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Epidemic in networked population with recurrent mobility pattern

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A B S T R A C T
The novel Coronavirus (COVID-19) has caused a global crisis and many governments have taken social measures, such as home quarantine and maintaining social distance. Many recent studies show that network structure and human mobility greatly influence the dynamics of epidemic spreading. In this paper, we utilize a discrete-time Markov chain approach and propose an epidemic model to describe virus propagation in the heterogeneous graph, which is used to represent individuals with intra social connections and mobility between individuals and common locations. There are two types of nodes, individuals and public places, and disease can spread by social contacts among individuals and people gathering in common areas. We give theoretical results about epidemic threshold and influence of isolation factor. Several numerical simulations are performed and experimental results further demonstrate the correctness of proposed model. Non-monotonic relationship between mobility possibility and epidemic threshold and differences between Erdős-Rényi and power-law social connections are revealed. In summary, our proposed approach and findings are helpful to analyse and prevent the epidemic spreading in networked population with recurrent mobility pattern.

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1. Introduction
Recently, the novel Coronavirus (COVID-19) has caused a global crisis and more than 4 million people of more than 200 countries have been infected up to now (14th May, 2020) [1]. The primary measures taken by many governments are home quarantine and maintaining social distance, with the aim to break transmission of this infective virus and halt the spread of pandemic. Although most public places are locked down, supermarkets and drugstores which are essential for daily life remain open. Different from commonly used homogeneous mixing approaches [2,3], we give an analysis of epidemic spreading in population following a structured network with recurrent mobility pattern in this work.

The influences of network structure and human mobility on epidemic spreading have received lots of attention in recent years. On one hand, homogeneous mixing assumption among individuals is often invalid and variations of social interaction network bring large differences in the propagation process of virus [4–10]. On the other hand, human mobility also greatly affects the peak and duration of epidemic outbreak [11–15], and recurrent patterns between people and their familiar locations (e.g. workplace, supermarket) often dominate the behaviour of human mobility [16,17]. However, the interactions of individuals in public places provide another route of virus transmission, which makes the analyse of epidemic spreading much more difficult.

One widely used approach to analyse epidemic spreading in complex networks is metapopulation model, which divides the whole population into several geographical structured parts [13,18], and contacts among individuals in the same subpopulation are assumed to be well-mixed. Though some challenges remain [19], a useful strategy of analysing the impact of human mobility behaviour on epidemic spreading is to integrate metapopulation model with reaction-diffusion process [20,21]. Each subpopulation can be formulated as a node and edges between different metapopulations indicate the mobility probabilities of individuals. Reaction-diffusion process has been widely studied in physics, for epidemic spreading, diffusion often refers to individual movements between different places and reaction indicates the contagion process within each place after human behaviour. In addition to the way of using a unipartite network to model metapopulations for analysis of epidemic spreading with recurrent mobility pattern, heterogeneous networks are adopted recently [22,23]. All these methods take the assumption individuals in the same sub-
population will contact with each other in a well-mixed fashion, however, each individual only interacts with his neighbors in real social networks.

In this paper, we utilize a heterogeneous network to represent the social connections among individuals and recurrent human mobility pattern between population and locations. There are two types of connections: one is the edge between any two individuals, and the other is connection between individual and common areas. The social network may follow different network structures, such as Erdős-Rényi or power-law networks. In order to model the dynamics of virus spreading, we apply the discrete-time Markov chain method in the context of susceptible–infected–susceptible (SIS) infection process like [8,20,23,24]. With a mobility possibility, each agent will choose to get into public places which he connects to or stay in social network. Since there are no fixed individuals and connections among them, we assume people gathering in a public area will have contacts with all other individuals. The agent who chooses to stay in social network will have contacts with his remaining neighbors, and he may get infected if and only if he has some contagious neighbors who do not go outside at that time step. In this paper, detailed formulation of epidemic model in this kind of networked population with recurrent mobility pattern is introduced, and we also give theoretical results of the epidemic threshold. Besides, the decay of epidemic infection below threshold and impact of isolation factor are presented. As far as we know, this is the first attempt to analyse epidemic spreading in networked population with human recurrent mobility by using social contacts network among individuals.

The remaining of this paper is organized as follows. In Section 2, we give the formulation of epidemic model for virus spreading in networked population with recurrent mobility pattern, along with theoretical results of epidemic threshold. The analysis of epidemic threshold from the view of non-linear dynamical system (NLDS) and its decay property are introduced in Section 3. Experimental results on two types of social networks, Erdős-Rényi and power-law networks are showed in Section 4, and we conclude this work with a summary in Section 5.

