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*Research Article*

# Nonlinear Dynamics Characteristic of Risk Contagion in Financial Market Based on Agent Modeling and Complex Network

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Risk contagion is becoming a research hotspot in the field of econophysics with the rise of interdisciplinary studies and gains more and more attention from theoretical circles and practical departments. This paper proposes a new research frame to discuss the microscopic mechanism of risk contagion based on agent modeling technology and complex network theory and reveals nonlinear dynamics characteristic of risk contagion from the perspective of market participants in financial market. Based on the proposed SICM model, financial risk can transmit from susceptible agents to infected agents, to contagious agents, or to immune agents. With the increases of contagious probabilities, the simulation experiments show that (1) the amount of susceptible agents continuously decreases; (2) the amount of infected agents increases first and then decreases; (3) the amount of contagious agents increases first and then decreases with a lower speed, relative to the amount of infected agents; and (4) the amount of immune agents continuously increases. The major contribution of this paper is a new method for studying nonlinear dynamics characteristic of risk contagion, which can be used as a theoretic basis for further researches on the behavioral features of microcosmic subject and the inner mechanisms of risk contagion.

## 1. Introduction

With the rapid development of society, the financial industry has gotten more attention and support from governments. Because the financial development can obviously promote the economy development in theory and practice, governments around the world have always committed to developing domestic finances, especially in financial markets and financial institutions. Many world economic centers can lead the development direction of globe economy because they also serve as world financial centers, such as New York, London, and Hong Kong. But due to the influence of financial risks, the development of finance has not been smooth. Many famous financial crises are still remembered by market participants, for example, Asian financial crisis in 1997, American subprime crisis in 2008, and European debt crisis in 2009. These serious international financial crises brought many destructive impacts on the world economy in the past. In essence, the outbreaks of financial crises are always the results of risk transmission and risk accumulation in financial markets. Therefore, the research of risk contagion not only has significant theoretical values to reveal the interaction mechanisms of participants in financial markets, but also great practical values for the prevention of financial risks for governments.

In the existing literature, a part of researchers mainly focus on the fluctuation of asset prices, which causes the transmission of financial risk from one market to another. Although these researches often adopt empirical analysis method, the behavioral features of microcosmic subject are not well revealed. And another part of researchers depend on complex network theory and agent modeling technology to analyze risk contagion in financial markets. These findings

are more focused on the design of network structure and the construction of artificial financial market.

The contributions of this paper are twofold. First, we propose a new model of risk contagion based on agent modeling and complex network, which can explain the nonlinear dynamics characteristics of risk contagion from the aspect of microcosmic subject in financial market, compared with the existing literature. In simulation experiments, we discuss the interaction process of investor behavior and the dynamic evolution mechanism of risk contagion. The second contribution of this paper is a theoretic expansion of risk contagion. The contagious model built in this paper is a basic theory model, which can explain risk contagion caused by interaction behaviors of market participants. For different types of financial markets, this theoretic model can be used as a basis for discussing the investor behavior and the resulting risk contagion. The remainder of this paper is organized as follows. Section 2 gives a brief review about the existing literature of risk contagion in financial market. Subsequently, Section 3 presents the theoretical model of risk contagion. Next, Section 4 conducts a series of simulation experiments that discusses the evolution characteristics of risk contagion. Finally, Section 5 concludes the work and points out the direction for future researches.

## 2. Literature Review

The underlying mechanism of risk contagion can hardly be explained from traditional finance theories because risk contagion shows some characteristics, such as global influences, systematic influences, and interactive influences [1]. Global influences indicate that financial risk may well transmit to neighbor region from original region and ultimately to spread from one country to another. And systematic influences emphasize that risk contagion probably causes multiple crises in many financial markets, such as debt crisis, liquidity crisis, and credit crisis. At last, interactive influences involve two parts: the interaction between commodity market and financial market and the interaction among different participants. For the researches on risk contagion in financial markets based on agent modeling and complex network, the literature reviews are shown from the following three aspects.

*2.1. The Researches of Risk Contagion in Financial Markets.* The early scholars often explained risk contagion in financial markets from the perspective of international trades, economic fundamentals, and international capital flows [2–8]. In international trades, a country could increase its international competitiveness of goods and services through a devaluation. And the successive devaluations of different currencies caused the international transmission of financial risks in international scenes [9, 10]. For example, it was easy to be found that financial risks transmitted among the countries having closer trade ties, after analyzing three decades of panel data coming from industrialized nations [11]. And the worse economic fundamentals easily led to risk contagion from economic fields to financial fields [12]. When a financial crisis broke out in a country, the economic fundamentals of neighbor countries began deteriorating generally. The process of risk contagion involved three effects: monsoonal effects, spillovers effects, and pure contagion effects [13]. However, international capital inflows could not always promote economic growth. When the domestic economy was overheating, international capital inflows would increase the appreciation pressure of domestic currency and then realize risk contagion among different countries [14]. Besides, more scholars increasingly focused on the behaviors of market participants to explain the inner mechanism of risk contagion. They gave up the hypothesis of rational economic man and insisted that the investor had the bounded rationality [15–18]. In the framework of bounded rationality, investors were mainly divided into two kinds: rational traders and noise traders [19–22]. Essentially, noise was a signal caused by the shortcomings of market mechanisms or the decision-making mistakes of market subjects [23]. A small-scale noise trader could increase market liquidity, but a large-scale noise trader was likely to cause excessive price fluctuations in financial markets [24–26]. Moreover, rational traders and noise traders were not unchangeable. And in some cases, rational traders likely turned into noise traders [27]. On the basis of the hypothesis of bounded rationality, the more researches explained risk contagion in financial markets from the perspective of investor behavior bias, such as equity premium puzzle [28–30], idiosyncratic volatility puzzle [31–33], closed-end fund puzzle [34–36], and dividend puzzle [37–39]. In addition, the cognitive bias and irrational behavior of investors were often associated with overconfidence theory. But the studies found that the irrational behaviors of overconfident investors did not generate excess returns in the long term [40, 41]. Furthermore, the behaviors of institutional investors could make a dramatic impact on risk contagion. As institutional investors had larger amounts of money and professional investment strategies, the herd behaviors of institutional investors were easily observed in financial markets [42, 43].

