Hindawi
Complexity
Volume 2018, Article ID 2929157, 11 pages
https://doi.org/10.1155/2018/2929157

WILEY | Hindawi

Research Article

Credit Risk Contagion Based on Asymmetric Information Association

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Received 8 February 2018; Revised 5 May 2018; Accepted 3 June 2018; Published 11 July 2018

Academic Editor: Andreas Flache

The study of the contagion law of credit risk is very important for financial market supervision. The existing credit risk contagion models based on complex network theory assume that the information between individuals in the network is symmetrical and analyze the proportion of the individuals infected by the credit risk from a macro perspective. However, how individuals are infected from a microscopic perspective is not clear, besides the level of the infection of the individuals is characterized by only two states: completely infected or not infected, which is not realistic. In this paper, a credit risk contagion model based on asymmetric information association is proposed. The model can effectively describe the correlation among individuals with credit risk. The model can analyze how the risk individuals are infected in the network and can effectively reflect the risk contagion degree of the individual. This paper further analyzes the influence of network structure, information association, individual risk attitude, financial market supervision intensity, and individual risk resisting ability on individual risk contagion. The correctness of the model is verified by theoretical deduction and numerical simulation.

1. Introduction

Credit risk refers to the risk of economic loss caused by the failure of the counterparty to fulfill the obligations stipulated in the contract, and it is the main type of financial risk. In recent years, the contagion effects of credit risk occur frequently in financial markets, which have caused severe impacts on the financial market and economic development of almost all market economy countries. The subprime mortgage crisis, which originated in the United States in 2008, eventually became a global financial crisis and shocked the global capital market. The global financial risks caused by the subprime mortgage crisis in the United States are directly reflected in the credit risks and contagion effects in the financial market. Credit is the cornerstone of the market economy, and the risk of the capital market is largely from the credit crisis. The contagion of credit risk will increase the complexity of credit risk in the capital market and reduce the transparency of credit risk in the capital market. At present, the introduction and rapid development of CRT (credit risk transfer) market make credit risk management more difficult, and the credit risk contagion is more extensive [1–4]. Therefore, the study of the contagion law of credit risk in the financial market has attracted much attention of researchers.

At present, the research on the contagion model of credit risk in the financial market mainly includes the following three categories: the simplified model, the structured model, and the complex network evolution model. The stochastic theory-based simplified model and structured model are used to describe the impact and contagion effects on the creditor under different circumstances of credit default strength and default loss rate of the debtor [5–10]. The method of structural model assumes that the dynamic process of corporate assets depends on a set of common state variables, and that the interfirm default correlation arises from the dynamic evolution of the firm's asset value [9]. The simplified model directly models the process of corporate default intensity, and the default correlation is determined by the intensity of the default process, without considering the relationship between the default and the company value. Comparing with

other models, the simplified model is easier to calculate the default intensity and becomes the main framework for the study of the contagion model of credit risk [10]. The simplified model and the structured model mainly assume that credit default is exogenous. The influence of endogenous factors such as psychological behavior, correlation mechanism, and network structure on the behavior of the model is not considered in the modeling of credit risk contagion for the simplified model and the structured model, while the process of credit risk contagion is the result of many endogenous factors and exogenous factors [11]. The latest research in behavioral finance believes that the psychological characteristics of investors seriously affect people's psychological expectations and decision-making behavior [12] and increase market risk and investors' attitudes and emotions can be transmitted in the market [13]. Therefore, in the study of credit risk contagion, the influence and function of economic subject's psychological and behavioral factors cannot be ignored [14, 15]. Many researches indicate that the network structure of credit risk holders also has an important impact on the spread of risk [16]. In addition, empirical research shows that market supervision has a strong inhibitory effect on the infection of credit risk [17]. Therefore, credit risk is not a simple credit default dependency contagion but mixed with endogenous and exogenous factors: psychological and behavioral factors, network structure of credit risk holders, and the market regulator.

The method of complex network is paid more and more attention in the research of financial risk contagion in recent years. The financial risk contagion system is a self-organized social system [18]. Complex networks can visually describe the complex relationship between credit risk individuals and can effectively analyze endogenous and exogenous factors that affect risk transmission [19, 20]. Cimini and Serri [21] defined a systemic risk metric that estimated the potential amplification of losses in interbank markets accounting for both credit and liquidity contagion channels. This work indicated that losses reverberate among banks and eventually were amplified because of the complex structure of interbank exposures, which lead to the occurrence of the financial crisis. Bardoscia et al. [22] proposed a dynamical "microscopic" theory of instability by iterating balance sheet identities of individual banks and by assuming that transfer of shocks from borrowers to lenders. Allen and Gale [23] pioneered a study of risk contagion in the interbank market. They believed that the transmission of financial risks mainly depended on the internal relations of the financial system, such as the structure of lending relationship. This work indicated that sparse networks were more likely to infect risks, and the reason was that the tight network dispersed the impact of single bank failures on the overall system, which was similar to a complex social system [24]. But the opposite view was that the tight network reduced the risk of a single bank but increased the correlation between banks, thereby increasing the risk of contagion [25, 26]. The work [27] formalized an extension of a financial network model originally proposed by Nier et al. [25]. Acemoglu's work [28] showed that the network structure is not a monotonic linear relationship with contagion effects. When the negative impact was less than a certain threshold, the tight network was more stable; while the negative impact was greater than a particular threshold, the weak link network was more stable. Upper [29] summarized the simulation methods of the spread of network risk in the interbank market, discussed the assumptions and applications of various simulation methods, and pointed out that infectious default could not be completely eliminated. Gai and Kapadia [30] pointed out that the high connectivity of the financial network can reduce the probability of infection but also increase the risk of infection when the problem occurs. Li's work [31] indicated that the increased connectivity between banks reduced contagion effects, but will lead to liquidity problems, causing the risk infection. Heise and Kühn [32] studied dynamic risk contagion in the financial network and pointed out that the derivative securities risk exposure was an additional channel of contagion, which could reduce losses but did not rule out very large tail risks, and that risk contagion and loss may be increased in stressful situations. Filiz et al. [33] used algebraic geometry technique and maximum likelihood estimation method to study the problem of bank related default in simple graphs. Mastromatteo et al. [34] used the information transfer method and the maximum entropy theory to study the systemic risk of financial network structure, which highlighted the sparsity and heterogeneity of financial networks. Glasserman and Young [35] used the complex network theory to investigate the bankruptcy costs and mark-to-market losses resulting from credit quality deterioration or a loss of confidence. Bardoscia's work [36] indicated that the origin of instability resided in the presence of specific types of cyclical structures. Tonzer [37] analyzed whether international linkages in interbank markets affected the stability of interconnected banking systems. Li and Sui [38] investigated contagion risk in an endogenous financial network. Deng's work [39] investigated how systemic risk was affected by the structure of the banking system.

