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AN EVOLVING NETWORK MODEL OF CREDIT RISK CONTAGION IN THE FINANCIAL MARKET

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Abstract. This paper introduces an evolving network model of credit risk contagion containing the average fitness of credit risk contagion, the risk aversion sentiments, and the ability of resist risk of credit risk holders. We discuss the effects of the aforementioned factors on credit risk contagion in the financial market through a series of theoretical analysis and numerical simulations. We find that, on one hand, the infected path distribution of the network gradually increases with the increase in the average fitness of credit risk contagion and the risk aversion sentiments of nodes, but gradually decreases with the increase in the ability to resist risk of nodes. On the other hand, the average fitness of credit risk contagion and the risk aversion sentiments of nodes increase the average clustering coefficient of nodes, whereas the ability to resist risk of nodes decreases this coefficient. Moreover, network size also decreases the average clustering coefficient.

Keywords: credit risk contagion, preferential node deletion, behavioral factors, evolving network model.

JEL Classification: A12, C15, C80, E22, E44, G32.

Introduction

In the modern financial market, an intricate web of credit relations links a wide variety of creditors and debtors in a complex system. Once credit relations are destroyed, credit events will occur in droves, thus the value of credit assets will declined sharply. This decline will lead to credit risk contagion in the financial market, particularly the credit risk transfer (CRT) market. Moreover, the advent of sophisticated financial products involving credit default swaps and collateralized debt obligations does not only increase the complexity of the population of credit risk investors, but also heightens the contagion effect of credit risk among credit risk investors. Complex credit connections between creditors

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and debtors have a double-edged effect. On one hand, complex credit connections increase the stability of a financial system, including those using the interbank market to develop relationships that can protect them against liquidity risk (Kahn, Santos 2005; Cocco *et al.* 2009), and those using the credit risk transfer market to decentralize the risk of a financial system (Baur, Joossens 2006; Wagner, Marsh 2006; Neyer, Heyde 2010). On the other hand, credit risk contagion and the corresponding fragility of a financial system also result from the complex credit connections between creditors and debtors (Kahn, Santos 2005; Allen, Carletti 2006; Santos 2006; Chen *et al.* 2015), particularly the contagion effect of credit risk among credit risk holders (Chen *et al.* 2014, 2015; Bo, Capponi 2015). In the financial market, values or price fluctuations of credit assets will induce the contagion effect of credit risk among credit risk holders, thus eventually leading to a financial crisis. Numerous studies show that the knock-on effect of an initial default of several financial institutions on asset prices can trigger further rounds of defaults as other financial entities are forced to write down their assets values because of complex interconnections between credit assets and the psychology bias of credit risk investors (Cifuentes *et al.* 2005; Shin 2008; Chen *et al.* 2013, 2015).

Behavioral factors of credit risk holders and financial market regulators have important effects on credit risk contagion, particularly investor sentiment. The market behavioral approach recognizes that investors are not "rational" but "boundedly rational", and that systematic biases in their beliefs cause them to trade on non-fundamental information, called "sentiment" (Zouaoui *et al.* 2011). Several financial economists also recognize that the market has mood swings. Soon, the link between asset valuation and investor sentiment becomes the subject of considerable deliberation among financial economists. Theories departing from rational asset pricing often posit the influence of investor sentiment (De Long *et al.* 1990), which leads to price fluctuation, and risk contagion generation. A number of theoretical studies offer models for establishing the relationship between investor sentiment and assets prices (De Long *et al.* 1990; Barberis *et al.* 1998; Kent *et al.* 2001; Baker, Wurgler 2007). Investors have two types in these models: rational arbitrageurs who are sentiment-free and irrational traders prone to exogenous sentiment (Baker, Stein 2004). Baker and Stein (2004) find that total sentiment, particularly the global component of total sentiment, is a contrarian predictor of country-level market returns. Baker and Wurgler (2006, 2007) predict that extensive waves of sentiment will exert greater effects on hard-to-arbitrage and hard-to-value stocks, which exhibit high "sentiment beta" (Glushkov 2005). Hence, given that sentiment influences valuation, taking a position opposite to the prevailing market sentiment can be expensive and risky. Meanwhile, several theoretical studies show that investor sentiment is the most relevant in the decision-making domain, which primarily affects investor personal investment decisions (Lin *et al.* 2006). Baker and Wurgler (2006) pointed out that sentiment-based mispricing is based on the uninformed demand of several investors, noise traders, and a limit to arbitrage. Mispricing can be persistent given that the length of period during which overly optimistic and pessimistic noise traders will continue exerting buying or selling pressures is unknown. Similarly, numerous significant studies in this area are available (Hertel *et al.* 2000; Loewenstein *et al.* 2001; Yuen, Lee 2003; Raghunathan, Corfman 2004). Recently, theoretical studies have found that investor sentiment is

contagious across markets (Baker *et al.* 2012), thus providing clues on how investor sentiment induces the spread of risk. The effect of the behaviors of credit risk investors have been a concern of credit risk contagion (Allen, Carletti 2006; Santos 2006; Neyer, Heyde 2010; Chen, He 2012; Chen *et al.* 2014, 2015). This concern is also our motivation in considering the effect of the risk sentiment of credit risk investors on the evolving network of credit risk contagion. In addition, we will introduce the ability to resist risk to analyze the effect of this ability of credit risk holders on credit risk contagion. Therefore, our paper will contribute to a deeper understanding of the effects of the risk aversion sentiments and the ability to resist risk of credit risk holders on credit risk contagion.