*2.2. The Application of Agent Modeling in Financial Markets.* The study of risk contagion was always based on efficient market hypothesis and rational expectation theory since long times ago. But in reality, the investor showed the obvious heterogeneity due to the differences of knowledge structure, analytical ability, and risk preference. And investor behavior became more complicated under the influence of interaction effects. It was difficult to reveal the dynamic characteristics of risk contagion for traditional analysis methods, such as theoretical analysis and empirical analysis [44–46]. However, agent modeling technology could solve this dilemma very well [47]. For example, a stochastic multiagent model was built to analyze risk contagion based on investors' mood fluctuations, and the results indicated that the return series showed the characteristics of heavy-tail distributions [48]. A key reason for heavy-tail distributions in financial markets was sheep-flock effect. After analyzing the relationship of peek distribution of return series, market transaction order, and investor imitation, the findings showed that the group decision was a common strategy among agents and the return series followed a power-law distribution [49, 50]. Chen and

Yeh modified the learning mechanism of SFI-ASM model and constructed an agent-based model of "school" in order to analyze the interaction mechanism of agents [51]. Based on [49], Iori paid more attention to individual decisions and found that agents tended to adjust their investment strategies according to an information set received at every time point [52]. Pascual et al. discussed the risk contagion and the heterogeneity of agents after adding psychological factors and emotive factors into artificial stock market model [53]. In addition, artificial stock market model was a common method to study risk contagion caused by the interactive behaviors of agents. For example, agents could be divided into three types: fundamental analysts, optimistic analysts, and pessimistic analysts, which transformed into any of types according to the changes of returns rates in financial markets [54]. Bertella constructed an artificial stock market including fundamental investors and technical investors and found that the volatility of stock price increased with the increasing heterogeneity of agents [55]. Besides, the high-frequency transaction data were usually used to build an artificial stock market in order to reveal the characteristics of risk contagion. By studying the relation between price restraint and risk contagion, the simulation experiment showed that both price ceiling and price floor led to the spillover effect of financial risks [56]. Based on SFI-ASM model, Liu and Han constructed a multiagent stock market model and found that the volatility of stock returns was decreasing with the increasing of simulation period [57].

*2.3. The Application of Complex Network in Financial Markets.* Complex network theory originated from the study on Seven Bridges of Königsberg [58]. Along with the increasing development of complex network theory, more and more scholars tried to apply this theory to financial markets and studied the inner mechanism of risk contagion [59–61]. Taking stock market as an example, a stock can be abstracted as an agent in a complex network, and the correlation coefficient between two stocks can be seen as an edge in this network. Thus, both agents and edges together form a complex network. Mantegna built a complex network model through the minimum spanning tree algorithm and analyzed risk contagion in stock market [62]. On the basis of [62], the results from Bonanno et al. indicated that the network structure of stock market followed a power-law distribution after analyzing the difference between the minimum spanning tree model and the real stock market model [63]. On a scale-free network, the network structure was changing with the adjustment of threshold value [64]. In addition, the closing price in financial market was often used for studying risk contagion. Emmertstreib and Dehmer used the daily closing price data to investigate the formation of network structure and the dynamic evolutionary process of financial network [65]. Some researchers found that financial market probably had the small-world feature. For example, after analyzing the correlation coefficients between different stocks in Chinese stock market, Li and Li found the obvious small-world feature in Chinese stock market and discussed the interaction effect among different stocks and the risk contagion caused by stock price variation [66]. The similar conclusions could be found from [67], which found the small-world feature of Chinese stock market by building a complex network model based on multiagent modeling. Besides, the changing investor attention could cause the fluctuation of asset price and the intensity of herd behavior in a small-world network [68]. Under the background of global financial crisis, financial risk was easier to transmit from one investor to another. By using stock prices in the Korean stock, Nobi discussed the characteristics of risk contagion, during and after 2008 global financial crisis, based on the hierarchical network and the minimum spanning tree algorithm [69]. Network linkage played a major role in the process of formatting network structure. The dynamic conditional correlations method could be used to study network linkage effects of financial market, especially in stock market. By using the GMM model, Qiao discussed the influences of inner nodes in different positions on stock returns and found that financial risks were easier to transmit from the central area to the marginal area of network [70].

