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Spatial spread of an epidemic through public transportation systems with a hub

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A B S T R A C T

This article investigates an epidemic spreading among several locations through a transportation system, with a hub connecting these locations. Public transportation is not only a bridge through which infections travel from one location to another but also a place where infections occur since individuals are typically in close proximity to each other due to the limited space in these systems. A mathematical model is constructed to study the spread of an infectious disease through such systems. A variant of the next-generation method is proposed and used to provide upper and lower bounds of the basic reproduction number for the model. Our investigation indicates that increasing transportation efficiency, and improving sanitation and ventilation of the public transportation system decrease the chance of an outbreak occurring. Moreover, discouraging unnecessary travel during an epidemic also decreases the chance of an outbreak. However, reducing travel by infectives while allowing susceptibles to travel may not be enough to avoid an outbreak.

1. Introduction

Inter-regional public transportation systems link all major regions on Earth. These systems make it possible for people to travel around the world and, at the same time, serve as a network through which an epidemic can spread worldwide. As indicated by recent epidemic outbreaks, such as SARS and H1N1, a disease can very quickly spread to distant locations through this network. Controlling this mode of disease spread is essential.

In order to understand the spatial spreading of an epidemic, researchers have analyzed related mathematical models. For example, Sattenspiel and Dietz [18] investigated the spread of infectious disease among discrete geographical regions with the assumption that people travel between them. Using demographic analysis adapted from [18], Arino and van den Driessche proposed a multi-city SIS epidemic model and found explicit bounds on the basic reproduction number $R_0$ [2]. Hyman and LaForce modeled the disease spread among multiple cities using a system of deterministic differential equations and analyzed the reproductive number and the sensitivity of the model [12]. Numerical simulation results of the model were also compared with the CDC influenza data. An n-patch SIS model with immigration of susceptible and infective individuals was proposed by Wang and Zhao [22] and a threshold which determines the persistence and extinction of the epidemic was obtained. This model was further investigated by Jin and Wang [13], and Zhang and Zhao [25]; the latter assumes that some coefficients in the model proposed in [22] are periodic with a common period. Recently, Yang et al. [24] studied both SIR and SIS epidemic models with bilinear incidence and migration of susceptible and infective individuals. These studies investigated the spatial spread of different kinds of diseases among two or more locations.

The role played by the transportation system in an epidemic was investigated by Castillo-Chavez et al. [6], who modelled the dynamics of smallpox and its control under the assumption that the disease is deliberately released in a large city with a mass transportation system. Cui et al. [8] proposed an SIS epidemic model to investigate disease transmission between two regions with infection during travel. This model was further studied by Takeuchi et al. [19], Using an SIQS model, Liu and Takeuchi [16] investigated the spread of disease with transport-related infection and entry screening. Wan and Cui [21], on the other hand, proposed an SEIS epidemic model to study the effect of transport-related infection on the spread and control of disease spreading. The effect of transport-related infection was also studied by Liu and Zhou [15] using an SIRS model.

The worldwide airline network consists of nodes (e.g. local airports) and hubs (e.g. airline hubs) [1,5,23]. Each hub in this network has direct connections to several nodes. If there are no direct connections between these nodes, passengers travelling from one node to another must go through a transportation hub.
Some airlines have only one hub while others have multiple hubs. A hub, its adjacent nodes and connecting edges constitute a sub-system of the world-wide transportation network. Such a transportation system can be modeled by a hub-and-spoke paradigm [1,5].

A transportation system with a single hub can be considered as a sub-system of the world-wide transportation network. We consider a disease spreading between multiple locations (nodes) through a transportation system with a single hub, ignoring any connections between this sub-system and the rest of the global transportation system. Understanding how the epidemic spreads through this sub-system is fundamental to studying the disease spread through the world-wide transportation network. In order to appreciate the effect that the transportation system may have on the epidemic spread, we also ignore any other transportation connections between these locations (nodes) and the hub (in particular, we assume there is no direct connection between any two nodes). Here we consider an SIRS disease model. That is, for each location, the population is divided into susceptibles, infectives and recovereds. Modifications based on other standard epidemiological factors (e.g. exposed classes) or the effect of specific travel routes can be incorporated into the model, but that lies beyond the scope of the current work.

In order to travel from node $i$ to node $j$, a passenger has to stay in the transportation vehicle from node $i$ to the hub for a period of time, spend some time at the hub checking in and waiting for another vehicle, and then stay in the vehicle from the hub to node $j$ for a while. Infection can take place in the public transportation system including the vehicles and the hub. Since people are often physically close to each other in the transportation system, the chance of being infected during travel may be very high.

To investigate the spatial spread of an epidemic among $N$ locations through a public transportation system linking these locations, we propose a mathematical model with the aims of revealing the influence of the public transportation system on the epidemic spread and of gaining insight into the relationship between travel and disease spread. A series of conclusions about the spread and control of an epidemic are obtained using mathematical analysis and numerical simulation.

The paper is organized as follows. In Section 2, we present the formulation of the epidemic model. Section 3 describes the spread and control of an epidemic. Epidemic spreading among identical cities (also called regions or locations) and among non-identical cities with standard incidence is discussed in Sections 4 and 5, respectively. In Section 6, we briefly consider additional interpretations of the model. Section 7 consists of a discussion of the results and their significance, including detailed numerical simulations. Finally, the conclusions are given in Section 8.

2. Formulation of the epidemic model

We consider a disease that has the potential to spread among multiple locations through a transportation system with a hub. Typical epidemics spreading through public transportation are airborne diseases (e.g. SARS and influenza including the H1N1 strain). Such diseases are caused by pathogenic microbial agents spreading through the air. Aerosolized viruses and germs can originate from a patient’s mouth and nose through sneezing, coughing, laughing or speaking [9,14,17]. After leaving the human body, these pathogens can remain suspended in the air contained in aerosol particles or tiny droplets, or stay on surfaces for an extended period of time [3,14,17]. Recent investigations indicate that the SARS coronavirus can survive on a surface for up to 3 days [4], while influenza A and B viruses are capable of remaining contagious on a surface for up to 48 h [3]. By touching such a surface, even without being close to the infectives, the susceptibles may become infected [3,4].

Thus, susceptibles can become infected by being close to the infectives or through sharing the same environment as infectives who have been there recently. Due to the limited space of the public transportation system, passengers are typically closer to each other, and system facilities such as restaurants, washrooms and waiting rooms are shared more often. The infection rate in the system is potentially much higher than that in each region linked by the public transportation.

The susceptibles in a transportation vehicle (e.g. an airplane) connecting a node to the hub can be infected not only by the infectives in the same vehicle but also by the infectives who have taken this vehicle previously. Since each aircraft might be assigned to different routes for different time periods, a healthy passenger in the vehicle might be infected by the infectives going from and/or to any location. Additionally, passengers have to spend a period of time in the transportation hub to check in and possibly to wait for another vehicle. Since there is a limited amount of room and facilities at the hub, the susceptibles have the chance to meet the infectives from any location and/or to any destination. Furthermore, since gates and service counters of the hub are assigned to different routes or airlines for different time periods, passengers to or from different locations have to share them and, as a result, indirect infection may happen. For these reasons, we make the modelling assumption that the hub and the vehicles are a single system with passengers mixing homogeneously throughout.

