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Highlights

- 1. We investigate the spread of awareness and risk on multiplex networks considering the heterogeneity of firm.
- 2. Threshold is affected by the cooperation intensity but not the local risk propagation prevalence.
- 3. The global awareness has two-stage effects on threshold ignoring the local awareness.
- 4. Threshold lies in three different areas by the common effects of the global awareness and the local awareness.

The impact of firm heterogeneity and awareness in modeling risk propagation on multiplex networks

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Abstract: Growing interest has emerged to understand the coupled awareness-epidemic dynamics in multiplex network. However, most previous studies usually assume that all the infected nodes have the same influence on the susceptible neighbors, without considering node's heterogeneity. In this paper, with the similarity between epidemic spreading and risk propagation, we apply the UAU-SIS model to investigate the interplay between awareness and risk propagation in R&D networks considering firms' heterogeneity. Here, the risk triggering probabilities are heterogenous and depend on two factors: cooperation intensity and local risk prevalence. The results reveal that the cooperation intensity can increase the risk propagation prevalence and decrease the risk propagation threshold, while the local risk prevalence can only increase the risk propagation prevalence. Moreover, we find that the risk propagation threshold undergoes an abrupt transition with a certain point of the local awareness ratio (the global awareness ratio) ignoring the global awareness (the local awareness ratio), which includes two-stage effects on risk propagation threshold. Furthermore, threshold lies in three different areas when considering both the global and local awareness. These results could provide a basis for managerial professionals to improve the robustness of interdependent R&D networks under risk propagation by taking effective measures.

Keywords: Risk propagation; Global awareness; Local awareness; Node heterogeneity; MMCA method.

1. Introduction

Research and Development (R&D) network [1] is a representation of the research and development alliances formed by formal joint ventures or more informal research agreements between firms in one or more industrial sectors within a given period, where nodes are firms and links represent R&D cooperation. R&D network has been an effective cooperative form that helps firms to gain advantages, e.g. obtaining the complementary resources, shortening the period of R&D project and sharing the risk [2-5]. It is thus not surprising that the number of R&D networks has grown very rapidly in recent decades [6], especially in high-tech industry. Although enterprises can benefit a lot from joining R&D networks, risk still exists in R&D network. Some researches [7-10] have shown that when firms occur risk, they might trigger the potential risk of their neighbors just like reaction chains. This phenomenon, which can be called risk propagation, might cause the majority of firms to fail and lead to the collapse of the R&D network. It is very meaningful to study risk propagation in R&D networks in order to provide theoretical basis for the

efficient risk management of R&D network.

Many researches [11-13] have studied risk propagation in inter-firm networks. However, to our knowledge, most of them are mainly concentrated on supply chain networks or financial networks, and there exist only a few literatures on risk propagation in R&D networks. The literatures about risk propagation in R&D networks can be divided into two categories: the spread of firms' risk state and the cascading failures induced by overload at firms. For the first research stream, the core idea of these studies [14-16] is the similarity between the risk diffusion among firms and the spread of epidemic. These researches show that by reducing risk transmission rate, shorting the cycle of risk appeared or reducing the heterogeneity of the network, one can eliminate risk propagation between firms. For the second stream, previous researches [7-10,17] are mainly applying the idea of load-capacity model by assuming that each firm has risk capacity and risk load. Only if the risk load exceeds risk capacity, the firm occurs risk and is removed from the network. Some interesting results are obtained from these researches, e.g. the uniform distribution of firms' capacities could improve the invulnerability of R&D networks, and the robustness of R&D network is more sensitive to the negative deviation than to the positive deviation from attack information.