## 3. Theoretical Model

The researches on transmission dynamics are always the focuses and forefront projects in the academic world. By means of complex network theory, many scholars explain microscopic mechanisms and dynamic characteristics of some practical issues in society. For example, complex network can be used to reveal the transmission mechanism of computer viruses [70–72], the spread process of epidemics [73, 74], and the diffusion intensity of rumor [72, 75]. For the researches on dynamics of complex network, many research achievements are based on the traditional model of virus spread, such as epidemic models. This part introduces the classic infection models at first and then constructs the new risk contagious model in financial market based on agent modeling and complex network.

*3.1. The Basis Theoretical Model.* In order to reveal the transmission mechanism of complex networks, some theoretical models are gradually developed on the basis of the theory of epidemics, such as SI model, SIS model, SIRS model, SEIR model, and so on. In above models, every agent is probably in a different state in different time points. The main states can be classified into susceptible states, infected states, exposed states, resistant states, etc. Concretely speaking, susceptible states indicate that agents are not infected yet in a model, but they are probably infected by viruses in subsequently time. Infected states show that agents are infected by viruses, and they tend to spread the viruses to others. Exposed states emphasize that agents may keep latent states instead of becoming the infected states, after contacting with viruses. Resistant states contain two types of situations: (1) agents gain the immunity to viruses owing to vaccine injection or rehabilitation and (2) agents are removed from a model owing to the death. No matter which type of situations, agents are no longer able to continue to spread the viruses.

In SI model, all agents are assumed not to be immune to viruses. Thus, they are in susceptible states or infected

states. Without immunity, an agent is likely to immediately transform into infected state from susceptible state, after virus exposure. Considering all agents are impossible to be infected at the same time in the practical studies, the SI model is only applicable for the early stages of virus outbreak. Suppose that the total of agents is invariant, written as N. The percentage of agents in susceptible states is s(t), and the percentage of agents in infected states is i(t). The contagious probability of agents from susceptible states to infected states is assumed as $\alpha$. That means the agents in infected states will increase $\alpha \cdot s(t) \cdot Ni(t)$. The relationship of above variables is shown in expression (1), which can be further simplified as expression (2):

$$\frac{dNi(t)}{dt} = \alpha \cdot s(t) \cdot Ni(t) \tag{1}$$

$$\frac{di(t)}{dt} = \alpha \cdot s(t) \cdot i(t) \tag{2}$$

Although SI model reflects the basic process of virus spread, it does not take into account the immunity to viruses for an agent. In SIS model, an infected agent is probably cured under a certain probability and returns to the susceptible state from infected state. In addition, an agent who is cured after catching a viral influenza is likely to be infected again in daily life. Let us suppose that $\beta$ is the transformation probability of agents from cured states to susceptible states. In comparison with SI model, the agents in infected states decrease $\beta \cdot Ni(t)$ in every moment in SIS model. So, the amount of agents in infected states can be expressed as follows:

$$\frac{dNi(t)}{dt} = \alpha \cdot s(t) \cdot Ni(t) - \beta \cdot Ni(t) \tag{3}$$

Obviously, expression (3) can be simplified and written as

$$\frac{di(t)}{dt} = \alpha \cdot i(t)\,[1 - i(t)] - \beta i(t) \tag{4}$$

Compared with SIS model, SIR model considers the problem of virus immune. A cured agent in infected state will gain immunity to virus and stop spreading virus to other agents. Let us suppose that $\gamma$ is the transformation probability of agents from infected states to resistant states, and $r(t)$ is the percentage of agents in resistant states. The dynamic characteristics of SIR model can be described as follows:

$$\begin{aligned} \frac{ds(t)}{dt} &= -\alpha i(t)\, s(t) \\ \frac{di(t)}{dt} &= \alpha i(t)\, s(t) - \gamma i(t) \\ \frac{dr(t)}{dt} &= \gamma i(t) \end{aligned} \tag{5}$$

However, agents in resistant states may lose their immunity with a probability and enter into susceptible states again. This probability is assumed as $p$ in SIRS model, where the agents in susceptible states increase $pr(t)N$ relative to SIR model. Conversely, the agents in resistant states decrease $pr(t)N$. So, it is not difficult to find that SIRS model is an extension of SIR model. The dynamic characteristics of SIRS model can be described as follows:

$$\begin{aligned} \frac{ds(t)}{dt} &= -\alpha i(t)\, s(t) + pr(t) \\ \frac{di(t)}{dt} &= \alpha i(t)\, s(t) - \gamma i(t) \\ \frac{dr(t)}{dt} &= \gamma i(t) - pr(t) \end{aligned} \tag{6}$$

