# SEIQR disease transmission on GA-network

W. Jumpen, S. Orankitjaroen, B. Wiwatanapathaphee and P. Boonkrong

***Abstract*****—** This paper aims to present a local network model of Susceptible-Exposed-Infectious-Quarantined-Recovered disease transmission taking into account the community structure of the population. The population structure is generated by genetic algorithm based on the network modularity concept for the heterogeneous property. The basic reproductive number of the model is derived and used to predict the epidemiological situation. In numerical simulation, the disease transmissions within and across the communities are considered. The results show that this approach is able to capture the essential feature of epidemic spreading in human community.

***Keywords*****—** complex network, genetic algorithm, community detection, disease transmission, multi-group epidemic model, network modularity, SEIQR network model, stability analysis

## I. Introduction

MATHEMATICAL theory of epidemic proposed by McKendrick and Kermack in 1927 has impacted on both understanding of epidemic scattering and public health planning via a long term forecasting from compartmental models [13], [14], [15], [28]. In these models, populations are divided to different classes based on the states of the disease and are able to change their status. The two standard compartmental models including Susceptible-Infectious-Recovered (SIR) and Susceptible-Exposed-Infectious-Recover (SEIR) epidemic models have been widely applied to study the spread of many infectious diseases such as smallpox, measles, cholera, influenza and tuberculosis [26]. To staunch the spread of infectious diseases, especially influenza, isolation strategy is often used as a practical intervention procedure to reduce the infectiousness from infected people to susceptibles. The quarantine class is thus taken into consideration in the SEIR model. This model is known as the SEIQR model [7], [9].

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W. Jumpen is with Department of Mathematics, Faculty of Science, Mahidol University, Cenre of Excellence in Mathematics, PERDO, CHE, Bangkok 10400, THAILAND.

S. Orankitjaroen is with School with Department of Mathematics, Faculty of Science, Mahidol University, Centre of Excellence in Mathematics, PERDO, CHE, Bangkok 10400, THAILAND. (corresponding author; e-mail: somsak.ora@mahidol.ac.th).

B. Wiwatanapataphee is with Department of Mathematics, Faculty of Science, Centre of Excellence in Mathematics, PERDO, CHE, Bangkok 10400, THAILAND.

P. Boonkrong is with Department of Mathematics, Faculty of Science, Mahidol University, Bangkok 10400, THAILAND.

In the compartmental models, uniform mixing is assumed, i.e., all susceptible individuals can catch the disease with the same infection rate. Thus, the compartmental models do not reasonably reflect the disease transmission in the human community. It has been realized that the structure of community is directly significant to the disease proliferation. In recent years, many researchers have focused on the study of disease transmission using the combination of the compartmental models and complex networks [1], [30], [34], [35]. The social contact at schools, offices and public places is the crucial factor for the propagation of infectious diseases like pertussis, SARS, H1N1 and H5N1, through the population [17], [33]. Dascalu et al. (2011) introduced the way to construct communities in a cellular automata for the study of disease transmission between children [5]. It is shown that the contact among people in the same community occurs a lot more than those among different communities. The way to study the transmission of epidemic should consider the community structure and the interaction of people in the communities. For community establishment in the network, most of recent algorithms for constructing a network use the network modularity as a practical measure to find clear partitions (communities) of the network [3], [6], [21], [22], [25], [27]. Girvan and Newman (2002) proposed an algorithm to generate community network using the concept of modularity to create the community in a complex network and their algorithm has nonlinear time-complexity with respect to the number of edges in the constructed network [20]. Tasgin and Bingol (2006) proposed a new community detection algorithm based on genetic algorithm (GA) which has linear time-complexity [19], [29]. For the application of complex network in epidemiology, many researchers have proposed epidemic network models in a discrete space domain [16], [18], [33], [36]. Barthélemy et al. (2005) presented SIS and SIR dynamical patterns and analyzed the time evolution of epidemic outbreaks in complex networks [2]. Likewise, Suna and Gao (2007) investigated SIR dynamic behavior of epidemics on scale-free networks with community structure [31]. Dangerfield et al. (2008) studied a Susceptible-Infectious-Susceptible (SIS) epidemic model by integrating stochasticity and network structure [4]. Jumpen et al. (2011) proposed SEIQR-SIS epidemic network model and its stability [10], [11]. Nodes in their network were classified into hubs and people, and they found that hubs had significant effect on the disease

transmission. None of these extensions had considered the role of people structure to the disease transmission on the network. The aforementioned researches show that the development of proper epidemic network model is still worldwide active.

