Impacts of information propagation on epidemic spread over different migration routes

Bing Wang · Min Gou · Yuexing Han

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Abstract Information propagation driven by the epidemic may cause individuals awareness to change their behavior to prevent themselves from being infected, as we observed in reality that aware individuals often migrate away from infected areas. In this work, we study the coupled interaction of epidemic spreading and information propagation over a two-layer metapopulation network, where aware and unaware individuals separately take different migration routes, and mainly explore how individual migration route affects the epidemic spreading. Combined with the transition probability tree of individual states, we use Markovian chain approach to derive the epidemic threshold for the proposed model. Through numerous Monte Carlo simulations, we verify the accuracy of the Markovian equations for the prediction of epidemic dynamics. The results show that the role of information transmission in suppressing the epidemic is limited. Further increase in the information transmission rate beyond some critical value will no longer affect the epidemic. Detailed analysis of information propagation has to consider the migration route of individuals, especially the aware individuals, and their mobility frequency. In addition, the initial population distribution is also a fundamental factor for the epidemic dynamic. With a heterogeneous population distribution, frequent mobility of individuals would delay the epidemic spread, while with the homogeneous population distribution, it does not. The study of the coupled interaction between epidemic and information over separate migration routes provides helpful guidance for the intervention of epidemic in reality.

Keywords Epidemic spreading · Information propagation · Metapopulation networks

Mathematics Subject Classification 00A69 · 97M10

1 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 among individuals to simulate the epidemic dynamics, such as the classical susceptible–infected–susceptible (SIS) model [4], susceptible–infected–recovery (SIR) model [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 network models to explore different factors that may affect epi-
demics, e.g., contact patterns between people [9,10], the immunity of particular individuals [11–13], migration patterns [14–16], etc.

When epidemic spreads in the population, disease-related information also propagates among individuals 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 infection risk. Funk et al. [20,21] studied the coupling transmission of epidemic and information in uniformly mixed population. They found that information dissemination significantly reduced the final infection ratio, while it had no effect on the epidemic threshold. Sahneh et al. [22,23] proposed the susceptible–alert–infected–susceptible (SAIS) model and found that the increase in individual alertness helps to slow down the epidemic spreading.

In reality, epidemic and information usually have different routes and forms of transmission. Based on this observation, Granell et al. [24,25] utilized two-layer multiplex networks [8] to study the interaction between epidemic and information. Furthermore, Zhao et al. [26] found that the stronger the degree correlation of nodes in multiplex networks, the smaller the epidemic threshold and the infected scale will be. 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 studies on the coupling dynamics of epidemic spreading and information propagation are carried out on the contact networks where nodes represent individuals and edges represent the association between individuals [30]. Thus, it is not suitable for the spatial spread of epidemic due to the omission of individual migration. While individual migration is a crucial factor for epidemic spreading, 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 fundamental tool to study spatial spread of epidemic when considering individual migration [33–37]. In this framework, nodes represent patches, while links denote transportation routes among patches. Inside each patch, a dynamic process of epidemic spreading or information propagation among individuals occurs. 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 a change in migration behavior contributes to the epidemic to healthy patches, thus promoting the global spread of the epidemic. Lima et al. [39] used real data to establish the mobility matrix and the communication matrix, and concluded that information campaigns might be effective countermeasures for disease control, but the theoretical analysis of the disease threshold was not given.

In order to further study the impact of behavioral changes in individual migration caused by information dissemination on epidemic spreading, we propose a two-layer metapopulation network model, in which aware and unaware individuals separately migrate on one of the two-layer network. We utilize the microscopic Markovian chain method [40,41] to derive the epidemic threshold of the proposed model, and numerical simulations show that the Markovian equations have high accuracy for the coupling spread of epi-

