Available online at http://scik.org
Commun. Math. Biol. Neurosci. 2026, 2026:46
https://doi.org/10.28919/cmbn/9889
ISSN: 2052-2541

A PROBABILISTIC AND GRAPH-THEORETIC APPROACH TO THE ANALYSIS OF CREDIT AND LIQUIDITY RISK CONTAGION WITH DELAY EFFECTS IN A BANKING SYSTEM

RACHID EL IBRAQUI[1,*], MOULAY OTHMAN ABOUTAFAIL[1], REDOUANE NOUIRA[2], HAMZA MOURAD[3]

[1]Ibn Tofail University, Kénitra, Morocco

[2]CRMEF Kénitra, Morocco

[3]Graduate High School of Biomedical Engineering and Health Techniques- SUPTECH SANTE, Morocco

Abstract. This paper proposes a probabilistic interpretation of a delayed banking contagion model driven by the interaction between credit risk and liquidity risk. Instead of restricting the analysis to a local study around an equilibrium, the banking system is represented by a weighted directed graph whose vertices are banks and whose edges describe bilateral exposures. This framework captures bank heterogeneity, exposure intensity, network structure, and delayed transmission effects in a unified way. Each institution evolves among four financial states: healthy, exposed to credit risk, exposed to liquidity risk, and defaulted. Transitions between these states are described by probabilities depending on neighborhood conditions, exposure weights, and delay parameters. We further introduce structural indicators, including centrality measures and a synthetic systemic vulnerability index, to identify the institutions most likely to amplify contagion. Numerical simulations show how small changes in transmission and recovery mechanisms may shift the system from a contained stress regime to a generalized crisis.

Keywords: probability; graph theory; interbank network; credit risk; liquidity risk; financial contagion; delay; systemic stability.

2020 AMS Subject Classification: 92D30, 91G45.

---

*Corresponding author

E-mail address: rachidelibraqui@gmail.com

Received March 24, 2026

1. Introduction

Recent banking crises have repeatedly shown that an institution that appears individually sound may become a source of systemic fragility as soon as it is embedded in a dense web of financial interdependencies. Risk assessment therefore cannot rely on a purely isolated reading of balance sheets. It must also take into account refinancing links, cross-exposures, and the mechanisms through which a local disturbance can spread to the entire banking system [1, 2, 3].

Within this perspective, credit risk and liquidity risk are deeply intertwined. Credit risk refers to the possibility that a counterparty may default, thereby generating immediate losses on the assets held by exposed institutions. Liquidity risk concerns the inability of a bank to meet its short-term funding needs without incurring excessive costs or selling assets under unfavorable conditions. In practice, these two forms of fragility reinforce one another. Asset deterioration weakens confidence and restricts access to funding, while a prolonged liquidity shortage may force fire sales that, in turn, aggravate default risk.

Three dimensions are especially important for understanding contagion dynamics. The first one is the topology of the interbank network: banks do not occupy equivalent positions, and some of them play a disproportionate role because of their centrality or the size of their exposures. The second dimension is temporal: stress is not transmitted instantaneously, because portfolio adjustments, prudential responses, and corrective interventions usually operate with non-negligible delays. The third dimension is uncertainty itself: even under similar network conditions, trajectories may differ according to information quality, market expectations, and the speed of institutional reaction.

Graph-theoretic and probabilistic tools provide a natural framework to articulate these three dimensions. Network analysis makes it possible to identify dominant nodes, preferential transmission channels, and topologies prone to shock amplification. Probabilistic modeling, in turn, captures the uncertainty associated with transitions across financial states more realistically than a fully deterministic framework [4, 5, 6, 7].

The purpose of the present study is not to replace classical dynamic analysis, but rather to extend it through a probabilistic graph-based reformulation. This viewpoint is intended to better capture heterogeneous bilateral exposures, the role of delays, and the dependence of contagion on the relational structure of the system. More precisely, our contribution relies on the following elements:

- we start from a delayed four-compartment model that distinguishes healthy banks, banks exposed to credit risk, banks exposed to liquidity risk, and defaulted banks;
- we describe interbank interactions by means of a weighted directed graph so as to incorporate bilateral exposures explicitly;
- we introduce transition probabilities that depend on both the network neighborhood and delayed transmission effects;
- we propose several indicators for measuring individual vulnerability and global systemic risk;
- we illustrate the behavior of the model through numerical simulations interpreted from an economic viewpoint.