In addition, agents in susceptible states are probably not immediately infected after contacting with viruses. In other words, agents may enter into exposed states. In SEIR model, there are two probability parameters, $\varepsilon$ and $\theta$. To be specific, agents enter into exposed states from susceptible states with probability $\varepsilon$, and then they enter into infected states from exposed states with probability $\theta$. Finally, they enter into resistant states with probability $\gamma$. Similarly, we assume the percentage of agents in exposed states is $e(t)$. The dynamic characteristics of SEIR model can be described as follows:

$$\begin{aligned} \frac{ds(t)}{dt} &= -\varepsilon i(t)\, s(t) \\ \frac{de(t)}{dt} &= \varepsilon i(t)\, s(t) - \theta e(t) \\ \frac{di(t)}{dt} &= \theta i(t)\, s(t) - \gamma i(t) \\ \frac{dr(t)}{dt} &= \gamma i(t) \end{aligned} \tag{7}$$

*3.2. The Risk Contagious Model.* Based on the above models, this paper assumes there are four kinds of agents in financial market: susceptible agents, infected agents, contagious agents, and immune agents. These four kinds of agents are abbreviated to S, I, C, and M. In a complex network structure, S, I, C, and M can also be seen as network nodes. S, as a susceptible agent, who is likely to receive some grapevine in market, has not yet received it. And I, as an infected agent, has received a grapevine, but has not made a decision about whether or not to spread this grapevine. If the final decision is spread, infected agent (I) will transform into contagious agent (C). If not, infected agent (I) will transform into immune agent (M). According to the relationship of different network nodes, the risk contagious model is constructed and written as SICM model. The process of risk contagion is shown in Figure 1.

In Figure 1, an agent is presumed to have an unofficial grapevine at the initial moment. This grapevine can be seen as a risk event in financial market. Other agents around this agent have an opportunity to get this grapevine and become susceptible agents. Then susceptible agents will transform into infected agents with probability $x_1$. If infected agents accept this grapevine, they will become contagious agents with probability $x_2$. And if not, they will become immune agents with probability $x_3$. After a while, contagious agents will lose interest in this grapevine and decide not to continue spreading it to other agents. At this time, contagious agents probably become immune agents with probability $x_4$.

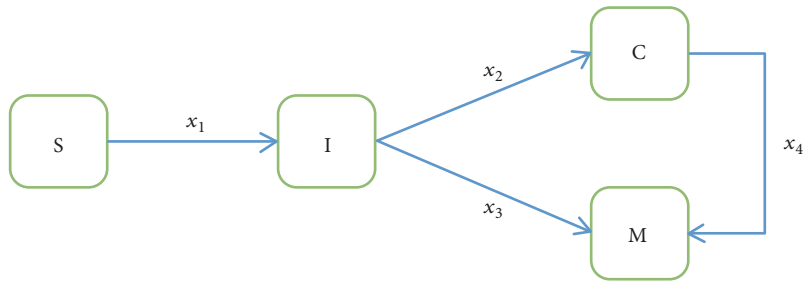


Figure 1: The process of risk contagion based on SICM model.

In our model, the sum total of market participants is assumed to be invariable at any time and written as N. The amount of each type of agents is expressed as S(t), I(t), C(t), and M(t) at time t, respectively. Similarly, the percentages of each type of agents are represented as s(t), i(t), c(t), and m(t) at time t, respectively. The above relationship can be described as follows:

$$S(t) + I(t) + C(t) + M(t) = N \tag{8}$$

$$s(t) + i(t) + c(t) + m(t) = 1 \tag{9}$$

According to the contagious mechanism of SICM model, the dynamic characteristics are shown as follows:

$$\begin{aligned} \frac{ds(t)}{dt} &= -x_1 s(t) c(t) \\ \frac{di(t)}{dt} &= x_1 s(t) c(t) - x_2 i(t) - x_3 i(t) \\ \frac{dc(t)}{dt} &= x_2 i(t) - x_4 c(t) \\ \frac{dm(t)}{dt} &= x_3 i(t) + x_4 c(t) \end{aligned} \tag{10}$$

Expression (10) actually includes four differential equations, which, respectively, represent the rate of change of s(t), i(t), c(t), and m(t) with time t. In order to reflect the microcosmic mechanism of risk contagion in financial market based on SICM model, we try to design the simulation experiments in the next section.

## 4. The Simulation Experiments

In our simulation experiments, there is only one agent in contagious state; the rest of agents are in susceptible states at initial time. On the basis of the risk contagious model, two basic conclusions can be drawn before the simulation experiments [76]. The first conclusion is that the number of susceptible agents has a decreasing trend. And the second conclusion is that the number of contagious agents has an increasing trend. As for the changing characters of infected agents and immune agents in risk contagion, the conclusions will be drawn after the simulation experiments. Thus, the contagious probabilities of agents are set at first. And then the dynamic characteristics of different types of agents are simulated in the experiments. The baseline configuration is S(0)=N-1, I(0)=0, C(0)=1, M(0)=0, N=3000, $x_1$=0.005, $x_2$=0.10, $x_3$=0.20, and $x_4$=0.15. In the simulation process, once the number of agents no matter in whatever state is less than 0.0005, the experiment will be set to automatically stop. In this case, we assume that some kind of agent has all become other kinds. In Figure 2, the curve of I(t) and the curve of C(t) have the similar trends increasing first and then decreasing. When the number of I(t) is equal to 0.0004906, the experiment stops at time step 150.