Thus, the spatial disease transmission among $N$ regions and a single hub is modeled as an SIRS system by the following set of differential equations:

$$
\begin{align*}
\dot{S}_k &= -B_k S_k I_k + \eta_k R_k + \phi \sum_{j=1}^{N} S_j - p S_k, \\
\dot{I}_k &= B_k S_k I_k - \gamma_k I_k + \phi \sum_{j=1}^{N} I_j - p I_k, \\
\dot{R}_k &= \gamma_k I_k - \eta_k R_k + \phi \sum_{j=1}^{N} R_j - p R_k, \\
\dot{S}_j &= A_k - B_k S_j I_k - \mu_k S_j + \eta_k R_k - \phi S_j - \omega_k S_j, \\
\dot{I}_j &= B_k S_j I_k - \mu_k I_j - \gamma_k I_j - \phi \eta_k I_j + \omega_k I_j, \\
\dot{R}_j &= \gamma_k I_j - \eta_k R_j - \mu_k R_j - \phi R_j + \omega_k R_j.
\end{align*}
$$

For $k = 1, \ldots, N$. Here $S_k, I_k$ and $R_k$, respectively, denote the numbers of susceptibles, infectives and recovereds in the population that are currently in the public transportation system. $\gamma_k$ and $\eta_k$ are the recovery rate of the infectives and $\eta_k$ is the rate at which recovered individuals lose immunity and enter the susceptible group in the transportation system. The numbers of susceptibles, infectives and recovereds in region $k$ are denoted by $S_k, I_k$ and $R_k$, respectively. For region $k = 1, \ldots, N A_k$ is the recruitment rate, $\mu_k$ is the natural mortality rate, $\gamma_k$ is the recovery rate of infectives and $\eta_k$ is the rate at which recovered individuals lose immunity and enter the susceptible group. It is assumed that people travel among the $N$ regions using the public transportation system. $\phi$ is the rate at which susceptibles and recovereds travel. We allow that a certain proportion of infective people may develop symptoms and choose not to travel. Thus the rate at which infective people travel becomes $\alpha_0 \phi$, where $0 < \alpha_0 < 1$ is the proportion of the infectives who do not develop serious symptoms.

We assume that, in region $k$, susceptible individuals contract the disease with incidence rate $B_k S_k I_k$, where the contact rate $B_k$ is a function of the total population $N_k = S_k + I_k + R_k$ in region $k$ (i.e. $B_k = B_0(N_k + k + R_k)$). Similarly, $B_k$, a function of the total population in transit $N_k = S_k + I_k + R_k$, is the contact rate in the public transportation system. We further assume that the functions $B_k$ and $B_k$ satisfy the following biologically reasonable conditions.

Finally, the conclusions are given in Section 8.
(i) \( B(N') > 0 \),
(ii) \( B'(N') \leq 0 \), for \( N' > 0 \).

For example, contact rates of the form
\[
B(N) = \frac{B_p}{N^3},
\]
where \( q \geq 0 \) satisfy (i) and (ii). These include the special cases of mass action (also called bilinear incidence with \( q = 0 \)) and standard incidence (\( q = 1 \)) [10].

The rate at which individuals in transit conclude their travel, joining one of the regional populations, is \( p \). Thus, \( 1/p \) is the average time spent in transit. The parameter \( p \) can be thought of as the transportation efficiency; the larger \( p \) is, the more quickly people arrive at their destinations. \( o_k \) is the rate at which individuals in transit arrive in location \( k \). The parameters \( p \) and \( o_k, k = 1, \ldots, N \) are related in that \( p = \sum_{k=1}^{N} o_k \).

In the absence of travel (i.e. \( o_k = \phi = 0 \)), the total population in region \( k \), \( N'_k = s_k + l_k + r_k \), satisfies
\[
\dot{N}'_k = A_k - A_k N'_k.
\]
Thus
\[
\dot{N}'_k(t) = \frac{A_k}{\mu_k} + e^{-\mu_k t} \left( N'_k(0) - \frac{A_k}{\mu_k} \right)
\]
and so \( \lim_{t \to \infty} N'_k(t) = \frac{A_k}{\mu_k} \). For simplicity, we use the equilibrium value \( \frac{A_k}{\mu_k} \) as an estimate of the size of region \( k \). We assume that each region attracts travelers in proportion to the region’s size. Thus,
\[
o_k = \frac{A_k/\mu_k}{\sum_{j=1}^{N} A_j/\mu_j} p,
\]
For the remainder of the paper, assume that the populations in all regions (or cities) are similar in terms of biology and behaviour except that the cities may differ in their sizes. That is, we assume there exists \( (\gamma, \alpha, \mu, \eta) \in R^4_{>0} \) and a function \( \beta : R_{>0} \to R_{>0} \) such that \( (\gamma_k, \alpha_k, \mu_k, \eta) = (\gamma, \alpha, \mu, \eta) \) and \( B_k(\cdot) = \beta(\cdot) \) for \( k = 1, \ldots, N \). A consequence of this assumption is that
\[
o_k = \frac{A_k}{\sum_{j=1}^{N} A_j} p.
\]
To make some of the expressions that follow more concise, let
\[
\bar{\alpha} = \frac{\sum_{j=1}^{N} A_j}{N}
\]
denote the average recruitment rate of the \( N \) regions. With \( \beta_T = Br \), we may now rewrite System (1) as
\[
\begin{align*}
\dot{S}_T &= -\beta_T S_T I_T + \eta_T R_T + \phi \sum_{j=1}^{N} S_j - pS_T, \\
\dot{I}_T &= \beta_T S_T I_T - \gamma_T I_T + \phi \sum_{j=1}^{N} I_j - pI_T, \\
\dot{R}_T &= \gamma_T I_T - \eta_T R_T + \phi \sum_{j=1}^{N} R_j - pR_T,
\end{align*}
\]
where \( \bar{\beta} = \beta(n) \in \bar{\beta}(\bar{\alpha}) + \beta'_{\bar{\alpha}} \bar{\alpha} \) and \( \bar{\beta}_T = \beta_T(n) \in \bar{\beta}_T(\bar{\alpha}) + \beta'_{\bar{\alpha}} \bar{\alpha} \).

The system has a unique disease-free equilibrium given by
\[
E_0 = \left( \frac{\phi AN}{\mu}, 0, 0, \frac{A_1}{\mu}, 0, 0, \frac{A_2}{\mu}, 0, \cdots, \frac{A_k}{\mu}, 0, \cdots \right).
\]
That is, \( I_T = I_1 = \cdots = I_k = 0 \), \( R_T = R_1 = \cdots = R_k = 0 \). \( S_T = \frac{\bar{\beta}N}{\mu} \) and \( S_k = \frac{\bar{\beta}N}{\mu} \) for \( k = 1, \ldots, N \).

