Measuring the Propagation of Financial Shocks across Southeast Asian Countries: An Interbank-Network Approach and Dynamic Simulation

Saiqa Ilham Akbar, R. Agus Sartono,
Kusdhianto Setiawan, Mamduh Mahmadah Hanafi

Faculty of Economics and Business, Universitas Gadjah Mada, Indonesia
saiqailham88@ugm.ac.id

Abstract. Financial contagion has been widely accepted to be a trigger for systemic risk in financial systems worldwide. However, the transmission mechanisms underlying this increased systemic risk have not been extensively studied. This study attempts to simulate cross-border interbank financial contagion using the context of Southeast Asian countries (ASEAN). This context is important considering that ASEAN countries have a strong and interconnected financial cooperation framework. The simulation is based on a network of interbank connections channeled through two transmission channels: the interbank liability channel and the market channel. The simulation was conducted using Monte Carlo analysis in both normal and crisis scenarios. Based on the number of bank defaults, equity losses and cascade length, we measured the magnitude of financial contagion from a shock on a single bank. The results also show that the amplification of distress transmitted across the system dominated by interbank channel both in normal and crisis. This study also found that the magnitude of the initial price shock becomes the main driver of systemic damage in crisis conditions only. Lastly, policy recommendation indicates the need of synchronized macroprudential actions across ASEAN to manage cross-border systemic risk.

Keywords: Financial Contagion, Systemic Risk, ASEAN Interbank Network, Monte Carlo Simulation, Macroprudential Policy.

1 Introduction

Contagion as a financial phenomenon has been observed since the 1997 Asian crisis started from currency crisis in Thailand [1]. The topic resurfaced among scholars when 2007–2008 global financial crisis demonstrated that the failure of a single large financial institution can destabilize the global financial system. The shock in US from subprime mortgage failure became the trigger of multiple banks and financial institution across the globe. The extensive interconnectedness between banks and financial institutions demonstrated the fragility of the modern financial system when faced with unexpected shocks [2, 3]. This suggests that financial contagion is closely linked to systemic risk and need to be further addressed.

© The Author(s) 2026
L. Warlina and S. Luckyardi (eds.), *Proceedings of the 9th International Conference on Business, Economics, Social Sciences & Humanities – Economics, Business and Management Track (ICOBEST-EBM 2026)*,
Advances in Economics, Business and Management Research 397,
https://doi.org/10.2991/978-94-6239-752-1_4

This issue of systemic risk is increasingly relevant in ASEAN. Banking in ASEAN is increasingly integrated, one of which is through ASEAN Banking Integration Framework (ABIF). The framework enables banks in ASEAN countries to become more connected and integrated [4]. However, unlike financial integration between other countries, integration in ASEAN is still partial and is faced with very high heterogeneity between countries. This creates an ideal setting to examine whether financial integration enhances resilience through risk sharing or amplifies shocks through cross-border banking, capital flows, asset prices, and liquidity channels. The literature highlights three channels of contagion [5, 6]. First, the capital market channel in which shocks transmitted through cross-exchange asset price movements. Second is the cross-border capital flow channel in capital market. Third, the interbank funding channel where the interbank lending network acts as a conduit for direct transmission [7, 8]. Differences in domestic policies, political stability, and development levels among member countries add to the heterogeneity of risk, making it crucial to map the most dominant channels for spreading risk.

Interbank network viewed as center mechanism of financial contagion in this study. At the bank level, interbank debt linkages can lead to defaults by one bank on another, directly harming its creditors [9]. In the market channel, liquidity pressures in one bank can force them to sale assets rapidly. These fire sales will lower market prices, causing value losses for other banks holding similar assets [10]. Recent empirical evidence suggests that the interaction of interbank lending channel and market channel can amplify financial contagion [11]. In other words, network topology and portfolio holdings together determine how far and how deeply shocks spread.

At the country level, the impact of contagion can spread beyond the financial sector. Concurrently increasing volatility across multiple markets during a crisis can impact the real economy. This will reduce confidence, delaying investment, and increasing the cost of capital. Therefore, understanding contagion in ASEAN is crucial to anticipate and minimize regional systemic risk. This understanding is also necessary for maintaining stable and inclusive growth. The need to understand this contagion drives research that can identify transmission channels, map network vulnerabilities, and evaluate financial system policies within a scalable framework.

