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Global stability of a transport-related infection model with general incidence rate in two heterogeneous cities

Lili Liu, Xianning Liu*

Key Laboratory of Eco-environments in Three Gorges Reservoir Region (Ministry of Education), School of Mathematics and Statistics, Southwest University, Chongqing 400715, China

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ABSTRACT

To further understand the effects of travel on disease spread, a transport-related infection model with general incidence rate in two heterogeneous cities is proposed and analyzed. Some analytical results on the global stability of equilibria (including disease free equilibrium and endemic equilibrium) are obtained. The explicit formula for the basic reproduction number R_0 is derived and it is proved to be a threshold for disease spread. To reveal how incidence rate and travel rate influence the disease spread, effects of general incidence rate and travel rate on the dynamics of system are shown via numeric simulations.

1. Introduction

Mathematical epidemiology, as the study field on the spread of diseases based on mathematical models, mainly aims at exploring factors that cause their occurrence, predicting the dynamics of infection and figuring out whether there is an effective measure to control their spread. Kermark and Mckendrick (1927) built up a system of ordinary differential equations to study disease transmission firstly. And then many epidemic models were widely proposed and studied (Kribs-Zaleta and Velasco-Hernández, 2000; Feng and Thieme, 1995; Sun and Yang, 2010), in which travels of population were not assumed. However, travel, as a common phenomenon in human society, plays a significant role on the spread of diseases such as in the case of influenza and SARS. In 2003, SARS emerged in Guangdong province of China and broke out at last in almost all parts of China and some other cities in the world due to the travel of infected individuals, and therefore is considered as one of main factors causing the outbreak of SARS (Wang and Ruan, 2004). More recently, the H1N1 influenza virus appeared in Mexico in 2009 and soon spread to other countries around the world (Yang and Xiao, 2010). Thus, it is more pragmatic to consider travels of population in epidemic models.

Hethcote (2000) introduced an epidemic model with population dispersal between two patches. Sattenspiel and Dietz (1995) discussed a model with the consideration of mobility among people and Sattenspiel and Herring (1998) applied the same model to the transmission of measles in the Caribbean subarctic, which can be thought of a closed population where travel is easily quantified. Experts have also discussed other models for the spread of diseases among two patches or n patches to show the population dispersal dynamics via the specific examples (Wang and Mulone, 2003; Wang and Ruan, 2004; Wang and Zhao, 2004). Allen et al. (2007) studied an SIS model with standard incidence rate on population dispersal among n patches and suggested that movements of susceptibles or infected individuals had the effects of enhancing or suppressing the spread of diseases, which depended on the heterogeneity and connectivity of the spatial environment. Furthermore, Arino et al. (2007) formulated a general SEIRS for multi-species on multi-patches and discussed the role of quarantine in the form of travel restriction.

* Corresponding author. Tel.: +86 23 68254726.
E-mail address: liuxn@swu.edu.cn (X. Liu).

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Note that all these researches ignored the possibility for the individuals to become infective during travel. Cui et al. (2006) proposed an SIS epidemic model with the effect of transport-related infection on disease spread as follows:

$$\begin{cases} S_j' = a - bS_j + dI_j - \beta N_j^{-1} S_j I_j - \alpha S_j + \alpha S_k - \gamma\alpha N_k^{-1} S_k I_k, \\ I_j' = \beta N_j^{-1} S_j I_j - (c + d + \alpha) I_j + \alpha I_k + \gamma\alpha_k N_k^{-1} S_k I_k, \end{cases} \tag{1}$$

for $j, k \in \{1, 2\}$ and $j \neq k$. System (1) showed that transport-related infection intensified the disease spread if infectious diseases broke out to cause an endemic situation in each city. However, (1) assumed that two cities were homogeneous, i.e. they had the same parameter values and the same pattern of infection (standard incidence rate) both within each city and during travel, which may be not so convincing in the real scenario. Wang and Yang (2011) formulated the following model

$$\begin{cases} S_j' = a_j - b_j S_j + d_j I_j - \beta_j N_j^{-1} S_j I_j - \alpha_j S_j + \alpha_k S_k - \gamma_k \alpha_k^2 S_k I_k, \\ I_j' = \beta_j N_j^{-1} S_j I_j - (b_j + d_j + \alpha_j) I_j + \alpha_k I_k + \gamma_k \alpha_k^2 S_k I_k, \end{cases} \tag{2}$$

for $j, k \in \{1, 2\}$ and $j \neq k$. System (2) considered that two cites were heterogeneous and the incidence rates both within each city and during travel were different, and revealed the importance of border screening. Based on the above discussions, this paper aims to extend current two-city transport-related infection in two ways. For one thing, two cities are heterogeneous and have different demographic parameter values. For another, a general incidence rate is introduced. To the best of our knowledge, no such work has been done for the models considering the effects of general incidence rate on the spread of transport-related infection in heterogeneous cities.

