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How the distance between regional and human mobility behavior affect the epidemic spreading

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HIGHLIGHTS

- The distance between regions and human mobility behavior are considered in the epidemic spreading.
- The epidemic threshold is theoretically calculated.
- The results show that wherever the virus originates from, the final infection size is similar.

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ABSTRACT

The distance between different regions has a lot of impact on the individuals' mobility behavior. Meanwhile, the individuals' mobility could greatly affect the epidemic propagation way. By researching the individuals' mobility behavior, we establish the coupled dynamic model for individual mobility and transmission of infectious disease. The basic reproduction number is theoretically obtained according to the next-generation matrix method. Through this study, we may get that the stability state of the epidemic system will be prolonged under a higher commuting level. The infection density is almost the same in different regions over a sufficiently long time. The results show that, due to the individual movement, the origin of virus can only speed up or delay the outbreak of infectious diseases, however, it have little impact on the final infection size.

1. Introduction

Human behavior is the driving force behind many complex social phenomena. The understanding of human behavior has always been the focus of sociology, psychology and economics. Quantitative analysis of human behavior is an important research topic of modern science [1–4]. Studying the human behavior could not only improve the human's understanding of their own behaviors, but improve the human's understanding for the social system.

Human society has always been in the long struggle with infectious diseases. Many of infectious diseases including Ebola virus, pestilence, influenza, plague, AIDS, cholera, SARS and avian flu spread over many areas of the world, and kill tens of thousands of people [5–7]. Therefore, researchers try their best to cover the epidemic spreading mechanism and find the best way to control the epidemic. The wanton transmission of infectious diseases will not only endanger the health of mankind itself, but bring great disaster to the people's livelihood [8–10]. Large-scale outbreak of each infectious disease in the history of mankind has brought physical and psychological suffering to people, while having a great hindrance on the development of human society [11]. In addition, the deterioration of the natural environment and the rapid development

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of social factors do not only provide opportunities for the transmission of existing and new infectious diseases, but lead to the emergence of the virus or bacteria variations, which will seriously endanger human life [12]. With the in-depth exploration of human behavior, some studies that involve the relationship between human behavior and transmission of infectious disease have been proposed in succession [13–15]. However, the ability to fully understand human behavior and study its impact on transmission of infectious disease remains an important project. By this study, to some extent, we could strengthen the human's recognition for behavioral evolution and formulate some significant strategies to curb the epidemic spread. Scholars have adopted a variety of theoretical methods to study the epidemic spreading, such as the percolation theory, mean field theory, game theory, stochastic processes, cellular automaton, etc. [16–20]. As the small-world network and the scale-free network are discovered, scholars have proposed a number of epidemic spread models that perform on those complex networks. The most classic models include: (1) the mean field theory proposed by Pastor-Satorras and Vespignani [21]; (2) the percolation model proposed by Newman [22]; (3) the discrete probability model proposed by Wang et al. [23]. Kihong investigate the generalized epidemic process on modular networks, the results show that the system exhibits a bond-percolation type continuous phase transition for weak social reinforcement [24]. Samue explore the effect of a prudent adaptive behavior on disease transmission. Their results indicate the effects of the prudent could accelerate spread [25]. Han research the epidemic process on activity-driven modular networks, their obtain that the final infected density in the original-infected-community shows different trends with the change of the response strength of vaccination and the spreading rate [26].

Based on the above analysis, this paper studies the influence of individual mobility on epidemic spreading. Firstly, a coupled dynamic model between individual mobility behavior and transmission of infectious diseases is established. Then, the next-generation matrix method is used to calculate the basic reproduction number. Finally, we perform the epidemic spreading on network to study the outbreak time and final epidemic size. The structure of this paper is as follows: in the second section, the new epidemic model is presented, and the basic reproduction number is theoretically calculated. The commuting level, infection rate and recovery rate are numerical studied in the third section. Finally, Section 4 gives the conclusion.

