Model of epidemic control based on quarantine and message delivery

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HIGHLIGHTS

- Propose two parameters, denoting different isolating rates on latent period and invasion period respectively.
- Propose the method of delivering specific messages.
- Experiments on different complex networks show that the method has great performance.
- A location-based social network is used in the experiments, which is justified to have 30% similarity with the real world.

ARTICLE INFO

ABSTRACT

The model provides two novel strategies for the preventive control of epidemic diseases. One approach is related to the different isolating rates in latent period and invasion period. Experiments show that the increasing of isolating rates in invasion period, as long as over 0.5, contributes little to the preventing of epidemic; the improvement of isolation rate in latent period is key to control the disease spreading. Another is a specific mechanism of message delivering and forwarding. Information quality and information accumulating process are also considered there. Macroscopically, diseases are easy to control as long as the immune messages reach a certain quality. Individually, the accumulating messages bring people with certain immunity to the disease. Also, the model is performed on the classic complex networks like scale-free network and small-world network, and location-based social networks. Results show that the proposed measures demonstrate superior performance and significantly reduce the negative impact of epidemic disease.

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1. Introduction

Though the medical conditions are improved significantly, epidemics have never been away from human world, especially in developing countries. Of growing concerns are adverse synergistic interactions between the emerging diseases and other infectious. Poor sanitation and lack of medical knowledge lead to the wide spreading of disease in developing countries, such as Ebola and MERS (Middle East Respiratory Syndrome Coronavirus). Besides, some of traditional contagions are resistant to drug treatments now, such as malaria, tuberculosis, and bacterial pneumonia. These kinds of diseases are defined as new emerging infectious disease (EID), which have increased in the past 20 years and will keep growing in the near future [1]. There are several remarkable characteristics in the EID: unpredictable, not preventable, irremediable, high mortality, rapid transmission and wide scope of influence. The expansion of disease spreading can lead to social panic at some level. It is hence imperative to study effective control strategies to prevent the disease diffusion.

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Table 1
Latent period of several common infectious diseases.

Disease	Between	And	Period
Cholera	0.5	4.5	Days [16]
Malaria	9	14	Days [17]
Ebola	1	21 (95%), 42 (98%)	Days [18]
Marburg	5	10	Days [19]
Measles	9	12	Days [20]
SARS	1	10	Days [21]
Smallpox	7	17	Days [22]
MERS	2	14	Days [23]
Average-day	4.3125	13.6503	Days

Many of researchers have tried to explore the prevention measures of EID. As a practical method, the information diffusion on epidemic dynamic has attracted much attention in recent years. A path-breaking work in this field was taken by Funk et al., who proposed an epidemiological model that considers the spread of awareness about the disease [2,3]. Lima et al. proposed that the propagation of disease can be reduced by the spreading of immune information, which make individuals resistant to disease and then work against the epidemic propagating [4]. A model of competing epidemic spreading over completely overlapping networks was proposed by Karrer and Newman, revealing a coexistence regime in which both types of spreading can infect a substantial fraction of the network [5]. Wang and Tang distinguish two types of disease spreading and proposed the dynamic model of asymmetrically interacting and disease spreading. Their research focuses on three problems: the different network structures and information spreading dynamics; the asymmetric effects of one type of spreading dynamics on another; the timing of the two types of spreading [6]. Moreover, several researches are also meaningful to us [7–12]. Trpevski et al. explored the rumors propagation in multiple networks [7]. Zhang et al. proposed a model considering time delay and stochastic fluctuations [8]. Ababou et al. investigated the spreading of periodic diseases and synchronization phenomena on exponential networks [9]. Rakowski et al. put forward an individual based model to study the effects of influenza epidemic in Poland. A simple transportation rule is established to mimic individuals' travels in dynamic route-changing schemes, allowing for the infection spread during a journey [10]. All the above efforts are worth approving but the specific ways of message spreading are ignored. This paper enriches the research in this area, and the proposed methods are shown to have a significant effect in epidemic prevention.

The paper proceeds as follows. In Section 2, we first describe the characteristics of epidemic, then the SIQM models are developed, which contain the disease prevention measures based on quarantine and message delivery. Section 3 gives the sensitivity analysis of the model on classical complex networks. In Section 4, the common regularity of human mobility and experiment results on location-based social network are given. The paper is concluded in Section 5.

2. Epidemic dynamic model

2.1. Characteristics of epidemic

Normally, the process of infectious disease can be divided into three stages: susceptible period, infection period, recovery period [13]. The infection period is further divided into latent period and invasion period. The former is characterized by the tiny indisposition; the latter is featured by onset of clinical signs and symptoms. Moreover, according to the research of Lessler et al., the latent period is essential to the investigation and control of infectious disease [14]. In this period, the infectious individuals have obtained the ability to spread diseases, yet without obvious symptom to arise attention, e.g., the latent period of influenza remains only one to three days, yet the epidemic can sweep through a city in less than six weeks. Furthermore, according to the health experts Sartwell's research, the period of latent period varies between individuals in the same regular fashion as do other biological characteristics [15]. The distribution of days seems to follow the "normal" curve. Based on their theory, we investigate several common infectious diseases and collect their incubation period in Table 1 and then get the average length of latent period which approximates to 7 days.

To sum up, two assumptions are proposed here as the basic precondition of model: (1) the infection period of epidemic is divided into latent stage and invasion stage in epidemiology. Because the disease in former stage is hard to observe and diagnose, we preset a relative low value to its isolating rate which ranges from 0.01 to 0.5. The rate in invasion period ranges from 0.5 to 1. (2) To simplify the model, the average length of latent period is uniformly set to 7 days, which is important to the experiments in Section 4. The value is deduced by results shown in Table 1.