Based on the above context and needs, this study aims to measure and model the transmission of financial shocks between banks in ASEAN. The modeling is conducted using a balance sheet-based interbank network approach and Monte Carlo-based dynamic simulation. This study develops a mathematical model of interbank contagion across ASEAN countries and integrates two main channels: the interbank liability network and the market channel. Furthermore, this study attempts to identify and compare the relative role of each transmission channel in explaining the contagion. This approach will bridge the gap between market-correlation-based studies and the need for analyses that explicitly account for transmission mechanisms. This approach will provide a quantitative basis for designing more targeted macroprudential policies at the regional level.

2 Literature Review

2.1 Theoretical Basis of Financial Contagion and Systemic Risk

Financial contagion refers to the transmission of financial distress between institutions or countries through co-movements and spillovers observed in prices and capital flows. Contagion as the rapid spread of market disturbances, reflected in co-movements in exchange rates, stock prices, and capital flows during a crisis. Relatedly, systemic risk is the potential for significant financial disruptions resulting from system-wide fragility, not just problems at the level of isolated firms. The International Monetary Fund, Bank for International Settlements, and Financial Stability Board (2009) conceptualize systemic risk as disruptions to essential financial services arising from a deterioration in the financial system, with serious consequences for the real economy. Therefore, tail risk, or the possibility that extreme losses in an institution become relevant to the system, has begun to be emphasized in systemic risk measurement frameworks [4,5].

Previous literature distinguishes between narrow and broader definitions of contagion. Broader definition of contagion often encompasses common shocks that simultaneously weaken multiple institutions [11, 12]. Although a common shock is conceptually distinct from direct contagion in the narrow definition, both can produce systemic impacts when vulnerabilities are widespread. Drivers of systemic risk are often associated with institutional characteristics such as size, interconnectedness, complexity, and common exposure conditions that shape how quickly losses spread across balance sheets and markets [3, 5, 11, 13]. These drivers are reflected in the Basel Committee on Banking Supervision's (2013) framework for identifying global systemic banks [14, 15].

2.2 Interbank Contagion Mechanism

Past literature focused on two main transmission channels. The first channel is the direct transmission through interbank claims. The second channel is indirect transmission through market valuation effects. The direct interbank channel is triggered by credit risk. Creditor banks experience losses when debtor banks default or fail to repay which potentially triggering a cascade of defaults. Theoretical and empirical contributions from previous research formalize this mechanism by showing how default cascades can emerge from bilateral exposure structures [8, 11]. Furthermore, the mechanism for bank failure to spread to creditor banks in the real-world interbank system has also been demonstrated [15]. Meanwhile financial distress spreads through low-priced asset sales and mark-to-market losses. This indirect transmission creates liquidity pressure that forces banks to liquidate assets. This action depress asset prices and inflicts valuation losses on other banks holding similar portfolios [9]. This mechanism becomes stronger when banks are under stress. When funding and market liquidity deteriorate simultaneously, deepening fire sales and asset price discounts [16]. Furthermore, high leverage further intensifies the dynamics of deleveraging and price declines [17].

Another important point to note is that these two channels can interact. Even in the absence of a contractual relationship, shared exposures can transmit stress between institutions [18]. Consistent with empirical evidence from crisis period, the combined

impact of the direct and indirect channels can produce nonlinear amplification. The combined effect exceed the impact of each channel separately [19]. The 2007 global financial crisis illustrates this interaction. Losses transmitted through counterparty exposures intersect with broad-based asset price declines and create liquidity pressures, thus strengthening systemic loss dynamics [4, 20].

2.3 The Role of Network Structure in Strengthening or Dampening Transmission

Based on network theory, systemic outcomes depend not only on the magnitude of the shock but also on the network topology, which is influenced by the connectivity, concentration, and centrality of nodes. Allen and Gale (2000) showed that systems with greater connectivity can diversify and distribute small losses more widely [11]. This often increases resilience to less severe shocks. However, the same connectivity can produce "robust but fragile" system behavior. In such a situation, the system appears stable under normal conditions but becomes highly vulnerable to large shocks or shocks affecting central institutions [3].

Theoretical research has focused more on the nonlinear and threshold nature of contagion, where outcomes change discontinuously when shocks exceed a critical level [21, 22]. From this perspective, centrality is a key determinant of the system's significance. Battiston and colleagues introduced the DebtRank variable to operationalize a node's position relative to the systemic impact of shocks and the potential for loss spread [23]. More broadly, dense and highly connected networks can mitigate small shocks through risk sharing but also expose the system to a cascade of disturbances that can spread widely [22, 24]. Beyond interbank relationships, overlapping portfolios and shared exposures to those portfolios can further accelerate contagion. Adverse price movements can impact multiple institutions simultaneously, effectively devaluing assets on their balance sheets through a generalized asset valuation [13].