2. Epidemic spreading model

In this section, we examine the effect of regional distance and human mobility behavior on epidemic spreading. According to the actual connectivity between regions, each region is regarded as a node in the network, and the people in this region are evenly mixed. That is to say, an individual in the same place has the same probability to communicate with any other individuals. The individuals in different places can access to any place of the connected networks. However, according to the actual situation, the probability for each individual to stay in different places is different. Here, the commuting form of an individual between place i and place j is set as $p_{ij} = \frac{1}{(1+d_{ij})^{\alpha}}$, which means that the probability for individuals in place i to stay in (access) the place j is p_{ij}. d_{ij} refers to the shortest distance between the place i and j; $\alpha \geq 0$ is a parameter, indicating the commuting level. To some extent, the commuting level indicates the degree that an individual would like to stay in the original region. In general, people would stay in the original region all the time while $\alpha = +\infty$. Without loss of generality, p_{ij} is normalized as follows:

$$\bar{p}_{ij} = p_{ij}(\sum_{j} p_{ij})^{-1} = (1 + d_{ij})^{-\alpha}(\sum_{j}(1 + d_{ij})^{-\alpha})^{-1} \tag{1}$$

It can be easily obtained from the formula (1) that $\sum_j \bar{p}_{ij} = 1, \ \ i = 1, 2, \ldots$. And we may get that there is almost no possibility for the individuals in the i place to stay in the j place, when $d_{ij} \to \infty$.

Due to the fact that individuals may be repeatedly infected with some flu viruses, we here consider the *SIRS* epidemiological transmission model, where S represents susceptible persons, I represents infected persons, and R represents recovery. Based on individual's movement behavior, the coupled epidemic spreading dynamic model is established as follows:

$$\begin{cases} \dfrac{dS_i}{dt} = -\sum\limits_{j=1}^{n}[S_i \text{ infected in community } j] + \gamma_i R_i \\ \dfrac{dI_i}{dt} = -\mu_i I_i + \sum\limits_{j=1}^{n}[S_i \text{ infected in community } j] \\ \dfrac{dR_i}{dt} = \mu_i I_i - \gamma_i R_i \end{cases} \tag{2}$$

Where, S_i, I_i and R_i refer to the susceptible person, infected person and recovery person in the i place, respectively. [S_i infected in place j] indicate the infection density of susceptible persons who comes from i and are infected in the place j; μ_i refers to the recovery rate in the place i; γ_i refers to the recovery rate in the place i, namely the probability that recovery person becomes susceptible person again due to loss of immunity to infectious disease.

Based on the individuals mobility behavior in different places, we can obtain the expression of the density of susceptible persons who comes from i and are infected in the place j:

$$[S_i \text{ infected in place } j] = \beta_j \bar{p}_{ij} S_i \frac{\sum_{k=1}^{n} \bar{p}_{kj} I_k}{\sum_{k=1}^{n} \bar{p}_{kj} N_k} \tag{3}$$

Where β_j indicates the probability that all susceptible persons who stay in the place j are infected; $\bar{p}_{ij} S_i$ refers to the susceptible density who comes from i and stay in the j; $\sum_{k=1}^{n} \bar{p}_{kj} I_k$ indicates the total infections in the place j; $\sum_{k=1}^{n} \bar{p}_{kj} N_k$ indicates the total density in the place j, and N_k indicates the population density of the place.

Let $S = (S_1, S_2, \ldots, S_n)^T$, $I = (I_1, I_2, \ldots, I_n)^T$, $\bar{\mathbf{P}} = [\bar{p}_{ij}]_{n\times n} = [(1+d_{ij})^{-\alpha}(\sum_j (1+d_{ij})^{-\alpha})^{-1}]_{n\times n}$, $\boldsymbol{\beta} = diag(\beta_1, \beta_2, \ldots, \beta_n)$, $\mathbf{N} = diag(N_1, N_2, \ldots, N_n)$ and $\boldsymbol{\mu} = diag(\mu_1, \mu_2, \ldots, \mu_n)$. Thus, the formula (3) is changed as follows:

$$\begin{cases} \dfrac{dS}{dt} = -S\bar{\mathbf{P}}\boldsymbol{\beta}(\bar{\mathbf{P}}\mathbf{N})^{-1}\bar{\mathbf{P}}I \\ \dfrac{dI}{dt} = -\boldsymbol{\mu}I + S\bar{\mathbf{P}}\boldsymbol{\beta}(\bar{\mathbf{P}}\mathbf{N})^{-1}\bar{\mathbf{P}}I \\ \dfrac{dR}{dt} = -\boldsymbol{\mu}I \end{cases} \tag{4}$$

Based on the formula (4), the disease-free equilibrium point $Q^0 = (S_1^0,\ S_2^0, \ldots, S_n^0,\ 0,\ 0, \ldots, 0,\ 0)$ can be calculated. Then, the next-generation matrix algorithm is used to calculate the basic reproduction number of the presented model, as shown below:

$$\Re^0 = \rho(\mathbf{M}) \tag{5}$$

Where $\rho(\mathbf{M})$ refers to spectral radius of $\mathbf{M}$, $\mathbf{M} = S^0\bar{\mathbf{P}}\boldsymbol{\beta}(\bar{\mathbf{P}}\mathbf{N})^{-1}\bar{\mathbf{P}}\boldsymbol{\mu}^{-1}$, $S^0 = (S_1^0,\ S_2^0, \ldots, S_n^0)$.

