Identifying the direct risk source to contain epidemics more effectively

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We investigate the impact of people's perceptions regarding the risk of an epidemic by analyzing the differences between local and global risk perceptions on affecting the epidemic threshold. Three issues are introduced to explain such differences: the indirect risk source, the heterogeneous global risk, and heterogeneity in individuals' intrinsic susceptibilities. When the direct risk source is completely undetected, the local risk perception tends to have no effect on the epidemic threshold, and the effect of the local risk is nearly equivalent to that of the global risk perception, thereby also suggesting a reason why global risk perception cannot affect the epidemic threshold. However, there is a surprising effect of the global risk perception: When its heterogeneity is sufficiently high, an increased epidemic threshold value sometimes may lead to a greater infected ratio.

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I. INTRODUCTION

Individual risk perception is believed to be a key factor that influences the spread of epidemics [1]. Empirical studies have demonstrated that when people perceive risk from infectious diseases, they will take positive actions to protect themselves [2–7]. These behaviors can significantly help prevent a disease, such as severe acute respiratory syndrome [8] and acquired immunodeficiency syndrome [9], from resulting in a large-scale epidemic [10].

People are embedded in social networks, and their self-protective responses are highly influenced by others. Such influence mainly comes from two sources: globally available information and locally available information [11]. Global information is broadcasted by health authorities and public media and is available to everyone. In contrast, locally available information is often obtained from a social or spatial neighborhood. Examples include spreading information by word of mouth [12] and assessing infection risk from a social neighborhood [5], etc. Both local and global risk information are of great importance for containing an epidemic.

However, the effects of the two types of information differ. Wu *et al.* found that locally based awareness could influence the epidemic threshold (an index that quantifies whether an infection can survive long term [13]), whereas globally based awareness could not [14]. Scholars may doubt the base of such differences. We define an individual's direct risk sources as the people who have the potential to spread the virus to him or her (e.g., the infected neighbors) and define indirect risk sources as those who carry the virus but are unable to spread the virus directly (e.g., the infected people outside his or her personal network). As described above, the local risk often comes from direct risk sources, whereas the global risk is usually from indirect risk sources. If the direct risk sources cannot be detected by the susceptible people, the local risk perception may not affect the epidemic threshold. Moreover, in contrast with local information, global information is often more homogeneously distributed, and such homogeneity may lead to the result that the global risk perception does not affect the epidemic threshold.

Therefore, we address the following issues to explore the above problems. First, we assume that direct risk sources are detected with a certain probability. Many infectious diseases, such as influenza, have an incubation period during which virus-exposed individuals are not easy to identify but can infect others [15]. Moreover, some mildly infected people may not display any disease symptoms until they become severely infected. Second, heterogeneity can be introduced into the global risk perception. Previous models have usually assumed that all susceptible people have identical global risk perceptions [14,16]. In fact, because of different personal habits, various sorts of media, and governments' biased policies, people's global risk perceptions may be heterogeneously distributed [17]. Third, another heterogeneity that should not be ignored is the intrinsic susceptibilities of the individuals. Reports have demonstrated that some people, such as pregnant women, are more vulnerable to diseases [2]. In addition, susceptibility heterogeneity is often due to genetic factors [18,19] or previous encounters with antigenically similar pathogens [19,20].

We propose a susceptible - mildly infected - severely infected - susceptible (SIIS) model to investigate the impact of people's risk perceptions on the course of an epidemic. When the direct risk source is completely undetected, both the infected ratio and the epidemic threshold remain nearly unchanged even when more confidence is assigned to the local risk perception. Once the direct risk source could be identified, the epidemic threshold value is immediately enhanced and has a highly positive correlation with the local confidence. However, a larger threshold cannot ensure a better controlling effect when global risk perceptions are heterogeneously distributed. Moreover, introduction of heterogeneous initial susceptibilities significantly reduces the infected ratio and makes the network less vulnerable to an epidemic.

