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Stability Analysis of a Credit Risk Contagion Model with Distributed Delay

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Abstract: This research investigates the stability and occurrence of Hopf bifurcation in a credit risk contagion model, which includes distributed delay, using the chain trick method. The model is a generalized version of those previously examined. The model is an expanded version of those previously studied. Comparative analysis showed that unlike earlier models, which only used the nonlinear resistance coefficient to determine the rate of credit risk infection, the credit risk contagion rate is also affected by the weight given to past behaviors of credit risk participants. Therefore, it is recommended to model the transmission of credit risk contagion using dispersed delays.

Keywords: time delay; credit risk; distributed delay; stability analysis; stability switch

MSC: 34K18; 91B62

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1. Introduction

The traditional perspective on credit risk involves borrowers failing to make timely payments, resulting in financial losses for the creditors [1]. The industrial and real economy sectors or firms with limited internal funds depend on the financial sector as a crucial source of funding, so their risk and return are significantly influenced by the unpredictability of the financial sector, especially in terms of their profitability and stability. According to Chen et al. [2], as the system's stability decreases, the infectious scale of credit risk and the wavy frequency of credit risk contagion increase. This leads to chaotic phenomena in the credit risk contagion system, and the increase becomes gradual as the time delay increases. Any credit risk contagion system includes inherent structural instability, meaning that any disruptions within the system can cause a qualitative or topological change in its dynamic properties [2]. Hence, it is crucial to develop a mathematical model with time delays to capture the dynamics of credit risk contagion in order to understand the impact of the internal complexity of the credit risk contagion system and to offer both theoretical and practical guidance for credit risk management [3].

The significance of time delay is widely recognized in mathematical models related to finance, economics, and population dynamics [4,5]. This is because differential equations alone cannot fully capture the complexities of economic and financial phenomena [3]. Time delays commonly occur in real financial markets due to finite information transmission speed, noise, and the transport of matter and energy [6]. Additionally, mathematical models incorporating time delays have been suggested in the relevant literature to account for system behavior at previous time points, such as those observed in economic and biological systems [4,5,7–9]. The time delay has had notable impacts on the system, causing a switch in stability from a stable state to an unstable one, as well as leading to fluctuations and Hopf bifurcation [10,11].

Chen et al. [12] modeled the mechanism of time delay and nonlinear resistance in credit risk contagion within the Newman–Watts length scale connection and long-distance connection. They presented its dynamics using the following time-delay differential equation:

$$\dot{N}(t) = \lambda k_1 - N(t) + \lambda k_2 N(t-\tau) - \mu\xi[\lambda k_2 N(t-\tau)]^2, \quad t \geq 0 \tag{1}$$

$$N(t) = c, \quad -\tau \leq t \leq 0$$

where $N(t)$ denotes the number of CRT activity participants who are affected by credit risk in the CRT market at time t. The parameters $\xi, k_1, k_2, \lambda, \mu, c$, and τ are all positive, and, respectively, represent the Newman–Watts length scale, the number of instances where the connection distance from a participant infected by credit risk matches the Newman–Watts length scale, the number of instances where the connection distance from an infected participant constitutes a long-distance connection, the effective contagion rate of credit risk in the credit risk transfer (CRT) market, the nonlinear resistance coefficient of the network of relationships among CRT market participants, a real parameter, and the time delay in credit risk contagion within the long-distance connection.

Chen et al. [2] investigated the following stochastic time-delayed system of credit risk contagion, which is influenced by correlated Gaussian white noise:

$$\dot{N}(t) = \lambda_1 N(t) + \lambda_2 N(t-\tau) - \mu\xi[\lambda_2 N(t-\tau)]^2 + N(t)\psi(t) + \eta(t), \tag{2}$$

where $N(t)$ refers to the number of credit activity participants affected by credit risk in the financial market, while the terms $\psi(t)$ and $\eta(t)$ are stochastic and represent noise in the system. The parameters λ_1 and λ_2, with $\lambda_1 > \lambda_2$, respectively, represent the effective spread of credit risk among various participants in credit activities, reflecting both direct and indirect business relationships in the financial market. τ represents the time delay for the spread of credit risk among participants through indirect connections. ξ is the Nelength scale and μ is the nonlinear resistance factor within the network of relationships among credit activity participants, indicating the diversity among participants, encompassing their offerings and psychological tendencies. For recent studies on credit risk contagion models, see [13–21].