In order to obtain the basic reproduction number, it is critical to calculate the spectral radius of the next-generation matrix. However, the matrix $\mathbf{M}$ is arbitrary, it is impossible to use strict and accurate theoretical analysis. Therefore, we deduce some special expressions of the basic reproduction number. In general, for any two places i and j, there is always an path to connect the two regions in the real world, namely $0 \le d_{ij} < \infty$. People from those two places can communicate with each other. Thus, we just discuss how the value of α affects the epidemic spreading. The epidemiological spreading threshold for a place i is discussed below according to different cases.

(i) When $\alpha = 0$, $\bar{p}_{ij} \equiv n^{-1}$, $\quad 1 \le i,\ j \le n$. It can be obtained from formula (2) that

$$\begin{aligned} \frac{dI_i}{dt} &= -\mu_i I_i + \sum_{j=1}^{n} \beta_j \bar{p}_{ij} S_i \frac{\sum_{k=1}^{n} \bar{p}_{kj} I_k}{\sum_{k=1}^{n} \bar{p}_{kj} N_k} \\ &= -\mu_i I_i + \frac{1}{n} \sum_{j=1}^{n} \beta_j S_i \frac{\sum_{k=1}^{n} I_k}{\sum_{k=1}^{n} N_k} \end{aligned} \tag{6}$$

Thus, the basic reproduction number is $\Re^0 = \sum_{i=1}^{n} \frac{\sum_{k=1}^{n} \beta_k N_i}{n\mu_i \sum_{k=1}^{n} N_k}$. When $\Re^0 > 1$, the epidemic disease will spread throughout the place.

(ii) When $0 < \alpha < \infty$, it can be obtained from formula (2) that

$$\begin{aligned} \frac{dI_i}{dt} &= -\mu_i I_i + \sum_{j=1}^{n} \beta_j \bar{p}_{ij} S_i \frac{\sum_{k=1}^{n} \bar{p}_{kj} I_k}{\sum_{k=1}^{n} \bar{p}_{kj} N_k} \\ &= -\mu_i I_i + \sum_{j=1}^{n} \beta_j \bar{p}_{ij} S_i \frac{\sum_{k=1,\ k\neq i}^{n} \bar{p}_{kj} I_k}{\sum_{k=1}^{n} \bar{p}_{kj} N_k} + I_i \sum_{j=1}^{n} \beta_j \bar{p}_{ij} S_i \frac{\bar{p}_{ij}}{\sum_{k=1}^{n} \bar{p}_{kj} N_k} \\ &> -\mu_i I_i + I_i \sum_{j=1}^{n} \beta_j \bar{p}_{ij} S_i \frac{\bar{p}_{ij}}{\sum_{k=1}^{n} \bar{p}_{kj} N_k} \end{aligned} \tag{7}$$

Let $\Re_i^0 = \mu_i^{-1} N_i \sum_{j=1}^{n} \beta_j \bar{p}_{ij} \frac{\bar{p}_{ij}}{\sum_{k=1}^{n} \bar{p}_{kj} N_k}$, we further obtain:

$$\left(\frac{dI_i}{dt}/I_i\right)|_{I_i=0} = (\Re_i^0 - 1)\mu_i = N_i \sum_{j=1}^{n} \beta_j \frac{\left((1+d_{ij})^{-\alpha}(\sum_j (1+d_{ij})^{-\alpha})^{-1}\right)^2}{\sum_{k=1}^{n} (1+d_{kj})^{-\alpha}(\sum_j (1+d_{kj})^{-\alpha})^{-1} N_k} - \mu_i \tag{8}$$

When $\Re_i^0 > 1$, $(\frac{dI_i}{dt}/I_i)|_{I_i=0} > 0$, Thus, $I(\infty) > 0$, Thus when $\Re_i^0 > 1$, there are always persons infected with epidemic disease in place i.

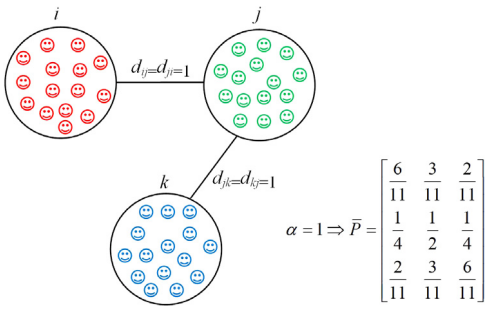

Fig. 1. Epidemiological transmission diagram that includes three places i, j and k. According to the distance among various communities, the mobile probability matrix $\bar{P}$ could be calculated when $\alpha = 1$.