Given the significant development of complex network theory, there are a number of scholars have looked for evidence of contagion risk in the financial system which results from complex credit connections. The most well-known contribution to contagion analysis through direct linkages in the financial system is Allen and Gale (2000). This work demonstrates that the spread of contagion depends crucially on the pattern of interconnectedness among banks through a network structure involving four banks. Since the publication of this work, numerous scholars have applied complex network theory to model the complex structure of the financial system and to analyze risk contagion in the financial system, particularly in banking systems. Several theoretical studies have found that the network structure is crucial to credit risk contagion (Barro, Basso 2010; Chen, He 2012), including the random (Iori *et al.* 2006; Chen, He 2012) and tiereds structures (Nier *et al.* 2007; Teteryatnikova 2009; Canedo, Jaramillo 2009; Georg, Poschmann 2010; Gai, Kapadia 2010; Li 2011). These theoretical studies examine risk contagion in banking systems via direct linkages among banks, whereas others analyze risk contagion via indirect linkages (Dasgupta 2004; De Vries 2005; Babus 2006; Vivier-Lirimont 2009; Jorion, Zhang 2009; Bo, Capponi 2015). The aforementioned studies show that the network structure can significantly affect credit risk contagion. Recently, a number of theoretical studies found that the effect of adding and deleting nodes and edges is prominent on the network structure. Bollobás and Riordan (2004b) consider the effect of deleting vertices from the basic preferential attachment model of Barabási and Albert (1999) and Bollobás and Riordan (2004a) on the network structure. Sarshar and Roychowdhury (2004) study ad hoc networks and demonstrate that even in limited equal insertion and deletion rates, true scale-free structures emerge, wherein the degree distribution obeys a power law with a tunable exponent. Cooper *et al.* (2004) study deletion of vertices within a dynamic setting. Moore *et al.* (2006) study the general process in which a network grows (or potentially shrinks) through the constant addition and deletion of vertices and edges. Deng *et al.* (2007) introduce a new type of network growth rule comprising addition and deletion of nodes, and propose an evolving network model to investigate the effect of node deletion on network structure. Gu and Sun (2008) study a local-world node-deleting evolving network. These theoretical studies consider node deletion as random, that is the probability of node deletion is constant. However, other minor studies have analyzed preferential node deletion. Deo and Cami (2007) investigate a dynamic random graph model that interweaves addition of nodes and edges with a preferential node deletion favoring removal of small-degree nodes. Prior studies show that network structure can affect risk contagion. Therefore, we will consider the

effect of the change in network structure on credit risk contagion, which primarily use the average fitness of nodes, the risk aversion sentiments, and the ability to resist risk of credit risk holders in describing the preferential deletion mechanism of nodes. Our objective is to understand the effect of the change in network structure resulting from the average fitness, the risk aversion sentiments, and the ability to resist risk of nodes on credit risk contagion.

The structure of this paper is as follows. In Section 1, we develop an evolving network model of credit risk contagion in the financial market. In Section 2, we conduct a theoretical analysis and numerical simulations to study the effects of the average fitness, the risk aversion sentiments, and the ability to resist risk of nodes on the evolving network model of credit risk contagion. The last section provides concluding remarks.

1. The evolving network model of credit risk contagion

Consider a network of credit risk contagion in the financial market that evolves through the preferential deletion of old nodes. In the initial state, the network has a small number of m_0 nodes that have been infected with credit risk by other nodes. Hence, the network of credit risk contagion in the financial market is evolved through the following scheme.

At each time step, we conduct either (a) or (b).

(a) Credit risk contagion effect. The algorithm is achieved as follows.

1) Contagion effect: infect a new node with m infected path connected to nodes infected with credit risk by other nodes in the network. The number of infected paths of node i gradually increases or decreases with the evolving credit risk contagion.
2) Preferential contagion: contagion is from the m edges because these edges connected to infectious nodes infected with credit risks by other nodes. The probability for node i being selected to connect with node j which has been infected with credit risk by other nodes in the network is:

$$\Pi_i = \frac{\eta^{(1-\beta_j)\log\theta_j} k_j}{\sum_h \eta^{(1-\beta_h)\log\theta_h} k_h}, \tag{1}$$

where η is the average fitness of credit risk contagion in the network, and η is chosen from a fitness distribution $f(\eta)$. β_i is the risk aversion sentiments of node i for credit risk contagion in the financial market, θ_i is the resistance of node i for credit risk contagion. $\eta^{(1-\beta_i)\log\theta_i}$ indicates the contagion power of the credit risk of node i. If θ_i is greater, then the credit risk contagion power of node i is smaller. Moreover, if β_i is greater, then the credit risk contagion power of node i is greater. Thus, behavioral factors β_i and θ_i of node i determine the heterogeneity of node i for credit risk contagion. Assuming $\beta_i \in [0,1]$ and $\theta_i \in [1,10]$, if β_i is greater, then the resistance of node i to credit risk contagion is stronger. If θ_i is greater, then the resistance of node i to credit risk contagion is stronger.

(b) Node deletion: the risk aversion sentiments β_i of node i will increase the probability of node i deletion, whereas resistance θ_i of node i will decrease the probability of node i dele-

tion. Thus, the effects of the risk aversion sentiments β_i and resistance θ_i of node i deletion on credit risk contagion are considered. Assuming that an old node i is deleted preferentially from the network and that all the edges attached to this node are also removed, then the preferential probability is:

$$p_i = \frac{\beta_i}{\theta_i}. \tag{2}$$

Therefore, we can easily determine that the probability of the preferential deletion of node i is high because the risk aversion sentiments β_i is big and resistance θ_i is small, i.e., the preferential probability of the deletion of node i is positively related to the risk aversion sentiments β_i, and negatively related to the resistance θ_i of node i for credit risk contagion. This finding agrees with the actual financial market in a certain sense.