II. MODELS

We divide our model into two processes, epidemic spreading and risk perception, and assume the two processes proceeding on the same network topology. Thus each node simultaneously plays two roles: spreading disease and diffusing risk information.

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The network model adopted is the Barabási-Albert (BA) network [21], which exhibits a power-law degree distribution. Because of the large number of neighbors, the hubs in the BA network may perceive less risk than normal nodes when a certain number of neighbors are infected and may not pay sufficient attention to self-protection. Thus, local risk perception may be unable to prompt self-protective actions, which implies that global risk perception may be more important to a hub than a normal node. Therefore, we propose a form of degree-based global risk perception in a BA network to let the hub nodes perceive increased global risk.

A. Epidemic spreading

The epidemiological SIIS model, which is an extension of the susceptible-infected-susceptible (SIS) model [22,23], is adopted in our epidemic spreading process. The original infection state is divided into two parts based on the severity scale: mild infection and severe infection. People at the mild infection state may be asymptomatic, which makes them difficult to detect by susceptible people, but they carry the disease virus and may be highly infectious. The severely infected people, however, are isolated from the rest of the population because once they exhibit symptoms they are immediately taken to receive medical treatment [15]. Thus, the susceptible individuals catch the disease via direct contact with their mildly infected neighbors.

We assume mildly infected nodes to grow to severe infection at rate μ and the severely infected nodes recover to the susceptible state at rate γ. A susceptible node catches the disease from one infected neighbor at an infection rate λ. We introduce a parameter ω to represent the probability that the mildly infected nodes can be detected. Once being detected, the mildly infected nodes are isolated from the rest of the population and lose the ability to infect others. For simplicity, we assume that the infection rate becomes $(1-\omega)\lambda$. A susceptible node will catch the disease (or be mildly infected) according to the following probabilistic equation:

$$P_{S\to I} = 1-(1-(1-\omega)\lambda\phi)^{n_1}. \tag{1}$$

Here ϕ denotes the node's susceptibility, and n_1 represents the number of its mildly infected neighbors.

B. Risk perception

People's responses are closely connected with their risk perceptions and often shift as an epidemic progresses [24]. When people perceive higher risk, they take more effective actions, consequently becoming less susceptible to infection. Consistent with this view, Bagnoli *et al.* [1] proposed the form $\phi = e^{-Jz^{\theta}}$ to describe the susceptibilities of self-protected individuals, where z represents the value of perceived risk, J is the precaution measure level, and θ denotes the special prophylaxis. In this paper, we adopt the following simplified version [14]:

$$\phi = \nu(1-z_t), \tag{2}$$

where ν is the intrinsic (or natural) susceptibility of an individual and z_t represents hisor her perceived risk at time t, which consists of two parts: local risk perception and global risk perception. Thus, z_t can be interpreted in the following form:

$$z_t = \alpha x_t + \beta y_t. \tag{3}$$

Here x_t and y_t represent the local and global risk perception at time t. $\alpha \in [0,1]$ and $\beta \in [0,1]$ $(\alpha+\beta=1)$ denote people's confidences in the two risk perceptions; we refer to the variables as the local and global confidence, respectively.

People's local risk perception is often denoted by the fraction of infected individuals in his or her neighborhood [1,14,25]. In our model, mildly infected neighbors are the direct risk source, and severely infected neighbors are the indirect risk source. People perceive local risk from both sources. Here we denote the risk perception by

$$x_t = (\omega n_1 + n_2)/k, \tag{4}$$

where k is the node degree and n_2 denotes the numbers of severely infected neighbors.

People with a larger number of friends may have more chances to receive global information and perceive higher global risk. Consequently, we take the heterogeneity of global risk perception into consideration and measure it as

$$y_t = \max(1,(\omega\rho_1+\rho_2)(k/\langle k\rangle)^{\xi}), \tag{5}$$

where ξ is a coefficient to determine the heterogeneity level of global risk perception (for simplicity, we call it the global heterogeneity coefficient) and $\langle k\rangle$ represents the average degree of all nodes. When $\xi > 0$, y_t has a positive correlation with the node degree and vice versa. Considering that some nodes have a large degree value, when $y_t > 1$, we set $y_t = 1$, which is the upper limit of perceived global risk. When the upper limit is reached, extra risk information may not increase the global risk perception. Large-degree nodes are more likely to obtain the maximum global risk perception. ρ_1 and ρ_2 are the densities of the mildly and severely infected nodes across the whole network, which have often been adopted in previous studies to represent the global risk perception [14,26].