### 3. The spread and control of an epidemic

In this section, we investigate the epidemic spreading among \( N \) regions and a hub, obtaining a sufficient condition for the epidemic to die out (see Theorem 1 below). From this result, it is clear that the sizes of different regions may have a significant influence on the spread of the epidemic.

Theorem 1 is based on estimating the basic reproduction number \( R_0 \) of the system (8). However, due to the complexity of the model, \( R_0 \) of the system cannot be obtained through an ad hoc approach. Instead, we use the next generation method discussed in [20] to study the basic reproduction number of the epidemic model. Furthermore, since the dimension of the system poses computational problems, a variant of the standard approach is used.

Using the standard notation [20], we obtain, for system (8),
\[
\mathcal{F} = \begin{bmatrix}
\beta_T S_T I_T \\
\beta_S s_1 \tau_S \\
\beta_S s_2 \tau_S \\
\vdots \\
\beta_S s_N \tau_S
\end{bmatrix}
\quad \text{and} \quad
\mathcal{V} = \begin{bmatrix}
\gamma_T I_T - \phi \sum_{j=1}^{N} I_j + pI_T \\
\mu_1 + \gamma_1 - \frac{\bar{\alpha}_1 \phi}{AN} + \phi \beta_1 \\
\mu_2 + \gamma_2 - \frac{\bar{\alpha}_2 \phi}{AN} + \phi \beta_2 \\
\vdots \\
\mu_N + \gamma_N - \frac{\bar{\alpha}_N \phi}{AN} + \phi \beta_N
\end{bmatrix}.
\]

Then,
\[
F = \begin{bmatrix}
\frac{\mu \gamma}{\mu + \gamma} & 0 & 0 & 0 & 0 \\
0 & \frac{\mu \gamma}{\mu + \gamma} & 0 & 0 & 0 \\
0 & 0 & \frac{\mu \gamma}{\mu + \gamma} & 0 & 0 \\
0 & 0 & 0 & \frac{\mu \gamma}{\mu + \gamma} & 0 \\
0 & 0 & 0 & 0 & \frac{\mu \gamma}{\mu + \gamma}
\end{bmatrix},
\]
where the contact rates are evaluated at the disease-free equilibrium i.e. \( (\beta_1 = \beta(N)I_1 = \beta(\mu) + \beta_T(N)I_1 = \beta_T(\alpha)) \) and
\[
V = \begin{bmatrix}
\gamma_T + p & -\phi \alpha & -\phi \alpha & \cdots & -\phi \alpha \\
-\frac{\bar{\alpha}_1 \phi}{AN} & \mu + \gamma + \phi \alpha & 0 & \cdots & 0 \\
\vdots & \vdots & \ddots & \ddots & \ddots \\
-\frac{\bar{\alpha}_N \phi}{AN} & 0 & \mu + \gamma + \phi \alpha & 0 & \cdots \\
0 & \cdots & \cdots & \cdots & \mu + \gamma + \phi \alpha
\end{bmatrix}.
\]

The basic reproduction number can then be calculated as
\[
R_0 = p(FV^{-1}),
\]
where \( \rho(M) \) denotes the spectral radius of a matrix \( M \) (i.e. the largest modulus of the eigenvalues of \( M \)).

From (11) and (12),
\[ FV^{-1} = \frac{1}{Q} \begin{bmatrix} L & 0 & \cdots & 0 \\ \frac{a_1}{b_1} & \frac{a_2}{b_2} & \cdots & \frac{a_N}{b_N} \\ \vdots & \vdots & \ddots & \vdots \\ \frac{a_1}{b_1} & \frac{a_2}{b_2} & \cdots & \frac{a_N}{b_N} \end{bmatrix} \] 

(14)

where \( Q = p_M \gamma^2 + p_\gamma \phi N \delta A \), \( a_i = \frac{\lambda_i \phi \gamma N}{b_i} \), \( b_j = \frac{\lambda_j \phi \gamma N}{b_j} \), and \( L = \frac{\lambda_j + \gamma + \phi x}{b_j} \). Since it is difficult to study the spectral radius of \( FV^{-1} \) directly due to its complexity, we use a modified version of the next generation method to investigate the basic reproduction number of our model. We first state a basic lemma, which will be used in the discussion that follows.

**Lemma 1.** If \( M \) is an invertible matrix with eigenvalue \( \lambda \), then \( \frac{1}{\lambda} \) is an eigenvalue of \( M^{-1} \).

By **Lemma 1**, we instead consider the inverse of \( FV^{-1} \) which is given by

\[ \begin{bmatrix} p_M \gamma^2 + p_\gamma \phi N \delta A & -\frac{\lambda_i \phi \gamma N}{b_i} & \cdots & -\frac{\lambda_i \phi \gamma N}{b_i} \\ -\frac{\lambda_j \phi \gamma N}{b_j} & \frac{\lambda_j \phi \gamma N}{b_j} & \cdots & 0 \\ -\frac{\lambda_j \phi \gamma N}{b_j} & 0 & \cdots & 0 \\ \vdots & \vdots & \ddots & \vdots \\ -\frac{\lambda_i \phi \gamma N}{b_i} & 0 & \cdots & 0 \end{bmatrix} \] 

(15)

Since \( FV^{-1} \) is similar to the symmetric real matrix \( M = P(FV^{-1})P^{-1} \), where \( P = \text{diag}(1, a_1, \ldots, a_N) \) and

\[ a_i = \frac{\phi \gamma N \delta A}{b_i}, \quad b_i > 0, \]

the eigenvalues of \( FV^{-1} \) (and hence those of \( FV^{-1} \)) are all real.

Thus by Gersgorin’s Theorem [11] applied to column sums, the eigenvalues of \( FV^{-1} \) are located in the union of the following \( N + 1 \) intervals

\[ x - p_M \gamma^2 + p_\gamma \phi N \delta A \leq \frac{\lambda_j \phi \gamma N}{b_j} \leq x - \mu + \gamma + \phi x, \]

(16)

for \( i = 1, \ldots, N \), where \( x \) denotes a point on the real line. Equivalently, each eigenvalue of \( FV^{-1} \) lies in the following union of intervals:

\[ \gamma + \lambda \begin{bmatrix} \gamma + 2 \gamma \phi x + p_\gamma \phi N \delta A \\ \gamma + 2 \gamma \phi x + p_\gamma \phi N \delta A \\ \gamma + 2 \gamma \phi x + p_\gamma \phi N \delta A \end{bmatrix} \cup \left\{ \gamma + \frac{\mu + \gamma + 2 \phi x}{\mu + \gamma} \right\} \]

(17)

Furthermore, by inverting, we find that the eigenvalues of \( FV^{-1} \) are located in

\[ \begin{bmatrix} \beta_1 \phi N \delta A \gamma \gamma + 2 \gamma \phi x + p_\gamma \phi N \delta A \\ \beta_1 \phi N \delta A \gamma \gamma + 2 \gamma \phi x + p_\gamma \phi N \delta A \\ \beta_1 \phi N \delta A \gamma \gamma + 2 \gamma \phi x + p_\gamma \phi N \delta A \\ \vdots \end{bmatrix} \cup \left\{ \beta_1 \phi N \delta A \gamma \gamma + 2 \gamma \phi x + p_\gamma \phi N \delta A \right\} \]

(18)

as guaranteed by **Lemma 1**. Without loss of generality, we can assume that \( \beta_1 A_1 \leq \beta_2 A_2 \leq \cdots \leq \beta_N A_N \). Then, each eigenvalue \( \lambda \) of \( FV^{-1} \) satisfies

\[ \min \left\{ \beta_1 \phi N \delta A \gamma \gamma + 2 \gamma \phi x + p_\gamma \phi N \delta A \right\} \leq \lambda \]

\[ \leq \max \left\{ \beta_1 \phi N \delta A \gamma \gamma + 2 \gamma \phi x + p_\gamma \phi N \delta A \right\}. \]

(19)

The basic reproduction number \( R_0 \) is the maximum of these eigenvalues and so Eq. (19) leads to the following theorem.