2.4 Monte Carlo Simulation and Sensitivity Analysis in Contagion Studies

Simulation-based approaches are widely used to characterize systemic outcomes beyond average-case dynamics. This approach used in this study to overcome the nonlinear and tail-driven nature of contagion. Monte Carlo simulation is particularly useful for capturing heavy-tail loss distributions and rare-but-severe cascades that are difficult to derive analytically [25]. Simulation studies also show that connectivity and capital buffers can affect contagion nonlinearly. Greater connectivity may increase domino risk in intermediate regimes while potentially reducing marginal risk at very high connectivity through diversification effects.

Sensitivity exercises conducted to complement the Monte Carlo simulation. Varying key parameters such as recovery conditions and shock intensity are paired with regression or correlation-based diagnostics to identify dominant drivers of systemic outcomes. In European network simulations, lower recovery assumptions are associated with meaningfully higher systemic losses [25]. This view reinforces the broader literature's emphasis on identifying which structural parameters strongly govern cascade se-

verity. Overall, prior work supports combining network-based simulation with sensitivity and regression-style diagnostics to reveal the mechanisms through which local disturbances escalate into system-wide distress under different regimes

2.5 Hypotheses Development

Financial contagion transforms into systemic events when banks are interconnected through contractual claims and correlated exposures. In banking system networks, distress is more than a function of individual balance-sheet weakness, but also a function where losses are transmitted through interbank obligations and market-based valuation effects. Consistent with the systemic risk perspective this study frames contagion in ASEAN as an interaction between direct balance-sheet interdependence and indirect amplification via asset prices under liquidity stress. This dual-channel view aligns with theoretical work showing that linkages can provide risk sharing under normal conditions while generating non-linear amplification once shocks cross critical thresholds. Interconnectedness between banks producing "robust-yet-fragile" outcomes in financial networks [3, 11, 19].

Network models of interbank markets imply that the failure of one bank can impose losses on counterparties and trigger further defaults. This happens when exposures are concentrated or when a shock hits a highly connected node [11]. Clearing and contagion frameworks formalize how payment shortfalls can propagate through the liability matrix and creating default cascades [8]. Moreover, non-linearities in networks imply that systemic outcomes may be rare but severe. Small shocks are often absorbed, whereas large shocks lead to widespread failures [19]. Given ASEAN's increasing financial integration and cross-border banking linkages, these mechanisms suggest that local shocks may escalate into system-wide distress. Therefore, the first hypothesis tests whether the simulated ASEAN banking network exhibits systemic contagion. The systemic contagion reflected in the emergence of multi-bank failures and large aggregate equity losses, particularly in the tails of the outcome distribution.

Hypothesis 1 (H1): There is financial contagion between banks in the ASEAN region, which is transmitted through interbank networks and joint asset ownership.

Previous literature distinguishes between direct transmission through interbank claims and indirect transmission through market prices. The interbank liability channel is triggered by the default risk of borrower banks. When borrower banks fail to meet their obligations, creditor banks experience a direct decline in asset values, which can trigger subsequent defaults [8, 11]. Conversely, the asset price channel amplifies distress when banks respond to liquidity shortages. When facing liquidity pressures, banks sell assets en masse, depressing market prices and incurring mark-to-market losses on other banks holding similar assets [9]. This market channel is particularly potent during liquidity crises because funding and market liquidity can deteriorate simultaneously, giving rise to forced selling and significant price discounts [15].

Network theory suggests that the relative importance of these two channels depends on institutional and market conditions. Direct interbank exposure creates a direct contractual loss mechanism that does not require large-scale trading to operate. On the

other hand, price-mediated contagion generally requires substantial fire sales and a sufficiently strong downward price impact to generate losses [9]. In many banking systems with material interconnectedness, counterparty losses can be a key pathway for a cascade of failures. Meanwhile, asset price effects act as an additional amplifier, particularly when market depth collapses. In the context of the increasingly integrated ASEAN financial system, this study hypothesizes that variations in the interbank liability channel should be able to explain systemic impacts more strongly than the market channel.

Hypothesis 2 (H2): The interbank liabilities channel is more dominant than the asset price decline channel in explaining cross-border interbank contagion in ASEAN

3 Methodology

This study builds a model based on bank balance sheets and the interbank liability network to simulate contagion through the interbank channel and the market channel. We build the model within a Monte Carlo simulation framework with realistic bank heterogeneity. We also simulate two macroprudential scenarios, baseline (normal) and crisis (stress). The three main outputs of the simulation are Final Defaults (the number of failed banks at post-shock equilibrium), Equity Loss (the aggregate loss of system capital), and Cascade Length (the number of rounds of contagion until the system reaches a new fixed point).

