Impacts of Information Propagation on Individual Migration Routes and Epidemic Spread Over Metapopulation Networks

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Impacts of information propagation on individual migration routes and epidemic spread over metapopulation networks

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Information propagation driven by the epidemic may cause the awareness of individuals to change their behavior, thus preventing themselves from being infected. For example, the aware individuals migrate away from areas with severe infection. In this paper, we study the coupling transmission of epidemic and information in metapopulation networks, and mainly explore how the change of individual migration behavior affects the epidemic spreading. Combined with the transition probability tree of individual states, we use Markov chain approach for theoretical analysis and derive the epidemic threshold. Through numerous Monte Carlo simulation, we verify the accuracy of Markov equations for the prediction of epidemic spreading. The results show that the role of information transmission in suppressing the epidemic in terms of the epidemic threshold and the infection scale is very limited. Further increase of information transmission rate beyond its critical value will no longer affect the epidemic. The initial population distribution is a fundamental factor in the epidemic dynamics, and in the case of heterogeneous distribution, an appropriate movement of individuals can delay the epidemic spread with a smaller threshold. In addition, topological homogeneity of individual migration route is beneficial for the epidemic control. This study analyzes the interaction between epidemic and information on the metapopulation network model, which can provide guidance for epidemic intervention in reality.

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I. INTRODUCTION

Epidemic spreading not only threatens human health and survival, but also hinders the development of society and economy, and has been extensively studied in different fields [1–3]. In earlier studies, various models were proposed under the assumption of uniformly mixing to simulate the epidemic dynamics, such as the classical susceptible-infected-susceptible model (SIS) [4], susceptible-infected-recovery model (SIR) [5], and so on [6, 7]. In recent years, the rise of the research in complex networks provides a new perspective for the study of epidemic spreading [8]. It is necessary to establish the corresponding network model for the study of different influencing factors of epidemics, e.g., the contact between people [9, 10], the immunity of particular individuals [11–13], the migration behavior [14–16], etc.

When epidemics spread in the population, disease-related information also propagates through social networks. Individuals who receive information become aware of the epidemic and take defensive measures, such as washing hands frequently, wearing masks [17, 18] and changing migration behaviors [19], to reduce the probability of being infected. Funk et al. studied the coupling transmission of epidemic and information in uniformly mixed population. They found that information dissemination significantly reduced the final infection ratio, but it had no effect on the epidemic threshold [20, 21]. Sahneh et al. proposed the Susceptible-Alert-Infected-Susceptible (SAIS) model and found that the increase of individual alertness helps to slow down the epidemic spreading [22, 23].

In reality, epidemic and information usually have different routes and forms of transmission. Based on this observation, Granell et al. [24, 25] utilized two-layers multiplex networks [8] to study the interaction between epidemic and information. Furthermore, Zhao et al. found that the stronger the degree correlation of nodes in multiplex networks, the smaller the epidemic threshold and the infected scale will be [26]. Considering the time-varying characteristics of physical contact network and communication network among individuals, Guo et al. [27] and Yang et al. [28] modeled the contact layer or the information layer as a temporal network [29]. They found that the time-varying network structure of information layer hinders the information dissemination and accelerates the epidemic spreading.

The above research on the coupling dynamics of epidemic and information is carried out on the contact networks where node represents the individual and edge represents the association between individuals [30]. Thus, it is not suitable for the study of spatial spread of epidemic due to the omission of individual migration. While individual migration is a crucial factor for epidemic spreading, since migration behavior may be affected by information transmission. For example, aware individuals may reduce the frequency of travel and change the path of mobility [31, 32], and so on. These changes in migration behavior will further influence the epidemic dynamics.

Metapopulation network model is a crucial tool to study spatially epidemic spreading considering individual migration [33–37]. In this framework, nodes represent patches and links denote transportation routes among patches. Inside each patch, there is a dynamic process of epidemic spreading or information spreading between individuals. Meloni et al. [31] and Wang et al. [32, 38] considered that aware individuals might migrate away from areas with severe infection. They found that such change in individual migration behavior contributes to the spread of the epidemic to healthy patches, thus promoting the global spread. Lima et al. [39] used the real data to establish the mobility matrix and the communication matrix, and concluded that the information campaigns might be effective countermeasures for disease control in metapopulation network, but the theoretical analysis of disease threshold was not given.

