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A Compartmental Model for Financial Systemic Risk: Extending an SIRS Model to Capture Mitigation and Protection Dynamics

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Abstract

Financial systemic risk refers to the transmission of distress among financial institutions, posing a significant threat to economic stability. Inspired by epidemiological modelling, this study develops an extended compartmental framework based on the classical SIRS model to analyse the spread and control of financial systemic risk within a banking network. The model introduces six compartments: susceptible, immune, infected, curated, mitigated, and removed to capture the diverse states of banks under systemic stress and regulatory intervention. Central bank actions such as curatorship, mitigation, and temporary protection are explicitly incorporated. The model is formulated as a system of ordinary differential equations, and analytical techniques are employed to derive the risk reproduction number, R_{sr}, which serves as a threshold parameter governing the system's long-term behaviour. Two equilibrium points are identified: the risk-free equilibrium, which is locally and globally asymptotically stable when $R_{sr} < 1$, and the endemic equilibrium, which persists when $R_{sr} > 1$. Numerical simulations demonstrate how variations in key parameters such as the rate of curatorship, mitigation, and protection affect the prevalence of financial contagion. While the model does not yield fundamentally new theoretical insights, it offers a structured framework for evaluating the impact of regulatory interventions. The findings underscore the utility of epidemiological modelling in financial risk analysis and highlight the importance of timely and targeted control measures to prevent cascading failures in the banking sector.

Keywords: Financial systemic risk, Contagion, Risk reproduction number, Model equilibria, Stability, Simulations.

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1. Introduction

Financial systemic risk refers to the "infection" of one financial institution that triggers instability in others, potentially destabilising the entire financial system [1]. By infection, we mean that if one bank experiences distress, the risk is transferred to other banks. This phenomenon has been the subject of considerable empirical research over the past century [2]. Once the banking sector is infected with financial systemic risk, the consequences can be severe, harming both the economy and society at large. Indeed, many banks worldwide face systemic risks that adversely affect economic development and the provision of financial services. Although the intensity of systemic risk varies across institutions, the interconnectivity of the banking sector ensures that the distress of one bank often affects several others. More broadly, systemic risk is a multifaceted phenomenon encompassing social, economic, political, and cultural dimensions, which makes its elimination extremely challenging [3].

Financial systemic risk has historically contributed to the collapse of banking sectors, often resulting in poverty for large segments of the population [4]. It manifests in different forms, including: (i) panic-driven crises arising from multiple equilibria; (ii) crises triggered by sharp declines in asset prices; (iii) contagion effects, whether systematic or idiosyncratic; and (iv) foreign exchange distortions within the banking system. To evaluate such risks, several methodologies have been developed, including tail risk measures, contingent claims analysis, network models, and dynamic stochastic macroeconomic models [5–10]. In recent decades, mathematical models have proven to be powerful tools for assessing the implications of financial systemic risk on economic stability and development [11].

The spread of financial systemic risk has often been likened to the spread of infectious diseases in epidemiology [12]. This analogy has motivated the application of mathematical epidemiological modelling to investigate the dynamic behaviour of systemic risk, which has the potential to undermine the stability of the financial system. In epidemiology, remedial measures such as treatment are typically aimed at slowing or halting disease progression, with the ultimate goal of eradication.

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In a similar way, interventions in the financial market are intended to control the spread and impact of systemic risk.

Several studies have applied epidemic models to analyse systemic risk contagion in financial markets [13–15]. For example, when systemic risk emerges, central banks often provide emergency funding to at-risk banks as a containment measure [16, 17]. In these cases, the classical "SIR" model of Kermack and McKendrick (1927) has been employed, with compartments representing susceptible (S), infected (I), and removed (R) institutions. While this model captures basic contagion dynamics, it does not account for additional interventions. More recent work [18] has combined epidemic models with complex network theory, extending the analysis to the "SIRS" model to study the spread of credit risk among institutions. Building on this, we propose further improvements by incorporating mitigation and protection strategies for banks, analogous to vaccination and hospitalisation in epidemiological models. These extensions are critical, as they reflect real-world interventions used by regulators and central banks to stabilise the financial system.

This paper aims to develop and analyse a novel epidemiological-inspired model for financial systemic risk that explicitly incorporates intervention strategies such as mitigation (central bank funding support) and protection (regulatory safeguards). Our objectives are threefold: (i) to formulate a theoretical model capturing the dynamics of financial systemic risk under remedial interventions; (ii) to derive the threshold parameter, termed the risk reproduction number (R_{sr}), which determines whether systemic risk persists or dies out; and (iii) to use numerical simulations to illustrate the impact of different intervention strategies on systemic stability.

The structure of the paper is as follows. In Section 2, we present the formulation of the theoretical model. Section 3 is devoted to the mathematical analysis of the model. In Section 4, we derive the risk reproduction number (R_{sr}). Section 5 provides numerical simulations of the model using Matlab, while Section 6 concludes with a discussion of the findings and their implications.

2. Model formulation

The model is formulated by categorizing the banks' population $N(t)$ into six compartments, namely, susceptible banks $S(t)$, which are banks that have never been infected by financial systemic risk; immune banks $V(t)$, banks that are protected from financial systemic risk irrespective of the circumstances around them; curated $C(t)$, banks which have liquidated for a specific period of time during which they cannot be infected with financial systemic risk and infect others, financial systemic risk infected banks $I(t)$, these are banks which are infected with financial systemic risk and are capable of influencing a susceptible bank to be infected with financial systemic risk, Mitigated banks $M(t)$, banks that obtained financial relief from the central bank, in order to remedy financial systemic risk and the removed banks

