Rendezvous effects in the diffusion process on bipartite metapopulation networks

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(Received 12 May 2011; revised manuscript received 25 August 2011; published 31 October 2011; publisher error corrected 8 November 2011)

Epidemic outbreaks have been shown to be closely related to the rendezvous-induced transmission of infection, which is caused by casual contact with infected individuals in public gatherings. To investigate rendezvous effects in the spread of infectious diseases, we propose an epidemic model on metapopulation networks bipartite-divided into two sets of *location* and *rendezvous* nodes. At a given transition rate $\gamma_{kk'}^{p}$, each individual transfers from location k to rendezvous p (where rendezvous-induced disease incidence occurs) and thereafter moves to location k'. We find that the eigenstructure of a transition-rate-dependent matrix determines the epidemic threshold condition. Both analytical and numerical results show that rendezvous-induced transmission accelerates the progress of infectious diseases, implying the significance of outbreak control measures including prevention of public gatherings or decentralization of a large-scale rendezvous into downsized ones.

DOI: 10.1103/PhysRevE.84.041936 PACS number(s): 87.23.Ge, 87.19.X−

I. INTRODUCTION

In the last decade, extensive attention has been paid to epidemics and their mathematical spreading models [1–11]. Modern human societies have undergone more and more serious crises of large-scale outbreaks of communicable diseases. One of the causes of epidemic outbreaks can be a concentrated population participating in gathering activities in public places. There are particular risks of infection arising from casual contact with infected individuals through use of public transportation, travel to certain areas, or participation in gatherings. Examples can be found in studies of the worldwide spread of severe acute respiratory syndrome (SARS) in 2003 [12–14]. The SARS case had stringent outbreak control measures that ranged from distributing health alert notices to visitors and travelers in countries with SARS to canceling or postponing academic courses, business meetings, sporting events, and other public gatherings. In this paper, we address the issue of modeling and analyzing gathering processes of individuals and their role in the spread of infectious diseases, which we may term the *rendezvous effects*.

Recently, metapopulation models for understanding the spread of infectious diseases have been the topic of intensive investigation [15–19]. In these models, the entire population is demographically divided into different geographic regions, allowing migration of individuals between these subpopulations or sublocations. Thus in a metapopulation network, nodes represent subpopulations at discrete locations, and links represent individual diffusions between location nodes. To incorporate rendezvous effects of public gatherings into epidemic dynamics on metapopulation networks, we introduce *rendezvous nodes* to provide pathways via which casual contact (which spreads infections of the disease) occurs to those individuals passing through the same rendezvous node. We extendedly associate each link between location nodes with one of rendezvous nodes representing the rendezvous places for casual contact of individuals when diffusing between the sublocations. Therefore we can use a bipartite graph to describe the metapopulation structure since there is no allowed link that directly connects with location or rendezvous nodes in the network, as shown in Fig. 1. In this paper, we focus our attention on the role of rendezvous-induced transmission in the spread of infectious diseases; our results show that the presence of rendezvous effects in a densely concentrated population of individuals can dramatically alter the spreading behavior of epidemics.

The remainder of this paper is organized as follows. In Sec. II, we explain our basic assumptions on an extended epidemic model on metapopulation networks. In Sec. III, we provide a theoretical analysis of our model using the mean-field approximation. We find that the epidemic threshold, given by the basic reproductive number R_0, relies on the leading eigenvalue and/or eigenvector of the transition-rate-dependent matrices. In Sec. IV, we carry out extensive numerical simulations to verify the validity of our analytical prediction. Further, we numerically study the role of both the rendezvous node number and the heterogeneous size distribution of populations concentrated at these rendezvous nodes in the spread of epidemics. Finally, Sec. V concludes our study.

II. MODEL ASSUMPTION

Consider a standard susceptible-infected-susceptible (SIS) epidemiological model on metapopulation networks. Individuals can only exist in two discrete states: susceptible (S) or infected (I), and the spread of infections is formulated as a reaction-diffusion-decay process: $S + I \to 2I$, $I \to S$. We extend the metapopulation network by introducing rendezvous nodes to mimic public places for gathering activities of individuals. In our model, the following assumptions are made:

(A1) The transfer of individuals from different locations occurs at different transition rates but at synchronous time steps.

(A2) The metapopulation network is bipartite. Individuals cannot directly transfer between location nodes because geographic regions of different locations are discrete. Also,

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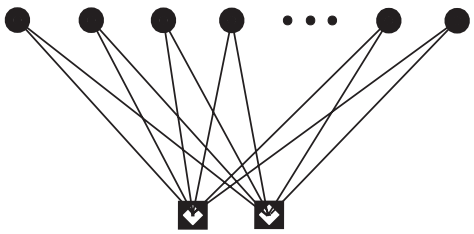

FIG. 1. An illustrative metapopulation structure described by a bipartite metapopulation network. Each individual in a location node (•) can transfer to another location through an associated rendezvous node (■); therein, one may make contact with other infected individuals, if any are present. This causes rendezvous-induced infection during the gathering process of individuals.

direct transfer between rendezvous nodes is disallowed, i.e., individuals leaving from a rendezvous node are assumed to be incapable of continuing to move on to another rendezvous node.

(A3) An individual can optionally stay at a location node without transfer to other locations, whereas an individual at a rendezvous node compulsively leaves from it at the next time step (that is, the individual transition rate of departure from a rendezvous place is a unit 1). This assumption arises from the simple fact that there are no nonmoving residents in public places.