We adopt the same approach as introduced in references (Gu, Sun 2008; Dorogovtsev, Mendes 2001; Sarshar, Roychowdhury 2004; Deng *et al.* 2007) for our analysis. In the financial market, a node deletion will affect property and behavior of credit risk contagion, thus causing several nodes to become infected with credit risk by other nodes because of the relationship among them. To obtain information on the effect of node deletion on credit risk contagion, let $D_i(j, t)$ denotes the probability that the node is infected with credit risk at time step j and has not been deleted until time step t is easy, where $t > j$. We use the independence of events corresponding to the preferential deletions of nodes at each time step, thus verifying that $D_i(j,t+1) = D_i(j,t)[1-(1-p_i)/N(t)]$ with $D_i(t,t) = 1$, $N(t) = (1-p_i)t$, where $N(t)$ is the total number of nodes that infected with credit risk in the network. Therefore, according to the continuous limit, the dynamic equation of $D_i(j, t)$ can be stated as follows:

$$\frac{\partial D_i(j,t)}{\partial t} = -p_i \frac{D_i(j,t)}{N(t)} = \frac{p_i}{p_i - 1}\frac{D_i(j,t)}{t}. \tag{3}$$

Which can obtain:

$$D_i(j,t) = \left(\frac{t}{j}\right)^{p_i/(p_i-1)}. \tag{4}$$

Thus, we obtain $D_i(j, t)$ with the risk aversion sentiments β_i and resistance θ_i, i.e.,

$$D_i(j,t) = \left(\frac{t}{j}\right)^{\beta_i/(\beta_i-\theta_i)}. \tag{5}$$

We can easily determine that $D_i(j, t)$ has a significant negative correlation with $t - j$, i.e., $D_i(j, t)$ decreases rapidly as $t - j$ increases. Moreover, $D_i(j, t)$ has a significant negative correlation with risk aversion sentiments β_i of node i, and a significant positive correlation with resistance θ_i of node i. We know that highly connected nodes, or hubs, have important roles in the properties and behavior of credit risk contagion. In the financial market, a huge portion of highly connected nodes or hubs is easier to delete during the evolution of credit risk contagion. This phenomenon is reason for the easy clustering of credit risk during credit risk contagion, and consequently causing financial assets to shrink sharply. To verify these finding, we have conducted numerous simulations for a wide range of β_i

and θ_i values. Figures 1 and 2 show the results for a wide range of β_i and θ_i values. In Figure 1, we provide the survival probability $D_i(j, t)$ as a function of different β_i and θ_i values in our model for $j = 100$. We determine that the survival probability $D_i(j, t)$ is a monotonic diminishing concave function of β_i and θ_i during the evolution of credit risk contagion. In Figure 2, we provide the survival probability $D_i(j, t)$ under the interaction effects of β_i and θ_i in our model, for $j = 100$ and different t values. We find similar results.

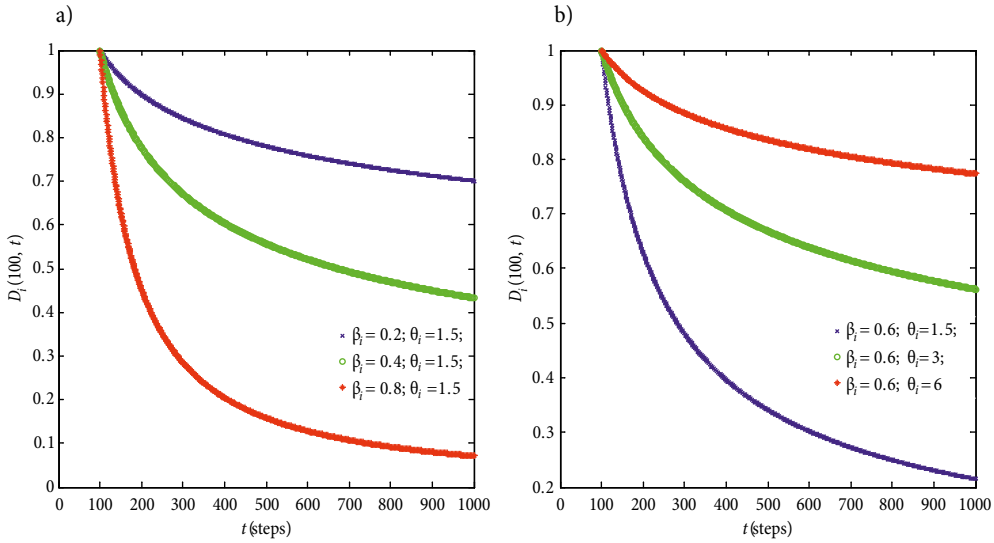

Fig. 1. The survival probability $D_i(j, t)$ that node i is infected with credit risk at time step 100, and has not been deleted until time step t as a function of β_i and θ_i, where $t > 100$