2. Epidemic model for networked population with recurrent mobility pattern

The heterogeneous graph of networked population in our paper consists of two different parts. One is composed of \( M \) individuals with specific social network structure, and the other is \( N \) public places. Different from previous works [20,21,23] which only consider connections between metapopulations and common areas, we discard the well-mixed assumption in metapopulation and suppose there is a contact network among individuals. We formulate an epidemic model of virus propagating in networked population with recurrent mobility pattern between individuals and public areas. The social connections are defined by a \( M \times N \) matrix \( A \), where \( A_{ij} = 1 \) if individual \( i \) has a contact with agent \( j \). The edges between individuals and locations are dependent on a \( M \times N \) matrix \( B \), where \( B_{ij} = 1 \) if agent \( i \) will visit place \( j \) if he goes out, and 0 otherwise. Here, we assume edges are unweighted, and undirected, hence \( A \) is a symmetric matrix.

In order to simulate the recurrent mobility pattern between individuals and public places, at each time step, individuals will go to all public places which they connect to with a possibility of \( p \). After movements of individuals, virus propagates in both remaining networked population and common areas independently. We also force individuals in common places to return back to social network at the end of step, for the purpose of ensuring recurrent mobility patterns of individuals. Fig. 1 gives an example of epidemic spreading at time \( t \) when some individuals get into public places with possibility \( p \). It should be noted that, the edges of both \( A \) and \( B \) for a specific heterogeneous network remain unchanged during the whole simulation. However, at each time step, since some individuals will go outside, the remaining \( A \) and resulting infection process based on \( B \) in common areas might be different. For dynamics of epidemic spreading among individuals, we adopt the popular SIS model. There are two states, susceptible and infective, for each individual. We assume the homogeneous spreading of virus where the infection rate is \( \beta \) and recovery rate is \( \mu \). A susceptible individual will become infected by a possibility of \( \beta \) when contacting with a contagious agent, and individuals who got infected at previous time steps will recover with possibility \( \mu \) and become susceptible again at each time step. The states of population at \( t + 1 \) are only dependent on those at \( t \), thus a discrete-time Markov chain can be taken to model the dynamics of epidemic spreading. The possibility individual \( i \) is infective at the beginning of time \( t \) is denoted as \( P_{\text{SIS}} \), and susceptible \( P_{\text{SIS}} \) where \( P_{\text{SIS}} = 1 - P_{\text{SIS}} \) for SIS model. The evolution of \( P_{\text{SIS}} \) can be formulated as

\[
P_{\text{SIS}, t+1} = (1 - \mu) P_{\text{SIS}, t} + \mu P_{\text{SIS}} \Pi_i(t)
\]

where the left part is possibility when \( i \) has already got infected and remains infective, and right is possibility that susceptible individual \( i \) becomes infective at time \( t \) where \( \Pi_i(t) \) is the infection possibility. Different from formulation in traditional networked populations [5,6,22,24,25]. \( \Pi_i(t) \) consists of two different components

\[
\Pi_i(t) = (1 - p) D_i(t) + p C_i(t)
\]

where \( p \) is the mobility possibility, \( D_i(t) \), \( C_i(t) \) are infection possibilities when agent \( i \) stays in social contact network or gets into public places.

In this paper, we define the possibility of a susceptible individual getting infected when interacting with \( k \) contagious people as

\[
P(k) = 1 - (1 - \beta)^k
\]

when each individual goes outside with possibility \( p \), the infection possibility \( D_i(t) \) turns into

\[
D_i(t) = 1 - (1 - \beta)(1 - p) \sum_{j \text{ neighbors of } i} A_{ij} P_{\text{SIS}}
\]

\[
= 1 - (1 - \beta)(1 - p) \sum_{j \text{ neighbors of } i} A_{ij} P_{\text{SIS}}
\]