In this paper, we propose a social network with community of the study of the SEIQR disease transmission. To mimic the real structure of human society, Genetic Algorithm (GA) is used to detect a suitable community of each person in the network. The rest of the paper ia organized as followed. Section II presents an implemented GA-network algorithm using a community detection method. In section III, the SEIQR network model is introduced and used in conjunction with the constructed GA-network to simulate the disease transmission. Then, some numerical results are given in section IV, followed by conclusion in section V.

## II. GA-network Algorithm

To simulate the social activities, we have developed a network with community structure. A partition of communities in the network is based on the GA. Our algorithm follows closely to the GA proposed by Tasgin and Bingolas [19]. The resulting network is then used to study the SEIQR disease spread. Details of our network construction are in **Step1** and **Step2**. GA algorithm for a community detection is in **Step3**.

Our network consists of people nodes together with connecting links inside a unit square. Let $N$, $C_n$ and $M$ be total numbers of people nodes, chromosomes and possible communities, respectively.

**Step1** Randomly select locations of $N$ people nodes in a unit square by setting the lower bound of shortest distance among people nodes.

**Step2** Search for neighbors of each people node inside the contact radius and generate connecting links between the node and its neighbors.

**Step3** Partition the network into communities based on the connecting links using GA as follows:

(i) **Initialization**: There are $C_n$ chromosomes and each chromosome contains $a_N$ cells stored the community identification (CommID) of $N$ people nodes. For every chromosome, a number of CommIDs are randomly selected. Each CommID is assigned to a random node (cell) together with its neighbors as displayed in Fig. 1.

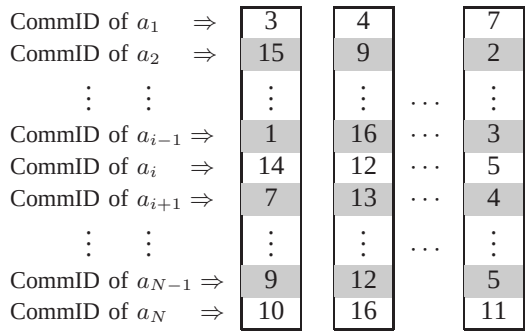



Fig. 1. Initialization of $C_n$ chromosomes

(ii) **Selection**: We select two chromosomes called as a source chromosome ($C_S$) and a destination chromosome ($C_D$) as follows:

(a) Calculate modularity $(\mathcal{H})$ of the chromosomes by the expression:

$$\mathcal{H} = \sum_g \left[ \frac{e_g}{e} - \left( \frac{\deg}{2e} \right)^2 \right]$$

where $e_g$ denotes the number of edges in community $g$, $\deg$ denotes the sum of degrees of each vertices of community $g$ and $e$ is the total number of edges in the network.

(b) Sort the chromosomes according to their modularity in descending order.

(c) Select two chromosomes. Number of their selections is based on their modularity. A source chromosome $C_S$ is the one with the higher modularity.

(iii) **Crossover**: We randomly select CommID named as ID-select. We then check the values in any cells in $C_S$. If the value equals ID-select, then the values in the corresponding cells $C_D$of $C_S$ are changed to be the same ID-select as shown in Fig. 2.

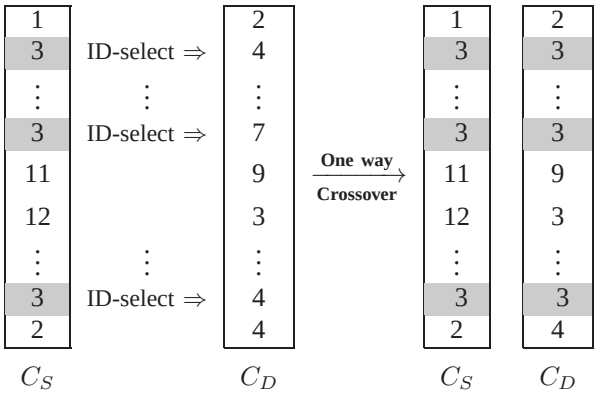



Fig. 2. One way crossover from source chromosome $C_S$ to destination chromosome $C_D$

(iv) **Mutation**: A node may be put into a random community in the network.