$R(t)$, banks that are ex-financial systemic risky which have reformed during the curatorship period and can still be infected by those banks with financial systemic risk.
The total bank population at any time t is given by

$$N(t) = S(t) + V(t) + I(t) + C(t) + M(t) + R(t).$$

Referring to the flow diagram in Figure 1, the class of the immune banks is

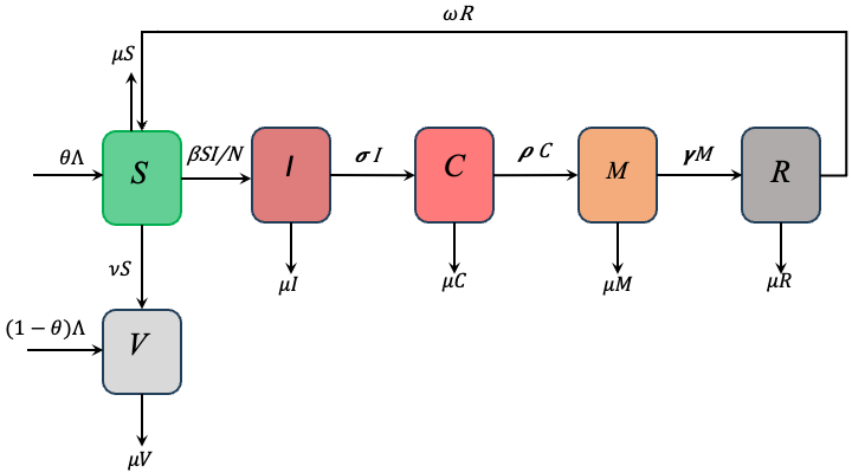


Figure 1: The flow diagram of our model that depicts the flow of banks as their status changes as a result of 'infection'.

generated from periodical recruitment of banks without the financial systemic risk at a rate $(1-\theta)\Lambda$, while those prone to infection with financial systemic risk progress to the susceptible compartment at a rate $\theta\Lambda$.
Susceptible banks become infected by infected banks and become infectious at a rate β, while financially systemic risky banks are curated at a rate σ. Curated and financially systemic risky banks in the removed category become susceptible after some time at a rate ω, while susceptible banks prone to financial systemic risk become immune due to central bank intervention. In the model, ρ is the rate at which banks are mitigated, and ν is the protection rate of the susceptible banks S. Banks under mitigation are allowed to recover at a rate γ. All classes are subject to collapse at a rate μ, that leads to removal from the financial system. In light of the above assumptions, extending the model in [18], the resultant system of ordinary differential equations is given by:

$$\frac{dS}{dt} = \theta\Lambda + \omega R - \frac{\beta SI}{N} - (\mu + \nu)S, \tag{1}$$

$$\frac{dV}{dt} = (1-\theta)\Lambda + \nu S - \mu V, \tag{2}$$

$$\frac{dI}{dt} = \frac{\beta SI}{N} - (\mu + \sigma)I, \tag{3}$$
$$\frac{dC}{dt} = \sigma I - (\mu + \rho)C, \tag{4}$$
$$\frac{dM}{dt} = \rho C - (\mu + \gamma)M, \tag{5}$$
$$\frac{dR}{dt} = \gamma M - (\mu + \omega)R, \tag{6}$$

subject to the initial conditions

$$S(0) = S_0 > 0, V(0) = V_0 \geq 0, I(0) = I_0 \geq 0, C(0) = C_0 \geq 0, M(0) = M_0 \geq 0, R(0) = R_0 \geq 0.$$

Equation (1) describes the dynamics of financial institutions that are susceptible to financial systemic risk. Equation (2) describes the dynamics of banks protected from financial systemic risk. Equation (3) describes the changes of banks that are impacted by financial systemic risk and impact susceptible banks when there is contagion. Equation (4) represents the dynamics of curated banks before any mitigation. Equation (5) describes the changes over time for banks that are under some intervention. The last equation, Equation (6), describes the changes over time for banks that recover from the financial systemic risk. The parameters and their hypothetical values are described in Table 1.

Table 1: Description of model parameters and their assumed values.

Parameter	**Description**	**Value**
θ	Proportion of new banks recruited into S.	0.9
Λ	Recruitment rate.	0.5
ν	Rate at which susceptible banks become immune.	0.001
ω	Rate at which removed banks return to S.	0.01
β	Infection (contagion) rate.	0.65
σ	Rate at which infected banks are curated.	0.08
ρ	Rate of mitigation by the central bank.	0.6
μ	Collapse rate (bank lifespan).	0.006
γ	Recovery rate of mitigated banks.	0.8

3. Analysis of the model

Positivity of solutions

Theorem 3.1. *Given the non-negative initial conditions*

$$S(0) > 0, \quad V(0) \geq 0, \quad I(0) \geq 0, \quad C(0) \geq 0, \quad M(0) \geq 0, \quad R(0) \geq 0,$$

the solutions $S(t), V(t), I(t), C(t), M(t), R(t)$ *of the system remain non-negative for all* $t \geq 0$.

Proof. We analyze each equation of the system using differential inequalities and separation of variables. Starting with Equation (1), observe that at $S = 0$:

$$\left.\frac{dS}{dt}\right|_{S=0} = \theta\Lambda + \omega R \geq 0.$$

Hence, the flow into the susceptible compartment is non-negative at the boundary. We derive the following differential inequality:

$$\frac{dS}{dt} \geq -(\mu + \nu)S \quad \Rightarrow \quad \frac{dS}{S} \geq -(\mu + \nu)\, dt.$$

Integrating both sides yields:

$$S(t) \geq S(0)e^{-(\mu+\nu)t} \geq 0.$$

From Equation (2), at $V = 0$, we have:

$$\left.\frac{dV}{dt}\right|_{V=0} = (1 - \theta)\Lambda + \nu S \geq 0.$$

Similarly, we obtain:

$$\frac{dV}{dt} \geq -\mu V \quad \Rightarrow \quad \frac{dV}{V} \geq -\mu\, dt,$$

which, upon integration, gives:

$$V(t) \geq V(0)e^{-\mu t} \geq 0.$$

Applying the same procedure to Equations (3)–(6) yields:

$$\begin{aligned} I(t) &\geq I(0)e^{-(\mu+\sigma)t} \geq 0, \\ C(t) &\geq C(0)e^{-(\mu+\rho)t} \geq 0, \\ M(t) &\geq M(0)e^{-(\mu+\gamma)t} \geq 0, \\ R(t) &\geq R(0)e^{-(\mu+\omega)t} \geq 0. \end{aligned}$$