where we use \( A_{ij} = 1 \) for neighbors of \( i \) only in the contact network \( A \) and assume probabilities \( P_{\text{SIS}} \) are independent of each other. For individuals in common areas, we take the assumption of well-mixed fashion used in [3,13,20,21,23], which means every one will have a contact with each other in the same place. Therefore,
we can get
\[ C_i(t) = 1 - \prod_{j \text{ neighbor of } i} (1 - \beta) P_{ij} e^{\sum_{j} b_{ij} P_{ij}} \]
\[ = 1 - (1 - \beta)^\sum_{j} b_{ij} P_{ij} \tag{3} \]
where \( j \) refers to a public place and \( B_{ij} \) takes the value of \( 1 \) for places \( j \) which individual \( i \) connects to. Combining Eq. 2 and Eq. 3 into \( \Pi_i(t) \), Eq. 1 can be rewritten as

\[ P_{i,t+1} = (1 - \mu) P_{i,t} + P_{S,i} \left( (1 - (1 - \beta)^{1-p}) \sum_{j} A_{ij} P_{ij} \right) \]
\[ + \mu (1 - (1 - \beta)^{1-p}) \sum_{j} b_{ij} P_{ij} \]
\[ \tag{4} \]

When whole population is near the critical onset of epidemic outbreak, the infective possibility for each individual and corresponding infection probability are negligible which means \( P_{i,t+1} = P_{i,t} ≈ 1 \) and \( \beta ≈ 1 \). By using approximations \((1 - \beta)^n ≈ 1 - n\beta\), and neglecting high-order terms \( O(\beta^2) \), we can reduce Eq. 4 into

\[ \mu P_{i,t} = \beta \left( (1 - p)^2 A + p^2 BB^T \right) P_{i,t} \]
\[ \text{if we use } P_i = [P_{1,i}, P_{2,i}, \ldots, P_{M,i}]^T \text{ to indicate infected possibilities of all } M \text{ individuals, Eq. 5 can be easily rewritten as} \]

\[ \mu P_i = \beta \left( (1 - p)^2 A + p^2 BB^T \right) P_i \]
\[ \text{hence, the infection threshold of epidemic spreading can be obtained } [5,6] \]

\[ \beta_{\text{min}} = \frac{\mu}{\lambda_{\text{max}}((1 - p)^2 A + p^2 BB^T)} \]
\[ \text{where } \lambda_{\text{max}}(Q) \text{ is the largest eigenvalue of matrix } Q. \]

Similar to [23], we consider the impact of isolation factor \( \gamma \) where \( 0 \leq \gamma \leq 1 \) which constrains infected individuals from going into common places and they can only have contacts with social neighbors who do not go outside. Therefore, we can get

\[ \tilde{D}_i(t) = 1 - (1 - \beta)^{(1-p)} \sum_{j} A_{ij} P_{ij} \]
\[ \tilde{C}_i(t) = 1 - (1 - \beta)^{p \sum_{j} b_{ij} P_{ij}} \]
and the epidemic threshold with isolation factor is

\[ \beta_{\text{min}} = \frac{\mu}{\lambda_{\text{max}}((1 - p)^2 A + \gamma p^2 BB^T)} \]
\[ \tag{8} \]

3. Analysis of epidemic threshold

In the last section, we derive the epidemic threshold for networked population with recurrent mobility pattern of individuals. On one hand, when \( p \) equals 0, no individual goes out and virus spreads only through contact network \( A \), Eq. 7 turns into situations in [5,6,24,25]. On the other hand, when all individuals get into corresponding common areas which means \( p = 1 \), Eq. 7 becomes dynamics of epidemic spreading in bipartite networks as discussed in [3,22]. Here we give some theoretical analysis of epidemic threshold from the view of non-linear dynamical system (NLS) and introduce the exponential decay property of infective individuals when \( \beta \) is below epidemic threshold.

3.1. NLS epidemic threshold

For SIS model, the transitions of individual \( i \) can be described by

\[ P_{S,i,t+1} = \mu P_{i,t} + P_{S,i} \left( 1 - P_{i}(t) \right) \]
\[ P_{i,t+1} = (1 - \mu) P_{i,t} + P_{S,i}, P_{i}(t) \]
\[ \tag{9} \]

When no one gets infected in system, the equilibrium point is \( P_0 = [0, \ldots, 0]^T \) and \( P_1 = [1, \ldots, 1]^T \) where the numbers of 0 and 1 are both \( M \). According to [26], stability of system at equilibrium point is related to the Jacobian matrix

\[ J = \begin{bmatrix} \frac{\partial b_{i,j+1}}{\partial P_{i,1}} & \frac{\partial b_{i,j+1}}{\partial P_{i,1}} \\ \frac{\partial b_{i,j+1}}{\partial P_{i,1}} & \frac{\partial b_{i,j+1}}{\partial P_{i,1}} \end{bmatrix} \]
\[ \text{where we can easily get } J |_{P_0} \]
\[ J |_{P_0} = \begin{bmatrix} \mu I - Q & \mu \mu - Q \\ 0_{M,1} & \mu (1 - \mu) I + Q \end{bmatrix} \]
\[ \text{where } I \text{ is the identity matrix and } Q = \beta (1 - p)^2 A + \beta p^2 BB^T. \]