**Theorem 1.** Assume the cities are ordered so that \( \beta_1 A_1 \leq \beta_2 A_2 \leq \cdots \leq \beta_N A_N \). If

\[ \max \left\{ \frac{\beta_1 \phi N \delta A \gamma \gamma + 2 \gamma \phi x + p_\gamma \phi N \delta A}{\gamma \gamma + 2 \gamma \phi x + p_\gamma \phi N \delta A} \right\} < 1, \]

(20)

then \( R_0 < 1 \) and the disease-free equilibrium \( E_0 \) of (8) is locally asymptotically stable. If

\[ \min \left\{ \beta_1 \phi N \delta A \gamma \gamma + 2 \gamma \phi x + p_\gamma \phi N \delta A \right\} > 1, \]

(21)

then \( R_0 > 1 \) and \( E_0 \) is unstable.

**Discussion of Theorem 1** This theorem presents a sufficient condition [20] for the disease-free equilibrium to be locally asymptotically stable. When there is no travel between the cities and hub (i.e. the cities are isolated), the basic reproduction numbers of the individual cities are \( \frac{A_1}{N_1} \leq \frac{A_2}{N_2} \leq \cdots \leq \frac{A_N}{N_N} \). Thus, if the additional condition \( \lim_{y \to 0} \beta_1(y)y = 0 \) is satisfied, then, for small \( \phi \) or large \( p \), conditions (20) and (21) imply that the disease-free equilibrium is locally asymptotically stable if the same is true when the cities are isolated. While that statement could be made through a continuity argument, Theorem 1 goes further, showing that the stability of the disease-free equilibrium persists at least until \( \beta_1 \frac{\phi N \delta A}{N} \) becomes as large as \( \gamma \). Notice that, in the remainder of this section, we explicitly show the dependence of \( \beta_1 \) as a function of \( \frac{\phi N \delta A}{N} \).

For \( \beta_1 \frac{\phi N \delta A}{N} < \gamma \), the stability of the disease-free equilibrium of system (8) is, to some extent, determined by the basic reproduction number of the largest region, region \( N \), in isolation, \( \frac{A_N}{N_1} \). Thus, increasing the public transportation efficiency \( p \) is an effective strategy to control or slow down the disease spreading among the \( N \) cities. Increasing the efficiency of the inter-regional public transportation system decreases the time that the passengers have to be in the public transportation system and as such reduces the possibility of passengers being infected. Condition (20) indicates that an efficient inter-regional public transportation system has stronger resistance to the spread of infectious diseases.

Similarly, decreasing the rate of travel (i.e. decreasing \( \phi \)) is another effective approach to control or slow down the spread of infectious disease through the inter-regional public transportation system. One possible way for policy makers and health agencies to decrease this rate is to provide up-to-date information about the spread of the epidemic disease and to warn against non-essential travel at the early stages of the epidemic outbreak. This will slow down the disease spread through the public transportation system if these warnings are heeded.

**Remark.** In the above discussion of the effects of the public transportation efficiency \( p \) and the proportion of the population who travel \( \phi \) on the spread of the epidemic, we considered the condition

\[ \lim_{y \to 0} \beta_1(y)y = 0. \]
Contact rates of the form $\beta(N) = \frac{\beta N^q}{N^q + 1}$ where $0 \leq q < 1$ satisfy this condition. As indicated in [10], such contact rates describe human disease infection with more accuracy than the widely used standard incidence ($q = 1$) for which the assumption fails.

Decreasing transmissibility in the public transportation system (i.e. decreasing $\beta_f(\frac{x_{\text{N}}}{x_{\text{RF}}})$) can help to control the spread of the disease. Improving sanitary conditions of the public transportation (through disinfecting of the system environment, installing ventilation equipment, and maintaining and cleaning vehicles regularly) will lower $\beta_f(\frac{x_{\text{N}}}{x_{\text{RF}}})$ by decreasing the amount of indirect disease transmission from touching contaminated surfaces or breathing contaminated air.

The sufficient condition given in Theorem 1, inequality (21), for the spread of a disease is not as useful from a practical perspective. In particular, it cannot be used to determine in what circumstances the transportation system will destabilize a stable disease-free equilibrium in the isolated cities. In this case, we would have $\frac{\beta_f(\frac{x_{\text{N}}}{x_{\text{RF}}})}{\mu} < 1$, and so inequality (21) could not hold. As we see from the simulations of Section 7 (cf. Fig. 2), it is the numerical evaluation of $R_0$ from the next generation matrix in (14) that is needed in these cases.

4. Epidemic spreading among identical cities

In this section, we assume that $A_1 = A_2 = \cdots = A_0 = A$ and so all cities have the same size. Thus, $\overline{A} = A$. Furthermore, with this assumption, the epidemic model (8) can be written as

\[
\begin{align*}
\dot{S}_r &= -\beta_f S_r I_r + \eta_r R_r + \phi \sum_{j=1}^N S_j - p S_r, \\
\dot{I}_r &= \beta_f S_r I_r - \gamma_r I_r + \phi \sum_{j=1}^N I_j - p I_r, \\
\dot{R}_r &= \gamma_r I_r - \eta_r R_r + \phi \sum_{j=1}^N R_j - p R_r, \\
\dot{S}_k &= A - \beta S_k I_k - \mu S_k + \eta R_k - \phi S_k + \frac{p S_r}{N}, \\
\dot{I}_k &= \beta S_k I_k - \mu I_k - \gamma_k I_k - \phi I_k + \frac{p I_r}{N}, \\
\dot{R}_k &= \gamma_k I_k - \eta R_k - \mu R_k - \phi R_k + \frac{p R_r}{N}.
\end{align*}
\]

The disease-free equilibrium is now given by

\[
E_0 = \left( \frac{\phi AN}{\mu}, 0, 0, \frac{A}{\mu}, 0, \frac{A}{\mu}, 0, \ldots, \frac{A}{\mu}, 0, 0 \right).
\]