Since each compartment satisfies a non-negative exponential lower bound and the vector field points inward on the boundary of the non-negative cone, it follows that:

$$S(t), V(t), I(t), C(t), M(t), R(t) \geq 0 \quad \text{for all } t \geq 0.$$

This establishes the positivity of the solutions. □

Feasible region

Define the region

$$\Omega = \left\{ (S, V, I, C, M, R) \in \mathbb{R}_+^6 : N = S + V + I + C + M + R \leq \frac{\Lambda}{\mu} \right\}.$$

We now show that Ω is positively invariant and attracts all feasible solutions of the system (1)–(6). The rate of change of the total population is given by:

$$\frac{dN}{dt} = \Lambda - \mu N.$$

Solving this linear ODE using an integrating factor yields:

$$N(t) = \frac{\Lambda}{\mu} + \left(N_0 - \frac{\Lambda}{\mu} \right) e^{-\mu t},$$

where $N(0) = N_0$. By the standard comparison theorem, it follows that:

$$N(t) \leq \frac{\Lambda}{\mu} \quad \text{if} \quad N_0 \leq \frac{\Lambda}{\mu}.$$

Thus, Ω is positively invariant. Moreover, all solutions with non-negative initial conditions eventually enter and remain in Ω. Therefore, the model is mathematically and epidemiologically well-posed in Ω.

Model steady states

Financial systemic risk-free equilibrium (RFE)

Setting the right-hand sides of (1)–(6) to zero and assuming $I = 0$, we find that $C = M = R = 0$. Solving for S and V gives:

$$S^0 = \frac{\theta\Lambda}{\mu + \nu}, \quad V^0 = \frac{\Lambda[(1-\theta)\mu + \nu]}{\mu(\mu + \nu)}.$$

Thus, the risk-free equilibrium (RFE) is:

$$E_0 = (S^0, V^0, 0, 0, 0, 0) = \left(\frac{\theta\Lambda}{\mu + \nu}, \frac{\Lambda[(1-\theta)\mu + \nu]}{\mu(\mu + \nu)}, 0, 0, 0, 0 \right),$$

with total population $N^0 = S^0 + V^0 = \frac{\Lambda}{\mu}$.

4. Risk reproduction number ($\mathcal{R}_{sr}$)

In analogy with epidemiological models, the risk reproduction number, denoted by R_{sr}, represents the expected number of secondary financially distressed banks

generated by a single infected bank introduced into an otherwise risk–free financial system. This threshold quantity determines whether financial systemic risk dies out or persists in the banking sector.
To derive R_{sr}, we employ the next–generation matrix approach [19, 20]. Let the vector of infected–related compartments be

$$X = (I, C, M, R)^T,$$

since only these classes are directly associated with the propagation or resolution of financial systemic risk. The system can be written in the form

$$\frac{dX}{dt} = \mathcal{F}(X) - \mathcal{V}(X),$$

where $\mathcal{F}(X)$ represents the rate of appearance of new infections, and $\mathcal{V}(X)$ denotes the transition terms among infected compartments and removals. The new infection terms are given by

$$\mathcal{F}(X) = \begin{pmatrix} \frac{\beta SI}{N} \\ 0 \\ 0 \\ 0 \end{pmatrix},$$

while the remaining transfer terms are

$$\mathcal{V}(X) = \begin{pmatrix} (\mu+\sigma)I \\ -\sigma I + (\mu+\rho)C \\ -\rho C + (\mu+\gamma)M \\ -\gamma M + (\mu+\omega)R \end{pmatrix}.$$

Evaluating the Jacobian matrices of $\mathcal{F}$ and $\mathcal{V}$ at the financial systemic risk–free equilibrium

$$E_0 = (S_0, V_0, 0, 0, 0, 0),$$

yields

$$F = \begin{pmatrix} 0 & \frac{\beta S_0}{N_0} & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \qquad V = \begin{pmatrix} \mu+\sigma & 0 & 0 & 0 \\ -\sigma & \mu+\rho & 0 & 0 \\ 0 & -\rho & \mu+\gamma & 0 \\ 0 & 0 & -\gamma & \mu+\omega \end{pmatrix}.$$

The risk reproduction number is defined as the spectral radius of the matrix FV^{-1}, that is,

$$R_{sr} = \rho(FV^{-1}).$$

A direct computation yields

$$R_{sr} = \frac{\beta S_0}{N_0(\mu + \sigma)}. \tag{7}$$

Substituting

$$S_0 = \frac{\theta\Lambda}{\mu + \nu} \quad \text{and} \quad N_0 = \frac{\Lambda}{\mu},$$

Equation (7) simplifies to

$$R_{sr} = \frac{\beta\theta\mu}{(\mu + \nu)(\mu + \sigma)}. \tag{8}$$

Expression (8) shows explicitly how regulatory interventions influence systemic risk transmission. In particular, increasing the curatorship rate σ or the protection rate ν reduces R_{sr}, thereby lowering the likelihood of sustained financial contagion.

Theorem 4.1. *The financial systemic risk–free equilibrium E_0 is locally asymptotically stable if $R_{sr} < 1$, and unstable if $R_{sr} > 1$.*

Proof. The result follows directly from the next–generation matrix theory [19]. When $R_{sr} < 1$, all eigenvalues of FV^{-1} have modulus less than unity, implying that perturbations away from E_0 decay over time. Conversely, if $R_{sr} > 1$, at least one eigenvalue exceeds unity, leading to the growth of financial systemic risk. □

The quantity R_{sr} therefore serves as a fundamental threshold parameter for the model, separating regimes of financial stability from those in which systemic risk persists.