In order to better reveal the influences of contagious probabilities on the process of risk contagion, we change the value of $x_1$, $x_2$, $x_3$, and $x_4$ in sequence and keep other parameters constant at the same time. The evolutionary characteristics of risk contagion are shown in Figures 3, 4, 5, and 6. The simulation experiments contain 5 cases in Table 1. And each case relates to each figure. For example, cases 1, 2, 3, 4 and 5 correspond to Figures 2, 3, 4, 5, and 6, respectively.

Firstly, $x_1$ is increased to 0.0010 from 0.0005 and other parameters remain unchanged, as described in Table 1. Likewise, the four curves of agents can be drawn and shown in Figure 3. The curve of S(t) has a similar feature in both Figures 2 and 3. In brief, the two curves of S(t) show a decreasing trend with a higher rate first and then a lower rate. But the other three curves have an obvious difference. In Figure 3, the amount of I(t) reaches a maximum with a faster speed at time step 25, and the curve of I(t) has a steeper trend. And the similar features are seen from the curve of C(t), which has a higher crest relative to Figure 2. For the curve of M(t), the amount of agents in infected states is greater than that in immune states before time step 22. Because of the increase of $x_1$, a grapevine can be spread to susceptible agents around a contagious agent with a higher probability. Considering that the grapevine is contacted by more agents in susceptible states, the running time of SICM model is shorted to time step 89 from time step 150 in Figure 3.

Secondly, $x_2$ is increased to 0.60 from 0.10, and other parameters are the same as the parameters in Figure 2. The change of $x_2$ directly affects the contagious effects from infected agents to contagious agents. More specifically, with a larger value of $x_2$, agents in infected states are likely to enter into contagious states with a higher probability. Thus, the curve of C(t) has a higher crest than the curve of I(t) in Figure 4, and the curve of C(t) in Figure 2. There are two intersections among the curve of M(t), the curve of I(t) and the curve of C(t). One is between the curve of M(t) and the

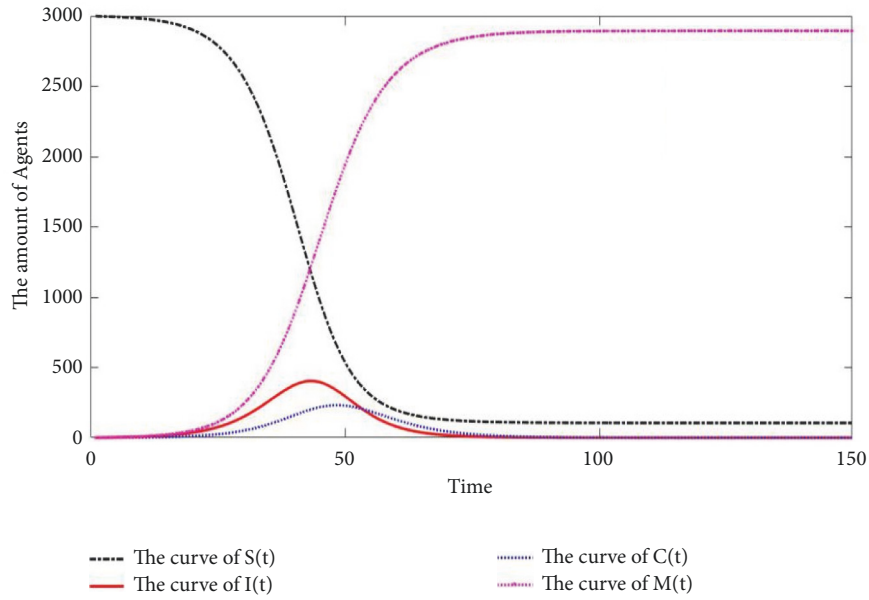


Figure 2: The evolutionary characteristics of risk contagion based on SICM model (Case 1).

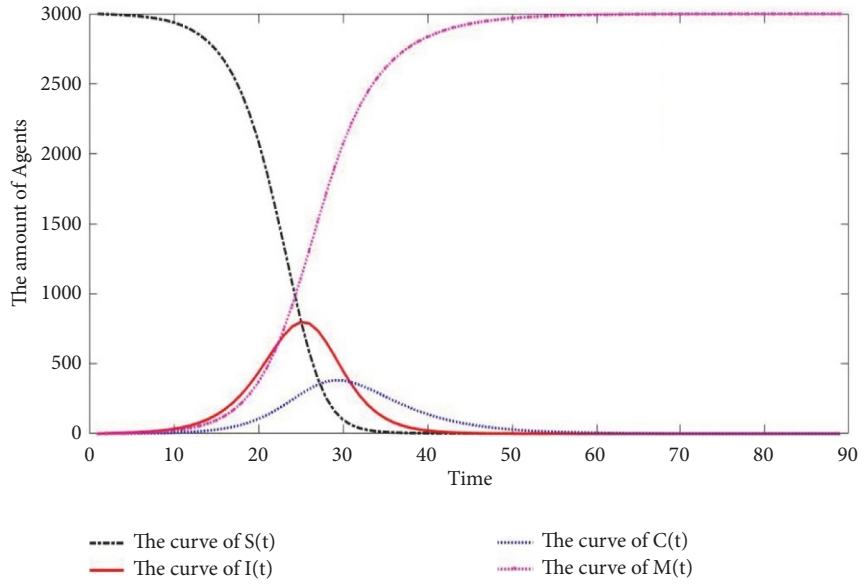


Figure 3: The evolutionary characteristics of risk contagion after increasing $x_1$ (Case 2).