In order to further study the impact of changes in individual migration behavior caused by information dissemination on epidemic spreading, we propose a two-layer metapopulation network model, in which aware and unaware individuals migrate in one layer of the network, respectively. We utilize the microscopic Markov chain method [40, 41] to derive the epidemic threshold of our model, and numerical simulations show that the Markov equations have high accuracy for the coupling spread of epidemic and information. The results show that information propagation plays a limited role in delaying the spreading and reducing the scale of infection, and further increasing information transmission rate does not affect the epidemic spreading any more. We also found that different initial population distribution will lead to different influences of individual migration on epidemic dynamics. In addition, individual migration route is a crucial factor for epidemic that the homogeneous route is conducive to the epidemic control.

The rest of this paper is organized as follows. In Sec. II we describe the two-layer metapopulation network model and dynamic process on it. In Sec. III, we provide simulation results to study effects of information transmission and individual migration under different conditions of the initial population distribution. In Sec. IV, we summarize our work.

II. MODEL DESCRIPTION

We apply a two-layer metapopulation network model to describe the coupling dynamics of epidemic spreading and information propagation, where aware and unaware individuals move in layer-\(U\) network and layer-\(A\) network, respectively, as shown in Fig. 1. For the sake of generality, we consider that the networks in the model are weighted.
FIG. 1. Framework framework of epidemic spreading and information propagation coupled in a two-layer metapopulation network. Aware and unaware individuals migrate on layer-A and layer-U network, where \( w_{ij}^{U/A} \) is the weight between patch \( i \) and \( j \) in layer-U/A. Individuals are divided into four states: unaware and susceptible (US), unaware and infected (UI), aware and susceptible (AS), aware and infected (AI). Firstly, individuals associated to patch \( i \) move to neighboring patch \( j \) with probability \( p \cdot R^{U/A}_{ij} \) according to their information state, where \( p \) is the individual mobility rate and \( R^{U/A}_{ij} \) is the transition matrix of layer-U/A. Then, the transmission of epidemics and information occurs simultaneously within each patch. Finally, individuals return to their home patch and the next reaction diffusion process starts.

and undirected, and the weight between patch \( i \) and patch \( j \) in layer-U/A is \( w_{ij}^{U/A} \). There are \( N \) patches and a total population of \( n \) individuals in the model.

The reaction-diffusion process [42] of individuals in our model can be described as follows. Individuals in patches \( i \) migrate with mobility rate \( p \). The aware individuals migrate to the neighboring patch according to the transition matrix \( R^A \) of layer-A, while the unaware individuals migrate based on the transition matrix \( R^U \) of layer-U, where 

\[
R^{U/A}_{ij} = \frac{w^{U/A}_{ij}}{\sum_k w^{U/A}_{ik}}.
\]

Then, epidemic spreading and information propagation occur simultaneously within patches, and individuals update their disease and information status. Finally, the individuals return to their home patches.

In the reaction stage, due to the simultaneous transmission of epidemic and information in the network, the individual states can be divided into four classes: aware and susceptible (AS), unaware and susceptible (US), aware and infected (AI), unaware and infected (UI). The SIS model is used for epidemic spreading. When an US individual contacts with an aware or unaware infected individuals, he/she will be infected with probability \( \beta^U (\beta^U = \beta) \) and become UI state, and then he/she will spontaneously change into AI state with probability \( \kappa \). While, if an individual is in AS state, he/she will be infected as an AI individual with probability \( \beta^A (\beta^A = \gamma \beta, 0 < \gamma < 1) \). \( \gamma \) can reflect the efficiency of information, that is, small value of \( \gamma \) means a high information efficiency. The recovery rate of infected individuals is \( \mu \). In addition, unaware-aware-unaware (UAU) model is used to describe the propagation of information. The unaware individuals get informed with rate \( \lambda \) by contacting with aware individuals, while aware individuals forget information with rate \( \sigma \).