Theorem 4.2. *The risk-free equilibrium E_0 is globally asymptotically stable in Ω if $\mathcal{R}_{sr} < 1$.*

Proof. Consider the Lyapunov function:

$$L = I.$$

Differentiating L with respect to time gives:

$$\frac{dL}{dt} = \frac{\beta SI}{N} - (\mu + \sigma)I.$$

Since $S \leq N \leq \frac{\Lambda}{\mu}$ and $S^0 = \frac{\theta\Lambda}{\mu+\nu}$, we have:

$$\frac{dL}{dt} \leq (\mu + \sigma)(\mathcal{R}_{sr} - 1)I.$$

Hence, $\frac{dL}{dt} \leq 0$ when $\mathcal{R}_{sr} \leq 1$, and $\frac{dL}{dt} = 0$ only when $I = 0$. By LaSalle's invariance principle [21], all solutions approach the largest invariant set where $I = 0$, which is E_0. Therefore, E_0 is globally asymptotically stable. □

Existence of the financial systemic risk endemic equilibrium

To find the endemic equilibrium, set the derivatives in (1)–(6) to zero and assume $I > 0$. From (3), we obtain:

$$\frac{S}{N} = \frac{\mu + \sigma}{\beta}.$$

Solving the system, we express all variables in terms of I. The endemic equilibrium exists if and only if $\mathcal{R}_{sr} > 1$, and is given by:

$$E_1 = (S^*, V^*, I^*, C^*, M^*, R^*),$$

where

$$\begin{aligned}
I^* &= Q(\mathcal{R}_{sr} - 1), \\
C^* &= \psi_1 I^*, \\
M^* &= \psi_2 I^*, \\
R^* &= \psi_3 I^*, \\
S^* &= \frac{\theta\Lambda + (\omega\psi_3 - \mu - \sigma)I^*}{\mu + \nu}, \\
V^* &= \frac{(1-\theta)\Lambda + \nu S^*}{\mu},
\end{aligned}$$

with

$$Q = \frac{\Lambda(\mu+\nu)(\mu+\sigma)(\mu+\rho)(\mu+\gamma)(\mu+\omega)}{\beta\mu^2\left[(\mu+\rho)(\mu+\sigma)(\mu+\omega) + \gamma(\mu+\rho)(\mu+\sigma) + \gamma\rho\omega\right]}.$$

Theorem 4.3. *The endemic equilibrium E_1 exists if and only if $\mathcal{R}_{sr} > 1$.*

Global stability of the endemic equilibrium

We now establish the global asymptotic stability of the endemic equilibrium of the system (1)–(6) under the following simplifying assumptions: the total population size N is constant, i.e., $\Lambda = \mu N$, and there is no waning immunity, i.e., $\omega = 0$. Under these assumptions, the model equations reduce to:

$$\begin{aligned}
\frac{dS}{dt} &= \theta\mu N - \frac{\beta SI}{N} - (\mu + \nu)S, \\
\frac{dV}{dt} &= (1-\theta)\mu N + \nu S - \mu V, \\
\frac{dI}{dt} &= \frac{\beta SI}{N} - (\mu + \sigma)I, \\
\frac{dC}{dt} &= \sigma I - (\mu + \rho)C, \\
\frac{dM}{dt} &= \rho C - (\mu + \gamma)M,
\end{aligned}$$

$$\frac{dR}{dt} = \gamma M - \mu R.$$

Theorem 4.4. *Consider the reduced system under the above assumptions. Then, the endemic equilibrium* $E_1 = (S^*, V^*, I^*, C^*, M^*, R^*)$ *is globally asymptotically stable in the interior of the feasible region* $\{\mathbf{x} > 0\}$.

Let E_1 be the endemic equilibrium of the system, which exists when $\mathcal{R}_{sr} > 1$. We construct the following Lyapunov function:

$$\mathcal{L} = \sum_{x \in \{S,V,I,C,M,R\}} a_x \left(\frac{x}{x^*} - 1 - \ln \frac{x}{x^*} \right),$$

where $a_x > 0$ are constants to be determined. This function is non-negative and equals zero if and only if $x = x^*$ for all compartments. It satisfies $\mathcal{L}(x^*) = 0$, $\mathcal{L}(x) > 0$ for $x \neq x^*$, and is convex in each variable. Differentiating $\mathcal{L}$ along the solutions of the system yields:

$$\dot{\mathcal{L}} = \sum_{x \in \{S,V,I,C,M,R\}} a_x \left(1 - \frac{x^*}{x} \right) \cdot \frac{1}{x^*} \cdot \frac{dx}{dt}.$$

Using the equilibrium conditions:

$$\begin{aligned}
\theta \mu N &= \frac{\beta S^* I^*}{N} + (\mu + \nu) S^*, \\
(1 - \theta) \mu N &= \nu S^* + \mu V^*, \\
\frac{\beta}{N} &= (\mu + \sigma) \frac{I^*}{S^*}, \\
\mu + \rho &= \frac{\sigma I^*}{C^*}, \\
\mu + \gamma &= \frac{\rho C^*}{M^*}, \\
\mu &= \frac{\gamma M^*}{R^*}.
\end{aligned}$$

Define the Lyapunov function:

$$\mathcal{L} = \frac{S}{S^*} - 1 - \ln \left(\frac{S}{S^*} \right) + \sum_{x \in \{V,I,C,M,R\}} a_x \left(\frac{x}{x^*} - 1 - \ln \frac{x}{x^*} \right),$$

and its time derivative:

$$\dot{\mathcal{L}} = -\frac{(\mu + \nu)}{S^*} \left(\frac{(S - S^*)^2}{S} \right) - \frac{\psi a_V}{V^*} (V - V^*) + f(S, V, I, C, M, R),$$

where we aim to show that $f(S, V, I, C, M, R) \leq 0$. Let

$$u = \frac{S}{S^*}, \quad v = \frac{V}{V^*}, \quad w = \frac{I}{I^*}, \quad x = \frac{C}{C^*}, \quad y = \frac{M}{M^*}, \quad z = \frac{R}{R^*},$$

then

$$\begin{aligned} f(u,v,w,x,y,z) = \frac{\beta I^*}{N}\left(1-\frac{1}{u}-uw+w+a_V\nu\frac{S^*}{V^*}\right)\left(u+\frac{u}{v}+\frac{1}{v}-1\right) \\ +(u+\sigma)a_I(uw-w-u+1) \\ +a_C\sigma\frac{I^*}{C^*}\left(w-x-\frac{w}{x}+1\right) \\ +\rho\frac{C^*}{M^*}\left(x-y-\frac{x}{y}+1\right) \\ +\gamma\frac{M^*}{R^*}a_R\left(y-z-\frac{y}{z}+1\right) \\ +\frac{I^*}{R^*}a_R\left(w-z-\frac{w}{z}+1\right). \end{aligned}$$