(v) **Clean-up**: For a node having a high community variance (CV), its CommID and its neighbors' CommID are changed to be the CommID of most neighbors.

(a) Randomly select a node for each chromosome. Calculate its community variance by the expression:

$$CV(i) = \frac{\sum_{(i,j)\in\mathcal{E}} f(i,j)}{\deg(i)}$$

where $\mathcal{E}$ is the set of all edges in the network and

$$f(i,j) = \begin{cases} 1, & \text{CommID}(i) \neq \text{CommID}(j) \\ 0, & \text{otherwise.} \end{cases}$$

(b) If $CV(i)$ is higher than the threshold value, then CommIDs of that node and its neighbors are changed to the one that occurs most often among them.

(vi) **Repetition**: The process is repeated from (ii) to (v) until the value of network modularity is between 0.3 and 0.7 exhibiting a good community structure. The expression $\mathcal{H} = 0$ if a community has no within-community edges whilst $\mathcal{H} = 1$ when all nodes are put into a single community [19], [29], [32].

Finally, the chromosome with the highest modularity is chosen to be the community structure of the social network which is incorporated to study the SEIQR model in the subsequent section.

## III. The SEIQR-network Model and Algorithm

The SEIQR-network model has been developed to study the spread of the infectious disease based on SEIQR dynamics and multi-group structure in the complex network [12]. This section illustrates the SEIQR-network model and its threshold parameter calculation, and guides an algorithm for the simulation of SEIQR-network model on GA-network presented in section II.

### A. SEIQR-network Model

In the network, there are $M$ communities. Each of which has $N_i$ people nodes for $i = 1, 2, \ldots M$. As can be seen in Fig. 3 considering the $i^{\text{th}}$ community, the total population size $N_i(t)$ is divided into five distinct epidemiological subclasses of individuals which are susceptible $S_i(t)$, exposed $E_i(t)$, infectious $I_i(t)$, quarantined $Q_i(t)$ and recovered $R_i(t)$. Fig. 3 can be inferentially interpreted

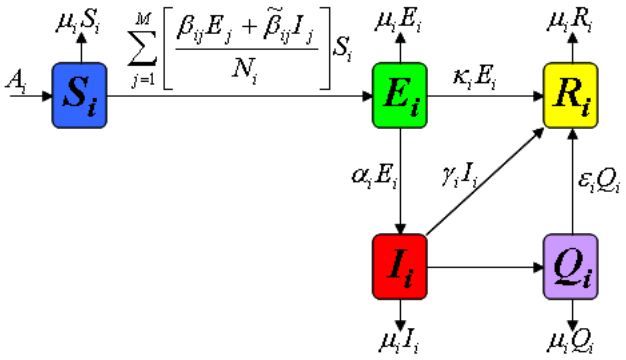



Fig. 3. Progression diagram for the SEIQR disease transmission in the $i^{\text{th}}$ community.

to the system of ordinary differential equations as follows.

$$\begin{aligned}
\frac{dE_i}{dt} &= \sum_{j=1}^{M}\left[\frac{\beta_{ij}E_j+\tilde{\beta}_{ij}I_j}{N_i}\right]S_i - (\alpha_i+\kappa_i+\mu_i)E_i,\\
\frac{dI_i}{dt} &= \alpha_i E_i - (\gamma_i+\delta_i+\mu_i)I_i,\\
\frac{dQ_i}{dt} &= \delta_i I_i - (\varepsilon_i+\mu_i)Q_i,\\
\frac{dR_i}{dt} &= \kappa_i E_i + \gamma_i I_i + \varepsilon_i Q_i - \mu_i R_i.\\
\frac{dS_i}{dt} &= A_i - \sum_{j=1}^{M}\left[\frac{\beta_{ij}E_j+\tilde{\beta}_{ij}I_j}{N_i}\right]S_i - \mu_i S_i,
\end{aligned} \tag{1}$$

The outbreak of the disease on the complex network occurs when infectious and exposed individuals transmit the disease to its susceptible neighbors via the edges with an infection rate $\beta$. It has been recognized that the transmission probability in the same community is higher than that between communities. Thus, the transmission probability within a community is assigned to be higher than the transmission probability between communities. After the susceptible individuals receive an amount of virus, their status become exposed. For the disease having incubation period of $1/\alpha$ days and sick period of $1/\gamma$ days, an exposed individual becomes an infectious individual at the transfer rate $\alpha$ and an infectious individual recovers at the transfer rate $\gamma$. From then, some infectious individuals are quarantined with the rate $\delta$ in order to reduce an infection. Finally, infectious and quarantined individuals will recover when they reach the sick period.