3.1 Balance Sheet Notation and Identities

Banks are indexed by $i = 1, \dots, N$ ($N = 10$). Each bank's balance sheet is defined as:

$$A_i = C_i + Y_i + I_i + S_i \tag{1}$$

$$L_i = D_i + U_i + B_i \tag{2}$$

$$E_i = A_i - L_i \tag{3}$$

where A_i is an asset with C_i denotes being cash/high-quality liquid assets (HQLA), Y_i credit to the real sector, I_i interbank receivables, and S_i a securities/market portfolio. L_i is a liability with D_i denotes third-party funds, U_i interbank liabilities, and B_i debt securities issued by the bank. E_i denotes an equity. To ensure consistency of the interbank system at the aggregate level, we impose $\sum_i I_i = \sum_i U_i = \mathcal{S}$. Bank heterogeneity is captured through cross-sectional variation in size, capital, liquidity buffers (HQLA), net cash outflow (NCOFi), and interbank exposure magnitudes.

A bank is considered to fail in round t if E_(i,t)<0 or " " 〖"LCR" 〗 _(i,t) is persistently lower than a specific value. In the baseline scenario, the primary default trigger is E_(i,t)<0; a violation of the minimum LCR value would trigger asset sales rather than an immediate default declaration.

3.2 Interbank Network and Counterparty-loss Channel

The gross liabilities of bank i to other banks are defined as $\bar{p}_i = \sum_j L_{ij}$ and the proportion matrix Π with $\Pi_{ij} = L_{ij}/\bar{p}_i$ (if $\bar{p}_i = 0$, then $\Pi_{ij} = 0$). The corresponding asset side $X_{ji} = L_{ij}$ represents the claim of bank j on bank i. Thus, $U_i = \bar{p}_i$ and $I_i = \sum_j X_{ij}$. Interbank payments at each round are governed by a clearing mechanism with recovery. With $\mathbf{a}_t$ denoting external assets available agter the market price correction and $0 \leq \rho_j \leq 1$ the recovery rate of debtor bank j, interbank clearing payments solve:

$$\mathbf{p}_t = \min\{\bar{\mathbf{p}}, \max\{0, \mathbf{a}_t + \Pi^\top \mathbf{p}_t\}\} \tag{4}$$

with effective loss-given-default $1 - \rho_j$ applied to flows from a defaulting debtor to its creditors. In the discrete implementation, creditor i's incremental interbank loss in round t is computed directly from exposures to newly defaulting debtors, and bank equity is updated accordingly. To avoid double counting of interbank losses across rounds, default events are tracked by a "new default" indicator; the full step-by-step accounting is provided in Appendix A.

3.3 Market Channel: Fire Sale and Price Impact

The market channel captures feedback from forced asset liquidation into market prices. Let $P_{a,0}$ be the initial price of asset a (or a single aggregate asset index). An initial price shock is applied at $t = 0$, reducing prices by ΔP_0. For $t \geq 1$, if banks collectively sell $\Delta Q_{a,t}$ units, the price evolves with a linear price-impact function:

$$P_{a,t+1} = P_{a,t}\left(1 - \lambda_a \frac{\Delta Q_{a,t}}{M_a}\right) \tag{5}$$

where M_a is an effective market size and λ_a is the market-depth (price-impact) parameter; larger λ_a implies a shallower market and stronger price declines for a given volume of sales. If bank i holds q_(i,a) units of asset a, its mark-to-market loss follows from the induced price change, and equity is updated aftermarket revaluation. For transparency and tractability, the baseline specification uses a single aggregate asset index.

3.4 Regulatory Constraints and Rules of Conduct

Liquidity Coverage Ratio (LCR) defined as:

$$\mathrm{LCR}_{i,t} = \frac{HQLA_{i,t}}{NCOF_{i,t}} \tag{6}$$

where $NCOF_{i,t}$ is net cash outflow and $HQLA_{i,t}$ the stock of high-quality liquid assets. Banks sell assets only when $LCR_{i,t} < 1$. The per-round asset sale quantity is determined by the shortfall needed to restore liquidity coverage, subject to an upper bound representing frictions or liquidation constraints. Defaulted banks cease to sell assets and are removed from subsequent balance-sheet adjustments. Implementation details and optional robustness constraints (e.g., per-round sale caps) are documented in Appendix A.