After simplification, we obtain:

$$a_V=\frac{\beta V^*I^*}{\nu NS^*},\quad a_I=\frac{\beta I^*}{(\mu+\sigma)N},\quad a_C=a_M=a_R=0,$$

which reduces the expression to:

$$f(u,v,w,x,y,z)=\frac{\beta I^*}{N}\left(1-\frac{1}{u}-\frac{u}{v}+\frac{1}{v}\right).$$

Using the inequality $1-k+\ln k\le 0$, we find:

$$\begin{aligned} 1-\frac{1}{u}-\frac{u}{v}+\frac{1}{v} &= \left(1-\frac{u}{v}\right)+\left(1-\frac{1}{u}\right)-\left(1-\frac{1}{v}\right)\le -\ln\left(\frac{u}{v}\right)-\ln\left(\frac{1}{u}\right)+\ln\left(\frac{1}{v}\right) \\ = \quad \ln\left(\frac{v}{u}\cdot u\cdot\frac{1}{v}\right) &= \nu. \end{aligned}$$

Thus,

$$\dot{\mathcal{L}}\le -\frac{(\mu+\nu)}{S^*}\left(\frac{(S-S^*)^2}{S}\right)-\frac{\mu\beta I^*}{\nu NS^*}\left(\frac{(V-V^*)^2}{V}\right).$$

Therefore, $\dot{\mathcal{L}}\le 0$, and $\dot{\mathcal{L}}=0$ if and only if $S=S^*, V=V^*, I=I^*, C=C^*, M=M^*, R=R^*$. By LaSalle's invariance principle, the omega-limit set of the solution is an invariant set contained in

$$\Omega=\{(S,V,I,C,M,R): S=S^*, V=V^*, I=I^*, C=C^*, M=M^*, R=R^*\}.$$

Thus, the only invariant set in Ω is the singleton $\{E_1\}$, which implies that every solution in $\mathbb{R}^6_+$ approaches the endemic equilibrium. This completes the proof.

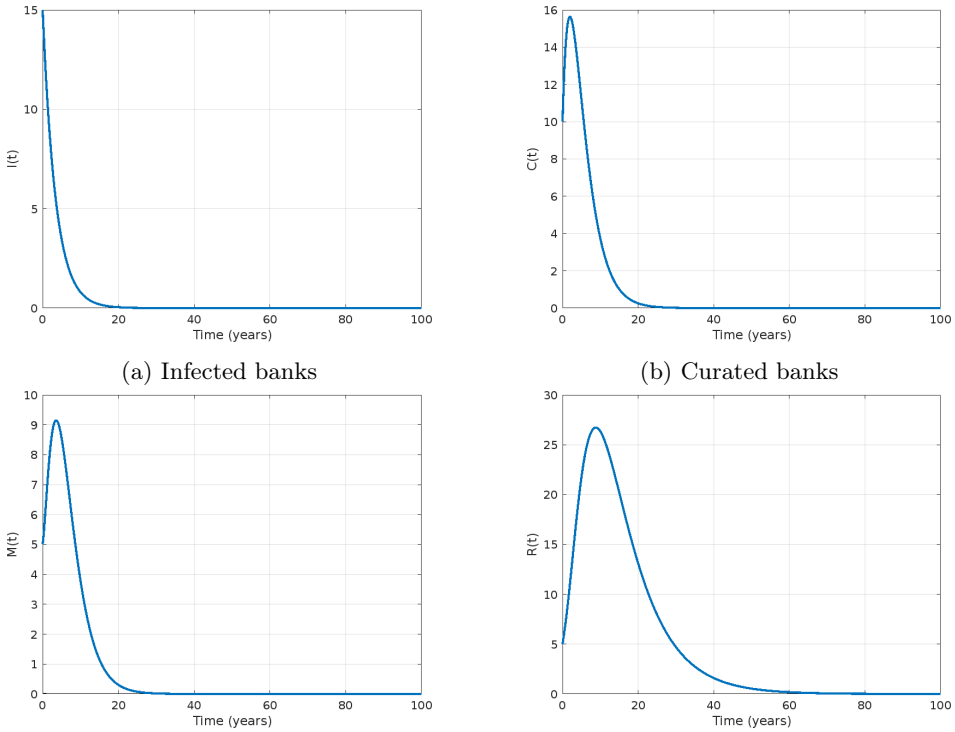
5. Simulations

Time series plots

We perform numerical simulations using the following initial conditions:

$$S(0) = 945, \quad V(0) = 20, \quad I(0) = 15, \quad C(0) = 10, \quad M(0) = 5, \quad R(0) = 5,$$

for a hypothetical bank population of $N = 1000$. Figure 2 (a) shows the trajectory



(a) Infected banks

(b) Curated banks

(c) Mitigated banks

(d) Recovered banks

Figure 2: Time series plots for selected state variables at the financial risk-free equilibrium. Parameter values used: $\theta = 0.9$, $\Lambda = 0.5$, $\omega = 0.1$, $\beta = 0.5$, $\mu = 0.009$, $\nu = 0.0009$, $\sigma = 0.75$, $\rho = 0.4$, $\gamma = 0.6$.

of infected banks over time. Starting with 15 infected banks, the number declines sharply and reaches zero in less than 20 years. Figure 2 (b) illustrates the dynamics of curated banks, which initially increase to approximately 15 within 6 years before declining to zero shortly after 20 years. A similar pattern is observed in Figure 2 (c) for mitigated banks. In contrast, Figure 2 (d) shows that the number of recovered banks gradually declines to zero between 55 and 60 years.