For the gathering process, using Assumptions (A1)–(A3) we have set up bipartite metapopulation networks for an extended metapopulation epidemic model with rendezvous-induced infection. For the spreading process, the following assumptions about rendezvous-node-related epidemiological properties are made:

(A4) Only simultaneously incoming and outgoing individuals gathering at the same rendezvous node can make contact with each other and, therefore, have risk of rendezvous-induced infection.

(A5) Compared to locations, rendezvous nodes have a relatively high transmission rate. This assumption relies on the fact that rendezvous nodes in which gatherings of individuals take place are typically characterized by a high population density and/or a high population mobility and, therefore, the per capita contact rate is much higher than that of location nodes (where infection spreads via, for example, household contact of patients). Here we further assume a positive linear dependence of the transmission rate on the population density (or the individual number) at the rendezvous node for simplicity.

(A6) Rendezvous nodes have a nearly zero rate of recovery, since an individual's sojourning time at rendezvous nodes is negligibly short compared to a disease's recovery period, according to Assumption (A3).

Until now, we have specified the epidemic diffusion processes accompanied with rendezvous of individuals on metapopulation networks.

III. ANALYTICAL RESULTS

A. Population dynamics

We first consider the population dynamics on bipartite metapopulation networks composed of n location nodes (labeled by the subscript $k = 1,2,\ldots,n$) and m rendezvous nodes (labeled by the superscript $p = 1,2,\ldots,m$). Introducing the transition matrix with respect to rendezvous p by $\mathbf{\Gamma}^p = (\gamma^p_{kk'})_{n\times n}$ whose entries denote the transition rates of individuals transferring from location k to location k' via rendezvous p, we obtain the population dynamics as follows:

$$\begin{aligned}\frac{d}{dt}x_k &= \sum_{p=1}^{m}\left(-x_k\gamma^p_{k,\circ} + \gamma^p_{\circ,k}y^p\right),\\ \frac{d}{dt}y^p &= \sum_{k=1}^{n}\left(x_k\gamma^p_{k,\circ} - \gamma^p_{\circ,k}y^p\right),\end{aligned} \tag{1}$$

where x_k and y^p denote the population sizes at location k and rendezvous p, respectively, $\gamma^p_{k,\circ} = \sum_{k'}\gamma^p_{kk'}$ is the transition rate of individuals at location k leaving for rendezvous p, and $\gamma^p_{\circ,k}$ is the transition rate of individuals at rendezvous p moving to location k. More precisely,

$$\gamma^p_{\circ,k} = \frac{\sum_{k'=1}^{n} x_{k'}\gamma^p_{k'k}}{\sum_{k'=1}^{n} x_{k'}\gamma^p_{k',\circ}}, \tag{2}$$

where according to Assumption (A3), $\gamma^p_{\circ,k}$ is a posterior probability derived from the Bayesian rule which satisfies the normalization condition $\sum_{k'}\gamma^p_{\circ,k'} = 1$ for every rendezvous p.

When the population distribution in the network reaches a steady state, the stationary metapopulation size is given by $\bar{x}_k = \sum_p \bar{\gamma}^p_{\circ,k}\bar{y}^p / \sum_p \gamma^p_{k,\circ}$ and $\bar{y}^p = \sum_k \bar{x}_k\gamma^p_{k,\circ}$, where $\bar{\gamma}^p_{\circ,k} = \sum_{k'}\bar{x}_{k'}\gamma^p_{k'k}/\bar{y}^p$. Eliminating $\bar{y}^p$ yields a set of linear equations with stationary population sizes $\bar{x}_k$ in n locations; rewriting in matrix form gives

$$\bar{\mathbf{x}} = \mathbf{A}\bar{\mathbf{x}}, \tag{3}$$

where $\bar{\mathbf{x}} = (\bar{x}_1,\ldots,\bar{x}_n)'$ [here $(\cdot)'$ denotes the transpose of the matrix] and the coefficient matrix $\mathbf{A}$ is given by

$$\mathbf{A}_{n\times n} = (a_{ij}) = \frac{\sum_{p=1}^{m}\gamma^p_{ji}}{\sum_{p=1}^{m}\sum_{k=1}^{n}\gamma^p_{ik}}. \tag{4}$$

Therefore, population dynamics in Eq. (1) converges to a stationary state $\bar{\mathbf{x}}$ proportional to $\mathbf{A}$'s leading eigenvector, and thus,

$$\bar{\mathbf{y}} = \mathbf{T}\bar{\mathbf{x}}, \tag{5}$$

where $\mathbf{T}_{m\times n} = (\gamma^p_{k,\circ})$. The stationary population distribution in the network is given by Eqs. (3) and (5) with the normalization condition $\sum_k \bar{x}_k + \sum_p \bar{y}^p = N$, where N is the population size on the entire metapopulation network.

Remarks. To interpret the coefficient matrix $\mathbf{A}$, we rewrite Eq. (4) in the form $\mathbf{A} = \mathbf{D}^{-1}\mathbf{\Gamma}'$ with $\mathbf{\Gamma} = \sum_p \mathbf{\Gamma}^p$, and $\mathbf{D} = \mathrm{diag}(\sum_p \gamma^p_{k,\circ})$.[1] Note that the entry in the ith row and the jth column of $\mathbf{\Gamma}'$ denotes the total transition rate at which individuals leaving location j arrive at location i via

[1] We assume that all diagonal elements of $\mathbf{D}$ are positive; that is, we do not take account of isolated metapopulations of individuals who never participate in any gatherings; the transition rate of individuals leaving location k for (at least one) rendezvous p is above zero, $\gamma^p_{k,\circ} > 0$.

any one of the rendezvous nodes; the ith diagonal entry of $\mathbf{D}$ denotes the total transition rate at which individuals leave location i. Therefore, we obtain from Eq. (3) that $\mathbf{\Gamma}'\bar{\mathbf{x}} = \mathbf{D}\bar{\mathbf{x}}$, implying a balanced population of immigration ($=\mathbf{\Gamma}'\bar{\mathbf{x}}$) and emigration ($=\mathbf{D}\bar{\mathbf{x}}$) in each location under a stationary population distribution.