C. Mean-field analysis

We use $\varphi_1(t)$ to denote the probability of a randomly chosen link pointing to a mildly infected individual and $\varphi_2(t)$ to denote the probability of pointing to a severely infected individual. Let $s_k(t)$, $i_{1k}(t)$, and $i_{2k}(t)$ be the susceptible, mildly infected, and severely infected densities among nodes with degree k at time t. As described by Pastor-Satorras and Vespignani, the probability that a link points to a node with k links is proportional to $kP(k)$, i.e., a randomly chosen link is more likely to be connected to a node with high connectivity [22], yielding

$$\varphi_\delta(t) = \frac{\sum_k kP(k)i_{\delta k}(t)}{\sum_k kP(k)} = \frac{\sum_k kP(k)i_{\delta k}(t)}{\langle k\rangle}, \tag{6}$$

where $\delta = 1,2$. The probability that a node of degree k has n_1 mildly infected neighbors and n_2 severely infected neighbors can be given by a trinomial distribution:

$$T(k,n_1,n_2) = \frac{k!}{n_1!n_2!(k-n_1-n_2)!}\varphi_1^{n_1}\varphi_2^{n_2} \times(1-\varphi_1-\varphi_2)^{k-n_1-n_2}, \tag{7}$$

where $n_1 + n_2 \leqslant k$. We use T as an abbreviation for $T(k,n_1,n_2)$. Thus, the node will catch the disease with an average probability,

$$P_{S\to I} = 1 - \sum_{n_1,n_2} [1 - (1-\omega)\lambda\nu(1 - \alpha x_t - \beta y_t)]^{n_1} T. \quad (8)$$

According to stochastic process theory, we can describe an epidemic spreading in a discrete-time process as

$$i_{1k}(t+1) = i_{1k}(t) - \mu i_{1k}(t) + P_{S\to I} \times s_k(t). \quad (9)$$

We then extend it to a continuous-time process to obtain the dynamical equations of the SIIS model. Assuming that a susceptible individual will be mildly infected with probability $(1-\omega)\lambda h\nu[1 - \alpha x_t - \beta y_t] + o(h)$ in an infinitesimal interval $(t,t+h]$ [14,16,27], we have

$$i_{1k}(t+h) = i_{1k}(t) - \mu h i_{1k}(t) + o(h) + s_k(t) \times \left\{1 - \sum_{n_1,n_2} [1 - (1-\omega)\lambda h\nu(1 - \alpha x_t - \beta y_t]^{n_1} T\right\}. \quad (10)$$

To solve Eq. (10), we let

$$\begin{aligned} M &= \lim_{h\to 0} \frac{1 - \sum_{n_1,n_2} T[1 - (1-\omega)\lambda h\nu(1 - \alpha x_t - \beta y_t)]^{n_1}}{h} \\ &= \sum_{n_1,n_2} (1-\omega)\lambda\nu n_1(1 - \alpha x_t - \beta y_t)T \\ &= (1-\omega)\lambda\nu[E(n_1) - \alpha\omega/kE(n_1^2) - \alpha/kE(n_1 n_2) \\ &\quad -\beta(\omega\rho_1 + \rho_2)(k/\langle k\rangle)^{\xi} E(n_1)]. \end{aligned} \quad (11)$$

