015.

[20] M. R. Welford, “Mortality clines, epidemic trans-mission velocities and transportation networks during medieval black death,” 2012.