B. Epidemic dynamics

Next we consider the process of epidemic diffusion. Thus the subpopulation at each location and rendezvous node is divided into two compartments: susceptible and infected. Under the mean-field approximation we have the following rate equations for epidemic dynamics:

$$\begin{aligned}
\frac{d}{dt}s_k &= -\alpha_k \frac{s_k i_k}{s_k + i_k} + \beta_k i_k + \sum_{p=1}^{m} \left(-s_k \gamma_{k,\circ}^{p} + \gamma_{\circ,k}^{p} s^p\right),\\
\frac{d}{dt}i_k &= \alpha_k \frac{s_k i_k}{s_k + i_k} - \beta_k i_k + \sum_{p=1}^{m} \left(-i_k \gamma_{k,\circ}^{p} + \gamma_{\circ,k}^{p} i^p\right),\\
\frac{d}{dt}s^p &= -\alpha^p \frac{s^p i^p}{s^p + i^p} + \sum_{k=1}^{n} \left(s_k \gamma_{k,\circ}^{p} - \gamma_{\circ,k}^{p} s^p\right),\\
\frac{d}{dt}i^p &= \alpha^p \frac{s^p i^p}{s^p + i^p} + \sum_{k=1}^{n} \left(i_k \gamma_{k,\circ}^{p} - \gamma_{\circ,k}^{p} i^p\right),
\end{aligned} \tag{6}$$

where s_k,i_k and s^p,i^p are the numbers of susceptible and infected individuals at location k and at rendezvous p, respectively; α_k and β_k are the transmission and recovery rates at location k; α^p is the transmission rate at rendezvous p [here we set the recovery rate at rendezvous nodes $\beta^p = 0$ under Assumption (A6)]; $\gamma_{k,\circ}^{p}$ and $\gamma_{\circ,k}^{p}$, defined the same as in the population dynamics Eq. (1), are given by

$$\gamma_{k,\circ}^{p} = \sum_{k'=1}^{n} \gamma_{kk'}^{p}, \quad \gamma_{\circ,k}^{p} = \frac{\sum_{k'=1}^{n} (s_{k'} + i_{k'})\gamma_{k'k}^{p}}{\sum_{k'=1}^{n} (s_{k'} + i_{k'})\gamma_{k',\circ}^{p}}. \tag{7}$$

Note that α_k, β_k, and $\gamma_{k,\circ}^{p}$ appear as constant parameters, and that α^p and $\gamma_{\circ,k}^{p}$ are state-dependent parameters in our model.

Under the assumption that the recovery period of diseases is much longer than the duration of individuals' transitions between locations and rendezvous nodes ($1/\beta_k \gg 1$), the population dynamics quickly evolves to the stationary state. Therefore, we shall essentially consider the epidemic dynamics on the network with a stationary population distribution, $(\bar{\mathbf{x}},\bar{\mathbf{y}})$, in which the transition rate $\bar{\gamma}_{\circ,k}^{p} = \sum_{k'} \bar{x}_{k'} \gamma_{k'k}^{p} / \bar{y}^p$ at which individuals at rendezvous p transfer to location k, as well as the rendezvous-induced transmission rate $\bar{\alpha}^p$ under $(\bar{\mathbf{x}},\bar{\mathbf{y}})$, are independent of system states.

Therefore, substituting $\bar{x}_k$, $\bar{y}^p$, $\bar{\alpha}^p$, and $\bar{\gamma}_{\circ,k}^{p}$ into Eq. (6) yields

$$\begin{aligned}
\frac{d}{dt}i_k &= \alpha_k i_k (1 - i_k/\bar{x}_k) - \beta_k i_k + \sum_{p=1}^{m} \left(-i_k \gamma_{k,\circ}^{p} + \bar{\gamma}_{\circ,k}^{p} i^p\right),\\
\frac{d}{dt}i^p &= \bar{\alpha}^p i^p (1 - i^p/\bar{y}^p) + \sum_{k=1}^{n} \left(i_k \gamma_{k,\circ}^{p} - \bar{\gamma}_{\circ,k}^{p} i^p\right),
\end{aligned} \tag{8}$$

where the equations with respect to s_k and s^p are omitted in the simplified rate equations, because the normalization conditions $s_k + i_k = \bar{x}_k$ and $s^p + i^p = \bar{y}^p$ hold in a stationary population distribution.

Here, we focus our attention on the epidemic threshold condition related to the stability of the disease-free equilibrium (DFE), $\bar{i}_k = \bar{i}^p = 0$. The epidemic threshold is given by the basic reproductive number R_0, which determines whether an initial outbreak of diseases will die out quickly ($R_0 < 1$), or survive longer and spread out appreciably to cover a population of a considerable size ($R_0 > 1$). The biological interpretation of R_0 is the expected number of secondary cases produced by one infected individual during his entire infectious period in a completely susceptible population [20–22].