From the system (1), certain assumptions for the model are described as follows:

(i) Indices $i, j = 1, 2, \ldots, M$ present the $i^{\text{th}}$ and $j^{\text{th}}$ communities of the sub-classes $S$, $E$, $I$, $Q$ and $R$.

(ii) All parameters are non-negative constants defined as follows:

(a) $\alpha_i$ is the rate at which an exposed individual $E_i(t)$ becomes infected individual $I_i(t)$;

(b) $\delta_i$ is the rate that individual $I_i(t)$ moves to the quarantined individual $Q_i(t)$;

(c) $\kappa_i, \gamma_i$ and $\varepsilon_i$ are the rates at which individuals in the $E_i(t)$, $I_i(t)$ and $Q_i(t)$ classes change their status to be in $R_i(t)$.

(iii) The model (1) is considered with a recruitment-death demographic structure such that

$$\sum_{i=1}^{M} \frac{dN_i}{dt} = \sum_{i=1}^{M} (A_i - \mu_i N_i),$$

$A_i$ is a constant recruitment in the $i^{\text{th}}$ community such that $A_i = b_i S_i(0)$, where $b_i$ and $\mu_i$ are the natural death rate.

(iv) $\beta_{ij}$ and $\tilde{\beta}_{ij}$ are the probabilities of catching the disease per contact to the infectious or exposed person, separately considered in two cases:

$$\beta_{ij} = \begin{cases} \beta_{\text{high}} & \text{when } i = j \text{ (intra-community)} \\ \beta_{\text{low}} & \text{when } i \neq j \text{ (inter-community)} \end{cases}$$

and $\tilde{\beta_{ij}}$ is defined as the same sense as $\beta_{ij}$.

### B. Threshold Parameter

Throughout this subsection, $i$ varies from 1 to $M$. To shorten our following computations, we denote

$$K_i = \alpha_i + \kappa_i + \mu_i \quad \text{and} \quad L_i = \gamma_i + \delta_i + \mu_i$$

for each $i$. If we introduce the state vector $X$ by

$$\begin{aligned} X &= (X_1, X_2, \ldots, X_{5M}) \\ &= (E_1, \ldots, E_M, I_1, \ldots, I_M, \\ &\quad Q_1, \ldots, Q_M, R_1, \ldots, R_M, S_1, \ldots, S_M), \end{aligned}$$

the first $2M$ equations after the arrangement of the system (1) according to the state vector $X$ correspond to the infectious compartments, namely, $E_i$ and $I_i$ whilst the last $3M$ equations correspond to the compartment $Q_i$, $R_i$ and $S_i$, respectively. The disease free equilibrium after the arrangement of the system (1) is in the form of

$$X_0 = (0, 0, \ldots, 0, A_1/\mu_1, A_2/\mu_2, \ldots, A_M/\mu_M),$$

and thus the solutions of the system stay in the region $[0, \infty)^{5M}$ given by

$$\begin{aligned} D = \{\, X \mid X_i + X_{M+i} + X_{2M+i} + X_{3M+i} + X_{4M+i} \\ = E_i + I_i + Q_i + R_i + S_i \leq A_i/\mu_i \,\}. \end{aligned}$$

Together with nonnegative initial condition $X(0) = X_0$, we can apply the next generation method to calculate the basic reproductive number of the system (1). We rewrite the system (1) as

$$\dot{X} = \mathcal{F}(X) - \mathcal{V}(X)$$

where

$$\mathcal{F} = \begin{bmatrix} \mathcal{F}_1 \\ \vdots \\ \mathcal{F}_k \\ \vdots \\ \mathcal{F}_{5M} \end{bmatrix}, \quad \mathcal{V} = \begin{bmatrix} \mathcal{V}_1 \\ \vdots \\ \mathcal{V}_k \\ \vdots \\ \mathcal{V}_{5M} \end{bmatrix}.$$

The function $\mathcal{F}_k(X)$, the rate of appearance of the next infection in $k^{\text{th}}$ compartment, is set to be

$$\mathcal{F}_k = \begin{cases} \displaystyle\sum_{j=1}^{M} \left[ \frac{\beta_{kj} E_j + \tilde{\beta}_{kj} I_j}{N_k} \right] S_k, & 1 \leq k \leq M, \\ \alpha_{k-M} E_{k-M}, & M+1 \leq k \leq 2M, \\ 0, & 2M+1 \leq k \leq 5M. \end{cases}$$