2.2. Definition of the model

Individual status in the model is divided into four types: susceptible, infected, in quarantine and in messaging. The susceptible means the people who are vulnerable to the disease. The infected denotes people who have already been infected consciously or unconsciously. When in quarantine, it means the infected ones have been isolated and will not spread disease anymore. When in "messaging" status, the specific persons, denoting the ones who have just been isolated or their directly
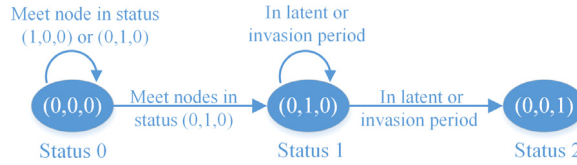
Fig. 1. The transition process of node status.

connected neighbors, would deliver specific messages to their neighbors. Timely isolation and message-delivery are the main prevention measures in our model, especially the latter. Just imagine that a new infectious disease outbreaks at a certain area and the messages about disease are open to public a few months later, it would be a large cost to control its scale, duration and damage.

Consider the epidemic SIR (Susceptible–Infectious–Recovery) model, which is firstly proposed by Kermack and McKendrick [24]. Now we improve the model by modifying the "recovery" status to "quarantine" status then constructing an enclosed and one-way evolution system. Infection rate is defined as u and isolation rate as v. Finally, the modified partial differential equations are given by:

$$
\begin{cases}
\dfrac{dS}{dt} = -uSI \\[4pt]
\dfrac{dI}{dt} = (1-u)\,SI - vI \\[4pt]
\dfrac{dQ}{dt} = vI,
\end{cases}
\tag{2.1}
$$

which are the basis of equations in (2.2).

Now, we will consider the specific situation of each individual in the population. Assume a closed population of N individuals, connected by a neighboring structure which is denoted by the undirected and unweighted graph $G = (V, E)$, with nodes (termed as V) representing individuals and edges (termed as E) representing connections between individuals. Let L_i represent the adjacency list of node i, i.e., if $(i, j) \in E$ set $j \in L_i$; $j \notin L_i$ otherwise. At time t, each node i can be in one of three statuses: susceptible, infectious and isolated. The status is described as a status vector, containing a single 1 in the position corresponding to the current state, and 0 everywhere else. Let

$$Status_i\,(t) = [s_i^0\,(t)\,, s_i^1\,(t)\,, s_i^2\,(t)].$$

The probability function of each status is set to

$$Prob_i\,(t) = [p_i^0\,(t)\,, p_i^1\,(t)\,, p_i^2\,(t)].$$

The state transition processes is shown in Fig. 1. Status 0 is the initial state, and status 2 is the final state. When connected to the node in status 1, nodes in status 0 may turn into status 1 at a certain rate. Nodes in status 1 could keep their status or convert into status 2 on either latent period or invasion period. Evolution of the model is given by the formulas

$$
\begin{cases}
p_i^0\,(t+1) = s_i^0\,(t)\,(1 - u_i\,(t)) \\[4pt]
p_i^1\,(t+1) = s_i^0\,(t)\,u_i\,(t) + (1 - v_i\,(t))\,s_i^1\,(t) \\[4pt]
p_i^2\,(t+1) = v_i\,(t)\,s_i^1\,(t) + s_i^2\,(t)\,,
\end{cases}
\tag{2.2}
$$

$$Status_i(t+1) = Multi\ Realize[Prob_i(t+1)], \tag{2.3}$$

where *Multi Realize* can be interpreted as a realization of random realization for the probability distribution $Prob_i\,(t)$, or a mapping from probability vector to status vector. For each node i, $u_i\,(t)$ and $v_i\,(t)$ represent its infecting rate and isolating rate respectively. In the study in Section 2.1, the isolating rate of disease is defined as $\{v_1 | v_1 \in (0, 0.5)\}$ when in latent period. When in invasion period, the isolating rate is defined as $\{v_2 | v_2 \in [0.5, 1)\}$. The infecting rate $u_i\,(t)$ is given by

$$u_i\,(t) = \left(1 - \prod_{j=1}^{N} \left(1 - \beta l_{ij} s_j^1\,(t)\right)\right)(1 - m_i\,(t))f\left(\frac{1}{\theta}\right). \tag{2.4}$$

Each node in status 1 tries to infect its neighbor node at time t. Each try may be successful with a rate β. If $j \in L_i$, set $l_{ij} = 1$, and $l_{ij} = 0$ otherwise. In formula (2.4), if $m_i\,(t) = 0$, set $f\left(\frac{1}{\theta}\right) = 1$; If $m_i\,(t) > 0$, set $f\left(\frac{1}{\theta}\right) = \frac{1}{\theta}$. Parameter θ could take any of values in the set $\{1, 2, 3, 4, 5, 6, 7, 8, 9, 10\}$, which stand for the quality of information. It could be measured by the characteristics of messages like authority, accuracy, availability, etc. The higher value corresponds to the higher quality. Moreover, the information quantity is defined by

$$m_i\,(t) = a \sum_{i=1}^{n_i^1} \frac{1}{2^i} + b \sum_{i=1}^{n_i^2} \frac{1}{2^i}\ (a + b = 1, a > b > 0)\,. \tag{2.5}$$

Lima et al. have proposed that the public-facing information distribution may cause a considerable amount of neglect [4]. Therefore, the directional message delivery is essential to warn the crowds who are under high risk of being infected. Simultaneously, as the receiving and accumulating of messages, the newly received messages perform a gradually decreasing effect. Accordingly, geometric series is adopted here to simulate the process. Owing to its convergence properties, $m_i(t)$ will never be over 1. n_i^1 and n_i^2, representing the total amount of direct and indirect messages respectively, are given by

$$\begin{cases} n_i^1(t) = \sum_{j=1}^{N} l_{ij} s_j^2(t) \\ n_i^2(t) = \sum_{j=1}^{N} \left(l_{ij} \sum_{k=1}^{N} l_{jk}\, \bar{l}_{ik}\, s_k^2(t) \right). \end{cases} \tag{2.6}$$

The messages, sent from one node (in status 2) to its direct-neighbors, are defined as direct messages, while the messages sent to the direct-neighbors' neighboring nodes are defined as indirect messages. a and b are the influence rates of two types of messages. For node i, $n_i^1(t)$ represents the total number of its direct-neighbors who are isolated, and $n_i^2(t)$ represents the total number of its neighbors's neighboring nodes who are isolated.