The remainder of the paper is organized as follows. Section 2 presents a brief review of the relevant literature. Section 3 introduces the delayed mathematical model. Section 4 establishes positivity and boundedness of solutions. Section 5 develops the probabilistic graph-theoretic reformulation. Section 6 is devoted to numerical simulations and their economic interpretation. The last section concludes and outlines possible extensions.

2. Related work

Early studies on financial contagion established that the stability of a banking system cannot be inferred solely from the inspection of individual balance sheets. Allen and Gale showed in particular that incomplete interbank market structures may transform a local shock into a system-wide disturbance [1]. This insight paved the way for network-based approaches in which the very architecture of financial interconnections becomes a key determinant of collective fragility.

Within this line of research, Gai and Kapadia highlighted the *robust-yet-fragile* nature of financial networks: such networks may absorb ordinary perturbations while becoming extremely vulnerable once specific thresholds are crossed [2]. Acemoglu, Ozdaglar, and Tahbaz-Salehi later showed that diversification of links may stabilize the system under small shocks, but may also accelerate the transmission of losses when shocks become sufficiently large [3].

At the same time, graph theory has provided a powerful language for representing interbank systems. Degree, weighted strength, centrality, and spectral radius make it possible to characterize the structural importance of a bank and to evaluate its ability either to absorb or to spread stress. Several studies on complex networks emphasize in particular the role of the spectral radius in the emergence of persistent diffusion regimes [4, 5, 6].

Analogies with contagion processes studied in other fields, especially epidemic spreading models, have also been methodologically fruitful. Without conflating banking crises with biological phenomena, this analogy formalizes a useful idea: an initial disturbance does not automatically generate a generalized crisis. Its impact depends on network structure, interaction intensity, and the mechanisms that drive the system back toward more stable states.

Despite the value of these contributions, several limitations remain. Many models are fully deterministic and therefore imperfectly capture uncertainty during crisis episodes. Delay effects are often treated in a simplified manner, even though regulatory responses, balance-sheet adjustments, and refinancing tensions rarely occur without temporal lags. Finally, heterogeneous bilateral exposures are not always modeled with sufficient granularity.

The present approach lies at the intersection of these strands of literature. We retain the structure of a delayed compartmental model while coupling it with a probabilistic graph-based reformulation designed to better describe interaction heterogeneity, propagation timing, and systemic vulnerability.

3. Delayed Mathematical Model

We consider a banking system divided into four compartments:

$$g_1(t) : \text{healthy banks}, \qquad g_2(t) : \text{banks exposed to credit risk},$$

$$g_3(t) : \text{banks exposed to liquidity risk}, \qquad g_4(t) : \text{defaulted banks}.$$

The delayed dynamic model is given by

$$\begin{aligned}
\frac{dg_1}{dt} &= -\alpha_1 g_1(t) g_2(t-\tau) - \beta_1 g_1(t) + \alpha_2 g_2(t) - \delta_1 g_1(t-\tau_1) - \lambda_1 g_1(t-\tau_2),\\
\frac{dg_2}{dt} &= \alpha_1 g_1(t) g_2(t-\tau) - \alpha_2 g_2(t) - \delta_2 g_2(t-\tau_1),\\
\frac{dg_3}{dt} &= \delta_1 g_1(t-\tau_1) + \delta_2 g_2(t-\tau_1) - \alpha_5 g_3(t) - \lambda_2 g_3(t-\tau_2),\\
\frac{dg_4}{dt} &= \lambda_1 g_1(t-\tau_2) + \lambda_2 g_3(t-\tau_2) + \alpha_5 g_3(t).
\end{aligned} \tag{1}$$

Initial conditions are assumed to be continuous and positive on the interval $[-\tau_{\max}, 0]$, where

$$\tau_{\max} = \max\{\tau, \tau_1, \tau_2\}.$$

The parameters are interpreted as follows:

- α_1: transmission rate of credit risk;
- α_2: recovery rate of banks exposed to credit risk;
- α_5: transition rate from liquidity stress to default;
- β_1: spontaneous exit rate from the healthy compartment;
- δ_1, δ_2: delayed effects linked to the accumulation of vulnerabilities;
- λ_1, λ_2: delayed effects associated with the propagation of liquidity stress;
- τ, τ_1, τ_2: transmission delays.