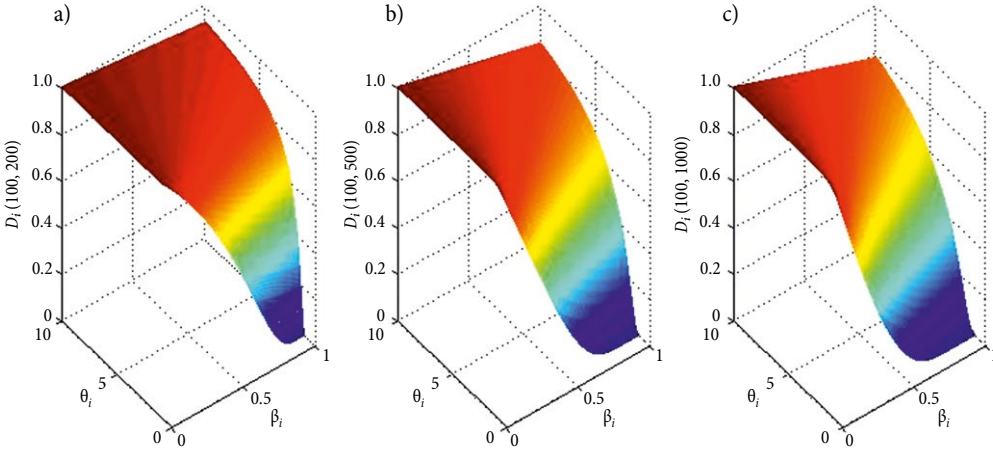

Fig. 2. The survival probability $D_i(j, t)$ of node i under the interaction effects of β_i and θ_i. (a) $j = 100$, $t = 200$; (b) $j = 100$, $t = 500$; (c) $j = 100$, $t = 1000$

2. Evolving network analysis of credit risk contagion with behavioral factors

We will provide a theoretical analysis and numerical simulations of the network of credit risk contagion to study the effect of the average fitness of credit risk contagion, the risk aversion sentiments of nodes, and the ability to resist risk of nodes on the infected path distribution $P(k)$ and the average clustering coefficient $\langle C \rangle$. Furthermore, we analyze the evolving properties of the network of credit risk contagion with behavioral factors β_i and θ_i of node i.

In the numerical simulations, we initialize the contagion network with $m_0 = 10$ nodes being infected with credit risk. If we decide to infect a new node at each time step t, then the nodes with $m = 5$ edges are chosen to connect with $m_0 = 10$ nodes infected with credit risk in the contagion network.

2.1. The infected path distribution and the effect of behavioral factors

Assuming that a node is infected at time step j, and the ith node is still in the network of credit risk contagion at time t. Let $k_i(j, t)$ be the infected path of the ith node at time t, and $t > j$. Therefore, according to the continuous limit, the rate of $k_i(j, t)$ during which the infected path is expected to increase is:

$$\frac{\partial k_i(j,t)}{\partial t} = m\frac{k_i(j,t)\eta^{(1-\beta_i)\log\theta_i}}{S(t)} - \frac{\beta_i}{\theta_i}\frac{k_i(j,t)}{N(t)}, \tag{6}$$

where

$$S(t) = \int_0^t D_i(j,t)k_i(j,t)dj. \tag{7}$$

The first term in Eq. (6) can be easily verified as the increasing number of the infected paths of node i during credit risk contagion resulting from the m preferential attachments made by the newly infected node. The second term in Eq. (6) accounts for the disappearance of an infected path of the ith node during preferential node deletion, which happens with the probability $k_i(j,t)/N(t)$.

To solve for $S(t)$, we initially multiply both sides of Eq. (6) by $D_i(j, t)$ and integrate j from 0 to t. Then:

$$\int_0^t D_i(j,t)\frac{\partial k_i(j,t)}{\partial t}dj = m\eta^{(1-\beta_i)\log\theta_i} - \frac{\frac{\beta_i}{\theta_i}}{N(t)}S(t). \tag{8}$$

The left side of Eq. (8) can be simplified as follows:

$$\int_0^t D_i(j,t)\frac{\partial k_i(j,t)}{\partial t}dj = \frac{\partial\int_0^t D_i(j,t)\frac{\partial k_i(j,t)}{\partial t}dj}{\partial t} - k_i(t,t)D_i(t,t) + \frac{\frac{\beta_i}{\theta_i}}{N(t)}\int_0^t D_i(j,t)k_i(j,t)dj. \tag{9}$$

Based on these equations, we know that $k_i(t,t) = m$, $D_i(t,t) = 1$, $N(t) = (1-p_i)t = \left(1 - \frac{\beta_i}{\theta_i}\right)t$, and $S(t) = \int_0^t D_i(j,t)k_i(j,t)dj$. Hence, we can obtain:

$$\int_0^t D_i(j,t)\frac{\partial k_i(j,t)}{\partial t}dj = \frac{\partial S(t)}{\partial t} - m + \frac{\frac{\beta_i}{\theta_i}}{N(t)}S(t). \tag{10}$$

We substitute Eq. (8) into Eq. (10) and obtain:

$$S(t) = m[1 + \eta^{(1-\beta_i)\log\theta_i}]\frac{\theta_i - \beta_i}{\theta_i + \beta_i}. \quad (11)$$

We substitute Eq. (11) into Eq. (6) and obtain:

$$\frac{\partial k_i(j,t)}{\partial t} = \frac{mk_i(j,t)\eta^{(1-\beta_i)\log\theta_i}}{m(1+\eta^{(1-\beta_i)\log\theta_i})\frac{\theta_i-\beta_i}{\theta_i+\beta_i}} - \frac{\beta_i}{\theta_i-\beta_i}\frac{k_i(j,t)}{t} = \frac{\theta_i\eta^{(1-\beta_i)\log\theta_i} - \beta_i}{(1+\eta^{(1-\beta_i)\log\theta_i})(\theta_i-\beta_i)}\frac{k_i(j,t)}{t}. \quad (12)$$