There is something in common between the financial system and the ecosystem. The contagion of financial risks is very similar to the spread of epidemics [40]. In recent years, epidemic models have been introduced into the field of economics and finance to study the diffusion effects of economic and financial risks [41]. Garas et al. [42] introduced the epidemic contagion mechanism into the actual financial network model. This work used the SIR epidemic model to simulate the contagion of the crisis in the global economic network combining with ecology, epidemiology, and complex network theory. Haldane [43] studied the relationship among network complexity, diversity, and financial vulnerability and explained the reasons for the vulnerability of the network structure. Chen and He [44] constructed a network model of credit risk contagion with related factors of credit principal behavior and revealed some relations among credit subjects in social networks. This work also studied the risk attitude of credit subject and the ability to resist credit risk. All above works show the advantages of complex network theory in the application of risk contagion. However, there are still some points needed to be improved: (i) Above models basically analyzed the characteristics of network risk contagion from a macro perspective, mainly analyzed the

proportion of individuals infected; however, there is no analysis of how individuals are infected in the network; (ii) The connection relation of network nodes is not well stated, and the relation among network nodes in most studies is symmetrical; (iii) In the above models, individuals are infected at two levels, that is, they are either completely infected or not infected, while in real financial networks, individuals are infected to varying degrees. Toivanen [45] used an epidemiologic SIR model to model the spreading of the contagion in the interbank network and analyzed the importance of individual bank-specific factors on financial stability. Brandi and Clemente [46] developed an Exposed-Distressed-Bankrupted model based on SIR model for the dynamics of liquidity shocked reverberation between banks. The above two works effectively analyzed the process of individual credit risk contagion from the perspective of liquidity, and banks were shown to be in three discrete states: exposed, distressed, and bankrupted. Moreover, the asymmetric risk contagion probability was established based on loan correlation.

Based on the above analysis, based on the existing correlation theory and the complex network theory, this paper establishes an asymmetric information association model. Considering micro behavior of investors, such as the risk attitude, the ability to resist risks, and the monitoring behavior of financial market supervisors, this paper studies the contagion behavior of individuals and its evolution mechanism. In this paper, the association between risk holders is asymmetric, which can analyze the evolution process of individual risk contagion. And the degree of individual infection is ranged from 0 to 1 instead of two levels: completely infected or not infected. Compared with the work of Toivanen and Brandi and Clemente [45, 46], the proposed model considers more factors and uses different infectious model, the definition of interbank relationship function is different, and banks are infected to varying degrees.

2. Credit Risk Contagion Model

In the financial market, the contagion of credit risk is a complex process related to social psychology, economic behavior, and information communication. In this process, the credit risk holder propagates the risk to other individuals through information association or interest association. Risk contagion is a game process of various factors. The strength of financial market supervision, individual ability to resist risks, and individual attitude to risk plays an important role in the process of risk transmission.

2.1. The Assumptions and Notation. In this paper, we assume that the network structure of credit risk propagation in the financial market remains unchanged. In the financial market, the probability of the individual being infected by the credit risk is related to four factors, such as the relationship among the credit risk holders, the risk attitude, the ability to resist risks, and the monitoring strength of the financial market regulators. At the same time, we assume that the number of individuals in the network is N, which is limited. All parameters used in this paper are defined as follows.

(i) λ is the average contagion rate of credit risk in the financial network, and $\lambda \in [0, 1]$.

(ii) u is the monitoring strength of the financial market regulators, and $u \in [0, \infty)$.

(iii) ζ is the effect strength of credit event, and $\zeta > 0$.

(iv) $r_i(\zeta), i = 1, 2, \ldots, N$, are individual attitudes and emotions to credit risk contagion, which can characterize the impact of credit events on individual behavior in financial markets. And $r_i(\eta) \geq 0$, $r_i'(\zeta) > 0$, and $r_i''(\zeta) > 0$, which indicate that the influence of credit events has an increasing marginal impact on individual risk aversion.

(v) $\theta_i, i = 1, 2, \ldots, N$, are the ability or resilience of individuals to resist credit risk contagion in financial markets, and $\theta_i \in [1, \infty)$.

(vi) $L_{ij}, i, j = 1, 2, \ldots, N$ are the information association between individuals with market risk holders, and $L_{ij} \in [0, 1]$. In the actual risk propagation network, the relation between individuals is bidirectional and asymmetrical. Thus, L is an asymmetric matrix. In fact, in this work, L_{ij} can be considered as bilateral exposures in a real financial network. This article focuses on the point of physical dynamics, so the expression "information association" is used. The lending relationship between financial institutions can be regarded as information association, that is to say, L_{ij} is the liability matrix. In real financial networks, L_{ij} can be obtained through maximum entropy [29], exponential random graphs [47, 48], or minimum density [49] based on the balance sheet of financial institutions.

(vii) $f_i(t), i = 1, 2, \ldots, N$, is the degree to which the credit risk is transmitted, and $f_i(t) \in [0, 1]$. Previous studies had only two states: infected or not infected, ignoring the degree of infection. In this paper, the degree of infection is taken into consideration.

(viii) η is the speed that individuals restore to the health status after being infected credit risk.