For non-identical cities, we obtained only approximations of the eigenvalues. However, for identical cities, the matrix $VF^{-1}$ has more structure, allowing exact calculation of the eigenvalues. It is easy to see that (15) can be written as

\[
VF^{-1} = \begin{bmatrix}
\frac{a}{N} & \frac{b}{N} & \frac{b}{N} & \cdots & \frac{b}{N} \\
\frac{c}{N} & \frac{d}{N} & 0 & \cdots & 0 \\
\frac{c}{N} & 0 & \frac{d}{N} & \cdots & 0 \\
\vdots & \vdots & \ddots & \ddots & \vdots \\
\frac{c}{N} & 0 & \cdots & 0 & \frac{d}{N}
\end{bmatrix}_{(N+1) \times (N+1)}
\]

with

\[
\begin{bmatrix}
a & b & b & \cdots & b \\
c & d & 0 & \cdots & 0 \\
c & 0 & d & \cdots & 0 \\
\vdots & \vdots & \ddots & \ddots & \vdots \\
c & 0 & \cdots & 0 & d
\end{bmatrix}
\]

\[
\begin{bmatrix}
\frac{p N}{\phi \mu} & \frac{\phi N}{\mu} & \frac{\phi N}{\mu} & \cdots & \frac{\phi N}{\mu} \\
\frac{p N}{\phi \mu} & \frac{\phi N}{\mu} & \frac{\phi N}{\mu} & \cdots & \frac{\phi N}{\mu} \\
\frac{p N}{\phi \mu} & \frac{\phi N}{\mu} & \frac{\phi N}{\mu} & \cdots & \frac{\phi N}{\mu} \\
\vdots & \vdots & \ddots & \ddots & \vdots \\
\frac{p N}{\phi \mu} & \frac{\phi N}{\mu} & \frac{\phi N}{\mu} & \cdots & \frac{\phi N}{\mu}
\end{bmatrix}
\]

where $\beta_f = \beta_f(\frac{x_{\text{N}}}{x_{\text{RF}}})$ and $\beta = \beta(\frac{x_{\text{N}}}{x_{\text{RF}}})$. Using (24), the eigenvalues of $VF^{-1}$ can be calculated directly. We find that $d$ is an eigenvalue of $VF^{-1}$ with multiplicity $N - 1$ and the remaining two eigenvalues are also eigenvalues of the $2 \times 2$ matrix

\[
\begin{bmatrix}
a & b \\
c & d
\end{bmatrix}
\]

Thus, $N - 1$ of the eigenvalues are equal to $\mu \frac{\phi N}{\mu} + d$ and the other two eigenvalues are roots of the quadratic

\[
0 = \lambda^2 - a_2 - d_2 - Nc_b + ad.
\]

Using (25), it follows that both roots of (27) are positive reals and that the smaller root $\lambda_1$ is less than $\lambda_2 = \frac{\mu \phi N}{\mu}$ Thus, all eigenvalues of $VF^{-1}$ are positive reals, and the smallest is $\lambda_1$. Since $R_0$ is the reciprocal of the smallest, we obtain

\[
R_0 = 2\frac{\beta A}{\mu} \frac{\beta N \phi}{\Gamma_1 - \sqrt{\Gamma_2^2 - \Gamma_2}},
\]

where

\[
\Gamma_1 = \beta p (\gamma_2 + p) + \beta N \phi (\mu + \gamma + \phi \alpha),
\]

\[
\Gamma_2 = 4 \beta p \phi \beta N \phi (\gamma_1 (\mu + \gamma + \phi \alpha) + \mu (\mu + \gamma)).
\]

We note that $\Gamma_2$ is strictly less than $\Gamma_1^2$.

By van den Driessche and Watmough (2002, Theorem 2), we have the following result.

**Theorem 2.** The disease-free equilibrium $E_0$ of (22) is locally asymptotically stable if $R_0 < 1$ and is unstable if $R_0 > 1$.

In order to discuss the effect that various model parameters have on $R_0$, we assume that the contact rate in the public transportation system $\beta_f$ satisfies $\lim_{y \to 0} \beta_f(y) = 0$. To further discuss the effect of public transportation efficiency $p$ on the spatial epidemic spreading, we investigate the case where $p$ tends to infinity. We find that

\[
\lim_{p \to \infty} R_0 = \frac{\beta A}{\mu (\mu + \gamma)} =: R_*.
\]

**Theorem 3.** For sufficiently large $p$, the disease-free equilibrium $E_0$ of (22) is locally asymptotically stable if $R_* < 1$ and is unstable if $R_* > 1$.

Note that $R_*$ is the same as the basic reproduction number of an isolated region. Theorem 3 indicates that, when public transportation efficiency is high enough, the disease-free equilibrium of (22) is locally asymptotically stable if the disease-free equilibrium for each of the cities without inter-regional public transportation is locally asymptotically stable (see Figs. 1(a)).

However, since the public transportation efficiency $p$ is determined by the average speed and number of vehicles in the public transportation system and these are both bounded, there is a limit as to how efficient the public transportation can be. As we will see (cf. Fig. 1(a)), it is then possible for an epidemic to spread in the full system even though the disease-free equilibrium is asymptotically stable in each isolated city.

\[1\] That is, for $p$ high enough, travel has no effect on the stability of the disease-free equilibrium in our model, which does not consider other modes of inter-regional transportation. As shown by [22,24], movement between regions can affect disease spread even when there is no transmission while travelling.
Intuitively, this latter scenario cannot occur when there are few people travelling. To see this, consider the limit as the rate at which people choose to travel goes to zero, that is as \( \phi \) tends to zero. In this case, Eq. (28) yields

\[
\lim_{\phi \to 0} R_0 = \frac{\beta A}{\mu (\mu + \gamma)} = R_c.
\]

(31)

**Theorem 4.** For sufficiently small \( \phi \), the disease-free equilibrium \( E_0 \) of (22) is locally asymptotically stable if \( R_c < 1 \) and is unstable if \( R_c > 1 \).

Theorem 4 indicates that, when the proportion of people who travel is sufficiently small, the stability of the disease-free equilibrium of (22) is determined by the stability of the disease-free equilibrium in the isolated cities.

Comparing Theorems 3 and 4, we see that the stability of the disease-free equilibrium is determined by the same quantity \( R_c \), if transmission during transport is minimized. For large \( p \), this transmission is minimized because individuals pass through the transportation system very quickly; for small \( \phi \), it is because very few individuals enter the transportation system.

On the other hand, the spread of a disease cannot always be controlled by decreasing the rate \( x \) at which infected people enter the transportation system (compared to non-infected people). We note that \( \lim_{x \to 0} R_0 = \max \left\{ R_c, \frac{\beta A N_{\text{in}}}{\mu (\mu + \gamma) x} \right\} \). This limit can be obtained directly from (28) and (29) or indirectly by considering (25) with \( \chi = 0 \). Thus, the disease can spread (i.e. \( R_0 > 1 \)) when \( R_c < 1 \) if, for example, the transportation efficiency \( p \) is low. In fact, even when no infectives travel (i.e. \( x = 0 \)), a disease can still spread by means of the transportation system when the disease-free equilibrium is asymptotically stable in each isolated city (Fig. 1(d)). In this case, the disease initially spreads from infected people within the transportation system (so \( l_i(0) > 0 \)) dispersing to the cities.\(^2\) To control the disease in these circumstances, infected people in the transportation system must be prevented from infecting people in the cities when they return there. For example, if infected people leaving the transportation system can be identified, then they could be placed in quarantine.