However, the above-mentioned researches ignore the risk perception of firm. Risk perception [18] refers to those ambiguities, as perceived by alliance partners, about the future events that may have negative impacts on the performance of the alliance, which can also call risk awareness. Risk awareness is very common in our daily life: When people are aware of the flu, they could reduce outdoor activities or take antiviral drugs to avoid getting it. The same happens to firms: When enterprise are aware of risks, they will take certain measures and countermeasures to avoid being triggered by these firms that have already occurred risk. In recent years, there is a growing interest in studying the dynamical interplay between epidemic spreading and awareness diffusion [19-26], which is typically modeled as two competing spreading in multiplex networks. Two diffusive processes are interacting with each other in a two-layer network, where the epidemic spreads on one layer and the awareness propagates on another one. Following this thought, Granell et al. proposed a UAU-SIS model to study the interplay of epidemic spreading and diffusion of awareness, and found that the spreading of awareness is able to control the onset of epidemic [19]. Different models have been proposed in order to extend the coupled awareness-epidemic dynamics corresponding to diverse realistic scenarios by considering other various factors, such as local awareness [20], individual heterogeneity [21,25,27], self-initiated awareness [22], global awareness [25,28], etc.

However, most studies assume that the susceptible individuals get infected by the infected individual with the same infection rate, and ignore the effect of individual heterogeneity. Although there exist a few researches that consider the individual heterogeneity, they are mainly focusing on the heterogeneous of aware individual precautions. In reality, there exists much heterogeneity in firms' influence on others in R&D network. This is because, on the one hand, when firms occur risk, they cannot continue to perform their own R&D tasks. Thus, the more cooperative firms occur risk, the more likely a firm will be triggered risk. On the other hand, a firm is usually influenced by those firms with the high intensity of cooperation than those with low cooperation intensity. Motivated by the above reasons, we explore the interplay between risk propagation and awareness in the R&D networks, considering the heterogeneity of firms.

The rest parts of this paper are as followings: In the second part, we describe our modelling

approach to the risk propagation in R&D network. In the third part, we conduct theoretical analysis by using microscopic Markov chain approach (MMCA). In the fourth part, we show the simulations of our risk propagation models, and in the last part we discuss about our findings.

2. Model

In this part, by taking the heterogeneity of firms, we model the dynamical process of risk propagation and awareness spreading in multiplex network. A sketch of the model is shown in Fig. 1. The two layers contain the same nodes (firms) but with different connections (relationship between firms). The upper layer is the information network. It has more links, because there are other inter-firm relationships expect for the R&D relationships, e.g. supply relationship or historical cooperation relations, etc. This layer is the network where the awareness spreading happens. The lower layer is the R&D network (contact network), where risk propagation happens.



Fig. 1. Example of the description of multiple network used in our work. The upper layer corresponds to the network which supports the spreading of awareness. Nodes are awareness (A) and unawareness (U) on this layer. The lower layer is the network where risk propagation happens and nodes also have two states: susceptible (S) and infected (I). Only three kinds of states can exist in the multiple network, namely unaware and susceptible (US), aware and susceptible (AS) and aware and infected (AI). In addition, the spreading models for the upper layer and the lower layer are different, with threshold model defined by the global information and local information for the awareness spreading, while the contagion model for risk propagation, respectively.

As for the awareness spreading, firms are either aware (A) or unaware (U) in the information layer. A firm needs enough information to become aware of the risk, before it will take any risk control actions. Thus, in this layer, we adopt a threshold model to describe awareness spreading. A firm can usually obtain information from the two sources: communication with the aware neighbors or gathering information from other aware firms. In this situation, an unaware firm becomes aware when its proportion of aware neighbors (local awareness) exceeds a certain point denoted by θ_1 , or the ratio of all aware firms in R&D network is larger than a certain point denoted by θ_2 . In addition, an aware firm might return to unawareness state with rate δ .

The lower layer is the R&D network where risk propagation happens. In this layer, we apply the susceptible-infected-susceptible (SIS) model to mimic the risk propagation process. A firm is either infected (I) or susceptible (S) in the layer. That is, a firm either occurs risk or not. The infected enterprises will take certain measures to mitigate risk, so that they can recover to susceptible state with probability μ . Differ from the epidemic spreading, susceptible firms can be influenced differently by different infected firms. Obviously, a firm might be more likely to be triggered risk by those cooperative firms with high cooperation intensity. Besides, the more neighbors are infected, the easier the firm to be infected. Here, we assume that the heterogeneity of firm depends on the cooperation intensity and the local risk prevalence. Then each susceptible firm will be triggered risk with a triggering rate that is defined as:

$$\beta_{ij}(t) = \frac{w_{ij}^{\alpha}(1 + a\rho_i(t))}{\max_{l=1...N} w_{lk}^{\alpha}(1 + a\rho_l(t))}\beta, \quad (\alpha \ge 0, \alpha \ge 0)$$
(1)