Therefore, the system is asymptotically stable at equilibrium point \( P \) if all the eigenvalues are less than 1 in absolute value. Assume the eigenvector is \([v_1, v_2]^T\), and corresponding eigenvalue is \( \lambda_{ij} \), we can get following equations

\[ v_1 + (\mu I - Q) v_2 = \lambda_{ij} v_1 \]
\[ ((1 - \mu) I + Q) v_2 = \lambda_{ij} v_2 \]
\[ \tag{10} \]

hence the eigenvalues of \( J \) are given by the eigenvalues of \( I \) (with \( v_2 = 0 \)) and \( (1 - \mu) I + Q \) (with \( v_2 \neq 0 \)).

Although when \( v_2 = 0, \lambda_{ij} = 1 \), it is related to \( \lambda_{ij} \) which does not cause instability when system is at equilibrium point \( P \). Then we just need \( \lambda_{\text{max}}((1 - \mu) I + Q) < 1 \) which means

\[ \beta \leq \frac{\mu}{\lambda_{\text{max}}((1 - p)^2 A + \gamma^2 p^2 BB^T)} \]
\[ \tag{11} \]

hence the obtained epidemic threshold is consistent with result of Eq. 7. By using the same strategy, we can also easily prove that epidemic threshold of SIR model is the same as SIS model.

3.2. Exponential decay of infection below threshold

Recall that we use approximations \((1 - \beta)^n ≈ 1 - n\beta\), if we take the high-order terms \( O(\beta^2) \) into account, the following results can be obtained

\[ (1 - \beta)^{(1-p)\sum_{j} A_{ij} P_{ij}} \geq 1 - \beta (1 - p) \sum_{j} A_{ij} P_{ij} \]
\[ (1 - \beta)^{p \sum_{j} b_{ij} P_{ij}} \geq 1 - \beta p \sum_{j} b_{ij} P_{ij} \]

By combining above results with Eq. 4, dynamic of infection possibility for individual \( i \) becomes

\[ P_{i,t+1} \leq (1 - \mu) P_{i,t} + \beta \left( (1 - p)^2 \sum_{j} A_{ij} P_{j,t} + p^2 \sum_{j} b_{ij} P_{j,t} \right) \]
\[ \text{and transitions for all people satisfy} \]
\[ P_{i,t+1} \leq WP_{i,t} \]
\[ \leq \sum_{j=1}^{M} \lambda_{ij}^{t+1} u_{j,0} u_{i,j}^{t+1} \tag{12} \]

where \( W = (1 - \mu)I + \beta (1 - p)^2 A + \beta p^2 BB^T \), \( \lambda_{ij}, u_{i,j} \) are the \( i \)-th largest eigenvalue and corresponding eigenvector of \( W \), and \( P_{i,0} \) is initial state of population. Since both \( A \) and \( BB^T \) are real symmetric matrix, all eigenvalues of \( W \) are real numbers. When \( \beta < \beta_{\text{min}} \) in Eq. 7, the equivalent form is

\[ \lambda_{\text{max}}((1 - \mu) I + \beta (1 - p)^2 A + \beta p^2 BB^T) < 1 \]
\[ \text{which indicates } \lambda_{ij} < 1 \text{ for every } i, \text{ therefore} \]
\[ P_{i,t+1} \leq \lambda_{ij}^{t+1} C \]
\[ \tag{13} \]

where \( C \) is a constant. Hence, the values of \( P_i \) will exponentially decrease over time if \( \beta \) is less than the epidemic threshold.
4. Numerical results

In order to validate the correctness of proposed model, we evaluate results from Monte Carlo simulations with theoretical predictions of Eq. 4. Two different types of network structures are used in the following experiments: Erdős-Rényi network and power-law network. We keep the edges between individuals and locations fixed, which means $B$ is unchanged while changing structure of social connections $A$ among individuals. In order to better analyse the impact of mobility possibility $p$ on different networks, total number of edges among individuals remains almost the same for Erdős-Rényi and power-law network. A comparison between simulation and theoretical results is showed in Fig. 2. The average infected ratio for the whole population is defined as $\eta = \frac{\sum_{j=1}^{M} p_{ij}}{M}$. We run 20 times for each mobility possibility $p$. For each simulation, we run 400 time steps and use average value of $\eta$ in the last 100 steps as the steady infection ratio.