To estimate the basic reproductive number R_0, consider the number i_0 ($\ll N$) of infected individuals initially introduced into a completely susceptible population. The infected individuals cause $\alpha_k i_0$ new cases if they are placed at location k with probability $\bar{x}_k/N$, and similarly, they cause $\bar{\alpha}^p i_0$ new cases if being posited at rendezvous p with probability $\bar{y}^p/N$. On the other hand, $\beta_k i_0$ infectives decrease because of recovery with probability $\bar{x}_k/N$ for location k, and $\beta^p i_0$ infectives recover to susceptibles with probability $\bar{y}^p/N$ for rendezvous p. Thus, an estimate of $\hat{R}_0$ can be obtained using the effective spreading rate, the ratio of the increase and decrease of the infectives as

$$\hat{R}_0 = \frac{\sum_k \alpha_k i_0 \bar{x}_k/N + \sum_p \bar{\alpha}^p i_0 \bar{y}^p/N}{\sum_k \beta_k i_0 \bar{x}_k/N + \sum_p \beta^p i_0 \bar{y}^p/N},$$

and eliminating i_0 and N yields

$$\hat{R}_0 = \frac{\sum_k \alpha_k \bar{x}_k + \sum_p \bar{\alpha}^p \bar{y}^p}{\sum_k \beta_k \bar{x}_k + \sum_p \beta^p \bar{y}^p}, \tag{9}$$

showing that R_0 is linearly dependent on both transmission rates α_k and $\bar{\alpha}^p$ at location and rendezvous nodes, respectively.

Remarks. Equation (9) implies that rendezvous effects (induced by additional infectious contact at rendezvous nodes that transmits infection) may cause epidemic outbreaks, $R_0 > 1$, even if infectious diseases will be eradicated without the presence of rendezvous effects (i.e., $\alpha_k/\beta_k < 1$ holds for every location k). A similar result is reported in Refs. [23,24].

More precisely, R_0 can be obtained by using the next generation matrix $\mathbf{F}\mathbf{V}^{-1}$ of Eq. (8), the spectral radius (the dominant eigenvalue) of which defines the basic reproductive number [16,25]

$$R_0 = \rho(\mathbf{F}\mathbf{V}^{-1}). \tag{10}$$

Here the matrix $\mathbf{F}$ is given by

$$\mathbf{F} = \left[\begin{array}{cc} \frac{\partial F_k}{\partial i_k} & \frac{\partial F_k}{\partial i^p} \\ \frac{\partial F^p}{\partial i_k} & \frac{\partial F^p}{\partial i^p} \end{array}\right]\Bigg|_{\mathrm{DFE}} = \left[\begin{array}{ccc|ccc} \alpha_1 & \cdots & 0 & & & \\ \vdots & \ddots & \vdots & & \mathbf{0} & \\ 0 & \cdots & \alpha_n & & & \\ \hline & & & \bar{\alpha}^1 & \cdots & 0 \\ & \mathbf{0} & & \vdots & \ddots & \vdots \\ & & & 0 & \cdots & \bar{\alpha}^m \end{array}\right], \tag{11}$$

where F_k (F^p) is the rate of appearance of new infections at location k (rendezvous p) due to casual contact with infected

individuals. And the matrix $\mathbf{V}$ is given by

$$\mathbf{V} = \left[\begin{array}{cc} \frac{\partial V_k}{\partial i_k} & \frac{\partial V_k}{\partial i^p} \\ \frac{\partial V^p}{\partial i_k} & \frac{\partial V^p}{\partial i^p} \end{array}\right]\Bigg|_{\mathrm{DFE}} = \left[\begin{array}{cccccc} \beta_1 + \sum_{p=1}^{m}\gamma_{1,\circ}^{p} & \cdots & 0 & -\bar{\gamma}_{\circ,1}^{1} & \cdots & -\bar{\gamma}_{\circ,1}^{m} \\ \vdots & \ddots & \vdots & \vdots & \ddots & \vdots \\ 0 & \cdots & \beta_n + \sum_{p=1}^{m}\gamma_{n,\circ}^{p} & -\bar{\gamma}_{\circ,n}^{1} & \cdots & -\bar{\gamma}_{\circ,n}^{m} \\ -\gamma_{1,\circ}^{1} & \cdots & -\gamma_{n,\circ}^{1} & \sum_{k=1}^{n}\bar{\gamma}_{\circ,k}^{1} & \cdots & 0 \\ \vdots & \ddots & \vdots & \vdots & \ddots & \vdots \\ -\gamma_{1,\circ}^{m} & \cdots & -\gamma_{n,\circ}^{m} & 0 & \cdots & \sum_{k=1}^{n}\bar{\gamma}_{\circ,k}^{m} \end{array}\right] \tag{12}$$

where $V_k = V_k^- - V_k^+$, and V_k^- (V_k^+) is the rate of diffusion of infected individuals out of (into) location k. Note that V_k^- includes the recovery process, $I \to S$, which also causes the decrease in the number of infectives. And V^p for every rendezvous p can be similarly defined. Both matrices $\mathbf{F}$ and $\mathbf{V}$ are evaluated at $\bar{i}_k = \bar{i}^p = 0$, related to the disease-free equilibrium.