Let us denote \( \rho_i^{US}(t) \), \( \rho_i^{UI}(t) \), \( \rho_i^{AS}(t) \), and \( \rho_i^{AI}(t) \) as the ratios of individuals associated to patch \( i \) in the states of the US, UI, AS, and AI at time \( t \), respectively. According to the transition probability tree given in Figure 2, the Markov evolution equations of the four different states can be described as follows [24]:
where $\pi$ and $\sigma$ is the transition probability that aware individuals forgetting information. Moreover, $\pi_{U/A}$ denotes the probability that unaware/aware individuals get infected. $\pi_{S/I}$ is the probability that susceptible/infected and unaware individuals are not informed the epidemics. The coupled dynamical processes of epidemics and information is synchronous.

\begin{align}
\rho_i^{US}(t+1) &= \rho_i^{UI} r_i(t) \mu + \rho_i^{AI} (t) \sigma \mu \\
&\quad + \rho_i^{US} (t) r_i^S (t) (1 - \pi_i^U (t)) + \rho_i^{AS} (t) \sigma (1 - \pi_i^U (t)), \quad (1)
\end{align}

\begin{align}
\rho_i^{UI}(t+1) &= \rho_i^{UI} r_i(t)(1 - \mu) + \rho_i^{AI} (t) \sigma (1 - \mu) \\
&\quad + \rho_i^{US} (t) r_i^S (t) \pi_i^U (t) + \rho_i^{AS} (t) \sigma \pi_i^U (t), \quad (2)
\end{align}

\begin{align}
\rho_i^{AS}(t+1) &= \rho_i^{UI}(1 - r_i(t)) \mu + \rho_i^{AI} (t) (1 - \sigma) \mu \\
&\quad + \rho_i^{US} (t) (1 - r_i^S (t)) (1 - \pi_i^A (t)) + \rho_i^{AS} (t) (1 - \sigma) (1 - \pi_i^A (t)), \quad (3)
\end{align}

\begin{align}
\rho_i^{AI}(t+1) &= \rho_i^{UI}(1 - r_i(t))(1 - \mu) + \rho_i^{AI} (t) (1 - \sigma)(1 - \mu) \\
&\quad + \rho_i^{US} (t) (1 - r_i^S (t)) \pi_i^A (t) + \rho_i^{AS} (t) (1 - \sigma) \pi_i^A (t), \quad (4)
\end{align}

where $\pi_{U/A} (t)$ represents the probability that unaware/aware individuals associated to patch $i$ get infected at time $t$, and $\pi_{S/I} (t)$ is the probability that the unaware individuals in susceptible/infected state are not informed. Specifically, $\pi_{U/A} (t)$ is given by:

$$\pi_{U/A} (t) = (1 - p) Q_{i}^{U/A} (t) + p \sum_{j=1}^{N} R_{ij}^{U/A} Q_{j}^{U/A} (t). \quad (5)$$

The first term of the right hand in Eq.(5) denotes that susceptible individuals associated to patch $i$ get infected in home patch, and the second term accounts for the probability that susceptible individuals become infected when moving to any neighboring patch $j$.

$Q_{i}^{U/A} (t)$ in Eq.(5) is the probability that unaware/aware individuals in patch $i$ are infected by contacting with any infected individuals inside patch $i$. This probability reads:

$$Q_{i}^{U/A} (t) = 1 - \prod_{j=1}^{N} (1 - \beta_{U/A}^{AI} (\rho_i^{AI}(t) + \rho_i^{UI}(t)))^{\delta_{j\rightarrow i}(t)}, \quad (6)$$

where $n_{j\rightarrow i}(t)$ represents the number of individuals moving from patch $j$ to patch $i$ at time $t$, given by

$$n_{j\rightarrow i}(t) = (1 - p) \delta_{ij} n_i + p [ \rho_j^A(t) R_{ji}^A + \rho_j^U(t) R_{ji}^U ] . \quad (7)$$

$\delta$ in Eq.(7) is the Kronecker delta function, $\delta_{ij} = 1$ if $i \neq j$ and $\delta_{ij} = 0$ otherwise.