Bianca and Guerrini [22] extended the work of Chen et al. [2] in the form of the following equation:

$$\dot{N}(t) = \lambda_1 N(t) + \lambda_2 N(t-\tau) - \mu\xi\lambda_2^2[N(t-\tau)]^2, \tag{3}$$

where $N(t)$ represents the density of individuals with credit activity in the financial sector who are at risk of credit exposure, λ_1 is the credit risk contagion rate associated with direct business relations, λ_2 is the credit risk contagion rate associated with indirect business relations, μ is alluded to as the Nelength scale, ξ symbolizes the nonlinear resistance factor in the network connecting financial market participants involved in credit activity, and $\tau \geq 0$ is the time delay of credit risk contagion. Bianca and Guerrini [22] used normal form theory and the center manifold argument to establish the presence of the Hopf bifurcation, as opposed to the numerical method employed by Chen et al. in [2]. They also identified the stability, direction, and period of the bifurcating periodic solutions with specific formulas and demonstrated that the examination of the deterministic aspect of model (3) is not limited to the scenario where $\lambda_1 > \lambda_2$.

In the current literature, time delays are categorized as either fixed or continuously distributed (referred to as distributed delay). Fixed delays apply to contexts where specific time gaps are stipulated for involved parties. In contrast, distributed delays are suitable for scenarios where delays among actors vary. A key challenge lies in the uncertain nature of these time intervals. Distributed delays represent a weighted average incorporating all historical data from time zero to the present, making them more accurate in describing time-delayed systems. As found by Elaiw and Algha in [23], the concept of a distributed

delay provides a more comprehensive and realistic way to model processes. Compared to models with a fixed time delay, it is evident that the latter oversimplifies the actual system process [24]. This assertion is bolstered in a study by Hu et al. [25] when a distributed delay was proposed to model time-delayed inherent biology systems. It was discovered that this model was sufficiently general to encompass a variety of characteristics exhibited by the system. Therefore, we generalize Equation (3) by substituting continuously distributed time delays for the discrete delays, as shown below, to assess if the distribution of time delays induces some changes in the rate of credit risk contagion compared to fixed time delay in the literature, which depends only on the nonlinear resistance coefficient. Thus, from the dynamics of the distributed delay model, the time delay of the number of individuals infected by credit risk varies among them, which is an extension of the effect of the discrete delay parameter in the previous models, which were the same for individuals infected by credit risk.

This paper is structured as follows. Section 2 deals with the formulation of the model, while basic properties of the model are derived in Section 3. In Section 4, the discussion revolves around the stability analysis, stability switch, and Hopf bifurcation analysis of the model. In Section 5, detailed numerical solutions and a stability analysis are conducted, comparing our model with the findings of Chen et al. [2]. Finally, Section 6 concludes.

2. Model Formulation

We propose the following credit risk contagion model with distributed delays:

$$\dot{N}(t) = \lambda_1 N(t) + \lambda_2 \int_{-\infty}^{t} N(r) f(t-r) dr - \mu \xi \lambda_2^2 \left[\int_{-\infty}^{t} N(r) g(t-r) dr \right]^2, \tag{4}$$

where the delay kernels $f(r)$ and $g(r)$ are non-negative bounded functions defined on $[0, \infty)$, satisfying the normalized conditions:

$$\int_0^\infty f(r) dr = 1,$$

$$\int_0^\infty g(r) dr = 1.$$

When

$$f(r) = g(r) = \delta(r),$$

where δ denotes a Dirac delta function, one recovers the discrete delay case (3). Another case of interest is when the kernels are gamma distributions (see Cushing [26]), namely

$$f(r) = g(r) = \frac{a^m r^{m-1} e^{-ar}}{(m-1)!},$$

with m being a positive integer that defines the form of the weighting function whose average delay is $T = m/a$. Notice that as $T \to 0$, the distribution functions f, g approach the Dirac distribution. In the literature, researchers focus on the special cases where the delay kernel functions f and g may take the so-called weak kernel form

$$f(r) = g(r) = ae^{-ar} \ (a > 0),$$

and strong kernel form

$$f(r) = g(r) = a^2 r e^{-ar} \ (a > 0).$$

The term weak kernel suggests that the significance of past events diminishes exponentially as one looks further into the past, while the term strong kernel indicates that a specific time in the past holds more importance than any other. In the next sections, we shall consider several special cases given by a combination of the special forms of the kernels.