As we can see, numerical solutions of proposed model have good correspondences with results of Monte Carlo simulations. For small mobility possibility $p = 2$, the epidemic threshold of power-law network is significantly smaller than that of Erdős-Rényi network. As explicated in [27], the heterogeneity of degree distribution in power-law network makes the largest eigenvalue larger than Erdős-Rényi network, and this makes virus more easily break out among individuals. When $p$ increases, the differences between Erdős-Rényi and power-law network both in epidemic threshold and steady infection ratio become less obvious. This can be explained by the fact that, when more individuals go outside, more and more spreads of epidemic take place in common areas while the connections $B$ between individuals and common locations are the same for both social contact networks.

In the next, we analyse the impact of recurrent mobility possibility $p$ on epidemic threshold $\beta_{\text{min}}$ with different numbers of contact edges. From Fig. 3, all curves show non-monotonic behaviours where epidemic threshold achieves its largest value $\beta_{\text{min}}^*$ for a specific value of $p^*$. Similar non-monotonic phenomena are also founded in [20,23]. Also, the largest epidemic thresholds in power-law network are smaller than those in Erdős-Rényi network. Besides, when total edges of social contacts ($E$) increase, $\beta_{\text{min}}$ decreases and $p^*$ increases for the synthetic networks.

After that, we investigate how isolation factor $\gamma$ influence epidemic spreading and the results are plotted in Fig. 4. When $\gamma$ is 1,
there are no isolations for infected individuals, and the curve behaves consistently with Fig. 3. For small isolation factors ($\gamma < 0.04$ in Fig. 4), the restriction of infected individuals makes $\beta_{\text{min}}$ monotonically increase with mobility possibility. Interestingly, in Erdős-Rényi network, all curves nearly intersect at one point and similar phenomenon is also founded in [23] which can be explained by the effective contact number of infected neighbors as discussed in [2,4], while curves in power-law network show more obvious dispersion due to the heterogeneity of degree distribution.

At last, we demonstrate the total number of infected individuals for different infection rates $\beta$ at different time steps in Fig. 5. As we can see, when $\beta < \beta_{\text{min}}$, the spread dies out exponentially, and becomes an epidemic otherwise. The experimental results have good agreements with outcomes of Eq. 13. We can also find that, for the same scale of threshold, such as $2\beta_{\text{min}}$ and $5\beta_{\text{min}}$, the number of steady infected individuals in power-law network is relatively smaller than Erdős-Rényi network. This indicates that although epidemic threshold in power-law network is usually smaller, there will be more infected population in Erdős-Rényi network due to the homogeneity of individual’s social contacts with their neighbors when infection rate increases by the same scale.

5. Conclusion

In this paper, we propose an epidemic model for networked population with recurrent mobility pattern. A heterogeneous network is used to represent the structure containing different connections. There are two types of edges, social connections among individuals and mobility connections between individuals and common areas. A detailed formulation by discrete-time Markov chain method to describe the dynamics of epidemic spreading is given, and we derive theoretical results about epidemic threshold. Several simulations on both Erdős-Rényi and power-law social networks are conducted and experimental results verify the correctness of our model and analysis. The non-monotonic relationship between epidemic threshold and mobility possibility indicates there is an optimal value which will make virus hard to spread. The influences of different values of isolation factor which restricts infected individuals from getting into common areas are analysed, and more obvious dispersion appears in power-law network because of heterogeneity of degree distribution. In addition, we demonstrate and prove the exponential decay of infection when epidemic rate is less than threshold. In summary, this pa-
per not only provides an approach to model epidemic spreading in networked population with recurrent mobility pattern, but offers a tool to analyse different network structures and social measures, such as restricting infected individuals.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

CRediT authorship contribution statement

Liang Feng: Writing - original draft, Investigation, Formal analysis, Methodology, Data curation, Conceptualization. Qianchuan Zhao: Conceptualization, Funding acquisition, Resources, Supervision, Writing - review & editing. Cangqi Zhou: Data curation, Methodology, Funding acquisition, Writing - review & editing.

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