The health condition of the public transportation system as measured by \( \beta_T \) also has significant influence on the epidemic spreading through such systems. On one hand, the limited room in these systems means passengers in the system are typically in close proximity to each other. On the other hand, shared facilities in the public transportation system increases the chance of indirect transmission. Thus, the transmission rate in the public transportation is potentially higher than that in a city. However, this transmission rate can still be decreased by regularly disinfecting the public transportation system environment, installing ventilation equipment, and maintaining and cleaning vehicles regularly.

\(^2\) However, if both \( l_i(0) = 0 \) and \( x = 0 \), the disease will die out.
An interesting relationship between the epidemic spreading and the health condition of the public transportation is revealed if we let $\beta_t$ approach 0 and obtain
\[
\lim_{\beta_t \to 0} R_0 = \frac{\beta A}{\mu + \gamma + \frac{N}{\beta_0 p}} = \frac{\beta A}{\mu + \gamma + \alpha \phi} \frac{1}{1 - \frac{\beta_0}{\mu + \gamma + \alpha \phi}}.
\]  
(32)
The final expression of the preceding equation can also be obtained by considering a newly infected individual undergoing a random walk through the infective classes. Since the cities are identical, the random walk may be considered to be back and forth between classes $I_t$ and $R_t$. The number of new infections expected caused by a single infective over the entire course of infectiousness in an otherwise susceptible population [10]. Because $\beta_0$ is tending to 0, $R_0$ is the product of the number of susceptibles in city 1 at the disease-free equilibrium $\hat{p}$, the incidence rate at the disease-free equilibrium $\hat{p}$, the average time spent in class $I_t$ per visit $\frac{1}{\mu + \gamma + \phi}$, and the expected number $K$ of visits to $I_t$ during the random walk. In order to calculate $K$, it is necessary to sum a geometric series
\[
K = 1 + q + q^2 + \cdots = \frac{1}{1 - q}
\]
where $q$ is the probability of leaving $I_t$ for $R_t$ and then returning to $I_t$ again. The quantity $q$ is a product of the probability that individuals leave $I_t$ for $R_t$ with the probability that individuals leave $I_t$ for $I_t$. These probabilities are calculated by taking the ratio of the required flow rate to the total exit flow rate. Thus $q$ is the product of $\frac{\beta_0}{\mu + \gamma + \phi}$ and $\frac{p}{\mu + \gamma + \alpha \phi}$. Therefore, $K$ is given by the final expression in (32).

We note that $\lim_{\beta_t \to 0} R_0 < R_*$, which indicates that, in principle, a transportation system with a small contact rate (i.e., $\beta_t$ close to 0) could be used to eliminate an epidemic spread. In theory, Theorem 2 then implies that the transportation system can eliminate a disease that would spread in isolated cities (i.e., $R_* > 1$). In order for this to be possible, it must be the case that
\[
\phi \alpha > (R_* - 1) (\mu + \gamma) \frac{p}{\mu + \gamma}.
\]
That is, sufficiently many infected people must be encouraged to leave the cities to travel in order to reduce the number infected in the cities below a critical value. Although this is an interesting mathematical consequence of the model, it seems quite unlikely such a public policy would be implemented in practice.

5. Epidemic spreading among non-identical cities with standard incidence

In this section, we consider the case that the epidemic spreads with standard incidence rate (i.e., $\beta(N) = \frac{\beta_0}{N}$ from (2)) in each city, where these cities may have different sizes. As in Section 4, we are able to determine the basic reproduction number precisely. Then, system (8) can be written as
\[
\begin{align*}
\dot{S}_t &= -\beta_t S_t R_t + \eta_t I_t + \phi \sum_{j=1}^{N} S_j - p S_t, \\
\dot{I}_t &= \beta_t S_t I_t - \gamma I_t + \phi \sum_{j=1}^{N} I_j - p I_t, \\
\dot{R}_t &= \gamma I_t - \eta R_t + \phi \sum_{j=1}^{N} R_j - p R_t, \\
\dot{S}_k &= A_k - \frac{\beta_0}{S_k + \eta R_k} S_k I_k - \mu R_k + \eta R_k - \phi S_k + A_k p S_T, \\
\dot{I}_k &= \frac{\beta_0}{S_k + \eta R_k} S_k I_k - \mu I_k - \gamma I_k - \phi \sum_{j=1}^{N} I_j + A_k p I_T, \\
\dot{R}_k &= \gamma I_k - \eta R_k - \mu R_k - \phi R_k + A_k p R_T.
\end{align*}
\]  
(34)
where $\beta_0$ is the transmission coefficient. We note that in the absence of travel, the isolated cities all have the same basic reproduction number $R_* = \frac{\beta_0}{\mu + \gamma}$. Similarto Section 4, $FV^1$ takes the form of Eq. (24) with
\[
\begin{pmatrix}
a & b \\
Nc & d
\end{pmatrix} = \begin{pmatrix}
\frac{\gamma I_j}{\beta_0} & p \phi \\
\frac{\gamma I_j}{\beta_0} & \frac{\beta_0}{\mu + \gamma + \phi}
\end{pmatrix}
\]  
(35)
where $\beta_t = \beta_t \frac{4}{\beta_0^2}$. Then, through a similar calculation, the basic reproduction number for (34) is obtained as
\[
R_{\infty} = 2\beta_0 + \frac{\beta_1 N \phi}{\Gamma_1 - \sqrt{\Gamma_1^2 - \Gamma_2}},
\]  
(36)
where
\[
\Gamma_1 = \frac{4\beta_0}{A} \frac{\beta_0 \mu (\gamma + \phi) + \beta_1 N \phi (\mu + \gamma + \phi \alpha)}{\mu (\gamma + \phi) + \phi \mu (\gamma + \phi)}
\]
Here $\Gamma_2$ is strictly less than $\Gamma_1$. The analogue of Theorem 2 is the following theorem.

Theorem 5. The disease-free equilibrium $E_0$ of (34) is locally asymptotically stable if $R_{\infty} < 1$ and is unstable if $R_{\infty} > 1$.

Next we investigate the effects that the public transportation efficiency $\phi$ and the proportion $\alpha$ of the population who travel have on the epidemic spread. We again assume that $\lim_{\beta_t \to 0} \beta_t \gamma = 0$ and so the incidence rate in the transportation system is not standard incidence (see the Remark following Theorem 1). However, it is still reasonable to assume standard incidence in the regions since these typically have a large population size. Specifically, it has been shown [10] that epidemic models based on standard incidence give acceptable approximations to data from epidemic spreading for five human diseases in a region with total population size greater than 1000.

With these assumptions, we obtain the following theorem.

Theorem 6. For sufficiently large $p$ or for sufficiently small $\phi$, the disease-free equilibrium $E_0$ of (34) is locally asymptotically stable if $\frac{\beta_0}{\mu + \gamma} < 1$ and is unstable if $\frac{\beta_0}{\mu + \gamma} > 1$.