According to multinomial distribution theory, we have $E(n_1) = k\varphi_1$, $E(n_1^2) = k^2\varphi_1^2 + k\varphi_1(1-\varphi_1)$, $E(n_1 n_2) = k^2\varphi_1\varphi_2 - k\varphi_1\varphi_2$; thus, we obtain the value of M:

$$\begin{aligned} M = {} & (1-\omega)\lambda\nu\varphi_1[k - \alpha\omega - \alpha(k-1)(\omega\varphi_1 + \varphi_2) \\ & -\beta k(k/\langle k\rangle)^{\xi}(\omega\rho_1 + \rho_2)]. \end{aligned} \quad (12)$$

Therefore, the dynamical equations are the following:

$$\begin{aligned} \frac{d}{dt}s_k(t) &= \gamma i_{2k}(t) - M \times s_k(t) \\ \frac{d}{dt}i_{1k}(t) &= -\mu i_{1k}(t) + M \times s_k(t). \\ \frac{d}{dt}i_{2k}(t) &= \mu i_{1k}(t) - \gamma i_{2k}(t) \end{aligned} \quad (13)$$

$s_k(t)$, $i_{1k}(t)$, and $i_{2k}(t)$ must obey the normalization condition $s_k(t) + i_{1k}(t) + i_{2k}(t) \equiv 1$. After imposing the stationary condition, $\frac{d}{dt}s_k(t) = \frac{d}{dt}i_{1k}(t) = \frac{d}{dt}i_{2k}(t) = 0$, we obtain $i_{2k} = \frac{\mu}{\gamma}i_{1k}$, $s_k = \frac{\mu}{M}i_{1k}$. Then, we obtain

$$i_{1k} = \frac{1}{1 + \mu/M + \mu/\gamma}. \quad (14)$$

Furthermore,

$$\varphi_1 = \frac{1}{\langle k\rangle}\sum_k kP(k)\frac{1}{1 + \mu/M + \mu/\gamma}. \quad (15)$$

Let $F(\varphi_1)$ denote the right-hand side of Eq. (15). A nonzero stationary prevalence ($\varphi_1 \neq 0$) can only be obtained when the left-hand side and right-hand side of Eq. (15) meet in the interval $0 < \varphi_1 < 1$, producing a nontrivial solution [28]. It is straightforward to deduce that a nontrivial solution requires that the inequality

$$\frac{dF(\varphi_1)}{d\varphi_1}|_{\varphi_1=0} \geqslant 1 \quad (16)$$

must be satisfied, namely

$$\frac{(1-\omega)\lambda\nu(\langle k^2\rangle - \alpha\omega\langle k\rangle)}{\mu\langle k\rangle} \geqslant 1. \quad (17)$$

Thus the epidemic threshold λ_c can be obtained as

$$\lambda_c = \frac{\mu\langle k\rangle}{(1-\omega)\nu(\langle k^2\rangle - \alpha\omega\langle k\rangle)}. \quad (18)$$

From Eq. (18), we observe that the epidemic threshold λ_c is highly positively correlated with the discovery probability for the mildly infected nodes, ω. When $\omega = 0$, the value of λ_c is reduced to $\mu\langle k\rangle/\nu\langle k^2\rangle$, and the epidemic threshold is unrelated to people's risk perceptions. This situation differs from those of previous studies [14,16], which stated that people's risk perceptions can enhance the threshold value. To explain the difference, we observe that people's risk information in our model does not come from the mildly infected nodes. When $\omega > 0$, λ_c can rebuild its relation to the risk perception and is increased with an enhanced local confidence α. Thus, we draw the conclusion that the influence of people's risk perceptions on the epidemic threshold depends on the detection of the direct risk source. If we cannot discover the direct sources, then whether the risk information diffuses may not change the threshold.

