Variations in epidemic distribution with some characteristic parameters*

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(Received 28 February 2012; revised manuscript received 5 April 2012)

Considering the spread of an epidemic among a population of mobile agents that can get infected and maintain the infection for a period, we investigate the variation in the homogeneity of the distribution of the epidemic with the remaining time of infection τ, the velocity modulus of the agent v, and the infection rate α. We find that the distribution of the infected cluster size is always exponential. By analyzing the variation of the characteristic infected cluster size coefficient, we show that the inhomogeneity of epidemic distribution increases with an increase in τ for very low v, while it decreases with an increase in τ for moderate v. The epidemic distribution also tends to a homogeneous state as both v and α increase.

Keywords: random walk, epidemic spread, homogeneity, cluster size

PACS: 89.75.–k, 87.23.Ge, 64.60.Ht **DOI:** 10.1088/1674-1056/21/7/078901

1. Introduction

Major outbreaks of several epidemics have been recorded in human history, e.g., the Black Death sweeping across 14th century Europe, SARS threatening the world in 2003, and bird flu hitting South-East Asia in 2004. The large-scale spread of epidemics has also occurred widely in animal populations. The spread of epidemics in populations has been studied for a long time. Recently, the rapid development of computer modeling techniques[1−5] has greatly improved the public's understanding of infection mechanisms. In these models, the individuals in a population are abstracted to agents, and each agent is assumed to be in either a susceptible state or an infected state.[6] Each susceptible agent can be infected by its infected neighbors with some probability, and each infected agent can recover to the susceptible state.

The agents were immobile in previous agent-based epidemic spread studies, so researchers only need to analyze the epidemic spread in static networks, where the nodes represent the agents and the links represent the interactions between the agents.[7−12] Later, mobile agent models[13−19] were introduced, since the individuals that may suffer and spread the epidemic usually move around in the real world. In such models, numerous agents representing the individuals move randomly in a limited square, and the agents can infect each other when they are close enough to each other.

In this study, we investigate the spread of an epidemic in the mobile agent model. Each susceptible agent can be infected by its infected neighbors in two modes. In Mode 1, a susceptible agent is infected with the probability of the infection rate α if it has at least one infected neighbor. In Mode 2, the probability increases with the number of its infected neighbors. Each infected agent can maintain the infection for a period before it becomes susceptible, and the remaining time of infection is denoted as τ hereinafter. This scenario concerns the fact that the infected individuals always need time to recover in the real world. The velocity modulus of the agent is denoted as v in this study.

When discussing epidemic spread, researchers often adopt the homogeneous mixing hypothesis, that is, each infected agent has the same probability of contact with any susceptible agent,[20] to obtain the analytical solution.[9,21−23] However, such a hypothesis is not strong and should be thoroughly discussed. In fact

*Project supported by the National Natural Science Foundation of China (Grant Nos. 61173183, 60973152, and 60573172), the Specialized Research Fund for the Doctoral Program of Higher Education of China (Grant No. 20070141014), and the Natural Science Foundation of Liaoning Province, China (Grant No. 20082165).

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our previous study has shown that, after the transition process, the distribution of an epidemic is always inhomogeneous.[24] In this study, we take a further step to investigate the change in the homogeneity of the epidemic distribution with τ, v, and α. The spatial distribution of the epidemic can be characterized by the infected cluster size,[19,24–26] namely, the number of infected agents that are connected together.

In the discussion, the Homogeneous Mode,[24] where all agents are static, infected and randomly distributed in the system, is used to represent the case where the epidemic is homogeneously distributed. To investigate the variation in the homogeneity of the epidemic spread, we introduce the characteristic infected cluster size coefficients of Mode 1 and Mode 2, which are defined as $\chi_1^{\rm C} = (\chi_1 - \chi_{\rm H})/\chi_{\rm H}$ and $\chi_2^{\rm C} = (\chi_2 - \chi_{\rm H})/\chi_{\rm H}$. Here, χ_1, χ_2, and $\chi_{\rm H}$ are the characteristic infected cluster sizes, which are the sizes of the largest infected clusters of Mode 1, Mode 2, and the Homogeneous Mode, respectively.