On the other hand, by linearizing dynamics Eq. (8) around the stationary state $(\bar{i}_1, \ldots, \bar{i}_n, \bar{i}^1, \ldots, \bar{i}^p)$ we calculate the Jacobian matrix $\mathbf{J}$ which is given by

$$\mathbf{J}|_{(\bar{i}_1,\ldots,\bar{i}_n,\,\bar{i}^1,\ldots,\bar{i}^p)} = \left[\begin{array}{cccccc} \left(1-\frac{2\bar{i}_1}{\bar{x}_1}\right)\alpha_1 - \beta_1 - \sum_{p=1}^{m}\gamma_{1,\circ}^{p} & \cdots & 0 & \bar{\gamma}_{\circ,1}^{1} & \cdots & \bar{\gamma}_{\circ,1}^{m} \\ \vdots & \ddots & \vdots & \vdots & \ddots & \vdots \\ 0 & \cdots & \left(1-\frac{2\bar{i}_n}{\bar{x}_n}\right)\alpha_n - \beta_n - \sum_{p=1}^{m}\gamma_{n,\circ}^{p} & \bar{\gamma}_{\circ,n}^{1} & \cdots & \bar{\gamma}_{\circ,n}^{m} \\ \gamma_{1,\circ}^{1} & \cdots & \gamma_{n,\circ}^{1} & \left(1-\frac{2\bar{i}^1}{\bar{y}^1}\right)\bar{\alpha}^1 - \sum_{k=1}^{n}\bar{\gamma}_{\circ,k}^{1} & \cdots & 0 \\ \vdots & \ddots & \vdots & \vdots & \ddots & \vdots \\ \gamma_{1,\circ}^{m} & \cdots & \gamma_{n,\circ}^{m} & 0 & \cdots & \left(1-\frac{2\bar{i}^m}{\bar{y}_m}\right)\bar{\alpha}^m - \sum_{k=1}^{n}\bar{\gamma}_{\circ,k}^{m} \end{array}\right]. \tag{13}$$

Thus the stationary state is locally asymptotically stable if all of the eigenvalues of $\mathbf{J}$ have a negative real part. Therefore the epidemic threshold specified by $R_0 < 1$ is equivalent to the stability of the epidemic dynamics locally linearized at the DFE, noting that Jacobian matrix $\mathbf{J}$, evaluated at the origin $\bar{i}_k = \bar{i}^p = 0$, satisfies

$$\mathbf{J}|_{\mathrm{DFE}} = \mathbf{F} - \mathbf{V}, \tag{14}$$

where $\mathbf{F}$ and $\mathbf{V}$ are the same as in the next generation matrix.

Remarks. It suffices to guarantee the outbreak of a disease if at least one of the following k conditions holds: $\alpha_k - \beta_k > 2\sum_p \gamma_{k,\circ}^p$ (implying an increase rate of infected individuals greater than twice the total transition rate for location k). In this case, it directly follows from the Gershgorin disk theorem that the Jacobian matrix Eq. (13) has at least one eigenvalue with a positive real part [26]. This also implies that the disease cannot be eliminated if the spreading rate in this metapopulation is sufficiently high, relative to their population-mobility-related properties, even if rendezvous nodes have nonzero recovery rates ($\beta^p > 0$).

IV. NUMERICAL SIMULATIONS

In this section, we perform agent-based computer simulations to mimic diffusion processes of individuals. Initially, all the individuals of a population of size $N = 10^4$ are uniformly distributed in the location nodes, and a number 10 (=0.1%) of the initially infected individuals are randomly dispersed in the population. At each time step, individuals at every location and rendezvous node successively undergo the spread processes $S + I \to 2I$ and the recovery process $I \to S$ to update their states according to epidemiological parameters of the location or rendezvous node, and then the state-updated individuals select to transfer to a next rendezvous or location node at the corresponding transition rate according to the basic Assumptions (A1)–(A3) about gathering processes in

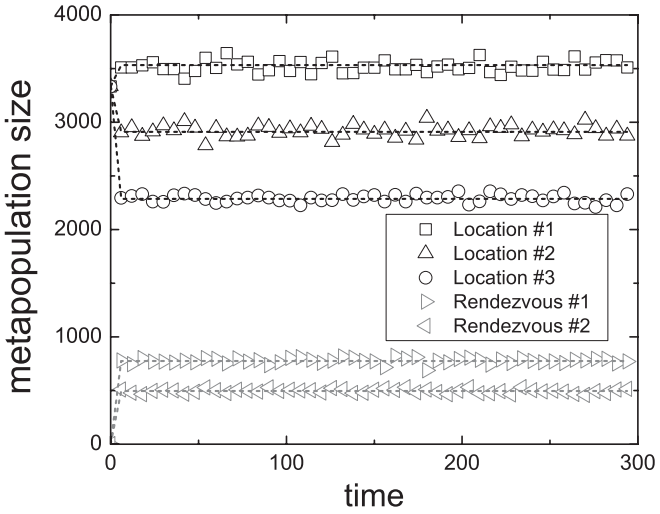

FIG. 2. Time course of the metapopulation size in each location and rendezvous during the epidemic spreading process on a bipartite network composed of $n = 3$ locations and $m = 2$ rendezvous nodes. This figure shows that simulation results agree well with the analytical prediction (dotted line) derived from the population dynamics of Eq. (1), and that the stationary population sizes in location nodes are proportional to the leading eigenvector $(\frac{17}{42}, \frac{1}{3}, \frac{11}{42})'$ of matrix **A** defined by Eq. (4), where the transition rate matrices via both rendezvous nodes are respectively set as $\Gamma^1 = \begin{pmatrix} 0.05 & 0.03 & 0.02 \\ 0.03 & 0.04 & 0.02 \\ 0.02 & 0.03 & 0.02 \end{pmatrix}$ and $\Gamma^2 = \begin{pmatrix} 0.03 & 0.01 & 0.02 \\ 0.02 & 0.02 & 0.01 \\ 0.04 & 0.01 & 0.01 \end{pmatrix}$.

our model. To confirm analytical predictions of the rate equations under the mean-field approximation, we first carry out simulations with a given set of model parameters, as shown in Figs. 2 and 3. The simulation results agree well