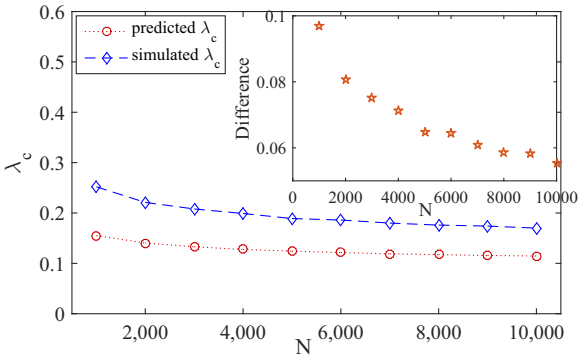

FIG. 1. Simulated epidemic threshold λ_c vs predicted λ_c under different network sizes. We generate 500 independent networks for each network size N and compute the value of $\langle k^2\rangle$. Then we obtain the average value for the predicted λ_c. The simulated epidemic thresholds are greater than the predicted ones. However, the difference between them decreases when the network size increases.

D. Simulation design

To verify our analysis, we use a multiagent intelligent software, REPAST [29,30], to perform the simulations. A scale-free BA network with network size $N = 1000$ and average degree $\langle k\rangle = 6$ is adopted. Without loss of generality, we set the recovery rate to $\gamma = 1.0$ and the average rate from mild infection to severe infection to $\mu = 1.0$. Because people may

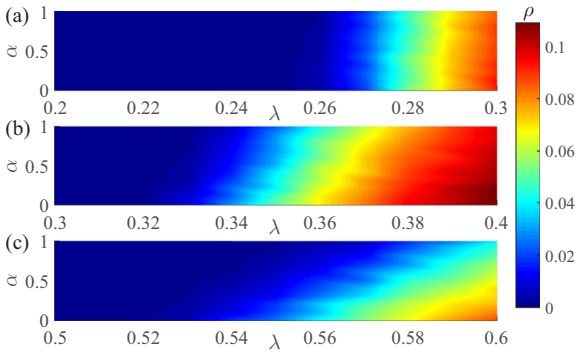

FIG. 2. The value of the infected ratio ρ in the parameter space λ-α. From (a) to (c), the values of ω are 0, 0.2, and 0.5. The selected ranges of λ are around the simulated epidemic thresholds, which are significantly enhanced with increasing ω. In (b) and (c), ρ_1 has a tendency to decrease with α and the tendency becomes clearer when ω is larger.

have a preference between local and global risk perception, we set $\alpha + \beta = 1$, as performed by Chen when considering the belief distribution between two types of incentives [26]. Under these conditions, we find that when people have more local confidence, they may have less global confidence and vice versa. To reduce the randomness of the simulation, we obtain the data by averaging over 500 independent runs. To start the simulation, we randomly choose 10 nodes to be initially mildly infected.

III. NUMERICAL RESULTS

We first check whether the simulated epidemic threshold is dependent on the value of the discovery probability of the mildly infected nodes ω and the local confidence α, which we predicted in the above analysis. As observed from Fig. 1, the simulated epidemic thresholds are larger than the predicted ones obtained from Eq. (18), which may be due to a distribution cutoff effect on a finite network [14,31], and the difference between them decreases as the network size increases. In

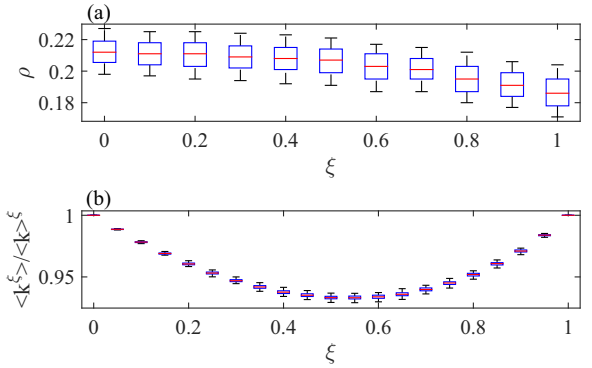

FIG. 3. Results when incorporating global risk perception heterogeneity. (a) Evolution of infected ratio ρ as a function of the global heterogeneity coefficient ξ. We set the parameters as follows: $\omega = 0.5$, $\alpha = 0.5$, and $\lambda = 1$. (b) Distribution of $\langle k^{\xi}\rangle/\langle k\rangle^{\xi}$ versus ξ. The value of $\langle k^{\xi}\rangle/\langle k\rangle^{\xi}$ first decreases and then increases when ξ changes from 0 to 1.0.

