Application of SIR Model on Bank Risk Contagion

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ABSTRACT

SIR model has been widely used in epidemiology and started to be used in various fields. This paper analyzes the risk contagion in banking system using a variation of SIR model. The differential equations based on the constructed model represent the dynamics of the risk contagion. The disease-free equilibrium and basic reproduction number, $\mathcal{R}_0$, is calculated in order to further analyze the model. The results show that the bank risk contagion needs to be considered both partially and systemically.

CCS CONCEPTS

• **Applied computing**; • **Law, social and behavioral sciences**; • **Economics**; • **Mathematics of computing**; • **Mathematical analysis**; • **Differential equations**; • **Ordinary differential equations**; • **Operations research**; • **Marketing**;

KEYWORDS

Bank risk, Risk contagion, SIR model, Systemic risk

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1 INTRODUCTION

The global financial crisis in 2008 caused severe damage to the international financial system, including the bankruptcy of several major investment and commercial banks [1]. Especially after Lehman Brother's failure, a large group of short-term bank creditors tried to withdraw their investments and caused a run on banks, which harmed the banks' capacity to roll over their short-term debt, and triggered the subsequent international banking crisis [2]. The interrelation between banks is one of the contributors to the spread and intensification of this crisis since banks are inextricably tied to each other in various ways. They may be linked directly via bilateral transactions or interlibrary loan and may also affect each other indirectly. For example, a failing bank's fire sale of assets may cause a decline in the asset value therefore result in mark-to-market losses for other banks [3]. Any impact on specific subject caused by the crisis cannot be isolated. Such impact is likely to generate shock waves that are transmittable to other subjects through chain reaction as the banks are tightly connected [4]. Therefore, what might seem as individual impact can potentially threaten the overall stability of the financial system as a whole. As a result, the close connectivity of banks in the market imposes danger of risk contagion, and this contagion in financial market may cause a systemic risk, which is defined as the risk of collapse of a system due to a series of failure a long a chain of financial institutions [5]. Therefore, it is necessary to understand the nature and characteristics of risk contagion in banking to control and minimize its negative effects.

Risk contagion spreads across banks like a disease. Many previous studies have shown the validity of analyzing the interbank risk contagion with an epidemiological model. In 2013, Toivanen applied an epidemiological model to the spread of risk contagion in banking networks and assessed how different characteristics of banks influence the contagion. Their results showed that the volume of interbank business crucially increase the risk contagion in a banking system. Based on their findings, they further suggested that the regulations should pay greater attention to the interconnection between banks rather than focusing only on the size of the bank [6]. In 2018, Kostylenko, Rodrigues, and Torres demonstrated that epidemiological models might be used to characterize the behavior of contagion spread of banking risks and presented simulating data from banks in Europe. Their approach provided qualitative specifications of contagion in banking and forecasted from a macroeconomic point of view [7]. They later proposed that a similar model may be applied in a broader context, that is, to understand the spread of financial virus globally [8]. Cao and Zhu discussed the infection threshold and immune strategy for banking crisis in their study in 2011. They concluded that to prevent risk contagion, it is more efficient to immunize the banks with large amount of assets and those have close connections with other banks [9].

Many researchers suggest that mathematical models simplify the process of understanding how an infection spreads over time. Between 1927 and 1933, Kermack and McKendrinck's theoretical studies on infectious disease models had a profound impact on creating mathematical epidemiology models. They presented the well-known compartmental model called SIR to simulate diseases such as chickenpox, measles, and other similar infections [10]. In 2001, Driessche and Watmough gave a definition of the basic reproduction number, $\mathcal{R}_0$, and its calculation. Their study claimed that $\mathcal{R}_0$ is a threshold parameter for the model and enabled better understanding and further analysis of the model [11]. Several researchers from across the world have claimed that the contagion spreads between banks are very similar to these diseases. However, this investigation has not been thoroughly examined. Most of the research did not carry out the calculation following construction of the models.

The purpose of this paper is to discuss the risk contagion in the banking industry using SIR model, an epidemiological model, and

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to find the method to control the spread based on the concept of dynamical system and differential equation models. The investigation of risks in banking system is classified into systemic risks and non-systemic risks. These risks are considered and modelled differently according to their features. The organization of this paper is as follows: in chapter 2, SIR model is briefly introduced. Chapter 3 describes the proposed model and its mathematical approach. In chapter 4, the disease-free equilibrium (DFE) and the basic reproduction number is calculated. Chapter 5 analyzes the results and give suggestions on how to control the risk spread. Chapter 6 concludes.