3. Existence and Uniqueness of Solution

We use the contraction mapping theorem (see, e.g., [27–29]) to determine the existence and uniqueness of the solution of Equation (4). Set $TN = N$ and rewrite Equation (4) as

$$\lambda_1 N = \dot{N} - \lambda_2 \int_{-\infty}^{t} Ng(t-r)dr + \mu\xi\lambda_2^2 \left[\int_{-\infty}^{t} Ng(t-r)dr\right]^2.$$

Since $g(t-r)$ is continuous, we have that $|g-r| \leq H$. Given any N_1 and N_2,

$$TN_1 = \frac{1}{\lambda_1}\left\{\dot{N}_1 - \lambda_2 \int_{-\infty}^{t} N_1 g(t-r)dr + \mu\xi\lambda_2^2 \left[\int_{-\infty}^{t} N_1 g(t-r)dr\right]^2\right\}$$

and

$$TN_2 = \frac{1}{\lambda_1}\left\{\dot{N}_2 - \lambda_2 \int_{-\infty}^{t} N_2 g(t-r)dr + \mu\xi\lambda_2^2 \left[\int_{-\infty}^{t} N_2 g(t-r)dr\right]^2\right\},$$

implying

$$\begin{aligned}
TN_1 - TN_2 &= \frac{1}{\lambda_1}\left\{-\lambda_2 \int_{-\infty}^{t} (N_1 - N_2)g(t-r)dr + \mu\xi\lambda_2^2 \left[\int_{-\infty}^{t} (N_1^2 - N_2^2)g^2(t-r)dr\right]\right\} \\
&= \frac{1}{\lambda_1}\left\{-\lambda_2 \int_{-\infty}^{t} (N_1 - N_2)g(t-r)dr \right. \\
&\qquad \left. +\mu\xi\lambda_2^2 \left[\int_{-\infty}^{t} (N_1 - N_2)(N_1 + N_2)g^2(t-r)dr\right]\right\} \\
&= \frac{1}{\lambda_1}(N_1 - N_2)\int_{-\infty}^{t} \left[-\lambda_2 g(t-r) + \mu\xi\lambda_2^2 (N_1 + N_2)g^2(t-r)\right]dr,
\end{aligned}$$

consequently, we obtain

$$\begin{aligned}
|TN_1 - TN_2| &= \frac{1}{\lambda_1}|N_1 - N_2| \int_{-\infty}^{t} \left[-\lambda_2 g(t-r) + \mu\xi\lambda_2^2 (N_1 + N_2)g^2(t-r)\right]dr \\
&\leq \frac{1}{\lambda_1}\left|\int_{-\infty}^{t} \left[-\lambda_2 g(t-r) + \mu\xi\lambda_2^2 (N_1 + N_2)g^2(t-r)\right]dr\right| \\
&\leq \frac{\beta}{\lambda_1}\left[-\lambda_2 g(t-r) + \mu\xi\lambda_2^2 (N_1 + N_2)g^2(t-r)\right]|N_1 - N_2| \\
&\leq \eta|N_1 - N_2|,
\end{aligned}$$

where

$$\eta = \frac{\beta}{\lambda_1}\left[-\lambda_2 g(t-r) + \mu\xi\lambda_2^2 (N_1 + N_2)g^2(t-r)\right].$$

For contraction,

$$\frac{\beta}{\lambda_1}\left[-\lambda_2 g(t-r) + \mu\xi\lambda_2^2 (N_1 + N_2)g^2(t-r)\right] \leq 1. \tag{5}$$

Since (5) holds, according to the contraction mapping theorem, it can be concluded that there is a single fixed point that serves as the only solution to the original integro-differential Equation (4). As a result, in cases where the parameters are chosen such that the kernel is small, ensuring contraction, the existence and uniqueness of the solution to Equation (4) are guaranteed.

4. Stability and Hopf Bifurcation Analysis

Since steady states are unaffected by the delay, Equation (4) admits the following unique positive equilibrium:

$$N_* = \frac{\lambda_1 + \lambda_2}{\mu\xi\lambda_2^2}. \tag{6}$$

In the following, we examine the stability and Hopf bifurcation of our model in certain specific scenarios. Our method utilizes the well-established linear chain trick (refer to [30]), which enables a reduction in a system with distributed delays to a system of ordinary differential equations.