3.5 Scenario Design and Parameter Distribution

Each Monte Carlo iteration simulates two regimes in parallel—baseline and crisis—with parameters drawn independently for each regime. The model focuses on three stochastic parameters: market depth (λ), interbank recovery rate (ρ), and initial price shock (ΔP_0). Parameter draws follow truncated normal distributions to respect economic bounds ($\lambda \geq 0,\ 0 \leq \rho \leq 1,\ \Delta P_0 \geq 0$). Regime-specific means are set by the scenario design (Table 1), while standard deviations are fixed within regime for consistency. In addition, each regime includes a targeted initial shock: a trigger bank is selected uniformly at random among the 10 banks, and the initial shock is applied through an indicator vector in the first contagion round. After parameters and triggers are assigned, the contagion process iterates until a fixed point is reached (or a conservative maximum number of rounds), and the three systemic outputs are recorded.

Table 1. Parameter distribution and truncation rules.

Scenario	Parameter	Mean	SD	Truncation
Baseline	λ_B	$\mu_{\lambda,B}$ (default 0,01)	0.01	$\lambda_B \leftarrow \max\,(o, \mathcal{N}(\mu_{\lambda,B}, 0.01))$
	ρ_B	$\mu_{\rho,B}$ (default 0,10)	0.10	$\rho_B \leftarrow \min\,\{1, \max\left(o, \mathcal{N}(\mu_{\rho,B}, 0.10)\right)\}$
	$\Delta P_{0,B}$	$\mu_{\Delta,B}$ (default 0,00)	0.03	$\Delta P_{0,B} \leftarrow \max\left(o, \mathcal{N}(\mu_{\Delta,B}, 0.03)\right)\}$
Crisis	λ_C	$\mu_{\lambda,C}$ (default 0,05)	0.05	$\lambda_C \leftarrow \max\,(o, \mathcal{N}(\mu_{\lambda,C}, 0.05))$
	ρ_C	$\mu_{\rho,C}$ (default 0,05)	0.05	$\rho_C \leftarrow \min\,\{1, \max\left(o, \mathcal{N}(\mu_{\rho,C}, 0.05)\right)\}$
	$\Delta P_{0,C}$	$\mu_{\Delta,C}$ (default 0,05)	0.05	$\Delta P_{0,C} \leftarrow \max\left(o, \mathcal{N}(\mu_{\Delta,C}, 0.05)\right)\}$

3.6 Hypothesis Testing and Statistical Analysis

Hypotheses are evaluated using a consistent set of empirical checks on the simulated output distributions. H1 assessed by comparing baseline versus crisis outcome distributions using summary statistics and tail quantiles (P50/P95/P99). Analysis complemented by difference-of-means tests and bootstrap confidence intervals for tail-risk measures. H2 tested using three methods. First, Pearson correlations between $\lambda, \rho, \Delta P_0$ and each systemic outcome. Second, standardized OLS regressions with heteroskedasticity-robust (HC1) standard errors estimated separately for baseline and crisis regimes. The last, targeted "toggle" robustness experiments that vary ρ versus $\lambda / \Delta P_0$ to compare marginal contributions under comparable parameter shifts.

3.7 Limitations

This model combines two main channels and minimal regulatory rules. several simplifications such as linear price impact, a single aggregate asset index, and a deterministic fire sale rule are made for transparency and replicability. The correlations presented are

reduced across a range of parameters. Therefore, standardized regression analysis supports the causal interpretation. Multi-asset extensions and endogenous recovery could add fidelity but are retained as future research agendas to ensure the calibration burden does not exceed the scope of this article.

4 Results and Discussion

4.1 The Existence of Systemic Contagion

Table 2 presents summary statistics from 1,000 Monte Carlo iterations conducted for two scenarios. The table displays the mean, standard deviation, and 50th, 95th, and 99th percentiles for the three primary metrics used in the analysis. Final Defaults capture the number of banks that fail after the propagation process is complete. Equity Loss capture the aggregate loss to system capital. The last, cascade Length capture the number of rounds of contagion before the system reaches new stability.

The comparison between the baseline and crisis columns aims to show the extent to which changes in stress parameters. This specifically compare the recovery rate (ρ), price impact (λ), and initial shock (ΔP_0) that affect the severity of the contagion and overall systemic risk.

Table 2. Simulation results summary.

Metrics	Statistics	Baseline	Crisis	Crisis/Baseline Ratio
Final Defaults (number of default banks)	Mean	3.79	9.07	2.40
	Std Dev	0.70	0.58	
	P50	4.00	9.00	
	P95	4.00	10.00	
	P99	7.01	10.00	
Equity Loss (% of system capital)	Mean	29.88%	91.09%	3.05
	Std Dev	10.67%	5.21%	
	P50	29.50%	90.74%	
	P95	45.06%	100.00%	
	P99	69.11%	100.00%	
Cascade Length (number of rounds)	Mean	1.05	2.01	1.91
	Std Dev	0.40	0.12	
	P50	1.00	2.00	
	P95	1.00	2.00	
	P99	4.00	3.00	

The simulation results indicate that contagion is present but typically contained in the baseline regime. The system experiences only limited failures (approximately 3–4

banks) and short cascades. This implies that in normal conditions the network can absorb moderate idiosyncratic shocks without triggering widespread collapse. However, baseline outcomes are not purely benign. The distribution remains right-skewed, implying that rare but larger loss realizations can still occur. This result consistent with the idea that interconnected systems may appear stable under typical shocks while retaining exposure to tail events.