At a certain time t, let $S(t) = \frac{1}{N}\sum_{i=1}^{N} s_i^0(t)$, $I(t) = \frac{1}{N}\sum_{i=1}^{N} s_i^1(t)$, $Q(t) = \frac{1}{N}\sum_{i=1}^{N} s_i^2(t)$ represent the ratio of nodes in status 0, 1 and 2 respectively; let $U(t) = \frac{1}{N}\sum_{i=1}^{N} u_i(t)$, $M(t) = \frac{1}{N}\sum_{i=1}^{N} m_i(t)$ stand for the average value of infectious rate and average information quantity respectively.

To facilitate understanding, a simple example is given in Fig. 2. Initialize the global parameters with $u_i = 0$, $v_1 = 0.2$, $v_2 = 0.9$. Set local variables as $m_i = 0$, $a = 0.6$, $b = 0.4$, $\theta = 5$, $n_1 = 0$, $n_2 = 0$. The length of latent period is defined as one time unit. Each node in status 1 could spread disease to its neighbors and get isolated at a random probability of v_1 (in latent period) or v_2 (in invasive period). Each node in status 2 sends messages to its neighbors and neighbors' neighboring nodes. Those received messages will improve the nodes immunity and decrease infection rate. Concretely, "Remark 1" is calculated by

$$u_i = \left(1 - (1 - 0.2)^2\right) \times (1 - 0.2) \times \frac{1}{5} = 0.0576.$$

"Remark 2" is obtained by

$$m_i = 0.6 \times \left(\frac{1}{2} + \frac{1}{4}\right) + 0.4 \times \left(\frac{1}{2} + \frac{1}{4} + \frac{1}{8}\right) = 0.8.$$

All the infectors are finally isolated, and several lucky nodes become immune to the disease like node 7.

3. Behavior of the model on complex networks

This section presents the results of model behavior on complex networks. BA scale-free networks and WS small-world networks are contained, generated by algorithms. All the experiments are performed initially with two nodes in status 1 and others in status 0. If there are N nodes sorted in number in network, then the $(N/3)$th, $(2N/3)$th nodes are picked up as the initial nodes in status 1.

3.1. BA scale-free network

First consider the scale-free network, which is generated by BA algorithm [25]. The network begins with m_0 nodes. Every time adding a new node, we connect it to m ($m < m_0$) nodes which are already in the network. Finally, the average degree of network $\langle k \rangle$ is equal to $2m$, and degree distribution p_k is approximate to k^3.

In Fig. 3, we present the results of model behavior under different conditions and situations. If without message diffusion (Fig. 3(a) and (b)), most of nodes become infected in the epidemic. Under quarantine measures, there is a red and unimodal curve shown in Fig. 3(b), which represents the number of nodes in status 1. Statistical results show that the speed of disease spreading is delayed fivefold, and the peak value is reduced by 20%. In Fig. 3(c), we present a more acceptable result, where over a third of population is preserved from the disease only if received several messages in a medium-degree quality, and the peak of infection is decreased by around 50%. Let

$$P_0(t) = \frac{1}{N}\sum_{i=1}^{N} p_i^1(t), \qquad P_1(t) = \frac{1}{N}\sum_{i=1}^{N} p_i^2(t), \qquad P_2(t) = \frac{1}{N}\sum_{i=1}^{N} p_i^3(t),$$

then we get three dash lines which are nearly overlapping the solid lines. It could be equivalently represented for $S(t) = P_0(t)$, $I(t) = P_1(t)$, $Q(t) = P_2(t)$. Moreover, a clear comparison can be seen in Fig. 3(d), where $U(t)$, which denotes the average infection rate, is investigated under three different situations as mentioned above.