The function $\mathcal{V}_k(X)$ is the difference in the transfer rate of individual into and out of the $k^{\text{th}}$ compartment. It is in the form

$$\mathcal{V}_k = \begin{cases} K_k E_k, & 1 \leq k \leq M, \\ L_{k-M} I_k, & M+1 \leq k \leq 2M, \\ 0, & 2M+1 \leq k \leq 5M. \end{cases}$$

If we denote Jacobian matrices $F$ and $V$ as

$$F = \left[ \frac{\partial \mathcal{F}_j}{\partial X_k} \right] (X_0) \quad \text{and} \quad V = \left[ \frac{\partial \mathcal{V}_j}{\partial X_k} \right] (X_0),$$

with $1 \leq j, k \leq 2M$, then $F$ and $V$ can be expressed as

$$F = \left[ \begin{array}{c|c} F_{1,1} & F_{1,2} \\ \hline F_{2,1} & F_{2,2} \end{array} \right], \quad V = \left[ \begin{array}{c|c} V_{1,1} & V_{1,2} \\ \hline V_{2,1} & V_{2,2} \end{array} \right]$$

where the submatrices of $F$ and $V$ are defined as follows:

$$F_{1,1} = \begin{bmatrix} \frac{\beta_{11} A_1}{\mu_1 N_1} & \frac{\beta_{12} A_1}{\mu_1 N_1} & \cdots & \frac{\beta_{1M} A_1}{\mu_1 N_1} \\ \frac{\beta_{21} A_2}{\mu_2 N_2} & \frac{\beta_{22} A_2}{\mu_2 N_2} & \cdots & \frac{\beta_{2M} A_2}{\mu_2 N_2} \\ \vdots & \vdots & \ddots & \vdots \\ \frac{\beta_{M1} A_M}{\mu_M N_M} & \frac{\beta_{M2} A_M}{\mu_M N_M} & \cdots & \frac{\beta_{MM} A_M}{\mu_M N_M} \end{bmatrix},$$

$$F_{1,2} = \begin{bmatrix} \frac{\tilde{\beta}_{11} A_1}{\mu_1 N_1} & \frac{\tilde{\beta}_{11} A_1}{\mu_1 N_1} & \cdots & \frac{\tilde{\beta}_{1M} A_1}{\mu_1 N_1} \\ \frac{\tilde{\beta}_{21} A_2}{\mu_2 N_2} & \frac{\tilde{\beta}_{22} A_2}{\mu_2 N_2} & \cdots & \frac{\tilde{\beta}_{2M} A_2}{\mu_2 N_2} \\ \vdots & \vdots & \ddots & \vdots \\ \frac{\tilde{\beta}_{M1} A_M}{\mu_M N_M} & \frac{\tilde{\beta}_{M2} A_M}{\mu_M N_M} & \cdots & \frac{\tilde{\beta}_{MM} A_M}{\mu_M N_M} \end{bmatrix},$$

$$F_{2,1} = \begin{bmatrix} \alpha_1 & 0 & \cdots & 0 \\ 0 & \alpha_2 & \cdots & 0 \\ \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \ldots & \alpha_M \end{bmatrix},$$

$$V_{1,1} = \begin{bmatrix} K_1 & 0 & \cdots & 0 \\ 0 & K_2 & \cdots & 0 \\ \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \cdots & K_M \end{bmatrix},$$

$$V_{2,2} = \begin{bmatrix} L_1 & 0 & \cdots & 0 \\ 0 & L_2 & \cdots & 0 \\ \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \cdots & L_M \end{bmatrix},$$

$$F_{2,2} = V_{1,2} = V_{2,1} = \begin{bmatrix} 0 & 0 & \cdots & 0 \\ 0 & 0 & \cdots & 0 \\ \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \ldots & 0 \end{bmatrix}.$$

The basic reproductive number can be determined by

$$\mathcal{R}_0 = \rho(FV^{-1})$$

where $\rho(J)$ is the spectral radius of a matrix $J$ [24]. If we further assume that $FV^{-1}$ has rank of $M+1$, then the spectral radius $\rho(FV^{-1})$ is of the form

$$\frac{1}{2}\sum_{i=1}^{M}\beta_{ii}B_i + \frac{1}{2}\sqrt{\left(\sum_{i=1}^{M}\beta_{ii}B_i\right)^2 + 4\sum_{i=1}^{M}\frac{\alpha_i\tilde{\beta}_{ii}B_i}{L_i}}$$

where

$$B_i = \frac{A_i}{K_i\mu_i N_i}.$$

Literally, the basic reproductive number can be used to consider the epidemiological situation, i.e., the disease will disappear when $\mathcal{R}_0 < 1$ whilst $\mathcal{R}_0 > 1$ informs that the disease will spread [8], [37].