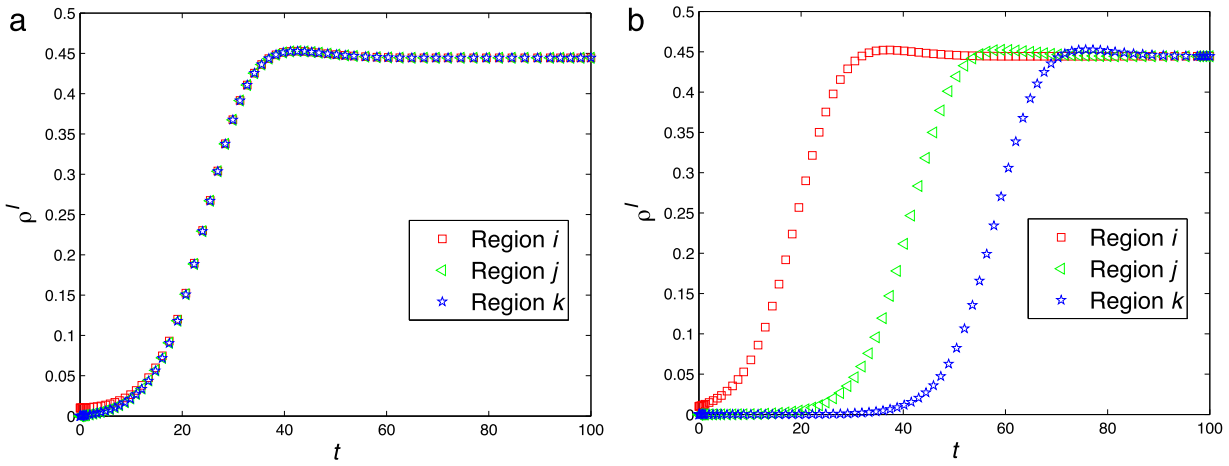

Fig. 2. Change chart of infected person density ρ^I over time in different places and at different commuting levels. At the initial time, the infectious disease emerges in place i, and the infection rate, recovery rate and restoration rate of each place are: $\beta_1 = \beta_2 = \beta_3 = 0.3$, $\mu_1 = \mu_2 = \mu_3 = 0.1$, $\gamma_1 = \gamma_2 = \gamma_3 = 0.2$. (a) commuting level $\alpha = 1$; (b) commuting $\alpha = 10$.

(iii) When $\alpha = \infty$, $\bar{p}_{ii} = 1$, $\bar{p}_{ii} = 0$, $i \neq j$, which indicates that there is no communication among individuals in the place. It can be obtained according to formula (2) that

$$\begin{aligned}\frac{dI_i}{dt} &= -\mu_i I_i + \sum_{j=1}^{n} \beta_j \bar{p}_{ij} S_i \frac{\sum_{k=1}^{n} \bar{p}_{kj} I_k}{\sum_{k=1}^{n} \bar{p}_{kj} N_k} \\ &= -\mu_i I_i + \beta_i (N_i - I_i) \frac{I_i}{N_i}\end{aligned} \tag{9}$$

The basic reproduction number $\Re_i^0 = \frac{\beta_i}{\mu_i}$ can be easily obtained. That is to say, when $\Re_i^0 > 1$, there are always persons infected with epidemic disease in place i.

3. Simulation results

Next, this paper performs numerical analysis of the epidemic spreading in three places (as shown in Fig. 1). Assuming that the number of population in three places i, j and k is 10 000 at the initial time. It can be seen from Fig. 1 that the geographical position of community i and community k is symmetrical. Thus, the outbreak of disease in place i and place j is studied in the following. Assuming that at the initial time, there are 1% of the individuals are infected. ρ_i^I, ρ_j^I and ρ_k^I respectively represent the infected person density in regions i, j and k.