Another finding is shown in Fig. 3(e) and (f). As the value of v_1, which denoted the isolation rate in latent period, grows from 0.01 to 0.5, the peak value of $I(t)$ reduces from 0.95 to 0.05 (Fig. 3(e)); With the same parameters, we change the value
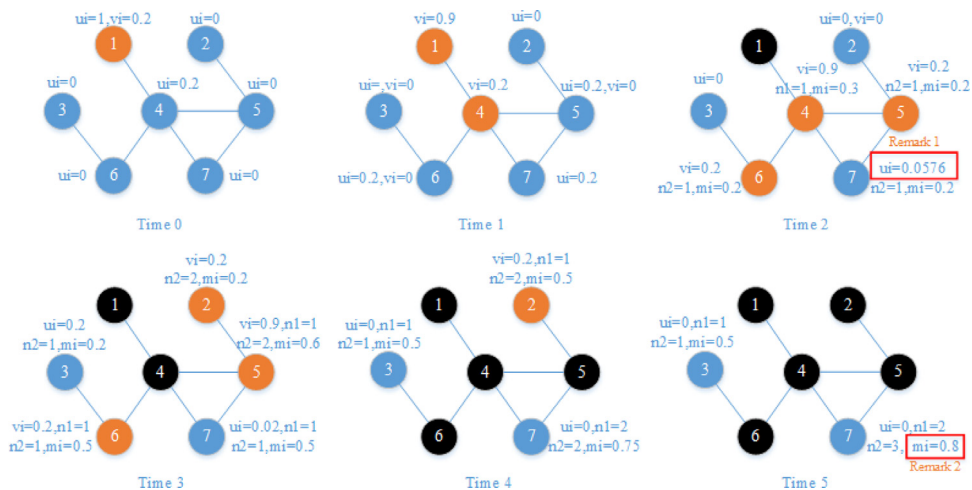
Fig. 2. A simple example of model operating. Node in status 0, 1, 2 is respectively dyed blue, orange and black. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)
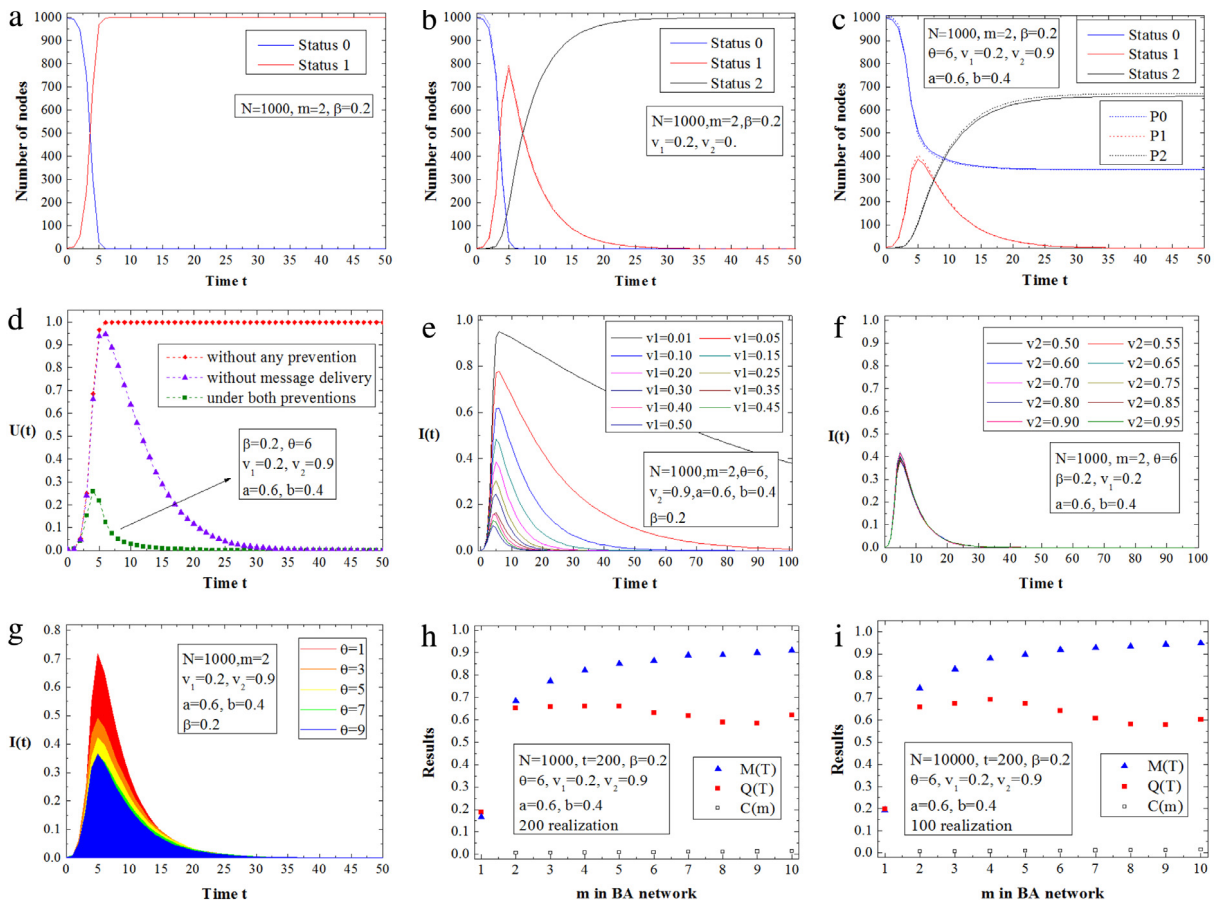
Fig. 3. (a)–(c): Number of nodes in status 0, 1 and 2 as time changes. Consider the disease dynamic process without any prevention measures (first panel), with quarantine measures (second panel) and with quarantine and message-delivery measures (third panel). (d)–(g): Behavior of the model with different parameters. Including value of the average probability to be infected (fourth panel), proportion of the nodes in status 1 by setting multiple value of v_1(fifth panel), v_2 (sixth panel) and θ (seventh panel). (h) and (i): The steady-status behavior of the model. Part of results is obtained by averaging over 200 network realizations, running for 100 time units on 1000 nodes (eighth panel). Others are performed on the average of 100 network realizations, running for 100 time units on 10,000 nodes (fifth panel). Time unit is defined on a week basis here. The same is below. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

of v_2 from 0.5 to 0.95, then obtain the peak value $I_{max}(t) \approx 0.4$ (Fig. 3(f)). There is a particular conclusion: the increasing of isolation rate in invasion period, as long as over 0.5, contributes little to the preventing of epidemic; the improvement of isolation rate in latent period is the key to control the spreading of disease.

Next in Fig. 3(g), the influences of message quality and isolation rate are measured. For the same parameters as before, the higher value of θ corresponds to the lower value of $I(t)$. It can be predicted by $\Delta I \sim -1/\theta^2$. That is

$$\frac{dI(t)}{d\theta} = \frac{dP_1(t)}{d\theta} = -\frac{1}{\theta^2}\Theta(t),$$

where

$$\Theta(t) = \frac{1}{N}\sum_{i=1}^{N} s_i^1(t)\left(1 - \prod_{j=1}^{N}\beta l_{ij}S_j^2(t)\right)(1 - m_i(t)).$$

Another conclusion is obtained here: even though a higher value of θ corresponds to the lower infection rate, once $\theta > 7$ the effect is changed slightly. It implies that the disease is easy to control as long as the messages achieve a certain quality.

In Fig. 3(h) and (i), we display the steady-status behavior of the model on BA networks with different value of m. Similar results are obtained by the experiments on 1000-nodes network and 10,000-nodes network. The clustering coefficient in scale-free network [26] is get by

$$C(m) = \frac{m^2(m+1)^2}{4(m-1)}\left(\ln\left(\frac{m+1}{m}\right) - \frac{1}{m+1}\right)\frac{\ln^2(N - m_0)}{N - m_0}.$$

It is obvious that there is not obvious characteristic of clustering in scale-free network. $M(T)$ represents the average information quantity on the stabilizing time T. $Q(T) = \sum_{t=1}^{T} I(t)$ denotes the ultimate scale of population in quarantine. They are used to measure the population immunity and magnitude of disease. As m changes from 1 to 10, the average information quantity presents exponential distribution, which illustrates that the flow of information is highly improved as the increasing of average degree $\langle k \rangle$. The proportion of isolators presents a fluctuating distribution. For $m = 1$, both $M(T)$ and $Q(T)$ are sustained at around 0.2. At this point, despite the weak immunity of population, the epidemic will not expand for the low connectivity of network.