FIG. 2. Transition probability trees for the four individual states. $\mu$ is the probability that infected individuals recover to susceptible individuals, and $\sigma$ is the transition probability that aware individuals forgetting information.
Similar to the calculation of $\pi_i^{U/A}(t)$, the probability $r_i^{S/I}(t)$ can be read as

$$r_i(t) = (1 - p) \prod_{j=1}^{N} [1 - \lambda \rho_j^A(t)]^{n_j \rightarrow i(t)} + p \sum_{j=1}^{N} R_{ij}^{U} \prod_{l=1}^{N} [1 - \lambda \rho_l^A(t)]^{n_l \rightarrow i(t)},$$

(8)

where $\rho_i^A(t) = \rho_i^{AS}(t) + \rho_i^{AI}(t)$. The first term of the right hand in Eq.(8) represents the probability that unaware individuals remain in patch $i$ with rate $1 - p$ and are not informed, while the second term denotes the probability that unaware individuals migrate to the neighboring patch $j$ with rate $p \cdot R_{ij}^{U}$ and do not become aware.

In order to derive the epidemic threshold $\beta_c$, it is necessary to explore the stationary solution of the system of Eq.(1)-(4). For the convenience of derivation, we denote $\rho_i(t)$ as the infection scale of patch $i$ at time $t$, i.e., $\rho_i(t) = \rho_i^{U}(t) + \rho_i^{AI}(t)$, we have

$$\rho_i'(t) = \rho_i(t)(1 - \mu) + \rho_i^{AS}(t)[(1 - \sigma) \pi_i^U(t)] + \rho_i^{AI}(t)[(1 - \sigma) \pi_i^A(t)].$$

(9)

When $t \rightarrow \infty$, $\rho_i^{US}$, $\rho_i^{AS}$, $\rho_i^{A}$ satisfy the relationship $\rho_i^{US} = \rho_i^{US} + \rho_i^{AS}$, $\rho_i^{A}$ satisfy the relationship $\rho_i^{US} = \rho_i^{US} + \rho_i^{IS}$, $\rho_i^{A}$ satisfy the relationship $\rho_i^{US} = \rho_i^{US} + \rho_i^{AS}$, and $\rho_i^{A} = \epsilon_i \ll 1$, then $Q_i^{U/A}(t)$ can be approximated as $Q_i^{U/A}(t) \approx \sum_{j=1}^{N} \beta_i^{U/A} \epsilon_j n_{j \rightarrow i}$. By substituting this approximation into Eqs.(1)-(4), (9) and omitting higher order items, we have

$$\begin{align*}
\rho_i^{US} & = \rho_i^{US} \rho_i^{S} + \rho_i^{AS} \rho_i, \\
\rho_i^{AS} & = \rho_i^{US} (1 - r_s^i) + \rho_i^{AS} (1 - \sigma), \\
\mu \epsilon_i^* & = (1 - r_i^S) \pi_i^U + r_i^S \pi_i^U, \\
\end{align*}$$

(10)

Afterwards, by analysing Eq.(10), we can get

$$\mu \epsilon_i^* = \rho_i^{US} \pi_i^U + \rho_i^{AS} \pi_i^A \approx (1 - \rho_i^A) \pi_i^U + \rho_i^A \pi_i^A.$$  

(11)

By inserting $\pi_i^{U/A}$ into Eq. (11), the stationary state of the epidemic can be written as

$$\begin{align*}
\mu \epsilon_i^* & = (1 - \rho_i^A) \sum_{j=1}^{N} \beta_i^{U} \epsilon_j n_{j \rightarrow i} + p \sum_{j=1}^{N} R_{ij}^{U} \sum_{l=1}^{N} \beta_i^{U} \epsilon_l n_{l \rightarrow j} \\
+ & \rho_i^A \sum_{j=1}^{N} \beta_i^{A} \epsilon_j n_{j \rightarrow i} + p \sum_{j=1}^{N} R_{ij}^{A} \sum_{l=1}^{N} \beta_i^{A} \epsilon_l n_{l \rightarrow j},
\end{align*}$$

(12)