Firstly, we investigate the type of epidemic distribution by analyzing the infected cluster size distribution for various τ, v, and α, and find that the infected cluster size always follows an exponential distribution. By computing $\chi_1^{\rm C}$ and $\chi_2^{\rm C}$, we show that the inhomogeneity of the epidemic distribution increases with the increase in τ when v is very low. However, for moderate v, the epidemic distribution becomes more homogeneous with the increase of τ. When v is very high, the epidemic almost homogeneously spreads regardless of τ. And with the increases of v and α, the epidemic distribution becomes more homogeneous.

2. Model

N agents walk randomly in a square of linear size L with periodic boundary conditions. Each agent has two states: S (susceptible) and I (infected). Initially, these agents are distributed randomly in the square with randomly distributed directions. Some agents are infected at the seed of the infection, and all the others are susceptible. And from then on, their positions and states are updated at each time step.

The position and motion direction of agent i at time t are denoted as $\boldsymbol{x}_i(t)$ and $\theta_i(t)$, respectively. Agent i moves according to the following equation:

$$\boldsymbol{x}_i(t+\Delta t) = \boldsymbol{x}_i(t) + \boldsymbol{v}_i(t)\Delta t,$$
$$\theta_i(t+\Delta t) = \xi_i,$$

where $\boldsymbol{v}_i(t) = (v\cos\theta_i(t), v\sin\theta_i(t))$ is the velocity of the agent, and v is the modulus of the agent motion velocity and is the same for all agents. ξ_i follows the uniform distribution in $[-\pi, \pi]$. Δt is the update interval and is set to be 1. For agents i and j, if $|\boldsymbol{x}_i - \boldsymbol{x}_j| \leq r_0$, where r_0 is the interaction radius, then i and j are neighbors and can infect each other.

For agent i, if it is currently susceptible, then it can be infected in two modes: in Mode 1, it is infected with probability α if it has the infected neighbor(s),[7–9] and in Mode 2, it becomes infected with probability $1-(1-\alpha)^{k(i)}$, where $k(i)$ is the number of its infected neighbor(s).[10,14] For each infected agent, the infection lasts τ time steps in both Mode 1 and Mode 2. That is, the infected agent recovers to the susceptible state after it remains infected for τ time steps.

In this work, the number of agents N is fixed to be 200 and L is measured in units of the radius $r_0 = 1$. The density of the system is denoted as $\rho = N/L^2$. When the density ρ is very small, the probability that two agents are connected would also be very small. In this case, the epidemic is not easy to spread and tends to disappear. On the contrary, when the density is very high, the probability that two agents are connected is also very high. Thus, most agents would be connected together and would be infected even though the infection rate is small. Since N is fixed, we need to choose a proper value of L to ensure that the epidemic will neither disappear, nor spread throughout most of the network under varied conditions. In this work, the value of L is set to be 14.

The simulation results show that, when α is very low, e.g., α tends to 0, no epidemic exists in the steady state. For such a reason, we do not use very low α. When α and τ are very large, most agents are infected in the steady state. This is not realistic, so we do not use very large α and τ. Our simulations also show that, for any given τ and α, when v is higher than a certain value, the infected density does not change with v. Therefore, we do not use very high v either. In this work, τ is limited to [1, 7], α is limited to [0.3, 0.6], and v is limited to [0.01, 2].

3. Results

The infected proportion is 5% in the initial state of the system. After the transient process, the system stabilizes at a steady state, in which the infected density fluctuates narrowly around a value. We measure the system in its steady state. The infected densities of Mode 1 and Mode 2 are denoted as $\rho_1 = N_1/L^2$

and $\rho_2 = N_2/L^2$, where N_1 and N_2 are the numbers of infected agents in Mode 1 and Mode 2, respectively.

There are three parameters that affect the dynamical process of the epidemic spread quite directly, that is, the remaining time of infection τ, the velocity modulus of the agent v, and the infection rate α. In this study, we mainly discuss the effects of these three parameters on the epidemic distribution. As can be seen from the previous studies,[24–26] the distribution of the epidemic can be well described by the infected cluster size. Here, the cluster is the subnet, in which the agents are connected, the infected cluster is the cluster that only includes the infected agents, and the infected cluster size means the number of infected agents in the infected cluster.