curve of I(t), and the other intersection is between the curve of M(t) and the curve of C(t). To be specific, the amount of infected agents is larger than the amount of immune agents before time step 17 (including time step 17). And before time step 21 (including time step 21), the amount of contagious agents is larger than the amount of immune agents. But in general, the curve of M(t) shows an increasing trend and the curve of S(t) shows a decreasing trend. As an increasing $x_2$, the curve of I(t) decreases faster after time step 19, and the model comes to a stop at time step 60.

Thirdly, $x_3$ is increased to 0.20 from 0.30, and other contagious probabilities, $x_1$, $x_2$, and $x_4$, maintain the initial values of baseline configuration in Table 1. The obvious differences in Figure 5 manifest the curve of S(t) and the curve of M(t) compared with Figure 2. These two curves can be divided into two parts according to the trend characteristics in Figure 5. The two curves show a dramatic decrease or increase in the first stage ($0 \leq$ time step $\leq 90$), and a very slow decrease or increase in the second stage ($90 \leq$ time step $\leq 179$). For the curve of I(t), there is not a significant change in Figure 5, compared with that in Figure 2. And a similar conclusion can be drawn after analyzing the curve of C(t). As the increase of $x_3$ implies that susceptible agents become immune agents with a higher probability, the increasing

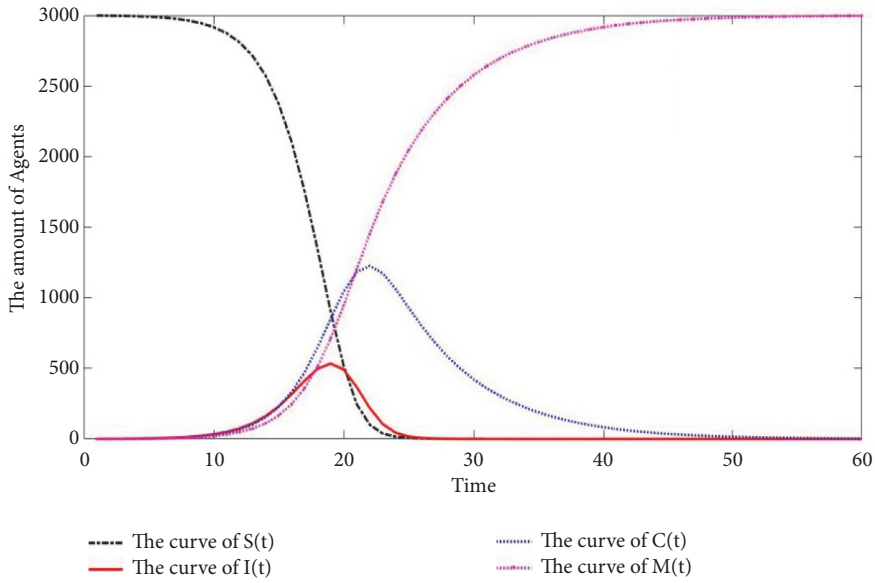


Figure 4: The evolutionary characteristics of risk contagion after increasing $x_2$ (Case 3).

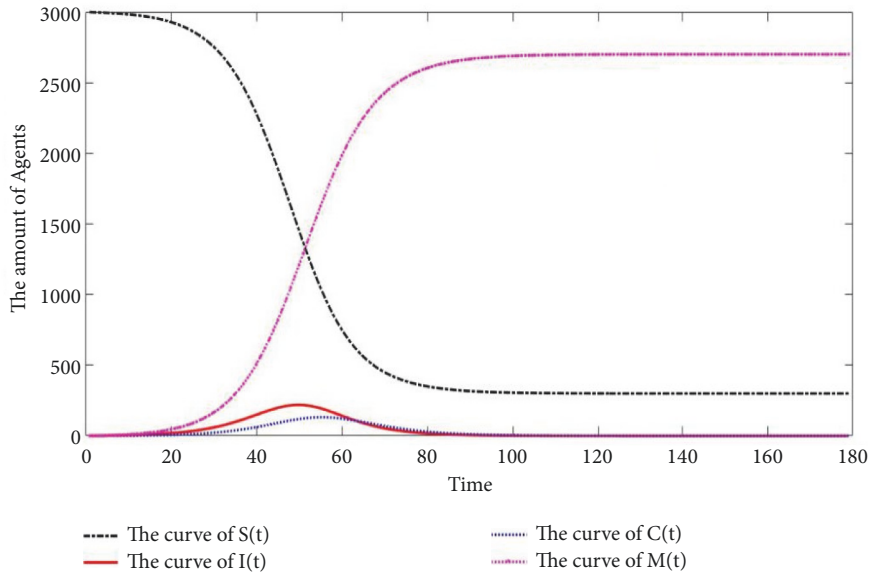


Figure 5: The evolutionary characteristics of risk contagion after increasing $x_3$ (Case 4).