2.2. The Credit Risk Contagion Model. The contagion mechanism of credit risk in the financial system is similar to the physical phenomenon of network flow. In financial markets, individuals who are strongly associated with individuals who have been infected by credit risk are more likely to be infected. In this work, the average intensity of infection for individual i by other infected individuals is defined as

$$\Theta_i(t) = \frac{\sum_j L_{ij} f_j(t)}{\sum_j L_{ij}}. \quad (1)$$

From the (1), the intensity of contagion $\Theta_i(t)$ monotonically increases with $f_j(t)$. For a fixed structure risk contagion network, the contagion process of credit risk can be regarded

as a Markov process. For any individual i, the degree of infection satisfies the differential equation as follows:

$$\frac{df_i(t)}{dt} = \frac{g\left(\sum_j L_{ij}\right)\lambda r_i(\zeta)[1 - f_i(t)]}{u\theta_i}\Theta_i(t) - \eta f_i(t), \quad (2)$$

where $g(x)$ is the monotonically increasing convex function. In this work, $g(x) = (x/1 + x) \in [0, 1)$ is used. The first item in the right of (2) indicates that the intensity increases in which individuals are infected by infected individuals, and the second item is the recovery of individuals who are infected by credit risk. This work models credit risk contagion drawing on virus infection model. On the one hand, individuals with a large degree are easy to be infected by credit risks. On the other hand, individuals who are highly associated with infected individuals are also susceptible to infection. Previous works [25, 27] indicated that this credit risk contagion mechanism in the financial system is similar to the physical phenomenon of network flow. $g(\sum_j L_{ij})\Theta_i(t)$ represents the effect of other related financial institutions to node i. $g(\sum_j L_{ij})$ reflects the connection strength of node i and other nodes, and the greater the association with other nodes, the easier the node i will be infected. $\Theta_i(t)$ is the average infection degree of the nodes connected to node i. The bigger $\Theta_i(t)$ is, the more likely the node i is to be infected. λ is the average contagion rate of credit risk in the financial network, and the bigger λ is, the greater the node i will be infected. $r_i(\zeta)$ represents individuals' attitudes and emotions towards credit risk contagion, depicting the impact of credit event influence on individual behavior in financial markets. The bigger ζ, the bigger $r_i(\zeta)$, the greater the credit risk will affect the individual, which makes the individual more susceptible to be infected. u is the strength of market regulation, the stronger the regulation is, the more stable the financial market is, and the lower the degree of individual credit risk infection. θ_i is the ability or resilience of individual to resist credit risk contagion. In the real financial system, which can be regarded as the fundraising capability and management capability of financial institutions in times of crisis. When $(df_i(t)/dt) = 0$, the contagion intensity of node i in the equilibrium state of credit risk contagion system can be obtained as

$$f_i = \frac{g\left(\sum_j L_{ij}\right)(\alpha(r_i(\zeta)/u\theta_i)\Theta_i)}{1 + g\left(\sum_j L_{ij}\right)(\alpha(r_i(\zeta)/u\theta_i)\Theta_i)}, \quad (3)$$

where $\alpha = \lambda/\eta$ is risk transfer rate of credit risk contagion. From (3), it can be obtained that $(\partial f_i/\partial r_i(\zeta)) > 0$, $(\partial^2 f_i/\partial (r_i(\zeta))^2) < 0$, $\partial f_i/\partial \zeta > 0$, $\partial^2 f_i/\partial \zeta^2 < 0$, $\partial f_i/\partial u < 0$, $\partial^2 f_i/\partial u^2 > 0$, $\partial f_i/\partial \theta_i < 0$, and $\partial^2 f_i/\partial \theta_i^2 > 0$. Obviously, the contagion intensity of credit risk is a monotonic increasing convex function of individual risk attitude and the influence of credit events, and the contagion intensity of credit risk is a monotonically decreasing concave function of financial market supervision intensity and individual risk resisting ability. Thus, risk aversion can increase the infection and impact of credit risk, and individual risk resistance and financial market regulation will reduce the contagion of credit risk. Then, we plug (3) into (1) and can get an autonomous equation:

$$\begin{bmatrix} \Theta_1 \\ \Theta_2 \\ \vdots \\ \Theta_N \end{bmatrix} = \begin{bmatrix} 0 & \frac{L_{12}}{\sum_j L_{1j}} & \cdots & \frac{L_{1N}}{\sum_j L_{1j}} \\ \frac{L_{21}}{\sum_j L_{2j}} & 0 & \cdots & \frac{L_{2N}}{\sum_j L_{2j}} \\ \vdots & \vdots & \cdots & \vdots \\ \frac{L_{N1}}{\sum_j L_{Nj}} & \frac{L_{N2}}{\sum_j L_{Nj}} & \cdots & 0 \end{bmatrix} \times \begin{bmatrix} \frac{T_1\Theta_1}{1 + T_1\Theta_1} \\ \frac{T_2\Theta_2}{1 + T_2\Theta_2} \\ \vdots \\ \frac{T_N\Theta_N}{1 + T_N\Theta_N} \end{bmatrix}, \quad (4)$$

where $T_j = g(\sum_j L_{ij})\alpha((r_j(\zeta))/u\theta_j)$. Equation (4) describes the influence strength of infected nodes on i node when the credit risk contagion system reaches equilibrium. This equation also describes the conditions and the factors that need to be satisfied when the credit risk contagion system reaches equilibrium. Obviously, $\Theta_i = 0, i = 1, 2, \ldots, N$, is the trivial solution of (4). The trivial solution $\Theta_i = 0, i = 1, 2, \ldots, N$, indicates that there is no risk contagion in the network. However, nontrivial solutions are not the concern of contagion, and nonzero nontrivial solutions $\Theta_i \neq 0$ are important for risk contagion networks.