Contrast to the normal regime, the crisis regime produces systemic, non-linear amplification. The average number of failed banks rises significantly (around nine failures), and tail realizations become substantially more severe. The crisis regime also generates much larger aggregate losses with equity loss is around three times higher than the baseline. The contagion process persists longer with cascade length extending to up to three rounds in stressed iterations. Overall, the pronounced deterioration in mean outcomes and, crucially, the expansion of the upper tail provide clear evidence of a robust-yet-fragile structure: the same network that absorbs shocks in normal times can transition into widespread failures once stress parameters push the system past critical thresholds [3, 11, 19].

Overall, the baseline–crisis comparison supports H1 by showing that the simulated ASEAN banking network can exhibit systemic contagion, particularly under adverse conditions where counterparty losses and market stress jointly amplify distress. This finding provides the empirical foundation for subsequent analyses that identify the dominant transmission channel and quantify cross-country spillovers.

4.2 The Influence of Interbank and Market Channels

The simulation results confirm that the relative magnitude of the interbank and market channels significantly influences the severity of contagion. A comparison between the baseline and crisis scenarios provides quantitative evidence on the role of the recovery rate (ρ) parameter in the interbank channel. The price-impact (λ) parameter and the magnitude of the initial price shock ($\Delta P0$) in the market channel also confirm it. The parameters are assumed to be moderate in the baseline scenario. On the other hand, the crisis scenario represents a stressful situation, with a lower recovery rate (ρ), a larger λ, and a deeper $\Delta P0$ reflecting market panic. Coordinated changes in these three parameters significantly increase contagion magnitude in the simulation.

All systemic risk metrics spike in the crisis scenario compared to the baseline. Average final defaults doubled from 3–4 banks to around nine bank failures during the crisis. This finding suggests that in a crisis, the same shock can trigger much broader contagion. This result in line with expectations that adverse scenarios expose hidden weaknesses not visible under normal conditions. The system's aggregate equity loss also increased significantly. Average equity losses in the crisis doubled more than baseline. The Cascade Length metric shows a striking difference in patterns. In the baseline scenario, propagation rarely extended beyond one round (P95 cascade length = 1, P99 = 4 rounds). However, in the crisis scenario, many simulations experienced multi-round contagion. The average cascade length increased to 2, with P95 reaching two rounds and P99 reaching three rounds. This finding suggests that bank failures cascaded for up to three waves before abating in crisis scenario. This statistical comparison consistently

demonstrates the crucial role of the parameters ρ, λ, and ΔP0. When the interbank channel is weakened (low ρ), and the market channel is strengthened (high λ, ΔP0), the shock's impact is significantly greater.

These two transmission channels reinforce each other nonlinearly. A low recovery rate means that each bank default results in maximum credit losses for its creditors in crisis scenario. It also increases the likelihood that creditors will fail as well. This transmission of losses triggers a large first wave of defaults through direct interbank losses. At the same time, the λ value and the sharp initial price decline of ΔP0 contribute to the market channel's transmission of distress. Banks that not directly affected by counterparty defaults still experience declining portfolio values due to asset fire sales by failing banks. Many previously healthy banks are under pressure with their equity. Some banks may cross the threshold for failure due to these mark-to-market losses. This combination creates a double feedback loop. Banks weakened by market losses may then fail, triggering further interbank losses for other banks, and so on. This pattern is evident in crisis simulations. A second wave of defaults that triggered by asset price depreciation, often follows the first wave. Subsequent waves cease only when neither the network effect nor the price decline triggers additional bank failures.

To identify which channel contributes more strongly to systemic outcomes, we first examine parameter sensitivity in the baseline regime using pearson correlation. Table 3 shows that the interbank recovery rate ρ_B exhibits the largest absolute correlations across all systemic outcomes (Final Defaults: -0.20; Equity Loss: -0.53; Cascade Length: -0.30). These results indicate that higher recovery substantially reduces bank failures, capital depletion, and cascade persistence. In contrast, λ_B is essentially uncorrelated with all outcomes while ΔP_(0,B) displays only modest positive correlations (0.09–0.21). These baseline results imply that under normal conditions the interbank liabilities channel dominates in explaining variation in contagion severity, while the market channel plays a secondary role.