This system describes a gradual deterioration of financial conditions. A bank that is initially healthy may become fragile because of its counterparties, credit stress may extend into liquidity stress, and severe liquidity difficulties may eventually lead to default. The delayed terms introduce memory into the system: a past shock does not vanish immediately, but continues to influence current dynamics. From an economic standpoint, this is consistent with the fact that latent losses, prudential adjustments, and market reactions usually materialize with a time lag.

4. Positivity and Boundedness of Solutions

Proposition 4.1. *Under positive initial conditions, every solution of system* (1) *remains positive for all* $t \geq 0$.

Proof. Let (g_1, g_2, g_3, g_4) be a solution of (1) generated by continuous positive initial data on $[-\tau_{\max}, 0]$. We show that each component remains nonnegative for all $t \geq 0$.

Assume by contradiction that there exists a first time $t^* > 0$ at which at least one component vanishes and then becomes negative. By definition of t^*,

$$g_i(t) \geq 0 \quad \text{for all } t \in [0, t^*], \qquad \text{and} \qquad g_i(t^*) = 0$$

for some index i.

We now inspect the system at time t^*. Every positive creation term depends on present or delayed components that, by minimality of t^*, are still nonnegative. Every loss term is proportional to the component under consideration. Hence:

- if $g_1(t^*) = 0$, then
$$\frac{dg_1}{dt}(t^*) = \alpha_2 g_2(t^*) \geq 0;$$
- if $g_2(t^*) = 0$, then
$$\frac{dg_2}{dt}(t^*) = \alpha_1 g_1(t^*) g_2(t^* - \tau) \geq 0;$$
- if $g_3(t^*) = 0$, then
$$\frac{dg_3}{dt}(t^*) = \delta_1 g_1(t^* - \tau_1) + \delta_2 g_2(t^* - \tau_1) \geq 0;$$
- if $g_4(t^*) = 0$, then
$$\frac{dg_4}{dt}(t^*) = \lambda_1 g_1(t^* - \tau_2) + \lambda_2 g_3(t^* - \tau_2) + \alpha_5 g_3(t^*) \geq 0.$$

In each case, the derivative at the instant where the component reaches zero cannot be strictly negative. Therefore, no component can cross the horizontal axis and become negative immediately after t^*, contradicting the hypothesis. The positive cone is thus invariant under the flow of the system, and every solution generated by positive initial conditions remains positive for all $t \geq 0$. □

Proposition 4.2. *Solutions of system* (1) *are bounded on every bounded interval and remain globally bounded whenever the parameters are positive.*

Proof. Let

$$N(t) = g_1(t) + g_2(t) + g_3(t) + g_4(t).$$

Summing the four equations of system (1), we obtain

$$\frac{dN}{dt} = -\beta_1 g_1(t).$$

It follows immediately that

$$\frac{dN}{dt} \leq 0,$$

so that $N(t)$ is nonincreasing on $[0,+\infty)$. Therefore,

$$0 \leq N(t) \leq N(0), \qquad \forall t \geq 0.$$

Since all components are nonnegative by the previous proposition and their sum is bounded above by $N(0)$, it follows that for every $i \in \{1,2,3,4\}$,

$$0 \leq g_i(t) \leq N(0), \qquad \forall t \geq 0.$$

Hence each compartment remains globally bounded. □

5. Probabilistic and Graph-Theoretic Analysis

In this section, we adopt a viewpoint that complements the local analysis based on the Jacobian matrix. The idea is no longer only to study the behavior of the system near an equilibrium, but also to represent explicitly the interactions among institutions by means of a weighted directed graph. Such a representation naturally incorporates heterogeneous exposures, asymmetric financial relationships, and the uncertainty attached to shock transmission [4, 7, 5].