Where the heterogeneity factor is defined as follows:

$$\varphi_{ij} = \frac{w_{ij}^{\alpha} (1 + a\rho_i(t))}{\max_{l=1,N} w_{lk}^{\alpha} (1 + a\rho_l(t))}$$
(2)

Where w_{ij} is the intensity of cooperation between firm *i* and firm *j*, ρ_i is the fraction of infected firm among i's neighbors. α and *a* stand for the impact strength of cooperation and local risk propagation prevalence on one's triggering rate, respectively.

The interplay of the UAU-SIS process is as follows: once a firm occurs risk, it will certainly become awareness. Moreover, these susceptible enterprises in awareness state will take measures to reduce their susceptibility. To distinguish the protective behaviors between aware enterprises and unaware enterprises, we assume that the risk propagation rates in aware state and unaware state are β_i^A and β_i^U , and β_i^A and β_i^U are linearly correlated [19,29], i.e. $\beta_i^A = \gamma \beta_i^U (0 \le \gamma \le 1)$, $\gamma = 0$ means that when firms are aware of risk, they are totally immune to it.

3. Theoretical analysis using the MMCA

In this section, the theoretical analysis of our model is based on MMCA approach, as it has a high accuracy in solving the interplay spreading in the multiplex networks [19, 28-33]. Defining

 a_{ij} and b_{ij} as the adjacency matrices of the information communication network and the R&D

network, respectively. According to the proposed model, at time t, the probability of firm i in one of the three states is denoted by $p_i^{AI}(t)$, $p_i^{AS}(t)$ and $p_i^{US}(t)$. Here, assuming that the transition probability of unaware firm i not becoming aware by the information obtained from their aware neighbors (the global aware firms) is $r_i(t)$ ($m_i(t)$), and unaware (aware) susceptible

firm *i* not being infected is $q_i^U(t)$ ($q_i^A(t)$), which are described as the following equations:

$$r_i(t) = \mathbf{H}[\theta_1 - \frac{\sum_j a_{ji} p_j^{\mathbf{A}}(t)}{k_i}]$$
$$m_i(t) = \mathbf{H}[\theta_2 - \frac{\sum_{j=1}^N P_j^{\mathbf{A}}(t)}{N}]$$

$$q_{i}^{A}(t) = \prod_{j} [1 - p_{j}^{AI}(t)b_{j,i}\beta_{ji}^{A}(t)]$$
$$q_{i}^{U}(t) = \prod_{j} [1 - p_{j}^{AI}(t)b_{ji}\beta_{ji}^{U}(t)]$$
(3)

Note that H(x) is a Heaviside step function. If x > 0, H(x) = 1, else H(x) = 0. In other words, the values of $r_i(t)$ $(m_i(t))$ can either be 0 when the fraction of its aware neighbors (the global aware nodes) surpasses the aware ratio θ_1 (θ_2) , or 1 if the fraction of its aware neighbors (the global aware nodes) is less than the aware ratio θ_1 (θ_2) .

Therefore, for each firm i, the transition probability trees for the three possible states are illustrated in Fig. 2.



Fig. 2. Transition probability trees for the three states (AI, AS and US) of the UAU_SIS dynamics in the multiplex per time step. The denotation of r_i , m_i , $q_i^{\rm U}$ and $q_i^{\rm A}$ are given in Eq. (3). Each time step is subdivided into two phases: awareness spreading (UAU process) and risk propagation (SIS process).