2 SIR MODEL

Compartmental models, significantly developed by Kermack and McKendrick in 1927, are often used to simulate infectious disease. In the models, the population is divided into different compartments, and individuals may move between the compartments. The basic compartmental model is called the SIR model which contains three compartments:

- Susceptible (S): individuals in this state may get infected if a "infectious contact" occurs between them and the infected individuals;
- Infected (I): in this state, individuals are infected and may infect susceptible individuals;
- Recovered or Resistant (R): individuals in this state are either recovered from infection or are immune.

The variables S, I, and R represent the number of individuals in the corresponding compartments and $S + I + R = N$, where N is the total number of individuals in the population. In order to illustrate the dynamics of the state variables over time, the variables are written as functions of t (time). A basic SIR system can be expressed as the following:

$$\begin{cases} \frac{dS}{dt} = -\frac{\beta IS}{N} \\ \frac{dI}{dt} = \frac{\beta IS}{N} - \gamma I \\ \frac{dR}{dt} = \gamma I \end{cases}$$

where β is the force of infection, and γ represents the recovery rate.

The basic model only represents certain diseases; however, different diseases have their own natures and characteristics. Moreover, epidemiological models are used as an innovated approach to describe situations in various fields, from health problems to marketing, informatics, and even sociology [12]. Therefore, variations of the basic model are used to satisfy different scenario.

3 METHODOLOGY

Consider a financial market, the banks in the market are divided into susceptible compartment, two infected compartments, carrier, and recovered or resistant compartment. The infected banks are those facing crisis, such as, improper coordination of bank assets and liabilities, low profitability, or lack of trust from clients. The infected banks are further divided into two compartments. Firstly, if the number of infected banks is not large enough to affect the whole financial system, then they will only infect those banks that are closely linked to them, for example, those with bilateral transactions. In this case, the susceptible banks which have close inter-connection with the infected banks may be influenced and move to the infected compartment, and these banks are denoted by I_n. If the number of infected banks is not controlled and exceeds a certain number, systemic risk occurs. In this case, any banks apart from the ones in the recovered or resistant compartment may be infected due to the systemic risks, and those infected banks are denoted by I_s.

Susceptible banks, denoted by S, are those that have defects in their risk control systems, such as taking excessive risks, but have not defaulted yet. These susceptible banks could be affected by infected banks, resulting in their own risk issues. However, if susceptible banks completed their risk management systems prior to infection, they would go directly to recovery or resistance stage, eliminating their exposure to systemic risks.

To simulate the risk contagion in banking market, a carrier state (C) is added to the basic model. In epidemiological situation, for some diseases, people may carry the infection while not suffering from it severely, and they may still infect others in this state. Similarly, banks may need a period of time to recover until fully functional. The banks that come up with effective recovery plans and adjust accordingly may start recovering and become carriers, C. During the recovery, banks are not fully functional and can only make low-risk investments. In this state, banks may fully recover and enter the recovered or resistant compartment, or they may be infected again and move to the infected compartments. The banks in the recovered or resistant compartment are those that gain immunity by either fully recover from the infection or directly by controlling the risks in advance, and they are denoted by R.

Moreover, for the sake of simplicity, the following assumptions are made:

(A1) The total number of banks in the market is fixed; that is, establishment of new banks and bankruptcy are negligible;

(A2) The infected banks may need certain period of time to recover from an infection, or they may enter recovered state directly;

(A3) The recovered banks have immunity against both type of the risks;

(A4) The recovered banks may lose the immunity and become susceptible again.

Based on the above discussion, the interbank infection in a financial market is illustrated in Figure 1

The number of banks at time t are denoted by $S(t)$, $I_n(t)$, $I_s(t)$, $C(t)$, and $R(t)$ and they sum up to the total number of banks in the financial market. The corresponding dynamic system of the model is formulated as follows:

$$\begin{cases} \frac{dI_n}{dt} \& = \beta_1 SI_n + \beta_3 CI_n - \beta_2 I_n I_s - (\gamma_1 + \lambda) I_n \\ \frac{dI_s}{dt} \& = \beta_2 SI_s + \beta_2 I_n I_s + \beta_2 CI_s - \omega I_s \\ \frac{dC}{dt} \& = \lambda I_n - \beta_3 CI_n - \beta_2 CI_s - \gamma_2 C \\ \frac{dS}{dt} \& = \omega I_s + \varepsilon R - \beta_1 SI_n - \beta_2 SI_s - \alpha S \end{cases}$$

where the parameters are described in Table 1

4 RESULTS

To further investigate the above model, the disease-free equilibrium (DFE) and the basic reproduction number, $\mathcal{R}_0$, need to be found. By Driessche, Pauline, and Watmough, a DFE of an epidemiological