### C. Algorithm

The system (1) is now considered in a social complex network. We assume that time step is one day and natural death rate is zeroes. Approximating the first derivative of each equation in the system (1) by forward finite different:

$$\frac{df}{dt} \approx \frac{f_{t+\triangle t} - f_t}{\triangle t}.$$

The system (1) becomes

$$\begin{aligned} E_{i,t+1} &= E_{i,t} + \left[\sum_{j=1}^{M}\frac{\beta_{ij}E_{j,t} + \tilde{\beta}_{ij}I_{j,t}}{N_i}\right]S_{i,t}\triangle t \\ &\quad - (\alpha_i + \kappa_i)E_{i,t}\triangle t, \\ I_{i,t+1} &= I_{i,t} + [\alpha_i E_{i,t} - (\gamma_i + \delta_i)I_{i,t}]\triangle t, \\ Q_{i,t+1} &= Q_{i,t} + [\delta_i I_{i,t} - \varepsilon_i Q_{i,t}]\triangle t, \\ S_{i,t+1} &= S_{i,t} - \left[\sum_{j=1}^{M}\frac{\beta_{ij}E_{j,t} - \tilde{\beta}_{ij}I_{j,t}}{N_i}S_{i,t}\right]\triangle t, \\ R_{i,t+1} &= R_{i,t} + [\kappa_i E_{i,t} + \gamma_i I_{i,t} + \varepsilon_i Q_{i,t}]\triangle t. \end{aligned}$$

Let $\tau$ be the maximum time for the simulation, and $\triangle t$ be the time step. The spread of the disease based on SEIQR dynamics is simulated by the following algorithm.

**Step1** At initial time step $t = 0$ day, there are $N$ people nodes including a few infected nodes say $\eta$ in the infectious class ($I$-class) and $N-\eta$ people nodes in the susceptible class ($S$-class).

**Step2** Set $t = t + \triangle t$.

**Step3** Updating stage of people nodes based on their neighbor status:

(i) The $S$-class nodes move to the $E$-class with the transmission probability $\beta_{\text{high}}$ if they are in the same community with $I$-class nodes and $\beta_{\text{low}}$ when they are in different communities;

(ii) The $E$-class nodes move to the $I$-class with the transfer rate $\alpha$;
The $E$-class nodes move to the $R$-class with the transfer rate $\kappa$;

(iii) The $I$-class nodes move to the $R$-class with the transfer rate $\gamma$;

(iv) The $I$-class nodes move to the $Q$-class with the transfer rate $\delta$;

(v) The $Q$-class nodes move to the $R$-class with the transfer rate $\varepsilon$.

**Step4** Repeating **Steps2** and **Step3** until the $\tau$ reaches or there is no infectious and quarantined node in the network.

## IV. Numerical results and discussion

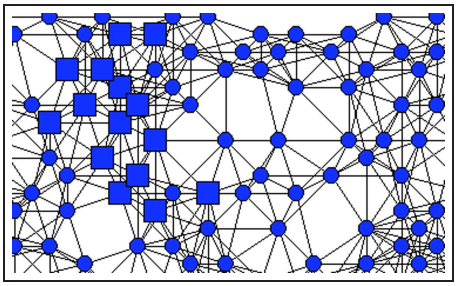
An experiment is brought up for studying the spread of SEIQR infections in the social complex network having 1,000 people nodes classified into 16 communities ($M = 16$) by GA-network algorithm. To understand how the disease spreads on the real-world population network, we randomly set five infectious nodes at the beginning and then simulate the SEIQR epidemic on the network over time using the SEIQR network algorithm presented in section III. Values of parameters used in this simulation are expressed in TABLE I.