\( \text{Fig. 1 Framework of the coupled interaction of epidemic spreading and information propagation on a two-layer metapopulation network. Unaware and aware individuals migrate on layer-}\ U\ \text{network and layer-}\ A\ \text{network, respectively, where } w_{ij}^{U/A} \text{ is the weight between patch } i \ \text{and } j \ \text{in layer-}\ U/A. \ \text{Individuals are divided into four states: unaware and susceptible (US), unaware and infected (UI), aware and susceptible (AS), aware and infected (AI). Firstly, individuals associated with patch } i \ \text{move to neighboring patch } j \ \text{with probability } p \times R_{ij}^{U/A} \text{ according to their information state, where } p \text{ is the mobility rate and } R_{ij}^{U/A} \text{ is the transition matrix of layer-}\ U\text{. Then, the transmission of epidemics and information occurs simultaneously within each patch. Finally, individuals return to their home patch and the next reaction–diffusion process starts.} \)
demic and information. The results show that information propagation plays a limited role in delaying the epidemic spread and reducing the infection scale, and further increase in information transmission rate does not affect the epidemic spreading any more. We also find that different initial population distributions will lead to different influences of individual migration on epidemic dynamics. In addition, individual migration route is a crucial factor for epidemic, for instance, the homogeneous route is conducive to the epidemic control.

The rest of this paper is organized as follows. In Sec. 2, we describe the two-layer metapopulation network model and dynamical process on it. In Sec. 3, we provide simulation results for the interaction of information transmission and individual migration under different conditions of the initial population distribution. In Sec. 4, we summarize our work.

2 Model description

We apply a two-layer metapopulation network model to describe the coupling dynamics of epidemic spreading and information propagation, where unaware and aware 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 network in the model is weighted and undirected, and the weight between patch i and patch j in layer-U/A is \( w_{ij}^{U/A} \). There are totally N patches and a total population of n individuals in the network.

The reaction–diffusion process [42] of individuals in our model can be described as follows. Individuals in patches i migrate with mobility rate \( p \). Specifically, aware/unaware individuals migrate to the neighboring patch j according to the transition matrix \( R_{ij}^{U/A} \) of layer-U/A, where \( R_{ij}^{U/A} = \frac{w_{ij}^{U/A}}{\sum_k w_{ik}^{U/A}} \). Then, epidemic spreading and information propagation occur simultaneously within patches, and individuals update their disease status and information status. Finally, individuals return to their home patches.

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

Let us denote \( \rho_{i,U}^r(t), \rho_{i,U}^I(t), \rho_{i,A}^S(t), \) and \( \rho_{i,A}^I(t) \) as the fractions of individuals associated with patch i in the states of the US, UI, AS, and AI at time t, respectively. According to the transition probability tree given in Fig. 2, the Markovian evolution equations for the four different states can be described as follows [24]:

\[
\begin{align*}
\rho_{i,U}^r(t+1) &= \rho_{i,U}^I(t) \mu + \rho_{i,A}^I(t) \sigma \mu \\
&+ \rho_{i,U}^I(t) \sigma (1 - \pi_i^U(t)), \\
\rho_{i,U}^I(t+1) &= \rho_{i,U}^I(t) (1 - \mu) + \rho_{i,A}^I(t) (1 - \mu) \\
&+ \rho_{i,U}^I(t) \pi_i^U(t) \\
&+ \rho_{i,A}^S(t) \sigma \pi_i^U(t), \\
\rho_{i,A}^S(t+1) &= \rho_{i,U}^I(t) (1 - \mu) \mu + \rho_{i,A}^I(t) (1 - \sigma) \mu \\
&+ \rho_{i,U}^I(t) (1 - \sigma) \pi_i^A(t) \\
&+ \rho_{i,A}^S(t) (1 - \sigma) \pi_i^A(t), \\
\rho_{i,A}^I(t+1) &= \rho_{i,U}^I(t) (1 - \mu) \\
&+ \rho_{i,A}^I(t) (1 - \sigma) \pi_i^A(t) \\
&+ \rho_{i,U}^I(t) \pi_i^S(t) \pi_i^A(t), 
\end{align*}
\]

where \( \pi_i^U(t) \) represents the probability that unaware/aware individuals associated with patch i get infected at time t, and \( r_i^S(t) \) is the probability that unaware individuals in susceptible/infected state are not informed. Specifically, \( \pi_i^U(t) \) is given by:

\[
\pi_i^U(t) = (1 - p) Q_i^U(t) + p \sum_{j=1}^{N} R_{ij}^{U/A} Q_j^U(t).
\]
The first term of the right hand in Eq. (5) denotes that susceptible individuals associated with 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 $j$. $Q_{ij}^{UA}(t)$ in Eq. (5) is the probability that unaware/aware individuals in patch $i$ are infected by contacting with infected individuals inside patch $i$ and reads as:

$$Q_{ij}^{UA}(t) = 1 - \prod_{j=1}^{N} (1 - \rho_{ij}^{UA} (\rho_{ij}^{AI}(t) + \rho_{ij}^{UI}(t)))^{n_{j\rightarrow i}(t)}, \quad (6)$$