Theorem 1. *In the incomplete market, when the credit risk system is in equilibrium status, there is only a unique equilibrium $f_i > 0$ for $i = 1, 2, \ldots, N$ at most in the credit risk contagion system.*

Proof. Let

$$F_1(\Theta_i) = \Theta_i, \quad (5)$$

$$F_2(\Theta_i) = \frac{\sum_j L_{ij}\left(T_j\Theta_j(\Theta_i)/1 + T_j\Theta_j(\Theta_i)\right)}{\sum_j L_{ij}}. \quad (6)$$

It obviously that the intersection point of (5) and (6) is the solution of (4). Solving first and two order derivatives of (6), we get

$$\frac{dF_2(\Theta_i)}{d\Theta_i} = \frac{\sum_j L_{ij}\left(d\Theta_j(\Theta_i)/d\Theta_i\right)\left(T_j/\left(1 + T_j{\Theta_j}^2\right)\right)}{\sum_j L_{ij}}, \quad (7)$$

$$\frac{d^2F_2(\Theta_i)}{d\Theta_i^2} = \frac{\sum_j L_{ij}\left(\left(\left(d^2\Theta_j/d\Theta_i^2\right)T_j\right)/1 + T_j{\Theta_j}^2\right) - 2L_{ij}\left(\left(d\Theta_j/d\Theta_i\right)^2 T_j^2/1 + T_j{\Theta_j}^3\right)}{\sum_j L_{ij}}. \quad (8)$$

From (4), it is easy to find that $d\Theta_j(\Theta_i)/d\Theta_i > 0$ and $d^2\Theta_j(\Theta_i)/d\Theta_i^2 < 0$. Thus, $dF_2(\Theta_i)/d\Theta_i > 0$, and $d^2F_2(\Theta_i)/d\Theta_i^2 < 0$. Equations (7) and (8) indicate that (6) is monotonic increasing convex function. Due to $\Theta_i = \sum_j L_{ij} f_j / \sum_j L_{ij} \le 1$, we get

$$F_2(\Theta_i) \le \frac{\sum_j L_{ij} g\left(\sum_j L_{ij}\right) \alpha(r_i(\zeta)/u\theta_i)/1 + g\left(\sum_j L_{ij}\right) \alpha(r_i(\zeta)/u\theta_i)}{\sum_j L_{ij} < \left(\sum_j L_{ij}/\sum_j L_{ij}\right)} = 1. \tag{9}$$

Equation (9) indicates that there are at most two fixed points of (4) in the interval $[0, 1]$ shown in Figure 1, in which $\Theta_i = 0$, when $\Theta_j = 0, j = 1, 2, \ldots, N$, is a fixed point. If (4) has nontrivial solutions $\Theta_i \neq 0$, the following conditions must be satisfied:

$$\left.\frac{dF_2(\Theta_i)}{d\Theta_i}\right|_{\Theta_i=0} > 1. \tag{10}$$

It can be obtained from (10) that

$$\alpha > \frac{1}{\sum_j \left(\left(L_{ij}L_{ji}\left(r_i(\zeta)r_j(\zeta)/u^2\theta_i\theta_j\right)\right)\left(g\left(L_j\right)g(L_i)\right)/L_iL_j\right)}, \tag{11}$$

where $L_i = \sum_j L_{ij}$. From the above, when (11) is satisfied, there is a unique equilibrium $\Theta_i > 0$. If there is a unique equilibrium $\Theta_i > 0$ for $i = 1, 2, \ldots, N$, then there is a unique equilibrium $f_i > 0$ for $i = 1, 2, \ldots, N$ in the credit risk contagion system. Theorem 1 is proved.

In the credit risk contagion network, the threshold of risk transfer rate is closely related to the node's afferent information. If the credit event has a greater impact on the individual behavior of the financial market, namely, the bigger $r_i(\zeta)$, then the smaller the threshold of risk transfer rate for the infection of node *i*. In addition, the threshold of risk transfer rate is directly related to the ability to resist the risk of infection and the regulation of financial markets.

Theorem 2. *If the average connection information $L^A_{\text{ave}}(i)$ ($L_{\text{ave}}(i) = \sum_j L_{ij}/k_i$, k_i is the degree of node i) of the network* A *is greater than the average connection information $L^B_{\text{ave}}(i)$ of the network B, at the same time, A and B have the same network structure, and other parameter's value is the same, then the infection intensity f_i^A of the network A is greater than the infection intensity f_i^B of the network B.*

Proof. We assume that if the average connection information $L^A_{\text{ave}}(i)$ of the network A is greater than the average connection information $L^B_{\text{ave}}(i)$ of the network B, there is $\Theta_i^A < \Theta_i^B$. Let

$$Q\left(L_{ij}\right) = L_{ij}\frac{g\left(\sum_j L_{ij}\right)\left(\alpha(r_i(\zeta)/u\theta_i)\Theta_i\right)}{\left(1 + g\left(\sum_j L_{ij}\right)\left(\alpha(r_i(\zeta)/u\theta_i)\Theta_i\right)\right)\sum_j L_{ij}}. \tag{12}$$

We defined $\alpha(r_i(\zeta)/u\theta_i)\Theta_i/((1 + g(\sum_j L_{ij})(\alpha((r_i(\zeta)/u\theta_i))\Theta_i))) = P_i$ and $\alpha(r_i(\zeta)/u\theta_i)\Theta_i = M_i$. It can be obtained that

$$\frac{dQ\left(L_{ij}\right)}{dL_{ij}} = \frac{P_i\left(\sum_j L_{ij} - L_{ij}\right)}{\left(\sum_j L_{ij}\right)^2} + \left(\frac{M_i L_{ij}}{\sum_j L_{ij}\left(1 + g\left(\sum_j L_{ij}\right)M_i\right)^2}\frac{dg\left(\sum_j L_{ij}\right)}{dL_{ij}}\right). \tag{13}$$

It obviously that $\sum_j L_{ij} - L_{ij} > 0$ and $dg(\sum_j L_{ij})/dL_{ij} > 0$. Thus, $dQ(L_{ij})/dL_{ij} > 0$, and $Q(L_{ij})$ is a monotonically increasing function. According to the stochastic dominance condition, if the average connection information of the network *A* is greater than the average connection information of the network *B*, then L_{ij}^A first-order stochastic dominates L_{ij}^B. Therefore, $\sum_j Q(L_{ij}^A) > \sum_j Q(L_{ij}^B)$ for any $\Theta_i^A = \Theta_i^B > 0$, that is, $(F_2(\Theta_i^A) = \Theta_i^A) > (F_2(\Theta_i^B) = \Theta_i^B)$. This result is contradictory, and the hypothesis is not tenable. Thus, if the average connection information $L^A_{\text{ave}}(i)$ of the network *A* is greater than the average connection information $L^B_{\text{ave}}(i)$ of the network *B*, there is $\Theta_i^A > \Theta_i^B$.