Thus, for a public transportation system that is either highly efficient or has a low number of passengers, the stability of the disease-free equilibrium is the same as for each isolated city.

6. Additional interpretations of the model

The primary interpretation of our systems (1), (8), (22) and (34) is as models of disease spread among $N$ regions where individuals move between these regions by means of a transportation system with a central hub within which disease transmission can also occur. These systems can be re-interpreted to consider the role that quarantines and hospitals play during an epidemic.

First, consider the case where a quarantine has been introduced as a control measure. This can be done in one of two ways. There is the sanatorium style of quarantine which has been used for tuberculosis, and in some sense is similar to the historical leper colonies. Here, infected individuals are grouped together, in an attempt to keep them from infecting the general population. The parameter $\phi$ is small, indicating that susceptibles enter the quarantine at a very slow rate, whereas $\eta$ is large indicating that infected individuals enter the quarantine at a higher rate. Let $\psi = \alpha \phi$, so that the two rates are decoupled. This allows us to consider $\phi$ to be zero.
while $\hat{\phi}$ is non-zero, indicating that only infectious enter the quarantine. Then, a careful analysis of (28) and (29) gives

$$R_0 = \frac{pA}{\mu} \frac{\gamma_I + p}{\gamma_1 (\mu + \gamma + \hat{\phi}) + p(\mu + \gamma)}$$

for the case of identical cities.

Alternatively, a home-based quarantine, as was used in the Toronto SARS outbreak of 2003, encourages any suspected infected individuals to stay home for a prolonged period of time, reducing potential transmission. Potentially, this will involve sending susceptibles into quarantine as well as infecteds. Ideally, though, $\alpha$ would be greater than one so that an infected individual is more likely to be quarantined than a susceptible individual. Since each individual in a home-based quarantine has a limited number of contacts, this corresponds to the case where $\beta_I$ is very small. In this case $R_0$ is given by (32).

An important area of disease management [7] is to control the spread of antibiotic resistant staph infections (which are primarily transmitted in hospitals) to the community at large. System (22) can be re-interpreted as a model of a central hospital (i.e. the hub) serving a community consisting of $N$ identical regions where $\beta_I$, the contact rate in the hospital, is much larger than in the community ($\beta$). Taking $\beta$ close to 0, Theorem 2 and Eq. (28) show how altering the number of community members (such as hospital staff and visitors) and their time spent at the hospital (i.e. by altering $\phi$ and $p$) affect the stability of the disease free equilibrium. Here,

$$R_0 = \frac{pAAN}{\mu} \frac{\phi(\mu + \gamma + \phi x)}{p[\gamma_1 (\mu + \gamma + \phi x) + p(\mu + \gamma)]}$$

7. Numerical simulations and discussions

In this section, we present some numerical simulation results to further illustrate the epidemic spreading among 3 discrete geographical regions through an inter-city public transportation system. Based on these simulation results and the mathematical analysis given in Sections 3–5, further discussions about the epidemic spread and control are given. The default parameters used for the numerical simulations come from [12] which provides a detailed analysis of influenza transmission dynamics assuming a standard incidence model for disease transmission within each city. In these simulations, population size is measured in thousands and time in days.

Epidemic spreading among identical cities

Fig. 1 shows the dependence of the stable equilibrium on the transportation parameters (i.e. it shows the bifurcation diagrams). First, the effect of public transportation efficiency $p$ on the epidemic spread is shown in bifurcation diagram Fig. 1(a). The equilibrium number of infectious in the population that are currently in the public transportation system decreases with increasing $p$. As a result, epidemic spread is slowed down in the three cities. The effect of the sanitation condition of the public transportation system (i.e. the effect of the contact rate $\beta_I$) on the epidemic spread is shown in Fig. 1(b). With improved sanitation, $\beta_I$ is expected to decrease and so fewer travelers will be infected during the journey, and as such epidemic spread in the three cities is lessered accordingly. The relationship between the rate $\phi$ at which susceptibles choose to travel and the epidemic spreading is illustrated in bifurcation diagram Fig. 1(c), which indicates the potential effectiveness of warning against unnecessary travel at controlling the epidemic. The effects of decreasing $\alpha$ on the epidemic spread will be discussed in the following subsection.

Installing clinical thermometers

Fever is a symptom of many epidemic diseases. Installing clinical thermometers at the entrances of the inter-city public transportation systems can then be used to decrease the rate infectious travel (i.e. to decrease $\alpha$) either through mandatory travel bans for individuals with a fever or through voluntary actions by infective travelers who self diagnose before entering the inter-city public transportation system. With fewer infectious entering the public transportation system, infection within the system will decrease. As a result, epidemic spread in the cities will decrease accordingly. However, as shown in Fig. 1(d), decreasing $\alpha$ by installing clinical thermometers at the entrances of the inter-city public transportation system does not always return the system to a stable disease-free equilibrium, even when the disease would die out in isolated cities. If the sanitary condition of the public transportation system is poor, then viruses will spread quickly within the system due to the high contact rate; that is $\beta_I$ will be high. Even with decreased $\alpha$, the number of infectious in the public transportation system might still be high and the effect of decreasing $\alpha$ might not be significant. However, in a cleaner public transportation system, the effect of decreasing $\alpha$ is more pronounced in decreasing the spread of the disease. As indicated in Fig. 1(d), for some parametric values related to transportation, the epidemic cannot be totally controlled by letting $\alpha = 0$. Assume that, from time $t_0$, $\alpha$ is reduced to 0. For a transportation system with low $p$ and high $\beta_I$, since the infecteds who entered the system before $t_0$ have to spend an extended period of time in the system, they could be still in the system after $t_0$ and as such the susceptibles who entered the system after $t_0$ can still get infected. In this case, the transportation hub acts as a reservoir for infectives.

Epidemic spreading among non-identical cities

In this section, we perform numerical simulations to verify the bound of the basic reproduction number $R_0$ given in Theorem 1. Here we assume that the disease-free equilibrium of each city is stable if there is no public transportation system involved. When these cities are linked by the public transportation system, with appropriately chosen parameter values, the disease-free equilibrium of the model is destabilized as shown in Fig. 2(a). Using the matrix $FV^{-1}$ in (14), we obtained the basic reproduction number of the system, $R_0 = 1.209$. Decreasing $\phi$ reduces $R_0$ to 0.980 and stabilizes the disease-free equilibrium (See Fig. 2(b)). Note that for these latter parameters, Theorem 1 gives $R_0 < \max \left\{ \frac{\alpha_0 AN}{\gamma_1 (\mu + \gamma)}, \frac{\alpha_0 AN}{\mu (\mu + \gamma)} \right\} = 0.985 < 1$. Similar effects can be obtained by varying of $\beta_I$ and/or $p$.

Epidemic spreading among non-identical cities with standard incidence

Next we consider the case that the epidemic spreads with standard incidence rate in three locations and all these cities have different sizes. We assume that the disease starts in the city with the lowest size and that the other two cities as well as the public transportation system are free of disease at time 0. All parameters are chosen such that the disease would die out in these cities if they were all isolated.