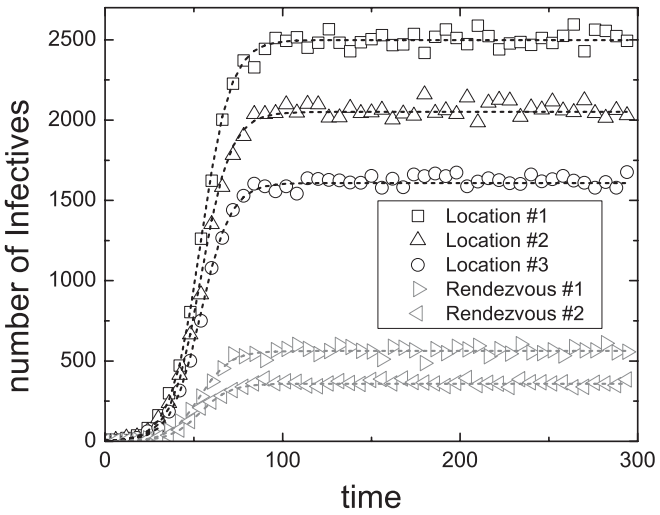

FIG. 3. Time course of the infective individual number at each location and at each rendezvous during the epidemic spreading process. The epidemiological parameters are set at $\alpha_k = 0.1$, $\beta_k = 0.05$ (homogeneously for all location nodes), and $\bar{\alpha}^p = 0.5$ (under the stationary population of both rendezvous nodes), and other parameters are set the same as those in Fig. 2. Simulation results agree well with the analytical prediction (dotted line) derived from epidemic dynamics of Eq. (6).

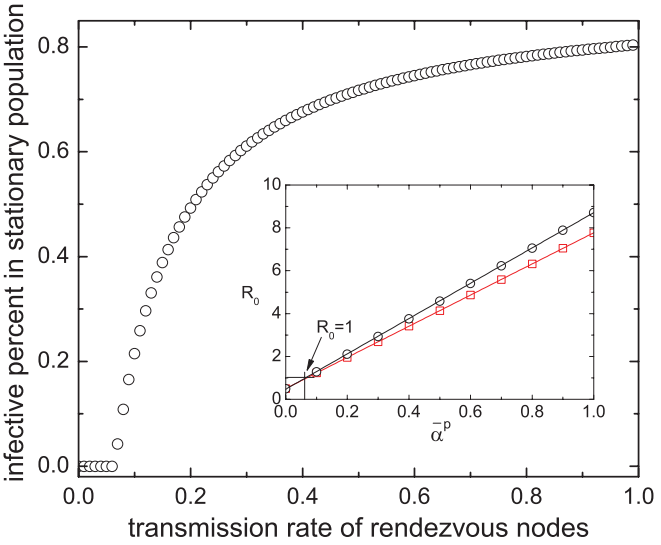

FIG. 4. (Color online) Percent of infective individuals in the stationary population $i(\infty)$ as an increasing function of the rendezvous-induced transmission rate $\bar{\alpha}^p$. The epidemic critical point separating the disease-free phase $[i(\infty) = 0]$ from the endemic phase $[i(\infty) > 0]$ corresponds to the condition $R_0 = 1$. The inset plots the basic reproductive number R_0 (circles) obtained using the next generation matrix Eq. (10), and its estimate $\hat{R}_0$ (boxes) given by Eq. (9). The epidemiological parameters are set at $\alpha_k = 0.1$ and $\beta_k = 0.2$ for all location nodes; other parameters are set the same as those in Fig. 2.

with the predictions given by population dynamics of Eq. (1) and epidemic dynamics of Eq. (6).

To visualize rendezvous effects in epidemic spreading, we study a population of individuals in which each location has a stable DFE (i.e., $\alpha_k < \beta_k$); we find that a sufficiently large rate $\bar{\alpha}^p$ of transmission occurrence that accompanies gathering processes of individuals can greatly affect the epidemic dynamics and even result in an endemic state in the entire population [the percent of infected individuals in the stationary population $i(\infty) > 0$], as shown in Fig. 4. The inset of Fig. 4 plots the basic reproductive number R_0; the critical point defined by $R_0 = 1$ gives the epidemic threshold that separates a disease-free state and an endemic state for the system.

Figure 5 plots the color-coded $i(\infty)$ and R_0 values versus the relative transmission rates in location and rendezvous nodes; this provides a more comprehensive view of effects of epidemiological parameters on the spread of infectious diseases. In particular, Fig. 5(b) shows that R_0 linearly depends (approximately) on both α_k and $\bar{\alpha}^p$, as given by Eq. (9).

Next, we study the role of transition rate matrices $\mathbf{\Gamma}^p$ in the spread of infectious diseases. These matrices determine the (stationary) number of individuals involved in rendezvous-induced contacts during gathering processes, $\bar{\mathbf{y}}$, and hence, affect the epidemiological parameter α^p, which relates to the rendezvous-induced transmission. According to Assumption (A6), we assume, for simplicity, a linear dependence of α^p on its metapopulation size in the form of $\alpha^p = \kappa y^p / N$, where κ is a positive parameter. We first observe the basic reproductive number as the number m of rendezvous nodes