This reformulation has a double advantage. First, it offers a more concrete description of the mechanisms through which risk is transmitted. Second, it helps identify the banks whose structural position makes them particularly influential in the propagation of stress. It is therefore well suited to systemic vulnerability analysis and to the identification of critical institutions.

5.1. Weighted graph representation of the banking system. We consider a weighted directed graph

$$G = (V,E,W),$$

where:

- $V = \{1,2,\ldots,n\}$ is the set of banks;
- $E \subseteq V \times V$ is the set of financial links between banks;

- $W = (w_{ij})_{1\le i,j\le n}$ is the exposure matrix, where $w_{ij} \ge 0$ measures the exposure of bank i to bank j.

The existence of an arc (i, j) indicates a financial relationship between banks i and j. The larger the weight w_{ij}, the more strongly a deterioration in bank j is likely to affect bank i. The exposure matrix therefore provides the support for constructing transition probabilities between financial states.

5.2. Probabilistic states of banks. At each time t, every bank i may occupy one of the following states:

- S: healthy bank;
- C: bank exposed to credit risk;
- L: bank exposed to liquidity risk;
- D: defaulted bank.

We denote the corresponding probabilities by

$$P_i^S(t), \quad P_i^C(t), \quad P_i^L(t), \quad P_i^D(t),$$

subject to the normalization condition

$$P_i^S(t) + P_i^C(t) + P_i^L(t) + P_i^D(t) = 1.$$

This representation preserves an individual-level reading of dynamics while introducing uncertainty explicitly. Even under similar exposure structures, two banks need not follow the same trajectory, because their evolution also depends on asset quality, access to refinancing, management choices, and market conditions.

5.3. Contagion probabilities on the network. The propagation of financial stress depends simultaneously on network organization and delay effects. To formalize this idea, we introduce three elementary transition probabilities.

5.3.1. *Transition from the healthy state to credit risk.* The probability that a healthy bank i becomes exposed to credit risk at time t is defined by

$$p_i^{SC}(t) = 1 - \exp\left(-\beta_c \sum_{j=1}^{n} w_{ji} P_j^C(t - \tau_1)\right).$$

This expression means that the credit pressure faced by bank i increases with the intensity of exposures coming from banks that are already financially weakened.

5.3.2. *Transition from the healthy state to liquidity risk.* The probability that a healthy bank i becomes exposed to liquidity risk is given by

$$p_i^{SL}(t) = 1 - \exp\left(-\beta_l \sum_{j=1}^{n} w_{ji}\big(P_j^L(t-\tau_2) + P_j^D(t-\tau_2)\big) \right).$$

Here liquidity stress depends not only on already illiquid banks but also on defaulted banks, which may disrupt funding channels and intensify distrust within the network.

5.3.3. *Transition from liquidity risk to default.* A bank under liquidity stress may enter default with probability

$$p_i^{LD}(t) = 1 - \exp\big(-\gamma_i(t)\big), \qquad \gamma_i(t) = \gamma_0 + \theta \sum_{j=1}^{n} w_{ij} P_j^D(t-\tau_2).$$

The term γ_0 represents baseline vulnerability, whereas the weighted sum captures the aggravating role of a defaulted neighborhood.

5.4. Probabilistic evolution system. A discrete formulation consistent with this logic reads

$$\begin{aligned} P_i^S(t+1) &= P_i^S(t)\big(1 - p_i^{SC}(t) - p_i^{SL}(t)\big) + \rho_c P_i^C(t) + \rho_l P_i^L(t), \\ P_i^C(t+1) &= P_i^C(t)\big(1 - \rho_c - \eta_i(t)\big) + p_i^{SC}(t) P_i^S(t), \\ P_i^L(t+1) &= P_i^L(t)\big(1 - \rho_l - p_i^{LD}(t)\big) + p_i^{SL}(t) P_i^S(t) + \eta_i(t) P_i^C(t), \\ P_i^D(t+1) &= P_i^D(t) + p_i^{LD}(t) P_i^L(t). \end{aligned} \tag{2}$$

System (2) should be interpreted as a mean-field approximation of individual random evolutions. From one period to the next, a change of state is not completely deterministic; it depends on the bank's own fragility, on the neighborhood to which it is exposed, and on the amount of stress already present in the network. This viewpoint is economically meaningful because the same environment does not necessarily generate identical reactions across institutions.