According to the scheme in Fig. 2, together with equations (3), we can develop the coupled processes for each node i by using MMCA method as:

$$p_{i}^{US}(t+1) = \delta p_{i}^{AS}(t)m_{i}(t)q_{i}^{U}(t) + p_{i}^{US}(t)r_{i}(t)m_{i}(t)q_{i}^{U}(t)$$

$$p_{i}^{AS}(t+1) = \mu p_{i}^{AI}(t) + p_{i}^{AS}(t)\{\delta[1-m_{i}(t)]q_{i}^{A}(t) + (1-\delta)q_{i}^{A}(t)\}$$

$$+ p_{i}^{US}(t)\{r_{i}(t)[1-m_{i}(t)]q_{i}^{A}(t) + [1-r_{i}(t)]q_{i}^{A}(t)\}$$

$$(t+1) = (1-\mu)p_{i}^{AI}(t) + p_{i}^{AS}(t)\{\delta m_{i}(t)[1-q_{i}^{U}(t)] + \delta[1-m_{i}(t)][1-q_{i}^{A}(t)] + (1-\delta)[1-q_{i}^{A}(t)]\}$$

$$+ p_{i}^{US}(t)\{[r_{i}(t)m_{i}(t)(1-q_{i}^{U}(t)] + r_{i}(t)[1-m_{i}(t)][1-q_{i}^{A}(t)] + [(1-r_{i}(t))(1-q_{i}^{A}(t)]\}$$

$$(4)$$

Where $p_i^{AI}(t) + p_i^{AS}(t) + p_i^{US}(t) \equiv 1$.

 p_i^{AI}

To calculate the risk propagation threshold, we need to explore the steady solution of the system constituted by the equations (4). When the system is in the stationary state, it satisfies the conditions $p_i^{AI}(t+1) = p_i^{AI}(t) = p_i^{AI}$, $p_i^{AS}(t+1) = p_i^{AS}(t) = p_i^{AS}$ and $p_i^{US}(t+1) = p_i^{US}(t) = p_i^{US}$. Noting that near the threshold, the infected probability of firm *i* can be assumed as $p_i^{AI} = \varepsilon_i \ll 1$, Consequently, $q_i^{U}(t)$ and $q_i^{A}(t)$ can simply approximate as $q_i^{U} \approx 1 - \beta_i^{U} \sum_j b_{ji}\varepsilon_j$ and

 $q_i^A \approx 1 - \beta_i^A \sum_j b_{ji} \varepsilon_j$. Inserting these approximation in Eqs. (3), we obtain,

$$p_{i}^{\mathrm{US}} = p_{i}^{\mathrm{US}} r_{i} m_{i} + p_{i}^{\mathrm{AS}} \delta m_{i}$$

$$p_{i}^{\mathrm{AS}} = p_{i}^{\mathrm{AS}} (1 - \delta m_{i}) + p_{i}^{\mathrm{US}} (1 - r_{i} m_{i})$$

$$\mu \varepsilon_{i} = (\beta_{i}^{\mathrm{U}} p_{i}^{\mathrm{US}} + \beta_{i}^{\mathrm{A}} p_{i}^{\mathrm{AS}}) \sum_{j} b_{ji} \varepsilon_{j}$$
(5)

Then, with $\beta_i^A = \gamma \beta_i^U$, $p_i^A = p_i^{AI} + p_i^{AS} \approx p_i^{AI}$, $p_i^U = p_i^{US}$, $p_i^A + p_i^U = 1$ and Eq. (1), we get

$$\mu\varepsilon_{i} = \beta\sum_{j} b_{ji}\varepsilon_{j} \left\{ (1 - p_{i}^{A} + rp_{i}^{A}) \frac{w_{ij}^{-\alpha} (1 + a\sum_{j} b_{ji}\varepsilon_{j} / \sum_{j} b_{ji})}{\max\left[w_{lk}^{-\alpha} (1 + a\sum_{k=1}^{N} b_{kl}\varepsilon_{k} / \sum_{k} b_{kl})\right]} \right\}$$
(6)
$$= \beta\sum_{j} b_{ji}\varepsilon_{j} \left\{ \left[1 - (1 - \gamma)p_{i}^{A} \right] \left[\frac{w_{ij}}{\max(w_{lk} \mid l = 1, 2, ..., N)} \right]^{-\alpha} \right\}$$

Therefore,

$$\sum_{j} \left\{ \left\{ \left[\left[1 - (1 - \gamma) p_i^A \right] \right] \left[\frac{w_{ij}}{\max(w_{ik} \mid l = 1, 2, ..., N)} \right]^{-\alpha} \right\} b_{ji} - \frac{\mu}{\beta} \sigma_{ji} \right\} \varepsilon_j = 0$$
(7)

Where σ_{ji} is the element of the identity matrix.