Equation (3) shows that f_i is a monotonically increasing function of Θ_i. Thus, if $\Theta_i^A > \Theta_i^B$, then $f_i^A > f_i^B$, namely, Theorem 2 is true.

From Theorem 2, we can see that if the network is more closely related, and the influence of credit risk contagion will be greater. The key reason is that the ability to interact with individuals is stronger with the stronger association of network. The individual's behavior convergence effect is stronger when credit risk occurs, the infection of credit risk is accelerated, and the influence of credit risk is increased.

3. Numerical Simulation Analysis

In order to further verify the effectiveness of the proposed model in characterizing the credit risk contagion, this paper simulates the model from different angles: (i) evolution process of risk contagion under different average degree $<k>$; (ii) evolution process of risk contagion under different average connection weights L_{ave} ($L_{\text{ave}} = \sum_{ij} L_{ij}/N < k >$); (iii) evolution process of risk contagion under different network structures; (iv) evolution process of risk contagion under

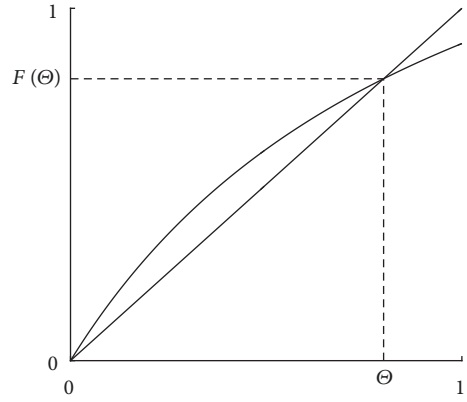

Figure 1: The equilibrium relationship graph of credit risk contagion system.

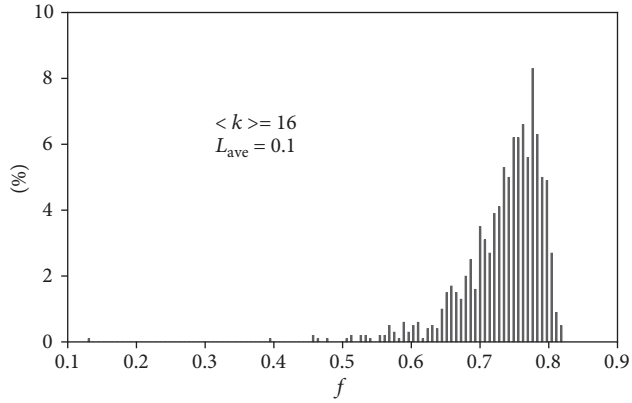

Figure 2: The distribution of the degree of individual infection in a random network with $<k> = 16$, $L_{ave} = 0.1$, and $r_i(\zeta)/u\theta_i = 1$.

different parameters' values of $r_i(\zeta)$, u, and θ_i. In this work, the parameters' values of λ and η are 0.9 and 0.1, respectively, and the risk transfer rate $\alpha = \lambda/\eta$ is 9. It can be assumed that only one random individual p is infected at the initial state, and the infection rate is $f_p = 0.5$.

In order to reflect effectively the evolution law of network under conditions of different value $<k>$ and L_{ave}, the value of $r_i(\zeta)/u\theta_i$ is firstly set as 1 firstly and the network structure is a random network. The method presented in this paper can reflect the degree of infection of individuals. Figure 2 shows the distribution of the degree of individual infection with $<k> = 16$ and $L_{ave} = 0.1$. From Figure 2, it is obvious that the degree of infection of individuals is not 0 or 1, and all the individuals are infected, but individuals are infected in different degrees, which is different from the existing models using complex network theory.

In a real financial network, the degree of individual infection is different. The model in this paper is more consistent with the real financial network. It is interesting that when all the other parameters are fixed, the results of network evolution are only related to the average degree of the network but not to the scale of the network. The network scale is defined as the total number of nodes in the network (N). Figure 3 shows that the average infected degree is same for

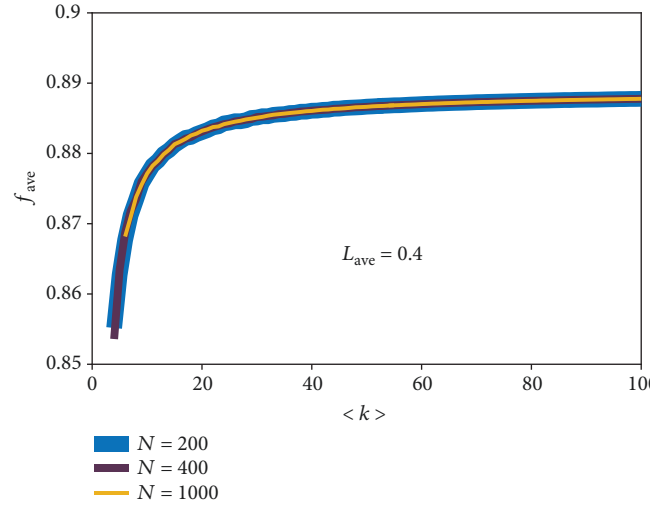

Figure 3: The average infected degree for different values of $<k>$ in different network.