Inserting $n_{j \rightarrow i}$ and $n_{l \rightarrow j}$ into Eq. (12), we obtain the expression

$$\begin{align*}
\frac{\mu}{\beta} \epsilon_i^* & = \sum_{j=1}^{N} M \epsilon_j,
\end{align*}$$

(13)

where $M$ is a matrix of $N \times N$, and its element $M_{ij}$ can be expressed as

$$\begin{align*}
M_{ij} = & (1 - p)^2 (1 - \rho_i^A + \gamma \rho_j^A) \delta_{ij} n_i \\
+ & p (1 - p) \{ (1 - \rho_i^A + \gamma \rho_j^A) (\rho_j^A R_{ij}^A + \rho_j^A R_{ij}^U) + \\
& [\gamma \rho_j^A R_{ij}^A + (1 - \rho_i^A) R_{ij}^U] n_j \\
+ & p^2 [\gamma \rho_j^A R_{ij}^A + (1 - \rho_i^A) R_{ij}^U] (\rho_j^A R_{ij}^A + \rho_j^U R_{ij}^U) n_j.
\end{align*}$$

(14)

Thus, the epidemic threshold $\beta_c$ can be obtained by solving the maximum eigenvalue of matrix $M$, given by

$$\beta_c = \frac{\mu}{\Lambda_{max}(M)}.$$  

(15)
Let us now analyze the elements of the matrix $M$ in Eq.(14). $M_{ij}$ corresponds to the probability that an individual associated to patch $i$ contacts with another one from patch $j$. The first term describes the situation that patch $i$ and $j$ are the same one and neither of the two individuals migrates. The second term describes that the individuals contact in patch $i$ or $j$ ($i \neq j$). And the third one denotes these two individuals migrate to the same patch which is different from their associated patches. More importantly, the influence of information propagation leads to individuals being divided into two states: aware individuals who migrate based on $R^A_{ij}$, and unaware individuals who migrate based on $R^U_{ij}$. From Eq.(14), it is clearly to see that each term considers the effect of migration route by both aware and unaware individuals.

In the case of without information diffusion, the value of $\rho^A_i$ is zero and naturally all individuals migrate in layer-$U$ network. Then, $M_{ij}$ can be simplified as:

$$M_{ij} = (1 - p)^2 \delta_{ij} n_i + p(1 - p)(R^U_{ji} + R^U_{ij}) n_j + p^2 R^U_{ij} R^U_{ji} n_j,$$

which is consistent with the result in Ref. [40].

![Graph](image)

**FIG. 3.** The final infected ratio $\rho^I$ versus $\beta$ for different information dissemination rate $\lambda$. The simulations are carried out under the homogeneous initial population distribution (HOD). (a) Results for the migration case of ER-BA; (b) Results for the migration case of BA-ER. The other parameters are set as $\mu = 0.3$, $\sigma = 0.3$, $\kappa = 0.3$, $\gamma = 0.5$, $p = 0.3$. The solid curves correspond to the numerical solutions of the Markovian evolution equations (Eq.(1)-(4)), whereas the points represent the results of Monte Carlo simulations which are the average of 100 simulations.

### III. SIMULATION RESULTS

We have performed extensive Monte Carlo simulations on two-layer metapopulation network, which reflects the characteristics that aware and unaware individuals have different migration trajectories. For simplicity, the two layer networks composed of Erdős-Rényi (ER) and Barabási Albert (BA) synthetic networks are considered. We use BA-ER to describe the individual migration that aware individuals move on BA network and unaware individuals move on ER network, while ER-BA represents the opposite situation. There are totally $N = 1000$ patches in each layer and the average degree of ER and BA networks set as $\langle k \rangle_{ER} = 5$ and $\langle k \rangle_{BA} = 5.96$, respectively. The edge weights are randomly assigned according to the uniform distribution in the range of $[1, 50]$. The simulations involve two types of initial population distribution, namely, homogeneous distribution (HOD) and heterogeneous distribution (HED). For the case of HOD, each patch contains same number of individuals, i.e., $n_i^{ER} + n_i^{BA} = 500$, $\forall i = 1, 2, \ldots, N$, where
FIG. 4. The stationary fraction of infected individuals as a function of information dissemination rate $\lambda$ and infection rate $\beta$. The initial population distribution is homogeneous (HOD). (a) The migration case of ER-BA; (b) The migration case of BA-ER. Other parameter settings are consistent with Figure 3. The phase diagrams are obtained by averaging 100 Monte Carlo simulations for each point in the grid $50 \times 50$.