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

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

where $\delta$ is the Kronecker delta function, $\delta_{ij} = 1$ if $i \neq j$ and $\delta_{ij} = 0$ otherwise.

Similar to the calculation of $\pi_{ij}^{UA}(t)$, the probability $r_{ij}^{SI}(t)$ can be read as

$$r_{ij}(t) = (1-p) \prod_{j=1}^{N} \left[ 1 - \lambda \rho_{ij}^{A}(t) \right]^{n_{j\rightarrow i}(t)} + p \sum_{j=1}^{N} R_{ij}^{U} \prod_{l=1}^{N} \left[ 1 - \lambda \rho_{ij}^{A}(t) \right]^{n_{l\rightarrow j}(t)}, \quad (8)$$

where $\rho_{ij}^{A}(t) = \rho_{ij}^{AS}(t) + \rho_{ij}^{AI}(t)$. The first term of the right hand in Eq. (8) denotes 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 $p R_{ij}^{U}$ 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 Eqs. (1)–(4). For the convenience of derivation, we denote $\rho_{ij}(t)$ as the infection scale of patch $i$ at time $t$, i.e., $\rho_{ij}(t) = \rho_{ij}^{UI}(t) + \rho_{ij}^{AI}(t)$, we have

$$\rho_{ij}(t) = \rho_{ij}^{I}(t)(1 - \mu) + \rho_{ij}^{AS}(t)(\sigma \pi_{ij}^{U}(t)) + (1 - \sigma) \pi_{ij}^{A}(t) + \rho_{ij}^{US}(t) [r_{ij}^{S}(t) \pi_{ij}^{U}(t) + (1 - r_{ij}^{S}(t)) \pi_{ij}^{A}(t)]. \quad (9)$$

When $t \to \infty$, $\rho_{ij}^{US}$, $\rho_{ij}^{AS}$, $\rho_{ij}^{I}$ satisfy the relationship $\rho_{ij}^{US}(t+1) = \rho_{ij}^{US}(t) = \rho_{ij}^{US}$, $\rho_{ij}^{AS}(t+1) = \rho_{ij}^{AS}$, and $\rho_{ij}^{I}(t+1) = \rho_{ij}^{I}(t) = \rho_{ij}^{I}$, respectively. When $\beta < \beta_c$, the number of infected individuals in patch $i$ is negligible, i.e., $\rho_{ij}^{I} = \epsilon_{ij}^{I} \ll 1$, then $Q_{ij}^{UA}(t)$ can be approximated as $Q_{ij}^{UA}(t) \approx \sum_{j=1}^{N} \beta_{ij}^{U/A} \epsilon_{ij}^{S} n_{j\rightarrow i}$. By substituting this approximation in Eqs. (1)–(4), (9) and omitting higher-order items, we have

$$\rho_{ij}^{US} = \rho_{ij}^{US} r_{ij}^{S} + \rho_{ij}^{AS},$$

$$\rho_{ij}^{AS} = \rho_{ij}^{US} (1 - r_{ij}^{S}) + \rho_{ij}^{AS} (1 - \sigma),$$