Before discussing the variation in the epidemic distribution, we analyze the type of infected cluster size distributions $P_1(k)$ (Mode 1) and $P_2(k)$ (Mode 2), where k is the infected cluster size. In Figs. 1(a) and 1(b), the linear-log plots of the infected cluster size distributions ($P_1(k)$ and $P_2(k)$) are plotted, respectively. We can see that Figs. 1(a) and 1(b) show an approximate linear relationship. According to our examinations, the linear-log plots of the accumulative distributions of the infected cluster sizes in Mode 1 and Mode 2 are also approximately linear. These indicate that the infected cluster sizes follow the exponential distribution, namely, $P_1(k) \sim \mathrm{e}^{-\lambda_1}$ and $P_2(k) \sim \mathrm{e}^{-\lambda_2}$.

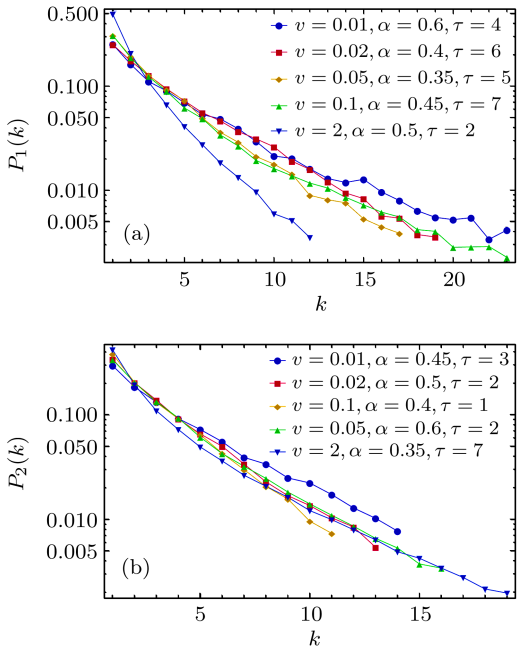


Fig. 1. (colour online) Linear-log plots of the infected cluster size distributions in Mode 1 (a) and Mode 2 (b). Each curve corresponds to a set of (v, α, τ) values. The error bars of the standard errors (not shown) are smaller than the symbol sizes. The results are averaged over 50 runs.

3.1. The variation of the epidemic distribution with τ

To investigate the homogeneity of the infection distribution, we introduce the Homogeneous Mode,[24] in which all agents are static, infected, and randomly distributed in the square. The infection distribution of the Homogeneous Mode is considered to be homogeneous, since the probability that each infected agent is located at any position is the same. According to our simulations, the distribution of the infected cluster size in the Homogeneous Mode is also exponential. Hereinafter, the infected density of the Homogeneous Mode is denoted as ρ_{H}. In this study, the largest infected cluster sizes of Mode 1, Mode 2 and the Homogeneous Mode are called the characteristic infected cluster sizes (CICSs), and are denoted as χ_1, χ_2 and χ_{H}, respectively. The simulations show that χ_1, χ_2 and χ_{H} increase with the increase of ρ_1, ρ_2 and ρ_{H}, respectively.

To discuss the change in the homogeneity of the epidemic distribution, we define $\chi_1^{\mathrm{C}} = (\chi_1 - \chi_{\mathrm{H}})/\chi_{\mathrm{H}}$ (with $\rho_1 = \rho_{\mathrm{H}}$) and $\chi_2^{\mathrm{C}} = (\chi_2 - \chi_{\mathrm{H}})/\chi_{\mathrm{H}}$ (with $\rho_2 = \rho_{\mathrm{H}}$) as the characteristic infected cluster size coefficients (CICSCs) of Mode 1 and Mode 2, respectively. For any infected density, the larger χ_1^{C} and χ_2^{C} are, the more inhomogeneous the epidemic distribution is.

Firstly, we investigate the influence of the remaining time of infection τ on the epidemic spread in Mode 1 and Mode 2. In Figs. 2(a) and 2(b), χ_1 and χ_2 are plotted as functions of τ, respectively. Different curves correspond to different v and α. It can be seen that χ_1 and χ_2 increase with the increase in τ. This is because the number of infected agents increases when the infection disappears more slowly. Moreover, since the infected agents only infect their neighbors, the infected clusters will become larger in the case where the other parameters remain unchanged.