FIG. 5. The final fraction of infected individuals $\rho^I$ as a function of different combinations of $\kappa$ and $\gamma$ under homogeneous initial population distribution. The top panels show the results in the case of ER-BA, while bottom panels is the results of the BA-ER situation. The values of $\gamma$ from left to right are $\gamma = 0$, $\gamma = 0.3$ and $\gamma = 0.7$, and other parameters are set to $\lambda = 0.001$, $\mu = 0.3$, $\sigma = 0.3$, $p = 0.3$. 
\( n_i^{ER/BA} \) denote the number of population of patch \( i \) in layer-ER/layer-BA network. For the case of the HED, the number of population contained in each patch is proportional to the sum of its edge weights in layer-BA network, i.e., \( \sum_j w_{ij}^{BA} \). The total population in the network is \( n_0 = 5 \times 10^5 \) for both cases of the HOD and the HED.

At the beginning of Monte Carlo simulation, the infection rate is set as \( \beta = 10^{-3} \), and there are 500 infected individuals as seeds for the start of epidemic spread. Similarly, the same initial condition is used to initialize the variables \( \rho^{I5}(0) \), \( \rho^{I4}(0) \), \( \rho^{A3}(0) \), and \( \rho^{AI}(0) \) in Markovian equations. The experimental results are obtained on the average of 100 MC simulations for each combination of the parameters \( (\beta, \mu, \lambda, \sigma, \kappa, \gamma, p) \) we testified.

A. Effects of the information dissemination on the epidemic

Firstly, we explore the influence of information propagation on epidemic spreading on metapopulation networks. The simulation is carried out under the condition of homogeneous initial population distribution. Figure 3 shows the final infected ratio \( \rho^I \) at the stable state versus \( \beta \) for different choices of \( \lambda \), where \( \rho^I = \sum_i^N \rho_i^I \). The dots represent the results obtained for Monte Carlo simulations and the solid curves are for the solutions of the Markovian equations. The high consistency between the two proves the correctness of Markov equations Eqs.(1)-(4).

As can be seen from Figure 3, when there is no communication between individuals \( (\lambda = 0) \), the epidemic threshold of individual migration with BA-ER is higher than that with ER-BA. Due to the lack of information dissemination, there are few aware individuals and more unaware individuals in the network. When the migration situation is BA-ER, the unaware individuals migrate in layer-ER network, which suppress the epidemic spreading due to the high homogeneity in ER networks, thus, the epidemic threshold is larger, compared with the case of ER-BA.

Further, we find that when the information transmission rate \( \lambda \) is small, with the increase of it, the proportion of infection decreases and the epidemic threshold increases. However, with the further increase in \( \lambda \), the difference between the curves is neglective. This is because if \( \lambda \) is higher than the value necessary for the full dissemination of information, it will no longer affect the epidemic spreading. In addition, compared with the case of individual migration of ER-BA, the effect of \( \lambda \) on epidemic is smaller when the migration is BA-ER. It is because that aware individuals migrate on the BA network in the situation of BA-ER, and the structural heterogeneity of BA network promotes the diffusion of information, thus the value of \( \lambda \) required for the full information spread will be smaller. Therefore, the impact of information transmission rate \( \lambda \) is less significant for the BA-ER situation.

In order to deeply understand the impact of information dissemination on the epidemic, Figure 4 illustrates the diagrams for the final infection scale for different values of \( \beta \) and \( \lambda \). It is obvious that the epidemic threshold of ER-BA migration is smaller when \( \lambda \) is small \( (\lambda \to 0) \), which is consistent with the result in Figure 3. Within a certain range of \( \lambda \), the epidemic threshold increases with \( \lambda \). We can clearly see that the impact of information transmission rate is more significant under the case of ER-BA.