$$\mu \epsilon_{ij}^{I} = \rho_{ij}^{US} \left[ (1 - r_{ij}^{S}) \pi_{ij}^{A} + r_{ij}^{S} \pi_{ij}^{U} \right] + \rho_{ij}^{AS} \left[ (1 - \sigma) \pi_{ij}^{A} + \sigma \pi_{ij}^{U} \right]. \quad (10)$$
Afterward, by analyzing Eq. (10), we can get
\[
\mu \epsilon^*_i = \rho_i U \pi_i U + \rho_i A \pi_i A \approx (1 - \rho_i A) \pi_i U + \rho_i A \pi_i A. \tag{11}
\]
By inserting \(\pi_i U/A\) into Eq. (11), the stationary state of the epidemic can be rewritten as
\[
\mu \epsilon^*_i = (1 - \rho_i A) \left[ (1 - p) \sum_{j=1}^{N} \beta U \epsilon^*_j n_{j\rightarrow i} + p \sum_{j=1}^{N} R_{ij} \sum_{l=1}^{N} \beta U \epsilon^*_l n_{l\rightarrow j} \right] \\
+ \rho_i A \left[ (1 - p) \sum_{j=1}^{N} \beta A \epsilon^*_j n_{j\rightarrow i} + p \sum_{j=1}^{N} R_{ij} \sum_{l=1}^{N} \beta A \epsilon^*_l n_{l\rightarrow j} \right], \tag{12}
\]
Inserting \(n_{j\rightarrow i}\) and \(n_{l\rightarrow j}\) into Eq. (12), we obtain the expression
\[
\frac{\mu}{\beta} \epsilon^*_i = \sum_{j=1}^{N} \mathbf{M} \epsilon^*_j, \tag{13}
\]
where \(\mathbf{M}\) is a matrix of \(N \times N\), and its element \(M_{ij}\) can be expressed as
\[
M_{ij} = (1 - p)^2 (1 - \rho_i A + \gamma \rho_i A) \delta_{ij} n_i + p(1 - p)((1 - \rho_i A + \gamma \rho_i A)(\rho_j A R_{ji} + \rho_j U R_{ji}) \\
+ [\gamma \rho_i A R_{ij} + (1 - \rho_i A) R_{ij} U]) n_j + p^2 [\gamma \rho_i A R_{ij} \\
+ (1 - \rho_i A) R_{ij} U](\rho_j A R_{ji} + \rho_j U R_{ji}) n_j. \tag{14}
\]
Thus, the epidemic threshold \(\beta_c\) can be obtained by solving the maximum eigenvalue of matrix \(\mathbf{M}\), given by
\[
\beta_c = \frac{\mu}{\lambda_{\text{max}}(\mathbf{M})}. \tag{15}
\]
Let us now analyze the elements of the matrix \(\mathbf{M}\) in Eq. (14). \(M_{ij}\) corresponds to the probability that an individual associated with patch \(i\) contacts with another one from patch \(j\). More in detail, the first term describes the situation that patches \(i\) 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 \(j\) \((i \neq j)\). 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 in two states, that is, aware individuals who migrate based on \(R_{ij}^A\) and unaware individuals who migrate based on \(R_{ij}^U\). From Eq. (14), it is easy to see that each term considers the effect of migration routes of both aware and unaware individuals.

In the case of no information diffusion, the value of \(\rho_i^A\) is zero and naturally all individuals migrate in layer-\(U\) network. Then, \(\mathbf{M}_{ij}\) is simplified as:
\[
M_{ij} = (1 - p)^2 \delta_{ij} n_i + p(1 - p)(R_{ij}^U + R_{ij}^U) n_j \\
+ p^2 R_{ij}^U R_{ji} n_j, \tag{16}
\]
which is consistent with the result in Ref. [40].

3 Simulation results

We have performed extensive Monte Carlo simulations on two-layer metapopulation networks, which reflect the characteristics that aware and unaware individuals follow different migration trajectories. In order to distinguish the migration routes that unaware and aware individuals take, we consider a two-layer metapopulation network, where one-layer network is Erdős–Rényi (ER) network, representing a homogeneous migration route, and the other-layer network is Barabási–Albert (BA) network, representing a heterogeneous migration route. Specifically, we use BA-ER to indicate 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 network, and the average degree of ER and BA networks is set as \((k)_{ER} = 5\) and \((k)_{BA} = 5.96\), respectively. It is to note that the choice of the average degree of the network does not affect the main results and an appropriate choice of the average degree has to guarantee the connectedness of the network. The edge weights are randomly assigned according to the uniform distribution in the range of \([1, 50]\). Since it has found that the distribution of initial population also affects the epidemic, especially when considering the recurrent mobility as in Ref. [40], here, we consider two types of initial population distributions, namely homogeneous distribution (HOD) and heterogeneous distribution (HED). For the case of HOD, each patch contains the same number of individuals, i.e., \(n_{ER}^i = n_{BA}^i = 500, \forall i = 1, 2, \ldots, N\), where \(n_{ER/BA}^i\) denotes the
number of population in 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 simulations. Similarly, the same initial condition is used to initialize the variables $\rho^{US}(0)$, $\rho^{UL}(0)$, $\rho^{AS}(0)$, and $\rho^{AL}(0)$ in Markovian equations. The experimental results are obtained on the average of 100 MC simulations for each combination of the parameters ($\beta$, $\mu$, $\lambda$, $\sigma$, $\kappa$, $\gamma$, $p$) that we testified.