Table 1: The features of parameters in different simulation environments.

| Case | Parameters features | Initial parameters | |
|---|---|---|---|
| | | $(x_1, x_2, x_3, x_4)$ | $(S(0), I(0), C(0), M(0))$ |
| Case 1 | To keep all parameter values constant | (0.0005, 0.10, 0.20, 0.15) | $S(0) = 2999$; $I(0) = 0$; $C(0) = 1$; $M(0) = 0$. |
| Case 2 | To increase the parameter value of $x_1$ | (0.0010, 0.10, 0.20, 0.15) | |
| Case 3 | To increase the parameter value of $x_2$ | (0.0005, 0.60, 0.20, 0.15) | |
| Case 4 | To increase the parameter value of $x_3$ | (0.0005, 0.10, 0.30, 0.15) | |
| Case 5 | To increase the parameter value of $x_4$ | (0.0005, 0.10, 0.20, 0.25) | |

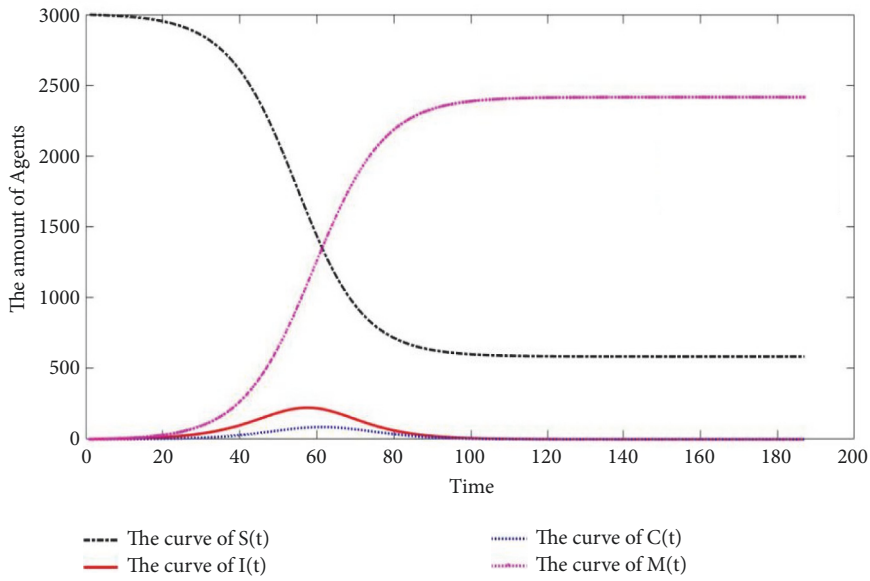


Figure 6: The evolutionary characteristics of risk contagion after increasing $x_4$ (Case 5).

Table 2: The evolutionary characteristics of agents after finishing simulation processes.

| Case | Parameters feature | Running time step | The amount of agents | | | |
|---|---|---|---|---|---|---|
| | | | S(t) | I(t) | C(t) | M(t) |
| Case 1 | To keep all parameter values constant | 150 | 105.1358 | 0.0004906 | 0.0017 | 2894.8621 |
| Case 2 | To increase the parameter value of $x_1$ | 89 | 1.3262 | 0.0004985 | 0.0566 | 2998.6168 |
| Case 3 | To increase the parameter value of $x_2$ | 60 | 0.1921 | 0.0004809 | 3.2472 | 2996.5603 |
| Case 4 | To increase the parameter value of $x_3$ | 179 | 299.5044 | 0.0004789 | 0.0000959 | 2700.4942 |
| Case 5 | To increase the parameter value of $x_4$ | 187 | 584.3215 | 0.0006814 | 0.0004612 | 2415.6774 |

quantity of immune agents is slow in the second stage. Simultaneously, the decreasing quantity of susceptible agents is also slow in this stage. In Figure 5, the evolution process of risk contagion finally stops at time step 179.

Fourthly, $x_4$ is increased to 0.25, and other parameters are unchanged. The evolutionary characteristics of risk contagion are still displayed by the curves of agents, including the curve of S(t), the curve of I(t), the curve of C(t), and the curve of M(t). It is easy to be seen that both Figures 5 and 6 are similar in the trends of the corresponding curves. Because no matter which contagious probability is increased, $x_3$ or $x_4$, the final result is the increasing quantity of immune agents. According to Figure 1, there are two ways to make infected agents become immune agents: (1) I⟶M and (2) I⟶C⟶M. As the second way includes two processes: I⟶C and C⟶M, $x_4$ has less impact on M(t) relative to $x_3$. Therefore, the final quantity of immune agents is smaller in Figure 6 than that in Figure 5 at the stop time. The curve of S(t) and the curve of M(t) are divided into two parts based on the trend of curves. The first part is the rapid change phase ($0 \leq$ time step $\leq 100$), and the second part is the slow change phase ($100 \leq$ time step $\leq 187$). During the whole simulation process, the amount of infected agents has little change, and the maximum is 222.7187 at time step 58. However, the amount of contagious agents has less change relative to infected agents, and the maximum is 86.3355, appearing at time step 61.