TABLE I
VALUES OF PARAMETERS USED IN FIG. 4–8

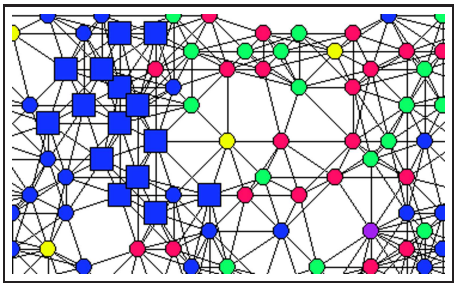
| Parameters | Biological Description | Value |
|---|---|---|
| $\mathcal{C}_r$ | Neighborhood contact radius | 0.060 |
| $\alpha$ | Transfer rate at which $E$ becomes $I$ | 0.250 |
| $\beta$ | Transmission rate between people nodes | 0.004 |
| $\gamma$ | Transfer rate at which $I$ and $Q$ become $R$ | 0.167 |
| $\delta$ | Quarantine rate | 0.100 |
| $\kappa$ | Transfer rate at which $E$ becomes $R$ | 0.020 |
| $\varepsilon$ | Transfer rate at which $Q$ becomes $R$ | 1.000 |

In Fig. 4, the topology of our network are presented at four different times in which square nodes represent

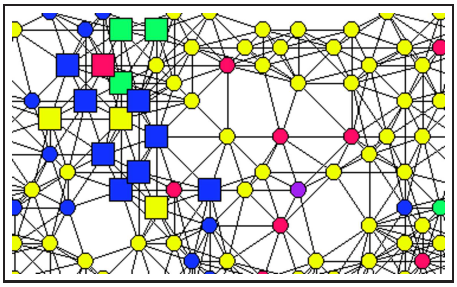
people in the largest community and circle nodes are in other communities.



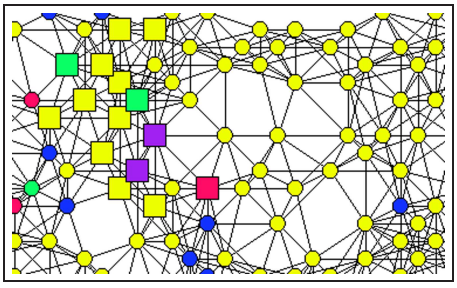
(a) $t = 0$ day



(b) $t = 20$ days



(c) $t = 30$ days



(d) $t = 40$ days

Fig. 4. The topology of a portion of our network at four different times.

Effect of transmission rate is considered. In Fig. 6, we choose three different transmission rates of 0.002, 0.004 and 0.006. The results indicate that an increase of transmission rate from 0.002 to 0.006 increases the peak of infection proportion from 0.038 to 0.401.

The influence of quarantine rate is also investigated. The result shows a faster spread of the disease for a lower quarantine rate as shown in Fig. 7. That is, a decrease of quarantine rate from 0.02 to 0.005 increases the peak

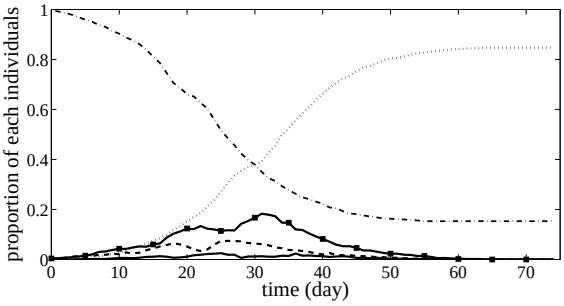
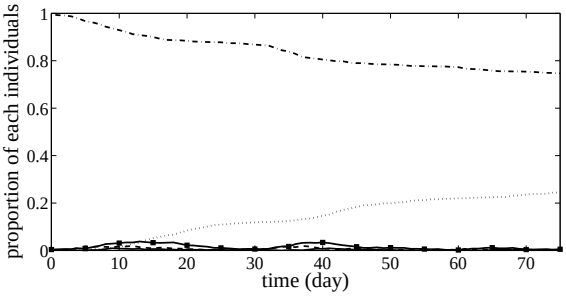
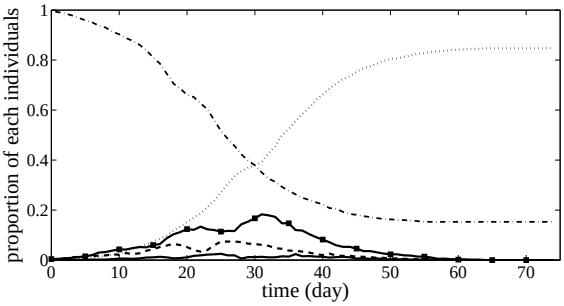