The solution to Eq. (12) with $k_i(j,j) = m$ is:

$$k_i(j,t) = m\left(\frac{t}{j}\right)^{\frac{\theta_i\eta^{(1-\beta_i)\log\theta_i} - \beta_i}{(1+\eta^{(1-\beta_i)\log\theta_i})(\theta_i-\beta_i)}}. \quad (13)$$

Eq. (6) is significant because it states that the infected path of a node in the network (when it is not deleted) depends on the average fitness η of credit risk contagion in the network, the risk aversion sentiments β_i, and the resistance θ_i of node i for credit risk contagion. These factors will determine the infected path of a node in an evolving network of credit risk contagion. To verify this statement, we have conducted numerous simulations for a wide range of β_i, θ_i and η. Figures 3 and 4 show the results for the different β_i, θ_i and η values.

In Figure 3, we plot $k_i(j, t)$ as a function of different β_i, θ_i and η values at time $j = 100$, where $t > j$. In Figure 4, we also plot $k_i(j, t)$ as a function of different β_i, θ_i and η values at time $j = 100$ and $t = 1000$. Based on a comprehensive analysis of Figures 3(a) and 4(a), we can see that the infected path $k_i(j, t)$ of node i has a significant positive relation with the risk aversion sentiments β_i of node i, and $k_i(j, t)$ is monotonic increasing concave function of the risk aversion sentiments β_i of node i. Figures 3(b) and 4(b) show that the infected path $k_i(j, t)$ of node i has a significant negative relation with the resistance θ_i of node i, and $k_i(j, t)$ is a monotonic diminishing concave function of the resistance θ_i of node i. From Figure 3(c), we can see that the infected path $k_i(j, t)$ of node i has a significant positive relation with the average fitness η of the network of credit risk contagion. From Figure 4(c), we can see that $k_i(j, t)$ is a monotonic increasing convex function of η. Therefore, Figures 3 and 4 effectively describe the effects of the average fitness η of the network of credit risk contagion, the risk aversion sentiments β_i, and the resistance θ_i of node i for credit risk contagion on the infected path $k_i(j, t)$ of node i.

To obtain the probability $P(k_i, t)$ that a randomly chosen node i at time t will have the infected path $k_i(j, t)$, we calculate the expected number of nodes at time t with the infected path $k_i(j, t)$ and divide it by the total number $N(t)$ of nodes, i.e., $P(k_i, t) = N_{k_i}(t)/N(t)$. Let $J_k(t)$ represent the set of all nodes with the infected path $k_i(j, t)$ at time t, then we can obtain:

$$P(k_i, t) = \frac{N_{k_i}(t)}{N(t)} = \frac{1}{N(t)}\sum_{j\in J_k(t)} D_i(j,t). \quad (14)$$

According to the continuous-time approach, we can obtain:

$$\sum_{j\in J_k(t)} D_i(j,t) = D_i(j,t)\left|\frac{\partial k_i(j,t)}{\partial j}\right|^{-1}_{j=j_k}. \quad (15)$$

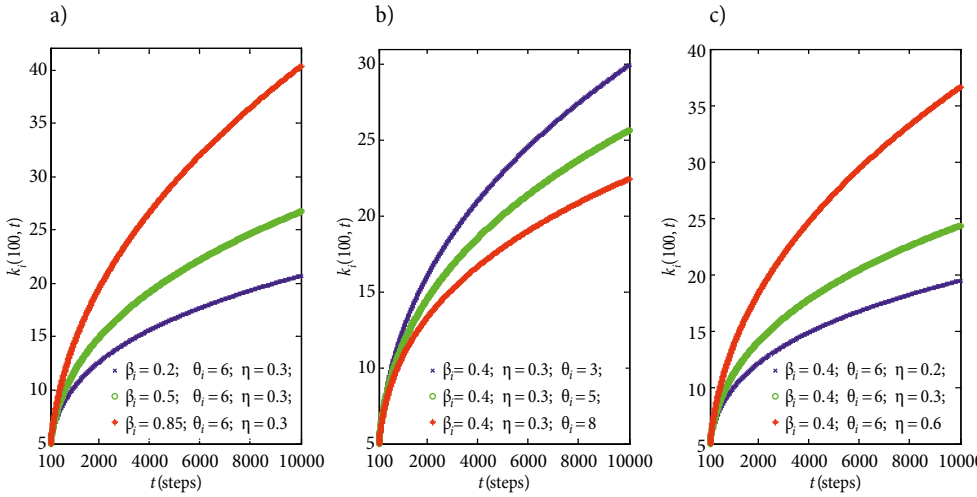

Fig. 3. The evolution of the infected path of a node infected at time $j = 100$ in the network of credit risk contagion in logarithmic scales. (a) The effect of the risk aversion sentiments β_i of node i on its infected path at $\theta_i = 6$ and $\eta_i = 0.3$. (b) The effect of the resistance θ_i of node i on its infected path at $\beta_i = 0.4$ and $\eta_i = 0.3$. (c) The effect of the average fitness η of credit risk contagion on its infected path at $\beta_i = 0.4$ and $\theta_i = 6$. The continuous curve is according to the analytical result of Eq. (13)