Then, defining matrix H with elements

$$h_{ij} = \left\{ \left[1 - (1 - \gamma) p_i^{A} \right] \left[\frac{w_{ij}}{\max(w_{lk} \mid l = 1, 2, ..., N)} \right]^{-\alpha} \right\} b_{ji}$$
(8)

the non-trivial solution of Eq. (8), that is the risk propagation threshold, reduces to an eigenvalue problem for the matrix H. The risk propagation threshold is the minimum value of β stratifying Eq. (8), which can be written as:

$$\beta_c = \frac{\mu}{\Lambda_{\max}(H)} \tag{9}$$

Here, $\Lambda_{\max}(H)$ is the largest eigenvalue of matrix H. According to Eq. (8), the risk propagation threshold depends on risk recovery rate μ , the structure of the R&D network (b_{ij}) , the parameter γ , the dynamics in the virtual communication networks (p_i^A) and the cooperation intensity (α) .

4 Simulations

After obtaining the analytic solution of risk propagation threshold β_c , we explore the vulnerability of the R&D network under risk propagation using numerical simulations. Previous

studies have shown the degree distribution of the real R&D network follows a power-law [1,34]. Since BBV model is with the properties as many real networks, e.g. power-law distribution of degree and weight, Thus, we first use BBV model [35] to generate the R&D network with N=300,

and other related parameters are fixed with $\sigma = 3.0$, $w_0 = 1.0$ and m = 3. The virtual

communication network is generated by adding 500 extra random links (nonoverlapping with the previous) in the R&D network, which is an unweighted network and the average degree is 7.3. Then, we use $\rho^{I} = \sum_{i=1}^{N} p_{i}^{AI} / N$ and $\rho^{A} = \sum_{i=1}^{N} (p_{i}^{AI} + p_{i}^{AS}) / N$ to represent the fraction of occurred risk

firms and aware enterprises when risk propagation in the multiplex networks reaches the steady state.

In the following parts, we mainly investigate the effects of awareness ratios and the heterogeneity of firms on the interplay between awareness and risk propagation in multiplex networks.

From the above model, we know that the critical awareness ratios θ_1 (local awareness) and θ_2 (global awareness) control the threshold of firms becoming aware in information layer. The effect of critical awareness ratio θ_1 on epidemic spreading has been studied without considering the nodes heterogeneity and global awareness. The research [20] shows that the critical awareness ratio has two-stage effect on epidemic threshold, and the corresponding transition point called θ_c is approximately 0.5. Thus, firstly, we explore how the firms' heterogeneity and global awareness θ_1 on risk propagation.

In Fig. 3 and Fig. 4, we plot the full phase diagrams $(\theta_1 - \beta)$ of ρ^1 and ρ^A , together with $(\theta_2 - \beta)$ of ρ^1 and ρ^A respectively. The results reveal that the density of infected firms ρ^1 decreases with the decreasing of either the local awareness ratio θ_1 or the global awareness ratio θ_2 . This is because that awareness is more likely to spread with a smaller awareness ratio. Then these aware firms will take actions to suppress risk propagation. In addition, we notice that the value of ρ^1 is even smaller with a larger β than a smaller β . This is because ρ^A suddenly increases with the increasing of β for some values of θ_1 or θ_2 , which result in ρ^1 abruptly decreases. In essence, it is a result of the coupled risk-awareness spreading processes. On the one hand, the increasing of β promotes risk spreading, which will promote awareness diffusion due to these occurred risk firms will automatically become aware. In return, the aware firm will take risk control measures to suppress risk propagation. Thus, when the effect of promoting awareness spreading overwhelms the effect of promoting risk propagation, the density of infected firms will be reduced. These results imply that promoting the awareness spreading among firms is an effective way to decrease risk propagation in R&D network.