3.2. WS small-world network

In this part, we consider the model behavior on small-world networks. The networks are generated by WS algorithm, which are firstly distributed in an irregular circular shape and contain N nodes. Every node connects to K nodes nearby, with $K/2$ in the clockwise and $K/2$ in the anticlockwise and $N >> K >> \ln(N) >> 1$. Later rewire each edge at probability p. Change the parameter p from 0 to 1 then the generating network is translated from regular network into random network. Self-loops or multiple edges between nodes are not allowed. Clustering coefficient in WS small-world network [27] is given by

$$C(K, p) = \frac{3(K-2)}{4(K-1)}(1 - p)^3.$$

The steady-status in WS network achieves the same level ($Q(T) \approx 0.65$) as in BA network by setting $p = 0.01$ and $K = 6$, which can be studied from Fig. 4(a) and (b). By increasing the value of p, the randomness of network is improved. The networks with high regularity and low degree are hard to spread disease, e.g. the networks in Fig. 4(a) with $p = 0.01$, $K = 4$; The increasing K contributes to the disease spreading theoretically but finally is balanced by the increasing value of $M(T)$; In Fig. 4(c), we find that in a limited range, the more irregular the network is, the more easy the disease spreads; Finally in Fig. 4(d), the small-world network is transformed into a completely random network by setting $p = 1$. There seems to be no remarkable differences between results in Fig. 4(b) and (d). It illustrates that the network topologies contribute little to disease spreading, as the generating parameter p is over 0.1. Fig. 4(e) and (f) give a comparison about the model behavior on scale-free networks and small-world networks. Results show that the infectious disease spreads more rapid in scale-free network. Epidemics in small-world networks appear three characteristics corresponding to the number of infectors: slower growth, slower convergence and lower peak.

4. Behavior of the model on social network

4.1. Regularity of human mobility

In most studies, the meta-population networks are fixed. Yet in actual world, the positions of individuals change every time. Thus it is meaningful to consider the regularity of human mobility when analyzing the disease spread in real world. Cho, Myers and Leskovec have made a research about this issue [28]. As they put forward, "short-ranged traveling is periodic both spatially and temporally and not effected by the social network structure, while long-distance travel is more influenced by social network ties". They have proved that social relationships can cover about 10%–30% of all human movements, while periodic behavior covers 50%–70%. We conclude their achievement systematically below as the experimental basis:
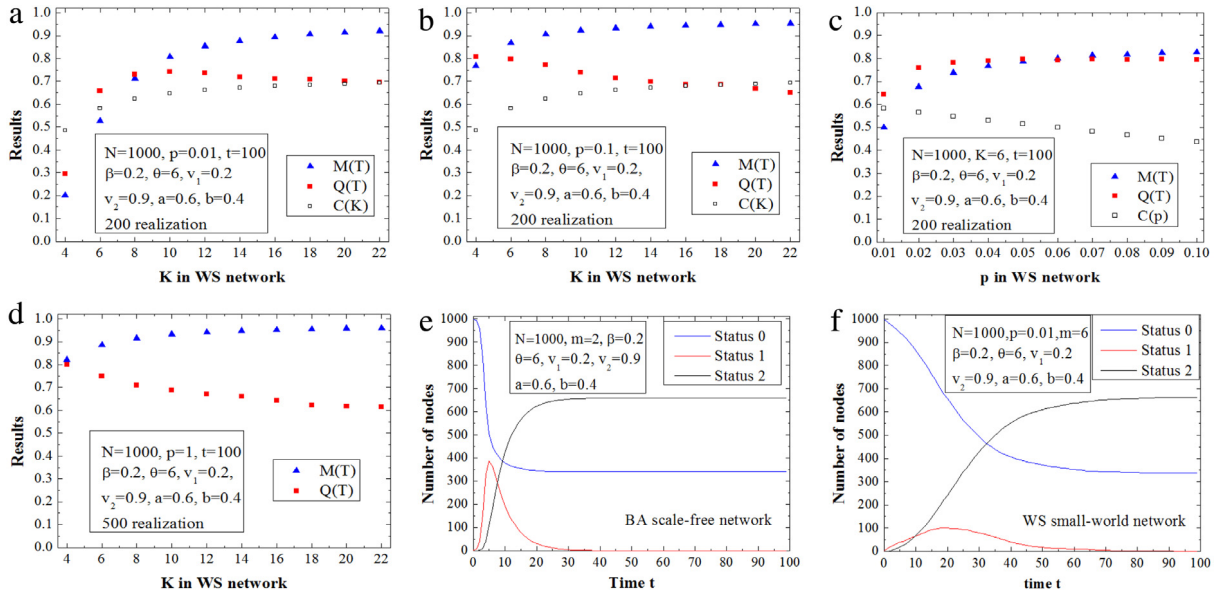
Fig. 4. (a)–(d): The steady-status behavior of the model. Respectively set different p and K. Results are obtained by averaging over 200 network realizations and running for $t = 100$ time units in each realization. (e) and (f): Comparison between BA scale-free network and WS small-world network. Blue, red and black curves represent the number of nodes in status 0, 1 and 2 respectively, as time changing. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

- Human activity presents the periodic prosperity and recession. The period is on a weekly basis. From Monday to Friday, human activity is highly consistent, i.e., moving from home to work station then going back home. On weekends, the activities are scattered and inconclusive.
- 100 km is the typical human radius of "reach" as it takes about 1–2 h to drive such distance. Because of the geographically non-uniform distribution over the earth, human tend to cluster in cities and the homes of friends at around 100 km present a similar kink.
- User home locations are not explicitly given yet can be inferred by defining the home location as the average position of check-ins within the scope of 25 km. This method to infer home locations is demonstrated with 85% accuracy. Likewise, we say that user A "visits" her friend B if A checks-in within radius r of B's home (set $r = 25$ km).
- Long-distance travel of people is usually influence by friends. The relative influence of a friend who lives 1000 km away is 10 times greater than the influence of a friend who lives 40 km away.
- There is a 30% possibility that a user who travels more than 100 km from her home will pay a visit to an existing friend's home. Moreover, the probability remains constant after the 100 km mark though the number of friends decreases with the distance.
- "Check-in" data is with a spatial accuracy of about 3 km.