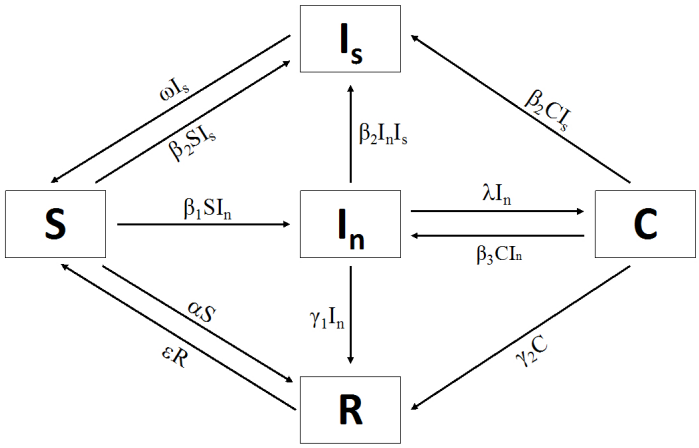

Figure 1: SICR Model for Interbank Infection.

Table 1: Parameter description

Parameter	Interpretation
ω	Systemic risk recovery rate
β_1	Non-systemic risk infection rate for susceptible banks
β_2	Systemic risk infection rate
β_3	Non-systemic risk infection rate for carrier banks
γ_1	Non-systemic risk recovery rate for infected banks
γ_2	Non-systemic risk recovery rate for carriers
λ	Rate of making effective adjustment and start recovering
ε	Rate of losing immunity and back to susceptible state
α	Rate of having a sound risk control system from the start

model is the state when there are no infections among the population. The basic reproduction number, $\mathcal{R}_0$, is a threshold parameter that determines the stability of the DFE [11].

4.1 The Disease-Free Equilibrium (DFE)

Let all the differential equations in the model equal to 0, and $I_n = I_s = C = 0$. Then,

$$\frac{dS}{dt} = \varepsilon R - \alpha S = 0 \implies \varepsilon R = \alpha S \Rightarrow R = \frac{\alpha}{\varepsilon} S$$

Therefore, the DFE is

$$x_0 = \begin{pmatrix} 0 & 0 & 0 & S_0 & \frac{\alpha}{\varepsilon} & S_0 \end{pmatrix}.$$

Without loss of generality, assume $S_0 = 1$ is a DFE. Therefore,

$$x_0 = \begin{pmatrix} 0 & 0 & 0 & 1 & \frac{\alpha}{\varepsilon} \end{pmatrix}.$$

4.2 The Basic Reproduction Number, $\mathcal{R}_0$

Decompose the system into F and V, where

$$\mathcal{F} = \begin{pmatrix} \beta_1 \mathrm{SI_n} \\ \beta_2 \mathrm{SI_s} \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

$$\mathcal{V} = \begin{pmatrix} -\beta_3 \mathrm{CI_n} + \beta_2 I_n I_s + (\gamma_1 + \lambda)\,\mathrm{I_n} \\ -\beta_2 I_n I_s - \beta_2 C I_s + \omega \mathrm{I_s} \\ -\lambda \mathrm{I_n} + \beta_3 \mathrm{CI_n} + \beta_2 C I_s + \gamma_2 \mathrm{C} \\ -\omega \mathrm{I_s} - \varepsilon \mathrm{R} + \beta_1 \mathrm{SI_n} + \beta_2 \mathrm{SI_s} + \alpha \mathrm{S} \\ -\alpha \mathrm{S} - \gamma_1 \mathrm{I_n} - \gamma_2 \mathrm{C} + \varepsilon \mathrm{R} \end{pmatrix}$$

According to Driessche and Watmough, the derivatives of F and V at x_0 are partitioned as

$$\mathrm{D}\mathcal{F}(x_0) = \begin{pmatrix} F & 0 \\ 0 & 0 \end{pmatrix}$$

$$DV(X_0) = \begin{pmatrix} V & 0 \\ J_3 & J_4 \end{pmatrix}$$

where J_3 is a 2 x 3 matrix and J_4 is a 2 x 2 matrix. Then

$$F = \begin{pmatrix} \beta_1 & 0 & 0 \\ 0 & \beta_2 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} \gamma_1 + \lambda & 0 & 0 \\ 0 & \omega & 0 \\ -\lambda & 0 & \gamma_2 \end{pmatrix}$$

and all eigenvalues of J_4 have positive real part.