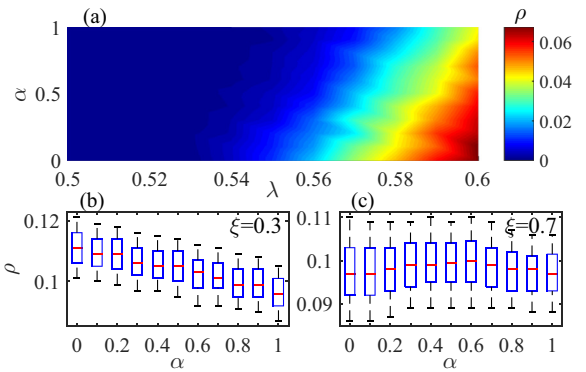

FIG. 4. (a) The value of ρ in the parameter space $\lambda - \alpha$ with $\omega = 0.5$ and $\xi = 0.5$. [(b) and (c)] Evolution of ρ versus the change of local confidence α with $\xi = 0.3$ and $\xi = 0.7$, respectively. The other parameters are set as follows: $\omega = 0.5$, $\lambda = 1$.

addition, as can be observed from Fig. 2, the simulated λ_c is apparently dependent on α when $\omega > 0$, as we predicted in the above analysis. Remarkably, this dependence decays as ω decreases and even vanishes when $\omega = 0$. Thus, the impact of the local risk perception on the epidemic threshold depends on whether the direct risk source can be detected.

We record the total infected ratio $\rho = \rho_1 + \rho_2$ as the prevalence size. When $\omega = 0$, as shown in Fig. 2(a), the value of α has little influence on the infected ratio ρ, thus suggesting that when the direct risk source is completely invisible, the local and global risk perceptions have nearly the same effect on people's self-protective responses, partly because they are both from the indirect risk sources. Detection of the direct risk source immediately increases the effect of local risk perception.

We next consider the impact of heterogeneity on the global risk perception by setting $\xi > 0$. This causes the hubs to perceive higher global risk. Figure 3(a) shows that this change increases the inhibitory impact on the disease. However, this better inhibitory impact does not result in higher global risk perception, and the risk perception sometimes decreases. The global risk that all individuals perceive at infection densities ρ_1 and ρ_2 can be calculated as $N\sum_k(\omega\rho_1 + \rho_2)(k/\langle k\rangle)^{\xi}P(k) = N(\omega\rho_1 + \rho_2)\langle k^{\xi}\rangle/\langle k\rangle^{\xi}$. With the infected densities remaining constant, a larger ξ may reduce the global risk perception amount, as inferred from Fig. 3(b). Thus, even if the whole population perceives less global risk, the epidemic might be better contained if the hub individuals perceive higher risk, which implies that better-organized global risk policies across the whole population may result in a better controlling effect and consume fewer publicity resources.

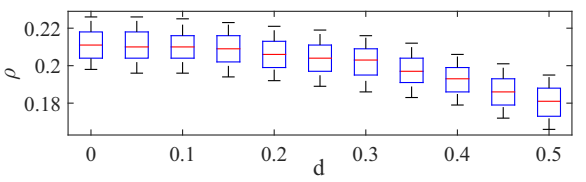

FIG. 5. Infected ratio versus heterogeneity in people's intrinsic susceptibilities. We set the parameters $\theta = 0.5, \alpha = 0.5$, and $\lambda = 1.0$.