3.1 Effects of information propagation on the epidemic spread over different migration routes

Firstly, we explore the influence of information propagation on epidemic spreading on two-layer metapopulation networks representing different migration routes, followed by aware and unaware individuals. Simulations are carried out under the conditions of both the HOD and the HED. Since the results are similar, here we only show the results with the HOD. Figure 3 shows the final infected ratio $\rho^I$ in the stable state versus $\beta$ for different choices of $\lambda$, where $\rho^I = \sum_{i=1}^{N} \rho^I$. The dots represent the results obtained from Monte Carlo simulations, and the solid curves are the solutions by iterating the Markovian equations. The high consistency between the curves and the dots verifies the correctness of the Markovian equations Eqs. (1)-(4).

The role of different migration route followed by aware and unaware individuals is demonstrated in Fig. 3. When there is no communication between individuals ($\lambda = 0$), the epidemic threshold of the BA-ER network is higher than that of ER-BA network. This is because that due to the lack of information dissemination, there are few aware individuals and more unaware individuals in the network. When the migration is BA-ER network, unaware individuals migrate in layer-ER subnetwork, which suppresses the epidemic spreading due to the high homogeneity in ER networks; thus, the epidemic threshold is slightly larger, compared with the case of ER-BA.

When the information propagation is involved, with the increase in the information transmission rate $\lambda$, the epidemic size decreases and the epidemic threshold increases, see Fig. 3a, b. It indicates that the involvement of information propagation between individuals suppresses the epidemic spread. However, further increase in $\lambda$ does not help suppress the epidemic, where the gap between the curves becomes 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 ER-BA network, the effect of $\lambda$ on epidemic is less when the migration is BA-ER. We can understand it as that when aware individuals migrate on the BA network, the structural heterogeneity of BA network promotes the information diffusion.
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(a) ER-BA network; (b) BA-ER network. Other parameter settings are consistent with Fig. 3. (Color figure online)

thus leading to a less $\lambda$ required for the full information dissemination. Therefore, the impact of information transmission rate $\lambda$ is less significant in the BA-ER situation.

In order to deeply understand the impact of information dissemination on the epidemic, Fig. 4 illustrates the diagrams for the final infection scale with combined values of $\beta$ and $\lambda$ over different migration routes. It clearly shows that for a smaller information transmission rate $\lambda$, the migration of unaware individuals in heterogeneous networks (ER-BA network) would promote the epidemic with a smaller epidemic threshold, compared with the case of BA-ER, which is consistent with the result in Fig. 3. Then, with the increase in $\lambda$, more individuals become aware, which helps delay the epidemic with an increased epidemic threshold. In addition, compared with the case of BA-ER, the impact of information transmission rate is more significant in the case of ER-BA.

In our proposed model, since the self-awareness of individuals in UI state into AI state controlled by parameter $\kappa$ will also promote the information dissemination, and the information efficiency on aware individuals is described by the factor $\gamma$ ($\beta^A = \gamma \beta$), we need to explore their combined effect on the epidemic. Figure 5 shows the epidemic size for different combinations of $\kappa$ and $\gamma$. With the increase in $\gamma$ (from right to left), the epidemic size decreases, while the epidemic threshold is almost unchanged. The smaller the value of $\gamma$ is, the stronger the conscious defense of aware individuals will be, thus inhibiting the epidemic spreading with a reduced infection scale. In addition, the increase of $\kappa$ can further reduce the infection scale (Fig. 5a, d), while the gap is neglectful when aware individuals still have high infection rate (large $\gamma$). It indicates that the information dissemination would have more influence on the epidemic only when the information efficiency is sufficiently high, suggesting that information propagation can enhance the conscious defense of individuals, thus contributing to the control of epidemic.