In Fig. 3, χ_1^{C} and χ_2^{C} are plotted as functions of τ. In each plot, α is fixed, and different curves correspond to different v. We can see that, when $v < 0.02$, χ_1^{C} and χ_2^{C} increase with the increase of τ; when $0.02 < v < 2$, χ_1^{C} and χ_2^{C} decrease with the increase of τ; and when $v = 2$, χ_1^{C} and χ_2^{C} are close to 0 regardless of τ. The simulations show that when $v > 2$, χ_1^{C} and χ_2^{C} are also close to 0. This indicates that the inhomogeneity of the epidemic distribution increases with the increase of τ when $v < 0.02$, and decreases with the increase

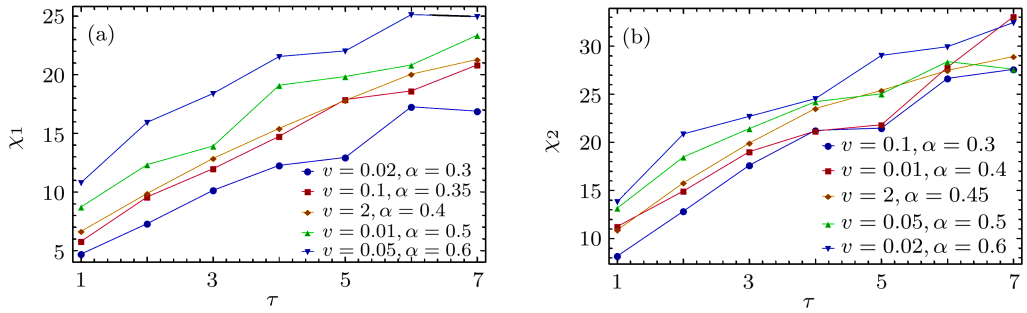


Fig. 2. (colour online) The characteristic infected cluster sizes (CICSs) as functions of the remaining time of infection τ in Mode 1 (a) and Mode 2 (b), respectively. Each curve corresponds to a set of (v, α) values. The error bars of the standard error (not shown) are smaller than the symbol sizes. The results are averaged over 50 runs.

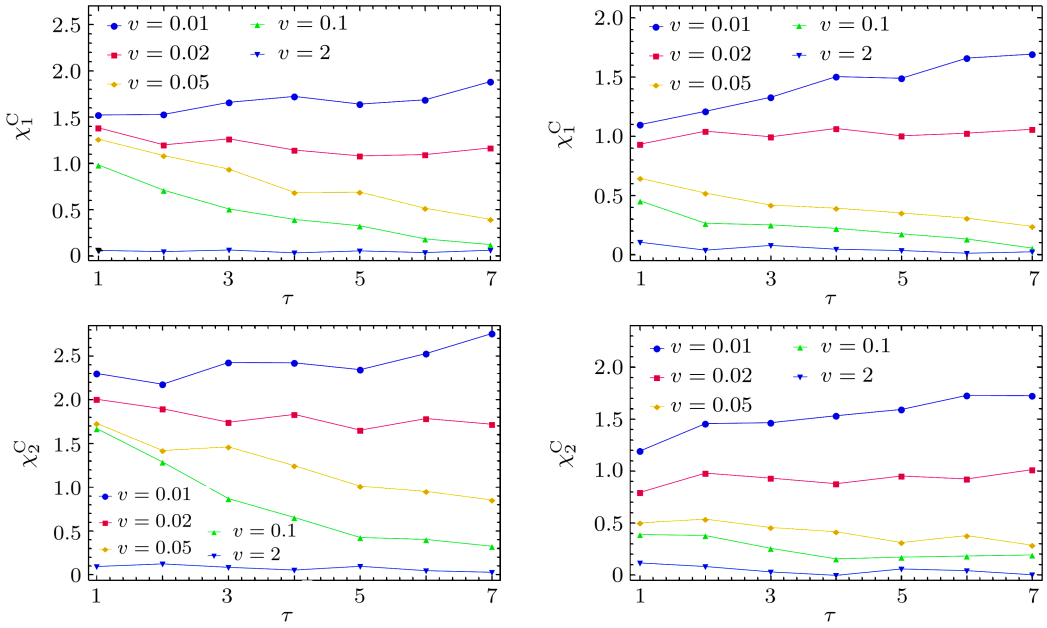


Fig. 3. (colour online) The characteristic infected cluster size coefficients (CICSCs) as functions of the remaining time of infection τ in Mode 1 ((a), (b)) and Mode 2 ((c), (d)), respectively. In each plot, α is fixed and each curve corresponds to a v value. The error bars of the standard errors (not shown) are smaller than the symbol sizes. The results are averaged over 50 runs.

of τ when $0.02 < v < 2$. In addition, when $v \geq 2$, the infection distribution is always homogeneous.