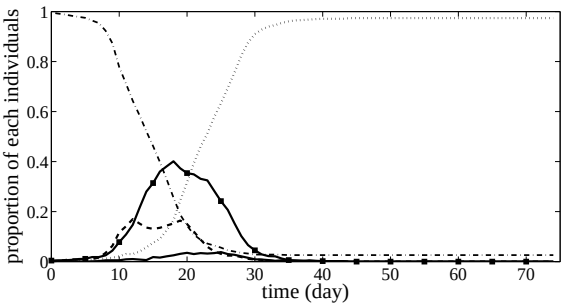

Fig. 5. Disease transmission obtained from the network model with $\mathcal{H} = 0.7$ where $S$, $E$, $I$, $Q$ and $R$ are respectively presented by dash-dot line, dashed line, solid-square line, solid line and dotted line.




(a) Transmission rate $\beta = 0.002$



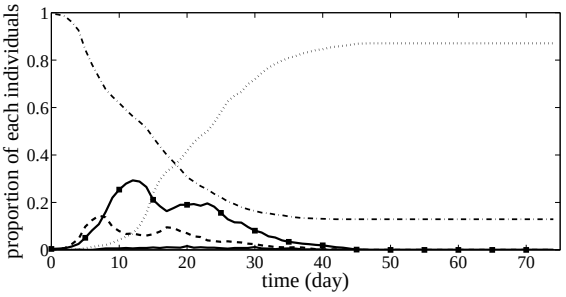

(b) Transmission rate $\beta = 0.004$



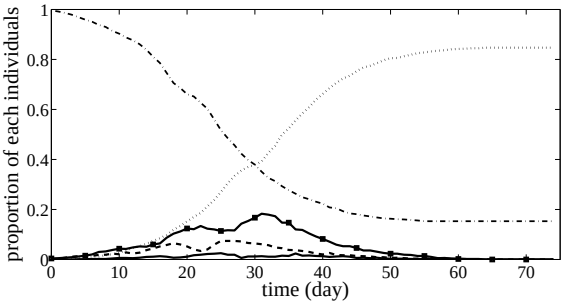

(c) Transmission rate $\beta = 0.006$

Fig. 6. Effect of three different transmission rates of the network on the SEIQR disease transmission where $S$, $E$, $I$, $Q$ and $R$ are respectively presented by dash-dot line, dashed line, solid-square line, solid line and dotted line: (a) $\beta = 0.002$; (b) $\beta = 0.004$ and (c) $\beta = 0.006$.

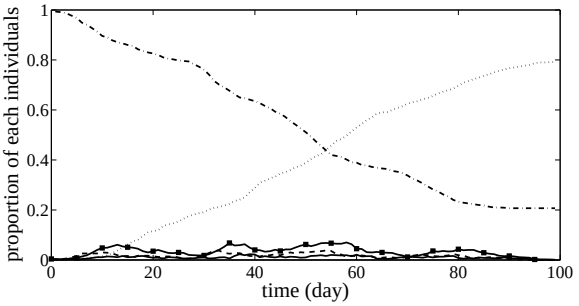
of infection proportion from 0.070 to 0.293. Decreasing quarantine rate yields a faster spread of the disease, a lower peak number of infections and a shorter period of the transmission.




(a) Quarantine rate $\delta = 0.05$




(b) Quarantine rate $\delta = 0.1$




(c) Quarantine rate $\delta = 0.2$

Fig. 7. Effect of three different quarantine rates of the network on the SEIQR disease transmission where $S$, $E$, $I$, $Q$ and $R$ are respectively presented by dash-dot line, dashed line, solid-square line, solid line and dotted line: (a) $\delta = 0.05$; (b) $\delta = 0.1$ and (c) $\delta = 0.2$.

## V. Conclusion

The appropriate structure of social complex network has been organized by the community detection method using GA-network. Then, it is cooperated with the SEIQR-Network model to study the spreading behavior of an epidemic. The simulation results point that the network modularity, the transmission rate and the quarantine rate have significant impacts on the disease transmission. The higher transmission rate and lower quarantine rate directly raise the rate of infection. The higher quarantine rate leads to the smaller number of infection. Therefore, the proposed network model can describe the essential feature of disease transmission.

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