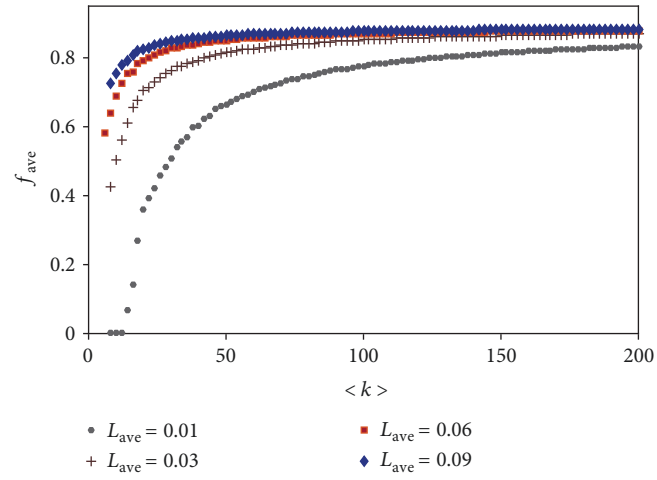

Figure 4: The evolution of the network varies with $<k>$ under different average connection weights L_{ave}.

different network scale with same $<k>$. In the following simulation, the scale of the network is set as 1000.

Figure 4 shows how the evolution of the network varies with $<k>$ under different average connection weights L_{ave}. Figure 4 indicates that the average infected degree f_{ave} increases with the increase of $<k>$. Under the same conditions, if the network is more intensive, then the level of credit risk contagion is higher. This is because the network is more intensive, the dissemination of information between individuals is more conducive, and the formation of psychological groups is easier.

Figure 4 also presents that the connection weight has an important influence on the evolution of the network. Figure 5 shows the evolution of credit risk contagion under two different average connection weights. The simulation results show that when the average connection weight is large, the distribution of individual infection is centralized, and the mean value of infected degree is large. This is because when the average connection weight increases, the ability of

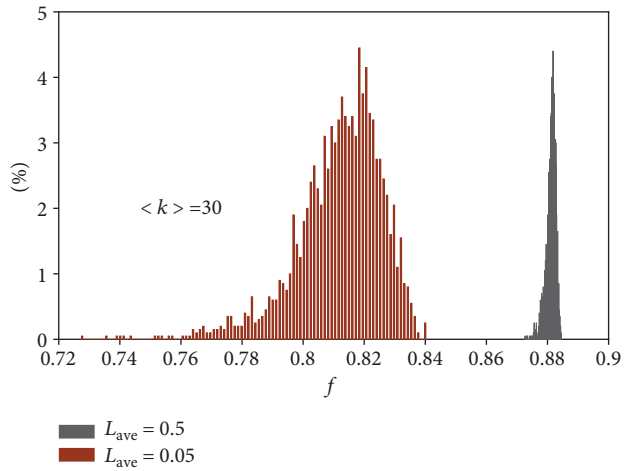

Figure 5: The evolution of credit risk contagion under two different average connection weights when $<k> = 30$.

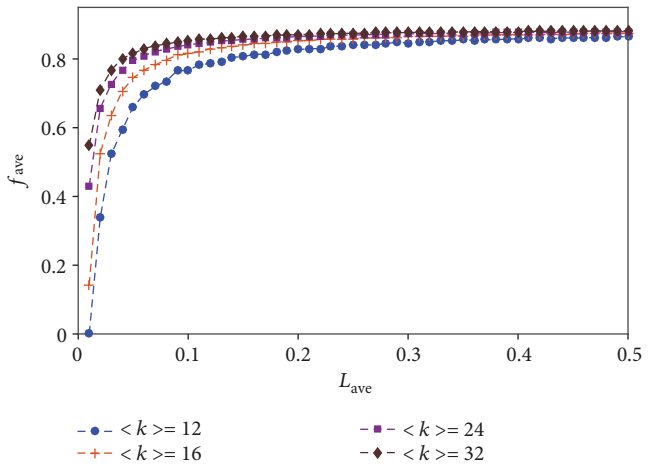

Figure 6: The evolution of the network varies with L_{ave} under different value of $<k>$.

risk contagion among individuals becomes stronger, and the degree of infection among individuals becomes greater. At the same time, the increase of average connection weight leads to the increase of convergence among infected individuals, which leads to a more concentrated distribution of infected degree.

Figure 6 shows how the evolution of the network varies with L_{ave} under different $<k>$. Figure 6 indicates that the average infected degree f_{ave} increases with the increase of L_{ave}, and as long as small information L_{ave} is associated, contagion can be large.

In order to explain more clearly the impact of the degree of information association between individuals on risk contagion, we set up the average amount of income information $L_{ave}(i) = \sum_j L_{ij}/k_i$ (k_i is the degree of node i) $(i = 1, 2, \ldots, N)$ for all individuals: 15% for $L_{ave}(i) = 0.1$, 15% for $L_{ave}(i) = 0.05$, 15% for $L_{ave}(i) = 0.025$, 15% for $L_{ave}(i) = 0.01$, and 40 % for $L_{ave}(i) = 1$. The simulation result is shown in Figure 7. As described in Figure 7, the degree of infection is also low for individuals with small average association levels.

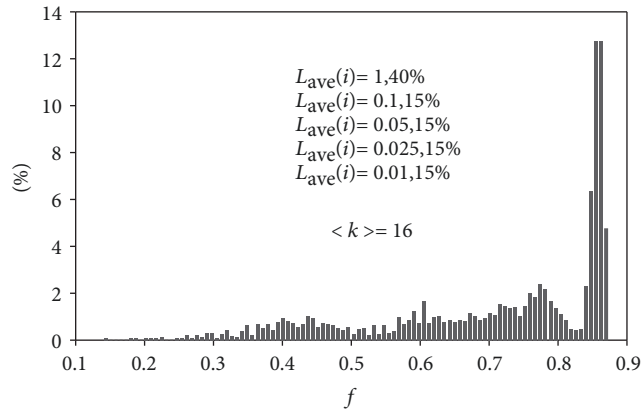

Figure 7: The infection distribution with different degrees of information association, when $<k> = 16$.