It can be seen from Fig. 2, that when the commuting level α is different, there is a great difference in the outbreak time in different regions. When α is small, it can be seen from Fig. 2(a) that the density of infected persons in the two places j and k that are far away from the initial outbreak place of the infectious diseases reaches its peak at a short time. The infection densities of such two places are almost the same. However, when α is large enough, it can be seen from Fig. 2(b) that the density of infected persons in the two places j and k which are far away from the initial outbreak place of the infectious

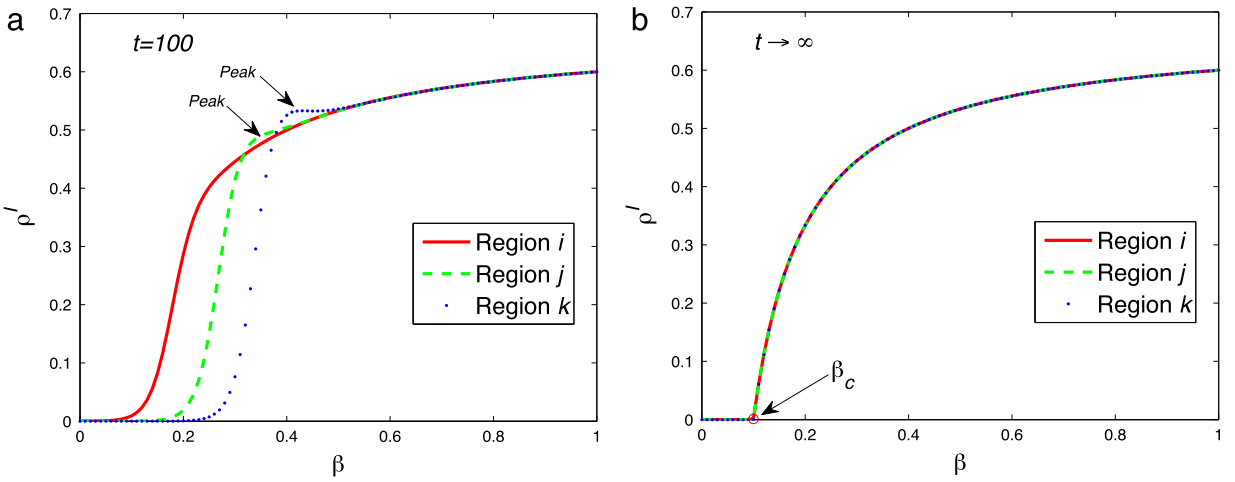

Fig. 3. Relationship between infection rate and transmission scale at different times. At the initial time, the infectious disease emerges in place i, and the recovery rate and restoration rate of each place are $\mu_1 = \mu_2 = \mu_3 = 0.1$, $\gamma_1 = \gamma_2 = \gamma_3 = 0.2$. Commuting level $\alpha = 10$. (a) The relationship between β and $\rho^I(t)$ at $t = 100$; (b) The relationship between β and $\rho^t(t)$ at $t \to \infty$.

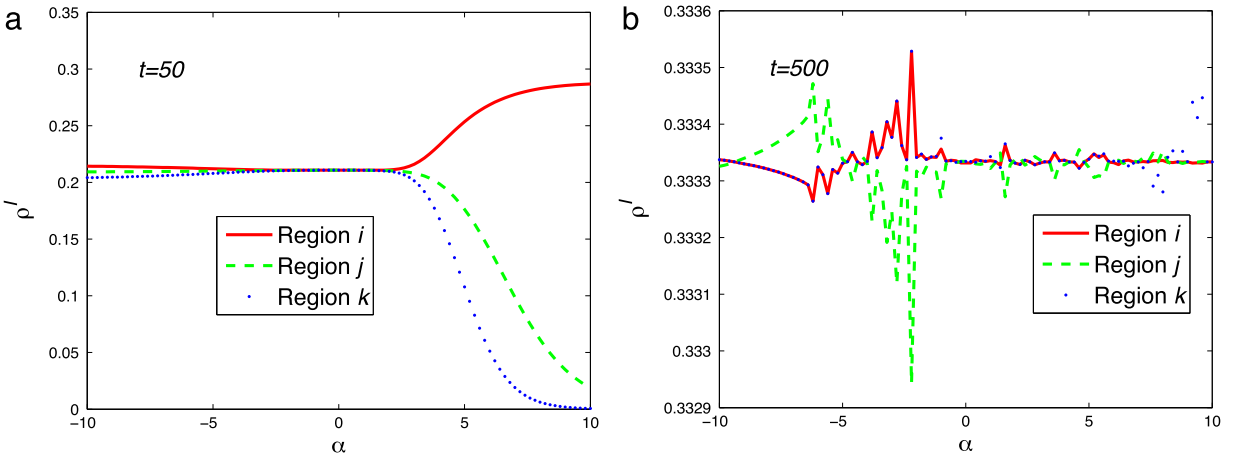

Fig. 4. Relationship between commuting level and infection density at different times. At the initial time, the infectious disease emerges in place i, and the infection rate, recovery rate and restoration rate of each place are $\beta_1 = \beta_2 = \beta_3 = 0.2$, $\mu_1 = \mu_2 = \mu_3 = 0.1$, $\gamma_1 = \gamma_2 = \gamma_3 = 0.2$. (a) The relationship between α and $\rho^I(t)$ at $t = 50$; (b) The relationship between α and $\rho^I(t)$ at $t = 500$.