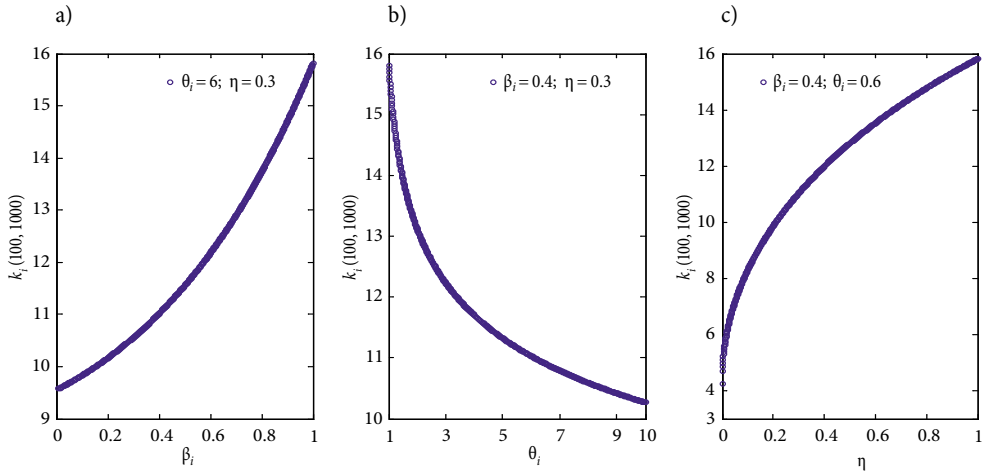

Fig. 4. The effect of the risk aversion sentiments β_i, the resistance θ_i, and the average fitness η of credit risk contagion in the network on the infected path of node i at $j = 100$ and $t = 1000$

From Eq. (13), we obtain:

$$j_k = t\left(\frac{k_i}{m}\right)\frac{(1+\eta^{(1-\beta_i)\log\theta_i})(\theta_i-\beta_i)}{\beta_i-\theta_i\eta^{(1-\beta_i)\log\theta_i}}. \tag{16}$$

When Eq. (16) is inserted into Eq. (5), we can obtain:

$$D_i(j_k, t) = \left(\frac{m}{k_i}\right)\frac{(1+\eta^{(1-\beta_i)\log\theta_i})\beta_i}{\beta_i-\theta_i\eta^{(1-\beta_i)\log\theta_i}}. \tag{17}$$

$$\left|\frac{\partial k_i(j,t)}{\partial j}\right|^{-1}_{j=j_k} = t\frac{(1+\eta^{(1-\beta_i)\log\theta_i})(\theta_i-\beta_i)}{m(\theta_i\eta^{(1-\beta_i)\log\theta_i}-\beta_i)}\left(\frac{k_i}{m}\right)^{\frac{2\theta_i\eta^{(1-\beta_i)\log\theta_i}+\theta_i-2\beta_i-\beta_i\eta^{(1-\beta_i)\log\theta_i}}{\theta_i\eta^{(1-\beta_i)\log\theta_i}-\beta_i}}. \quad (18)$$

When Eqs (17) and (18) are inserted into Eq. (14), we can obtain:

$$P(k_i,t) = \frac{(1+\eta^{(1-\beta_i)\log\theta_i})\theta_i}{m(\theta_i\eta^{(1-\beta_i)\log\theta_i}-\beta_i)}\left(\frac{k_i}{m}\right)^{-\frac{2\theta_i\eta^{(1-\beta_i)\log\theta_i}+\theta_i-\beta_i}{\theta_i\eta^{(1-\beta_i)\log\theta_i}-\beta_i}}. \quad (19)$$

Which is a power-law distribution with the exponent:

$$\gamma = 1+\frac{\theta_i\eta^{(1-\beta_i)\log\theta_i}+\theta_i}{\theta_i\eta^{(1-\beta_i)\log\theta_i}-\beta_i}. \quad (20)$$

Therefore, the infected path k_i of the credit risk contagion network does not depend on time step t, but on the average fitness η of the network of credit risk contagion, the risk aversion sentiments β_i, and the resistance θ_i of node i on the infected path $k_i(j, t)$ of node i, which follows a power law. We will provide the numerical simulations of the infected path distribution $P(k)$. Hence, we verify the aforementioned theoretical analysis and analyze further the effects of the average fitness η of the network of credit risk contagion, the risk aversion sentiments β_i, and the resistance θ_i of node i on the infected path $k_i(j, t)$ of node i through numerous simulations. In Figure 5, we provide the infected path distributions $P(k)$ of the network of credit risk contagion with different β_i, θ_i and η values. From Figure 5, the infected path distribution $P(k)$ of the network of credit risk contagion is power-law with changing β_i, θ_i, and η values. As β_i and η increase, Figures 5(a) and 5(c) show an interesting transition process for $P(k)$, i.e., the values of $P(k)$ increase as β_i and η increase. This shows that the probability of a node infected with credit risk increases gradually as β_i and η gradually increase. Moreover the effect of η on the infected path distribution $P(k)$ is more significant than that of β_i. From Figure 5(b), we can see that the effect of θ_i on