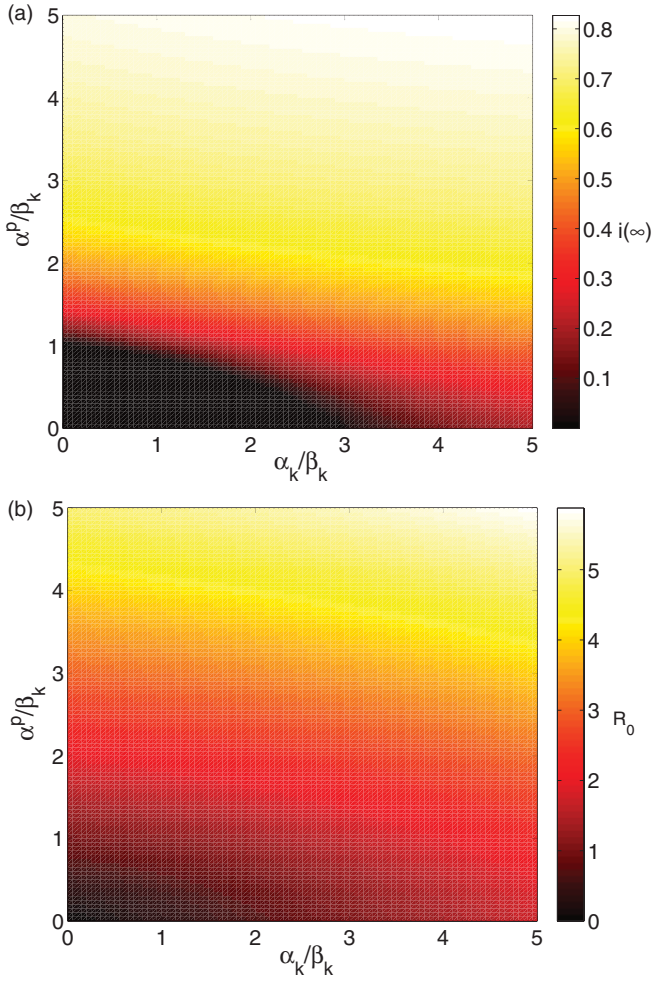

FIG. 5. (Color online) (a) Stationary infective percent $i(\infty)$ for various transmission rates α_k and $\bar{\alpha}^p$ relative to the recovery rate β_k. Here, α_k, β_k, and $\bar{\alpha}^p$ are homogeneously set for all location and rendezvous nodes. (b) Color-coded plot of the analytically predicted basic reproductive number R_0 value Eq. (10) as a function of relative transmission rates in location and rendezvous nodes. Other parameters are set the same as those in Fig. 2.

varies. Randomly splitting a given transition rate matrix $\mathbf{\Gamma}$ into m non-negative matrices $\mathbf{\Gamma}^p$ yields a group of transition rate matrices for all rendezvous nodes ($\sum_p \mathbf{\Gamma}^p = \mathbf{\Gamma}$),[2] and hence under one such set of parameters, R_0 can be analytically given by the next generation matrix Eq. (10); the final infective percent in the stationary population $i(\infty)$ can be obtained by carrying out agent-based simulations. We vary the number of rendezvous nodes, fixing the total transition matrix in the form of $\mathbf{\Gamma} = t\mathbf{1}/n$, where $\mathbf{1}$ denotes the matrix for which each entry is 1, and t denotes the probability of individuals participating in gatherings per unit time. Figure 6 shows that the rendezvous

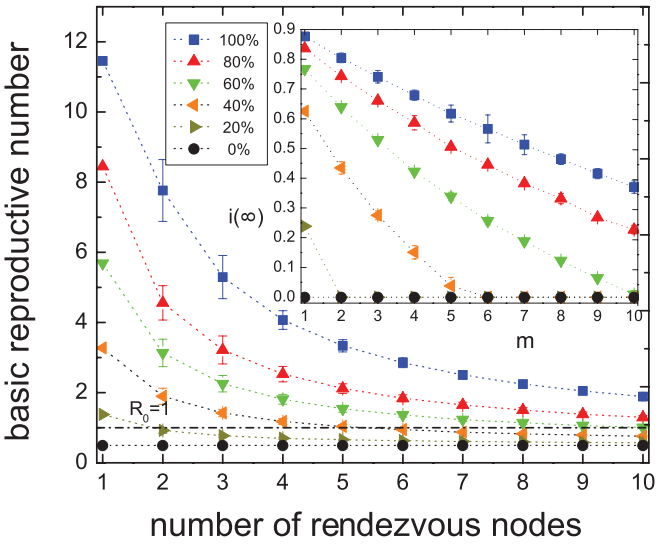

FIG. 6. (Color online) Basic reproductive number R_0 and the stationary infective percent $i(\infty)$ (inset) as decreasing functions of the number m of rendezvous nodes. Each line corresponds to a different transition matrix $\mathbf{\Gamma} = t\mathbf{1}/n$; the rendezvous-induced transmission rate $\alpha^p = \kappa y^p/N$ with the proportionality factor $\kappa = 2$ (see text). Other parameters are set as follows: $N = 10^4$, $n = 3$, $\alpha_k = 0.05$, and $\beta_k = 0.1$. Each data point is obtained by averaging over 100 independent realizations; the length of the bar indicates the maximum and minimum values.

effect is reduced as the number of rendezvous nodes increases (accordingly, the concentrated populations in these rendezvous nodes are reduced in size), which is of benefit to the outbreak control of infectious diseases.

We further study the role of heterogeneity of rendezvous sizes in the epidemic spreading process. Many real networks, unlike homogeneous or structureless ones, usually exhibit the scale-free feature with heterogeneous degree distribution which typically obeys a power law, $P(k) \sim k^{-\gamma}$ [27–29]. To generate heterogeneous size distribution of populations gathered at rendezvous nodes, we consider that transition rates for different rendezvous nodes satisfy $\gamma^p_{kk'} \sim p^{-\delta}$.[3] Figure 7 plots the basic reproductive number R_0 as a function of parameter δ, which shows that heterogeneously distributed $\bar{y}^p$ usually forms a few densely populated, hublike rendezvous nodes (as shown in the inset of Fig. 7), and thus, induces rendezvous effects, which greatly accelerate the progress of infectious diseases.