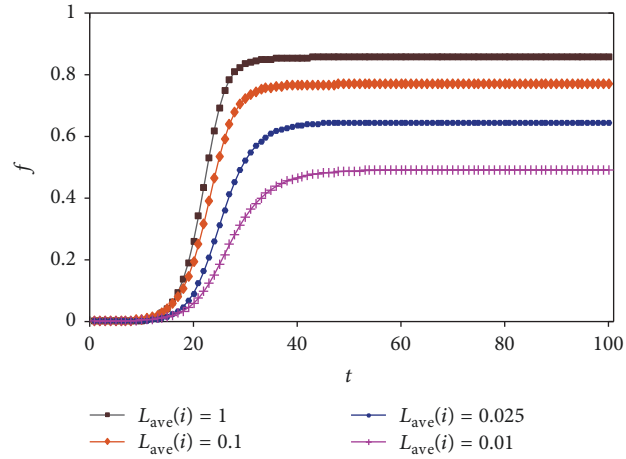

Figure 8: The evolution process of individuals with different average degree of association in simulation of Figure 7.

This is in line with the actual credit risk network. In the real credit risk network, if the average correlation degree of an individual is small, then the individual is less affected by the risk contagion. Figure 8 shows the evolution process of individuals with different average degree of association for the individual. The simulation indicates that the greater the average connection weight, the faster the individual is infected. However, the time to reach stability is consistent for all individuals.

In order to further depict the law of credit risk contagion and its evolution characteristics, according to the heterogeneity of the network, two other networks are selected to compare the simulation experiments: WS network (Watts-Strogatz network) and BA network (Barabasi network). Figure 9 shows the evolution of risk contagion for three different networks under the same parameters: $<k> = 16$ and $L_{ave} = 0.01$. In this simulation, the reconnection probability of WS network is 15%. The initial network node number of BA network is 22, the initial network is a random connection, and the number of nodes generated by each added node is 8. As shown in Figure 9, the distribution of individual infections in WS networks is minimal,

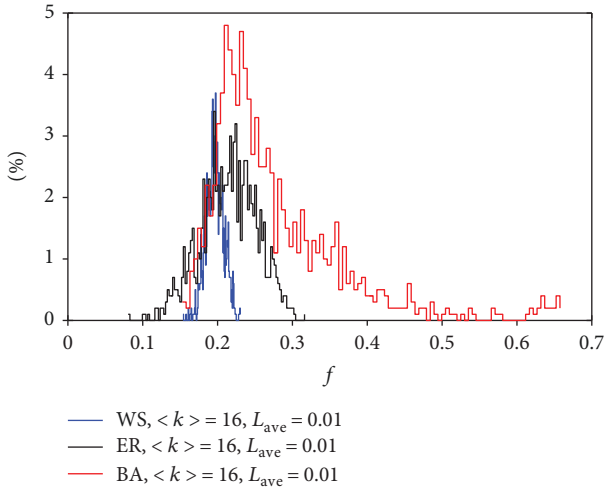

Figure 9: The evolution of risk contagion for three different networks with $<k> = 16$ and $L_{ave} = 0.01$.

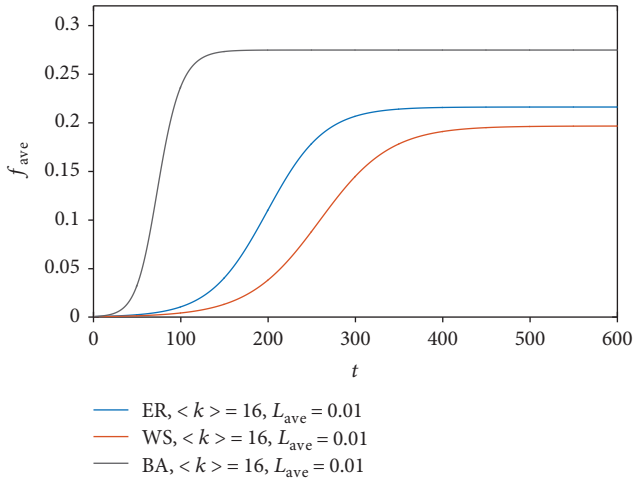

Figure 10: The variation of the average infection degree f_{ave} with time for three networks.

and the distribution of individual infections in BA networks is the most widely distributed.

Figure 10 shows the variation of the average infection degree f_{ave} with time for three networks. In the simulation, the average infection rates are 0.1962, 0.2136, and 0.2751 for WS network, random network, and BA network, respectively. It can be seen that the average risk of infection among individuals in the BA network is highest in three networks when the average connection weight is small, and that the nodes with high degree in the BA network are more highly infected. In the WS network, the risk individuals are infected to a small extent, and the difference between individuals is not large. Figure 10 also indicates that under the same conditions, the risk propagation speed of BA network is the fastest, and the risk propagation speed of WS network is the slowest. The simulation shows that if the network structure is more regular, the risk contagion speed is smaller, and the degree of contagion is lower. Relevant studies

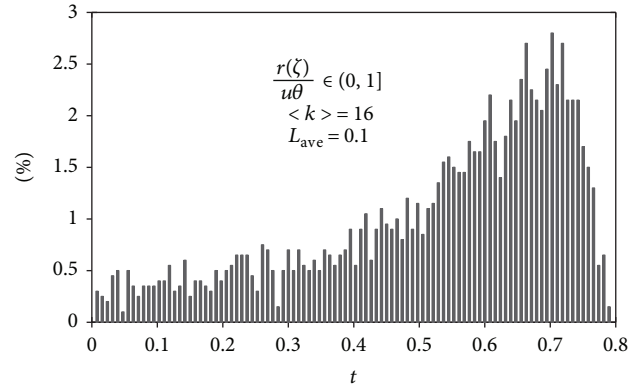

Figure 11: The infection distribution of individuals when y_i is randomly selected in $(0, 1]$.