diseases reaches its peak at a long time, and the infection density in place j is closer to the initial outbreak place. In addition, by comparing Fig. 2(a) and (b), we could find that the higher commuting level leads a longer time to reach stability state.

During the transmission of an infectious disease, it is necessary to focus on both the final number of infections and the number of infections in time. This can help us better to control the epidemic spreading. Assuming that the infection rate in each place is the same, the following sections will study the relationship between the infection rate β and the size of infectious diseases at different time. In general, the greater the infection rate is, the more likely the individual is infected. By comparing Fig. 3(a) and (b), it can be found that with the increase of the infection rate, the infection density in three places tends to be stable at a shorter time. When the commuting level α is larger, the individuals are reluctant to leave their hometown for another place. Thus, it can be found from Fig. 3(a) that the time for the epidemic size reaching peak is different in the three places. In addition, Fig. 3(b) illustrates that when the infection rate β is less than the threshold β_c, there will be no infected person in the last.

Commuting level α reflects whether the individual's desire to stay in the original place. A higher commuting level reveals the increasing tendency of individuals to stay in the original place of residence. Fig. 4 shows the variation of infection density $\rho^I(t)$ in different places is observed at different time points. Fig. 4(a) presents that as the commuting level increases, there is a large difference in the infection density among different places. When the observation time is short, the probability that the infected person leaves the original place of residence is less, that reduce the risk of infection in other places in the initial stage. However, it can be seen from Fig. 4(b), when the time is longer enough, the infection density is almost the same among

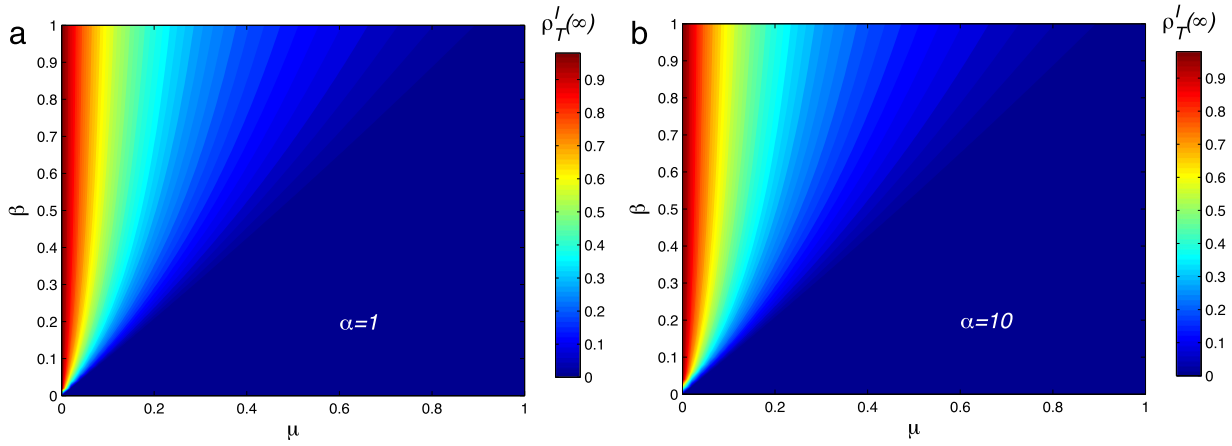

Fig. 5. The change of the final infection density $\rho_T^I(\infty)$ throughout the entire place over the changing infection rate and recovery rate at different commuting levels α. At the initial time, the infectious disease emerges in place i, and the recovery rate is $\gamma_1 = \gamma_2 = \gamma_3 = 0.1$. (a) $\alpha = 1$; (b) $\alpha = 10$.