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Fig. 3. The full phase diagram $(\theta_1 - \beta)$ of ρ^A or ρ^1 for the same multiplex network described before. Where $\gamma = 0.4$, $\theta_2 = 1.1$, $\alpha = 0.2$ and a = 0.5. (a)-(b) $\mu = 0.4$, $\delta = 0.6$; (c)-(d) $\mu = 0.6$, $\delta = 0.4$. The simulations are averaged by 100 realizations.



Fig. 4. The full phase diagram $(\theta_2 - \beta)$ of ρ^A or ρ^I for the same multiplex network described before. Where $\gamma = 0.4$, $\theta_1 = 1.1$, $\alpha = 0.2$ and a = 0.5. (a)-(b) $\mu = 0.4$, $\delta = 0.6$; (c)-(d) $\mu = 0.6$, $\delta = 0.4$. The simulations are averaged by 100 realizations.

Secondly, we explore how the awareness ratios influence on the risk propagation threshold with considering the heterogeneity of nodes. In Fig. 5 (a), one can see that there is a good agreement between the MMCA approach and MC simulations in calculating the risk propagation threshold β_c . Thus, we have decided to only use MMCA in Fig. 5 (b) and Fig 6. As can be seen in Fig 5 (a), the risk propagation threshold β_c has a sudden transition at a certain value of local awareness ratio denoted as θ_{1c} without considering the effect of global awarenss. Obviously, when the local awareness ratio θ_1 is smaller (bigger) than θ_{1c} , β_c is with a larger (smaller) value. The two values of β_c are denoted as $\beta_c^{\rm H}$ (higher β_c) and $\beta_c^{\rm L}$ (lower β_c), respectively. In addition, from Fig.5 (b), we could conclude that the global awareness also has the two-stages effects on the risk propagation threshold β_c , ingoring the effect of local awareness. Moreover, we can find that the transition point θ_{1c} and θ_{2c} are not always 0.5 when considering the heterogeneity of firms and global awareness.



Fig. 5 (a). The comparison of risk propagation threshold β_c using the MMCA approach and Monte Carlo (MC) simulations as a function of θ_1 , the blue square is using the MMCA and the red circle is obtaining by MC simulations, where $\theta_2 = 1.1$, $\mu = 0.4$, $\delta = 0.6$, a = 0.5, $\gamma = 0.4$ and $\alpha = 0.2$ (b). The risk propagation threshold β_c as a function of θ_2 . Here $\theta_1 = 1.1$, $\mu = 0.4$, $\delta = 0.6$, a = 0.5, $\gamma = 0.4$ and $\alpha = 0.2$ (b). The risk propagation threshold β_c as a function of θ_2 . Here $\theta_1 = 1.1$, $\mu = 0.4$, $\delta = 0.6$, a = 0.5, $\gamma = 0.4$ and $\alpha = 0.2$

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Fig. 6. The full phase diagram $(\theta_1 - \theta_2)$ of β_c for the same multiplex network described before. Here $\mu = 0.4$, $\delta = 0.6$, a = 0.5, $\gamma = 0.4$ and $\alpha = 0.2$.

Thirdly, we explore how the local awareness and global awareness influence on the risk propagation threshold. From Fig. 6, we can see that the threshold β_c lies in three different areas with considering both the effects of θ_1 and θ_2 . Here, we use two groups of four numbers of θ_1 and θ_2 to describe the three areas, which are denoted as θ_{11} , θ_{12} , θ_{21} and θ_{22} . Obviously, when $\theta_1 < \theta_{11}(\theta_2 < \theta_{21})$, the threshold is always the same with a larger value, no matter what values of $\theta_2(\theta_1)$ is, this is because that the awareness will spread all over the network. In addition, when $\theta_1 \ge \theta_{12}$ and $\theta_2 \ge \theta_{22}$, no matter what values of θ_1 and θ_2 are, the values of threshold are still the same but with a smaller value. The reason is that the onset of risk propagation is independent of awareness spreading in this situation and remains at a lower level. In the other cases, the values of the threshold are not always the same, which is influenced by the coupled interplay between θ_1 and θ_2 . The general trend is that when $\theta_1(\theta_2)$ is fixed, β_c decreases with the increasing of $\theta_2(\theta_1)$.