4.1. Case f, g are Weak Kernels

Let

$$f(r) = g(r) = ae^{-ar},\ a > 0.$$

Equation (4) writes as

$$\dot{N}(t) = \lambda_1 N(t) + \lambda_2 \int_{-\infty}^{t} N(r)ae^{-a(t-r)}dr - \mu\xi\lambda_2^2\left[\int_{-\infty}^{t} N(r)ae^{-a(t-r)}dr\right]^2. \tag{7}$$

Introducing the variable

$$u(t) = \int_{-\infty}^{t} N(r)ae^{-a(t-r)}dr,$$

by the linear chain technique, Equation (7) gets converted to the equivalent system below:

$$\begin{cases} \dot{N}(t) &= \lambda_1 N(t) + \lambda_2 u(t) - \mu\xi\lambda_2^2[u(t)]^2, \\ \dot{u}(t) &= a[N(t) - u(t)]. \end{cases} \tag{8}$$

Linearizing (8) at its equilibrium point (N_*, u_*), where $u_* = N_*$, with N_* as in (6), we obtain the Jacobian matrix:

$$J = \begin{pmatrix} \lambda_1 & \lambda_2 - 2\mu\xi\lambda_2^2 N_* \\ a & -a \end{pmatrix}. \tag{9}$$

Let ρ denote a characteristic root of (9). The related characteristic equation is of second order in ρ,

$$\rho^2 + (a - \lambda_1)\rho + a\left(2N_*\mu\xi\lambda_2^2 - \lambda_1 - \lambda_2\right) = 0, \tag{10}$$

resulting in

$$\rho^2 + (a - \lambda_1)\rho + a(\lambda_1 + \lambda_2) = 0.$$

The determinant of J equals the product of the eigenvalues and is given by

$$\rho_1\rho_2 = a - \lambda_1,$$

while the trace of J is equal to the sum of the eigenvalues

$$\rho_1 + \rho_2 = a(\lambda_1 + \lambda_2) > 0.$$

The model's local asymptotic stability is assured when both roots have negative real parts, leading to the following outcome.

Theorem 1. *For $a > 0$, system (8) is locally asymptotically stable if $a > \lambda_1$, unstable if $a < \lambda_1$.*

We now use the Hopf bifurcation theorem to demonstrate that a limit cycle could start at $a = \lambda_1$. This theorem says that we can prove the existence of a cyclic solution if the characteristic equation has a pair of purely imaginary roots and the real parts of these roots switch signs with a bifurcation parameter. Suppose $\rho = i\omega$, $\omega > 0$, is a root of (10). Substituting it into (10), we have

$$-\omega^2 + (a - \lambda_1)(i\omega) + a(\lambda_1 - \lambda_2) = 0.$$

Separating the real and imaginary parts, we obtain

$$-\omega^2 + a(\lambda_1 + \lambda_2) = 0,$$

$$(a - \lambda_1)\omega = 0,$$

which yield

$$a = \lambda_1 = a_*,$$

$$\omega = \sqrt{\lambda_1(\lambda_1 + \lambda_2)} = \omega_*.$$

Accordingly, we conclude that Equation (10) has a pair of purely imaginary roots $\rho = \pm i\omega_*$ when $a = a_*$.

Next, we select a as a bifurcation parameter and consider the eigenvalue as a function of a, $\rho = \rho(a)$. By implicitly differentiating Equation (10) with respect to a, we obtain

$$\frac{d\rho}{da} = -\frac{\rho + \lambda_1 + \lambda_2}{2\rho + a - \lambda_1},$$

then,

$$Re\left(\frac{d\rho}{da}\right)_{\rho = i\omega_*} = Re\left(-\frac{i\omega_* + \lambda_1 + \lambda_2}{2i\omega_*}\right) = -\frac{1}{2}.$$

so that

$$sign\left[Re\left(\frac{d\rho}{da}\right)_{\rho = i\omega_*}\right] < 0.$$

As the value of a decreases, the roots of the characteristic equation will transition from the right side to the left side of the imaginary axis. Based on the prior examination, we can draw the following conclusions.