After the comparative analyses of Figures 2–6, the evolutionary characteristics of agents in SICM model are summarized in Table 2. The running time step indicates that the increase of $x_1$ or $x_2$ directly reinforces the effect of risk contagion, but the increase of $x_3$ or $x_4$ reduces this effect. In consequence, the running time step is shortened in cases 2 and 3, relative to case 1. Conversely, cases 4 and 5 have a longer running time step compared with that in case 1. When the simulation process automatically stops, the quantitative structures of different kinds of agents are displayed in Table 2. As the amount of I(t) decreases at the highest speed in the second part of simulation processes, the amount of I(t) is lower than the threshold in the shortest time, which is supposed in the baseline configuration of SICM model. In addition, the amount of M(t) is largest at stop time for each case, because most of other agents have been immune to a grapevine after the behavioral interactions of agents again and again.

Considering that the curve of I(t) and the curve of C(t) having similar fluctuation characteristics in SICM model, the amount of agents has a maximum for two curves. Table 3 shows the maximum values of I(t) and C(t) and the

Table 3: The maximum of infected agents and contagious agents in simulation processes.

| Case | Parameters feature | The amount of I(t) | | The amount of C(t) | |
|---|---|---|---|---|---|
| | | Maximum | Time step | Maximum | Time step |
| Case 1 | To keep all parameter values constant | 403.3906 | 43 | 231.3881 | 48 |
| Case 2 | To increase the parameter value of $x_1$ | 796.9925 | 25 | 382.0501 | 29 |
| Case 3 | To increase the parameter value of $x_2$ | 534.3893 | 19 | 1181.6496 | 21 |
| Case 4 | To increase the parameter value of $x_3$ | 219.2371 | 50 | 131.4064 | 55 |
| Case 5 | To increase the parameter value of $x_4$ | 222.7187 | 58 | 86.3355 | 61 |

corresponding time steps. It is easy to be seen that time steps are less in the curve of I(t), rather than the curve of C(I). Namely, the curve of I(t) reaches a peak first; then the curve of C(t) reaches another peak. Only in Figure 4, the curve of C(t) has a higher peak. However, the curve of C(t) has a lower peak in other figures in Table 3. Owing to a grapevine spreading to contagious agents from infected agents, the maximum value of I(t) is often larger than that of C(t) in cases 1, 2, 4, and 5. But an exception appears in case 3. To be specific, more contagious agents become infected agents when the contagious probability $x_2$ is increased in case 3.

## 5. Conclusions

In this paper, we employ agent modeling technology and complex network theory to build a new contagious model to analyze the nonlinear dynamics characteristic of risk contagion in financial market. In the model, market participants are divided into four types: susceptible agents, infected agents, contagious agents, and immune agents. Based on the proposed SICM model, a grapevine can spread in four kinds of agents with the different probabilities. According to different configuration parameters, the simulation experiments are conducted. The results show that the same type of curves has similar volatility characteristics without changing any of contagious probabilities. For example, the curve of S(t) has a decreasing trend and the curve of M(t) has an increasing trend in the model. However, the curves of I(t) and C(t) show an ascending trend first and then descending trend. In addition, the results also indicate that the running time of model is shortened after increasing $x_1$ or $x_2$. But if $x_3$ or $x_4$ is increased, the model needs more running time. From cases 1 to 4, the curves of I(t) reach a peak first, compared with the curve of C(t). In all cases, the amount of M(t) is the most at the stop time, and the amount of I(t) is the least below the stop threshold. In short, the simulation experiments are automatically finished because the amount of I(t) declines rapidly.

Different from other researches, this paper designs the SICM model of risk contagion in financial market and reveals the interaction features of agents under the influences of different contagious probabilities. The conclusions of this paper can offer a theoretical foundation for future studies about the behavioral features of market participants and the contagious mechanisms of financial risks. As an agent-based model, SICM model can also be developed further in future studies. On the one hand, each agent may have a heterogeneous probability of infection. It means that the contagious probability probably is not a constant. For example, each agent has different risk preferences, different understandings of market information, and irrational investment motivations. Because different agents in the same class may have different probabilities of infection, the function of contagious probability should be built in the future. On the other hand, the transmission speed of the grapevine is probably changeable in different network topologies, considering that degree distribution and average path length are not invariable in financial markets. As the transmission speed of the grapevine can affect the available information of agents in unit time, agents may make decisions to buy or sell financial assets more quickly or slowly. And the price of financial asset is likely to be changed, because of the change of the market demand and supply. Based on the above two parts, we will further extend the SICM model in future studies, by building the function of contagious probability, affected by the price of financial asset. In addition, financial data has important significance for the research of risk contagion in a real financial market. In our future works, we will try to import the market data into the model and analyze risk contagion in the real financial market.

## Data Availability

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

## Conflicts of Interest

All authors declare that they have no conflicts of interest regarding the publication of this paper.

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