This paper is organized as follows. A transport-related infection model with general incidence rate in two heterogeneous cities is introduced in the next section. In Section 3, the basic reproduction number is calculated and the global dynamics are studied. In Section 4, some comparisons of the main results with those in Cui et al. (2006) and Wang and Yang (2011) are made and some interesting simulations are provided. And it the last section, our model and results are briefly discussed and some concluding comments are given.

2. Model formulation

We now introduce a transport-related infection model. Assume that there are two heterogeneous cities and we divide the total population N of each city into two compartments: the susceptible S, who are healthy individuals able to contract the infection; and the infected I, who are infectious individuals able to spread the disease. Then $N = S + I$.

The incidence rate has been modelled by using many forms in the literatures, in which mass action incidence rate βSI and standard incidence rate $\beta N^{-1} SI$ are commonly used (Kermark and Mckendrick, 1927; Kribs-Zaleta and Velasco-Hernández, 2000; Feng and Thieme, 1995; Hethcote, 2000; Cui et al., 2006; Diekmann et al., 1990; Liu and Takeuchi, 2006; Liu et al., 2011). A general incidence rate (proposed in Hethcote (2000)) takes the form of $\beta(S+I)^{\theta-1}SI$, where $\theta \in [0, 1]$. This general incidence rate is more realistic since it can model the incidence rates between the standard incidence corresponding to $\theta = 0$ and the mass action incidence corresponding to $\theta = 1$. This means that the two incidences are two extreme cases. Obviously, this general incidence characterizes the continuous transitions from the mass action incidence to the standard incidence and can simulate behavior changes of populations from random mobility in a fixed area to the mobility with a fixed population density (Li et al., 2007). Thus, it is more practical and precise to describe the disease of infection according to this general incidence. Motivated by the above ideas, we assume that the incidence rate is modelled by the form $\beta(S+I)^{\theta-1}SI$ for $\theta \in [0, 1]$.

Denote S_j and I_j as the number of susceptible and infected individuals in city j ($j \in \{1, 2\}$), respectively. Let a_j be the recruitment rate, b_j the natural death rate, d_j the recovery rate of the infected individuals. The disease is transmitted horizontally in city j according to the incidence rate $\beta_j N_j^{\theta_C - 1} S_j I_j$, where β_j is the transmission rate in city j and $\theta_C \in [0, 1]$. The susceptible individuals of city j leave to city k at the rate α_j. When the individuals travel from city j to city k, disease is transmitted with the incidence rate $\gamma_j(\alpha_j N_j)^{\theta_T - 1}(\alpha_j S_j)(\alpha_j I_j) = \gamma_j \alpha_j^{\theta_T + 1} N_j^{\theta_T - 1} S_j I_j$, where γ_j is the transmission rate during travel from city j to city k and $\theta_T \in [0, 1]$. From the biological point of view, the new infection during travel should not be more than the traveling susceptible, namely, $\alpha_j S_j - \gamma_j \alpha_j^{\theta_T + 1}(S_j + I_j)^{\theta_T - 1} S_j I_j \geq 0$. We will give conditions to ensure this inequality always holds in the following Remark 2.1. Assume that there is no additional mortality induced by disease and the latent period of disease is negligible. These assumptions lead to the dynamical system as follows:

$$\begin{cases} S_j' = a_j - b_j S_j + d_j I_j - \beta_j N_j^{\theta_C - 1} S_j I_j - \alpha_j S_j + \alpha_k S_k - \gamma_k \alpha_k^{\theta_T + 1} N_k^{\theta_T - 1} S_k I_k, \\ I_j' = \beta_j N_j^{\theta_C - 1} S_j I_j - (b_j + d_j + \alpha_j) I_j + \alpha_k I_k + \gamma_k \alpha_k^{\theta_T + 1} N_k^{\theta_T - 1} S_k I_k, \end{cases} \tag{3}$$

for $j, k \in \{1, 2\}$ and $j \neq k$. The initial conditions are given by

$$S_j(0) > 0, \quad I_j(0) \geq 0, \quad I_1(0) + I_2(0) > 0. \tag{4}$$

From system (3), the total population is described by the equations

$$N_j' = a_j - (b_j + \alpha_j) N_j + \alpha_k N_k, \quad j, k \in \{1, 2\}, \quad j \neq k. \tag{5}$$

For system (5), we have the following lemma:

Lemma 2.1. *System (5) admits a unique positive equilibrium which is globally asymptotically stable.*

Proof. It is easy to obtain the unique positive equilibrium of system (5), denoted by (N_1^*, N_2^*), where

$$N_1^* = \frac{a_1(b_2 + \alpha_2) + a_2\alpha_2}{b_1\alpha_2 + b_2\alpha_1 + b_1 b_2}, \quad N_2^* = \frac{a_2(b_1 + \alpha_1) + a_1\alpha_1}{b_1\alpha_2 + b_2\alpha_1 + b_1 b_2}.$$