Theorem 2. *The equilibrium point of (7) bifurcates to a limit cycle through a Hopf bifurcation when $a = \lambda_1$.*

4.2. Case f, g are Strong Kernels

Let

$$f(r) = g(r) = a^2 r e^{-ar},\ a > 0.$$

Equation (4) becomes

$$\dot{N}(t) = \lambda_1 N(t) + \lambda_2 \int_{-\infty}^{t} N(r) a^2 (t - r) e^{-a(t-r)} dr - \mu\xi\lambda_2^2 \left[\int_{-\infty}^{t} N(r) a^2 (t - r) e^{-a(t-r)} dr\right]^2. \quad (11)$$

To apply the linear chain trick on the integro-differential Equation (11), we define

$$u(t) = \int_{-\infty}^{t} N(r) a^2 (t - s) e^{-a(t-r)} ds, \quad v(t) = \int_{-\infty}^{t} N(r) a e^{-a(t-r)} ds,$$

so that (11) transforms into

$$\begin{cases} \dot{N}(t) &= \lambda_1 N(t) + \lambda_2 u(t) - \mu\xi\lambda_2^2 u(t)^2, \\ \dot{u}(t) &= a[v(t) - u(t)], \\ \dot{v}(t) &= a[N(t) - v(t)]. \end{cases} \tag{12}$$

The equilibrium point of (12) takes the form (N_*, u_*, v_*), with $u_* = v_* = N_*$, N_* defined in (6). To determine its stability, the characteristic equation of the linearized system is taken into consideration, leading to the following polynomial equation in ρ:

$$\rho^3 + (2a - \lambda_1)\rho^2 + a(a - 2\lambda_1)\rho + a^2(\lambda_1 + \lambda_2) = 0. \tag{13}$$

The Routh–Hurwitz criterion implies that the real parts of the solutions of (13) are negative if and only if the following conditions hold:

$$2a - \lambda_1 > 0,$$

$$a^2(\lambda_1 + \lambda_2) > 0,$$

$$(2a - \lambda_1)a(a - 2\lambda_1) - a^2(\lambda_1 + \lambda_2) > 0.$$

These can be reduced to

$$a > \frac{\lambda_1}{2} = a_0$$

and

$$2a^2 - (6\lambda_1 + \lambda_2)a + 2\lambda_1^2 > 0. \tag{14}$$

The discriminant of the left-hand side of (14) is given by

$$\Delta = (10\lambda_1 + \lambda_2)(2\lambda_1 + \lambda_2) > 0.$$

Thus, the inequality (14) is solved by

$$\begin{aligned} a &< \frac{6\lambda_1 + \lambda_2 - \sqrt{(10\lambda_1 + \lambda_2)(2\lambda_1 + \lambda_2)}}{4} = a_1, \\ a &> \frac{6\lambda_1 + \lambda_2 + \sqrt{(10\lambda_1 + \lambda_2)(2\lambda_1 + \lambda_2)}}{4} = a_2 \end{aligned} \tag{15}$$

Noticing that $0 < a_1 < a_0 < a_2$, we arrive at the following result.

Proposition 1. *Let a_2 be defined as in (15). The equilibrium point of (12) is locally asymptotically stable if $a > a_2$, unstable if $a < a_2$.*

We will now go back to (13) and demonstrate via the Hopf bifurcation theorem that a limit cycle might begin at $a = a_2$. At the value $a = a_2$, where one has

$$(2a_2 - \lambda_1)a_2(a_2 - 2\lambda_1) = a_2^2(\lambda_1 + \lambda_2),$$

the characteristic Equation (13) can be rewritten as

$$\rho^3 + (2a_2 - \lambda_1)\rho^2 + a_2(a_2 - 2\lambda_1)\rho + (2a_2 - \lambda_1)a_2(a_2 - 2\lambda_1) = 0,$$

which factors as

$$(\rho + 2a_2 - \lambda_1)\left[\rho^2 + a_2(a_2 - 2\lambda_1)\right] = 0. \tag{16}$$

Thus, one of (16) is real and negative, i.e., $\rho = 2a_2 - \lambda_1$, and the other two are purely imaginary:

$$\rho = \pm i\sqrt{a_2(a_2 - 2\lambda_1)} = \pm i\omega_*,$$

where $\omega_* > 0$. By choosing a as the bifurcation parameter, taking the derivative of (13) with respect to a, we derive

$$\frac{d\rho}{da} = -\frac{2\lambda^2 + 2(a - \lambda_1)\lambda + 2a(\lambda_1 + \lambda_2)}{3\lambda^2 + 2(2a - \lambda_1)\lambda + a(a - 2\lambda_1)}.$$