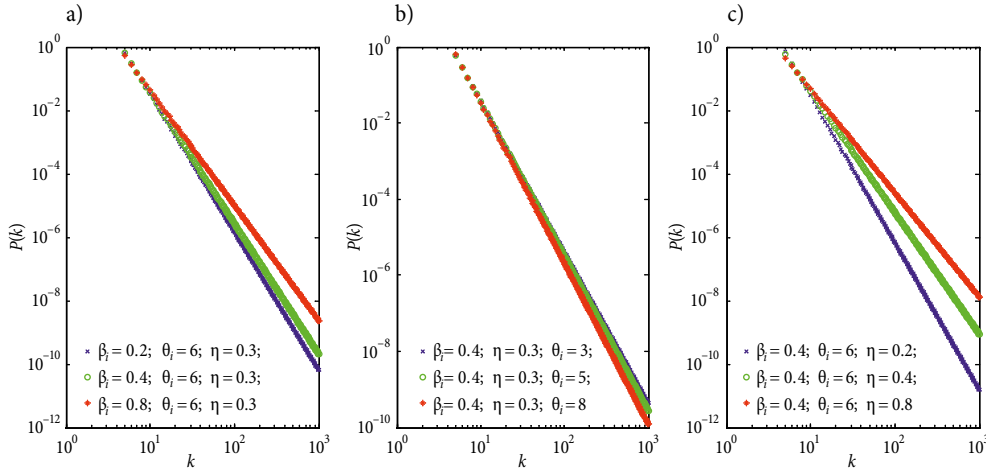

Fig. 5. The path distribution of nodes $P(k)$ infected with credit risk at network size M = 1000 of credit risk contagion and different values of β_i, θ_i and η in logarithmic scales

the infected path distributions $P(k)$ is opposite to those of β_i and η, i.e., the value of $P(k)$ decreases as θ_i increases. Therefore, the numerical simulation results are consistent with that of the theoretical analysis.

2.2. The average clustering coefficient of credit risk contagion and the effect of behavioral factors

In the network of credit risk contagion, the average clustering coefficient $\langle C \rangle$ can quantify the extent to which nodes adjacent to an infected node are infected with credit risk. For a selected node i with the infected path k_i in the network, let E_i denote the number of infected paths among its k_i neighbor nodes, then the clustering coefficient C_i of node i is defined as follows:

$$C_i = \frac{2E_i}{k_i(k_i - 1)}. \tag{21}$$

Therefore, the average clustering coefficient $\langle C \rangle$ of the network of credit risk contagion can be defined as follows:

$$\langle C \rangle = \frac{\sum_i C_i}{M} = \frac{1}{M} \sum_i \frac{2E_i}{k_i(k_i - 1)}. \tag{22}$$

$\langle C \rangle$ is closely related to the network size M of credit risk contagion, the average fitness η of credit risk contagion in the network, the risk aversion sentiments β_i of node i, and the resistance θ_i of nodes i. Then, we conduct numerous simulations to intuitively describe the effect of M, η, β_i, and θ_i on the average clustering coefficient $\langle C \rangle$ of the network of credit risk contagion. In Figure 6, we plot the $\langle C \rangle$ of the giant component in the network of credit risk contagion as a function of network size M for different η, β_i, and θ_i values. In Figure 7, we plot $\langle C \rangle$ as a function of η, β_i, and θ_i for different M values. In Figures 6 and 7, we can see that the average clustering coefficient $\langle C \rangle$ of the network of credit risk

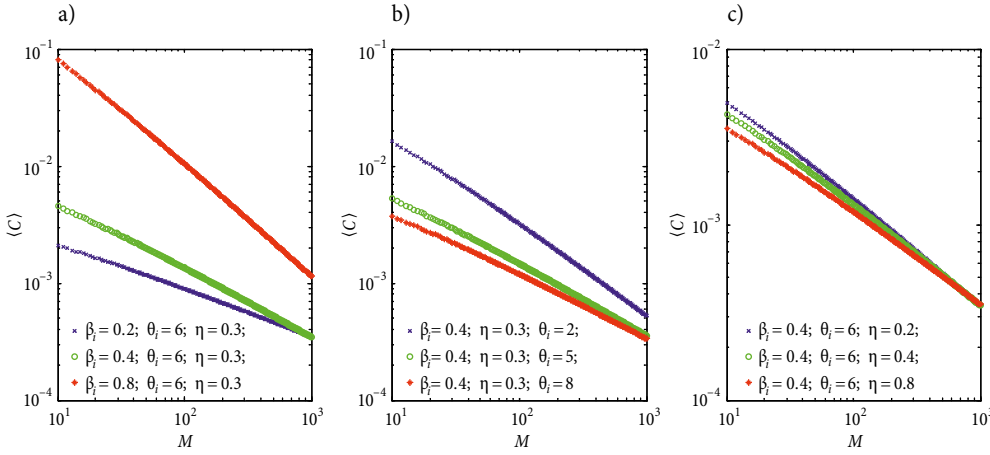

Fig. 6. The average clustering coefficient $\langle C \rangle$ of the network of credit risk contagion as a function of network size M, for (a) the average fitness η of credit risk contagion in the network, (b) the risk aversion sentiments β_i of node i on credit risk contagion, and (c) the resistance θ_i of node i on credit risk contagion

contagion decreases as the network size of credit risk contagion M gradually increases. When the network size M of credit risk contagion is constant, the average clustering coefficient $\langle C \rangle$ of the network of credit risk contagion increases as β_i and η increase, and the effect of θ_i is more significant than that of η. However, the effect of θ_i on the average clustering coefficient $\langle C \rangle$ is opposite to those of β_i and η, i.e., the average clustering coefficient $\langle C \rangle$ gradually decrease as θ_i increases. From Figure 7(a), we can see that the effect of β_i on the average clustering coefficient $\langle C \rangle$ is a concave and monotonically increasing curve. However, the effect of θ_i on the average clustering coefficient $\langle C \rangle$ is a concave and monotonic decreasing curve, as shown in Figure 7(b). From Figure 7(c), we can see that