Above all, individuals can be geographically divided into: the intimates (with a largest probability to be infected) within 25 km; the fellows (with a larger probability to be infected) within the range of 25–100 km; the potential contacts (with a medium probability to be infected) beyond 100 km but within 1000 km; the distant contacts (with small probability to be infected) beyond 1000 km. Four types could transform into each other as distance changes.

4.2. Experiments based on SNAP dataset

The dataset is obtained from Stanford Network Analysis Project (SNAP), named "loc-Brightkite". It collects a total of 4,491,143 check-ins over the period of Apr. 2008–Oct. 2010, consisting of 58,228 nodes and 214,078 edges. The network was originally directed but then constructed as a network with undirected edges. To keep structural integrity and information adequacy, we have weeded out the incomplete data during years of 2008 and 2010, meanwhile, removed the items losing location messages and taken an average of all the check-ins per day as the final position. After processing, 35 418 nodes and 428 158 directed edges are picked out and constitute the relationship network. The statistical properties of the network are shown in Table 2.

In Fig. 5 we show the geographic position (left panel) and relational network (right panel), where nodes are positioned using the geographic locations of Mar. 2009. Initially, all the individuals are distributed around the world, mostly in North America, Europe, Japan and New Zealand. It seems like that people who lived in America, Europe and Japan tend to keep a close contact with each other. Especially, there are most lines scattered from America to other districts. It is supposed that
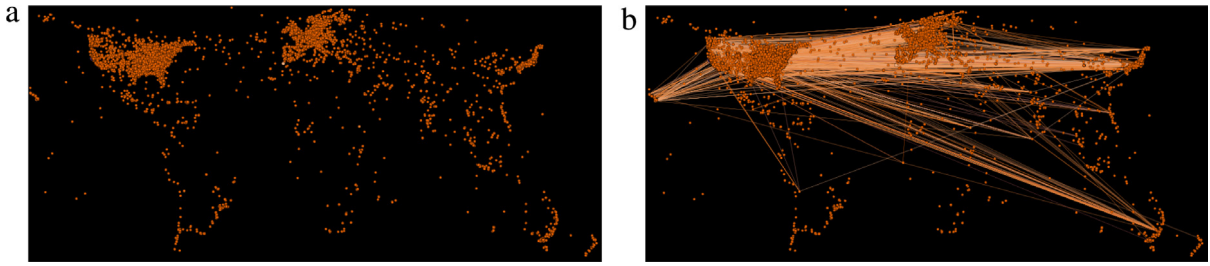
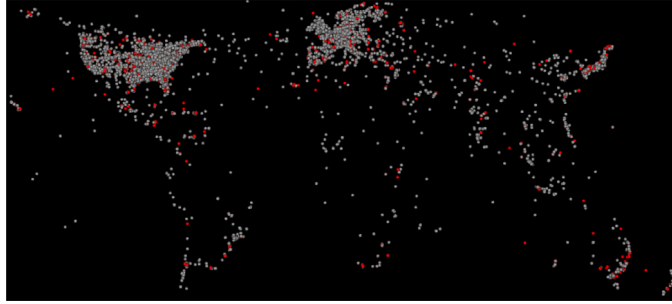**Fig. 5.** (a) Geographic traces of population; (b) Network of contacts.

Fig. 6. The result that epidemic spreads for three months.
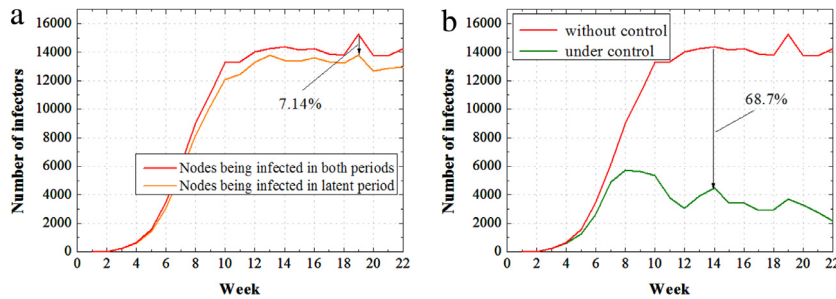

Fig. 7. The spread trend of epidemic in population. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Americans tend to travel around the world and live in foreign countries, meanwhile keeping a close touch with their internal friends.

The mechanism of disease spreading and preventing is as follows. For node i, which has been infected, the first week is defined as latent period, when it is hard to observe the obvious symptoms of disease. The infected node would keep its periodic activities in this period, and most of neighbors within scope of 25 km would be infected unconsciously. Until the next week (invasion period), the node would be isolated at a high probability of v_2. If isolated, the node would deliver immune messages to its direct and indirect neighboring nodes. If not, it would continue to spread disease within the scope of 100 km. The process is derived from Eqs. (2.2) and (2.3).

Without any prevention measures, the disease would spread all over the world within three months (Fig. 6). It can be attributed to the convenient and fast movement of human beings. In Fig. 7(a), the red line describes the diseases which are infectious in both latent period and invasion period, while the orange line depicts diseases which are infectious only in latent period. Making a comparison, the former is over 7.14% than the latter. After analyzing, we summarize two reasons to express the weak distinction: firstly, most neighboring nodes have been infected and the space of infection becomes smaller in invasion period; secondly, the high isolation rate reduces the number of infectors. The green line in Fig. 7(b) represents the number of infectors while taking preventive measures. Compare to the previous red line, number of infectors decreases over 68.7%.