The reason for this is that when $v < 0.02$, the infected agents move little at each time step and their neighbors are almost unchanged. So the persistence of infection only enlarges the old infected clusters rather than causing new infected clusters. Thus, the increase in τ enhances the inhomogeneity of infection. When $0.02 < v < 2$, the infected agents can jump out of their infected clusters with a non-ignorable probability to cause the new infected clusters. Thus, when ρ_1, ρ_2, and ρ_H increase by the same amount, χ_1 and χ_2 increase less than χ_H. This leads to the result that χ_1^C and χ_2^C decrease with increasing τ. When $v \geq 2$, the infected agents always jump out of their infected clusters. This causes the infected agents to tend to be homogeneously distributed in the square regardless of τ.

3.2. The variation of the epidemic distribution with v and α

In this subsection, we first investigate the effect of the velocity modulus of the agent v on the epidemic spread in Mode 1 and Mode 2. χ_1 and χ_2 are plotted as functions of v for various τ and α in Figs. 4(a) and 4(b), respectively. It can be seen that χ_1 and χ_2 almost do not change with v. The reason for this fact is that along with the increase of v, the probability that the infected agents jump out of their infected

clusters also increases. Thus, they can infect more susceptible agents. On the other hand, the increasing v prevents the formation of the large infected clusters, so it does not enlarge the infected clusters. Moreover, $\chi_{\rm H}$ steadily increases with $\rho_{\rm H}$, so we can expect that $\chi_1^{\rm C}$ and $\chi_2^{\rm C}$ would both decrease with the increasing v. This is verified obviously as shown in Figs. 4(c) and 4(d).

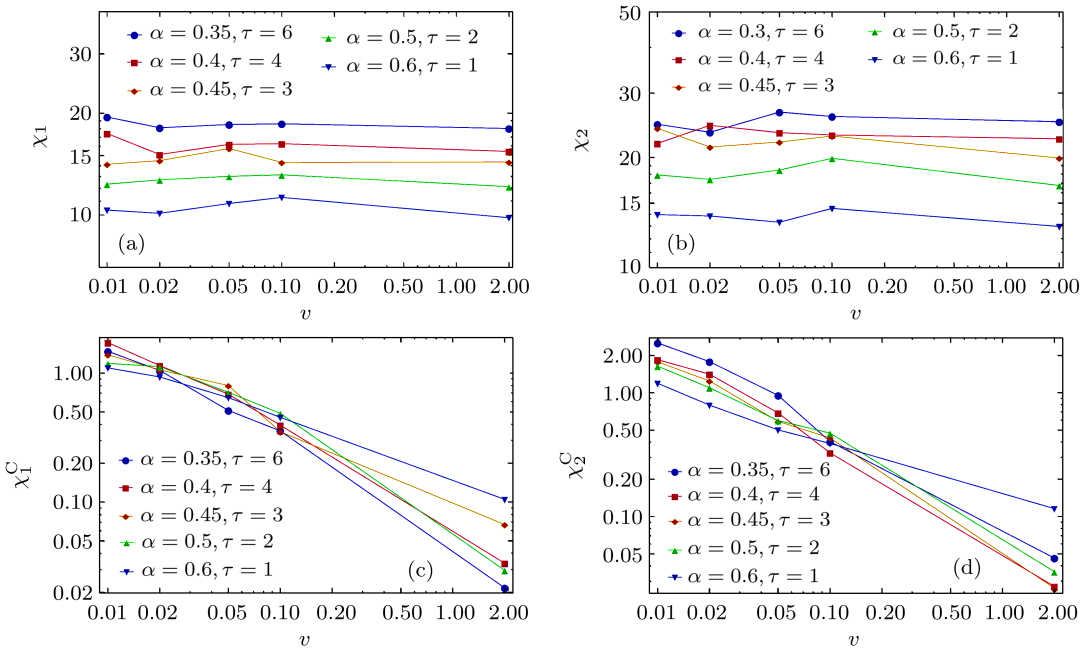


Fig. 4. (colour online) The characteristic infected cluster sizes (CICSs) and the characteristic infected cluster size coefficients (CICSCs) as functions of the velocity modulus of the agent v in Mode 1 ((a), (c)) and in Mode 2 ((b), (d)), respectively. Each curve corresponds to a set of (α, τ) values. The error bars of the standard errors (not shown) are smaller than the symbol sizes. The results are averaged over 50 runs.