Impact of varying key parameters

Figure 3 presents simulations of infected banks under varying values of σ, the rate at which infected banks are curated, while keeping all other parameters constant. Initially, all curves follow a similar trajectory. For smaller values of σ, the infected population peaks higher due to slower removal. As σ increases, the peak diminishes, indicating more effective curatorship. After peaking, all curves exhibit exponential decay. Additionally, reducing the infection rate β in equation (3) further lowers the number of infected banks. Figure 4 explores the effect of varying ν,

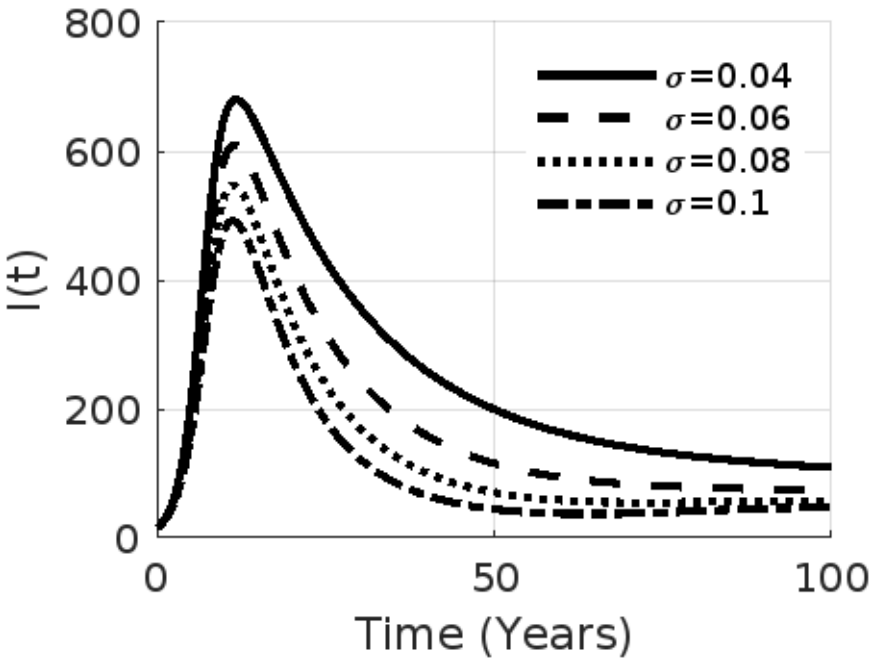


Figure 3: Impact of varying σ on the infected banks compartment. Parameter values: $\theta = 0.9,\ \Lambda = 0.5,\ \omega = 0.01,\ \beta = 0.65,\ \mu = 0.006,\ \nu = 0.001,\ \rho = 0.6,\ \gamma = 0.8$.

the rate at which banks become immune. The simulation results are qualitatively similar to those in Figure 3, with differences primarily in the peak values. This suggests that both curatorship (σ) and immunity (ν) play comparable roles in reducing the infected population. Figure 5 shows the effect of varying ρ, the rate of mitigation. All curves behave similarly up to the peak, but diverge thereafter. Around the 20-year mark, differences in decay rates become apparent, indicating that higher mitigation rates accelerate the decline of infected banks.

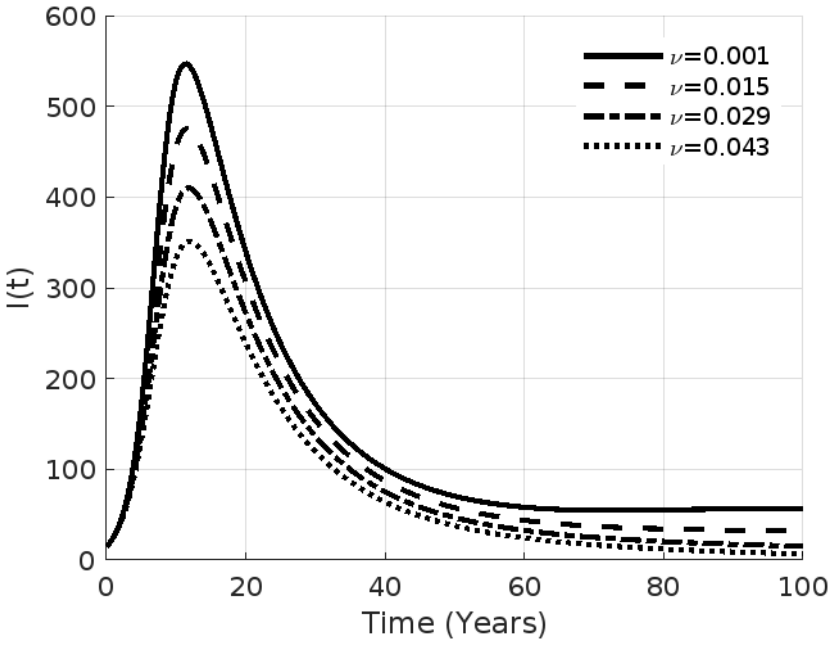


Figure 4: Impact of varying ν on the infected banks compartment. Parameter values: $\theta = 0.9,\ \Lambda = 0.5,\ \omega = 0.01,\ \beta = 0.65,\ \mu = 0.006,\ \sigma = 0.08,\ \rho = 0.6,\ \gamma = 0.8$.

Contour plots

Contour plots are useful for visualizing the relationship between a response variable and two independent variables. Typically, the independent variables are represented on the x- and y-axes, while the response variable is depicted through contour levels.

In Figure 6, the independent variables are θ and μ. As μ decreases, the contours become more closely spaced, indicating a rapid change in the reproduction number. For larger values of μ, the contours spread out, suggesting slower variation. Figure 7 illustrates the effect of σ and ν on the reproduction number. Initially, the level curves are tightly packed, but they become more spaced as the values of σ and ν increase.