We then choose the transportation system parameters, $p$, $\phi$ and $\beta_I$ such that the disease-free equilibrium $E_0$ of system (34) is unstable (See Fig. 3(a)). This follows from Theorem 5 since $R_0 = 1.177$ for these parameters. As indicated in Fig. 3(a) where the epidemic initially starts in city 1, the disease spreads through all three cities within a short period of time by way of the public transportation system. The infective travelers infect the susceptibles in the public transportation system with contact rate $\beta_I (N_I)$.

Increasing public transportation efficiency decreases the average time passengers spend in the public transportation system and as such lowers the chance of infection. On one hand, when the infectives spend less time in the transportation system, their chance of meeting susceptibles is lowered. On the other hand, the healthy passengers also spend less time in the system and thus have less chance of being infected. With increased public
transportation efficiency $p$, the disease-free equilibrium $E_0$ of system (34) is locally asymptotically stable, i.e., the epidemic dies out in all three cities as well as in the public transportation system (See Fig. 3(b)). Similar effects can be obtained by decreasing $b_T$ and/or $\phi$.

**Sensitivity analysis**

To investigate how the basic reproduction number as given in (36) is related to properties of transportation including the efficiency and sanitation condition of the public transportation system, and to the proportions of the population and the infectives who travel, we perform a sensitivity analysis. We assume $b_T(N_T) = \frac{b_T}{N_T}$ and $\phi(b) = b$. As shown in Figs. 4(a) and (b), increasing $p$, decreasing $b_T$, and decreasing $\phi$ are effective strategies in controlling the epidemic spreading. With the decrease of $\alpha$, the basic reproduction number $R_0$ decreases accordingly. However, decreasing $\alpha$ alone does not always guarantee the elimination of the epidemic when the disease-free equilibria of all the isolated cities are stable.

**Cost-effective epidemic control strategies**

As discussed above, reducing unnecessary travel (e.g. by issuing warnings that are heeded when epidemics start) and increasing public transportation efficiency are effective strategies in the epidemic control. However, reducing travel and increasing the number of vehicles in the public transportation system are not always applicable, especially when the budget is limited. On one hand, it is unreasonable to expect warning against travel to continue to be effective for a long period of time, especially for business travel. On the other hand, the cost of increasing the number of vehicles and hiring more drivers will be high. Thus, developing a cost-efficient epidemic control strategy is essential.

As indicated in Fig. 3(a), the most populated city is also the city most sensitive to epidemic spread. Thus, the epidemic situation of the most populated city might serve as a forecast for all the other cities. Using this forecast information to determine when warning against unnecessary travel should be announced
might be an efficient and cost-effective way to decrease the epidemic spread through the inter-city public transportation system. For instance, a threshold value $I_{\text{threshold}}$ can be set for the number of infectives in the largest city. When the number of the infectives in this city is above this threshold, warning against unnecessary travel will be announced (and so $\phi$ decreased) until the number is under this threshold value. Based on the above discussion, the rate at which susceptibles choose to travel $\phi$ is defined as

$$
\phi = \begin{cases} 
\phi_1 & I_{\text{threshold}} < I_N \\
\phi_2 & I_N \leq I_{\text{threshold}} 
\end{cases}
$$

(38)
where $\phi_1 < \phi_2$. This strategy is effective in epidemic control without decreasing the number of travelers for a long period of time (see Figs. 5(a) and (b)).

The number of vehicles put in use in the public transportation system can also be adjusted according to the epidemic forecast. More vehicles will be put in use when the number of the infectives in the most populated city is above the threshold value until the number is under or equal to this value. Thus, the transportation efficiency $p$ is given by

$$p = \begin{cases} p_1 & I_N \leq I_{\text{threshold}} \\ p_2 & I_{\text{threshold}} < I_N, \end{cases}$$

(39)

where $p_1 < p_2$. As simulated in Figs. 5(a) and (c), the strategy decreases the epidemic spreading greatly without increasing the number of vehicles in the public transportation system for a long period of time.

8. Conclusion

In this paper, we have studied a model of disease transmission amongst multiple regions or cities through a transportation system with a single hub. Due to the properties of airborne diseases and the fact that the vehicles, and public transportation facilities such as gates and service counters in the transportation hub are assigned to different routes at different times, passengers from/to any location might have the chance to meet passengers from/to any location, directly or indirectly. For simplicity, we explicitly assume that all individuals in the transit system may interact with each other and so include the transit system as a single additional location where infection may occur. The goal of the study is to investigate the influence that the transportation system (including the time spent in the transit system, the proportions of the susceptibles and the infectives who travel, and the health condition of the location where infection may occur) and the characteristics of the transportation system (such as gates and service counters in the transportation hub) assigned to different routes at different times have on the epidemic outbreak. The effects of different controlling strategies such as discouraging unnecessary travel during an epidemic, regular disinfecting of the public transportation system environment, installing ventilation equipment, and installing clinical thermometers at the entrances of the inter-city public transportation system are also discussed.

Using a modified next generation method, the basic reproduction numbers are calculated precisely for the cases of identical cities with arbitrary incidence rates and of non-identical cities with standard incidence. In this context, it is shown that it is possible for an outbreak to occur for the connected system even though the isolated regions cannot support the disease. However, if the transit system is sufficiently efficient (i.e., as $p \to \infty$) or if the population travels sufficiently rarely (i.e., as $\phi \to 0^-$), then the prospect of disease outbreak for the linked regions is the same as for a single isolated region; that is, the basic reproduction number for the meta-population model is greater than one precisely when the basic reproduction of an isolated region is greater than one.

On the other hand, simply reducing the likelihood that infectives travel (i.e., letting $z$ decrease), even to zero, may not be sufficient to prevent an outbreak (cf. Fig. 1(d)) where a significant decrease in $z$ results in only a marginal decrease in disease prevalence. Instead, even if the disease would die out in isolated regions and infectives are prevented from travelling, an epidemic could be maintained by an infective population within the transit system. Here, infective travelers still conclude their travel like everyone else, but on average infect enough susceptibles during their normal travel that an outbreak occurs. This possibility requires transmission to be relatively easy within the transit system (i.e., $\beta_f$ or $\phi$ is large, or $p$ is small) and also requires infectives to be already within the transit system before control measures are put in place to prevent them from entering. (For a model with an exposed or latent phase, this may not be the case.)

In the case where the cities or regions differ in size and the contact rate takes the general form, the basic reproduction number $R_0$ is estimated (Theorem 1). A condition on the parameters, which guarantees the stability of the disease-free equilibrium, is given, as is another condition, which guarantees its instability.

In Figs. 2(a) and 3(a), the disease outbreak occurred earliest in the largest city, even though the disease was introduced in a smaller region. Potentially, early detection of outbreaks may benefit from focusing resources on the more populated regions, especially if the transmission rates are small enough that the disease cannot spread in the isolated cities. Further studies should be performed to explore the validity of this notion in a range of meta-population models.

The analysis here is based on a coupled SIRS model. The corresponding results for a coupled SIS model can be obtained by allowing $\eta_1$ and $\eta_2$ to tend to infinity. We note, though, that we have performed the same analysis on the SIS model, and obtained these results directly.

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