In our model, the self-awareness of UI individuals will also promote the diffusion of information by changing their state into AI with probability \( \kappa \). Moreover, the infection rate of aware individuals decreases with factor \( \gamma \) \( (\beta^A = \gamma \beta) \). Figure 5 shows the final infected fraction for different combinations of \( \kappa \) and \( \gamma \). With the increase of \( \gamma \) from left to right in Fig. 5, the infection scale increases but the epidemic threshold is almost unaffected. The smaller the value of \( \gamma \), the stronger the conscious defense of aware individuals, thus, inhibiting the epidemic spreading with a reduced infection scale. In addition, the increase of \( \kappa \) can further reduce the infection scale, but its effect gradually weakens with the increase of \( \gamma \). It can be explained that the information dissemination has more influence on the epidemic dynamics when the information efficiency is high, and vice versa. It suggests that information propagation can enhance the conscious defense of individuals, thus contributing to the control of epidemic.

B. Effects of the individual migration on the epidemic with information propagation

In this part, we discuss the influence of individual migration on the coupling transmission of epidemic and information in metapopulation networks under the conditions of homogeneous (HOD) and heterogeneous population distribution (HED), respectively. In the case of HOD, the ratio of infection in steady state for different mobility rate \( p \) are shown in Fig. 6, it is clear that the agreement between simulations and the equations is almost exact. Firstly, whether the individual migration is ER-BA or BA-ER, the increase of \( p \) will reduce the epidemic threshold and increase the proportion of infection, that is, individual migration promotes the epidemic spread.

When the individual migration is ER-BA, the smaller the value of information transmission rate \( \lambda \) (Fig. 6 (a)), the greater the impact of mobility rate \( p \) on epidemic dynamics. Since smaller \( \lambda \) is not conducive to information dissemination, it results in a larger proportion of unaware individuals migrating on the BA network with hub patches. On the contrary, with the increase of \( \lambda \) (Fig. 6 (b)), more aware individuals migrate in the ER network which is more
FIG. 6. The final fraction of infected ratio $\rho^I$ versus $\beta$ for different mobility rate $p$ when the initial population is homogeneously distributed (HOD). (a) and (c) are the results that the individual migration is ER-BA, while (b) and (d) show the results of the BA-ER situation. The solid curves and the points are represent results of the iteration of Markov equations (Eq.(1)-(4)) and the Monte Carlo simulations respectively. Each point is the average results of more than 100 individual based simulations. Other parameters are set to $\mu = 0.3$, $\sigma = 0.3$, $\kappa = 0.3$, $\gamma = 0.5$.

FIG. 7. The final fraction of infected individuals obtained as a function of $p$ and $\beta$ with homogeneous initial population distribution (HOD). The heatmap represents the fraction of infected individuals in the steady state obtained from Monte Carlo simulations. The experimental conditions for the four phase diagrams are same with Fig. 6. The four graphs are obtained by averaging 100 Monte Carlo simulations for each point in the grid $50 \times 50$.

homogeneous, thus the effect of individual mobility is neglectable. A detailed analysis of mobility rate $p$ is shown in Fig. 7 (a) and (b).

Contrary to the case of ER-BA, when the individual migration is BA-ER, the larger value of information transmission rate $\lambda$ leads to more obvious influence on the epidemic. This phenomenon can be understood in a similar way as in ER-BA situation. For smaller mobility rate $p$ ($p = 0.3$), the epidemic can be delayed by the introduction of information propagation. Higher information transmission rate further delays the epidemic with a higher threshold. With frequent mobility (increase of $p$), even a higher information transmission rate can not suppress the epidemic spread due to the frequent mobility of aware individuals in BA networks, which promotes the epidemic spread. More details can be found in Fig. 7 (c) and (d).

In the case of heterogeneous population distribution (HED), the effect of mobility rate $p$ on epidemic dynamic is illustrated in Fig.8, where points represent the results obtained for Monte Carlo simulations from the epidemic process and the solid lines are the iteration of the Markovian equations. In order to ensure the heterogeneity of population distribution, each patch contains the number of individuals proportional to the sum of the edge weights in layer-BA.
FIG. 8. The infection scale $\rho^i$ for different values of mobility rate $p$ under heterogeneous initial population distribution (HED). (a)(b) The individual migration is ER-BA; (c)(d) The individual migration is BA-ER. The curves represent the results of Markovian equations solution, and the points are obtained from Monte Carlo simulations. Each point is the average results of more than 100 individual based simulations. Other parameters are the same as Figure 6.