We display the details of disease spreading process in Fig. 8. Green points represent the susceptible people; red points stand for the infected people; blue points denote both the isolators and immune people (who collect enough messages). Source of infection is located in North America. To simulate the free spreading process of disease, there is no prevention measure in the beginning. As we can see from the first four panels, the uncontrolled disease spreads from America to the Europe and Asia successively. In fact, the prevention measures are started at the fourth week but remain with a weak and
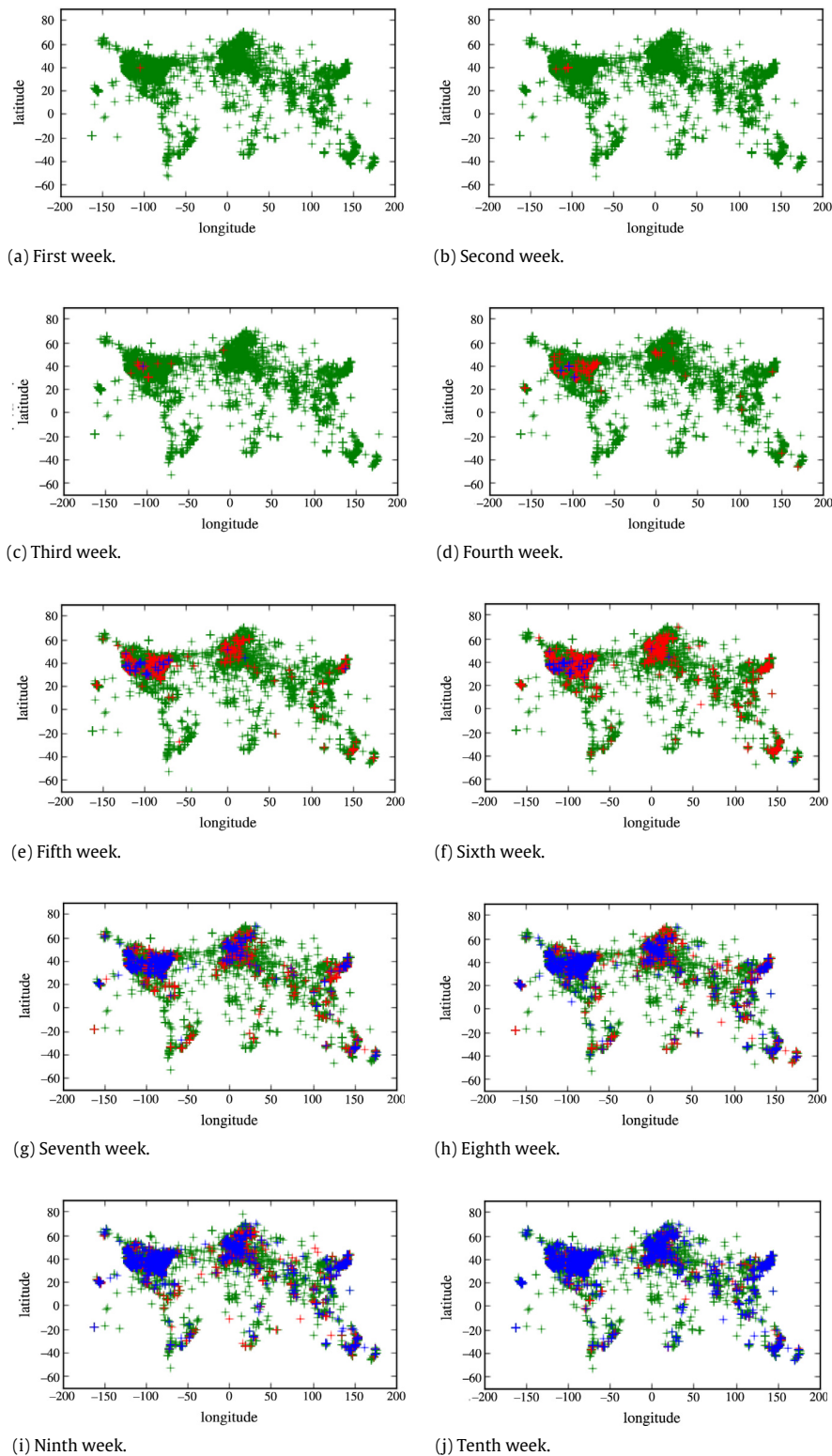
Fig. 8. Epidemic dynamics under measures of quarantine and message-delivery. The process is developed by the model in Section 2.2. Set $v_1 = 0.2$, $v_2 = 0.9$, $a = 0.6$, $b = 0.4$, $\theta = 7$. First begin with a random node in North America as source of infection. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Table 2
Statistical properties of the network.

Parameter	Value
Nodes	35,418
Directed-edges	428,158
Total check-ins	978,118
Average clustering coefficient	0.016
Average degree	2.647
Diameter (longest shortest path)	16
Average path length	41.466
Modularization	0.965
Network diameter	501

limited power. We attribute it to the latent period of disease, which is set to a week basis. Later in the fifth and sixth weeks, the regional infectious disease evolves into a global outbreak. Most of patients are experiencing the invasion period clinically and may be isolated at a very high rate. The immune measures show their great effects right now. The covering square of blue points is expanding. After nine weeks, the epidemic is under control completely.

5. Conclusion

In this paper we provide a novel model about the control of infectious diseases and perform it on different networks. The main conclusions are as follows.

First, in the model, two kinds of disease prevention measures are proposed: quarantine and message delivery. Under the premise of medical research, we assume the rates of patients who are isolated in latent period and invasion period are different. Besides, the specific messages sent from the isolators to their direct and indirect neighbors are originally proposed. Information quality and information accumulating effect are also considered in the model.