A direct calculation implies

$$\begin{aligned} Re\left(\frac{d\rho}{da}\right)_{\lambda=i\omega_*} &= Re\left(-\frac{-2\omega_*^2 + 2a(\lambda_1 + \lambda_2) + 2(a - \lambda_1)i\omega_*}{-3\omega_*^2 + a(a - 2\lambda_1) + 2(2a - \lambda_1)i\omega_*}\right) \\ &= -\frac{\lambda_1^2 + 3a_2^2 - 6\lambda_1 a_2 - \lambda_2 a_2}{(2a_2 - \lambda_1)^2 + a_2^2(a_2 - 2\lambda_1)} \\ &= -\frac{(10\lambda_1 + \lambda_2)(2\lambda_1 + \lambda_2) + \sqrt{(10\lambda_1 + \lambda_2)(2\lambda_1 + \lambda_2)}(6\lambda_1 + \lambda_2)}{8(2a_2 - \lambda_1)^2 + a_2^2(a_2 - 2\lambda_1)}, \end{aligned}$$

namely

$$sign\left[Re\left(\frac{d\rho}{da}\right)_{\rho=i\omega_*}\right] < 0.$$

As a result, the imaginary axis is crossed by the roots at $a = a_2$ from left to right as a decreases. In summary, the outcome is as follows.

Theorem 3. *Let a_2 be as in (15). The equilibrium point of (12) undergoes a Hopf bifurcation at $a = a_2$.*

5. Numerical Simulations

The first part of the numerical analysis of Chen et al. [2] was restricted to the deterministic part of the model (3) using the following parameters: time delay $\tau = 4$, Nelength scale $\xi = 3$, spread of credit risk among different participants engaged in credit activities is closely linked to their direct business relationships $\lambda_1 = 0.12$, transmission rate of credit risk among various participants in credit activities is associated with their indirect business relationships $\lambda_2 = 0.07$, and the initial number of credit activity participants who are infected by credit risk in the financial market N_0. We keep $\tau = 4$ and vary the nonlinear resistance coefficient μ and the weight γ on the previous activities of the credit risk participant to assess the dynamics of the credit risk contagion in the financial market on the assumption that $\lambda_1 = 0.12 > \lambda_2 = 0.07$, as we compare model (4) to model (3) (see [2]).

5.1. Time Process Diagrams of Credit Risk in Financial Market on $\lambda_1 > \lambda_2$

The actual number of credit activity participants infected by credit risk in the financial market can be determined by multiplying the simulated value from model (4) by 10 from the $N(t)$ axis compared to the values in Chen et al. [2].

From Figure 1, it is observed that at a time delay of $\tau = 4.0$ and nonlinear resistance coefficient of $\mu = 0.15$, the range of credit risk and the infectious scale are diminished compared to the subsequent simulation analysis of the diagrams in the study.

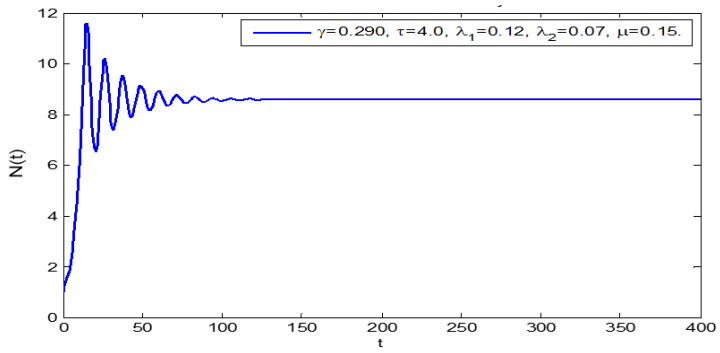

Figure 1. Dynamics of credit risk contagion under nonlinear resistance coefficient of $\mu = 0.15$.

It is observed in Figure 2 that as the nonlinear resistance coefficient is reviewed downward to $\mu = 0.08$ from $\mu = 0.15$, at a time delay of $\tau = 4.0$, the infectious scale and range of credit risk are gradually increased.

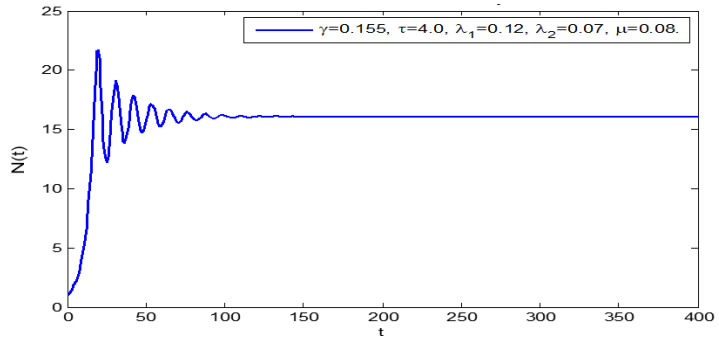

Figure 2. Dynamics of credit risk contagion under nonlinear resistance coefficient of $\mu = 0.08$.