FIG. 9. The final fraction of infected individuals under different combination values of $p$ and $\beta$ with heterogeneous initial population distribution (HED). The heatmap denotes the stationary fraction of infected individual obtained from Monte Carlo simulations. The experimental conditions for the phase diagrams are same as Fig. 6. The results are obtained by averaging 100 Monte Carlo simulations for each point in the grid $50 \times 50$.

network ($\sum_j w_{ij}^{BA}$). Compared with the situation of HOD in Fig. 6, HED is more beneficial to epidemic transmission by the lower epidemic threshold as shown in Fig. 8. In addition, we find that the threshold increases with mobility rate $p$, that is, individual’s mobility delays the epidemic spread. This counter-intuitive phenomenon is called detrimental effect in Ref. [40]. Because the epidemic threshold is related to the maximum eigenvalue of matrix $M$ (Eq.(14)), the eigenvalue expression of $M$ can be obtained by using perturbation theory [43], which can be regarded as a quadratic equation with respect to $p$ [40]. Therefore, there is an optimal mobility rate $p^*$ such that the maximum eigenvalue of $M$ is minimized.

For the migration route of ER-BA (Fig. 8 (a)(b)), the impact of $p$ is slightly different under various values of information transmission rate $\lambda$. With the increase of $p$, the epidemic threshold first increases and then decreases when $\lambda$ is small, but it increases monotonically for large $\lambda$. The detailed analysis can be found in Fig. 9 (a) and (b). Similar to the analysis of Fig. 3, higher information transmission rate $\lambda$ will increase the proportion of aware individuals who migrate in the ER network with uniform topology, which can slow down the spread of disease. Thus, it indicates that when the epidemic breaks out, guiding the aware individuals to migrate according to the homogeneous route to avoid people gathering is helpful for the control of epidemic.
In addition, when the individual migration is BA-ER (Fig. 8 (c)(d)), the impact of individual migration on epidemic dynamics is similar for different values of $\lambda$, and the disease threshold increases monotonically with the increase of mobility rate $p$ (Fig. 9 (c)(d)). It can be understood that aware individuals migrate on the BA network, both the heterogeneity of network structure and heterogeneous population distribution will promote information dissemination, which needs a less value of $\lambda$ to achieve the full dissemination of information. As a result, $\lambda$ has no obvious effect on epidemic transmission with the value we tested in the simulation, which is consistent with the result in Fig. 3.

IV. CONCLUSIONS AND DISCUSSIONS

As a summary, we study the coupled interacting of epidemic spread and information propagation on the metapopulation network, and mainly explore how aware individual’s behavior change in migration route affects the epidemic spreading. We propose a two-layer multipopulation network model to reflect the different migration routes that aware and unaware individuals take. Then, based on the transition tree, we build the dynamical model with the Markov equations accordingly. The expression of disease threshold can be obtained by solving an eigenvalue problem. The high consistency between the iterative of Markov equations and Monte Carlo simulations verifies the accuracy of theoretical analysis.

First, we have investigated how the dissemination of relevant information affects the epidemic spread on metapopulation framework. Our results show that the information propagation leads to larger epidemic threshold and smaller final infection scale, thus it can suppress the epidemic spread. However, the inhibitory effect of information propagation on epidemic is limited and it depends on the migration route that aware individuals take. Then, we have found that the role of mobility in delaying or promoting the epidemic based on the initial population distribution, where the HOD promotes the epidemic spreading while the HED does not. In addition, the individual migration route is another significant factor which affects the epidemic dynamics, where a homogeneous migration route would be beneficial to epidemic control. Overall, our work points out a new way to explore factors such as the behavior change in migration that may alternate the epidemic dynamic, and also provides guidance for the intervention in epidemic spread.

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COMPLIANCE WITH ETHICAL STANDARDS

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

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Figures

Figure 1

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