[2]For convenience of comparison, we fix the total transition rate matrix $\mathbf{\Gamma}$, and thus the stationary population distribution $\bar{\mathbf{x}}$ in locations is proportional to the leading eigenvalue of matrix $\mathbf{A}$ of Eq. (4), which solely depends on $\mathbf{\Gamma}$. Therefore the total number of individuals who are temporally located in all rendezvous nodes and participating in gatherings keeps invariant given a fixed $\mathbf{\Gamma}$.

[3]The construction of the power-law distributed $\lambda^p_{kk'}$ is as follows: First randomly split $\mathbf{\Gamma}$ into transition rate matrices $\mathbf{\Gamma}^p$ for each rendezvous node; then by weighing factor $p^{-\delta}$ for the transition rate matrix $\mathbf{\Gamma}^p$ of rendezvous p, the randomly split $\mathbf{\Gamma}^p$ is rescaled to $ap^{-\delta}\mathbf{\Gamma}^p$, where a is a normalization constant obtained by $a = \sum_p \bar{y}^p / \sum_p p^{-\delta}\bar{y}^p$, where $\bar{y}^p$ is the stationary population size at rendezvous p and is given by Eq. (5). As a result of the heterogeneous distribution of transition rates $\mathbf{\Gamma}^p$, a few rendezvous (with smallest superscripts p) act as hub nodes that are selected by most individuals for their rendezvous places.

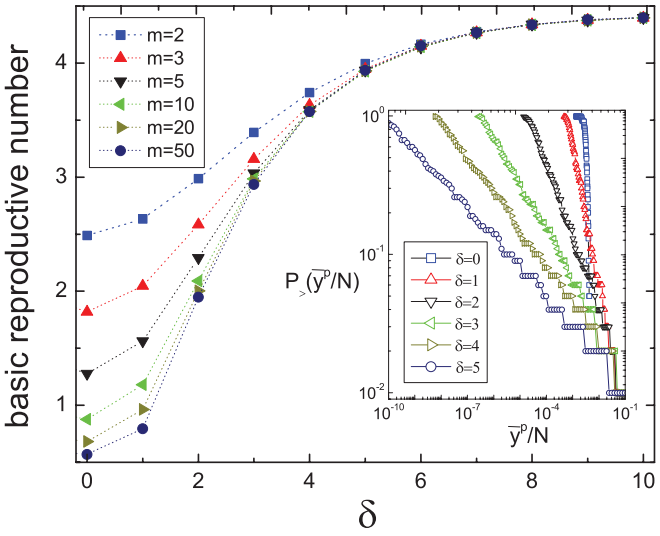

FIG. 7. (Color online) Basic reproductive number R_0 as a function of the exponent δ under different rendezvous numbers m. We adopt $t = 50\%$ and $\gamma_{kk'}^{p} \sim p^{-\delta}$ (see Footnote 3); other parameters are set the same as those in Fig. 6. Each data point is obtained by averaging over 100 independent realizations. The inset shows the accumulative distribution $P_{>}(y)$ of (relative) population size $\bar{y}^p/N$ at rendezvous nodes in a logarithmic plot, implying a power-law distributed population size at rendezvous nodes. One can verify that the distribution function reads $P(y) \sim y^{-\gamma}$ with the power-law exponent $\gamma \approx 1 + 1/\delta$.

V. CONCLUSION

In summary, we have proposed an extended metapopulation epidemic model which includes migration of individuals between discrete locations. In our model, we consider gathering and rendezvous processes of individuals, where additional transmission of infectious diseases occurs due to casual contact at rendezvous nodes of the network during diffusion processes of individuals. We have both analytically and numerically evaluated the rendezvous effects in the spread of infectious diseases, showing that a highly concentrated population at rendezvous nodes can importantly affect the dynamic spreading behavior of metapopulation networks.

In particular, we also find that a heterogeneously distributed population size at rendezvous nodes can enhance rendezvous effects and accelerate the spreading of epidemics greatly. Our results are consistent with the basic finding by Eubank *et al.* [30] at the level of contact networks whose nodes represent individuals and whose links represent pairwise contacts between individuals. Here we have quantitatively obtained similar results but related to the diffusion model defined at the metapopulation level, in which network nodes represent subpopulations or locations, and network links represent individual transitions between metapopulations.

In this paper, we have made several assumptions. For example, the recovery rate at rendezvous nodes is assumed to be zero ($\beta^p = 0$). This condition can be relaxed, assuming that some rendezvous nodes are associated with a relatively high recovery rate to mimic places like hospitals, in which case Eqs. (9) and (10) still hold for the estimation or calculation of the basic reproductive number R_0. To make a population-level analysis tractable, we have also considered the assumptions that both susceptible and infected individuals have the same diffusion rate, and that individuals' sojourning times at rendezvous places are negligible in our model. There are many other conceivable rules of rendezvous processes, which provide a new possibility for altering the dynamical behavior of both physical and social systems by partly coupling a small portion of the system's components. These issues deserve further study.

ACKNOWLEDGMENTS

L.C. and X.L. thank Lin Wang and Xiang Li for their helpful discussions and valuable suggestions. This research is partially supported by the Japan Society for the Promotion of Science (JSPS) through the "Funding Program for World-Leading Innovative R&D on Science and Technology (FIRST Program)," initiated by the Council for Science and Technology Policy (CSTP) (K.A.), and also through a Grant-in-Aid for JSPS Fellows (21-09275, B.W.).

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