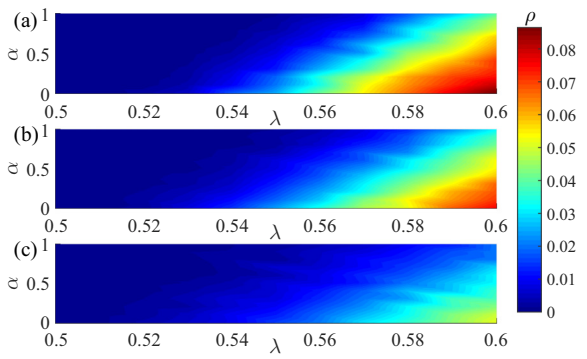

FIG. 6. Evolution of the infected ratio when considering heterogeneous intrinsic susceptibilities. The values of ξ and θ are set to 0 and 0.5, respectively. From (a) to (c), the values of d are 0, 0.25, and 0.5. The increasing dependence of ρ_1 on α remains.

Increasing the local risk confidence can enhance the epidemic threshold, and increasing the heterogeneity level of global risk perception can result in a greater inhibitory impact. When considering these factors together, some unexpected results arise. In Fig. 4(a), the epidemic threshold λ_c has a positive dependence on the local confidence α with the heterogeneous global risk perception. Thus, greater local confidence makes it easier for the network to eradicate an epidemic. Nevertheless, if the epidemic is ineradicable, increasing local confidence may result in different outcomes, as shown in Fig. 4(b) and Fig. 4(c). When global risk heterogeneity is relatively low ($\xi = 0.3$), increasing the local confidence can reduce the infected ratio. However, when the global risk heterogeneity is relatively high ($\xi = 0.7$), the infected ratio does not decreases with the increasing value of the local confidence, which may result in a paradox. Larger local confidence can enhance the epidemic threshold, which means increasing resistance to epidemic invasion. However, sometimes this effect may lead to greater epidemic outbreak. Thus, the global risk heterogeneity may weaken the importance of increasing the epidemic threshold, and the protection effect that an increased threshold provides may be somewhat "limited."

In Fig. 5, we explore the scenario of individual differences in susceptibility. We adopt a radius d to propose a uniform distribution of intrinsic susceptibilities. Accordingly, the natural susceptibility of an individual m is set to a random value $\nu_m \in [\nu - d, \nu + d]$ instead of a constant value ν. A larger d can yield a higher heterogeneity, which results in a smaller infected ratio. In addition, this individual heterogeneity can influence the epidemic threshold. Figure 6 shows that higher values of d result in larger λ_c and that the observed dependence of λ_c on α remains positive.

IV. CONCLUSIONS AND DISCUSSION

This paper has examined the impact of risk perceptions on epidemic spreading. People's risk perceptions in each disease season, or even each day in a single season, are not fixed but rather fluctuate with changes in the epidemic information, resulting in corresponding levels of self-protection. This self-protection can help to contain the epidemic in two ways: by enhancing the epidemic threshold and by decreasing the infected ratio. However, why only local risk perception enhances the threshold but global risk perception does not remains to be explored. It will be interesting to compare our results with those of previous studies. We considered three issues that are influential, namely the indirect risk source, heterogeneous global risk perceptions, and heterogeneous natural susceptibilities.

Our results demonstrate that the discovery probability of the direct risk source significantly enhances the epidemic threshold. When this probability is greater than 0, the threshold is further enhanced if people have more local confidence. However, if the probability is reduced to 0, then the local risk perception may not influence the epidemic threshold and may have nearly an equal effect as the global risk perception. A larger value for the epidemic threshold may not ensure a better controlling effect when considering the global risk heterogeneity and may lead to more people being infected because of insufficient global confidence. Thus, future studies should identify which factor is more important for containing diseases: enhancing the epidemic threshold or increasing the network heterogeneity. For the intrinsic susceptibility heterogeneity, increasing the heterogeneity increases the epidemic threshold and makes the network less vulnerable to an epidemic.

The major threat is often not from the easily detected targets but rather from invisible targets. The government should use new technology to accurately detect all risk sources. Moreover, proper strategies to let the hub individuals perceive more global risk and get more global protection should be developed; such strategies may cut off the main contagious paths of the disease. Individuals should be aware of the latest epidemic information and take positive actions to protect themselves. A remaining limitation of our research is that we only propose a "fraction" definition of the risk perception. Future work can explore the situations when defining the infected number as the risk perception.

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