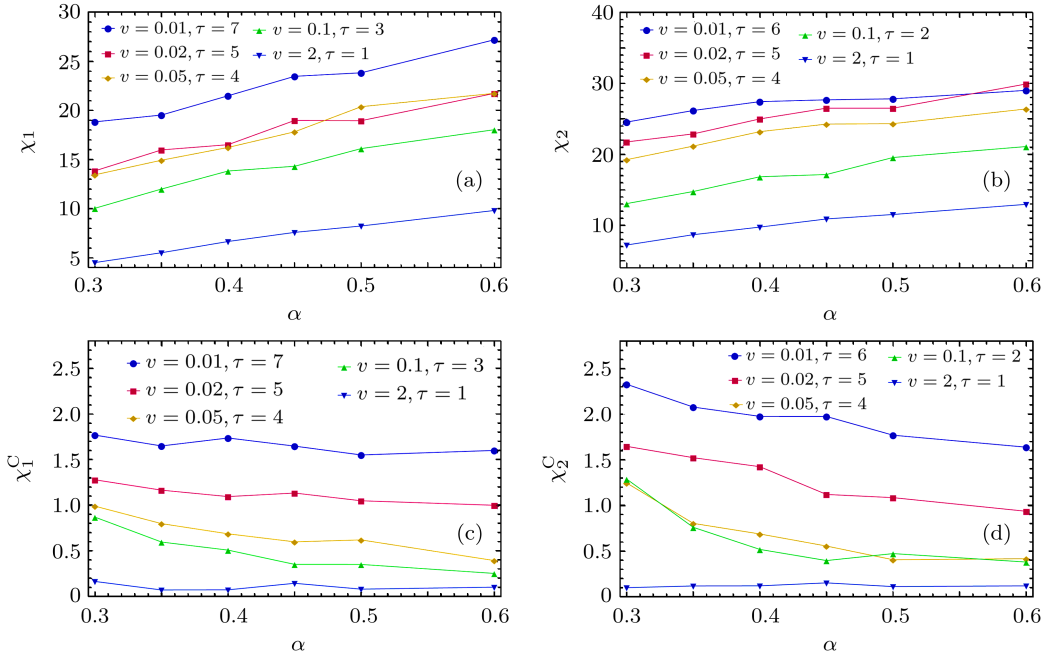


Fig. 5. (colour online) The characteristic infected cluster sizes (CICSs) and the characteristic infected cluster size coefficients (CICSCs) as functions of the infection rate α in Mode 1 ((a), (c)) and in Mode 2 ((b), (d)), respectively. Each curve corresponds to a set of (v, τ) values. The error bars of the standard errors (not shown) are smaller than the symbol sizes. The results are averaged over 50 runs.

Then we analyze the variation of the epidemic spread with the infection rate α in Mode 1 and Mode 2. In Fig. 5, χ_1, $\chi_1^{\rm C}$, χ_2, and $\chi_2^{\rm C}$ are plotted as functions of α for various v and τ, respectively. As shown in Figs. 5(a) and 5(b), χ_1 and χ_2 both increase with the increase of α. The reason for this is that when the infection rate α is high, the probability that a susceptible agent is infected by its infected neighbors is also high. Thus, more susceptible agents get infected. Moreover, the infected agents only infect their neighbors, so the CICS is also larger in the case where the other parameters remain unchanged. More simulations show that when ρ_1, ρ_2, and $\rho_{\rm H}$ increase by the same amount, χ_1 and χ_2 increase by a smaller amount than $\chi_{\rm H}$. This leads to the decrease of $\chi_1^{\rm C}$ and $\chi_2^{\rm C}$, and the more homogeneous distribution of the epidemic (see Figs. 5(c) and 5(d)).

4. Conclusions

In this paper, we investigate epidemic distribution by analyzing the infected cluster size when the infection of agents remains in τ time steps. It is found that the infected cluster size follows an exponential distribution. The simulation results show that the system parameters affect the homogeneity of the epidemic distribution in different ways: 1) for the fixed velocity modulus of the agent v and the infection rate α, the inhomogeneity of the epidemic spread increases with the increase of τ when v is very low, and decreases with the increase of τ when v is moderate; 2) when τ and α are fixed, the inhomogeneity of the epidemic distribution decreases with the increase of v; and 3) for constant τ and v, the inhomogeneity of the epidemic distribution decreases with the increase of α.

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