In Figure 3, the nonlinear resistance coefficient is further reviewed downward to $\mu = 0.05$ with all other parameters held at the same level compared to Figures 2 and 3, and the rate of credit contagion increased to 26.25 from 8.75 in Figure 1.

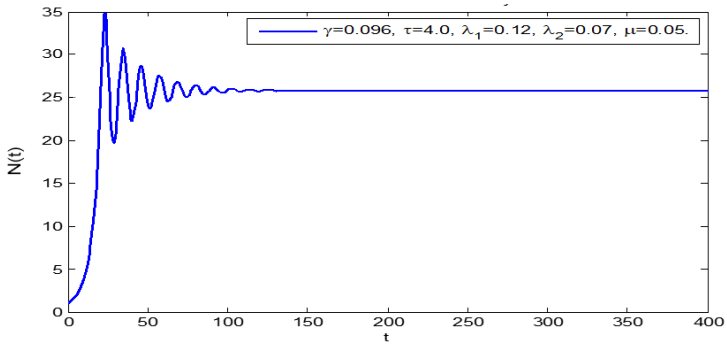

Figure 3. Nonlinear resistance coefficient influence on credit risk contagion at $\mu = 0.05$.

5.2. Time Process Diagrams of Credit Risk in Financial Market on $\lambda_1 < \lambda_2$

In this section, we maintain the parameter values used in Figures 1–3 on the condition that $\lambda_1 = 0.07 < \lambda_2 = 0.12$ and vary the weight γ on the previous activities of the credit risk participant to assess its effects on the credit risk contagion in the financial market as opposed to the assumption $\lambda_1 > \lambda_2$ of Chen et al. [2].

It is observed in Figure 4 that at a time delay of $\tau = 4.0$, on the condition that $\lambda_1 < \lambda_2$, the weight on the previous activities of the credit risk participant was reduced from $\gamma = 0.290$ to $\gamma = 0.149$ to keep the infectious scale at 8.75 compared to Figure 1. This implies that the generalized model (4) is not adherent to the condition $\lambda_1 > \lambda_2$ as claimed in Chen et al. [2].

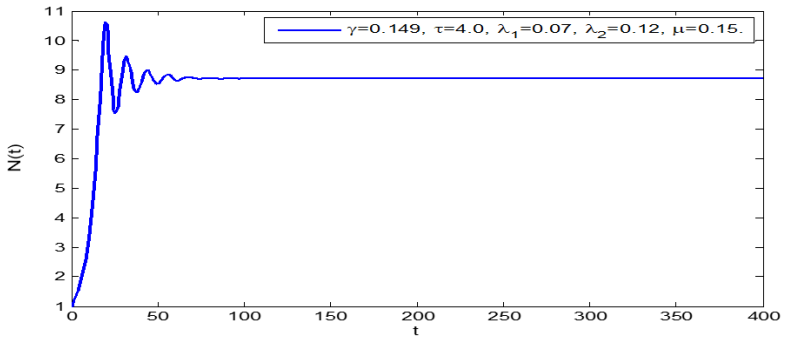

Figure 4. Effects of past credit risk activities on credit risk contagion at $\gamma = 0.149$.

It is observed in Figure 5 that at a nonlinear resistance coefficient of $\mu = 0.08$ and a time delay of $\tau = 4.0$, little initial oscillations were observed as the infectious scale converged to 16.25 on the condition that $\lambda_1 = 0.07 < \lambda_2 = 0.12$, as the weight on past credit risk activities is reduced by fifty one (51%).

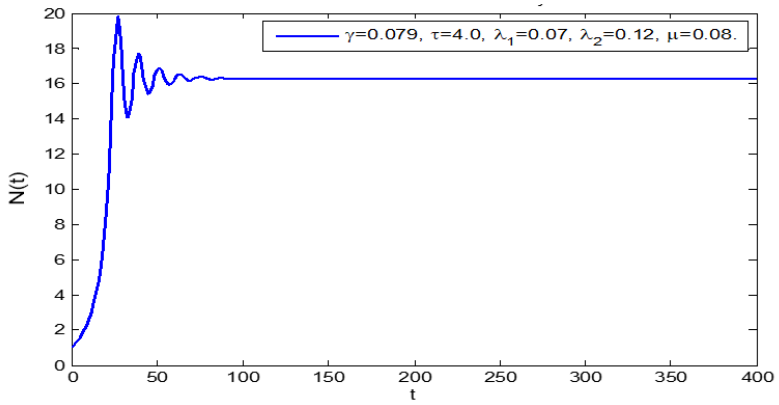

Figure 5. Effects of past credit risk activities on credit risk contagion at $\gamma = 0.079$.