3.2 Effects of mobility rate on the epidemic spread with information propagation

In this part, we explore the role of individual mobility over different migration route in the coupling transmission of epidemic and information under the conditions of homogeneous (HOD) population distribution and heterogeneous population distribution (HED), respectively. In the case of HOD, the fraction of infection at steady state for different mobility rate $p$ is shown in Fig. 6. We see a perfect agreement between simulations and the equations. Independent of the migration route being ER-BA network or BA-ER network, the increase in mobility rate $p$ will promote the epidemic spread in terms of a reduced epidemic threshold and an increased infection scale.

When the migration route is composed of ER-BA networks, the smaller the information transmission rate $\lambda$ is, the greater the impact of mobility rate $p$ on the
Fig. 5 Epidemic size $\rho^I$ as a function of $\beta$ for different combinations of $\kappa$ and $\gamma$ over migration routes of ER-BA network and BA-ER network. The initial population distribution is homogeneous (HOD). (Top) The migration route of ER-BA networks. (Bottom) The migration route of BA-ER networks. The values of information efficiency are $\gamma = 0$ (left column), $\gamma = 0.3$ (middle column) and $\gamma = 0.7$ (right column). Other parameters are set as $\lambda = 0.001$, $\mu = 0.3$, $\sigma = 0.3$, $p = 0.3$.

Fig. 6 Epidemic size $\rho^I$ versus $\beta$ for different mobility rates $p$ when the initial population is homogeneously distributed (HOD). $a$ and $c$ the results for the migration route of ER-BA networks, while $b$ and $d$ the results for the migration route of BA-ER networks. The solid curves and the dots are the results by iterating Markovian equations (Eqs. 1–4) and by the Monte Carlo simulations, respectively. Each dot is the average results over more than 100 realizations. Other parameters are set as $\mu = 0.3$, $\sigma = 0.3$, $\kappa = 0.3$. The information efficiency over infection is $\gamma = 0.5$. (Color figure online)
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**Fig. 7** Epidemic size as a function of $p$ and $\beta$ with homogeneous initial population distribution (HOD). The heatmap represents the fraction of infected individuals at the steady state obtained from Monte Carlo simulations. The experimental conditions for the four phase diagrams are same as in Fig. 6. Each dot is the average results over 100 realizations.

**Fig. 8** Epidemic size $\rho^I$ versus for the epidemic transmission rate $\beta$ for different mobility rate $p$ under heterogeneous initial population distribution (HED). a and b The migration route is ER-BA; c and d the migration route is BA-ER. The curves represent the iteration results of Markovian equations, and the dots are obtained from Monte Carlo simulations. Each dot is the average results of over 100 independent simulations. Other parameters are the same as in Fig. 6.
epidemic dynamic (Fig. 6a), e.g., the epidemic threshold decreases with the increase in \( p \). This is because that a smaller \( \lambda \) is not conducive to information dissemination, resulting in more unaware individuals who migrate on the BA network with hub patches. The heterogeneous network structure of BA networks brings a more obvious effect of mobility on the epidemic, leading to a large gap between different curves (Fig. 6a). With the increase in \( \lambda \), more aware individuals migrate on ER networks, which further suppresses the epidemic spread with an increased epidemic threshold. Due to the homogeneous structure of ER network, mobility of aware individuals over ER networks delays the epidemic overall, while it has less effect on the epidemic, leading to a less gap between curves, see Fig. 6b. The effect of mobility rate \( p \) can be found in Fig. 7a, b.