Second, several valuable results are obtained by simulating the model in scale-free and small-world networks: (i) The increasing of isolation rate in invasion period, as long as over 0.5, contributes little to the prevention of epidemic; the improvement of isolation rate in latent period is key to control the spread of disease. (ii) Infectious diseases are easy to control as long as the propagating message achieves a certain quality. (iii) In the scale-free network with a very low degree ($\langle k \rangle = 2$), the epidemic will not diffuse on the network despite the weak immunity in population. (iv) Compared to scale-free network, epidemics in small-world network appear three characteristics corresponding to the number of infectors: slow growth, slow convergence and lower peak.

Finally, in general, the mobility and communication of population can be time-varying. Based on the specific research about human periodic activity, we constitute a social network which has a certain similarity with the real world, in which the activity of human presents periodic trend and certain randomness. Experimental results show that the proposed strategies have great effect on the control of disease.

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References

[1] L.H. Taylor, S.M. Latham, E.J. Mark, Risk factors for human disease emergence, Philos. Trans. R. Soc. Lond. B Biol. Sci. 356 (1411) (2001) 983–989.
[2] S. Funk, E. Gilad, C. Watkins, et al., The spread of awareness and its impact on epidemic outbreaks, Proc. Natl. Acad. Sci. 106 (16) (2009) 6872–6877.
[3] S. Funk, E. Gilad, V.A.A. Jansen, Endemic disease, awareness, and local behavioural response, J. Theoret. Biol. 264 (2) (2010) 501–509.
[4] A. Lima, M. De Domenico, V. Pejovic, et al., Disease containment strategies based on mobility and information dissemination, Sci. Rep. 5 (2015) 10650.
[5] V. Marceau, P.A. Noël, L. Hébert-Dufresne, et al., Modeling the dynamical interaction between epidemics on overlay networks, Phys. Rev. E 84 (2) (2011) 026105.
[6] W. Wang, M. Tang, H. Yang, et al., Asymmetrically interacting spreading dynamics on complex layered networks, Sci. Rep. 4 (2014) 5097.
[7] D. Trpevski, K.S. Tang Wallace, L. Kocarev, Model for rumor spreading over networks, Phys. Rev. E 81 (5) (2010) 056102.
[8] Y. Zhang, Q. Zhang, X.G. Yan, Complex dynamics in a singular Leslie–Gower predator–prey bioeconomic model with time delay and stochastic fluctuations, Physica A 404 (2014) 180–191.
[9] M. Ababou, N. Vandewalle, N. Moussa, et al., Spreading of periodic diseases and synchronization phenomena on networks, Physica A 392 (10) (2013) 2526–2531.
[10] F. Rakowski, M. Gruziel, Ł. Bieniasz-Krzywiec, et al., Influenza epidemic spread simulation for Poland—a large scale, individual based model study, Physica A 389 (16) (2010) 3149–3165.
[11] C. Lagorio, M.V. Migueles, L.A. Braunstein, et al., Effects of epidemic threshold definition on disease spread statistics, Physica A 388 (5) (2009) 755–763.
[12] K. Afassinou, Analysis of the impact of education rate on the rumor spreading mechanism, Physica A 414 (2014) 43–52.
[13] P.H.N. Wood, The mathematical theory of infectious diseases and its applications, Immunology 34 (5) (1978) 955–956.
[14] J. Lessler, N.G. Reich, R. Brookmeyer, et al., Incubation periods of acute respiratory viral infections: a systematic review, Lancet Infect. Dis. 9 (5) (2009) 291–300.
[15] P.E. Sartwell, The Distribution of incubation periods of infectious diseases, Am. J. Hyg. 51 (3) (1950) 310–318.
[16] J. Satsuma, R. Willox, A. Ramani, et al., Extending the SIR epidemic model, Physica A 336 (3) (2004) 369–375.

[17] A.K. Singh, V. Rajendran, A. Pant, et al., Design, synthesis and biological evaluation of functionalized phthalimides: A new class of antimalarials and inhibitors of falcipain-2, a major hemoglobinase of malaria parasite, Bioorg. Med. Chem. 23 (8) (2015) 1817–1827.
[18] E.M. Leroy, B. Kumulungui, X. Pourrut, et al., Fruit bats as reservoirs of Ebola virus, Nature 438 (7068) (2005) 575–576.
[19] V.E. Friedewald, Marburg hemorrhagic fever, in: Clinical Guide to Bioweapons and Chemical Agents, 2008, pp. 138–141.
[20] S.J. Russell, K.W. Peng, Measles virus for cancer therapy, in: Measles, Vol. 330, Springer, Berlin, Heidelberg, 2009, pp. 213–241.
[21] W.H. Seto, D. Tsang, R.W.H. Yung, et al., Effectiveness of precautions against droplets and contact in prevention of nosocomial transmission of severe acute respiratory syndrome (SARS), Lancet 361 (9368) (2003) 1519–1520.
[22] I.K. Damon, C.R. Damaso, G. McFadden, Are we there yet? The smallpox research agenda using variola virus, PLoS Pathog. 10 (5) (2014) e1004108.
[23] A. Assiri, A. McGeer, T.M. Perl, et al., Hospital outbreak of Middle East respiratory syndrome coronavirus, N. Engl. J. Med. 369 (5) (2013) 407–416.
[24] W.O. Kermack, A.G. Mckendrick, A contribution to the mathematical theory of epidemics, Proc. R. Soc. Lond. 115 (772) (1927) 700–721.
[25] A.L. Barabási, R. Albert, Emergence of scaling in random networks, Science 286 (5439) (1999) 509–512.
[26] A. Fronczak, P. Fronczak, J.A. Hołyst, Mean-field theory for clustering coefficients in Barabási–Albert networks, Phys. Rev. E 68 (4) (2003) 046126.
[27] A. Barrat, M. Weigt, On the properties of small-world network models, Eur. Phys. J. B 13 (3) (2000) 547–560.
[28] E. Cho, S.A. Myers, J. Leskovec, Friendship and mobility: user movement in location-based social networks, in: Proceedings of the 17th ACM SIGKDD International Conference on Knowledge Discovery and Data Mining, 2011, pp. 1082–1090.