Following Diekmann and Driessche and Watmough, the generation matrix FV^{-1} needs to be calculated, and its spectral radius is said to be the basic reproduction number [11], that is

$$FV^{-1} = \begin{pmatrix} \frac{\beta_1}{\gamma_1+\lambda} & 0 & 0 \\ 0 & \frac{\beta_2}{\omega} & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

which has eigenvalues

$$\lambda_1 = 0$$

$$\lambda_2 = \frac{\beta_1}{\gamma_1 + \lambda}$$

$$\lambda_3 = \frac{\beta_2}{\omega}$$

Thus, the basic reproduction number $\mathcal{R}_0 = \frac{\beta_1}{\gamma_1+\lambda}$ when the number of infected banks is within control, and β_1 is significantly greater than β_2.

$\mathcal{R}_0 = \frac{\beta_2}{\omega}$ when β_2 is significantly greater than β_1, and there are plenty of infected banks that cause systemic risks.

5 DISCUSSION

In epidemiological cases, the basic production number, $\mathcal{R}_0$, is the average number of secondary cases caused by a single case in an entirely susceptible population. Because the value of $\mathcal{R}_0$ enables one to predict the work required to avoid an infection or to remove a disease from a community, it is essential to calculate $\mathcal{R}_0$ for a disease in a particular population [13]. When the value of $\mathcal{R}_0$ is greater than one, the virus will continue to spread among the susceptible hosts. When $\mathcal{R}_0$ is smaller than one, the virus will extinct naturally. Mathematically, by Diekmann and Driessche and Watmough, the DFE is locally asymptotically stable when $\mathcal{R}_0 < 1$, and it is unstable when $\mathcal{R}_0 > 1$ [11]. Therefore, from both epidemiological and mathematical points of view, in order to control the infection, the value of $\mathcal{R}_0$ needs to be minimized. In banking system, by above calculation, $\mathcal{R}_0$ has two possible values. When β_1 is significantly greater, $\mathcal{R}_0 = \frac{\beta_1}{\gamma_1+\lambda}$. In this case, non-systemic risks infection needs to be controlled and the recovery rate need to be increased. β_1 is the rate of infection between the infected banks and the banks that are closely tied to them. To lower the value of β_1, linkage between banks need to be reduced, for example, less interbank credit exposure, or financial service dependencies [3]. To increase the value of γ_1 and λ, the banks need to work on how to propose efficient recovery plan and be resilient to changes in the financial environment.

At this stage, systemic risks need to be prevented. Banking panics, assets price declines, and contagion are the main reasons of systemic risks [14]. For example, a bank may want to take higher risks in return for higher profit, and other banks may follow its steps which may lead to systemic risks. Regulations like Basel III are needed in order to prevent the systemic risks. When there are systemic risks in the market, and β_2 is significantly greater than β_1, $\mathcal{R}_0 = \frac{\beta_2}{\omega}$. To reduce the infection rate and increase the recovery rate of systemic risks, the government needs to step in and make adjustment from a macroeconomic point of view. Since systemic risks can influence every institution within the financial system the infection is very likely to spread across the market and cause financial crisis.

Furthermore, immunity of the individuals and the whole market should be strengthened. For example, a bank needs to assure its reserve before making high-risk investment. The susceptible banks should improve their management and try to be immune from the start. Risks should be monitored within the market by regulatory authorities using technologies like artificial intelligence and big data. If there are infections in a submarket, the rest of the banks should try to disconnect to prevent contagion.

6 CONCLUSION

Systemic risks need to be controlled to prevent financial crisis. Based on the calculation of the basic reproduction number, the risk contagion in banking system mainly depends on the interconnection between banks, the number of infected banks, the possibility of recovering from infection, and risk control ability of individual banks.

This paper mainly focuses on the construction of the model and risks analysis. However, it is essential to define the parameters used in the model in order to run simulations and apply to real world situations. Further studies could assign formulas to each parameter with variables that can be observed directly from the market. In this way, the $\mathcal{R}_0$ can be represented in more details and therefore calculated explicitly.

Furthermore, establishment and bankruptcy of banks are negligible in the model to keep the total number unchanged. The establishment and bankruptcy are represented by birth rate and death rate in the SIR model respectively. It would be more realistic if the model is improved and includes these factors.

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