When the migration route is BA-ER network, i.e., aware individuals migrate on BA networks. Whether the information propagation will help suppress or promote the epidemic spread depends on the individual’ mobility frequency. For instance, for a smaller mobility rate \( p = 0.3 \), the epidemic can be delayed by the introduction of information propagation. A higher information transmission rate will further delay the epidemic with a higher threshold as shown in Fig. 6c, d (blue curves). While when individuals migrate frequently (e.g., \( p = 1 \)), the acceleration of information dissemination will increase the number of aware individuals moving in the BA network, which further promotes the epidemic spread (Fig. 6d, e.g., orange curves). In all, although information dissemination partly suppresses the epidemic spread, frequent mobility of individuals still promotes the epidemic spread. Hence, appropriate intervention of mobile individuals can effectively help suppress the epidemic. Thus, the role of information dissemination has to be demonstrated with the mobility of individuals. More details can be found in Fig. 7c, d.

When the initial population distribution is heterogeneous (HED), the effect of mobility rate \( p \) on epidemic dynamic is illustrated in Fig. 8, where dots represent the results obtained for Monte Carlo simulations and the solid curves are the iteration results of the Markovian equations. Compared with the situation of HOD in Fig. 6, the heterogeneous population distribution (HED) promotes the epidemic spread with a lower epidemic threshold as shown in Fig. 8. In addition, we find that the epidemic threshold increases with mobility rate \( p \), that is, individual’s mobility delays the epidemic spread. This counterintuitive phenomenon is called detrimental effect as in Ref. [40]. Because the epidemic threshold is related to the maximum eigenvalue of matrix \( M \) (Eq.(14)), which can be solved with perturbation theory [43] and regarded as a quadratic equation with respect to \( p \) [40]. Therefore, there exists an optimal mobility rate \( p^* \) such that the maximum eigenvalue of \( M \) is minimized.

For the migration route of ER-BA (Fig. 8a, b), the impact of \( p \) is slightly different under various values of information transmission rate \( \lambda \). When \( \lambda \) is small, with the increase in \( p \), the epidemic threshold first increases and then decreases, while it increases monotonically for large \( \lambda \). The detailed analysis can be found in Fig. 9a, b. Similar to the analysis of Fig. 3, a higher information transmission rate \( \lambda \) will bring more aware individuals who migrate in ER network with homogeneous topology, which slows down the disease spread. Thus, it indicates that when the epidemic breaks out, guiding aware individuals to migrate over a more homogeneous route is able to avoid the gathering of individuals and helpful for the control of epidemic.

In addition, when the migration route is BA-ER (Fig. 8c, 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 in mobility rate \( p \) (Fig. 9c, d). It can be understood that aware individuals migrate on the BA network, both the heterogeneity of network structure and the heterogeneous population distribution will promote information dissemination, which needs a less \( \lambda \) to achieve the full dissemination of information. As a result, \( \lambda \) has less effect on epidemic with the value we tested in the simulation, which is consistent with the result in Fig. 3.

4 Conclusions and discussions

As a summary, we study the coupled interaction of epidemic spread and information propagation on the metapopulation network, and mainly explore how aware individuals’ behavioral change in migration route affects the epidemic spreading. We propose a two-layer multipopulation network model to reflect different migration routes that aware and unaware individuals take. Then, based on the transition tree, we build the dynamical model with the Markovian equations accordingly. The expression of the disease thresh-
old can be obtained by solving an eigenvalue problem. The high consistency between the iteration of Markovian 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 networks. Our results show that the information propagation leads to a larger epidemic threshold and a 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 found that the role of mobility in delaying or promoting the epidemic depends on the initial population distribution. In the case of the HOD, for a medium mobility rate, the information propagation will delay the epidemic spread on both the migration routes of ER-BA network and BA-ER network. Further frequent mobility of individuals will promote the epidemic spread with a reduced epidemic threshold, even though more individuals get aware the epidemic. The heterogeneous migration route of aware individuals further speeds up the epidemic spread. In the case of the HED, frequent mobility of individuals suppresses the epidemic with an increased epidemic threshold. In all, when studying the effect of information dissemination, one has to take both individuals’ mobility frequency and individuals’ (especially aware individuals’) migration route into account. Overall, our work points out a new way to explore factors such as the behavioral change in migration that may alternate the epidemic dynamics, and also provides guidance for the intervention in epidemic spread.

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Availability of data and material This work has no associated data.

Declaration

Conflict of interest The authors declare that they have no conflict of interest.

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