In the context of scalar fields, contour lines represent loci of constant function values, meaning that every point along a given contour corresponds to the same output of the underlying function. Importantly, the visual spacing between contour lines does not necessarily reflect uniform changes in function values; equally spaced contour values may yield unevenly spaced contours depending on the gradient of the field. Regions where contour lines are closely packed indicate steep gradients or rapid variation in the function, whereas widely spaced contours denote areas

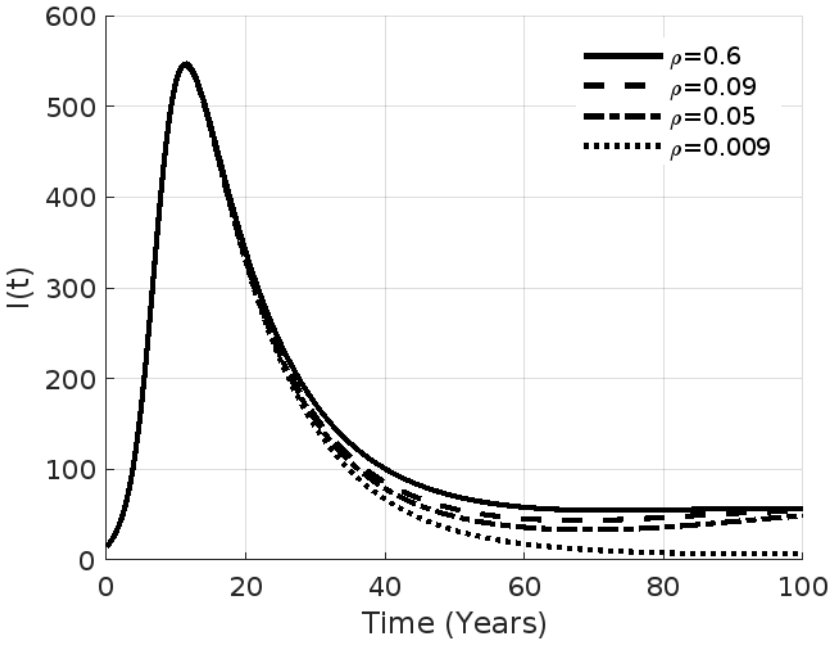


Figure 5: Impact of varying ρ on the infected banks compartment. Parameter values: $\theta = 0.9$, $\Lambda = 0.5$, $\omega = 0.01$, $\beta = 0.65$, $\mu = 0.006$, $\nu = 0.001$, $\sigma = 0.08$, $\gamma = 0.8$.

of gradual change. Collectively, the set of contour values delineates the functional range across the domain.

Sensitivity analysis

Table 2 presents the classification of correlation coefficients used to interpret the scatter plots in Figure 8.

Figure 8 illustrates the relationship between selected model parameters and the infected banks (I). Based on Table 2, the following observations are made:

- The first plot shows a weak positive correlation ($r = 0.12$) between the recruitment rate Λ and the infected banks I, indicating that recruitment is not strongly linked to infection.
- The second plot shows a weak negative correlation between the infected banks I and their lifespan μ, suggesting that infection does not necessarily lead to bank extinction.
- The third plot reveals a very strong positive correlation ($r = 0.94$) between the infected banks I and the rate ω at which recovered banks become sus-

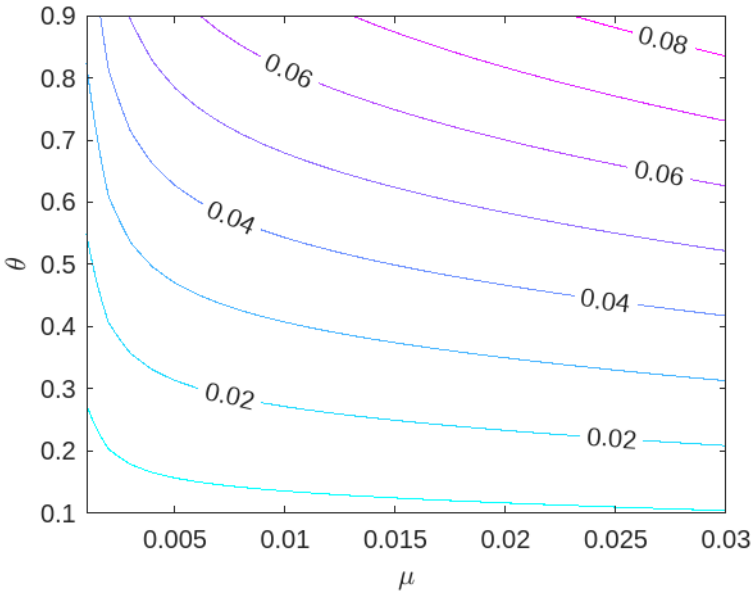


Figure 6: Contour plot showing the impact of parameters μ (bank lifespan) and θ (proportion recruited into the susceptible class) on the reproduction number. Other parameter values are: $\Lambda = 0.5$, $\omega = 0.01$, $\beta = 0.9$, $\nu = 0.001$, $\sigma = 0.08$, $\rho = 0.6$, $\gamma = 0.8$.

Table 2: Correlation coefficient ranges and their interpretation for the scatter plots in Figure 8.

Correlation Coefficient	**Correlation Strength**	**Correlation Type**
-0.7 to -1	Very Strong	Negative
-0.5 to -0.7	Strong	Negative
0 to -0.3	Weak	Negative
0 to 0.3	Weak	Positive
0.3 to 0.5	Moderate	Positive
0.5 to 0.7	Strong	Positive
0.7 to 1	Very Strong	Positive

ceptible. This reflects the realistic scenario where banks do not recover with permanent immunity.

- The final plot shows a weak negative correlation ($r = -0.15$) between the infected banks I and the curation rate σ, possibly influenced by the lifespan parameter μ.