Then, we explore how the parameter α and parameter a that control the heterogeneity of firm influence the risk propagation. As can be seen in Fig 7, we analyze the impact of α on the stationary risk propagation layer as a function of β . The results in Fig. 5 show that the density of infected firms ρ^{1} decreases effectively with the increasing of α , while the threshold β_{c} augments as well. Since $w_{ij} / w_{max} < 1$, the smaller the parameter α is, the more quickly risk propagates. Besides, when $\alpha = 0$, the risk propagation network degenerates to unweighted network under the same conditions, which is consistent with the results in single network [36]. Furthermore, the impact of α on risk spreading threshold β_{c} has been also



explored and shown in Fig. 8. We find that the risk spreading threshold increases with the parameter α .

Fig. 7. ρ^{I} as a function of β for different values of α . Here, $\gamma = 0.4$, $\mu = 0.4$, a = 0.5 and $\delta = 0.6$. (a) $\theta_{1} = 0.1$, $\theta_{2} = 0.9$; (b) $\theta_{1} = 0.9$, $\theta_{2} = 0.1$; (c) $\theta_{1} = 0.4$, $\theta_{2} = 0.6$; (d) $\theta_{1} = 0.6$, $\theta_{2} = 0.4$. Each curve is obtained by average 100 realizations.



Fig. 8. β_c as a function of the parameter α for different values of θ_1 and θ_2 . Other parameters are:

 $\gamma=0.4$, $\mu=0.4$, a=0.5 and $\delta=0.6$.

Finally, the stationary risk prevalence as a function of β under different values of a is shown in Fig. 9. The results indicate that ρ^{I} slightly decreases with the increasing of parameter a, while the threshold β_{c} is not influenced by the changing of a. This is because that the prevalence of risk is 0 near the threshold β_{c} . In sum, the risk propagation threshold is not affected by the prevalence of the infected neighbors. The result is consistent with the theoretical analysis, which we can see that a absents from the Eqs. (8) and (9) to determine the risk propagation threshold.



Fig. 9 ρ^1 as a function of β for different values of α . Other parameters are fixed with $\gamma = 0.4$, $\mu = 0.4$, $\delta = 0.6$, $\theta_1 = 0.5$ and $\theta_2 = 0.5$; (a) $\alpha = 0$; (b) $\alpha = 0.2$; (c) $\alpha = 0.6$; (d) $\alpha = 1$. Each curve is obtained by average 100 realizations.

5 Conclusion

The interrelation between epidemic spreading and awareness diffusion in multiplex network

has drawn a lot of attention. However, the heterogeneity of nodes has been ignored in many previous studies. In this paper, by taking firm's heterogeneity into account, we have investigated the interplay between the spreading of risk and the diffusion of awareness. The results reveal that the local awareness ratio θ_1 and the global awareness ratio θ_2 both can lead to the different risk propagation sizes, while the local awareness ratio θ_1 has the two-stage effects on risk propagation threshold β_c without considering of global awareness no matter what values of other parameters. While the global awareness ratio θ_2 has the same phenemon on the effects of threshold β_c ingnoring the local awareness. However, when considering both the effects of global awareness and local awareness, three different areas exsit accoring to two gropus of different values of θ_1 and θ_2 .

By assuming that the level of firm's risk triggering probability increases with cooperation intensity and local risk prevalence, our simulations show that the cooperation intensity can decrease the risk propagation threshold and promote the final risk propagation size effectively. However, the local risk prevalence can only augment the final risk propagation size, which cannot alter the risk propagation threshold β_c . Furthermore, our results could provide some useful suggestions on the prevention and control risk propagation. For instance, encouraging information communication between neighbors and developing a shared-information platform to realize information in whole R&D network are effective ways to decrease local (global) awareness ratio.

There are some limitations in this research. Frist, only the heterogeneity of firm in risk propagation layer has been considered in the model. However, heterogeneity also exists in the awareness perception, thus the heterogeneity in information layer should consider in the future research. Secondly, since some firms might join or quit R&D network over time, which causes the changing of topology structure. In this situation, considering temporal networks could serve as a good point in the future direction.

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