It is indicated in Figure 6 that at a time delay of $\tau = 4.0$, on condition that $\lambda_1 = 0.07 < \lambda_2 = 0.12$, the same rate of credit risk contagion of the participants (refer to Figure 3) can be obtained at a few wavy frequencies if the initial weight on previous credit risk activity is reduced by fifty four percent (54%).

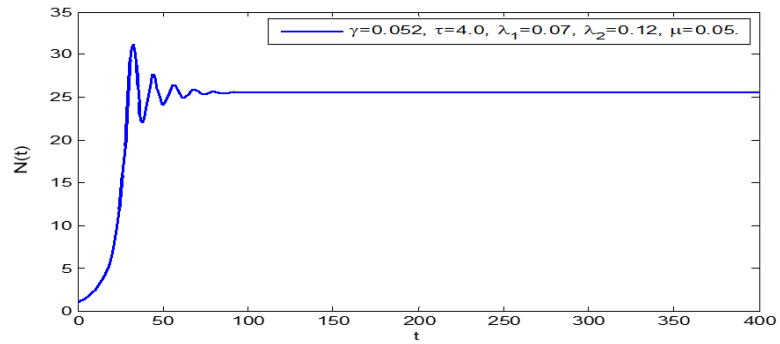

Figure 6. Effects of past credit risk activities on credit risk contagion at $\gamma = 0.052$.

5.3. Real-World Application of the Model

The practical usefulness of model (4) lies in its ability to help financial creditors control the spread of credit risk contagion. This can be achieved by reviewing the weight assigned to past activities of various credit activity participants, regardless of whether they are involved in direct business relationships (λ_1) or indirect business relationships (λ_2).

It is obvious in Figure 7 that when the weight on past activities of the credit risk population is increased from 0.290 to 0.390, the infectious scale of credit risk is reduced to 7.37 from 8.75 compared to Figure 1, even under the restricted condition of $\lambda_1 > \lambda_2$. In all cases, the oscillations of the infectious scale decreased drastically compared to the time process diagrams in Chen et al. [2].

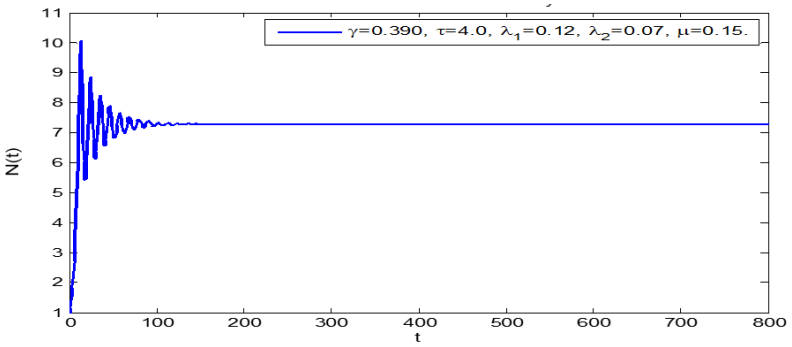

Figure 7. Dynamics of higher weight on contagion rate of credit risk at $\gamma = 0.390$.

6. Conclusions

This study examines the stability of a credit risk contagion model that incorporates distributed delay. The model is a generalized version of the models that were previously examined. The comparative research revealed that, in contrast to the previous models that solely used the nonlinear resistance coefficient to calculate the rate of credit risk infection, the weight placed on the past behaviors of credit risk participants also influences the credit risk contagion rate. This study establishes that existing models perform best when credit risk individuals with direct business relationships have a higher contagion rate than those with indirect business relationships. Nonetheless, regardless of whether the credit risk individuals' contagion rate is higher or lower in direct or indirect business relationships, the distributed delay model can quantify the infection rate. Our model (4) is practically useful as the financial creditors can check the spread of the contagion rate of credit risk by reviewing the weight they put on past activities among different credit activity participants exposed to risk. By doing this, the financial institution can lessen losses brought on by the borrowers' failure to make the agreed-upon payments. As a result, modeling the transmission of credit risk contagion with dispersed delays is advised.

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