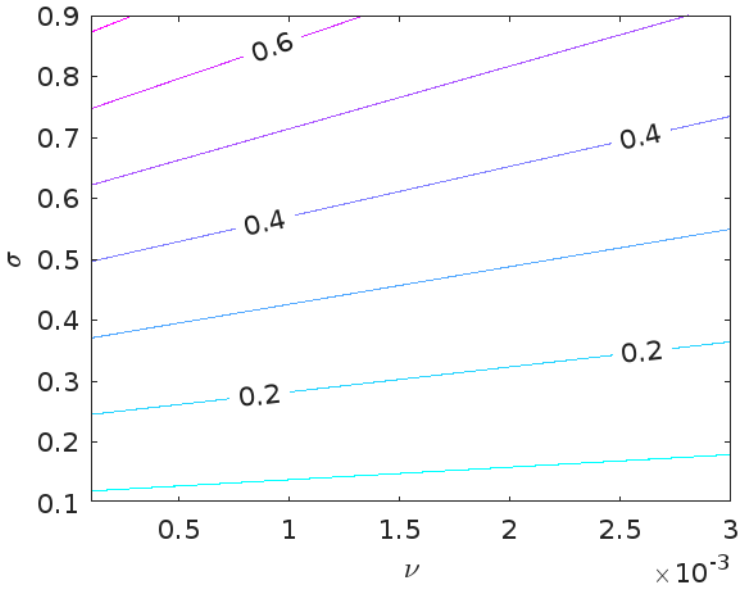
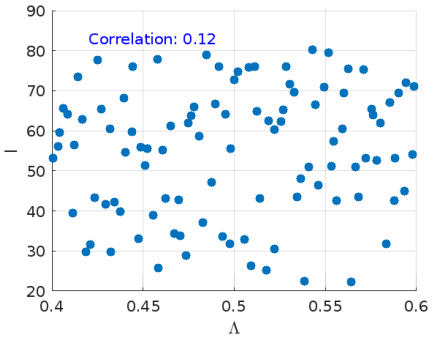


Figure 7: Contour plot showing the impact of parameters σ (curation rate) and ν (immunity rate) on the reproduction number. Other parameter values are: $\theta = 0.9,\ \Lambda = 0.5,\ \omega = 0.01,\ \beta = 0.9,\ \mu = 0.006,\ \rho = 0.6,\ \gamma = 0.8$.

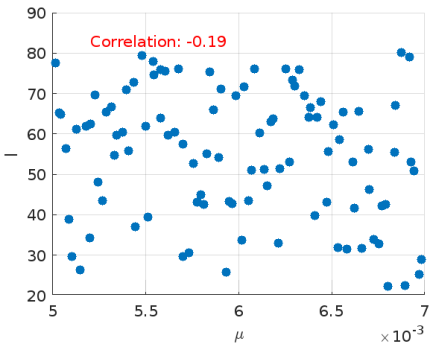
6. Discussion and conclusion

This study introduces a novel compartmental model inspired by infectious disease dynamics to analyse the spread and control of financial systemic risk among banking institutions. By incorporating six distinct compartments, these are susceptible, immune, infected, curated, mitigated, and removed. The model captures the complexity and heterogeneity of real-world financial contagion processes, extending previous models in the literature. The analysis reveals the existence of two key equilibrium points. The model's threshold parameter, the risk reproduction number $\mathcal{R}_{sr}$, governs the long-term behaviour of the system. When $\mathcal{R}_{sr} < 1$, the financial system is predicted to stabilise without persistent fiancial systemic risk. Conversely, when $\mathcal{R}_{sr} > 1$, financial contagion is expected to persist among banking institutions. These theoretical insights are supported by numerical simulations, which demonstrate the dynamic impact of various parameters on the system's trajectory.

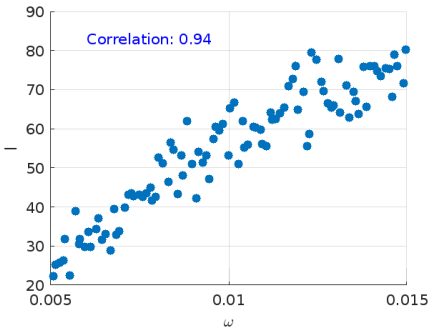
From a policy perspective, the model highlights the importance of timely interventions, specifically, curatorship (similar to quarantine), mitigation (comparable to treatment), and protection (analogous to vaccination in epidemiological models). Simulation results suggest that increasing the rates of mitigation and protection significantly reduces the number of infected financial institutions, thereby



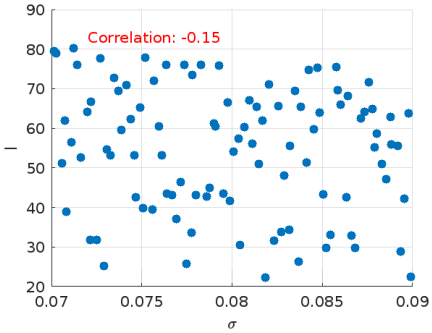
(A) Recruitment rate Λ vs. infected banks.



(B) Lifespan μ vs. infected banks.



(C) Susceptibility rate ω vs. infected banks.



(D) Curation rate σ vs. infected banks.

Figure 8: Scatter plots showing correlations between selected parameters and the infected banks compartment.

lowering the overall burden of financial systemic risk. These findings offer a quantitative foundation for regulatory strategies aimed at stabilising the financial sector.

This work contributes to the growing body of literature on financial contagion by enhancing the epidemiological modelling framework with greater granularity in state transitions and remedial actions. Future research may explore extensions such as stochastic effects, network-based interactions, or delay differential equations to account for latent risks and time-dependent interventions. Additionally, empirical calibration using real banking data would improve the model's predictive accuracy and practical relevance.

In conclusion, the extended SIRS model developed in this study provides valuable insights into the dynamics of financial systemic risk and underscores the critical role of proactive control measures. The mathematical framework presented here offers a foundation for future interdisciplinary research at the intersection of

financial economics and epidemiological modelling.

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Conflicts of Interest. The authors declare that there is no conflict of interest concerning the publication of this article.

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