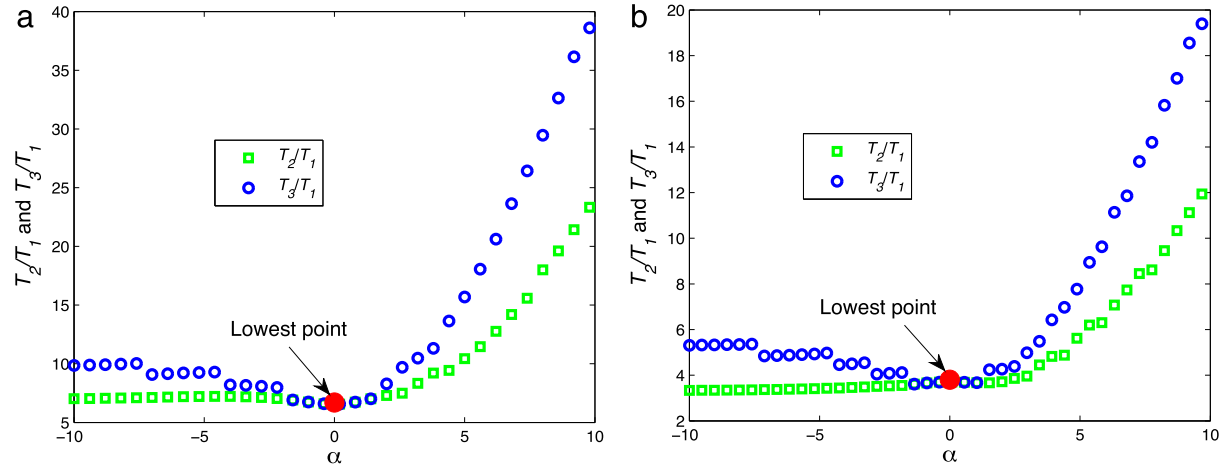

Fig. 6. Ratio of the time when the infection density of each place reaches 1% at different commuting levels α. At the initial time, the infectious disease emerges in place i, and the recovery rate and restoration rate of each place are $\mu_1 = \mu_2 = \mu_3 = 0.1$, $\gamma_1 = \gamma_2 = \gamma_3 = 0.2$. (a) Infection rate: $\beta_1 = \beta_2 = \beta_3 = 0.3$; (b) Infection rate: $\beta_1 = \beta_2 = \beta_3 = 0.5$.

different places. When commuting level α is larger, and if there are infected persons in a place, these infected people will stay here for a long time, which results in a significant increase of the infection density in this place.

Next, we investigate the change of the final infection density with the change of infection rate β and recovery rate μ at different commuting levels α. By comparing Fig. 5(a) and (b), it can be found that under different commuting levels and with a given infection rate β and recovery rate μ, the final infection density in all places is almost identical. In addition, according to Fig. 5(a) and (b), it can be found that in the plane $\beta - \mu$, the infection density is almost 0 blow the diagonal line. As the infection rate increases, the final infection density $\rho_T^I(\infty)$ gradually increases.

Assuming that the infection density of the place i is 1% at the initial time $T_1 = 1$, the ratio between T_1 and the time when the infection density of other places reaches 1% is studied then. T_1 and T_2 represent the time when the infection density reaches 1% in the places j and k. It can be seen from Fig. 6(a) and (b) that, almost when $\alpha = 0$, the minimum of T_2/T_1 and T_3/T_1 is reached. When $\alpha = 0$, the probability that individuals stay in any one place is the same, which maximize the efficiency by which the infected people arrive at any one place and stay in this place. Therefore, the time when the infection density reaches 1% in each place is minimized finally. In addition, by comparing Fig. 6(a) and (b) it can be found that with the increase of infection rate, the time when the infection density reaches its expected value in each place is greatly reduced.

In the following, we focus on the change of final infection density $\rho_T^I(\infty)$ under different recovery rates γ. It can be seen from Fig. 7 that with the increase of the recovery rate γ, the final infection density $\rho_T^I(\infty)$ gradually increases. In addition, at the same recovery rate γ, with the increase in infection rate β, the final infection density $\rho_T^I(\infty)$ is gradually improved. Even if there are individuals who are willing to stay in the original residence place, the whole regions will be influenced by the infectious disease. According to theoretical analysis, the basic reproduction number $\Re_i^0$ is not related to the recovery