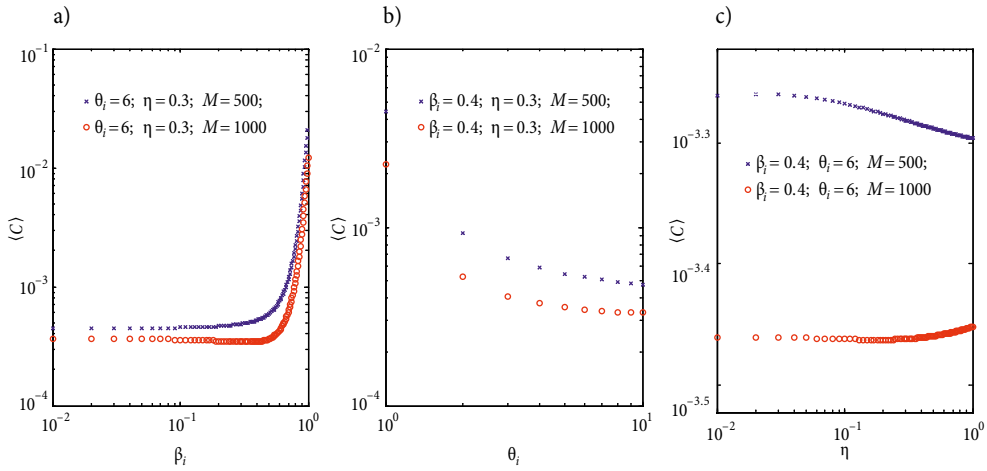

Fig. 7. The average clustering coefficient $\langle C \rangle$ of the network of credit risk contagion as a function of (a) the average fitness η of credit risk contagion in the network, (b) the risk aversion sentiments β_i of node i on credit risk contagion, and (c) the resistance θ_i of node i on credit risk contagion, for the network size M = 500 and M = 1000

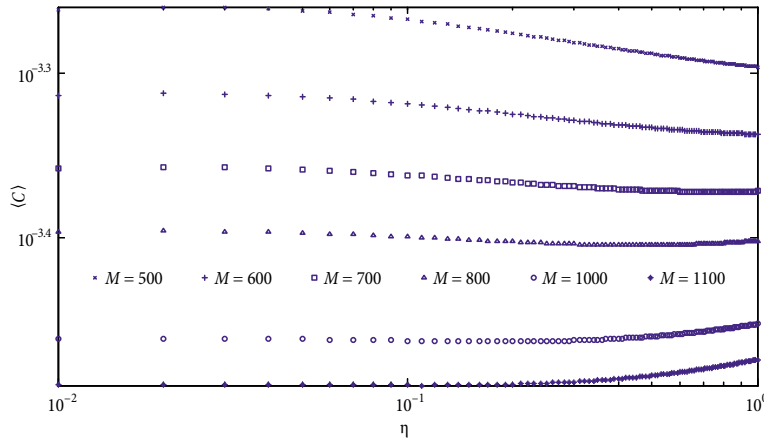

Fig. 8. The average clustering coefficient $\langle C \rangle$ of the network of credit risk contagion as a function of the average fitness η of credit risk contagion in the network, for different network size M, where β_i = 0.4 and θ_i = 0.6

the curve of the average clustering coefficient $\langle C \rangle$ exhibits tail-raising characteristics as η increases at credit risk contagion network size $M = 1000$. However, at credit risk contagion network size $M = 500$, the curve of the average clustering coefficient $\langle C \rangle$ exhibits a tail-falling phenomenon. The transition process, which is the gradual change process during which the network size of credit risk contagion M gradually increases is shown in Figure 8.

Conclusions

In this paper we introduced a new network contagion rule which comprises credit risk contagion effect and preferential node deletion with the average fitness of credit risk contagion, the risk aversion sentiments of nodes, and the ability to resist risk of nodes. We discussed the effects of the average fitness of credit risk contagion, the risk aversion sentiments of nodes, and the ability to resist risk of nodes on credit risk contagion through a series of theoretical analysis and numerical simulations. First, the survival probability $D_i(j, t)$ of node i is a monotonic diminishing concave function of β_i and θ_i during the evolution of credit risk contagion. Second, the infected path distribution $P(k)$ of the network of credit risk contagion exhibits a series of power-law properties as the average fitness of credit risk contagion in the network, the risk aversion sentiments, and the resistance of node change. $P(k)$ increases as the average fitness of credit risk contagion in the network and the risk aversion sentiments of node increase, but it decreases as the resistance of node increases. Third, we found that the average fitness of credit risk contagion in the network and the risk aversion sentiments of nodes increases the average clustering coefficient $\langle C \rangle$ of the network of credit risk contagion, but the resistance of node will decrease the average clustering coefficient $\langle C \rangle$. This finding occurs because the risk aversion sentiments of nodes increases the credit risk contagion effect and the preferential probability of node deletion. However, the resistance of nodes decreases the credit risk contagion effect and the preferential probability of node deletion. Thus, preferential node deletion will remove a huge portion of potential hubs with greater risk aversion sentiments and weaker resistance of nodes during the network evolution of credit risk contagion. In addition, the network size M of credit risk contagion can also decrease the average clustering coefficient $\langle C \rangle$.

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