5.5. Graph-theoretic indicators. The analysis of the interbank network relies on several structural indicators.

In-degree and out-degree. For each bank i, define

$$d_i^{in} = \sum_{j=1}^{n} \mathbf{1}_{\{w_{ji}>0\}}, \qquad d_i^{out} = \sum_{j=1}^{n} \mathbf{1}_{\{w_{ij}>0\}}.$$

The in-degree measures how many banks may transmit a shock to bank i, while the out-degree measures its potential ability to spread a shock to the rest of the system.

Weighted strength. The incoming and outgoing weighted strengths are defined by

$$s_i^{in} = \sum_{j=1}^{n} w_{ji}, \qquad s_i^{out} = \sum_{j=1}^{n} w_{ij}.$$

These quantities account not only for the number of links but also for their intensity.

Eigenvector centrality. Eigenvector centrality evaluates the systemic importance of bank i inside the network. It is defined by

$$c_i = \frac{1}{\lambda} \sum_{j=1}^{n} w_{ij} c_j,$$

where λ is the largest eigenvalue of the exposure matrix W [4, 5]. A bank has high centrality when it is connected to other highly influential banks.

5.6. A probabilistic threshold for systemic contagion. To separate more clearly the credit and liquidity channels, one may introduce two interaction matrices $W^{(C)}$ and $W^{(L)}$. The effective contagion matrix is then defined by

$$M = \beta_c W^{(C)} + \beta_l W^{(L)}.$$

In the simplest case one may take $W^{(C)} = W^{(L)} = W$.

The spectral radius $\rho(M)$ plays a central role in the analysis of the system [6, 5].

Proposition 5.1. *If $\rho(M) < 1$, then the average propagation of risk remains controlled and the system evolves in a subcritical diffusion regime.*

Proof. The idea is that matrix M measures the average effect of one transmission cycle over the network. When $\rho(M) < 1$, repeated iterations of this dynamics do not amplify shocks in a persistent way. In particular, powers of M do not generate explosive accumulation of average fragility, which means that each contagion episode produces, on average, less than one persistent effective transmission. The system therefore remains in a regime where disturbances may spread locally without turning into a self-sustained cascade. □

Remark 5.2. *When $\rho(M) > 1$, the system may enter a systemic contagion phase characterized by the amplification of defaults and by strong sensitivity to network structure. This condition should not be viewed as an absolute criterion, but rather as a global indicator of vulnerability.*

5.7. Systemic risk index. To evaluate the systemic importance of bank i, we introduce the synthetic index

$$\mathscr{R}_i = \alpha s_i^{out} + \beta c_i + \delta P_i^D(t),$$

where s_i^{out} measures propagation capacity, c_i captures structural position in the network, and $P_i^D(t)$ is the instantaneous default risk.

A large value of this index indicates that a bank combines several systemic risk factors: strong propagation ability, a central position in the network, and an already advanced level of fragility. The indicator may therefore be used to prioritize monitoring within a macroprudential perspective.

5.8. Economic interpretation of the probabilistic analysis. The graph-based probabilistic reading highlights several economically relevant facts. First, systemic risk does not depend solely on balance-sheet size; it also depends on the position occupied by the institution within the network of interconnections. Second, an apparently diversified network may remain highly vulnerable when the most important links are concentrated around a small set of central nodes. Third, the explicit introduction of delays shows that a crisis may continue to spread even after the initial shock has seemingly passed, as long as delayed effects remain active.

From a prudential standpoint, this suggests that effective prevention should combine several instruments: regulation of excessive exposures, stronger monitoring of highly central banks, reduction of intervention delays, and reinforcement of emergency liquidity facilities. The proposed framework may therefore support network-oriented stress testing and the identification of institutions whose failure would generate the strongest spillovers.