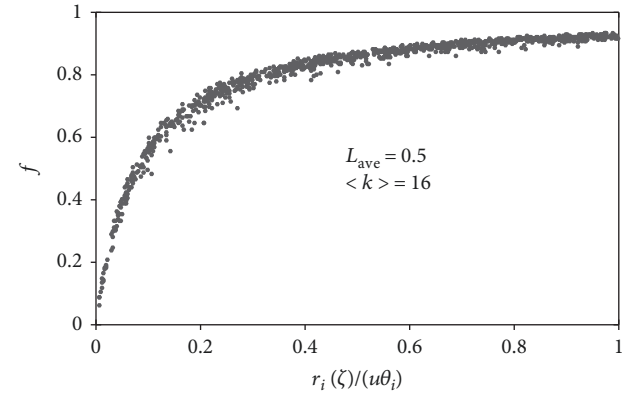

Figure 12: The relationship between the degree risk contagion and y_i.

shown that financial networks had significant scale-free network characteristics [50]. In scale-free networks, a randomly selected node tends to connect key nodes or nodes with large degree, so the node with large degree is easy to be infected, which is why the nodes with large degree are highly infectious in Figure 9. Then, the nodes with large degree are used as seed nodes to infect other nodes, which results in higher risk contagion and faster spread of credit risk than the homogeneous network.

Finally, we discuss the impact of parameters $r_i(\zeta)$, θ_i, and u on risk contagion. In this paper, individual risk attitudes $r_i(\zeta)$, individual ability to resist risks θ_i, and the regulatory strength of financial markets u constitute $y_i(r_i(\zeta), \theta_i, u) = r_i(\zeta)/\theta_i u$, which is a monotonically proportional increasing function of $r_i(\zeta)$, and is a monotonically proportional decreasing function of θ_i and u. Therefore, we study the impact of y_i on risk contagion, and we can get the influence of various parameters on risk contagion. Figure 11 shows the infection distribution of individuals when y_i is randomly selected in $(0, 1]$. From the simulation, compared with the simulation in Figure 2, we can see that because of the different risk attitude and risk resistance ability, the degree of risk contagion varies widely.

Figure 12 shows the relationship between the degree risk contagion and y_i. It can be seen that the degree of individual

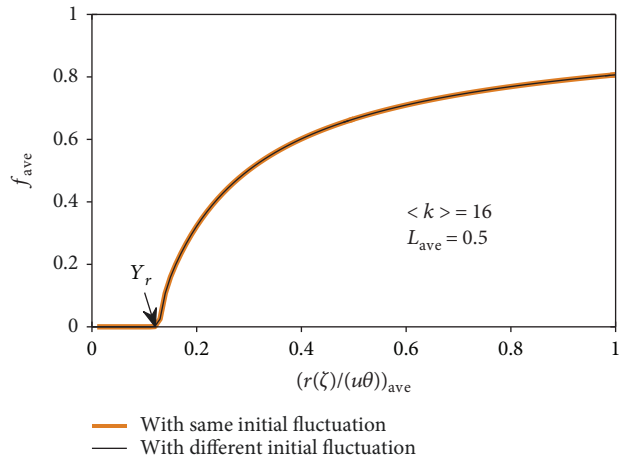

FIGURE 13: The average intensity of individual risk contagion with different average value y_{ave}.

risk contagion increases with the increase of y_i. Figure 13 shows the average intensity of individual risk contagion in the network under different average value y_{ave} ($y_{ave} = \sum_{i=1}^{N} y_i / N$) in two conditions: with same initial fluctuation, $f_p(0) = 0.5$ (p is a fixed value); with random fluctuation, $f_i(0)$ is the random number in $[0, 1]$ for $i = 1, 2, \ldots, N$. From the simulation results, we can see that the risk in the network is suppressed when the average value y_{ave} is less than the threshold value y_r. That is, the risk can be effectively controlled if the market regulation capability is strong enough or the individual's ability to resist risks is adequate. When the value of y_{ave} is larger than the threshold value y_r, the degree of the risk infection of the whole network is increased with the increase of y_{ave}. That is to say, if the individual risk attitude is stronger, then the individual is more likely to be infected by the risk. In addition, it can be seen from the simulation comparison that even if the initial values of the f_i are different, and the network converges to the same stable point with the same parameters, which is in accordance with the Theorem 1.

4. Conclusion and Discussion

In this paper, the theory of propagation dynamics in complex networks is introduced into the study of credit financial risk contagion. This paper focuses on asymmetric information association among individuals. Considering the individual risk attitude, individual risk resisting ability, and financial market supervisor's monitoring strength, a network model of credit risk contagion is established. Furthermore, this paper uses numerical simulation to study the influence and mechanism of these factors on credit risk contagion. Through theoretical deduction and numerical simulation, this paper gets a series of conclusions which has an important theoretical value and management significance. The main points are as follows: (i) In the risk network, individuals are infected to varying degrees, which is consistent with the characteristics of the real financial network; (ii) In an incomplete financial market, there exists only positive equilibrium point of credit risk contagion system, and the contagion intensity of credit risk is a monotonic increasing convex function of individual risk attitude and the influence of credit events, and the contagion intensity of credit risk is a monotonically decreasing concave function of financial market supervision intensity and individual risk resisting ability; (iii) The greater the amount of information the individual receives from each other, the greater the degree of risk contagion; (iv) The denser the network, the higher the average infection rate of individuals in the network; (v) The greater the average association information of the network, the higher the average infection rate of individuals in the network; (vi) The stronger the heterogeneity of the network, the faster the speed of credit contagion, and the greater the average level of credit contagion.

These conclusions have important theoretical value and practical significance for credit risk management practice. The network theory of financial risk contagion is a cross field of comprehensive finance, network theory, and system dynamics. The conclusions in this paper need to be further demonstrated in practice. This article is only a preliminary exploration of the infection of credit risk, and there are many points that can continue to be discussed. In a real financial network, the greater degree of the individual, the greater the transmission of information to other individuals, which has not been taken into account in this paper. In addition, the interbank market with overlapping portfolios will promote the spread of risk [51, 52]. Furthermore, the measurement of the quantity of information transfer between individuals can be analyzed by means of transfer entropy and mutual information. In addition, the real credit risk contagion network may be of community structures and multiplex network, which can be empirically studied on the basis of the proposed model in the future.

Data Availability

The data used to support the findings of this study are available from the corresponding author upon request.

Conflicts of Interest

No potential conflict of interest was reported by the authors.

Acknowledgments

This work is supported in part by the National Natural Science Foundation of PR China (71371046) and the Natural Science Foundation of Jiangsu Province (BK20161533).

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