Table 3. Parameter sensitivity to baseline scenario output (Pearson correlation).

Parameters	Final Defaults	Equity Loss	Cascade Length	Range (min – max)
λ_B	0.00	-0.01	0.01	0.102 – 0.172
ρ_B	-0.20	-0.53	-0.30	0.509 – 1
ΔP_{0B}	0.09	0.21	0.12	0 – 0.103

The sensitivity pattern shifts in crisis regime. This reflects the transition from "normal" to "stress" dynamics. Table 4 shows that the magnitude of the initial market shock becomes a key amplifier. The $\Delta P_{0,C}$ correlates strongly with Final Defaults (0.48) and Equity Loss (0.52). Meanwhile, ρ_C remains negatively correlated with systemic outcomes (Final Defaults: -0.13; Equity Loss: -0.14), although with smaller magnitudes than in baseline. This results consistent with the interpretation that once recovery is already low across banks, marginal variation in ρ still matters but is less powerful than the scale of the initial price disruption. Importantly, λ_C remains near zero for Final Defaults and Equity Loss, indicating that endogenous market depth (price impact) does

not materially explain cross-iteration variation relative to the initial shock magnitude. For Cascade Length, correlations remain close to zero for all parameters, suggesting that once crisis conditions are triggered, cascade duration is governed primarily by threshold and network-feedback effects rather than by small parameter variations.

Table 4. Parameter sensitivity to crisis scenario output (Pearson correlation).

Parameters	Final Defaults	Equity Loss	Cascade Length	Range (min – max)
λ_C	0.00	0.00	-0.01	0.554 – 0.900
ρ_C	-0.13	-0.14	0.02	0 – 0.189
ΔP_{0C}	0.48	0.52	-0.04	0 – 0.349

Taken together, Tables 3–4 provide strong support for H2 in a regime-contingent sense. the interbank liabilities channel (via ρ) is the most consistent and structurally important determinant of contagion propagation. These channels dominating the baseline regime and remaining directionally stabilizing in crisis. On the other hand, the market channel becomes influential mainly through the exogenous shock magnitude ΔP_0 rather than through the endogenous price-impact parameter λ. In other words, large asset-price shocks can magnify losses during crisis, but the persistence and transmission of distress across banks are still fundamentally anchored in counterparty-loss dynamics embedded in the interbank liabilities network.

5 Conclusion and Recommendation

The conclusion of this study confirms that integrating the interbank network model, Monte Carlo simulations, and empirical regression analysis provides a deeper understanding of the mechanisms of systemic risk transmission in the ASEAN region. Through this approach, the study successfully identified how local shocks can develop into regional systemic risk. This study also found the factors that determine the severity and direction of such transmission. The simulation results indicate that the ASEAN financial system is robust-yet-fragile. It means the system is relatively stable against small shocks but highly vulnerable to large shocks affecting central banks within the network. Baseline scenario simulation shows that transmission remains limited. Meanwhile in the crisis scenario, the number of bank failures and aggregate equity losses increase sharply. These increase indicates the potential for systemic contagion. Sensitivity and regression analyses confirm that the interbank liabilities channel has the greatest influence on systemic losses, with a significant negative correlation, whereas the market parameter shows no significant impact. These results confirm that the interbank channel is the primary mechanism of risk transmission in the ASEAN region. Meanwhile asset price shocks only become a major driver under extreme conditions.

Overall, this study makes three significant contributions. First, this study integrates a financial network approach and Monte Carlo simulation with econometric analysis to

identify measurable contagion mechanisms. Second, this study strengthens the evidence that the interbank liabilities channel is the primary source of contagion in the ASEAN region. In contrast, the asset price channel is secondary and dependent on market conditions. Third, this study provides an empirical basis for policymakers to strengthen regional macroprudential policy coordination through the ASEAN Banking Integration Framework (ABIF) and the Chiang Mai Initiative Multilateralization (CMIM).

For further research, several development directions are possible. First, the model can be expanded by adding multi-asset dimensions and portfolio heterogeneity to reflect the dynamics of fire-sale contagion better. Second, the interbank recovery mechanism can be modeled endogenously based on banks' liquidity ratios and asset quality, thereby reflecting more dynamic market conditions. Third, the analysis can be extended to the non-bank financial institutions (NBFIs) sector and cross-border capital flows to capture broader interconnectedness within the regional financial system. Finally, dynamic network regression or agent-based modeling can be an alternative for observing the evolution of systemic risk over time. With these development directions, future research is expected to strengthen empirical understanding and support the design of regional financial policies that are more resilient to cross-border systemic risk.