6. Numerical Simulation and Graphical Illustration

To make the analysis more transparent, we present an aggregated numerical simulation derived from a mean-field version of the probabilistic system (2). The curves are generated directly in the manuscript from numerical coordinates. The objective is not to reproduce a particular empirical dataset, but rather to illustrate the qualitative behavior of the system when transmission intensity, default sensitivity, and recovery effectiveness vary.

6.1. Simulation scheme. We consider the aggregate proportions S_t, C_t, L_t, and D_t of healthy, credit-risk, liquidity-risk, and defaulted banks. The discrete scheme is given by

$$\begin{aligned} S_{t+1} &= S_t(1 - p_t^{SC} - p_t^{SL}) + \rho_c C_t + \rho_l L_t, \\ C_{t+1} &= C_t(1 - \rho_c - \eta) + p_t^{SC} S_t, \\ L_{t+1} &= L_t(1 - \rho_l - p_t^{LD}) + p_t^{SL} S_t + \eta C_t, \\ D_{t+1} &= D_t + p_t^{LD} L_t, \end{aligned}$$

with

$$p_t^{SC} = 1 - \exp(-\beta_c k C_{t-\tau_1}), \qquad p_t^{SL} = 1 - \exp(-\beta_l k (L_{t-\tau_2} + D_{t-\tau_2})),$$
$$p_t^{LD} = 1 - \exp\big(-(\gamma_0 + \theta D_{t-\tau_2})\big).$$

This approximation remains consistent with the graph-based logic of the model: parameter k represents an effective average degree. The larger k is, the more numerous transmission opportunities become. Conversely, larger values of ρ_c and ρ_l correspond to a stronger stabilizing capacity.

6.2. Scenario parameters. We compare two scenarios: a moderate propagation scenario and a systemic contagion scenario. Initial conditions are common to both cases:

$$S_0 = 0.92, \qquad C_0 = 0.06, \qquad L_0 = 0.02, \qquad D_0 = 0.$$

TABLE 1. Parameters used in the numerical simulations.

Parameter	Moderate scenario	Severe scenario
β_c	0.20	0.52
β_l	0.10	0.36
γ_0	0.008	0.030
θ	0.12	0.55
ρ_c	0.14	0.07
ρ_l	0.10	0.04
η	0.04	0.09
τ_1	2	2
τ_2	3	4
k	4	4

6.3. Trajectory graphs. Figure 1 shows that under moderate conditions the proportion of healthy banks decreases progressively, but the dynamics remains contained: the increase in default is slow and the system preserves a large fraction of institutions capable of absorbing stress. Figure 2 displays a sharp acceleration of contagion; the proportion of defaulted banks becomes dominant by the end of the horizon, which corresponds to a self-sustained systemic crisis. Finally, Figure 3 compares the aggregate systemic risk index $\mathscr{R}(t) = 0.8C_t + 1.2L_t + 1.8D_t$ in the two scenarios and reveals a widening separation between a regime of relative stability and a regime of runaway risk.

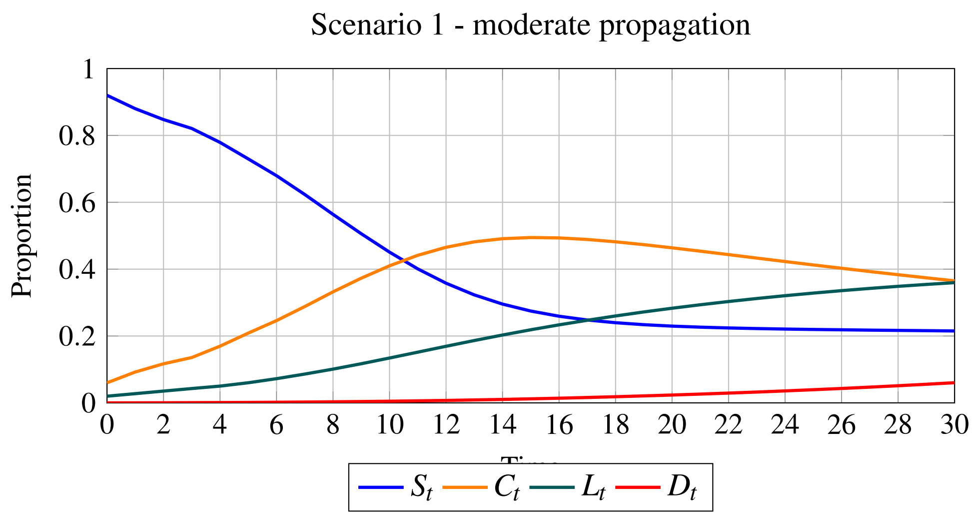

FIGURE 1. Aggregate trajectories in the moderate propagation scenario.

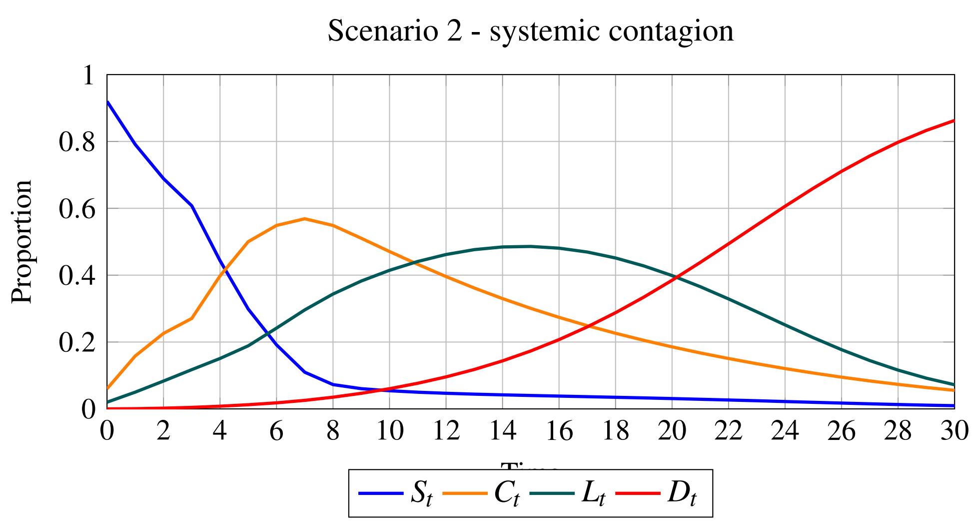

FIGURE 2. Aggregate trajectories in the systemic contagion scenario.

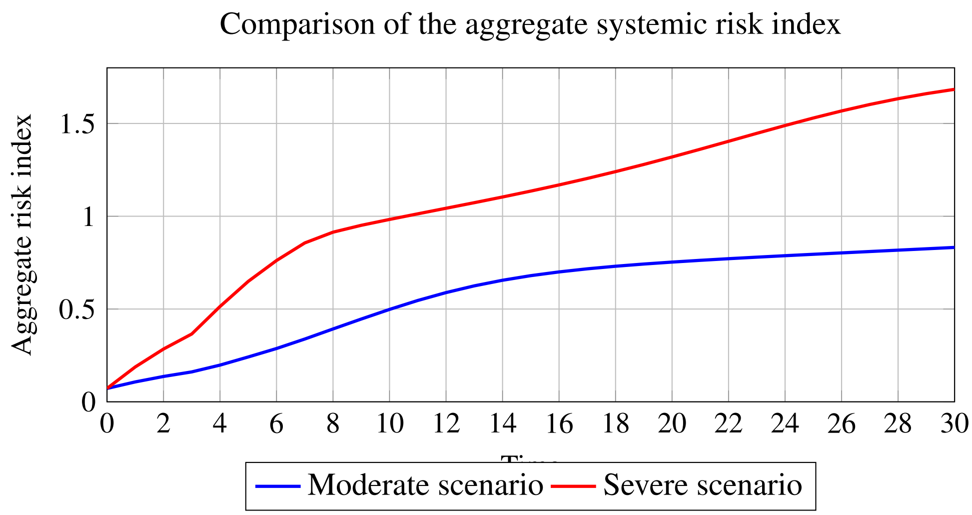

FIGURE 3. Comparison of the aggregate systemic risk index $\mathscr{R}(t) = 0.8C_t + 1.2L_t + 1.8D_t$.

6.4. Economic interpretation. The preceding graphs lead to several observations.

(1) When the transmission parameters β_c and β_l remain moderate, contagion stays diffuse but controlled. The system gradually becomes more fragile without immediately shifting toward mass default. Recovery mechanisms then play a visible stabilizing role.

(2) When transmission intensities rise and the default channel becomes more sensitive, the dynamics changes in nature. The proportion of defaulted banks grows rapidly, which means that the network no longer acts as a shock absorber but becomes itself a crisis multiplier.

(3) The aggregate systemic risk index clearly separates the two regimes. It may be interpreted as an early-warning indicator because it synthesizes the accumulation of credit stress, the rise in liquidity stress, and the increase in default.

(4) Delay effects are ambivalent. They may initially slow down the immediate visibility of the shock, but they also favor a silent accumulation of vulnerabilities, which may later trigger a much more abrupt rupture.

These results confirm that the evaluation of banking stability must jointly incorporate network structure, exposure intensity, and the effective timing of contagion. They also suggest that relevant prudential supervision should combine institution-specific solvency indicators with relational and spectral indicators of diffusion.

7. Conclusions

We proposed a graph-based probabilistic reformulation of a delayed banking contagion model involving credit risk and liquidity risk. The main interest of this framework is to bring together, within a single setting, three dimensions that are often studied separately: interbank network structure, uncertainty in financial transitions, and the temporal nature of stress propagation.

From a methodological perspective, the contribution establishes a bridge between a delayed compartmental model and a probabilistic reading that is better suited to heterogeneous bilateral exposures. From an analytical point of view, it highlights the role of centrality, weighted strengths, and spectral radius in the assessment of systemic vulnerability. From a numerical point of view, the simulations show that relatively small changes in transmission and recovery parameters may be enough to shift the system from a contained stress regime to a systemic crisis.

An important lesson of the study is that a bank may become systemically important not only because of its size, but also because of its position inside the network. A highly connected institution, or an institution located on key transmission paths, may amplify a shock well beyond its own balance-sheet weight. In this sense, graph-based analysis provides information that a purely local equilibrium study may fail to reveal with the same clarity.

Several extensions can be envisaged. Incorporating real interbank exposure data would allow a more refined calibration of the matrices used in the model. It would also be relevant to integrate optimal control mechanisms, stochastic macroeconomic shocks, or explicit central-bank interventions. Finally, the study of multilayer networks distinguishing credit exposures, liquidity dependencies, and market channels would provide a more faithful representation of the complexity of modern financial systems.

Conflict of Interests

The authors declare that there is no conflict of interests.

REFERENCES

[1] F. Allen, D. Gale, Financial Contagion, J. Polit. Econ. 108 (2000), 1–33. https://doi.org/10.1086/262109.

[2] P. Gai, S. Kapadia, Contagion in Financial Networks, Proc. R. Soc. A: Math. Phys. Eng. Sci. 466 (2010), 2401–2423. https://doi.org/10.1098/rspa.2009.0410.

[3] D. Acemoglu, A. Ozdaglar, A. Tahbaz-Salehi, Systemic Risk and Stability in Financial Networks, Am. Econ. Rev. 105 (2015), 564–608. https://doi.org/10.1257/aer.20130456.

[4] M. Newman, Networks: An Introduction, Oxford University Press, Oxford, 2010.

[5] P. Van Mieghem, Graph Spectra for Complex Networks, Cambridge University Press, Cambridge, 2011.

[6] R. Pastor-Satorras, C. Castellano, P. Van Mieghem, A. Vespignani, Epidemic Processes in Complex Networks, Rev. Mod. Phys. 87 (2015), 925–979. https://doi.org/10.1103/revmodphys.87.925.

[7] M.O. Jackson, Social and Economic Networks, Princeton University Press, Princeton, 2008.