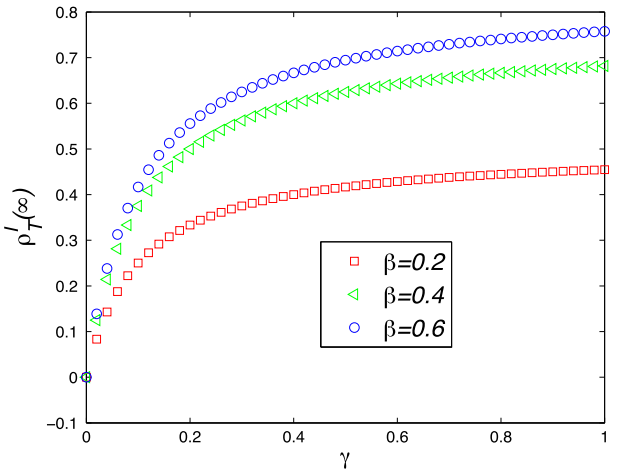

Fig. 7. Final infection density of all places at different recovery rates. At the initial time, the infectious disease emerges in place i, and the recovery rate and commuting level of each place are $\mu_1 = \mu_2 = \mu_3 = 0.1, \alpha = 1$. Here, $\beta_1 = \beta_2 = \beta_3 = \beta$.

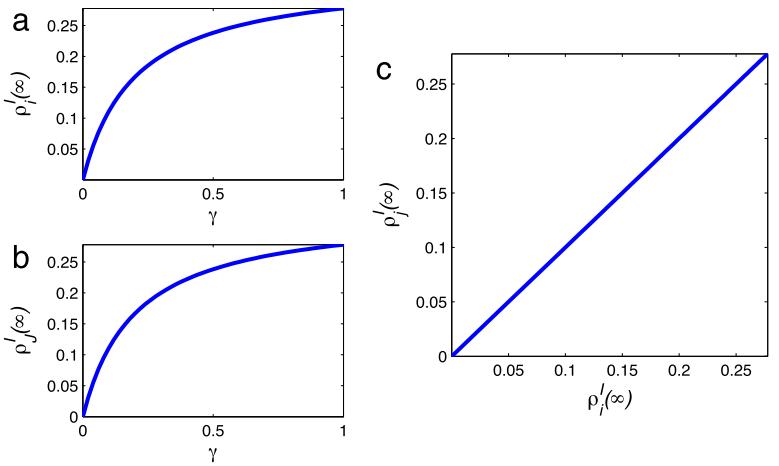

Fig. 8. The effect of the original infected regions on the final infection density with different recovery rates γ. $\rho_i^I(\infty)$ and $\rho_j^I(\infty)$ respectively refer to the total infection density of i and j, either of which is an initial infected place. The infection rate, recovery rate and commuting level are: $\beta_1 = \beta_2 = \beta_3 = 0.3$, $\mu_1 = \mu_2 = \mu_3 = 0.2, \alpha = 5$. (a) The relationship between the final total infection density $\rho_i^I(\infty)$ and the recovery rate γ; (b) The relationship between the final infection density $\rho_j^I(\infty)$ and the recovery rate γ. (c) The relationship between the final total infection density $\rho_i^I(\infty)$ and $\rho_j^I(\infty)$.

rate γ. However, it can be seen from Fig. 7 that if the recovered individual is likely to be infected again, the final number of infected persons in the entire areas will be greatly improved. Thus, the recovery rate has a big impact on the final epidemic size. However, if infected individuals are cured and they will not be infected again, the number of infected people in the entire area will be greatly reduced.

Next, the impacts of infectious disease that outbreaks in different places on the final infection density are compared. $\rho_i^I(\infty)$ and $\rho_j^I(\infty)$ represent the final infection density in place i and j, respectively. Either of which is an initial infected place. It can be seen from Fig. 8 that, at different recovery rates γ, the outbreak of initial infectious disease in different places almost has no impact on the final infection density. In other words, once the entire region is connected, the individual can travel between sub-regions. If there are no effective measures to be taken to prevent the epidemic spreading, the final epidemic size is almost kept at the same level. (It can be seen from Fig. 8(c) that the relationship between $\rho_j^I(\infty)$ and $\rho_i^I(\infty)$ is linear.) Thus, as long as the individuals' travels, the difference in initial infected places can only speed up or delay the epidemic outbreak.

4. Conclusions

To find an effective strategy to curb the epidemic spreading is a problem that should be solved quickly. Considering the human behavior and the regional distance, in this paper we establish an coupled epidemic spreading model. Using the next-generation matrix method, the basic reproduction number is theoretically calculated. The results show that the epidemic

will widely spread if the basic reproduction number $\Re_i^0 > 1$. Moreover, a higher commuting level will prolong the time to achieve the stability state. In addition, as the commuting level increases, the total density of infected individuals in each regions are different at a fixed observation time. However, when the time is sufficiently long, the infection density of all places is almost the same. Therefore, the difference of original infected places could only speed up or delay the outbreak of infectious disease.

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