References

1. Seth, N., Sighania, M.: Financial market contagion: selective review of reviews. Qualitative Research in Financial Markets. **9**, 391–408 (2017).
2. Bullard, J., Neely, C.J., Wheelock, D.C.: Systemic Risk and the Financial Crisis: A Primer. Review. **91** (2009).
3. Haldane, A.G., May, R.M.: Systemic risk in banking ecosystems. Nature. **469**, 351–355 (2011).
4. Adrian, T., Brunnermeier, M.K.: CoVaR. American Economic Review. **106**, 1705–1741 (2016).
5. Acharya, V.V., Pedersen, L.H., Philippon, T., Richardson, M.: Measuring Systemic Risk. Review of Financial Studies. **30**, 2–47 (2016).
6. Schwerter, S.: Basel III's ability to mitigate systemic risk. Journal of Financial Regulation and Compliance. **19**, 337–354 (2011).
7. Shahrier, N.A.: Contagion effects in ASEAN-5 exchange rates during the Covid-19 pandemic. The North American Journal of Economics and Finance. **62**, 101707 (2022).
8. Trihadmini, N., Falianty, T.A.: Contagion effect and stock market co-movement: ASEAN and the global financial crisis. Jurnal Ekonomi dan Pembangunan, **28**(2), 191–206 (2020).
9. Eisenberg, L., Noe, T.H.: Systemic Risk in Financial Systems. Management Science. **47**, 236–249 (2001).
10. Cifuentes, R., Ferrucci, G., Shin, H.S.: Liquidity Risk and Contagion. Journal of the European Economic Association. **3**, 556–566 (2005).
11. Allen, F., Gale, D.: Financial Contagion. Journal of Political Economy. **108**, 1–33 (2000).
12. Kaufman, G.G., Scott, K.E. What is systemic risk, and do bank regulators retard or contribute to it? The Independent Review. **7**(3), 371–391 (2003).
13. Billio, M., Getmansky, M., Lo, A.W., Pelizzon, L.: Econometric measures of connectedness and systemic risk in the finance and insurance sectors. Journal of Financial Economics. **104**, 535–559 (2012).

14. Basel Committee on Banking Supervision. Basel III: A global regulatory framework for more resilient banks and banking systems (rev. June 2011). Bank for International Settlements (2011).
15. Basel Committee on Banking Supervision. Global systemically important banks (G-SIBs): Updated assessment methodology and the higher loss absorbency requirement. Bank for International Settlements. (2013).
16. Upper, C., Worms, A.: Estimating bilateral exposures in the German interbank market: Is there a danger of contagion? European Economic Review. **48**, 827–849 (2004).
17. Brunnermeier, M.K., Pedersen, L.H.: Market Liquidity and Funding Liquidity. Review of Financial Studies. **22**, 2201–2238 (2008).
18. Adrian, T., Shin, H.S.: Liquidity and leverage. Journal of Financial Intermediation. **19**, 418–437 (2010).
19. European Systemic Risk Board. EU-wide stress test 2016: Methodological note (2016).
20. Glasserman, P., Young, H.P.: How likely is contagion in financial networks? Journal of Banking & Finance. **50**, 383–399 (2015).
21. Brownlees, C., Engle, R.F.: SRISK: A Conditional Capital Shortfall Measure of Systemic Risk. Review of Financial Studies. **30**, 48–79 (2016).
22. Acemoglu, D., Ozdaglar, A., Tahbaz-Salehi, A.: Systemic Risk and Stability in Financial Networks. American Economic Review. **105**, 564–608 (2015).
23. Battiston, S., Puliga, M., Kaushik, R., Tasca, P., Caldarelli, G.: DebtRank: Too Central to Fail? Financial Networks, the FED and Systemic Risk. Scientific Reports. **2**, (2012).
24. Gai, P., Kapadia, S.: Contagion in financial networks. Proceedings of the Royal Society A: Mathematical, Physical and Engineering Sciences. **466**, 2401–2423 (2010).
25. Halaj, G., Kok, C.: Modelling the emergence of the interbank networks. Quantitative Finance. **15**, 653–671 (2015).

Open Access This chapter is licensed under the terms of the Creative Commons Attribution-NonCommercial 4.0 International License (http://creativecommons.org/licenses/by-nc/4.0/), which permits any noncommercial use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons license and indicate if changes were made.

The images or other third party material in this chapter are included in the chapter's Creative Commons license, unless indicated otherwise in a credit line to the material. If material is not included in the chapter's Creative Commons license and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder.