



## **Financial Contagion in Interbank Networks**

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*“Learn from yesterday, live for today, hope  
for tomorrow. The important thing is not to  
stop questioning”*

*Albert Einstein*

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## ABSTRACT

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The United States subprime mortgage crisis of 2007/8as well as the European sovereign debt crisis of 2009/10, revealed the weaknesses of financial institutions worldwide and the crucial role financial interconnectedness plays in the transmission of financial distress. Although interconnectedness contributes to efficient risk sharing, it may lead to contagious episodes of default following an initial shock in the financial system.

The complexity of the financial system has led many academics to utilize the network theory to study the effects of interconnectedness and network topology on financial stability. Studying the financial system as a network is one of the methods that have been used to investigate the emergence of systemic risk through the connections of banks.

In this thesis, we argue that a proper assessment of systemic risk should include a thorough analysis of the network of financial linkages that exist between the various financial institutions. In such network structure, every node represents a bank and the connections between banks are represented by edges. A robust interbank market plays an important role in the stability of the financial system. Through the interbank market, banks which suffer liquidity shortages can borrow from banks with liquidity surpluses. Thus, the interbank market can have a stabilizing effect on the financial system by redistributing funds in an effective way among banks, however, at the same time, it can make the system prone to financial contagion through the existing interbank linkages.

The role connectivity plays in the stability of the interbank network depends also on how the structure of the network interacts with additional factors which are specific to the interbank market. Heterogeneity on banks' balance sheet sizes or in degree distributions (incoming and outgoing links) among them can change the role played by connectivity within the financial system.

For decades, among the various suspects for destabilizing the financial system were large financial institutions whose failure would be disastrous to the greater economic system (“too big to fail” theory). Such financial institutions must be supported by the government when they face financial distress due to their systemic importance and interconnectedness. However, smaller financial institutions but with lots of

connections in the interbank market can have an even larger impact on the financial system if they fail. Higher interconnectedness of the interbank network can reduce the probability of default due to the fact that transmission of a shock can be shared by many counterparties and thus it dissipates faster. On the other hand, when the magnitude of the shock has crossed a critical threshold, due to increased interconnectedness the shock will spread into a large part of the system which can cause a large cascade of defaults. This is the so-called “robust-yet-fragile” property that financial systems exhibit (Haldane, 2009; Acemoglu et al., 2015).

In this Thesis, we develop a better understanding of systemic risk according to 2 basic pillars/dimensions:

- The analysis of the interplay between interbank contagion and several crucial drivers such as network topology, leverage, interconnectedness, heterogeneity and homogeneity across bank sizes and interbank exposures
- Applications on the central banks’ policy and the monitoring of systemic risk.

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As Constantine Cavafy states in the most distinguished Greek poem of the 20th century, called Ithaka, “*As you set out for Ithaka hope the voyage is a long one, full of adventure, full of discovery. Laistrygonians and Cyclops, angry Poseidon—don’t be afraid of them:[ ]..Ithaka gave you the marvelous journey. Without her you would not have set out. She has nothing left to give you now. And if you find her poor, Ithaka won’t have fooled you. Wise as you will have become, so full of experience, you will have understood by then what these Ithakas mean.*”

Again, thank you all for this amazing and full of experiences journey!!

Kalliopi Loukaki

Athens, 2020

*To my beloved nieces,  
Mary & Constantina*

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## List of Abbreviations

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Basel I, BCBS' first accord

Basel II, BCBS' second accord

Basel III, BCBS' third accord

BCBS, Basel Committee on Banking Supervision

BIS, Bank for International Settlements

CDS, Credit Default swap

CoVaR, Conditional Value-at-Risk

EBA, European Banking Authority

ECB, European Central Bank

E-MID, Electronic market for interbank deposits in the Euro area and the USA

FED, Federal Reserve System (Central Bank of United States)

G10, Group of countries that agreed to participate in the General Arrangements to Borrow (GAB), an agreement to provide the International Monetary Fund (IMF) with additional funds to increase its lending ability.

GFC, Global Financial Crisis

G-SIBs, Global Systemically Important Banks

IMF, International Monetary Fund

ME, Maximum Entropy

MES, Marginal expected shortfall

NPLs, Non-Performing Loans

OLS, Ordinary Least Squares

OTC, Over the counter

SD, Standard Deviation

TIER 1, Common Stock + Retained Earnings

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## 1. Introduction

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### 1.1. Motivation of Thesis

*“And the lessons from the global financial crisis are of course many and varied. But among the most important is also perhaps the simplest: to safeguard against systemic risk, the financial system needs to be managed as a system.”*

in the speech “Rethinking the financial network” by Andrew G Haldane, Executive Director, Financial Stability, Bank of England, at the Financial Student Association, Amsterdam, 28 April 2009.

The global financial turmoil that occurred in 2007 has highlighted the crucial role that existing linkages among banks and financial institutions plays in channeling and amplifying shocks once an initial shock hits the system. The bankruptcy of Lehman Brothers and the sequence of events which unfolded in 2008 forced public sectors to intervene in order to bail out many financial institutions and restore financial stability. This action, though, put pressure on public finances in many countries. The high costs associated with those interventions demonstrated the need for a clear understanding of interconnected financial systems and their potential to induce/facilitate systemic crises (May, 2008;Haldane, 2009).

A starting point to study and analyze systemic risk among the agents in a financial system is to pay attention to the interaction structure among banks and financial institutions in the propagation of an initial shock that hits the system. A better analysis of such a structure should therefore help regulators to better evaluate systemic risk and prevent financial distress and domino effects in the market.

To address these fundamental issues concerning the complexity of the interactions among banks and the dynamics of an initial shock propagating into the system, network theory comes into play. This is so, because the skeleton of any complex system can be described by a network arising from the interactions of the parts that is consisted of. Indeed, the connections between the agents of the interbank system can be studied as a network and be represented as a graph where banks are the nodes and edges represent the existence of lending/borrowing relationships between any two agents. Directionality identifies the borrower and the lender while the weight of the link represents the loan amount. In general, the interbank market is much richer and complex than a simple weighted graph. However, network theory provides useful tools to characterize the structure of such graphs, to identify systemically significant nodes and test the effectiveness of macro-prudential policies.

According to Upper (2011), interbank contagion can take place through a multiple of channels. The channels through which a shock spread can be broken down into two groups: indirect and direct contagion channels. Direct contagion channel results from the direct interbank linkages between banks and can happen when an idiosyncratic

shock travel through the network of banks and affect the balance sheets of multiple agents while indirect contagion is created by indirect linkages between banks.

The Thesis is organized as follows. Chapter 1 deals with the systemic risk and contagion in financial systems with a special focus on interbank markets from a theoretical perspective. In Chapter 2, we discuss the scope of this Thesis and the related literature. Empirical findings on the structure of interbank networks and aspects such as heterogeneity in bank sizes and interbank exposures are analyzed in order to obtain a clear understanding of systemic risk in such financial networks.

In Chapter 3, we introduce our basic methodological tools we are going to use in the next chapters. This chapter involves basic concepts from network theory and our simulation methodology that we follow.

In Chapter 4 we develop an interbank network model and demonstrate how contagion propagates under various scenarios concerning the degree of the system's heterogeneity, the balance sheet composition and the level of connectivity among banks. This chapter is based on the paper "Simulating financial contagion dynamics in random interbank networks", which is a joint work with Mr Vassilios G. Papavassiliou and has previously been published as Leventides et al. (2019). Under this particular framework, we assume that the network structure in our model is arbitrary, that is, the network of interbank claims/obligations forms randomly. The assumption of randomness in the network structure has the advantage that our model contains any possible structure that may emerge in the real world and this is what makes our analysis distinct from the earlier literature. We use a direct channel of contagion resulting from the direct interbank linkages among banks which takes effect when an idiosyncratic shock travels through the network of banks and affects the balance sheets of multiple agents. Our findings show that heterogeneity in bank sizes and interbank exposures matters a great deal in the stability of the financial system, as its absorption capacity is enhanced. Also, the level of interconnectedness hugely impacts on the system's resilience, especially in smaller and highly interconnected interbank networks. Finally, we provide evidence that highly leveraged banks form the main channel through which financial shocks propagate within the system and such effect is more pronounced in large interbank networks than in smaller ones.

In Chapter 5, we extend the model developed in Chapter 4 to include a wide variety of network topologies and provide a better understanding of the relation between network structure, banks' characteristics and interbank contagion. While the focus of the previous chapter is on the various factors that affect interbank contagion such as bank capital ratios, leverage, interconnectedness and homogeneity across banks' sizes, the model lacks flexibility as far as the variability of the networks links is concerned. In order to circumvent this problem, we introduce the Erdős-Rényi probabilistic network model in our study to provide a wider vicinity of scenarios concerning the network structure of the interbank system and study how homogeneity within the interbank network affects the propagation of financial distress from one institution to the other parts of the system through bilateral exposures. Our findings indicate a non-monotonic relation between diversification and interbank contagion across the

different sizes of interbank networks for all scenarios tested. While for small or medium interbank networks, connectivity can act as an absorbing barrier, such that interbank systems of these sizes can withstand the initial shock, for large network systems connectivity does not seem to provide an effective shield against capital losses. Our results, for the four scenarios tested, indicate that small and thus more concentrated interbank network systems are more prone to contagion. In these cases, we observe that the size of total capital losses is, in most cases, larger than that documented in medium and large sized systems, which is in line with the findings of Nier et al.(2007). As far as heterogeneity is concerned, our results clearly suggest that, it plays a significant role in the stability of the financial system. Similar to Leventides et al. (2018), we still find that when heterogeneity is introduced with respect to the size of each bank, the system's shock absorption capacity is enhanced. In addition, when we allow for heterogeneity on interbank exposures in our model, we observe additional resilience to the interbank network as an initial shock dissipates more easily than in the case of homogeneous interbank claims.

Chapter 6 provides applications of complex network analysis for systemic risk monitoring and policy formulation while Chapter 7 concludes this Thesis.

## 1.2 Background

### 1.2.1 Contagion: The spread of systemic risk in financial networks

The recent financial crisis has brought to the fore the need for a better understanding of systemic risk and an immediate reform of the financial regulatory framework. It became clear that unregulated systemic risk can pose a real threat to the global financial stability<sup>1</sup> and economic growth. However, according to ECB(2009) and IMF (2009) there is no commonly accepted definition of systemic risk. The European Central Bank (ECB,2009) defines systemic risk as the risk inherited with the possibility of an institution failing to meet its obligations, prompting the same failure on other agents in the system causing wider effects due to illiquidity and credit constraints. In its Financial Stability Review in December (2009), ECB states for systemic risk that “ *one perspective is to describe it as the risk of experiencing a strong systemic event. Such an event adversely affects a number of systemically important intermediaries or markets (including potentially related infrastructures). The trigger of the event could be an exogenous shock (idiosyncratic, i.e. limited in scope, or systematic, i.e. widespread), which means from outside the financial system. Alternatively, the event could emerge endogenously from within the financial system or from within the economy at large. The systemic event is strong when the*

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<sup>1</sup>Financial stability is formally defined by ECB “*as a condition in which the financial system – which comprises financial intermediaries, markets and market infrastructures – is capable of withstanding shocks and the unravelling of financial imbalances. This mitigates the prospect of disruptions in the financial intermediation process that are severe enough to adversely impact real economic activity*”.

*intermediaries concerned fail or when the markets concerned become dysfunctional (in theoretical terms this is often a non-linearity or a regime change)."*

Billio et al. (2012) state that although most regulators and policymakers believe that systemic events can be identified after the fact, a precise definition of systemic risk seems remarkably elusive. However, Billio et al. (2012) suggest that systemic risk involves the financial system, a collection of interconnected institutions that have mutually beneficial business relationships through which illiquidity, insolvency, and losses can quickly propagate during periods of financial distress.

A recent definition of systemic risk comes from Constâncio et al. (2019) on an ECB occasional paper where systemic risk is defined as “*the risk that financial instability significantly impairs the provision of the financial products and services required by the financial system to a point where economic growth and welfare may be materially affected.*” The authors also highlight the importance of network interdependencies analysis in order to properly assess and evaluate systemic risk.

As Georg (2011) states, one of the lessons from the recent Global Financial Crisis is that systemic risk can take many forms. Systemic risk can take the form of financial contagion in the interbank market, where due to interconnectedness among banks, an initial shock that hits one bank can trigger and amplify contagious defaults to other banks in the market. Furthermore, there has been observed that the fear of interbank contagion may reduce interbank lending and, in turn, impair liquidity provision among banks. This fear has been detected over this period with the drying up of liquidity in the interbank market due to the counterparty default risk in the recent financial crisis. Liquidity dry-ups not only impact the banking sector but also the real economy since banks no longer fulfill their role as financial intermediaries facilitating the circulation of money in the market.

### **1.2.2 The interbank market**

The interbank lending market allows financial institutions to lend or borrow money in order to meet their liquidity requirements. Borrowing in the interbank market is the most immediate source a bank can resort to in meeting its liquidity needs (e.g funding outflows or investment opportunities) while the smooth functioning of this market is a key function in ensuring the stability of the banking system and the global financial system as well.

The majority of trading in the interbank market takes place over-the-counter (OTC), directly between any two banks. Banks lend or borrow funds and repay them over a short period of time, usually overnight. At any point in time, a bank may be involved in multiple lending or borrowing relationships thus forming multiple connections with different counterparties. According to the seminal paper of Allen and Gale (2000), in equilibrium banks will optimally use interbank market to insure themselves against liquidity risk by keeping deposits at other banks. However, this insurance comes with an additional risk, the counterparty default risk. Thus, for a bank holding

interconnections with other banks always implies dealing with the trade-off between risk sharing and risk of contagion.

The default of a single bank with multiple borrowing connections may jeopardize the functioning of the interbank market in case the creditors of the bank defaulting are unable to absorb the losses. In this case, the initial shock of the defaulting bank may potentially spread into the whole system and cause serious default cascades. Interbank markets therefore may represent an important channel of contagion through which problems affecting one bank or one country may spread to other banks or other countries.

Should the initial distress from a bank propagate into the banking system, several policy responses need to be made. According to Leitner (2004) financial agents in the system may be willing to bail out other agents to prevent the collapse of the whole network, therefore avoiding the intervention of the financial authorities or the regulatory institutions. However, according to Elliott, Golub and Jackson (2013), default cascades introduce a moral hazard problem. Financial organizations have an incentive to bail out a large failed bank in order to avoid failure costs to themselves, which then incentivizes failing firms to increase these costs in order to be bailed out.

Another policy response is the mandatory recapitalization of the shareholders by injecting new equity to the bank or to sell part of the bank's assets to pay off current liabilities. However, as Gaffeo and Mollinary (2014) state this practice is hardly feasible in time of systemic crisis and is subject to problems of inter-temporal inconsistency. This is why in many cases distressed institutions have been bailed out by cash injections or explicit guarantees financed by the government. The American International Group (AIG) bailout by the US authorities in September 2008 was motivated by the fear that its default would have significant repercussions in the global financial system since many of the biggest financial institutions had become exposed to it via derivative contracts, the Credit Default Swaps (CDS). This action, though, put pressure on public finances in many countries. The high costs associated with those interventions demonstrated the need for a clear understanding of interconnected financial systems and their potential to induce/facilitate systemic crises (May, 2008; Haldane, 2009). Thus, one of the main issues that academics and regulators had to fully understand, deal with and regulate was interconnectedness.

### **1.2.3 Looking at systemic risk through a network lens**

A starting point to study and analyze systemic risk among the agents in a financial system is to pay attention to the complex interaction structure among banks and financial institutions. This complex structure of the banking system can be captured particularly well by a network representation of financial systems. The use of network models can be instrumental in capturing the externalities that the risk related with a financial institution may create for the entire financial system. Indeed, such models provide useful insights for financial policy and supervision. The analysis of interbank systems under the prism of network theory can identify systemic importance or

vulnerability of financial institutions and thus provide a useful tool to the regulators for prudent and efficient supervision. Huser (2015) highlights the ability of network models to capture the “financial fragility hypothesis” that De Bandt et al. (2012) put forward, arguing that systemic risk and potential contagion effects are of special concern in the financial system. This hypothesis outlines three interrelated features of the financial system that can threaten financial stability. These are: (a) the complex network of exposures among banks, (b) the importance of balance sheet composition due to the maturity transformation role played by banks and (c) the informational and control intensity of financial contracts.

Network theory has gained significant momentum in the field of financial stability, supervision and regulation during the last decade. The rapid advances in computing power and the increasing availability of data concerning the exposures of the market players has helped central bankers and market practitioners to identify systemic vulnerabilities and design appropriate measures and policies to safeguard financial stability. Networks can capture the structural features of financial systems; exposures can be visualized by links where the direction represent lending-borrowing relationships and the weight of each link is associated with monetary quantities and transaction volumes. Finally, network theory can also describe the different types of links between financial institutions. As Huser (2015) and Langeld and Soramaki (2014) highlight, there are hundreds of different type of transactions that financial institutions engage in, such as deposits, derivatives, foreign exchange exposures, etc. In order to encapsulate the different kinds of possible connections among banks, recent empirical literature focus on multilayer networks where each layer of the system represent a particular kind of transaction and is characterized by its own topology and its own propensity for the propagation of eventual shocks (Aldasoro and Alves (2015), Montagna and Kok (2013) and Bargigli et al.(2014) ).

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## 2. Scope of the Thesis and Literature Review

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### 2.1 Scope of the Thesis

In this Thesis, we focus our attention on the direct contagion channel and study the effectiveness of various drivers on interbank contagion. The flourishing literature which ensued in recent years has developed theoretical models aimed at addressing the various issues concerning systemic risk. Counterfactual simulations on data have been employed to study interbank contagion under different scenarios related to the topology of the interbank network, the size of interbank exposures, the degree of heterogeneity and interconnectedness within the network. In what follows, we develop two distinct models with banks linked to one another by their interbank claims and investigate by means of Monte Carlo simulations how complexity of an interbank network structure affects interbank contagion under different testable scenarios. Our analysis belongs to the strand of the theoretical literature as we employ computer simulations to construct a large number of bank networks involving entities with interlocking interbank claims/obligations. Using tools from complex network theory, we model how shocks of an initial default may spread from one institution to another (simulated financial networks to study contagion phenomena have also been employed by Nier et al., 2007 and Gai and Kapadia, 2010).

This Thesis contributes to the literature in a number of ways. First, we study the interplay of several crucial drivers on interbank contagion, such as bank capital ratios, leverage, network topology, interconnectedness and homogeneity across banks' sizes. Along these lines, we address the following questions: Does heterogeneity, network topology, leverage and interconnectedness matter for systemic risk and the propagation of contagion? If so, in what respect? In order to answer these questions, we build two interbank network models and demonstrate how contagion propagates under various scenarios concerning the degree of the system's heterogeneity, the balance sheet composition and the level of connectivity among banks.

Second, we utilize only two components from a bank's balance sheet, that is, equity and interbank loans in order to construct a parsimonious regression model. Our regression model is used for testing the impact of crucial drivers recorded in our simulation experiments on interbank contagion. Regression analysis has also been used by Krause and Giasante (2012) to assess the role played by the network's topological features and balance sheet positions in the transmission of bank failures. The authors utilize a scale-free network model to study interbank contagion with large parameter ranges and many parameters to initialize a balance sheet. However, their model becomes inflexible in operation as it is difficult to compare simulations by varying one of the parameters. On the contrary, our models are easily explainable, reproducible and more amenable to analysis and interpretation.

Third, unlike most studies in the recent literature (Nier et al., 2007; Gai and Kapadia, 2010; Chinazzi et al., 2015; Amini et al., 2016) we define the term contagion as the situation in which the initial failure of a bank leads to the failure of at least one other



bank, while the extent of contagion is measured by the total capital loss in the banking system due to the failure of at least one bank. In other words, we are mostly interested in detecting the magnitude of capital losses in the banking network rather than the number of banks that were adversely affected.

Our analysis also differs from networks based on entropy methods which are able to capture unidimensional features of the network structure only, and not dynamic and multifaceted network patterns. Maximum entropy (ME) approaches may also not be very reliable in assessing the severity of financial contagion as, depending on the interbank market structure, can lead to undervaluation or overvaluation of the extent of contagion (Mistrulli, 2011). Moreover, we circumvent the problem of data unavailability as real data on interbank exposures are generally only available to central bankers and regulators, thus rendering the empirical analysis of networks problematic.

The first model, which is part of the paper Leventides et al. (2019), examines the knock-on effects an initial default can bring into the interbank network under the assumption of randomness in the link formation. The assumption that the network of interbank claims and obligations forms randomly, enables us to capture all possible scenarios that may appear in real-world situations.

The second model extends the study presented in Leventides et al. (2019) to include a wide variety of network topologies and provide a better understanding of the relation between network structure, banks' characteristics and interbank contagion. While the focus of this paper is on the various factors that affect interbank contagion such as bank capital ratios, leverage, interconnectedness and homogeneity across banks' sizes, the model lacks flexibility as far as the variability of the networks links is concerned. In order to circumvent this problem, we introduce the Erdős-Rényi probabilistic network model in our study to provide a wider vicinity of scenarios concerning the network structure of the interbank system and study how homogeneity within the interbank network affects the propagation of financial distress from one institution to the other parts of the system through bilateral exposures.

Finally, our findings have interesting implications for policymaking and further research. Useful insights resulting from this study can provide early alert signs of weakness of the interbank system, identifying vulnerabilities of the system as a whole.

## **2.2. Literature Review**

There is an increasing number of studies that investigate the numerous facets of contagion in the interbank network. Many researchers showed that the structure of the network plays a crucial role in the generation and propagation of systemic risk in the interbank market.

In their influential work, Allen and Gale (2000) using a simple network model of four banks, show that in equilibrium banks hold interregional claims on other banks to provide insurance against liquidity risk. These interbank connections, however, can make them vulnerable to counterparty default risk. The failure of one bank can cause

the default of other banks and several contagious events, thus jeopardizing the whole financial system. The authors distinguish three structures: (i) the complete structure, (ii) the incomplete market structure and (iii) the disconnected incomplete market structure. In the case of a complete structure the bank has symmetric linkages with all other banks in the interbank network system while in the case of incomplete structure the linkages that are observed are among the neighboring banks. Finally, in the case of disconnected structure, there are regions that are particularly disconnected or isolated. Allen and Gale (2000) show that a financial system with a complete network structure is more robust to contagion than incomplete structures. The reason for the above conclusion is that when the network is fully connected (complete network) the amount of interbank deposits held by each bank is evenly spread over all other banks. Thus, in case a negative shock hit the network, its impact is gradually fade away since every bank in the network share a small loss and there is no contagion. However, in case of an incomplete network structure, the system seems to be more fragile since the impact of the shock is concentrated among neighboring banks. Allen & Gale (2000) conclude that the interconnection in the interbank market always implies a trade-off between risk sharing and risk of contagion.

Babus (2007) builds on the framework proposed by Allen and Gale (2000) and develops a model with endogenous formation of network where banks form links with each other as an insurance mechanism to reduce the risk of contagion. The authors agree with Allen and Gale (2000) that better connected networks are more resilient to contagion.

Nier et al.(2007) study the extent to which the resilience of an interbank network depends on a combination of variables characterizing the network topology, banks' characteristics in terms of net worth and interbank exposures, and market concentration. Using Monte Carlo simulation experiments in random graphs, they find that the effect of the degree of connectivity is non-monotonic. Specifically, a small initial increase in connectivity increases the chance of contagion defaults. However, after a certain threshold value, connectivity improves the capacity of a banking system to withstand shocks. In addition, the authors find that the banking system is more resilient to contagious defaults if its banks are better capitalized and this effect is non-linear. Finally, the size of interbank liabilities tends to increase the risk of default cascades, even if banks hold capital against such exposures and more concentrated banking systems are shown to be prone to larger systemic risk.

Thus, interconnectedness acts first to strengthen contagion as it increases the number of channels (i.e. interbank links) through which an idiosyncratic shock can propagate through the network of counterpart exposures. Nonetheless, the residual shock passed on to any interconnected single institution becomes necessarily smaller as the number of links increases. Hence, interconnectedness also contributes to risk-sharing. As a matter of fact, beyond a certain threshold (or tipping point), this latter effect prevails and eventually enhances the resilience of the system. There is, however, a downside to the interconnectedness of the banking system. As the burst of the last financial

crisis showed, interbank markets display what Haldane (2009) denotes as a “robust-yet-fragile” or “knife-edge” property, specifying in a more precise way how connectivity influences stability. In normal times, interconnectedness may lead to an enhanced liquidity allocation and an increased risk sharing between banks but in times of a crisis interconnectedness can amplify shocks and propagate the crisis all over the network. Thus, connections can serve at the same time as shock-absorbers and shock-amplifiers. Higher interconnectedness may reduce the probability of default when contagion has not started yet. However, when contagion begins, higher interconnectedness amplifies initial shocks and increases the probability of having large default cascades.

Battiston et al. (2012) study also the trade-off between risk sharing (diversification) and network externalities (contagion) identifying, for some values of their model’s parameters, a U-shaped relation linking connectivity and probability of default. The authors argue that the stabilizing role of risk diversification prevails only when connectivity is low. If connectivity is already high, a further increase may have the perverse effect of amplifying the shock due to distress propagation and financial accelerator. Thus, for a bank, being interconnected with other banks always implies dealing with the trade-off between risk sharing and risk of contagion.

A more interconnected network scheme implies that a negative shock can be more easily absorbed when there are multiple counterparties. In addition, connectivity may induce banks to bail out each other in order to prevent contagion-avoiding thus the intervention of government or the central bank. However, a highly interconnected bank will also face the risk of being hit by a large negative shock through one of its neighbors. Thus, studying the role of the level and form of connectivity in the interbank market is crucial to understand how direct contagion works.

Gai and Kapadia (2010) using a network model of a banking system study how the probability and potential impact of contagion is influenced by aggregate and idiosyncratic shocks, network structure and liquidity. The authors agree with Haldane (2009) concerning the “robust-yet-fragile” property that the financial system exhibit. Even when the probability of contagion is very low, its effects can have tremendous consequences to the financial system. Higher connectivity may reduce the probability of default when contagion has not started yet but it may also increase the probability of having large default cascades when contagion begins.

We have seen that the level of connectivity plays a crucial role on the stability of the financial system. However, the role played by connectivity depends also on how the structure of the network interacts with some other factors such as the heterogeneity of banks, liquidity, capital requirements, incentives to misbehave and indirect contagion via price changes on common assets. All these can modify the role played by connectivity within the financial system.

### **2.2.1 The role of heterogeneity in the interbank network structure**

The role connectivity plays in the stability of the interbank network depends also on how the structure of the network interacts with additional factors which are specific to the interbank market. Heterogeneity on banks' balance sheet sizes or in degree distributions (incoming and outgoing links) among them can change the role played by connectivity within the financial system.

Iori et al. (2006), for instance, use a simulation model of 400 banks (banking systems with homogeneous banks, as well as systems in which banks are heterogeneous) in the interbank market in which the lending and borrowing are endogenously generated. In this model, each bank faces fluctuations in liquid assets and stochastic investment opportunities that mature with delay, creating the risk of liquidity shortages. Thus, the banks resort to overnight interbank borrowing only when facing a temporary liquidity shortage. The authors find that contagion probability is lower in case the interconnected institutions are homogeneous, i.e. they have similar characteristics such as size or investment opportunities, as thus no institution becomes significant for either borrowing or lending. Finally, Iori et al. (2006) conclude that with heterogeneity, knock-on effects become possible, but the stabilizing role of interbank lending remains so that the interbank market can play an ambiguous role. The authors also suggest, as Allen and Gale (2000), that as the connectivity increases the system becomes more stable.

Sachs (2010) assesses the interaction between completeness, interconnectedness and equality of distribution of interbank claims (as measured by entropy) finding that high levels of completeness, together with an equal distribution of exposures, can stabilize the interbank system. Studying different structures of interbank connections (the complete network, a network where banks form their linkages randomly and a money center model), Sachs (2010) finds that within a money center model, an increasing concentration of assets on core banks decreases the stability of the system. Comparing the above structures of interbank connections, the author concludes that money center systems with asset concentration among core banks are more unstable than networks with banks of homogeneous size that form their links randomly.

Caccioli et al. (2012) study the role of heterogeneity in degree distributions (the number of incoming and outgoing links), balance sheet size and degree correlations between banks. They study the probability of contagion conditional on the failure of a random bank, the most connected bank and the biggest bank. They find that networks with heterogeneous degree distributions are shown to be more resilient to contagion triggered by the failure of a random bank, but more fragile with respect to contagion triggered by the failure of highly connected nodes. The authors analyze the comparison in the interbank network literature between the "too big to fail" and the "too interconnected to fail" by comparing the probability of contagion conditional on the failure of the most connected versus the biggest bank for a system with power law degree distributions of connectivity versus asset size. Caccioli et al. (2012) show that when average degree (connectivity) is low, the probability of contagion due to failure

of the highly connected bank is higher than that due to the failure of the biggest node. However, when average degree is high, the opposite holds. Since the second scenario seems to be more realistic (networks with high connectivity), it seems that having "too big to fail" banks is more effective in the elimination of a shock.

Ladley(2013) develops a partial equilibrium model of a closed economy in which heterogeneous banks interact with borrowers and depositors through the interbank market. Banks in the model are subject to regulation and the aim of the model is to qualitatively show how regulation and network structure can constrain or enhance the risk of contagion. The results show that for high levels of connectivity the system is more stable when the shock is small, while the contagion effects are amplified in case of larger initial shocks. However, in the case of small shocks, higher interbank connections promote risk sharing. Finally, as far as regulatory actions are concerned, Ladley (2013) finds that increases in the equity ratio seem to reduce contagion while increases in the reserve ratio have the opposite effect as banks use the interbank market more to meet their liquidity needs creating stronger interbank linkages. He also considers how constraints on the amount a lender may lend to a particular borrower can help stabilizing the interbank system. For larger shocks this policy tends to reduce contagion but for smaller shocks the effect is increased. If this constraint is very tight, bankruptcies are uniformly reduced but so is lending to non-bank borrowers.

Amini et al. (2013) assess the role of heterogeneity due to connectivity in the network. They focus on bank heterogeneity not in terms of their size but in terms of their number and size of their interconnections with other banks. (heterogeneous node degree and exposures). They conclude that the most heterogeneity is introduced, the least the resilience of the network. Contrary to the findings of Amini et al. (2013) is the study from Georg and Poschmann (2010) who don't find any significant evidence that the heterogeneity of the financial system has a negative impact on financial stability.

Montagna and Lux (2013) study systemic risk in scale-free interbank networks. They construct a Monte Carlo framework features, taking into account various stylized facts from the recent empirical literature, via what is called a fitness algorithm. With a particular choice of such a function as a generating mechanism they construct an artificial banking sector which displays a power law degree distribution, disassortative behavior and heterogeneity in the banks' sizes. Montagna and Lux (2013) show how the percentage of net worth and the percentage of interbank assets (both on total assets) affect the spread of an idiosyncratic shock in the system. Their results indicate a shell structure in the diffusion of losses in the network, i.e. creditors banks of the defaulted entity fail mostly before the others, and it is possible to classify defaults of the different shells in the cascade events. Finally, they also find that random networks or networks constructed on the base of a maximum entropy principle lead to fewer contagious defaults than their scale-free networks which is justified by the underestimation of the contagion risk that these networks usually exhibit.

Chinazzi et al. (2015) make a significant contribution to the debate on macro-prudential regulation concerning which structure of the financial system is more resilient to exogenous shocks, and which conditions, in terms of balance sheet compositions, capital requirements and asset prices, guarantee the higher degree of stability. In order to explore the interplay between heterogeneity, network structure and balance sheet composition in the spreading of contagion, the authors develop two distinct models of contagion: a benchmark model and an extended model. The benchmark model is based on a simple framework where the financial system is modeled as a static network of credit exposures between banks while in the extended model, the interbank market is composed of two layers: a network of long-term exposures and a network of short-term exposures and banks can dynamically adapt their short-term exposures in response to liquidity shocks. For the above models, the authors consider different parametrizations where they let vary: capital requirements, banks' balance sheet composition, fire-sale prices, network topology and types of shocks hitting the system. By analyzing the five scenarios chosen (homogeneous banks with homogeneous exposures, homogeneous banks with heterogeneous exposures, heterogeneous banks with homogeneous exposures where the system is generated using a fitness model and heterogeneous banks with heterogeneous exposures), the authors show how different features characterize the financial system and its stability.

As far as heterogeneity is concerned and it regards the link weights, the authors observe a widening of the interval of connectivity levels in which contagion may occur. When heterogeneity concerns the size of the banks, which means that there are big banks which act as shock absorbers then the original shock fades away making contagion a less likely phenomenon. Heterogeneity in connectivity provides additional resilience to the system when the initial default is random. However, this is not always the case when highly connected or large institutions get distressed and this can raise the possibility of contagion risk.

Furthermore, Chinazzi et al. (2015) prove that the too-connected-to-fail banks are more dangerous than the too-big-to-fail ones and should be the primary concern for policy makers since their distress can trigger systemic breakdowns. Finally, the authors also find that larger capital requirements are effectively able to stabilize the system, while larger liquid reserves, despite providing a buffer in case of liquidity run, induce banks to keep lower capital buffers, thus making them vulnerable to contagion.

### **2.2.2 Empirical findings on the structure of interbank networks**

Empirical studies now provide a plethora of valuable stylized facts on the interbank network topology which can be used for theoretical modeling of contagion risk.

The first feature is the **degree distribution** of the nodes that represents the number of incoming and/or outgoing links per node, i.e. the number of bank's counterparties. There is a lot of evidence that many interbank networks often exhibit a **scale-free topology**, i.e. they are characterized by the presence of hubs, that are nodes with a

degree that is much higher than the mean degree of the other banks. Thus, in a scale-free network, there is a high probability that many transactions would take place through one of the high-degree nodes-often called as money center bank- of the network. As we have seen before, the presence of such highly interconnected hubs make systems in general more prone to a break-down in case of targeted attacks.

Scale-free degree distributions have been frequently reported in many interbank markets. Some examples are Boss et al. (2004) for the Austrian interbank market, Inaoka et al. (2004) for the Japanese interbank market, Soramaki et al. (2007) for the US Fedwire system, Alves et al. (2013) for the European interbank market for large banks, while there exist conflicting findings for the Italian interbank market (Iori et al. (2008), Fricke and Lux (2015a)).

Furthermore, Bech and Atalay (2010) found for the US Federal Funds market that the number of interconnections per bank follows a fat-tailed distribution, with most banks having many interconnections.

According to Iori et al. (2008) and Fricke and Lux (2015a), the degree distribution is not necessarily best represented by a power law distribution. More specifically, Iori et al. (2008) and Fricke and Lux (2015b) find no evidence in favor of scale-free networks in the e-MID<sup>2</sup> market. Iori et al. (2008), for example, find that the degree distribution, may not be a scale-free but it is still heavier tailed than a purely random network while Fricke and Lux (2015b) find that the e-MID market data are best described by negative binomial distributions.

Since evidence concerning the degree-distribution seems to be mixed, it hasn't be settled yet whether real-world interbank networks fall into the category of scale-free networks.

Knowing the degree distribution is of paramount importance for policy makers who could identify the systemic and vulnerable nodes and take actions on the knock on effects of turbulent banks. One step towards this action could be the availability of more granular data and a sound statistical analysis of the distributional properties of interbank data.

Empirical evidence shows also that banks tend to be organized in communities and the networks, they form, tend to be disassortative (Bech and Atalay, 2010; Soramäki et al., 2007). In a disassortative network, less connected nodes have a tendency to be connected with higher connected nodes. This often reflects the economic rationale that smaller banks, rather than transacting with each other, typically use a small set of money center banks as intermediaries.

A simple way to identify such a behavior is to study the distribution of the average degree of the neighbours of the vertices belonging to the network. In the case of

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<sup>2</sup>The e-MID is an electronic trading platform for unsecured deposits based in Milan and mainly used by Italian banks for overnight interbank credit. It started offering multilateral trading for interbank deposits in Italy in 1990 and now it connects to the market 170 banks from 28 countries, including 30 viewers among Central Banks and Ministries of Finance.

disassortative structure, this distribution should be a decreasing function in the degree of the nodes, as a consequence of the attitude of high-degree vertices to link with low-degree ones, and vice versa. The empirical evidence of the disassortative behavior include Boss et al. (2004) for the Austrian interbank market, Soramäki et al. (2006) for the US Fedwire Network and Iori et al. (2008) for the Italian interbank market.

Due to lack of data availability, many researchers either disregarded the heterogeneity of interbank relations or focused only on one type of transaction, implicitly assuming that the network of the selected type of credit transactions is a good proxy for the networks of other types. Data from the overnight unsecured market have been extensively used to study interbank contagion (Iori et al. (2008), Furfine (2003), Gabrieli (2010) ). According to Bargigli et al. al. (2013), focusing only in one type of the various transactions that banks engage in, may provide biased results. Instead of focusing only on one type of transaction -layer-, an analysis which could consist different types of transactions could be a more realistic representation of the interbank market. In order to encapsulate the different kinds of possible connections among banks, recent empirical studies focus on multilayer networks. A multilayer network is a system where the same set of nodes belong to different layers, and each layer is characterized by its own kind of edge, topology and its own dynamics in the spread of the initial shock.

### **2.2.3 Tiering in the interbank market**

According to Craig and von Peter (2014), an interbank market is tiered, operating in a hierarchical fashion, when few banks (core banks) intermediate between other banks (periphery banks) that do not transact with each other. Tiering is considered to be a structural property of the network, not a property of any individual bank. Recent empirical studies find evidence that a lot of interbank markets have a core-periphery structure.

Craig and von Peter (2014), for example, find a core-periphery structure in the German banking network , in which the core comprises only 2% of the banks in the system. This core-periphery structure appears to be stable over time. The authors also find that bank-level features such as connectedness and balance sheet size reliably predict which banks position themselves in the core and which remain in the periphery. The finding that big and well-connected banks are more likely to be located in the core strengthens the notion that core-periphery structure is more realistic for banking systems.

Fricke and Lux (2015), using a dataset of the overnight interbank transactions on the e-MID trading platform from January 1999 to December 2010, find distinct core-periphery structure in the Italian interbank network. The identified core is very persistent over time, consisting of approximately 28 % of sample banks. The authors compare their findings with these of Craig and von Peter (2014), the substantial differences in the German and Italian interbank market data and conclude that the finding of a structure close to a core-periphery network is unlikely to be a



coincidence. Instead, they expect that other interbank markets display a similar hierarchical structure, which might be classified as a new "stylized fact" of modern interbank networks.

Aldasoro and Alves (2016) using data on exposures between large European banks broken down by both maturity and instrument type find also a core periphery structure which comprises a large core and positively correlated multiplexity. They also argue that these results highlight the importance of an institution's role in the channel of transmission in determining the global importance of such institution.

Other empirical studies that report a core-periphery structure for various interbank markets are: Langfield et al.(2014) for the UK interbank system, and Veld and van Lelyveld (2014) for the Dutch interbank market. In case of a core-periphery structure, system's fragility depends largely on the position of the affected node within the system. Intuitively, the failure of a core bank rather than the failure of a periphery bank can be detrimental in the stability of the whole system. Thus, it is of paramount importance for researchers and policy makers to be able to identify the systemically important banks.

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### 3. Theoretical Framework

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#### 3.1 Introduction

The global financial system can be represented as a large complex network in which banks, hedge funds and other financial institutions are interconnected to each other through direct and indirect financial linkages. In normal times, institutions' connections may result in efficient risk sharing but in turbulent periods these connections can harm financial stability and facilitate contagion as initial shocks may lead to chains of defaults or liquidity shortages with repercussions on the real economy. During the last decade, a lot of attention has been paid to the understanding of the structure of this network and the extent to which it contributes to systemic fragility. Broadly speaking, the system becomes fragile and breaks down when existing financial links turn from being a means of risk diversification to channels for the propagation of risk across financial institutions.

#### 3.2 Financial Linkages and Contagion

According to Upper (2011), interbank contagion can take place through a multiple of channels. The channels through which a shock spread can be broken down into two groups: indirect and direct contagion channels.

Direct contagion channel results from the direct interbank linkages between banks and can happen when an idiosyncratic shock travel through the network of banks and affect the balance sheets of multiple agents. This shock can be due to inability of some banks to meet their obligations (failures of some banks which had lost funds due to defaults by their debtors) or -to put it more simply-when interbank exposures are large relative to the lender's capital.

The possibility of the occurrence and spread of direct contagion depends mainly on the structure and size of the interbank market. According to the recent literature on interbank contagion, there are three major types of interbank market structure. Allen and Gale (2000) state that the interbank structure can be complete or incomplete, with contagion being less likely in the case of a complete structure, i.e the case where the bank has symmetric linkages with all other banks in the banking system. There is also a third type of interbank market structure which is defined as the money center structure<sup>3</sup>.

On the other hand, indirect contagion is created by indirect linkages between banks. Allen and Babus (2008) argue that these linkages in interbank network include identical assets, portfolio returns and overlapping portfolios. If, for example, a bank

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<sup>3</sup>This structure which has been developed by Freixas et al. (2000) implies a symmetrical linkage of a "money centre" bank to other banks, but without any mutual links among other (peripheral) banks. In this network structure, the failure of a money centre bank-the core bank- can cause interbank contagion, while the failure of a peripheral bank can only affect the neighboring banks.

holds identical assets with other banks the correlation between their portfolios can cause fire sales in the market during a crisis period, depressing thus overall prices in the market and inducing significant losses for all the participants. The fear of losses on interbank loans or the uncertainty of the counterparty can make banks hesitant to extend credit and even induce them to hoard liquidity. With liquidity hoarding, it becomes harder for banks that were previously borrowing from other banks to comply with their own liquidity requirements.

Distinguishing between the various contagion channels is crucial if the intention is to prevent contagion, since this will affect which policy measures are likely to be effective. Since bailouts are undesirable because of moral hazard considerations, ex ante measures should be considered in order to limit the possibility of contagion in case any shocks hit the interbank system.

### **3.3 Network Theory**

A promising approach to study and assess systemic risk in various financial systems originates from network theory and has been widely applied to ecology, neuroscience, statistical physics, epidemiology, sociology and computer science. Applications of network theory include logistical networks, the World Wide Web, Internet, metabolic networks, etc.

The foundation of the field of network theory dates back to the 18<sup>th</sup> century when Swiss mathematician Leonhard Euler (1707-1783) solved the problem of how best to circumnavigate the Bridges of Königsberg. There were several contributions in the last two centuries with the most prominent being that of Erdős-Rényi (1960). The two Hungarian mathematicians Paul Erdős and Alfred Rényi reestablished network theory with papers on random graphs and paved the way for further development on the network theory.

Continuous development of complex network theory research and computing power since the late 1990s has offered a whole new dimension for studying connections in large, complex and dynamically evolving network systems and has already started to be applied to fields like physics, biology, computer science, sociology, epidemiology, and economics among others. Even though many relationships exist between financial institutions, we focus on banks and the connections that form between other banks through interbank lending or borrowing.

During the last decade, many researchers have applied the tools of network theory that is ideally suitable for the analysis of interconnected systems and the study of systemic events. An interbank network can be described by a set of nodes-one for each bank-and a set of weighted and directed links representing the various interbank relationships between those banks.

Among the many relationships between banks, lending relationship plays an important liquidity insurance tool and facilitates the flow of credit between them. For the lending bank the loan will be on the asset side of its balance sheet, while for the

borrowing bank the loan will be on the liability side of its balance sheet. The representation of this relationship can be done through a link, for example an outgoing link of bank A to bank B and this means that bank A lends money to bank B and bank B borrows money from bank A. In the same way, the weight attributed to each edge is equal to the size of the loan.

In order to describe the network topology of a banking system, one can resort to measures from network theory. There are four properties that are usually used to describe a network. The first one is the size of the network, given by the number of nodes in the network and the edges which represent the existence of credit/lending relationship between two parties. The weight of each edge might be proportional to the magnitude of the exposure between two banks, while the directionality of each edge shows who is the creditor and who is the lender.

The second measure is the connectivity of the interbank market. Connectivity or connection level is described as the fraction of actual edges to possible edges between nodes. It can range from 0 (no interconnections) to 1 (every bank is connected to every other bank). In normal times a high connection level can lead to an enhanced liquidity allocation and increased risk sharing in the banking system.

The third quantity that is used to determine the structure of the interbank system is the average path length, which is defined as the average number of connections that is needed to transfer liquidity from one bank to another (or the average distance of a node to every other node in the network-or the average length of shortest paths for all pairs of nodes). In normal times a small average path length indicates a well connected system, where liquidity can easily be transferred from one bank to another. In times of crises, however, a short average path length also implies that an exogenous contagious event can spread faster through the system.

Finally, the fourth measure of the network topology is the clustering coefficient<sup>4</sup>, which is defined as the probability of two banks being exposed to each other, if both of them are exposed to a common third bank. A high clustering coefficient indicates a well connected interbank system where in normal times banks can distribute liquidity and share risk widely in the system while in times of crises, high clustering coefficient amplify the contagion effects and increases the risk of joint bank defaults.

The flourishing literature which ensued has developed two distinct methodologies that use complex networks to analyze issues related to financial stability and shock propagation. The first methodology applies counterfactual simulations to assess the danger of contagion in a range of national banking systems while the other analyses the topological structure of interbank networks in order to assess their stability. Over the last few years, the network structure of banking systems in different countries has been studied by many academics. Iori et al. (2008) studied the network topology of the Italian banking system, Boss et al.(2004) studied the network structure of the Austrian interbank market and Wells (2002) analyzed the UK interbank system. The

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<sup>4</sup> According to Husser (2015), clustering coefficients measure the tendency of linked nodes to have common neighbors.

second methodology that describes the topological structure of a network tries to explain how bank and market characteristics such as bank heterogeneity, moral hazard, price changes and capital requirements interact with network connectivity in determining the stability and resilience of the financial system.

### **3.4 Network Formation models**

Analyzing and fully understanding the network formation processes is of paramount importance for someone who wants to study the implications of interbank contagion risks. Understanding the emergence process of the interbank networks can be critical to control and mitigate these risks. According to the recent interbank networks literature, there are three ways to model network formation.

The first area of the literature, which will monopolize our attention, uses random link formation which is based on network growth models. These growth models which are randomly produced are based on the notion that new nodes are generated over time and form attachments to nodes that already exist when they are generated. Thus, an empty network with no links between the nodes is given and then nodes are connected in a random manner following a stochastic process or a process that takes account some characteristics of the nodes. In this category, one can discern four network models: Erdős–Rényi random graph model, the small world model, the scale-free model and the -recently introduced in the relative literature-fitness model.

Erdős–Rényi random graph model which is one of the earliest theoretical network models was introduced by Erdős and Rényi (1960). In this random graph, each possible link between any two nodes can occur with a certain independent and identical probability-the Erdős and Rényi probability. The Erdős and Rényi (1960) random graph model is a model in which has been extensively applied for the study of contagion in financial networks, e.g. in the contributions from Nier et al. (2007), Gai and Kapadia (2010), Iori et al. (2006) and Montagna and Kok (2013). A number of alternatives models have been recently developed that differ in the probability law governing the distribution of links between nodes.

The small world model which was introduced by Watts and Strogatz (1998) is a graph network model that has two main features: small average shortest path length and a clustering coefficient significantly higher than expected by random chance. More specifically, this model has the so called "small world property" which refers to networks where, although the network size is large and each node has a small number of direct neighbors, the distance between any two nodes is very small compared to the network size. The small world model is a model in which has been extensively applied for the study of contagion in financial networks, e.g. in the contributions from Boss et al. (2004), Gai and Kapadia (2011) and Pegoraro (2012).

The property of a fat tail in the degree distribution has been observed in many types of networks and has led to the development of scale-free models by Barabási and Albert (1999). Scale free networks exhibit a degree-distribution that follows a power law and are often characterized by growth and preferential attachment. It has been observed

that the number of nodes in these models increases over time and each of them enter the network adding new edges (“growth”) which are then linked to the existing nodes according to a particular pattern –usually referred as preferential attachment. Preferential attachment by banks could result from the wish to interact with the most reliable counterparties. Banks who initially have the largest number of interactions will attract more linkages over time.

The distinctive feature of a scale free network is the existence of nodes with very different degree, and in particular the existence of hubs with a large number of connections. This property can have a large impact on the resilience of the system in the case of the failure of a hub. However, scale free networks are generally more resilient than other network models, but are extremely fragile if the most connected institution is in distress. In the literature, it is often argued that a more adequate model of a financial system is a scale-free network (see, for example, Boss et al. (2004) and Soramäki et al. (2007)).

Differently from the other network formation models belonging in the first category, the fitness model generates a network structure where the attachment rules are governed by intrinsic node attractiveness, usually termed fitness. This fitness is a measure of attractiveness of a node and so of the probability of forming a link. Specifically, every node in the network is endowed with a fitness parameter and then connections are formed between nodes with a probability which is a function of the fitness of the nodes. Thus, fitness-based models can mimic a variety of network topologies and subject to some constraints, they can be tuned to reproduce a given type of degree distributions and even degree correlation functions. The fitness model has been used by De Masi et al. (2006) and Montagna and Lux (2013) in order to match the empirical features of real interbank networks.

The second area of the literature uses strategic network formation, where financial institutions form links at once, taking into account the repeated nature of interactions over links and the discounted value of future gains obtained through these links. A game theoretical modeling and analysis requires that agents know the game they play. In other words, players need to be aware of the shape of the network they belong to and the impact of the network on their gains. The models that use network game techniques may be useful in understanding how banks decide on the level of mutual exposures towards each other, for a given pattern of interconnections. Some of these decisions are rollover decisions(e.g rollover a loan after receiving a signal about the solvency of the borrower) by banks which are often modeled using game theory tools. Farboodi (2014) and Acemoglu et al. (2015), using game theoretical modeling, show how equilibrium networks may exhibit excessive counterparty risk.

Finally, in the third area of the existing literature, network formation is based on the grounds of portfolio optimization by banks. The basic notion is that banks allocate their interbank exposures while balancing the return and risk of counterparty default risk and then links are generated taking into account funding diversification benefits.

Thus, links emerge endogenously from the interaction of banks' borrowing and lending decisions.

### **3.5 Simulation methodology**

A starting point for someone who wants to study interbank contagion is to describe the links along which contagion may take place. In the banking system these links represent credit exposures between the various banks in the system. The structure of these exposures can be represented either graphically or in a matrix form via an exposure matrix.

An exposure matrix is a  $N \times N$  representation of bilateral exposures in the interbank market with zeros on the diagonal due to the fact that banks do not lend to themselves. For example, the issuance of a loan from bank  $i$  to bank  $j$  is denoted as the loan size in row  $i$  and column  $j$ . Owing to the limitations of data sources, interbank exposure matrices can only be estimated indirectly.

Some sources of this information on bilateral exposures can be found in reports provided by banks to their supervisors or credit registers or in balance sheet data. Another way bilateral exposures can be estimated is through payment data. This approach introduced in the literature by Furfine (2003) and it is based in the idea that any loan with a maturity of, say, one day, involves both a transfer of funds from the lender to the borrower on day zero and a payment of opposite sign on day one. Thus, one has to search all transactions of a payment database for possible repayments and then identify whether there has been a payment of the same amount minus interest but the opposite sign on the previous day. Using payment databases, one is able to determine the complete matrix of interbank exposures while in balance sheet data (for each bank) the only information that can be obtained is the aggregate amount lent to or borrowed from all banks.

On the other hand, when using balance sheet data the maximum entropy estimation is the most widely used method in determining the interbank exposure matrix. In this method, the aggregated interbank assets and liabilities disclosed in balance sheets are the only input information and the exposure matrix can be derived by maximizing its entropy. Some authors claim that this method is the least biased given that only limited information of the interbank market structure, namely the aggregated interbank assets and liabilities are available. To draw inferences on bilateral exposures, one has to make assumptions on how banks spread their interbank lending across their counterparties. The most common assumption in the literature according to Upper (2007) is to assume that banks spread their lending as evenly as possible given the assets and liabilities reported in the balance sheets of all other banks. Technically, this amounts to the maximization of entropy of the network's linkages. The concept for the maximization of entropy is exactly the same as in a Bayesian estimation. When the researcher is agnostic about a parameter, he tends to use a uniform distribution. The selection of a uniform distribution is to provide no information that would influence the estimates.

However, there are some drawbacks from using the entropy maximization tool. Some of these are the inability to reproduce some properties of real world interbank markets which tend to underestimate the possibility of contagion. The entropy maximization approach assumes that banks aim to maximize the dispersion of their interbank activity. This framework could alter the original market structure, i.e a concentrated money centre structure towards a complete structure. Another drawback from maximum entropy is that it requires access to the balance sheets of all potential counterparties. Practically, this limits maximum entropy tool to domestic exposures only, since the data collected by central banks tend to give a full picture only of domestic institutions. On the other hand, commercial datasets tend to include large banks only and thus neglect other potential counterparties. To assume that contagion is only driven by domestic exposures leads again to an underestimation of both the possibility and severity of contagion.

Some of the above drawbacks, according to Upper (2007), can be mitigated by combining the method of maximum entropy with other sources of information. Additional information that can be incorporated has to do with particular elements of the exposure matrix that are known exactly. This information include the maximum size of particular elements of exposure matrix that are also known due to regulatory constraints and finally the overall idea of the structure of the market e.g. the presence of tiering. The latter which cannot be illustrated in terms of simple equality constraints can arise if balance sheets cover different types of exposures rather book loans only without distinguishing between collateralized and uncollateralized exposures.

After having estimated the exposure matrix, the next step is the simulation process. The simulation starts by assuming a bank is unable to repay its obligations in the interbank market. The losses of the creditor banks are calculated. Contagious defaults generally arise when the losses as a result of the exposures to the defaulting banks exceed the capital (Tier I capital) of a creditor bank. After a bank in the interbank network defaults, the effects on other banks are calculated. The size of the effect depends on the size of the exposures between the banks represented by the value and the loss rate. Many researchers use a range of loss rates between 0 and 1 and compare the differing results while others endogenize the loss rate in their models (Elsinger et al. 2006). As each bank failure weakens the banks that survived in the first step, it may end up causing a chain reaction of defaults, resembling the fall of domino pieces. To estimate the number of banks that will default due to contagion is a difficult task because any further defaults reduce the value of assets and the losses given default of the defaulted banks. Eisenberg and Noe (2001) and Furfine (2003) develop default algorithms to deal with this problem.

According to Upper (2007), counterfactual simulations help to identify which banks are critical to the stability of the interbank system. This criticality is determined by the bank's size, the structure of their balance sheet and their location in interbank network. However, performing counterfactual simulations involves a lot of simplistic



assumptions (e.g. bank's limited liability, equal share across the lenders of losses on interbank assets, seniority of nonbank liabilities to interbank liabilities), some of which might bias downwards or upwards the results. As we have discussed above, insufficient data poses also a strong obstacle in the application of simulations. Finally, as many researchers would say, the counterfactual simulation methodology lacks behavioural foundations which makes it less suited for the analysis of policy options in contagion defaults. Better quality of data and robustness tests of the models would allow researchers to obtain more accurate and reliable estimates.

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## 4. Simulating Financial Contagion Dynamics in Random Interbank Networks

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This Chapter is part of the paper Leventides et al. (2019) “Simulating Financial Contagion Dynamics in Random Interbank Networks” published in the *Journal of Economic Behavior and Organization*. The purpose of this study is to assess the resilience of financial systems to exogenous shocks using techniques drawn from the theory of complex networks. We investigate by means of Monte Carlo simulations the fragility of several network topologies using a simple default model of contagion applied on interbank networks of varying sizes. We trigger a series of banking crises by exogenously failing each bank in the system and observe the propagation mechanisms that take effect within the system under different scenarios. Finally, we add to the existing literature by analyzing the interplay of several crucial drivers of interbank contagion, such as network topology, leverage, interconnectedness, heterogeneity and homogeneity across bank sizes and interbank exposures.

### 4.1 Set up

Our model is tailored to simulate default cascades triggered by an exogenous shock in an interbank network, in which the various financial institutions are randomly linked to one another by their bilateral claims. We first introduce the interbank network model, describe the default cascades initiated by a random negative shock on this network and analyze the parameters that affect interbank contagion.

#### 4.1.1 The interbank network

We assume that the banking system contains  $i=1, \dots, N$  banks. Every bank has its own balance sheet and the accounting equation holds at all times. Total assets are divided in three categories: interbank assets  $A_i^{IB}$ , other assets  $A_i^{OT}$  and cash reserves  $C_i$ . On the liabilities side of the balance sheet we have included: interbank liabilities  $L_i^{IB}$ , other liabilities  $L_i^{OT}$  and equity capital  $E_i$ . A schematic overview of the balance sheet is given in Table 4. 1. Although the proposed balance sheet structure does not capture all elements of a bank balance sheet, it includes all those positions that are relevant to our study.

Let's consider a finite set  $V = \{v_1, v_2, \dots, v_n\}$  of unspecified elements and let  $V \otimes V$  be the set of all ordered pairs  $[v_i, v_j]$  of the elements of  $V$ , where a relation on the set  $V$  is any subset  $E \subseteq V \otimes V$ . Following Gutman and Polanski (1987), we define a simple interbank network as the pair  $G = (V, E)$ , where  $V$  is a finite set of nodes and  $E$  is a symmetric relation on  $V$ . We consider the Hilbert space of squared summable functions on the set of nodes  $V$  of the network  $H := l^2(V)$ , and let  $\{|i\rangle, i \in V\}$  be a

complete orthonormal basis of  $l^2(V)$ . Estrada (2011) shows that the adjacency operator of the network acting in  $l^2(V)$  is defined as:

$$(Af)(u) := \sum_{v \in E} f(v), \quad f \in H, \quad i \in V \quad (4.1)$$

<b>Assets <math>A_i</math></b>	<b>Liabilities <math>L_i</math></b>
Interbank Assets $(A_i^{IB})$	Interbank Liabilities $(L_i^{IB})$
Other Assets $(A_i^{OT})$	Other Liabilities $(L_i^{OT})$
Cash $(C_i)$	Equity Capital $(E_i)$

Table 4. 1: Stylized Balance sheet structure.

The table presents a stylized balance sheet structure in the interbank network. Total assets are divided in three categories: Interbank assets  $(A_i^{IB})$ , other assets  $(A_i^{OT})$ , and cash reserves  $(C_i)$ . Total liabilities include: Interbank liabilities  $(L_i^{IB})$ , other liabilities  $(L_i^{OT})$ , and equity capital  $(E_i)$ . It is assumed that the accounting equation holds at all times.

We further consider  $A$  as a  $|V| \times |V|$  matrix. For our network  $G = (V, E)$  the entries of the adjacency matrix are defined as

$$A_{ij} = \begin{cases} 1 & \text{if } i, j \in E \\ 0 & \text{otherwise} \end{cases}$$

The  $u$ th row or column of  $A$  has  $k_u$  entries, where  $k_u$  is the *degree* of the node  $u$ , which is simply the number of nearest neighbours that  $u$  has. Denoting by  $\mathbf{1}$  a  $|V| \times 1$  vector, the column vector of node degrees  $\kappa$  is given by

$$\kappa = (\mathbf{1}^T A)^T = A\mathbf{1} \quad (4.2)$$

We define the *indegree* as the number of links pointing toward a given node, and the *outdegree* as the number of links departing from the corresponding node. Specifically:

$$\kappa^{in} = (\mathbf{1}^T A)^T \quad (4.3)$$

$$\kappa^{out} = A\mathbf{1} \quad (4.4)$$

Thus, our interbank network of credit exposures between  $n$  banks can be visualized by a graph  $G = (V, E)$  where  $V$  represents the set of financial institutions – nodes, and  $E$  is the set of the edges linking the banks, that is, the set of ordered couples  $(i, j) \in V \otimes V$  indicating the presence of a loan made by bank  $i$  to bank  $j$ . The

number of nodes defines the size of the interbank network. Every edge  $(i, j)$  is weighted by the face value of the interbank claim and the representation of interbank claims is made by a single weighted  $N \times N$  matrix  $X$ :

$$X = \begin{bmatrix} 0 & \cdots & x_{1j} & \cdots & x_{1N} \\ \vdots & \ddots & \vdots & \ddots & \vdots \\ x_{i1} & \cdots & 0 & \cdots & x_{iN} \\ \vdots & \ddots & \vdots & \ddots & \vdots \\ x_{N1} & \cdots & x_{Nj} & \cdots & 0 \end{bmatrix}$$

where  $x_{ij}$  is the credit exposure of bank  $i$  vis-à-vis bank  $j$  and  $N$  is the number of banks in the network. Interbank assets are represented along the rows while columns represent interbank liabilities. Once  $X$  is in place, the interbank entries of each bank are given according to the following rules:

- (i)  $A_i = \sum_{j=1}^N x_{ij}$  (horizontal summation), where  $A_i$  is the total interbank assets of bank  $i$ .
- (ii)  $L_j = \sum_{i=1}^N x_{ij}$  (vertical summation), where  $L_j$  is the summation of the total interbank liabilities of bank  $j$ .

One can observe that the diagonal line contains zeros due to the fact that banks do not lend to themselves. In this framework, a random direct network of interbank loans is generated, in which we let vary the outdegree (number of outgoing links) of each node in the system. The outdegree of a bank corresponds to the number of debtors, the indegree corresponds to the number of creditors, while the sum of these two measures gives the degree of each node. The degree of a node is a measure of connectivity which can be both a risk sharing and a risk amplification device<sup>5</sup>. An example of an interbank network, consisting of  $n=20$  banks is provided in Figure 4. 1.

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<sup>5</sup>Following Somaräki et al. (2007), connectivity ( $p$ ) can be defined as the unconditional probability that two nodes share a link and equals  $p=m/n(n-1)$  for a directed network, where  $n$  is the number of nodes and  $m$  the number of links within the network.

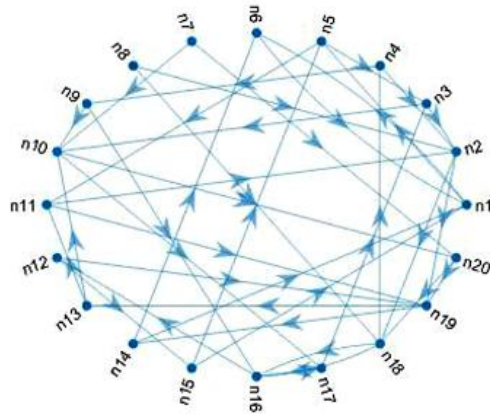


Figure 4. 1. The graph of an interbank network consisting of  $N=20$  banks and two outgoing/incoming links across banks in the network.

The network structure has been generated randomly and the arrows in the graph indicate the direction of links: incoming links represent assets, outgoing links represent liabilities.

#### 4.1.2 Shock propagation & contagion dynamics

The failure of a bank can affect other banks through their interbank connections. Below, we describe the mechanism through which an initial shock affecting a bank propagates onto its counterparties along the network. Contrary to the recent literature, the term contagion here translates into total capital losses due to multiple default cascades. The cascade dynamics we use in this study are straightforward to implement and enable us to run a great number of simulations on a variety of different scenarios.

The default procedure starts with an exogenous shock being simulated, typically by setting to zero the equity of one randomly chosen bank  $i$  and the cascade of defaults proceeds on a timestep-by-timestep basis, assuming zero recovery for shock transmissions. The zero recovery assumption, which is a realistic one in the short run, is often used in the literature to analyze worst case scenarios and refers to a situation where creditor banks lose all of their interbank assets held against a defaulting bank (Gai and Kapadia, 2010; Chinazzi et al., 2015). A bank's default implies that it is no longer able to meet its interbank liabilities to its counterparties. Since these liabilities constitute other banks' assets, the banks that get into trouble affect simultaneously their counterparties, leading to write-downs in their balance sheets. The interbank asset loss due to failure of bank  $i$  is subtracted from the bank's  $j$  capital. Bank  $j$  will fail if its exposure against bank  $i$  exceeds its equity. A second round of bank failure occurs if bank  $s$  creditors cannot withstand the losses realized due to its default and eventually, contagion stops if no additional bank goes bankrupt, otherwise a third round of contagion takes place. An initial shock can be amplified through banks' interconnections and further transmitted to other institutions, such that the overall effect on the system goes largely beyond the original shock. As Upper and Worms (2004) demonstrate, in response to a liquidity shock banks prefer to withdraw their deposits at other banks instead of liquidating their long-term assets, creating further instability and liquidity dry-ups in the financial system.

A general mathematical description of the dynamical system expressing the shock propagation mechanism is presented hereafter. We consider a network consisting of  $N$  banks numbered from 1 to  $N$ . We define  $b_i$  as the capital possessed by bank  $i$  in the network and

$$b_0 = (b_1, b_2, \dots, b_N) \quad (4.5)$$

stands for the initial vector of bank capital.  $X$  is defined as a  $N \times N$  matrix with entries:

$x_{ij}$  = the credit exposure of bank  $i$  vis-à-vis bank  $j$  in the network

$$x_{ii} = b_i \quad (4.6)$$

We consider the case where some of the banks (one or more) collapse. We wish to study how the crisis travels through the bank network and when exactly it comes to a fixed point. The collapse of banks  $i_1, i_2, \dots, i_k$  (where  $k \leq N$ ), can be described in the following way. Consider the element  $x_0 \in Z_N^2 = \{0, 1\}^N$  which has zero entries everywhere except the positions  $i_1, i_2, \dots, i_k$  where  $x_0$  takes on the value 1. Then,

$$b_1 = b_0 - X \cdot x_0 \quad (4.7)$$

is the new vector of capital of the  $N$  banks. We now take

$$x_1(i) = \begin{cases} 1, & b_1(i) \leq 0; \\ 0, & b_1(i) > 0. \end{cases} \quad (4.8)$$

Then  $x_1 \in Z_2^N$  and  $x_1$  indicates the banks that have collapsed after the bankruptcy of the first  $k$  banks. The vector  $x_1$  takes on the value 1 in the positions  $i_1, i_2, \dots, i_k$ . If  $x_1 \neq x_0$ , this indicates that the collapse of the first  $k$  banks has adversely affected other banks leading them to bankruptcy. Similarly, from  $x_1$  we take:

$$b_2 = b_0 - X \cdot x_1 \quad (4.9)$$

and then

$$x_2(i) = \begin{cases} 1, & b_2(i) \leq 0; \\ 0, & b_2(i) > 0. \end{cases} \quad (4.10)$$

The vector  $x_2$  indicates the banks that collapse after the bankruptcy of the banks of  $x_1$ . Therefore, we have a map:

$$F: Z_2^N \rightarrow Z_2^N \quad (4.11)$$

$$x \rightarrow F(x) = f(b_0 - X \cdot x) \quad (4.12)$$

The map  $F(x)$  defines a dynamical system  $x_{n+1} = F(x_n)$  which describes the evolution of contagion in the interbank network.

The mechanics of contagion can be illustrated by a simple example. We assume that we work with an interbank network consisting of  $i=1,2,3,4$  four banks, which are equipped with a simple internal structure representing their balance sheet. The balance sheet information for each bank and the interbank relationships among banks can be represented in matrix form. We assume that the banks' equity is given by a random vector  $b_i, i=1, \dots, 4$  and their interbank exposure by a squared matrix  $A$  with zeros off the diagonal due to the fact that banks do not lend to themselves. We also assume that the outdegree for each bank is 2, which is set randomly among banks.

$$b = \begin{bmatrix} 10 \\ 20 \\ 30 \\ 40 \end{bmatrix}, \quad A = \begin{bmatrix} 0 & 20 & 0 & 10 \\ 20 & 0 & 30 & 0 \\ 0 & 30 & 0 & 10 \\ 20 & 0 & 10 & 0 \end{bmatrix}$$

The above matrix forms a stylized interbank network that allows the representation of a system of interbank claims by a single weighted  $4 \times 4$  matrix, in which interbank assets are shown along the rows and interbank liabilities along the columns. The total capital of the system is defined as the summation of vector  $b$  and all magnitudes are expressed in money terms, e.g. euros.

We initially assume that a negative shock wipes out the equity of the first bank – bank 1. Banks that are interconnected with this bank immediately record losses as bank 1 is unable to repay its liabilities, represented by the summation of the first column of the squared matrix. Note that bank 1 has borrowed the amount of 20€ from bank 2, denoted by the entry in the first column and second row, and the amount of 20€ from bank 4 denoted by the corresponding entry in the first column and the fourth row. Thus, both banks 2 and 4 record losses that reduce their equity. The interbank loans of bank 1 that cannot be repaid represent the loss of capital as a percentage of the total capital in the system, while bank's 1 equity is regarded as the initial loss of capital in the network.

Subsequent to bank's 1 default, bank 2 becomes insolvent while the amount of 20€ is subtracted from bank's 4 equity ( $40€ - 20€ = 20€$ ) making it more vulnerable to subsequent shocks.

Thus, the updated vector of equity is now given by vector:  $b' = \begin{bmatrix} 0 \\ 0 \\ 30 \\ 20 \end{bmatrix}$

The distress caused from the initial default of bank 1 continues to propagate within the interbank network due to banks' interconnectedness. Bank 2 has borrowed money from bank 1 and bank 3 that cannot be repaid, thus, the amount that bank 2 owes to bank 3 has to be subtracted from the equity of bank 3 ( $30\text{€}-30\text{€}=0\text{€}$ ). This domino effect continues with the default of bank 3, as bank's 2 default has wiped out its equity.

The updated vector of equity is now given by vector:  $b'' = \begin{bmatrix} -20 \\ 0 \\ 0 \\ 20 \end{bmatrix}$

Bank 3 has borrowed funds from bank 2, which has already gone bankrupt, and from bank 4. The amount borrowed from bank 4 (10€) cannot be repaid and has to be subtracted from the updated equity of bank 4 ( $20\text{€}-10\text{€}=10\text{€}$ )

A new updated vector  $b'''$  of banks' equity is:  $b''' = \begin{bmatrix} -20 \\ -30 \\ 0 \\ 10 \end{bmatrix}$

The default of bank 1 has caused a total loss of 60€ or 60% of the total capital in the system. The initial loss of capital by defaulting bank 1 is 10% of the total capital in the system while the loss of capital at the first stage (interbank loans that cannot be repaid) by defaulting bank 1 as percentage of the system's total capital is 40%. The leverage of the network system, which is defined as total interbank exposure over the total capital in the interbank network, is 1.50 or 150% which explains the default cascades in this network ( $(20+10+20+30+30+10+20+10)/100$ ).

In the above example, banks rely heavily on interbank borrowing which makes the network more vulnerable to a random financial shock. We have described how exactly the default of a single bank can propagate through the interbank network and cause other banks to fail due to contagion effects. The same procedure is repeated for the  $n$  bank in the interbank network which is impacted by the initial random shock.

## 4.2 Monte Carlo simulations

In this section we apply Monte Carlo simulations in four different stages. In the first stage, we specify the model that will be used. Moreover, we choose the probability distribution specified for the errors. We have selected to work under a student's  $t$  symmetric distribution. The second stage involves estimating the parameters of interest, i.e. the value of the coefficients in the regression model. In the third stage the test statistics of interest are saved, while in the fourth stage we go back to the first stage and repeat  $N$  times.



The quantity  $N$  is the number of replications which should be as large as is feasible. As Monte Carlo is based on random sampling from a given distribution (with results equal to their analytical counterparts asymptotically), setting a small number of replications will yield results that are sensitive to odd combinations of random number draws. Generally speaking, the sampling variation is measured by the standard error estimate, denoted  $S_x = \sqrt{\text{var}(x)/N}$ , where  $x$  denotes the value of the parameter of interest and  $\text{var}(x)$  is the variance of the estimates of the quantity of interest over the  $N$  replications.

In order to provide a general assessment of the various parameters that affect financial stability and can trigger contagion in an interbank network, we consider four different scenarios, in line with Chinazzi et al. (2015), where we let vary the degree of heterogeneity in the system, the balance sheet composition and the connectivity among banks. The four scenarios tested are as follows:

- Scenario 1:*
  - **Heterogeneous banks with homogeneous exposures.** In this scenario, we construct interbank networks where banks have different equity size and their interbank claims are evenly distributed among the outgoing links.
- Scenario 2:*
  - **Heterogeneous banks with heterogeneous exposures.** In this scenario, the interbank networks allow for heterogeneous bank sizes and heterogeneous interbank claims among banks.
- Scenario 3:*
  - **Homogeneous banks with heterogeneous exposures.** In this scenario, we construct interbank networks where banks have the same equity size and unevenly distribute their exposures across creditor banks.
- Scenario 4:*
  - **Homogeneous banks with homogeneous exposures.** In this last scenario, we construct interbank networks where banks have the same equity size and interbank claims are evenly distributed across creditor banks.

In each case, we allow the connectivity among banks to vary and the number of outgoing links of each bank lies within the range of 2 to 4 links. We examine banking systems consisting of small banks with low, medium and large interbank exposures, as well as systems of large banks with corresponding exposure levels.

We consider a basic model that uses only two components from a bank's balance sheet, that is, equity and interbank loans – in the words of May and Arinaminpathy (2010) ‘*a caricature for banking ecosystems*’. We generate our model in two separate steps. First, we construct a model structure of  $N$  nodes representing the banks in our system and randomly assign directed edges to represent lending-borrowing relationships, while in a second step, we assign each node to a stylized balance sheet

structure. Once the banking networks are created, the default propagation dynamics are implemented to examine the effects of an idiosyncratic shock hitting one bank.

The effect of a shock is simulated, typically by setting to zero the equity of the affected bank. We estimate the initial loss of capital by letting the first bank default and subsequently record the loss as percentage of the total capital in the system. Consequently, the defaulted bank will be unable to repay its creditors and the interbank loans that were granted will be written-off, as we have selected to work under a zero recovery assumption. This bad debt will be recorded and expressed as percentage of the total capital in the system. Moreover, the creditors of the defaulted bank will experience a shock on their balance sheets and the recorded losses will be subtracted from their equity.

If at any time the total losses realized by a bank exceed its net worth, the bank is deemed in default and is removed from the network. Note that timesteps are modeled as being discrete and there is the possibility that many banks default simultaneously in each timestep. These shocks propagate to their creditors and take effect in the next timestep. When no further failures are observed, the default procedure terminates and the total losses are recorded and expressed as percentage of the total capital in the financial system. Figures 4.A.1 and 4.A.2 in the Appendix formalize the aforementioned mechanism in a pseudocode which simulates the default cascade in the interbank network.

### 4.3 Computer experiments

Having generated banking systems via a network structure framework and balance sheet allocation, the dynamics of an initial shock affecting a bank within the interbank network can be investigated. Given the complexity of the interbank network outlined above, it is extremely difficult to derive analytical solutions. In order to obtain data to describe the variables that affect contagion, we employ several Monte Carlo simulations. In each realization, we construct an interbank network with  $N \in [20, 50, 80, 100]$  nodes. In a second step, we test the four scenarios mentioned before by varying the equity size of banks and the interbank exposure structure across creditor banks.

For each scenario tested we let the depth of connectivity across banks to vary, such that each bank can have two, three or four outgoing links with other banks. When homogeneity across bank sizes is considered, all banks are assumed to have the same equity size and thus, each bank is endowed with a balance sheet that consists of 100 units of equity. On the other hand, when homogeneity is present with respect to interbank exposures, interbank claims are randomly allocated within the interbank network and are categorized as follows: small loans granted (10 units), medium loans (20 units) and large loans (35 units). With respect to scenarios tested where heterogeneity of bank size is introduced, the amount of equity of each bank is drawn from a uniform distribution in the range:  $b_i \in [0, 100]$ , whereas when heterogeneity is

introduced with respect to interbank claims, credit is allocated in the following ranges:  $a_{ij} \in [0,10]$ ,  $a_{ij} \in [0,20]$ ,  $a_{ij} \in [0,35]$ <sup>6</sup>. Then balance sheets are assigned to each node, consistent with each specific scenario tested. We randomly choose a single bank in the system to default due to an exogenous shock and the default cascades proceed sequentially, assuming zero recovery. When no further failures are observed, results are recorded and another realization may begin soon after. We then impose another shock on the second bank in the network and this procedure continues until all banks in the interbank network are hit by an exogenous shock.

For each scenario tested and for each network size we have nine cases in which we allow the number of outgoing links ( $i = 2,3,4$ ) and the weight of outgoing links (small, medium and large interbank claims) to vary among banks. Each case gives us 2,000 realizations or, to put it differently, 2,000 banking crises. We deem that 2,000 realizations provide a satisfactory number of runs and robustness to our analysis. Thus, for each scenario tested and each network size we employ  $2,000 \times 9 = 18,000$  realizations using the following variables in each realization:

- Total loss of capital due to contagion as percentage of total capital in the system (**CATEND**). This variable can be written in algebraic form as follows:

$$CATEND = \frac{\sum_{i=1}^N b_i l_i}{\sum_{i=1}^N b_i}, \text{ where } l_i \text{ is either 1 or 0 depending on whether bank } i \text{ defaults at the end of the contagion process.}$$

- Initial loss of capital by defaulting bank  $i$  as percentage of total capital in the system (**CATIN1**), i.e. bank's  $i$  depleted equity divided by the total equity in the network:  $CATIN1 = \frac{b_i}{\sum_{i=1}^N b_i}$ , where  $b_i$ ,  $i=1, \dots, N$  is a random vector

$$\text{representing bank's equity.}$$

- Loss of capital at the first stage (interbank loans that cannot be repaid) by defaulting bank  $i$  as percentage of total capital in the system (**CATIN2**), i.e. total amount of loans granted to bank  $i$  that cannot be repaid divided by the

$$\text{total equity in the network: } CATIN2 = \frac{L_i}{\sum_{i=1}^N b_i} = \frac{\sum_{j=1}^N x_{ij}}{b_i}, \text{ where } L_i \text{ is the}$$

summation of the total interbank liabilities of defaulting bank  $i$ . Due to zero recovery assumptions, these liabilities that constitute other banks' assets are written-down from their balance sheets and are removed from the interbank

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<sup>6</sup>Although those ranges have been selected arbitrarily, they are not sensitive to any regression model employed in the following analysis and thus, our regression results will be unaffected in a qualitative manner if different ranges are used.

network. One could say that this is the loss of capital at the first stage of the contagion process.

- Leverage of the interbank network (**LEVIN**), i.e. total interbank exposures as measured by the sum of matrix's A elements, divided by the total capital in the

$$\text{network } LEVIN = \frac{\sum_{i=1}^N \sum_{j=1}^N x_{ij}}{\sum_{i=1}^N b_i}, \text{ where } x_{ij} \text{ is the credit exposure of bank } i \text{ vis-à-vis}$$

bank  $j$ .

- Number of outgoing links of bank  $i$  (**NOUTGOING**), i.e. the degree of a bank  $i$  which corresponds to the number of its creditors in the network. It is defined as the summation of the  $i$ th column of the adjacency matrix A.
- Shock propagation variable (**COUNT**) which measures the number of rounds needed until no further bank defaults.
- Variance of capital (equity) (**VARCAP**) used in those scenarios tested where only heterogeneous bank sizes are considered
- Variance of interbank loans (**VARLOANS**) used in those scenarios tested where only heterogeneous interbank loan exposures are considered

Our selection of variables is motivated by economic intuition and by the findings of previous studies on the dynamics of systemic risks (Nier et al., 2007). In order to study the effect the aforementioned variables have on contagion risk, we estimate the following ordinary least squares (OLS) models:

$$CATEND = \beta_1 CATIN1 + \beta_2 CATIN2 + \beta_3 LEVIN + \beta_4 NOUTGOING + \beta_5 COUNT + \beta_6 VARCAP \quad (4.13)$$

$$CATEND = \beta_1 CATIN1 + \beta_2 CATIN2 + \beta_3 LEVIN + \beta_4 NOUTGOING + \beta_5 COUNT + \beta_6 VARCAP + \beta_7 VARLOANS \quad (4.14)$$

$$CATEND = \beta_1 CATIN2 + \beta_2 LEVIN + \beta_3 NOUTGOING + \beta_4 COUNT + \beta_5 VARLOANS \quad (4.15)$$

$$CATEND = \beta_1 CATIN2 + \beta_2 LEVIN + \beta_3 NOUTGOING + \beta_4 COUNT \quad (4.16)$$

The model described in equation (4.13) is applied to scenarios involving heterogeneous bank sizes with homogeneous exposures in the network structure, equation (4.14) refers to a situation where emphasis is placed on heterogeneous interbank loan exposures combined with heterogeneous bank sizes, equation (4.15) takes into account homogeneous banks with heterogeneous exposures while equation (4.16) considers only homogeneous bank sizes and interbank claims. The variable CATIN1 has been omitted from equations (4.15)-(4.16) due to the fact that banks in the interbank system are homogeneous, i.e. we keep constant the equity of each bank and thus CATIN1 remains stable during our simulation runs.

#### 4.4 Simulation results

In this section, we discuss the regression results of all four scenarios. Since our variables are measured on different scales, we cannot directly infer which independent variable has the largest effect on the dependent variable. In order to circumvent this problem we standardize our series to have zero mean and unit variance. Table 4.3 presents the regression results of the first scenario using the OLS model described in equation (4.13), where heterogeneous banks distribute evenly their interbank claims across the outgoing links of a network consisting of  $N = 20, 50, 80$  and  $100$  banks. All regressor coefficients are found to be statistically significant in all cases regardless of the size of the network.  $R$ -squared coefficients take on large values ranging from 72 to 76 percent and highlight the ability of our selected variables to explain financial distress in interbank networks.

The variable CATIN1 captures the initial effect defaulting bank  $i$  exerts on the network, whereas the magnitude of interconnectedness across the banks that comprise the interbank network is measured through parameter CATIN2. Financial shocks will propagate into the defaulting bank's counterparties along the network, erode their capital and make them more vulnerable to subsequent shocks. The magnitude of the positive relationship between the parameter CATIN2 and CATEND – the dependent variable - seems to decrease as the size of the interbank network increases. This finding implies that as we move from smaller to larger network settings, systemic risk and the likelihood of contagion declines. Figure 4.2 visually illustrates the extent of contagion as a function of the percentage loss of capital due to bank's  $i$  default. It is shown that as the network size increases capital losses decline, confirming the findings from the regression model.

As expected, we also find that there is a positive relationship between the leverage of the network and the capital losses due to contagion. This result is in line with the findings of Nier et al. (2007) who provide evidence that systemic risk increases when system-wide leverage increases. Highly leveraged banks in the interbank network are clearly more exposed to the risk of default on interbank loans. The greater the size of default on debt is, the larger the losses are that banks transmit to their neighbors, other things being equal. Thus, highly leveraged banks contribute more to systemic risk as they become a vehicle for transmitting shocks within the network. Moreover, it is shown that the magnitude of the positive relationship between the network's leverage and contagion risk increases as we move from smaller to larger interbank networks (illustrated in Figure 4.3).

Our results also suggest that connectivity, expressed in our experiments as the outdegree of the first bank that defaults, has a negative effect on interbank contagion. The fact that we have allowed connectivity in the network to vary, has provided additional resilience to it. Interestingly, the magnitude of connectivity decreases as the size of the network increases. In relatively small interbank networks, a high level of connectivity will allow an efficient absorption of shocks, whereas in larger networks the increased connectivity will spread the shock throughout the system, potentially

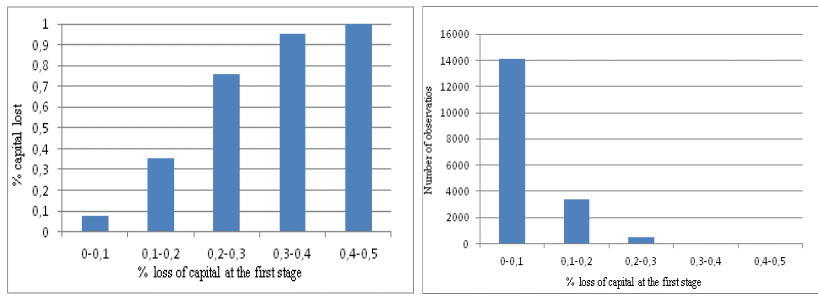
leading to market-wide collapses. Our regression analysis also shows that the COUNT variable which measures the number of rounds until no further bank defaults, has a positive impact on interbank contagion and this relationship becomes more statistically significant as the size of the network increases.

The size of heterogeneity expressed as the variance of capital exhibits a negative and statistically significant relationship with interbank contagion, showing that size heterogeneity can have positive effects on the stability of an interbank network. An interbank network consisting of banks of various sizes can more easily withstand a negative shock, therefore no institution becomes significant for either borrowing or lending. Furthermore, in such network both smaller and larger banks can act as shock absorbers when an initial shock hits the banking system, making contagion a less likely phenomenon. This finding is in line with the results of Iori et al. (2006) concerning bank size heterogeneity.

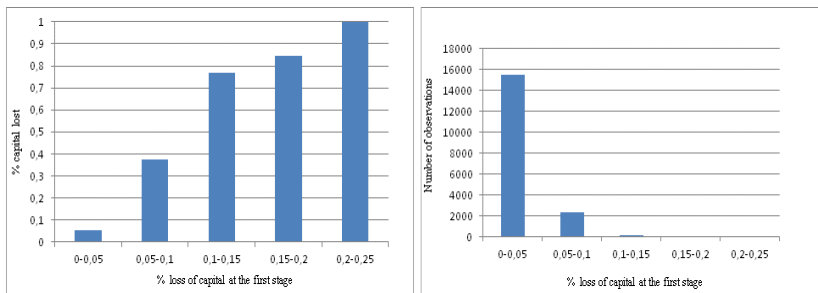
Table 4. 2: Summary statistics

The mean, median, and standard deviation are depicted for interbank networks consisting of 20, 50, 80, and 100 banks, respectively. Four scenarios are included: (a) Heterogeneous Banks – Homogeneous Exposures; (b) Heterogeneous Banks – Heterogeneous Exposures; (c) Homogeneous Banks – Heterogeneous Exposures; (d) Homogeneous Banks – Homogeneous Exposures. The variables are: CATEND, defined as total loss of capital due to contagion as percentage of total capital in the system; CATIN1, defined as bank's  $i$  depleted equity divided by the total equity in the network; CATIN2, defined as the total amount of loans granted to bank  $i$  that cannot be repaid, divided by the total equity in the network; LEVIN, defined as the leverage of the interbank network; NOUTGOING, defined as the number of outgoing links of bank  $i$ , which corresponds to the number of its creditors in the network; COUNT, defined as the number of rounds needed until no further bank defaults; VARCAP, defined as the variance of bank capital; VARLOANS, defined as the variance of interbank loans.

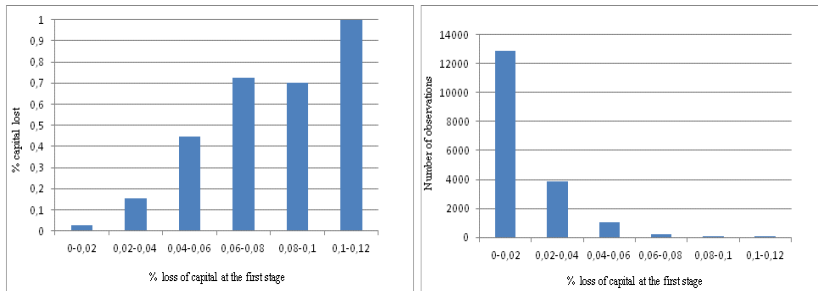
	Variable	HeterogeneousBanks – HomogeneousExposures			HeterogeneousBanks – HeterogeneousExposures			HomogeneousBanks – HeterogeneousExposures			HomogeneousBanks – HomogeneousExposures		
		Mean	Median	SD	Mean	Median	SD	Mean	Median	SD	Mean	Median	SD
N=20 banks	Catend	0.15	0.06	0.26	0.06	0.06	0.04	0.13	0.05	0.24	0.34	0.05	0.44
	Catin1	0.05	0.05	0.03	0.05	0.05	0.03	0.05	0.05	0.01	0.05	0.05	0.01
	Catin2	0.06	0.05	0.05	0.03	0.02	0.03	0.06	0.05	0.06	0.13	0.10	0.11
	Levin	1.32	1.16	0.76	0.66	0.57	0.38	1.30	1.19	0.74	2.60	2.40	1.45
	Noutgoing	3.00	3.00	1.78	3.00	3.00	1.76	3.00	3.00	1.78	3.00	3.00	1.77
	Count	2.31	1.00	2.17	1.44	1.00	0.93	1.73	1.00	1.93	2.21	1.00	1.92
	Varcap	823.65	816.14	180.86	832.28	831.65	173.46	-	-	-	-	-	-
	Varloans	-	-	-	48.24	33.08	40.86	47.63	33.19	40.16	-	-	-
N=50 banks	Catend	0.10	0.03	0.25	0.02	0.02	0.01	0.08	0.02	0.22	0.32	0.02	0.45
	Catin1	0.02	0.02	0.01	0.02	0.02	0.01	0.02	0.02	0.01	0.02	0.02	0.01
	Catin2	0.03	0.02	0.02	0.01	0.01	0.01	0.03	0.02	0.02	0.05	0.04	0.04
	Levin	1.31	1.20	0.74	0.65	0.59	0.37	1.30	1.20	0.73	2.60	2.41	1.46
	Noutgoing	3.00	3.00	1.86	3.00	3.00	1.87	3.00	3.00	1.86	3.00	3.00	1.88
	Count	2.61	1.00	2.92	1.44	1.00	0.98	1.94	1.00	2.78	2.61	1.00	2.57
	Varcap	833.07	828.49	106.61	832.60	828.68	106.71	-	-	-	-	-	-
	Varloans	-	-	-	48.22	33.43	40.27	48.22	33.13	40.43	-	-	-
N=80 banks	Catend	0.02	0.02	0.25	0.01	0.01	0.01	0.07	0.01	0.21	0.31	0.01	0.45
	Catin1	0.01	0.01	0.007	0.01	0.01	0.007	0.01	0.01	0.01	0.01	0.01	0.01
	Catin2	0.01	0.01	0.01	0.008	0.006	0.007	0.02	0.02	0.01	0.03	0.02	0.03
	Levin	1.22	1.22	0.73	0.65	0.60	0.37	1.30	1.21	0.74	2.60	2.40	1.46
	Noutgoing	3.00	3.00	1.88	3.00	3.00	1.86	3.00	3.00	1.89	3.00	3.00	1.88
	Count	1.00	1.00	3.62	1.48	1.00	1.06	2.00	1.00	3.01	2.81	1.00	2.87
	Varcap	830.24	830.24	82.45	833.91	838.49	90.70	-	-	-	-	-	-
	Varloans	-	-	-	47.87	32.77	40.06	47.79	33.48	39.54	-	-	-
N=100 banks	Catend	0.10	0.01	0.27	0.01	0.01	0.01	0.06	0.01	0.20	0.31	0.01	0.45
	Catin1	0.01	0.01	0.006	0.01	0.01	0.006	0.01	0.01	0.01	0.01	0.01	0.01
	Catin2	0.01	0.01	0.01	0.007	0.005	0.006	0.01	0.01	0.01	0.03	0.02	0.02
	Levin	1.31	1.16	0.74	0.66	0.60	0.38	1.30	1.20	0.73	2.60	2.41	1.45
	Noutgoing	3.00	3.00	1.87	3.00	3.00	1.88	3.00	3.00	1.90	3.00	3.00	1.90
	Count	3.10	1.00	4.27	1.49	1.00	1.18	2.04	1.00	3.23	2.88	1.00	3.00
	Varcap	836.04	841.54	72.45	836.40	836.88	70.78	-	-	-	-	-	-
	Varloans	-	-	-	48.22	33.43	40.06	47.86	33.49	39.48	-	-	-



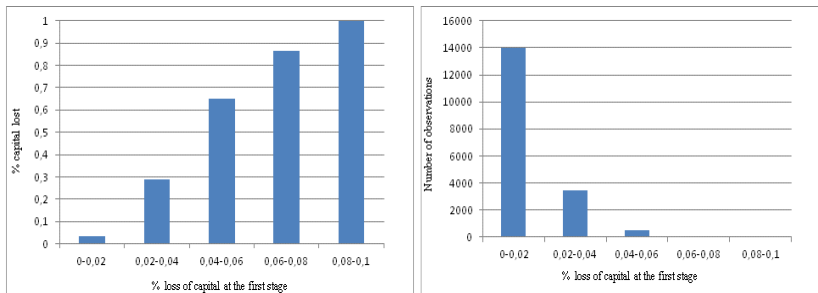
(a)  $N=20$  banks



(b)  $N=50$  banks



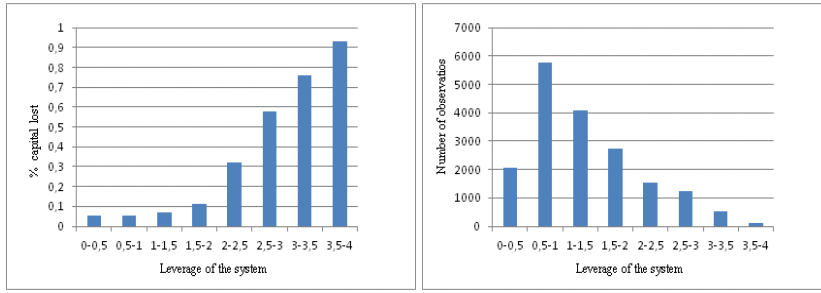
(c)  $N=80$  banks



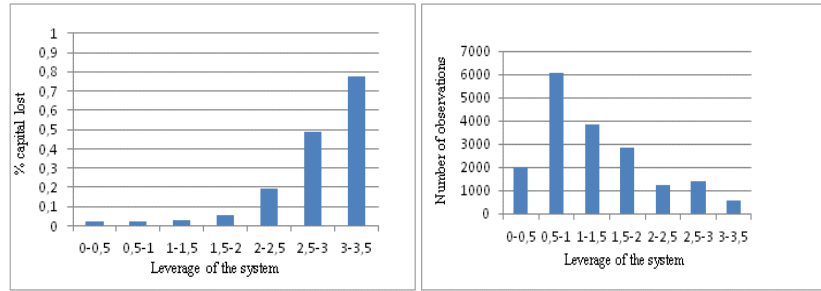
(d)  $N=100$  banks

Figure 4. 2: Scenario 1- Heterogeneous Banks with homogeneous exposures | Extent of contagion (expressed as the total capital lost from the banking system due to the failure of at least one bank) as a function of the % loss of capital at the first stage due to default of the first bank. Panels (a)-(d) show the relation between the % loss of capital and the extent of contagion across interbank networks with different number of banks.

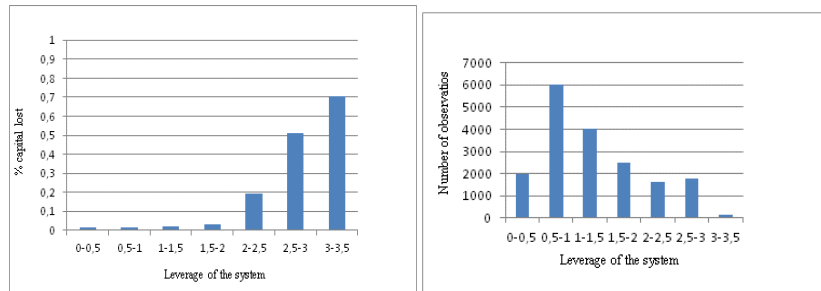




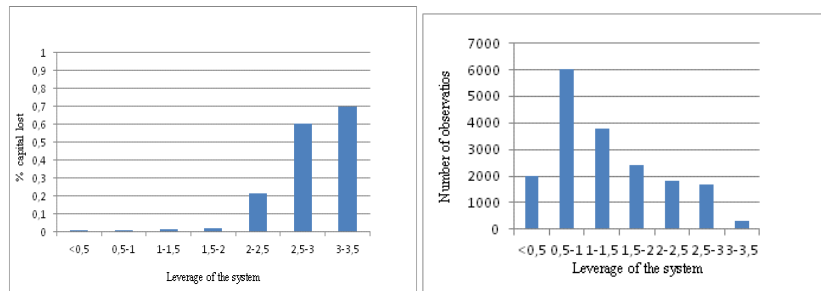
(a)  $N=20$  banks



(b)  $N=50$  banks



(c)  $N=80$  banks



(d)  $N=100$  banks

Figure 4. 3: Scenario 1- Heterogeneous Banks with homogeneous exposures | Extent of contagion (expressed as the total capital lost from the banking system due to the failure of at least one bank) as a function of the leverage of the system.

Panels (a)-(d) show the relation between the leverage of the system and the extent of contagion across interbank networks with different number of banks.

	<b>N=20</b>	<b>N=50</b>	<b>N=80</b>	<b>N=100</b>
<b>CATIN 1</b>	0.080 (23.029)***	0.030 (7.671)***	0.024 (6.245)***	0.017 (4.591)***
<b>CATIN2</b>	0.208 (24.266)***	0.088 (9.191)***	0.020 (2.109)**	0.023 (2.568)**
<b>LEVIN</b>	0.078 (13.376)***	0.131 (20.473)***	0.127 (19.898)***	0.160 (25.829)***
<b>NOUTGOING</b>	-0.147 (-25.061)***	-0.085 (-12.220)***	-0.023 (-3.212)***	-0.013 (-1.963)**
<b>COUNT</b>	0.721 (152.451)***	0.734 (147.324)***	0.762 (155.048)***	0.749 (158.296)***
<b>VARCAP</b>	-0.103 (-45.749)***	-0.063 (-39.920)***	-0.048 (-38.314)***	-0.042 (-39.818)***
<b>Adjusted R<sup>2</sup></b>	0.760	0.717	0.716	0.745

Table 4. 3: OLS regression analysis for Scenario 1 (Heterogeneous banks with homogeneous exposures).

The table presents the regression results for Scenario 1. The dependent variable is CATEND measured as the total loss of capital due to contagion as percentage of total capital in the network. Explanatory variables are CATIN1, CATIN2, LEVIN, NOUTGOING, COUNT and VARCAP. Each cell displays the OLS standardized coefficients along with the corresponding *t*-statistics (shown in parentheses). The sample comprises of 18,000 realizations (simulated banking crises). \*\* and \*\*\* denote significance at the 5 and 1 percent level, respectively.

	<b>N=20</b>	<b>N=50</b>	<b>N=80</b>	<b>N=100</b>
<b>CATIN 1</b>	0.670 (223.788)***	0.727 (271.376)***	0.640 (187.087)***	0.592 (177.760)***
<b>CATIN2</b>	0.121 (20.243)***	0.085 (15.635)***	0.064 (9.387)***	0.003 (0.524)
<b>LEVIN</b>	0.014 (2.616)***	-0.004 (-0.878)	0.068 (9.952)***	0.027 (4.186)***
<b>NOUTGOING</b>	-0.119 (-25.986)***	-0.081 (-18.756)***	-0.089 (-15.751)***	-0.060 (-11.101)***
<b>COUNT</b>	0.530 (145.530)***	0.541 (171.862)***	0.579 (147.590)***	0.674 (178.780)***
<b>VARCAP</b>	-0.101 (-53.995)***	-0.068 (-61.788)***	-0.061 (-50.322)***	-0.050 (-54.927)***
<b>VARLOANS</b>	-0.057 (-10.622)***	-0.023 (-4.303)***	-0.080 (-11.667)***	-0.034 (-5.396)***
<b>Adjusted R<sup>2</sup></b>	0.823	0.865	0.782	0.796

Table 4. 4: OLS regression analysis for Scenario 2 (Heterogeneous banks with heterogeneous exposures).

The table presents the regression results for Scenario 2. The dependent variable is CATEND measured as the total loss of capital due to contagion as percentage of total capital in the network. Explanatory variables are CATIN1, CATIN2, LEVIN, NOUTGOING, COUNT, VARCAP and VARLOANS. Each cell displays the OLS standardized coefficients along with the corresponding *t*-statistics (shown in parentheses). The sample comprises of 18,000 realizations (simulated banking crises). \*\*\* denotes significance at the 1 percent level.

Table 4.4 presents the regression results of the second scenario using the model described in equation (4.14), where banking institutions with heterogeneous bank sizes are linked to one another via heterogeneous interbank claims. The regressor coefficients are statistically significant in almost all cases and the *R*-squared values are quite high and lie in the vicinity of 78 to 86 percent, highlighting the good explanatory power of the model. CATIN1 impacts in a statistically significant manner the dependent variable in all network segments and the magnitude of standardized coefficients exceeds the corresponding magnitude of those derived from the first scenario. In other words, an initial shock from defaulting bank *i* will dissipate more easily and will not spillover in the network as intensively as in the first

scenario. Again, CATIN2 has a large positive impact on contagion risk, however, its magnitude fades away as we move from smaller to larger networks – in the last case of  $N = 100$  banks it becomes statistically insignificant. It should also be highlighted that the CATIN2 coefficients are smaller than those recorded in the first scenario when it comes to small and medium-sized networks, while the reverse holds for larger interbank markets. An initial shock following the default of bank  $i$  does not seem to contribute much to a banking crisis scenario within small and medium-sized networks and the size of total capital losses is smaller than that documented in the first scenario. Figure 4.4 depicts the extent of contagion as a function of the percentage loss of capital due to default of the first bank and confirms the results recorded in Table 4.4.

The results also show that there still exists a positive relationship between leverage and contagion, however, the coefficient estimates are much smaller than those recorded in the previous scenario. Moreover, the magnitude of the leverage coefficients increases as the number of banks in the interbank network increases, with the only exception being the 50 bank network segment which follows an autonomous path and is inversely related to contagion (although statistically insignificant). Results on connectivity are qualitatively similar to those of the first scenario, showing that connectivity negatively impacts contagion risk especially in larger interbank networks. The number of rounds until no further bank defaults positively impacts contagion risk and contributes the most to total capital losses in the banking system when large interbank networks are formed.

Under this scenario, the heterogeneity allowed on both bank sizes and interbank exposures has had a great impact on the resilience of the network system. Heterogeneity impacts negatively on interbank contagion although its intensity decreases as the size of the network increases. Moreover, we provide evidence that heterogeneity of bank size contributes more to the resilience of the interbank network than heterogeneity of interbank exposures. The heterogeneity of interbank exposures acts as a diversification tool and contributes to a smaller extent to an unfolding crisis compared to the scenario where homogeneous banks are interconnected via heterogeneous exposures (shown in Table 4.5).

	<b>N=20</b>	<b>N=50</b>	<b>N=80</b>	<b>N=100</b>
<b>CATIN2</b>	0.238 (51.837)***	0.166 (31.641)***	0.124 (21.978)***	0.085 (15.126)***
<b>LEVIN</b>	0.053 (11.723)***	0.080 (15.699)***	0.084 (15.145)***	0.089 (15.489)***
<b>NOUTGOING</b>	-0.186 (-61.481)***	-0.162 (-44.323)***	-0.147 (-37.350)***	-0.133 (-33.313)***
<b>COUNT</b>	0.875 (258.811)***	0.901 (247.480)***	0.911 (232.873)***	0.927 (241.892)***
<b>VARLOANS</b>	-0.199 (-41.960)***	-0.229 (-41.527)***	-0.235 (-39.356)***	-0.232 (-37.024)***
<b>Adjusted R<sup>2</sup></b>	0.887	0.856	0.834	0.835

Table 4. 5: OLS regression analysis for Scenario 3 (Homogeneous banks with heterogeneous exposures).

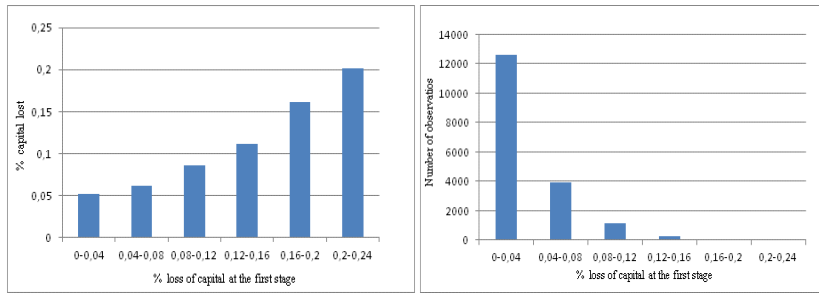
The table presents the regression results for Scenario 3. The dependent variable is CATEND measured as the total loss of capital due to contagion as percentage of total capital in the network. Explanatory

variables are CATIN2, LEVIN, NOUTGOING, COUNT and VARLOANS. Each cell displays the OLS standardized coefficients along with the corresponding  $t$ -statistics (shown in parentheses). The sample comprises of 18,000 realizations (simulated banking crises). \*\* and \*\*\* denote significance at the 5 and 1 percent level, respectively.

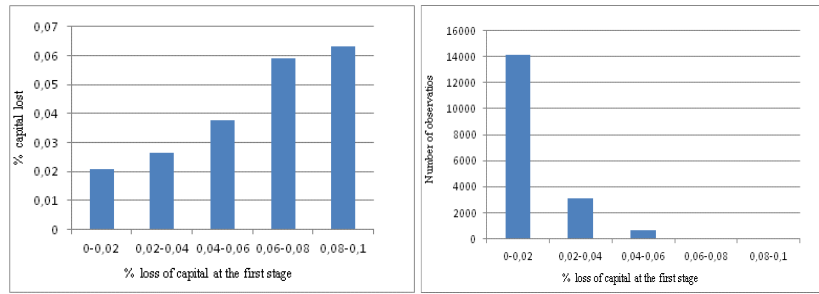
Table 4.5 depicts the results of the third scenario as described in equation (4.15). In this scenario, we construct network systems where banks have the same equity size and unevenly distribute their exposures across creditor banks. We note that an initial shock fades away with the failure of the first bank and does not spillover to other banks within the network. This is mainly due to our choice of parameters regarding the equity of each bank, the links among banks and the interbank claims among creditor banks. In order to observe default cascades we relax our initial assumptions and lower the equity of each bank in the network system. Specifically, each bank is now endowed with a balance sheet that consists of 25 units of equity and interbank claims among creditor banks are distributed in the following ranges:  $a_{ij} \in [0,10]$ ,  $a_{ij} \in [0,20]$ ,  $a_{ij} \in [0,35]$ .

Similar to the previous scenarios, the regressor coefficients are statistically significant in most cases and the  $R$ -squared values are still large, in fact the largest of all three scenarios tested. Variable CATIN2 has a highly significant positive impact on systemic risk that fades away as the network system gets larger. The same observation holds for the level of connectivity in the banking system i.e. a strong negative impact that dissipates as  $N$  increases. The leverage of the system has a positive impact on systemic risk and its magnitude increases as the size of the network increases. The standardized coefficients are much larger than those reported in the second scenario, implying that highly leveraged banks are less capable of absorbing negative shocks, something that can amplify the initial impact of a shock that is transmitted to neighbor banks via interlinkages. Figures 4.6 and 4.7 illustrate the third scenario as a function of the percentage loss of capital due to default of the first bank in the network and as a function of leverage in the banking system, respectively.

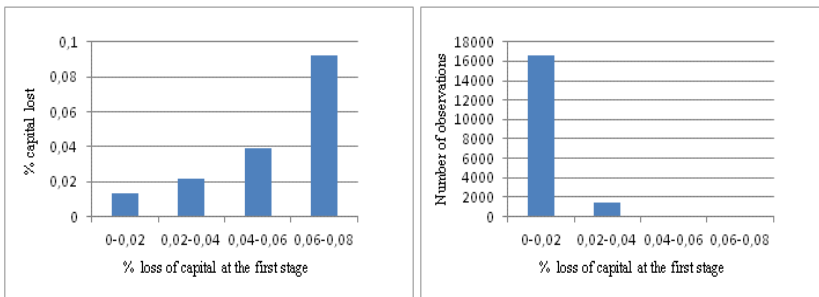
As in the previous cases, we find the number of rounds until no further bank defaults to affect contagion risk positively and statistically significantly, and such impact is magnified in relatively larger interbank networks. We also note that the standardized coefficients are more statistically significant than those reported in the first and second scenario. The heterogeneity of interbank exposures plays a significant role in the stability of the financial network and its impact declines with the number of banks included in the network, and such impact is stronger than that found in heterogeneous bank network settings.



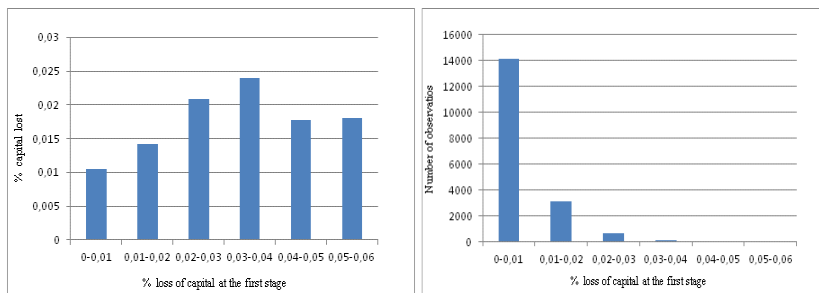
(a)  $N=20$  banks



(b)  $N=50$  banks



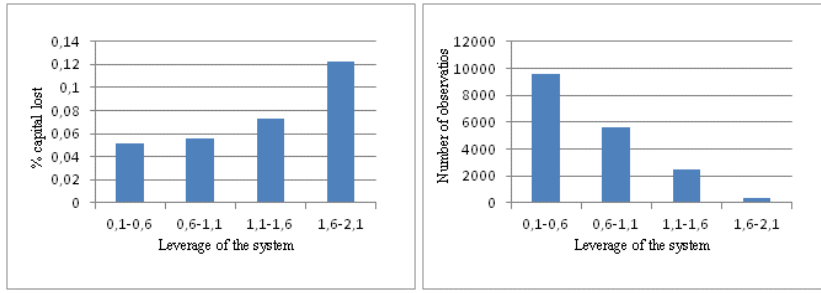
(c)  $N=80$  banks



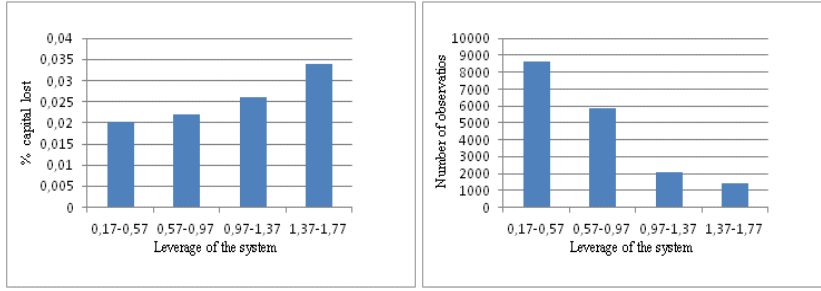
(d)  $N=100$  banks

Figure 4. 4: Scenario 2 - Heterogeneous Banks with heterogeneous exposures | Extent of contagion (expressed as the total capital lost from the banking system due to the failure of at least one bank) as a function of the % loss of capital at the first stage due to default of the first bank.

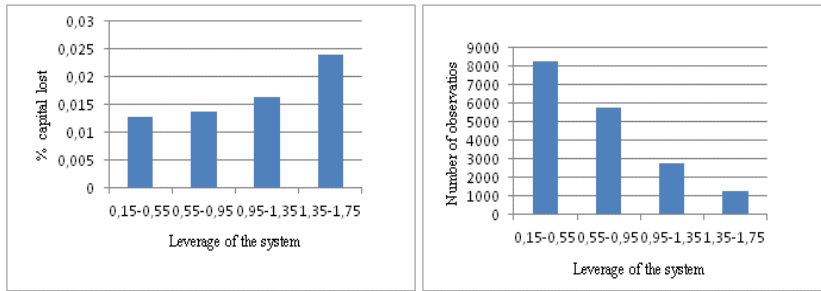
Panels (a)-(d) show the relation between the % loss of capital and the extent of contagion across interbank networks with different number of banks.



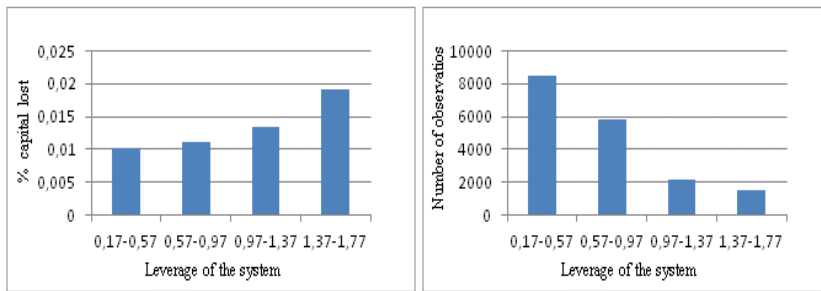
(a)  $N=20$  banks



(b)  $N=50$  banks



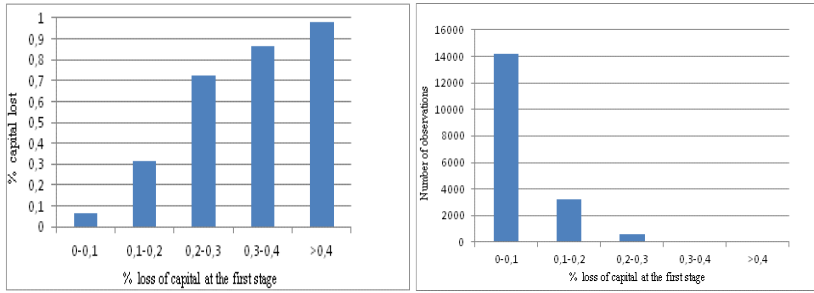
(c)  $N=80$  banks



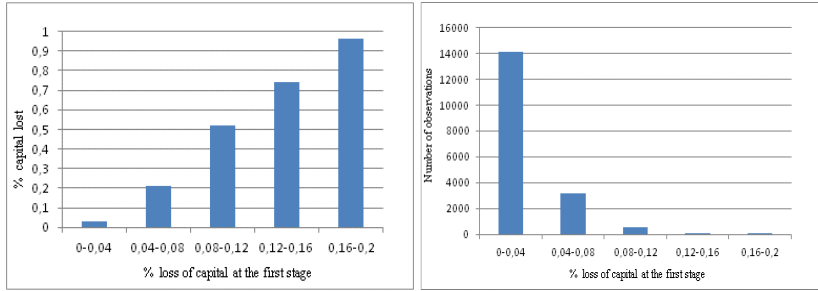
(d)  $N=100$  banks

Figure 4. 5: Scenario 2 - Heterogeneous Banks with heterogeneous exposures | Extent of contagion (expressed as the total capital lost from the banking system due to the failure of at least one bank) as a function of the leverage of the system.

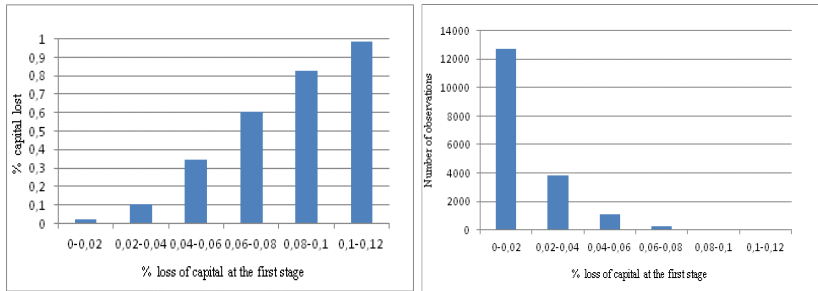
Panels (a)-(d) show the relation between the leverage of the system and the extent of contagion across interbank networks with different number of banks.



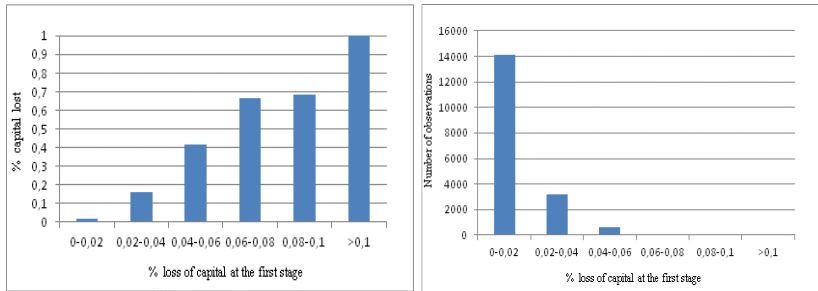
(a)  $N=20$  banks



(b)  $N=50$  banks



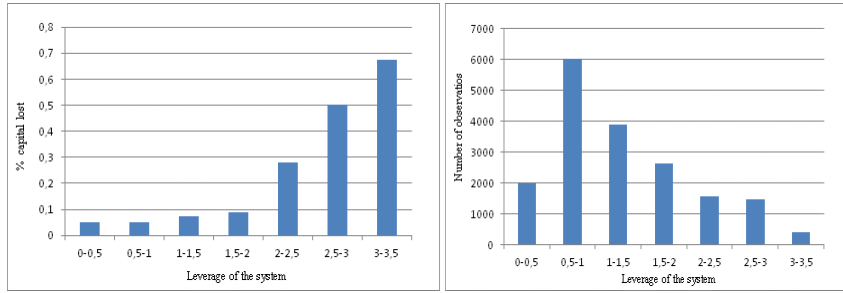
(c)  $N=80$  banks



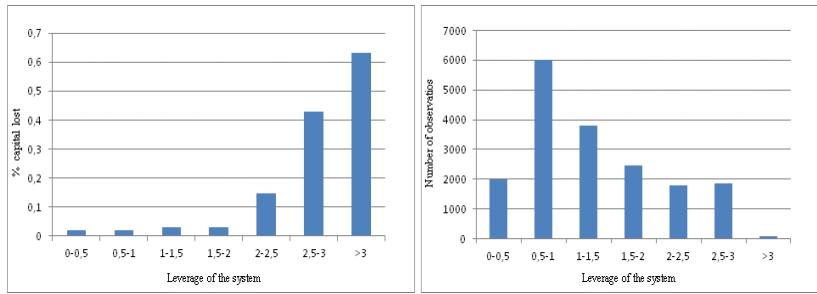
(d)  $N=100$  banks

Figure 4. 6: Scenario 3 - Homogeneous banks with heterogeneous exposures (expressed as the total capital lost from the banking system due to the failure of at least one bank) as a function of the % loss of capital at the first stage due to default of the first bank.

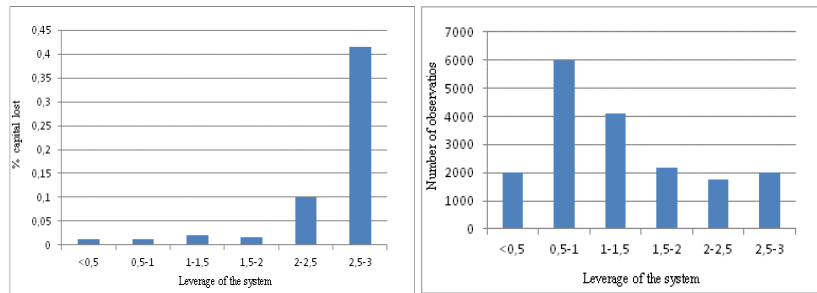
Panels (a)-(d) show the relation between the % loss of capital and the extent of contagion across interbank networks with different number of banks.



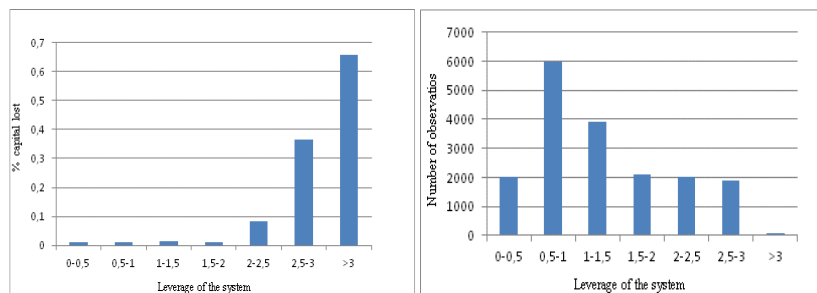
(a)  $N=20$  banks



(b)  $N=50$  banks



(c)  $N=80$  banks

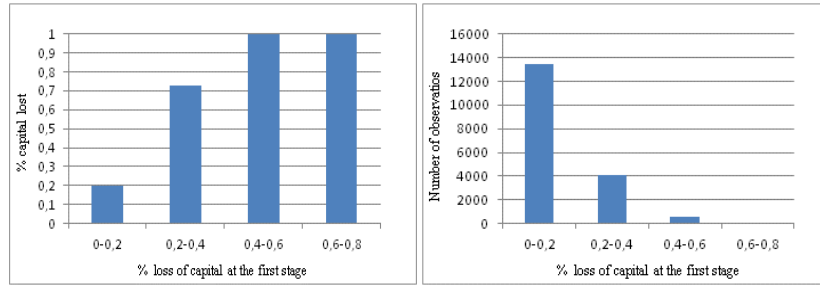


(d)  $N=100$  banks

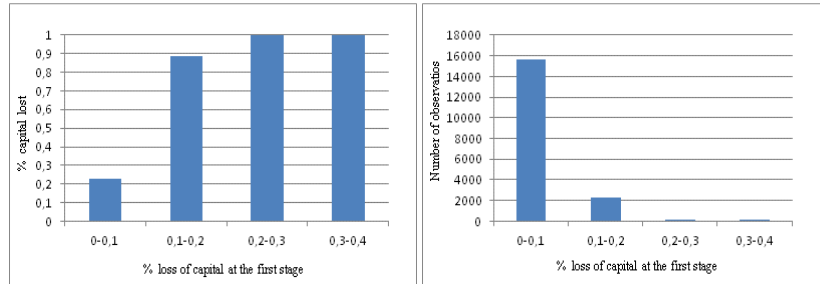
Figure 4. 7: Scenario 3 - Homogeneous banks with heterogeneous exposures | Extent of contagion (expressed as the total capital lost from the banking system due to the failure of at least one bank) as a function of the leverage of the system.

Panels (a)-(d) show the relation between the leverage of the system and the extent of contagion across interbank networks with different number of banks.

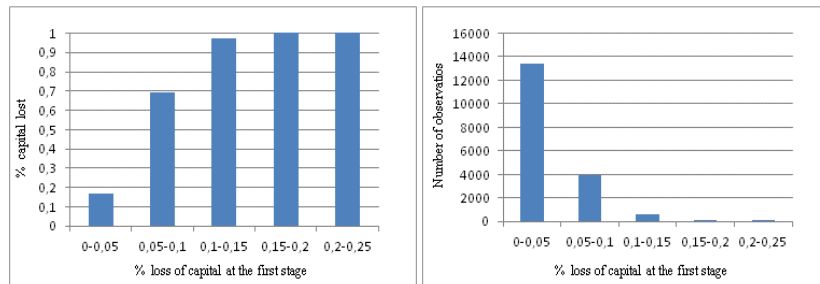




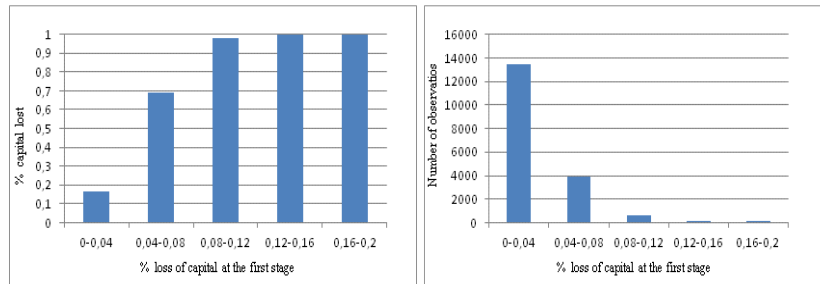
(a)  $N=20$  banks



(b)  $N=50$  banks

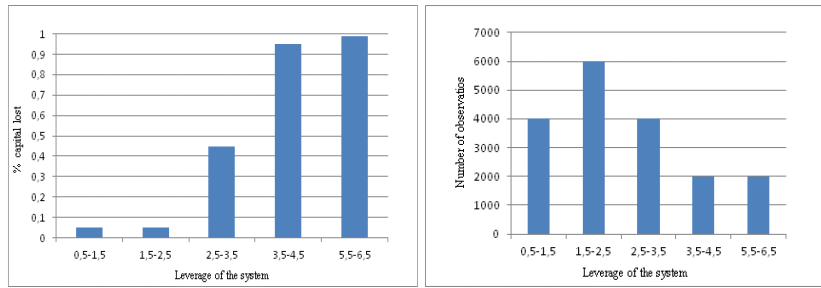


(c)  $N=80$  banks

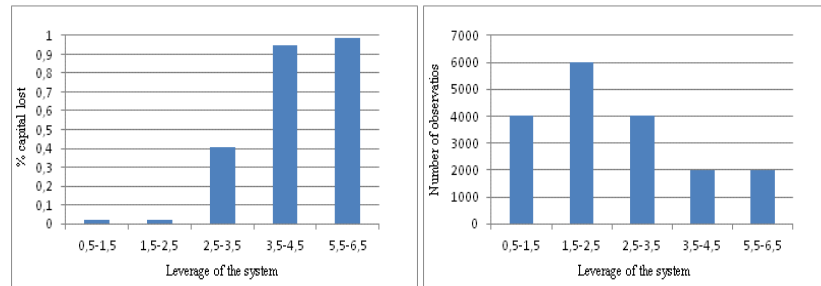


(d)  $N=100$  banks

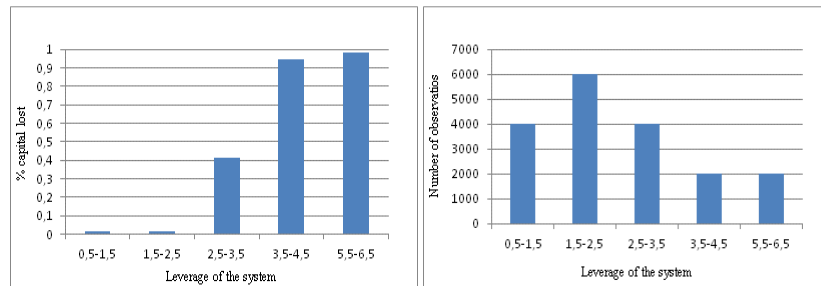
Figure 4. 8: Scenario 4 - Homogeneous banks with homogeneous exposures | Extent of contagion (expressed as the total capital lost from the banking system due to the failure of at least one bank) as a function of the % loss of capital at the first stage due to default of the first bank. Panels (a)-(d) show the relation between the % loss of capital and the extent of contagion across interbank networks with different number of banks.



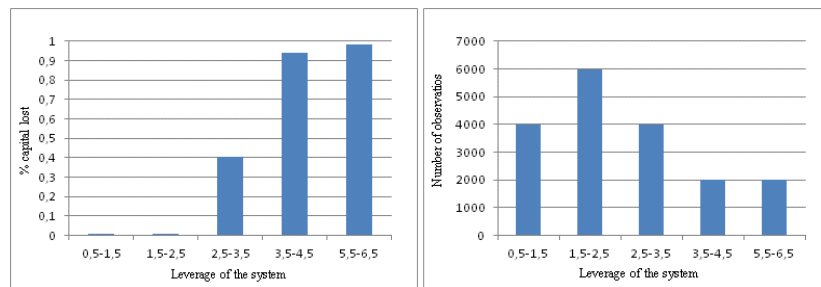
(a)  $N=20$  banks



(b)  $N=50$  banks



(c)  $N=80$  banks



(d)  $N=100$  banks

Figure 4. 9: Scenario 4 - Homogeneous banks with homogeneous exposures | Extent of contagion (expressed as the total capital lost from the banking system due to the failure of at least one bank) as a function of the leverage of the system.

Panels (a)-(d) show the relation between the leverage of the system and the extent of contagion across interbank networks with different number of banks.

Finally, Table 4.6 depicts the results of the fourth scenario as described in equation (4.16). In this scenario, we construct network systems where banks have the same equity size and interbank claims are evenly distributed across creditor banks. We acknowledge the fact that this scenario is a bit unrealistic as banks in real-world interbank networks do not have the same equity size and do not necessarily distribute their interbank claims evenly across their creditors. However, by varying the topology of the interbank market and the degree of heterogeneity of the system we are in a position to effectively examine the effect of different calibrations on systemic risk. Thus, although this scenario can be regarded as a special case with magnifying effects, it provides useful insights on interbank market resiliency during periods of stress.

The variable CATIN2 has a strong positive impact on systemic risk that dissipates as the network system gets larger. Simulations show that this scenario yields qualitatively similar results with the previous three scenarios in relation to the leverage of the network, that is, leverage positively and significantly affects contagion risk and such effect becomes stronger progressively when the number of constituent banks in the network increases. Figure 4.8 illustrates that the more leveraged a banking system is, the less resilient it becomes once a

	<b>N=20</b>	<b>N=50</b>	<b>N=80</b>	<b>N=100</b>
<b>CATIN2</b>	0.513 (136.409)***	0.484 (122.675)***	0.471 (122.258)** *	0.465 (121.843)***
<b>LEVIN</b>	0.006 (2.250)***	0.021 (8.188)***	0.027 (11.317)** *	0.024 (10.425)***
<b>NOUTGOING</b>	-0.340 (- 143.844)***	-0.335 (- 129.911)***	-0.331 (- 129.930)***	-0.326 (- 128.265)***
<b>COUNT</b>	0.652 (255.154)* **	0.660 (246.926)***	0.668 (257.520)** *	0.676 (262.331)** *
<b>Adjusted R<sup>2</sup></b>	0.933	0.929	0.932	0.934

Table 4. 6: OLS regression analysis for Scenario 4 (Homogeneous banks with homogeneous exposures).

The table presents the regression results for Scenario 4. The dependent variable is CATEND measured as the total loss of capital due to contagion as percentage of total capital in the network. Explanatory variables are CATIN2, LEVIN, NOUTGOING and COUNT. Each cell displays the OLS standardized coefficients along with the corresponding *t*-statistics (shown in parentheses). The sample comprises of 18,000 realizations (simulated banking crises). \*\*\* denotes significance at the 1 percent level.

random shock hits. For instance, for the less leveraged network systems (0.5% - 1.5%) and as the number of banks increases the total loss of capital due to contagion as percentage of total capital in the system drops to nearly 0%.

Figure 4.9 illustrates that the extent of contagion as a function of the percentage loss of capital in the network is magnified in this last scenario as capital losses exceed those documented in the previous scenarios. Connectivity impacts negatively on interbank contagion and follows a similar pattern to that of previous scenarios and dissipates as the number of banks in the network increases, although at a much slower rate than in previous cases. Finally, the number of rounds until no further bank

defaults affects contagion risk in a statistically significant manner especially when large interbank networks are considered.

The main intuition behind these results is that higher interconnectedness of a homogeneous interbank network can reduce the probability of contagion in case the first bank defaulting is less leveraged, as the shock will be absorbed by many counterparties and will dissipate at a faster rate. However, if the first bank defaulting is highly leveraged, the shock absorption capacity of the network will decrease and default cascades will prevail.

Tables 4.7–4.10 depict robustness tests on all four scenarios based on random sampling. We have performed second run Monte Carlo simulations in order to examine whether the new results differ from the previous ones, thus checking how random sampling affects our main conclusions. We observe qualitatively similar results in all four cases to those from the first run providing evidence that our findings are stable across different simulation scenarios.

	<b>N=20</b>	<b>N=50</b>	<b>N=80</b>	<b>N=100</b>
<b>CATIN 1</b>	0.082 (22.879)***	0.026 (6.695)***	0.025 (6.463)***	0.016 (3.958)***
<b>CATIN2</b>	0.195 (22.067)***	0.114 (11.875)***	0.036 (3.733)***	0.034 (3.453)***
<b>LEVIN</b>	0.071 (11.675)***	0.094 (14.620)***	0.140 (21.751)***	0.141 (21.143)***
<b>NOUTGOING</b>	-0.133 (-21.911)***	-0.090 (-12.822)***	-0.030 (-4.168)***	-0.039 (-5.291)***
<b>COUNT</b>	0.717 (146.422)***	0.744 (150.589)***	0.746 (151.926)***	0.748 (149.410)***
<b>VARCAP</b>	-0.103 (-44.718)***	-0.057 (-34.856)***	-0.047 (-37.580)***	-0.040 (-34.388)***
<b>Adjusted R<sup>2</sup></b>	0.747	0.722	0.715	0.709

Table 4. 7: Robustness tests: OLS regression analysis for Scenario 1(Heterogeneous banks with homogeneous exposures).

The table presents the regression results for Scenario1 applied on a second run of Monte Carlo simulations based on random sampling as robustness test. The dependent variable is CATEND measured as the total loss of capital due to contagion as percentage of total capital in the network. Explanatory variables are CATIN1, CATIN2, LEVIN, NOUTGOING, COUNT and VARCAP. Each cell displays the OLS standardized coefficients along with the corresponding t-statistics (shown in parentheses). The sample comprises of 18,000realizations (simulated banking crises).\*\*\* denotes significance at the 1% level.

	<b>N=20</b>	<b>N=50</b>	<b>N=80</b>	<b>N=100</b>
<b>CATIN 1</b>	0.632 (190.142)***	0.578 (175.421)***	0.631 (194.298)***	0.787 (278.811)***
<b>CATIN2</b>	0.146 (21.936)***	0.040 (6.178)***	0.065 (9.922)***	0.103 (18.207)***
<b>LEVIN</b>	0.043 (6.901)***	0.033 (5.275)***	0.023 (3.447)***	0.031 (5.694)***
<b>NOUTGOING</b>	-0.134 (-26.504)***	-0.081 (-15.491)***	-0.082 (-15.340)***	-0.087 (-18.819)***
<b>COUNT</b>	0.522 (126.635)***	0.664 (174.035)***	0.630 (168.235)***	0.440 (136.971)***
<b>VARCAP</b>	-0.101 (-47.761)***	-0.073 (-55.374)***	-0.059 (-53.394)***	-0.047 (-52.748)***
<b>VARLOANS</b>	-0.091 (-15.361)***	-0.049 (-7.591)***	-0.042 (-6.088)***	-0.036 (-6.457)***
<b>Adjusted R<sup>2</sup></b>	0.781	0.796	0.804	0.853

Table 4. 8: Robustness tests: OLS regression analysis for Scenario 2 (Heterogeneous banks with heterogeneous exposures).

The table presents the regression results for Scenario2 applied on a second run of Monte Carlo simulations based on random sampling as robustness test. The dependent variable is CATEND measured as the total loss of capital due to contagion as percentage of total capital in the network. Explanatory variables are CATIN1, CATIN2, LEVIN, NOUTGOING, COUNT, VARCAP and VARLOANS. Each cell displays the OLS standardized coefficients along with the corresponding t-statistics (shown in parentheses). The sample comprises of 18,000 realizations (simulated banking crises). \*\*\* denotes significance at the 1% level.

	<b>N=20</b>	<b>N=50</b>	<b>N=80</b>	<b>N=100</b>
<b>CATIN2</b>	0.239 (51.293)***	0.128 (26.334)***	0.108 (19.087)***	0.104 (17.898)***
<b>LEVIN</b>	0.061 (13.241)***	0.040 (8.251)***	0.073 (13.123)***	0.085 (14.721)***
<b>NOUTGOING</b>	-0.183 (-60.118)***	-0.138 (-41.188)***	-0.137 (-34.767)***	-0.138 (-33.711)***
<b>COUNT</b>	0.865 (251.275)***	0.936 (271.463)***	0.921 (240.695)***	0.914 (230.058)***
<b>VARLOANS</b>	-0.199 (-41.179)***	-0.198 (-38.059)***	-0.227 (-37.398)***	-0.235 (-37.253)***
<b>Adjusted R<sup>2</sup></b>	0.884	0.874	0.836	0.825

Table 4. 9: Robustness tests: OLS regression analysis for Scenario 3 (Homogeneous banks with heterogeneous exposures).

The table presents the regression results for Scenario3 applied on a second run of Monte Carlo simulations based on random sampling as robustness test. The dependent variable is CATEND measured as the total loss of capital due to contagion as percentage of total capital in the network. Explanatory variables are CATIN2, LEVIN, NOUTGOING, COUNT and VARLOANS. Each cell displays the OLS standardized coefficients along with the corresponding t-statistics (shown in parentheses). The sample comprises of 18,000 realizations (simulated banking crises).\*\*\*denotes significance at the 1%level.

	<b>N=20</b>	<b>N=50</b>	<b>N=80</b>	<b>N=100</b>
<b>CATIN2</b>	0.510 (135.692)***	0.489 (123.998)***	0.466 (123.931)***	0.463 (121.890)***
<b>LEVIN</b>	0.007 (2.899)***	0.018 (7.392)***	0.021 (9.066)***	0.038 (15.968)***
<b>NOUTGOING</b>	-0.340 (-143.814)***	-0.337 (-129.913)***	-0.327 (-130.803)***	-0.327 (-130.132)***
<b>COUNT</b>	0.652 (254.600)***	0.661 (248.526)***	0.678 (266.148)***	0.668 (262.001)***
<b>Adjusted R<sup>2</sup></b>	0.933	0.929	0.936	0.934

Table 4. 10: Robustness tests: OLS regression analysis for Scenario4 (Homogeneous banks with homogeneous exposures).

The table presents the regression results for Scenario4 applied on a second run of Monte Carlo simulations based on random sampling as robustness test. The dependent variable is CATEND measured as the total loss of capital due to contagion as percentage of total capital in the network. Explanatory variables are CATIN2, LEVIN, NOUTGOING and COUNT. Each cell displays the OLS standardized coefficients along with the corresponding t-statistics (shown in parentheses).The sample comprises of 18,000 realizations (simulated banking crises).\*\*\* denotes significance at the 1% level.

#### 4.4 Conclusions

This chapter investigates how complexity of an interbank network structure affects interbank contagion. In particular, we explore the interplay between heterogeneity, network structure and balance sheet composition in the spreading of contagion using four basic scenarios. Our findings clearly indicate that heterogeneity plays a significant role in the stability of the financial system. In our numerical simulations, we observe that when heterogeneity is introduced with respect to the size of each bank, the system's shock absorption capacity is enhanced. An interbank network consisting of banks of different sizes can more easily withstand a random shock,

making contagion a less likely phenomenon. Furthermore, when we allow for the presence of heterogeneous interbank exposures in our model, we observe additional resilience to the interbank network as an initial shock dissipates more easily than in the case of homogeneous interbank claims. We also find that the likelihood of contagion declines as we move from smaller to larger network settings. As far as connectivity is concerned, our analysis reveals that interconnectedness has a large impact on the resilience of the interbank network. Financial shocks will be absorbed more efficiently in relatively small and highly interconnected interbank networks, where as in larger systems increased connectivity will spread the shock into a large part of the system causing a cascade of defaults. Highly leveraged banks are more exposed to default risk and thus contribute more to systemic risk, especially to that of large interbank networks.

Avenues for future research can include the study of non-performing loans (NPLs) in relation to contagion risk in a unified framework. A second objective within this setting would be to test how asset devaluations and haircuts depicted on bank balance sheets can affect interbank contagion. Under such setting various weaknesses of network systems can be identified and additionally, the role systemic banks play in causing market-wide effects can be further explored. This becomes extremely relevant to the case of the European sovereign debt crisis whose aftermath is still fresh in the financial system.

## Appendix

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### Simulation algorithm:-Set up of the interbank network

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- *Define the number of banks ( $n$ ) in the interbank network system*
  - *Define the complexity (number of outgoing links of each bank) of the network system*
  - *Assign directed edges to represent lending-borrowing interbank relationships (link formation follows a uniform distribution)*
  - *Allocate balance sheet components among banks (equity and interbank loans)*
  - *Generate the interbank matrix of bilateral exposures (consistent with each scenario tested)*
  - *Generate the banks' equity vector (consistent with each scenario tested)*
- 

Figure 4.A. 1: Simulation algorithm: Set up of the interbank network

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**Simulation algorithm:-Contagion procedure**

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**for** each of the T realizations

- *Set up the interbank network;*
- *Estimate the leverage of the interbank network; (**levin**)*
- *Estimate the variance of capital of the interbank network (used in those scenarios tested where only heterogeneous bank sizes are considered); (**varcap**)*
- *Estimate the variance of interbank loans (used in those scenarios tested where only heterogeneous interbank loan exposures are considered); (**varloans**)*
- *Shock the system with the exogenous default of bank i;*
- *Estimate the initial loss of capital by defaulting bank i as percentage of total capital of the system; (**catin1**)*
- *Estimate the loss of capital at the first stage (interbank loans that cannot be paid back) by defaulting bank i as percentage of total capital of the system; (**catin2**)*
- *Estimate the number of outgoing links(outdegree) of bank i; (**noutgoing**)*

**While** at least one bank defaulted do

**for** every bank i do

**if** counterparty losses occurred **then**

*update equity of defaulting bank's creditors (subtract losses from creditors' equity);*

**end**

**if** equity  $\leq 0$  **then**

*default bank i;*

**end**

**end**

**end**

*-Estimate the total loss of capital due to contagion as percentage of total capital of the system; (**catend**)*

*- Estimate the shock propagation variable which measures the number of rounds needed until no further bank defaults; (**count**)*

*-Record **levin**, **varcap**, **varloans**, **catin1**, **catin2**, **noutgoing**, **count**, **catend**;*

**end**

\*After performing a satisfactory number of realizations for each scenario tested, regression analysis is employed in order to test the effect of the aforementioned variables on contagion risk.

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Figure 4.A. 2: Simulation algorithm: Contagion Procedure



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## 5. The case of Erdős-Rényi network model

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In this chapter, we extend the model developed in the previous chapter to include a wide variety of network topologies and provide a better understanding of the relation between network structure, banks' characteristics and interbank contagion. While the focus of the previous chapter is on the various factors that affect interbank contagion such as bank capital ratios, leverage, interconnectedness and homogeneity across banks' sizes, the model lacks flexibility as far as the variability of the networks links is concerned. In order to circumvent this problem, we introduce the Erdős-Rényi probabilistic network model in our study to provide a wider vicinity of scenarios concerning the network structure of the interbank system and study how homogeneity within the interbank network affects the propagation of financial distress from one institution to the other parts of the system through bilateral exposures.

### 5.1 Introduction

The introduction of the Erdős-Rényi probabilistic network model provides us with a wider vicinity of scenarios concerning the network structure of the interbank system. Under this framework, we build up multiple scenarios of various network structures that include a satisfactory number of cases via Monte Carlo simulations. In every single network that we construct, we investigate the dynamics of cascading defaults from an initial random shock that hits the system. Erdős-Rényi random graph model which is one of the earliest theoretical network models was introduced by Erdős and Rényi (1960). In this random graph, each possible link between any two nodes can occur with a certain independent and identical probability-the Erdős and Rényi probability. The Erdős and Rényi (1960) random graph model is a model in which has been extensively applied for the study of contagion in financial networks, e.g. Iori et al. (2006), Nier et al. (2007), Gai and Kapadia (2010), May and Arinaminpathy (2010) and Amini et al. (2016). However, a number of alternatives have been recently developed that differ in the probability law governing the distribution of links between nodes. Using the Erdős-Rényi network structure, the degree distribution or the connectivity among banks can vary with respect to the chosen probability  $p$ . Thus, each random network generated with the same parameters  $N$ ,  $p$  looks slightly different. Not only the detailed wiring network graph changes between realizations, but so does the number of links. Random graphs or Erdős-Rényi graphs are useful for modeling, analysis, and solving of structural and algorithmic problems arising in mathematics, theoretical computer science, statistical mechanics, natural sciences, and even in social sciences. However, the utility of an Erdős-Rényi model lies mainly in its mathematical simplicity, not in its realism. Virtually, the comparison with real-world networks indicates that the random network model does not capture the degree distribution of real networks but it provides a useful baseline for more complicated network models. One significant property of the real networks that is not captured by the Erdős-Rényi random model is the existence of hubs. In real networks, there is

often observed a significant number of highly connected nodes and large differences in node degrees.

### **5.1.1 Related Literature**

The most common network structures that are either found in real-world data or used in some theoretical studies of interbank contagion are the Erdős-Rényi random network structure, introduced in Erdős-Rényi (1960), the small-world structure, introduced in Watts and Strogatz (1998) and the scale-free structure, introduced in Barabasi and Albert (1999).

The Erdős-Rényi network structure, which is applied in our study, can be obtained by connecting any two nodes with a fixed and independent probability  $p$ . Thus, in an Erdős-Rényi network structure the degree or the number of links of a node is  $p(n-1)$ . The expected degree distribution for such networks is Binomial, converging to Poisson for large  $n$ . The Erdős-Rényi(1960) random graph model is a model in which has been extensively applied for the study of contagion in financial networks, e.g. in the contributions from Iori et al. (2006), Nier et al. (2007), Gai and Kapadia (2010), May and Arinaminpathy(2010) and Amini et al. (2016). A number of alternatives models have been recently developed that differ in the probability law governing the distribution of links between nodes.

Nier et al.(2007) study the extent to which the resilience of an interbank network depends on a combination of variables characterizing the network topology, banks' characteristics in terms of net worth and interbank exposures, and market concentration. Using Monte Carlo simulation experiments in Erdős-Rényi random graphs, they find that the effect of the degree of connectivity is non-monotonic. Specifically, a small initial increase in connectivity increases the chance of contagion defaults. However, after a certain threshold value, connectivity improves the capacity of a banking system to withstand shocks. In addition, the authors find that the banking system is more resilient to contagious defaults if its banks are better capitalized and this effect is non-linear. Finally, the size of interbank liabilities tends to increase the risk of default cascades, even if banks hold capital against such exposures and more concentrated banking systems are shown to be prone to larger systemic risk.

Gai and Kapadia (2010) using a network model of a banking system study how the probability and potential impact of contagion is influenced by aggregate and idiosyncratic shocks, network structure and liquidity. The authors agree with Haldane (2009) concerning the “robust-yet-fragile” property that the financial system exhibit. Even when the probability of contagion is very low, its effects can have tremendous consequences to the financial system. Higher connectivity may reduce the probability of default when contagion has not started yet but it may also increase the probability of having large default cascades when contagion begins.

May and Arinaminpathy (2010) apply an Erdos-Renyi network structure of which they build on the models of Nier et al. (2007) and Gai and Kapadia (2010) and study

the interplay between the characteristics of individual banks and the overall behavior of the network. The authors consider that banks interact through different asset classes and study contagion between those asset classes. May and Arinaminpathy (2010) find that increasing the level of connectivity is beneficial only when the initial shock has been caused by a default on interbank loans. However, by contrast, the opposite holds in case of liquidity shocks since they do not experience attenuation and for a given asset class, they tend to grow as more and more banks hold the failing asset. Finally, the authors emphasize the importance of having large capital buffers that will make for greater robustness both of individual banks and of the system as a whole.

Amini et al. (2016) test the impact of heterogeneity in an interbank network structure and the relation between resilience and connectivity using three different network models; a scale-free network with equal and heterogeneous weights and an Erdős-Rényi network with equal weights. The main result of this study is that the most heterogeneity is introduced, the least the resilience of the network.

The small world model which was introduced by Watts and Strogatz (1998) is a graph network model that has two main features: small average shortest path length and a clustering coefficient significantly higher than expected by random chance. More specifically, this model has the so called "small world property" which refers to networks where, although the network size is large and each node has a small number of direct neighbors, the distance between any two nodes is very small compared to the network size. The small world model is a model in which has been applied for the study of contagion in financial networks, e.g. in the contributions from Boss et al. (2004), Gai and Kapadia (2011) and Pegoraro (2012).

However, the property of a fat tail in the degree distribution has been observed in many types of real networks and has led to the development of scale-free models by Barabási and Albert (1999). Scale free networks exhibit a degree-distribution that follows a power law and are often characterized by growth and preferential attachment. It has been observed that the number of nodes in these models increases over time and each of them enter the network adding new edges ("growth") which are then linked to the existing nodes according to a particular pattern –usually referred as preferential attachment. Preferential attachment by banks could result from the wish to interact with the most reliable counterparties. For example, banks who initially have the largest number of interactions will attract more linkages over time. The distinctive feature of a scale free network is the existence of nodes with very different degree, and in particular the existence of hubs with a large number of connections. This property can have a large impact on the resilience of the system in the case of the failure of a hub. However, scale free networks are generally more resilient than other network models, but are extremely fragile if the most connected institution is in distress. In the literature, it is often argued that a more adequate model of a financial system is a scale-free network (see, for example, Boss et al. (2004) and Soramäki et al. (2007)).

### 5.1.2 Erdős–Rényi random graph Model

The random graph model which is one of the earliest theoretical network models was introduced by Erdős and Rényi (1960). In this random graph, each possible link between any two nodes can occur with a certain independent and identical probability,  $p$ . This model is typically denoted  $G(n, p)$  and has two parameters:  $n$  the number of vertices and  $p$ , the probability that each simple edge  $(i, j)$  exists, which is constant for each pair nodes.

The adjacency matrix of a random graph is given by

$$\forall i > j, A_{ij} = A_{ji} = \begin{cases} 1, & \text{edge } (i, j) \text{ exists; prob } (p) \\ 0, & \text{edge } (i, j) \text{ does not exist; prob } (1 - p) \end{cases}$$

In other words, each edge is included in the graph with probability  $p$ , independent from every other edge. The probability to create randomly a graph with  $n$  nodes and  $m$  edges is given by  $p^m(1 - p)^{\binom{n}{2} - m}$ . Furthermore, the probability  $p$  serves as the parameter of our model and as  $p$  increases, the graph is more likely to have more edges.

The restriction of  $i > j$  appears because edges are undirected or to put it differently, the adjacency matrix is symmetric across the diagonal, and there are no self loops. In the network there are  $n(n - 1)$  possible links to be created, resulting in an expected number of edges in the network equal to  $pn(n - 1)$ , so that the (expected) average degree is  $p(n - 1)$ . Thus, the degree distribution of such a graph is given by

$$p(k) = \binom{n - 1}{k} p^k (1 - p)^{n - 1 - k} \quad (5.1)$$

The mean degree,  $c$ , in the  $G(n, p)$  graph model is given by

$$c = (n - 1)p \quad (5.2)$$

In other words, each vertex has  $(n-1)$  possible partners and each of these exist with the same independent probability  $p$ . Asymptotically, as  $n \rightarrow \infty$ , the degree distribution of a random graph converges to a Poisson ( $c$ ) distribution

$$p(k) = \frac{e^{-c} c^k}{k!} \quad (5.3)$$

Due to the above property, the Erdős–Rényi random graph model is sometimes referred as Poisson random graph or random graph. The Erdős–Rényi (1960) graph model results in networks with small diameters and short average path lengths, capturing very well the "small-world" property, observed in many real networks. The clustering coefficient of an Erdős–Rényi graph model is equal to the probability of an edge's existence between two nodes,  $p$ . The Erdős–Rényi (1960) random graph model is a model in which has been extensively applied for the study of contagion in

financial networks, e.g. in the contributions from Iori et al. (2006), Nier et al. (2007), Gai and Kapadia (2010) and Montagna and Kok (2013).

In an Erdős –Rényi model we begin with  $n$  isolated nodes as presented in the first snapshot in Figure 1. Then, with probability  $p > 0$  each pair of nodes is connected by a link. Therefore, in this model the network is determined only by the number of nodes  $n$ , and edges,  $m$ , and usually an Erdős –Rényi random graph is written as  $G(n, m)$  or  $G(n, p)$ . In Figure 1 we present some examples of Erdős –Rényi random graphs with the same number of nodes and different linking probabilities. It is easy to understand that if we repeat the process for the same number of nodes and the same probability, we will not necessarily get the same network.

However, a number of alternatives models have been recently developed that differ in the probability law governing the distribution of links between nodes. Since, the Erdős-Rényi probability,  $p$ , is assumed to be equal and constant across all pairs of nodes, the resulting network structure does not present marked heterogeneity. Thus, modeling interbank networks using the Erdős-Rényi structure fails to mimic the heterogeneity observed in real interbank network systems.

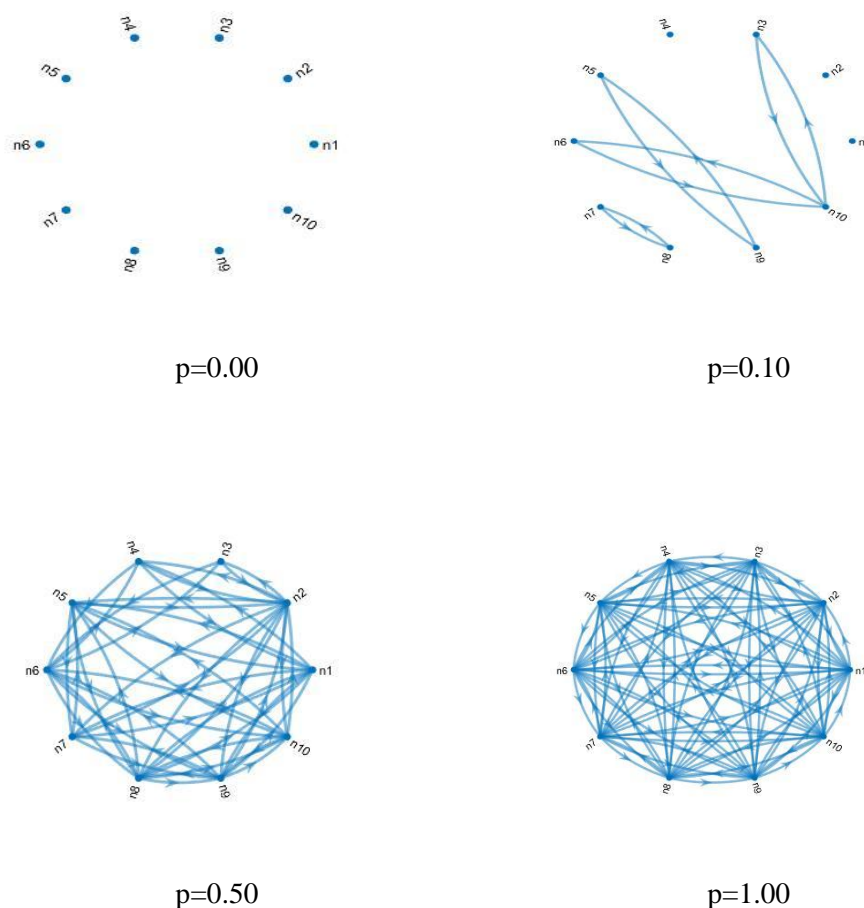


Figure 5. 1. Erdős –Rényi random networks: Erdős –Rényi random networks with ten nodes and different probabilities of connecting a pair of nodes.

In order to fully understand the heterogeneity of an Erdős-Rényi random network, we now consider one particular random realization of an Erdős-Rényi random network with 1,000 nodes and  $p=0.04$ , that is  $G(n=1000, p=0.04)$  and plot the probability  $p(k)$  of finding a node of degree  $k$ , versus the degree, we obtain Figure 2, where it can be seen that the maximum of the distribution is about the value  $k=(n-1)p=39$ . Obviously, the probability  $p(k)$  follows a binomial distribution of the form represented in equation (5.1). As we explained above, for large values of  $n$ , the degree distribution of a random graph converges to a Poisson ( $\lambda$ ) distribution. Figure 2 displays the heterogeneity plot for  $G(1000, 0.04)$ , where two characteristic features of the Erdős-Rényi networks are observed. The first is a typical dispersion of the points around the value  $x=0$ , and the second is the very small value of  $\rho(G)$ , which in this case is 0.0066.

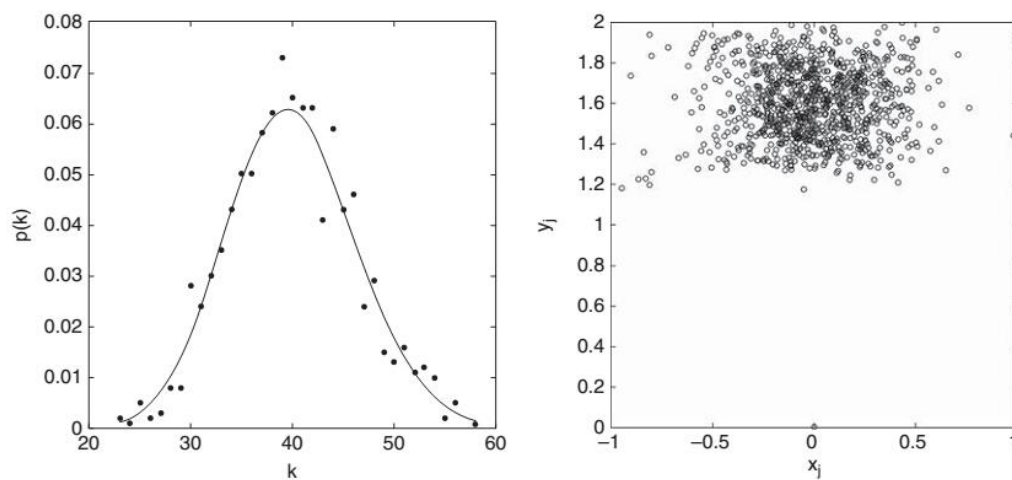


Figure 5. 2. Heterogeneity of Erdős-Rényi random networks. A typical Poisson degree distribution of an Erdős-Rényi random network with 1,000 nodes and  $p=0.04$  (left), and the characteristic heterogeneity plot for the same network. (Source: Estrada (2011) )

## 5.2 The mathematical description of the contagion model

In this section we study the case of an Erdős-Rényi network model in which, as we stated earlier, all nodes have the same probability of being connected to another node in the network. Our model is tailored to simulate default cascades triggered by an exogenous shock in an interbank network as in Leventides et al. (2019). We first introduce the interbank network model, describe the default cascades initiated by a random negative shock on this network and analyze the parameters that affect interbank contagion.

### 5.2.1 The interbank network

As in Leventides et al. (2019), we assume that the banking system contains  $i=1, \dots, N$  banks. Every bank has its own balance sheet and the accounting equation holds at all times. Total assets are divided in three categories: interbank assets  $A_i^{IB}$ , other assets  $A_i^{OT}$  and cash reserves  $C_i$ . On the liabilities side of the balance sheet we

have included: interbank liabilities  $L_i^{IB}$ , other liabilities  $L_i^{OT}$  and equity capital  $E_i$ . A schematic overview of the balance sheet is given in Table 1. Although the proposed balance sheet structure does not capture all elements of a bank balance sheet, it includes all those positions that are relevant to our study.

Assets $A_i$	Liabilities $L_i$
Interbank Assets ( $A_i^{IB}$ )	Interbank Liabilities ( $L_i^{IB}$ )
Other Assets ( $A_i^{OT}$ )	Other Liabilities ( $L_i^{OT}$ )
Cash ( $C_i$ )	Equity Capital ( $E_i$ )

Table 5. 1: Stylized Balance sheet structure.

The table presents a stylized balance sheet structure in the interbank network. Total assets are divided in three categories: Interbank assets ( $A_i^{IB}$ ), other assets ( $A_i^{OT}$ ), and cash reserves ( $C_i$ ). Total liabilities include: Interbank liabilities ( $L_i^{IB}$ ), other liabilities ( $L_i^{OT}$ ), and equity capital ( $E_i$ ). It is assumed that the accounting equation holds at all times.

We introduce a standard notation for our model and we define a simple interbank network as  $G = (V, E)$ , where  $V$  represents the nodes of the graph while  $E$  represents the edges. We further consider  $A$ , the adjacency matrix of the graph, defined as

$$\forall i > j, A_{ij} = A_{ji} = \begin{cases} 1, & \text{edge } (i, j) \text{ exists} \\ 0, & \text{edge } (i, j) \text{ does not exist} \end{cases}$$

The  $u$ th row or column of  $A$  has  $k_u$  entries, where  $k_u$  is the *degree* of the node  $u$ , which is simply the number of nearest neighbours that  $u$  has. Denoting by  $\mathbf{1}$  a  $|V| \times 1$  vector, the column vector of node degrees  $\kappa$  is given by

$$\kappa = (\mathbf{1}^T A)^T = A^T \mathbf{1} \quad (5.4)$$

We define the *indegree* as the number of links pointing toward a given node, and the *outdegree* as the number of links departing from the corresponding node. Specifically:

$$\kappa^{in} = (\mathbf{1}^T A)^T = A^T \mathbf{1} \quad (5.5)$$

$$\kappa^{out} = A \mathbf{1} \quad (5.6)$$

Thus, our interbank network of credit exposures between  $n$  banks can be visualized by a graph  $G = (V, E)$  where  $V$  represents the set of financial institutions – nodes, and  $E$  is the set of the edges linking the banks, that is, the set of ordered couples  $(i, j) \in V \times V$  indicating the presence of a loan made by bank  $i$  to bank  $j$ . The number

of nodes defines the size of the interbank network. Every edge  $(i, j)$  is weighted by the face value of the interbank claim and the representation of interbank claims is made by a single weighted  $N \times N$  matrix  $X$ :

$$X = \begin{bmatrix} 0 & \cdots & x_{1j} & \cdots & x_{1N} \\ \vdots & \ddots & \vdots & \ddots & \vdots \\ x_{i1} & \cdots & 0 & \cdots & x_{iN} \\ \vdots & \ddots & \vdots & \ddots & \vdots \\ x_{N1} & \cdots & x_{Nj} & \cdots & 0 \end{bmatrix}$$

where  $x_{ij}$  is the credit exposure of bank  $i$  vis-à-vis bank  $j$  and  $N$  is the number of banks in the network. Interbank assets are represented along the rows while columns represent interbank liabilities. Once  $X$  is in place, the interbank entries of each bank are given according to the following rules:

- (iii)  $A_i = \sum_{j=1}^N x_{ij}$  (horizontal summation), where  $A_i$  is the total interbank assets of bank  $i$ .
- (iv)  $L_j = \sum_{i=1}^N x_{ij}$  (vertical summation), where  $L_j$  is the summation of the total interbank liabilities of bank  $j$ .

One can observe that the diagonal line contains zeros due to the fact that banks do not lend to themselves. In this framework, a random network is generated based on two parameters, the size of the network (number of nodes/banks) and the probability  $p_{ij}$  that there is a lending/borrowing link between two nodes/banks. Thus, each possible link between two nodes exists with an independent and identical probability, which is often called the Erdős –Rényi probability.

Although, we have undirected edges in this framework, we cannot really speak of undirected links, since the two directions of the same link are given different weights.

### 5.2.2 Shock propagation & contagion dynamics

The failure of a bank can affect other banks through their interbank connections. Below, we describe the mechanism through which an initial shock affecting a bank propagates onto its counterparties along the network. Contrary to the recent literature, the term contagion here translates into total capital losses due to multiple default cascades. The cascade dynamics we use in this study are straightforward to implement and enable us to run a great number of simulations on a variety of different scenarios.

The default procedure starts with an exogenous shock being simulated, typically by setting to zero the equity of one randomly chosen bank  $i$  and the cascade of defaults proceeds on a timestep-by-timestep basis, assuming zero recovery for shock



transmissions. The zero recovery assumption, which is a realistic one in the short run, is often used in the literature to analyze worst case scenarios and refers to a situation where creditor banks lose all of their interbank assets held against a defaulting bank (Gai and Kapadia, 2010; Chinazzi et al., 2015). A bank's default implies that it is no longer able to meet its interbank liabilities to its counterparties. Since these liabilities constitute other banks' assets, the banks that get into trouble affect simultaneously their counterparties, leading to write-downs in their balance sheets. The interbank asset loss due to failure of bank  $i$  is subtracted from the bank's  $j$  capital. Bank  $j$  will fail if its exposure against bank  $i$  exceeds its equity. A second round of bank failure occurs if banks' creditors cannot withstand the losses realized due to its default and eventually, contagion stops if no additional bank goes bankrupt, otherwise a third round of contagion takes place. An initial shock can be amplified through banks' interconnections and further transmitted to other institutions, such that the overall effect on the system goes largely beyond the original shock. As Upper and Worms (2004) demonstrate, in response to a liquidity shock banks prefer to withdraw their deposits at other banks instead of liquidating their long-term assets, creating further instability and liquidity dry-ups in the financial system.

A general mathematical description of the dynamical system expressing the shock propagation mechanism is presented hereafter. We consider a network consisting of  $N$  banks numbered from 1 to  $N$ . We define  $b_i$  as the capital possessed by bank  $i$  in the network and

$$b_0 = (b_1, b_2, \dots, b_N) \quad (5.7)$$

stands for the initial vector of bank capital.  $X$  is defined as a  $N \times N$  matrix with entries:

$x_{ij}$  = the credit exposure of bank  $i$  vis-à-vis bank  $j$  in the network

$$x_{ii} = b_i \quad (5.8)$$

$$x_{ii} = b_i \quad (5.9)$$

We consider the case where some of the banks (one or more) collapse. We wish to study how the crisis travels through the bank network and when exactly it comes to a fixed point. The collapse of banks  $i_1, i_2, \dots, i_k$  (where  $k \leq N$ ), can be described in the following way. Consider the element  $x_0 \in Z_N^2 = \{0, 1\}^N$  which has zero entries everywhere except the positions  $i_1, i_2, \dots, i_k$  where  $x_0$  takes on the value 1. Then,

$$b_1 = b_0 - X \cdot x_0 \quad (5.10)$$

is the new vector of capital of the  $N$  banks. We now take

$$x_1(i) = \begin{cases} 1, & b_1(i) \leq 0; \\ 0, & b_1(i) > 0. \end{cases} \quad (5.11)$$

Then  $x_1 \in Z_2^N$  and  $x_1$  indicates the banks that have collapsed after the bankruptcy of the first  $k$  banks. The vector  $x_1$  takes on the value 1 in the positions  $i_1, i_2, \dots, i_k$ . If  $x_1 \neq x_0$ , this indicates that the collapse of the first  $k$  banks has adversely affected other banks leading them to bankruptcy. Similarly, from  $x_1$  we take:

$$b_1 = b_0 - X \cdot x_1 \quad (5.12)$$

and then

$$x_2(i) = \begin{cases} 1, & b_2(i) \leq 0; \\ 0, & b_2(i) > 0. \end{cases} \quad (5.13)$$

The vector  $x_2$  indicates the banks that collapse after the bankruptcy of the banks of  $x_1$ . Therefore, we have a map:

$$F: Z_2^N \rightarrow Z_2^N \quad (5.14)$$

$$x \rightarrow F(x) = f(b_0 - X \cdot x) \quad (5.15)$$

The map  $F(x)$  defines a dynamical system  $x_{n+1} = F(x_n)$  which describes the evolution of contagion in the interbank network.

### 5.3 Monte Carlo simulations

In this section we apply Monte Carlo simulations in four different stages. As in Leventides et al.(2019), we introduce randomness in three areas: amount of capital, interbank claims and network structure. The stochasticness introduced in our model provides us with a wide vicinity of scenarios that may come across in real world. Using the Erdős–Rényi network structure, the degree distribution or the connectivity among banks can vary with respect to the chosen probability  $p$ . Thus, each random network generated with the same parameters  $N, p$  looks slightly different.

The second stage involves estimating the parameters of interest, i.e. the value of the coefficients in the regression model. In the third stage the test statistics of interest are saved, while in the fourth stage we go back to the first stage and repeat  $N$  times. The quantity  $N$  is the number of replications which should be as large as is feasible. As Monte Carlo is based on random sampling from a given distribution (with results equal to their analytical counterparts asymptotically), setting a small number of

replications will yield results that are sensitive to odd combinations of random number draws. Generally speaking, the sampling variation is measured by the standard error estimate, denoted  $S_x = \sqrt{\text{var}(x)/N}$ , where  $x$  denotes the value of the parameter of interest and  $\text{var}(x)$  is the variance of the estimates of the quantity of interest over the  $N$  replications.

Similar to Leventides et al.(2019), we consider four different scenarios, in line with Chinazzi et al. (2015), where we let vary the balance sheet composition, the size of the network and the link probability among banks which is held constant for each pair of nodes. The four scenarios tested are as follows:

- Scenario 1:*
  - **Heterogeneous banks with homogeneous exposures.** In this scenario, we construct interbank networks where banks have different equity size and their interbank claims are evenly distributed across the outgoing links.
- Scenario 2:*
  - **Heterogeneous banks with heterogeneous exposures.** In this scenario, the interbank networks allow for heterogeneous bank sizes and heterogeneous interbank claims among banks.
- Scenario 3:*
  - **Homogeneous banks with heterogeneous exposures.** In this scenario, we construct interbank networks where banks have the same equity size and unevenly distribute their exposures across creditor banks.
- Scenario 4:*
  - **Homogeneous banks with homogeneous exposures.** In this last scenario, we construct interbank networks where banks have the same equity size and interbank claims are evenly distributed across creditor banks.

In each case, we do not control the number of outgoing links as in Leventides et al.(2019) but for each network that is generated a random probability, which is constant for each pair of nodes, defines the lending/borrowing relation of each bank. The probability  $p_{ij}$  is assumed to be equal and constant across all pairs  $(i,j)$ . For simplicity, we denote the probability, termed as the Erdős-Rényi probability, by  $p$ . Since the probability of forming a link is homogeneous, the resulting network structure does not present marked heterogeneity.

We examine banking systems consisting of small banks with low, medium and large interbank exposures, as well as systems of large banks with corresponding exposure levels. We consider a basic model that uses only two components from a bank's balance sheet, that is, equity and interbank loans—in the words of May and Arinaminpathy(2010) '*a caricature for banking ecosystems*'. We generate our model in two separate steps. First, we construct a model structure of  $N$  nodes representing

the banks in our system and randomly choose the probability  $p$  of forming a link between each of the  $\binom{N}{2}$  possible links.

For all the possible couples of nodes, a link is created with probability  $p$  which represent lending/borrowing relationship, while in a second step, we assign each node to a stylized balance sheet structure. Once the banking networks are created, the default propagation dynamics are implemented to examine the effects of an idiosyncratic shock hitting one bank. The effect of a shock is simulated, typically by setting to zero the equity of the affected bank. We estimate the initial loss of capital by letting the first bank default and subsequently record the loss as percentage of the total capital in the system. Consequently, the defaulted bank will be unable to repay its creditors and the interbank loans that were granted will be written-off, as we have selected to work under a zero recovery assumption. This bad debt will be recorded and expressed as percentage of the total capital in the system. Moreover, the creditors of the defaulted bank will experience a shock on their balance sheets and the recorded losses will be subtracted from their equity.

If at any time the total losses realized by a bank exceed its net worth, the bank is deemed in default and is removed from the network. Note that timesteps are modeled as being discrete and there is the possibility that many banks default simultaneously in each timestep. These shocks propagate to their creditors and take effect in the next timestep. When no further failures are observed, the default procedure terminates and various contagion indicators<sup>7</sup> are calculated based on the contagion map as described in subsection 5.3.

## 5.4. Main findings

This section discusses the main findings of this study. Subsection 5.4.1 describes in full detail the computer experiments conducted while subsection 5.4.2 discusses the simulation results of all four scenarios considered.

### 5.4.1 Computer experiments

Having generated banking systems via an Erdős-Rényi network structure framework and balance sheet allocation, the dynamics of an initial shock affecting a bank within the interbank network can be investigated. Given the complexity of the interbank network outlined above, it is extremely difficult to derive analytical solutions. In order to obtain data to describe the variables that affect contagion, we employ several Monte Carlo simulations. In each realization, we construct an interbank network with  $N \in [20, 50, 80, 100]$  nodes under the rewiring process of the Erdős-Rényi

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<sup>7</sup>We refer the interested reader to Appendix in Leventides et al.(2019) for a formalization of the aforementioned mechanism in a pseudocode which simulates the default cascade in the interbank network.

methodology. In a second step, we test the four scenarios mentioned before by varying the equity size of banks and the interbank exposure structure across creditor banks. For each scenario tested we check a wide range of link probabilities, such that we can observe dense or sparse interbank network systems. Since the probability of forming a link is homogeneous, the resulting network structure does not present marked heterogeneity.

When homogeneity across bank sizes is considered, all banks are assumed to have the same equity size and thus, each bank is endowed with a balance sheet that consists of 100 units of equity. On the other hand, when homogeneity is present with respect to interbank exposures, interbank claims are randomly allocated within the interbank network and are categorized as follows: small loans granted (4 units), medium loans (8 units) and large loans (14 units). With respect to scenarios tested where heterogeneity of bank size is introduced, the amount of equity of each bank is drawn from a uniform distribution in the range:  $b_i \in [0,100]$ , whereas when heterogeneity is introduced with respect to interbank claims, credit is allocated in the following ranges:  $a_{ij} \in [0,4]$ ,  $a_{ij} \in [0,8]$ ,  $a_{ij} \in [0,14]$ <sup>8</sup>. Interbank exposures are set 60% lower than these in Leventides et al.(2019). This is due to the fact that we cannot control the connectivity across banks since the link probability is randomly selected. The interbank exposure decrease was set by trial and error in order not to observe enormous high leveraged systems. In addition, we control the leverage of the system by setting the rule that the maximum leverage ratio of each network system cannot exceed five. Then, balance sheets are assigned to each node, consistent with each specific scenario tested. We randomly choose a single bank in the system to default due to an exogenous shock and the default cascades proceed sequentially, assuming zero recovery. When no further failures are observed results are recorded before another realization begins. We then impose another shock on the second bank in the network and this procedure continues until all banks in the interbank network are hit by an exogenous shock.

For each scenario tested and for each network size we have three cases in which we allow the weight of outgoing links (small, medium and large interbank claims) to vary among banks. Each case gives us 6,000 realizations or, to put it differently, 6,000 banking crises. We deem that 6,000 realizations provide a satisfactory number of runs and robustness to our analysis. Thus, for each scenario tested and each network size we employ  $6,000 \times 3 = 18,000$  realizations using the following variables in each realization:

- Total loss of capital due to contagion as percentage of total capital in the system (**CATEND**)

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<sup>8</sup>Although those ranges have been selected arbitrarily, they are not sensitive to any regression model employed in the following analysis and thus, our regression results will be unaffected from a qualitative point of view if different ranges are used.

- Initial loss of capital by defaulting bank  $i$  as percentage of total capital in the system (**CATIN1**), i.e. bank's  $i$  depleted equity divided by the total equity in the network
- Loss of capital at the first stage (interbank loans that cannot be repaid) by defaulting bank  $i$  as percentage of total capital in the system (**CATIN2**), i.e. total amount of loans granted to bank  $i$  that cannot be repaid divided by the total equity in the network
- Leverage of the interbank network(**LEVIN**), i.e. total interbank exposures as measured by the sum of matrix's A elements, divided by the total capital in the network
- Number of outgoing links of bank  $i$  (**NOUTGOING**), i.e. the outdegree of a bank  $i$  which corresponds to the number of creditors in the network. It is defined as the summation of the  $i$ th column of the adjacency matrix A.
- Shock propagation variable (**COUNT**) which measures the number of rounds needed until no further bank defaults
- Variance of capital (equity) (**VARCAP**) used in those scenarios tested where only heterogeneous bank sizes are considered
- Variance of interbank loans (**VARLOANS**) used in those scenarios tested where only heterogeneous interbank loan exposures are considered
- Erdős –Rényi probability  $p_{ij}(\mathbf{p})$  that there is a lending/borrowing link between two nodes/banks.

Our selection of variables is motivated by economic intuition and by the findings of previous studies on the dynamics of systemic risks (Nier et al., 2007) and Leventides et al. (2019). In order to study the effect the aforementioned variables have on contagion risk, we estimate the following ordinary least squares (OLS) models:

$$CATEND = \beta_1 CATIN1 + \beta_2 CATIN2 + \beta_3 LEVIN + \beta_4 NOUTGOING + \beta_5 COUNT + \beta_6 VARCAP + \beta_7 p \quad (5.16)$$

$$CATEND = \beta_1 CATIN1 + \beta_2 CATIN2 + \beta_3 LEVIN + \beta_4 NOUTGOING + \beta_5 COUNT + \beta_6 VARCAP + \beta_7 VARLOANS + \beta_8 p \quad (5.17)$$

$$CATEND = \beta_1 CATIN2 + \beta_2 LEVIN + \beta_3 NOUTGOING + \beta_4 COUNT + \beta_5 VARLOANS + \beta_6 p \quad (5.18)$$

$$CATEND = \beta_1 CATIN2 + \beta_2 LEVIN + \beta_3 NOUTGOING + \beta_4 COUNT + \beta_5 p \quad (5.19)$$

The model described in equation (5.16) is applied to scenarios involving heterogeneous bank sizes with homogeneous exposures in the network structure, equation (5.17) refers to a situation where emphasis is placed on heterogeneous interbank loan exposures combined with heterogeneous bank sizes, equation (5.18) takes into account homogeneous banks with heterogeneous exposures while equation (5.19) considers only homogeneous bank sizes and interbank claims. The variable

CATIN1 has been omitted from equations (5.18)- (5.19) due to the fact that banks in the interbank system are homogeneous, i.e. we keep constant the equity of each bank and thus CATIN1 remains stable during our simulation runs. There is an explanation in the next subsection concerning the fact that in our experiments we have selected to work with standardized variables—both dependent and independent variables—and have not included the intercept term in the regression models as it will be zero. Our concern is to measure effects not in terms of the original units of the dependent variable or the independent variables, but in standard deviation units<sup>9</sup>.

#### 5.4.2 Simulation results

In this section, we discuss the regression results of all four scenarios. Since our variables are measured on different scales, we cannot directly infer which independent variable has the largest effect on the dependent variable. In order to circumvent this problem we standardize our series to have zero mean and unit variance. Table 5.2 presents the regression results of the **first scenario** using the OLS model described in equation (5.16), where heterogeneous banks distribute evenly their interbank claims across the outgoing links of a network consisting of  $N = 20, 50, 80$  and  $100$  banks. Almost all regressor coefficients are found to be statistically significant for all the sizes of the network. We discern only two cases where regressor coefficients are found to be statistically insignificant and has to do with CATIN1 variable and one case that has to do with CATIN2.  $R$ -squared coefficients take on large values ranging from 74.9 to 80 percent and highlight the ability of our selected variables to explain financial distress in interbank networks.

	N=20	N=50	N=80	N=100
<b>CATIN 1</b>	0.051 (16.198)***	-0.002 (-0.459)	-0.001 (-0.347)	-0.007 (-2.044)**
<b>CATIN2</b>	0.098 (4.195)***	0.004 (0.170)	0.179 (8.073)***	0.104 (5.059)***
<b>LEVIN</b>	0.389 (17.018)***	0.413 (19.043)***	0.260 (12.205)***	0.315 (15.935)***
<b>NOUTGOING</b>	-0.080 (-3.915)***	0.097 (2.773)***	-0.170 (-4.933)***	-0.053 (-1.534)
<b>COUNT</b>	0.602 (138.571)***	0.572 (134.093)***	0.576 (136.735)***	0.540 (124.326)***
<b>VARCAP</b>	-0.088 (-53.348)***	-0.075 (-61.005)***	-0.053 (-53.890)***	-0.054 (-57.165)***
<b>P</b>	-0.101 (-5.089)***	-0.080 (-2.338)**	0.165 (4.885)***	0.107 (3.148)***
<b>Adjusted R<sup>2</sup></b>	0.800	0.763	0.756	0.749

Table 5. 2:OLS regression analysis for Scenario 1 (Heterogeneous banks with homogeneous exposures).

The table presents the regression results for Scenario 1. The dependent variable is CATEND measured as the total loss of capital due to contagion as percentage of total capital in the network. Explanatory variables are, CATIN1, CATIN2, LEVIN, NOUTGOING, COUNT, VARCAP and P, the probability for a link to exist between two nodes. Each cell displays the OLS standardized coefficients along with the corresponding  $t$ -statistics (shown in parentheses). The sample comprises of 18,000 realizations (simulated banking crises).\*, \*\* and \*\*\* denote significance at the 10, 5 and 1 percent level, respectively.

<sup>9</sup>See Wooldridge(2003) for an interesting discussion on standardization and explanation of the absence of the standardized intercept.

Table 5. 3: Summary statistics

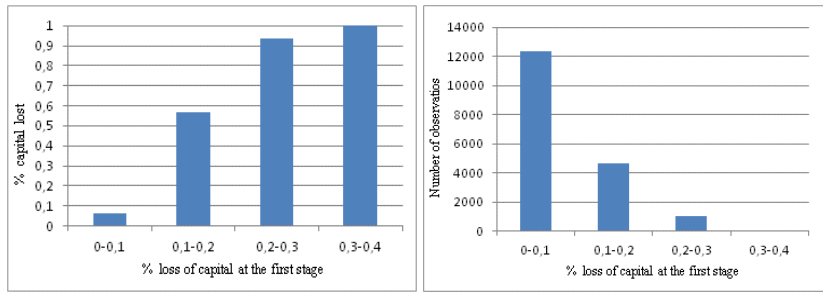
The mean, median, and standard deviation are depicted for interbank networks consisting of 20,50,80,and100 banks, respectively. Four scenarios are included: (a) Heterogeneous banks–homogeneous exposures; (b) Heterogeneous banks–heterogeneous exposures; (c) Homogeneous banks–heterogeneous exposures;(d) Homogeneous banks–homogeneous exposures. The variables are: CATEND, defined as total loss of capital due to contagion as percentage of total capital in the system;CATIN1,defined as bank’s i depleted equity divided by the total equity in the network;CATIN2,defined as the total amount of loans granted to bank i that cannot be repaid, divided by the total equity in the network; LEVIN, defined as the leverage of the interbank network; NOUTGOING, defined as the number of outgoing links of bank i, which corresponds to the number of its creditors in the network; COUNT, defined as the number of rounds needed until no further bank defaults; VARCAP, defined as the variance of bank capital; VARLOANS, defined as the variance of interbank loans and  $p$ , the Erdős–Rényi probability  $p_{ij}$  that there is a lending/borrowing link between two nodes/banks

	Variable	Heterogeneous banks– homogeneous exposures			Heterogeneous banks– heterogeneous exposures			Homogeneous banks– heterogeneous exposures			Homogeneous banks– homogeneous exposures			
		Mean	Median	Std. Dev.	Mean	Median	Std. Dev.	Mean	Median	Std. Dev.	Mean	Median	Std. Dev.	
<b>n=20</b>	CATEND	0.241	0.067	0.368	0.185	0.063	0.313	0.220	0.050	0.350	0.251	0.050	0.367	
	CATIN1	0.050	0.050	0.028	0.050	0.050	0.029	0.050	0.050	0.000	0.050	0.050	0.000	
	CATIN2	0.079	0.063	0.062	0.070	0.052	0.067	0.147	0.130	0.108	0.175	0.160	0.124	
	COUNT	2.624	1.000	2.342	2.278	1.000	1.999	1.916	1.000	1.893	2.029	1.000	1.876	
	LEVIN	1.572	1.273	1.193	1.406	1.081	1.241	2.935	2.679	1.888	3.510	3.510	2.046	
	P	0.491	0.486	0.286	0.459	0.438	0.278	0.402	0.371	0.253	0.276	0.214	0.226	
	NOUTGOING	9.303	9.000	5.729	8.701	8.000	5.595	7.601	7.000	5.139	5.228	4.000	4.603	
	VARCAP	829.371	824.415	171.611	836.673	832.512	172.930	-	-	-	-	-	-	
	VARLOANS	-	-	-	38.407	8.230	45.561	47.449	33.195	40.430	-	-	-	-
	<b>n=50</b>	CATEND	0.364	0.034	0.459	0.352	0.033	0.453	0.177	0.020	0.348	0.215	0.020	0.367
CATIN1		0.020	0.020	0.011	0.020	0.020	0.011	0.020	0.020	0.000	0.020	0.020	0.000	
CATIN2		0.047	0.044963	0.029	0.046	0.045	0.030	0.068	0.063	0.047	0.068	0.064	0.051	
COUNT		4.078	2.000	3.473	4.026	2.000	3.532	2.209	1.000	2.618	2.451	1.000	2.767	
LEVIN		2.330	2.320	1.365	2.310	2.336	1.347	3.380	3.392	1.996	3.401	3.240	2.002	
NOUTGOING		16.754	14.000	12.604	18.478	14.000	14.272	10.092	8.000	9.003	5.025	4.000	4.562	
P		0.342	0.290	0.250	0.377	0.270	0.286	0.206	0.159	0.176	0.103	0.079	0.084	
VARCAP		834.415	832.740	106.833	830.829	829.254	115.273	-	-	-	-	-	-	
VARLOANS		-	-	-	38.207	8.299	44.715	47.688	33.015	39.929	-	-	-	-
<b>n=80</b>		CATEND	0.383	0.022	0.469	0.359	0.021	0.460	0.180	0.012	0.362	0.198	0.012	0.3676
	CATIN1	0.012	0.012	0.007	0.012	0.012	0.007	0.012	0.012	0.000	0.012	0.012	0.000	
	CATIN2	0.031	0.030	0.020	0.029	0.028	0.020	0.045	0.042	0.031	0.040	0.035	0.032	
	COUNT	4.638	3.000	4.259	4.662	2.000	4.442	2.359	1.000	2.906	2.455	1.000	2.882	
	LEVIN	2.461	2.429	1.464	2.365	2.297	1.430	3.623	3.595	2.099	3.209	3.120	2.065	
	NOUTGOING	17.752	15.000	14.422	19.403	14.000	16.368	10.829	8.000	9.556	4.860	3.000	4.777	
	P	0.225	0.196	0.178	0.246	0.164	0.203	0.137	0.104	0.115	0.061	0.046	0.055	
	VARCAP	820.667	820.990	85.218	822.902	815.162	86.217	-	-	-	-	-	-	
	VARLOANS	-	-	-	38.955	8.376	46.160	47.854	33.478	39.566	-	-	-	-
	<b>n=100</b>	CATEND	0.370	0.017	0.468	0.382	0.018	0.468	0.149	0.010	0.332	0.220	0.010	0.385
CATIN1		0.010	0.010	0.006	0.010	0.010	0.006	0.010	0.010	0.000	0.010	0.010	0.000	
CATIN2		0.025	0.024	0.015	0.025	0.025	0.017	0.035	0.033	0.024	0.036	0.032	0.026	
COUNT		4.649	3.000	4.261	4.823	3.000	4.291	2.383	1.000	3.212	2.830	1.000	3.469	
LEVIN		2.474	2.458	1.423	2.555	2.629389	1.515	3.539	3.615	2.006	3.600	3.552	1.967	
NOUTGOING		18.732	14.000	15.964	21.815	15.00000	18.283	10.898	8.000	9.488	5.267	4.000	4.565	
P		0.189	0.142	0.157	0.220	0.141615	0.181	0.110	0.079	0.092	0.053	0.041	0.040	
VARCAP		822.626	821.214	80.128	827.379	826.1881	76.812	-	-	-	-	-	-	
VARLOANS		-	-	-	37.755	8.359417	44.330	48.180	33.479	39.959	-	-	-	-

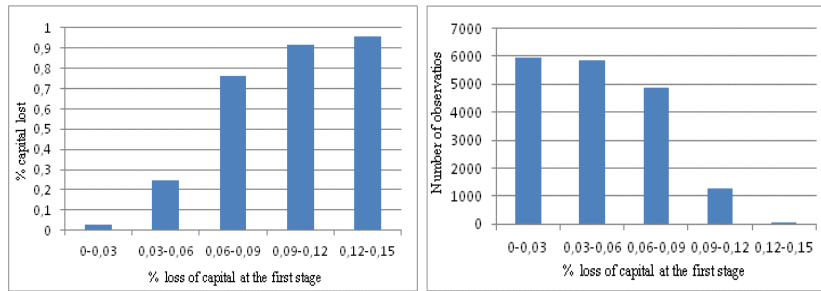


The variable CATIN1 captures the initial effect defaulting bank  $i$  exerts on the network, whereas the magnitude of interconnectedness across the banks that comprise the interbank network is measured through parameter CATIN2. As we observe from Table 5.2, variable CATIN1 does not seem to affect much the dependent variable, whereas two regressor coefficients are found to be insignificant. Financial shocks will propagate into the defaulting bank's counterparties along the network, erode their capital and make them more vulnerable to subsequent shocks. The magnitude of the positive relationship between CATIN2 and CATEND – the dependent variable – seems to increase as the size of the interbank network increases with the only exception being the N=50 bank network segment which follows an autonomous path (although statistically insignificant). The increasing magnitude of the above relationship seems to cease as we move from the case of n=80 banks to the case of n=100 banks. This finding implies that as we move from smaller to larger network settings, systemic risk and the likelihood of contagion increases. However, when we move from the case of n=80 banks to the case of n=100 banks the likelihood of contagion seems to decrease. Figure 5.3 visually illustrates the extent of contagion as a function of the percentage loss of capital due to bank's  $i$  default. It is shown that as the network size increases from small to medium sized networks, we observe that capital losses rise, confirming the findings from the regression model. As we can observe from Figure 5.3, as we move from the n=80 interbank network scheme to n=100 the likelihood of contagion seems to decrease since we have very few cases that cause systemic break downs and defaults.

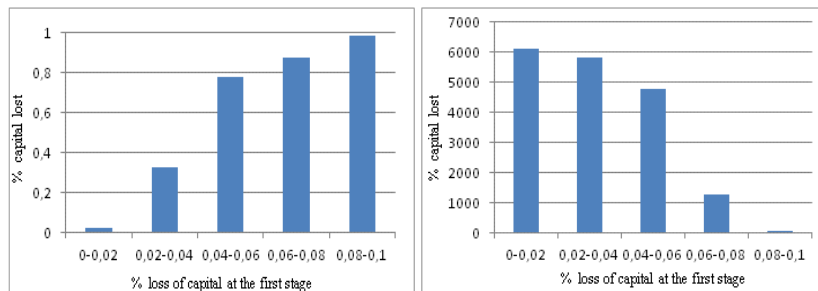
As expected, we also find that there is a positive relationship between the leverage of the network and the capital losses due to contagion. This result is in line with the findings of Nier et al. (2007) who provide evidence that systemic risk increases when system-wide leverage increases. Highly leveraged banks in the interbank network are clearly more exposed to the risk of default on interbank loans. The greater the size of default on debt is, the larger the losses are that banks transmit to their neighbors, other things being equal. Thus, highly leveraged banks contribute more to systemic risk as they become a vehicle for transmitting shocks within the network. Moreover, it is shown that the magnitude of the positive relationship between the network's leverage and contagion risk increases as we move from smaller to larger interbank networks (illustrated in Table 5.2) with the only exception being the n=80 bank



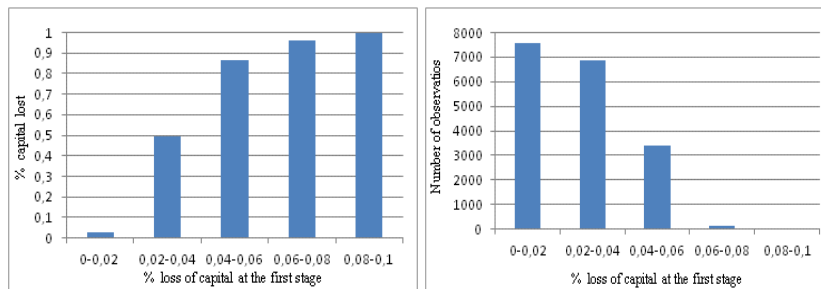
(a) N=20 banks



(b) N=50 banks



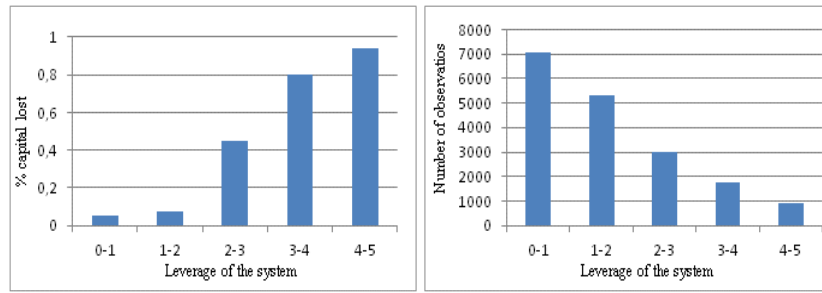
(c) N=80 banks



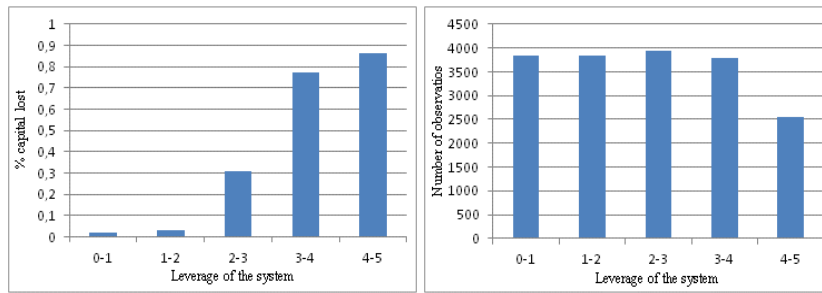
(d) N=100 banks

Figure 5. 3 : Scenario 1: Heterogeneous Banks with homogeneous exposures | Extent of contagion (expressed as the total capital lost from the banking system due to the failure of at least one bank) as a function of the % initial loss of capital due to default of the first bank.

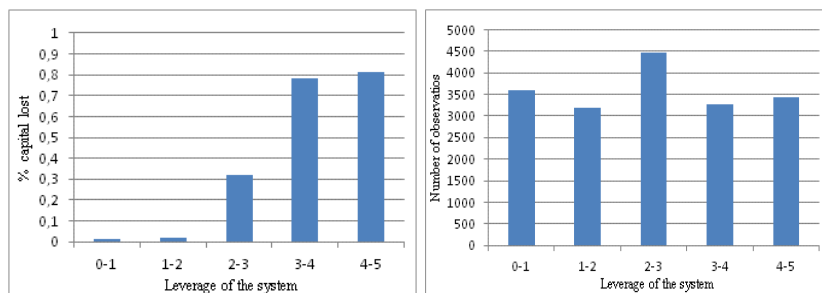
Panels (a)-(d) show the relation between the % initial loss of capital due to default of the first bank and the extent of contagion across interbank networks with different number of banks.



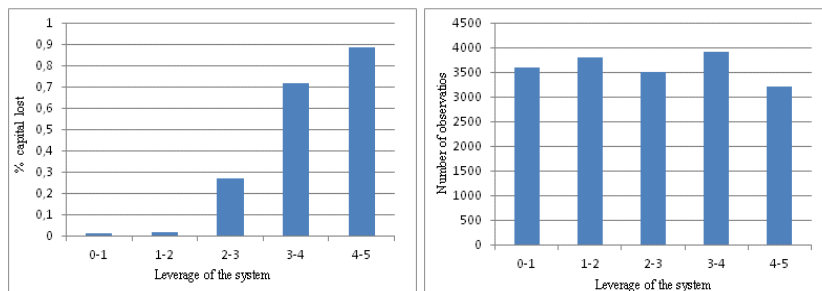
(a) N=20 banks



(b) N=50 banks



(c) N=80 banks



(d) N=100 banks

Figure 5. 4 : Scenario 1: Heterogeneous Banks with homogeneous exposures | Extent of contagion (expressed as the total capital lost from the banking system due to the failure of at least one bank) as a function of the leverage of the system.

Panels (a)-(d) show the relation between the leverage of the system and the extent of contagion across interbank networks with different number of banks.

network scheme where the magnitude of the standardized coefficients seems to decrease.

Our results also suggest that connectivity, expressed in our experiments as the outdegree<sup>10</sup> of the first bank that defaults, has a negative effect on interbank contagion with the only exception being the case of  $n=50$  banks where we can observe a positive relationship between contagious defaults and connectivity.

Interestingly, as we move from small networks consisted of twenty banks to networks consisted of fifty banks the effect of connectivity to interbank contagion turns from negative to positive and after then connectivity keeps affect negatively the systemic risk of the network. Thus, as we move from network systems consisted of fifty banks to networks consisted of 100 banks this negative relationship seems to decrease. In relatively small interbank networks, a high level of connectivity will allow an efficient absorption of shocks, whereas in medium size networks the increased connectivity will spread the shock throughout the system, potentially leading to many default cascades. The link probability, that is assumed to be equal across all pairs, seems to contribute to the resilience of the system for small and medium size networks. However, as we move from medium to large size networks this effect turns negative to the resilience of the system as it seems to contribute positively to systemic risk.

Our regression analysis also shows that the COUNT variable which measures the number of rounds until no further bank defaults, has a positive impact on interbank contagion. Heterogeneity expressed as the variance of capital exhibits a negative and statistically significant relationship with interbank contagion, showing that size heterogeneity can have positive effects on the stability of an interbank network.

However, the positive magnitude seems to decrease as we move from small to large interbank networks. An interbank network consisting of banks of various sizes can more easily withstand a negative shock, therefore no institution becomes significant for either borrowing or lending. Furthermore, in such network both smaller and larger banks can act as shock absorbers when an initial shock hits the banking system, making contagion a less likely phenomenon. This finding is in line with the results of Iori et al. (2006) concerning bank size heterogeneity.

Table 5.4 presents the regression results of the **second scenario** using the model described in equation (5.17), where banking institutions with heterogeneous bank sizes are linked to one another via heterogeneous interbank claims. The regressor coefficients are statistically significant in almost all cases and the  $R$ -squared values are quite high and lie in the vicinity of 75 to 83 percent, highlighting the good explanatory power of the model.

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<sup>10</sup>It should be highlighted that in the Erdős-Rényi network structure the outdegree equals the indegree since we have an undirected network structure. However, in our framework, the two directions of the same link are given different weights.

	<b>N=20</b>	<b>N=50</b>	<b>N=80</b>	<b>N=100</b>
<b>CATIN 1</b>	0.070 (23.660)***	0.007 (2.024)**	0.000 (0.047)	-0.001 (-0.283)
<b>CATIN2</b>	0.201 (19.541)***	0.113 (11.183)***	0.106 (9.669)***	0.071 (6.015)***
<b>LEVIN</b>	0.653 (58.484)***	0.346 (30.847)***	0.321 (26.320)***	0.399 (30.132)***
<b>NOUTGOING</b>	-0.136 (-11.540)***	-0.150 (-6.539)***	-0.052 (-2.253)**	0.038 (1.575)
<b>COUNT</b>	0.456 (111.687)***	0.630 (156.274)***	0.577 (141.939)***	0.573 (131.397)***
<b>VARCAP</b>	-0.032 (-18.897)***	-0.067 (-50.848)***	-0.053 (-52.027)***	-0.041 (-40.597)***
<b>VARLOANS</b>	-0.246 (-45.472)***	-0.091 (-14.882)***	-0.018 (-3.113)***	-0.082 (-12.307)***
<b>P</b>	-0.254 (-21.462)***	0.038 (1.620)	0.064 (2.678)***	-0.110 (-4.311)***
<b>Adjusted R<sup>2</sup></b>	0.830	0.796	0.776	0.751

Table 5. 4: OLS regression analysis for Scenario 2 (Heterogeneous banks with heterogeneous exposures).

The table presents the regression results for Scenario 2. The dependent variable is CATEND measured as the total loss of capital due to contagion as percentage of total capital in the network. Explanatory variables are the constant term CATIN1, CATIN2, LEVIN, NOUTGOING, COUNT, VARCAP, VARLOANS and P, the probability for a link to exist between two nodes. Each cell displays the OLS standardized coefficients along with the corresponding *t*-statistics (shown in parentheses). The sample comprises of 18,000 realizations (simulated banking crises). \*, \*\* and \*\*\* denote significance at the 10, 5 and 1 percent level, respectively.

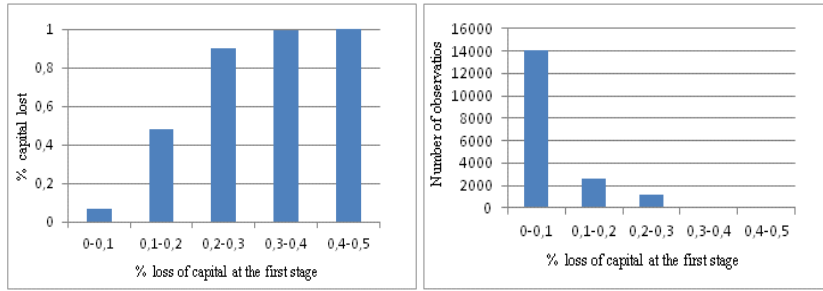
CATIN1 does not seem to impact much the dependent variable in all network segments and the regressor coefficients in the relatively large interbank networks becomes statistically insignificant. The magnitude of standardized coefficients is almost the same with the corresponding magnitude of those derived from the first scenario. In other words, an initial shock from defaulting bank *i* will spill over more easily in the network. Thus, the first bank defaulting has the dynamics to spread the initial shock and contaminate the entire interbank network. CATIN2 has a large positive impact on contagion risk, however, its magnitude fades away as we move from smaller to larger networks. It should also be highlighted that the CATIN2 coefficients are much larger than those recorded in the first scenario in all network sizes. An initial shock following the default of bank *i* seems to contribute much to a banking crisis scenario within small and medium-sized networks and the size of total capital losses is smaller than that documented in the first scenario. Figure 5.5 depicts the extent of contagion as a function of the percentage loss of capital due to default of the first bank and confirms the results recorded in Table 5.4.

The results also show that there still exists a positive relationship between leverage and contagion; however, the coefficient estimates are larger in almost all cases than those recorded in the previous scenario. Moreover, the magnitude of the leverage coefficients decreases as the number of banks in the interbank network increases, with the only exception being the 100 bank network segment where one can observe a slight increase compared to the 80 bank network segment.

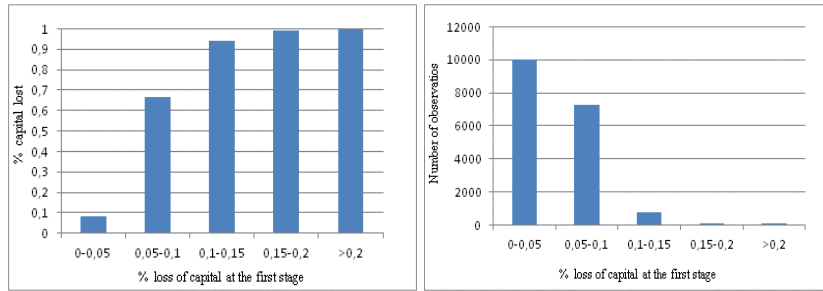
Results on connectivity are qualitatively similar to those of the first scenario, showing that connectivity negatively impacts contagion risk especially in small and medium interbank networks with the only exception being the 100 bank network segment

which follows an autonomous path and is positively related to contagion (although statistically insignificant).

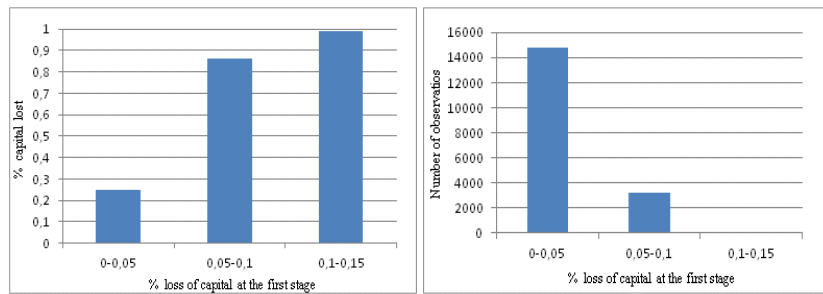
As far as the link probability is concerned, we can observe a different pattern from that of the first scenario. For small and large sized networks, link probability seems to contribute negatively to systemic risk while for medium sized networks there is a positive relationship between link probability and contagion. The number of rounds until no further bank defaults positively impacts contagion risk and contributes the most to total capital losses in the banking system when medium and large interbank networks are formed. Under this scenario, the heterogeneity allowed on both bank sizes and interbank exposures has had a great impact on the resilience of the network system. Heterogeneity impacts negatively on interbank contagion although its intensity decreases as the size of the network increases. Moreover, as we can see from the Table 5.4 heterogeneity of bank size contributes less to the resilience of the interbank network than heterogeneity of interbank exposures when it comes to small and medium sized networks.



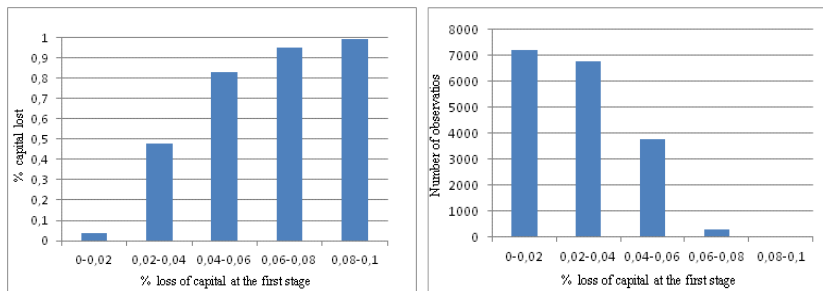
(a) N=20 banks



(b) N=50 banks



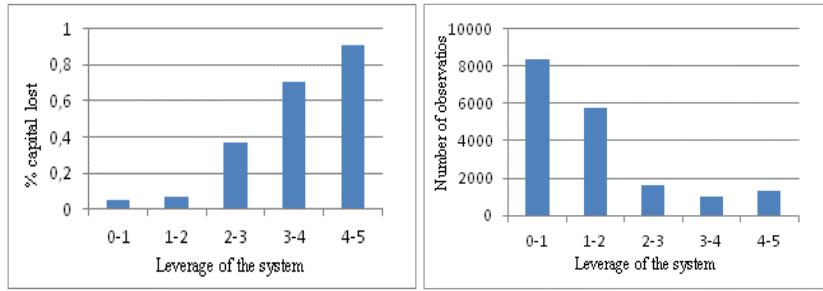
(c) N=80 banks



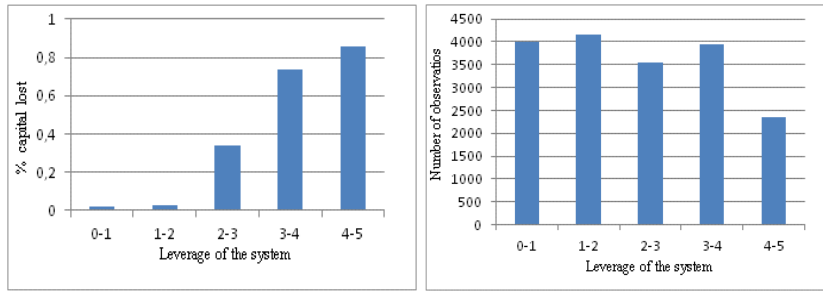
(d) N=100 banks

Figure 5. 5 : Scenario 2: Heterogeneous Banks with heterogeneous exposures | Extent of contagion (expressed as the total capital lost from the banking system due to the failure of at least one bank) as a function of the % initial loss of capital due to default of the first bank.

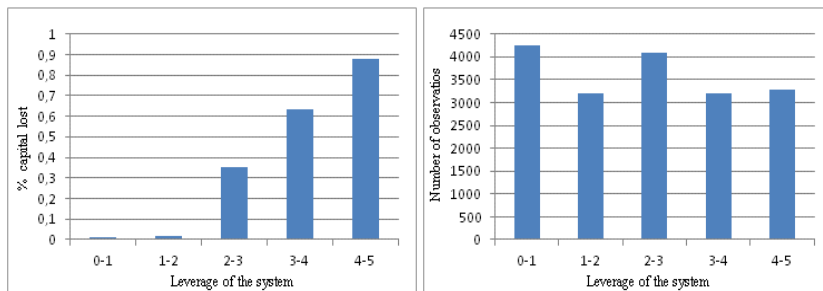
Panels (a)-(d) show the relation between the % initial loss of capital due to default of the first bank and the extent of contagion across interbank networks with different number of banks.



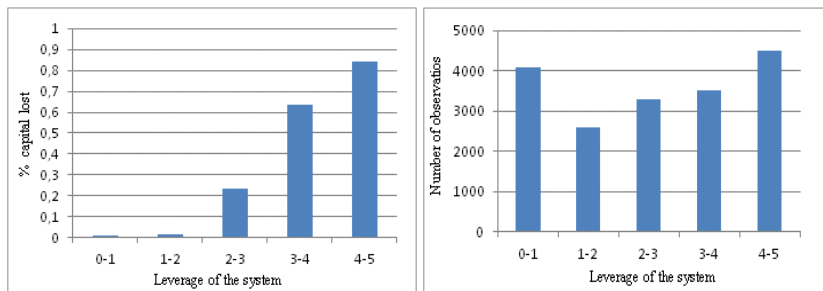
(a) N=20 banks



(b) N=50 banks



(c) N=80 banks



(d) N=100 banks

Figure 5. 6 : Scenario 2: Heterogeneous Banks with heterogeneous exposures | Extent of contagion (expressed as the total capital lost from the banking system due to the failure of at least one bank) as a function of the leverage of the system.

Panels (a)-(d) show the relation between the leverage of the system and the extent of contagion across interbank networks with different number of banks.

The heterogeneity of interbank exposures acts as a diversification tool and contributes to a smaller extent to an unfolding crisis compared to the scenario where homogeneous banks are interconnected via heterogeneous exposures (shown in Table 4).



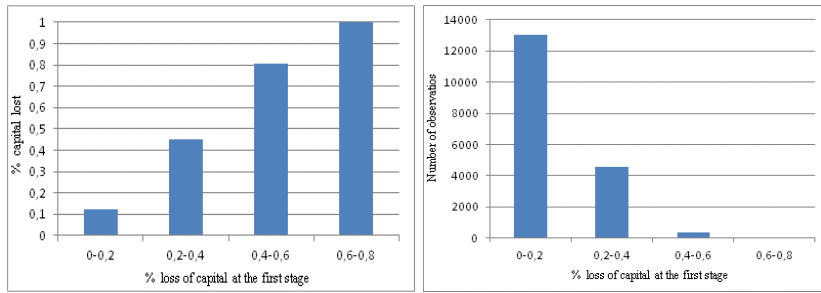
Table 5.5 depicts the results of the **third scenario** as described in equation (5.18). In this scenario, we construct network systems where banks have the same equity size and unevenly distribute their exposures across creditor banks. We note that an initial shock fades away with the failure of the first bank and does not spillover to other banks within the network. This is mainly due to our choice of parameters regarding the equity of each bank, the links among banks and the interbank claims among creditor banks. In order to observe default cascades we relax our initial assumptions and lower the equity of each bank in the network system.

	<b>N=20</b>	<b>N=50</b>	<b>N=80</b>	<b>N=100</b>
<b>CATIN2</b>	0.196 (25.178)***	0.143 (16.422)***	0.125 (15.232)***	0.088 (9.806)***
<b>LEVIN</b>	0.324 (39.268)***	0.298 (32.475)***	0.275 (31.619)***	0.279 (30.578)***
<b>NOUTGOING</b>	-0.163 (-15.308)***	-0.168 (-10.438)***	-0.126 (-8.707)***	-0.087 (-5.561)***
<b>COUNT</b>	0.736 (191.690)***	0.761 (175.841)***	0.790 (195.383)***	0.793 (186.247)***
<b>VARLOANS</b>	-0.175 (-43.977)***	-0.190 (-44.390)***	-0.180 (-46.723)***	-0.167 (-41.937)***
<b>P</b>	-0.253 (-24.270)***	-0.313 (-19.153)***	-0.322 (-21.833)***	-0.339 (-21.575)***
<b>Adjusted R<sup>2</sup></b>	0.860	0.823	0.845	0.809

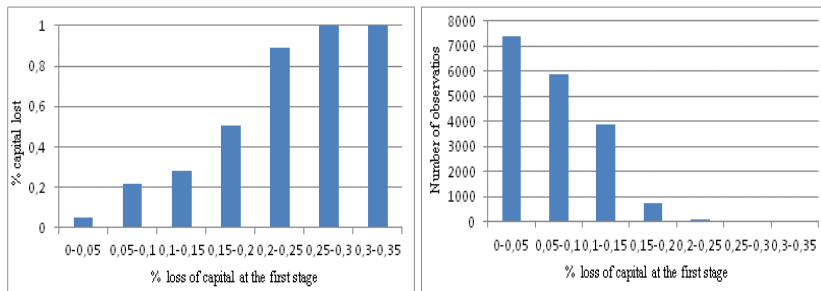
Table 5. 5: OLS regression analysis for Scenario 3 (Homogeneous banks with heterogeneous exposures).

The table presents the regression results for Scenario 3. The dependent variable is CATEND measured as the total loss of capital due to contagion as percentage of total capital in the network. Explanatory variables are the constant term CATIN2, LEVIN, NOUTGOING, COUNT, VARLOANS and P, the probability for a link to exist between two nodes. Each cell displays the OLS standardized coefficients along with the corresponding *t*-statistics (shown in parentheses). The sample comprises of 18,000 realizations (simulated banking crises). \*, \*\* and \*\*\* denote significance at the 10, 5 and 1 percent level, respectively.

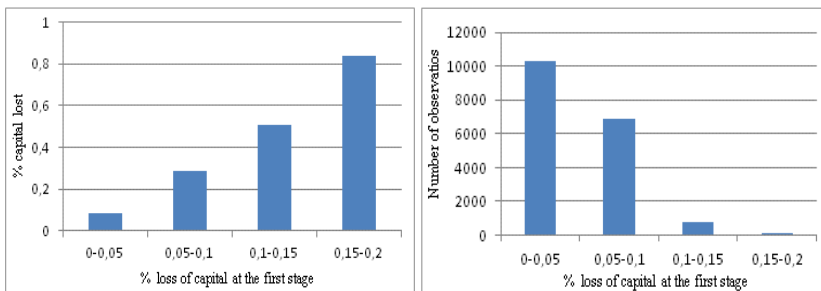
Specifically, each bank is now endowed with a balance sheet that consists of 25 units of equity and interbank claims among creditor banks are distributed in the following ranges:  $a_{ij} \in [0,10]$ ,  $a_{ij} \in [0,20]$ ,  $a_{ij} \in [0,35]$ . Interbank exposures levels were kept the same as in Leventides et al. (2019). Moreover, we control the leverage of the system by setting the rule that the maximum leverage ratio of each network system cannot exceed seven. Similar to the previous scenarios, the regressor coefficients are statistically significant in all cases and the *R*-squared values are still large, in fact the largest of all three scenarios tested. Variable CATIN2 has a highly significant positive impact on systemic risk that fades away as the network system gets larger. The same observation holds for the level of connectivity in the banking system i.e. a strong negative impact on contagion risk that dissipates as *N* increases.



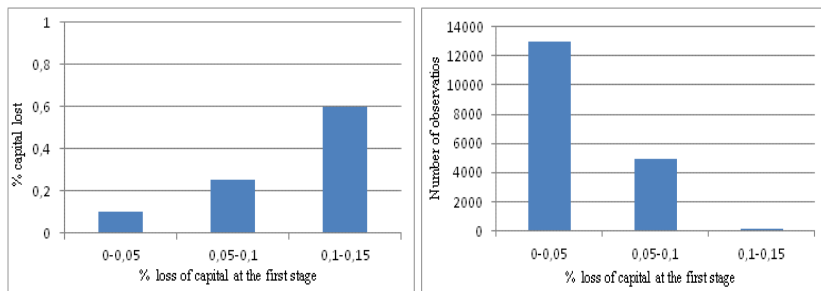
(a) N=20 banks



(b) N=50 banks



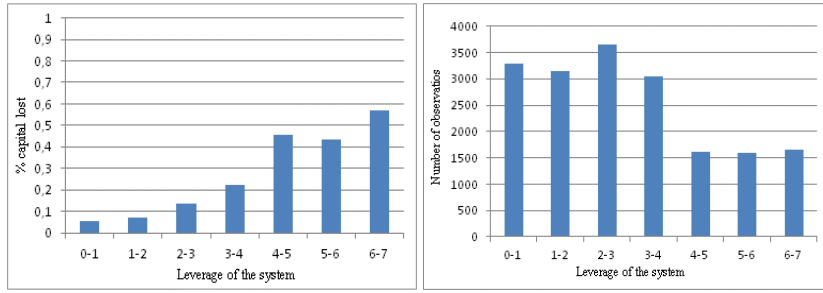
(c) N=80 banks



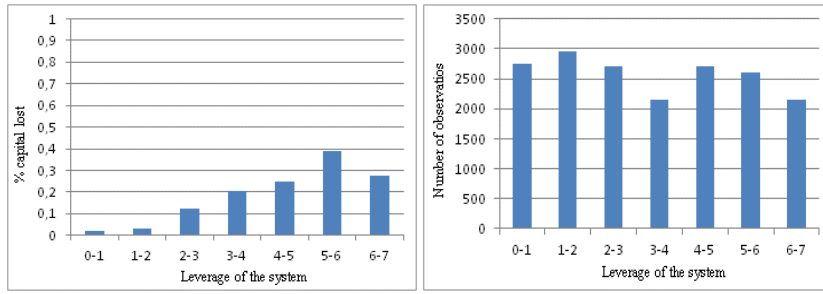
(d) N=100 banks

Figure 5. 7 : Scenario 3: Homogeneous banks with heterogeneous exposures (expressed as the total capital lost from the banking system due to the failure of at least one bank) as a function of the % initial loss of capital due to default of the first bank.

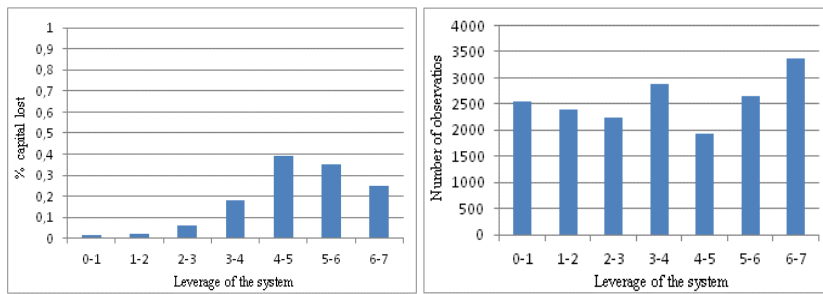
Panels (a)-(d) show the relation between the % initial loss of capital due to default of the first bank and the extent of contagion across interbank networks with different number of banks.



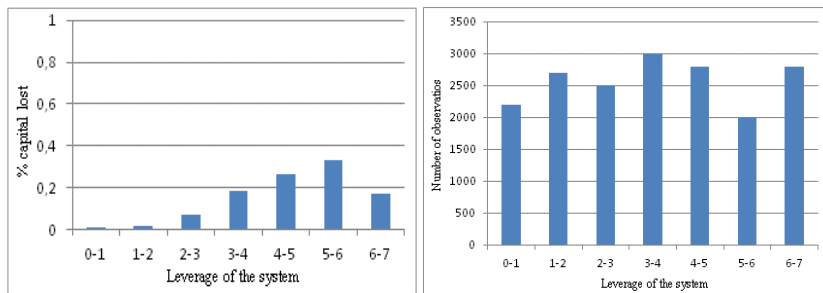
(a) N=20 banks



(b) N=50 banks



(c) N=80 banks



(d) N=100 banks

Figure 5. 8 : Scenario 3: Homogeneous banks with heterogeneous exposures | Extent of contagion (expressed as the total capital lost from the banking system due to the failure of at least one bank) as a function of the leverage of the system.

Panels (a)-(d) show the relation between the leverage of the system and the extent of contagion across interbank networks with different number of banks.

The leverage of the system has a positive impact on systemic risk and its magnitude decreases as the size of the network increases. Figures 6 and 7 illustrate the third scenario as a function of the percentage loss of capital due to default of the first bank in the network and as a function of leverage in the banking system, respectively. As in the previous cases, we find the number of rounds until no further

bank defaults to affect contagion risk positively and statistically significantly, and such impact is magnified in relatively larger interbank networks. The heterogeneity of interbank exposures plays a significant role in the stability of the financial network especially in the medium sized interbank networks.

Finally, Table 5.6 depicts the results of the **fourth scenario** as described in equation (5.19). In this scenario, we construct network systems where banks have the same equity size and interbank claims are evenly distributed across creditor banks. We acknowledge the fact that this scenario is a bit unrealistic as banks in real-world interbank networks do not have the same equity size and do not necessarily distribute their interbank claims evenly across their creditors. However, by testing a wide range of link probabilities between any two nodes, we are in a position to effectively examine the effect of different calibrations on systemic risk. Thus, although this scenario can be regarded as a special case with magnifying effects, it provides useful insights on interbank market resiliency during periods of stress.

The variable CATIN2 has a strong positive impact on systemic risk that dissipates as the network system gets larger. Simulations show that this scenario yields qualitatively similar results with the previous three scenarios in relation to the leverage of the network, that is, leverage positively and significantly affects contagion risk. However, in this scenario, we observe that this effect becomes stronger progressively when the number of constituent banks in the network increases. Figure 5.9 confirms the results recorded in the Table 5.6 concerning the relationship between the extent of contagion and the percentage loss of capital in the network. For instance, the likelihood of systemic breakdowns seems to decrease as we move from smaller to larger network systems since we have very few cases that cause large capital losses. Connectivity impacts negatively on interbank contagion, although this negative impact dissipates as the number of banks in the interbank networks increases. As expected, the link probability has the same negative impact as connectivity on the interbank contagion. Contrary to the previous findings concerning connectivity, the negative impact of the link probability on interbank contagion seems to scale up as we move from smaller to larger interbank networks.

	<b>N=20</b>	<b>N=50</b>	<b>N=80</b>	<b>N=100</b>
<b>CATIN2</b>	0.228 (21.978)***	0.153 (14.098)***	0.137 (12.902)***	0.105 (9.426)***
<b>LEVIN</b>	0.137 (14.890)***	0.268 (28.512)***	0.352 (37.106)***	0.352 (37.707)***
<b>NOUTGOING</b>	-0.257 (-15.906)***	-0.146 (-9.715)***	-0.130 (-8.719)***	-0.095 (-6.262)***
<b>COUNT</b>	0.645 (198.356)***	0.617 (172.925)***	0.568 (150.736)***	0.573 (148.381)***
<b>P</b>	-0.156 (-10.231)***	-0.304 (-21.593)***	-0.378 (-26.723)***	-0.379 (-27.197)***
<b>Adjusted R<sup>2</sup></b>	0.834	0.806	0.817	0.779

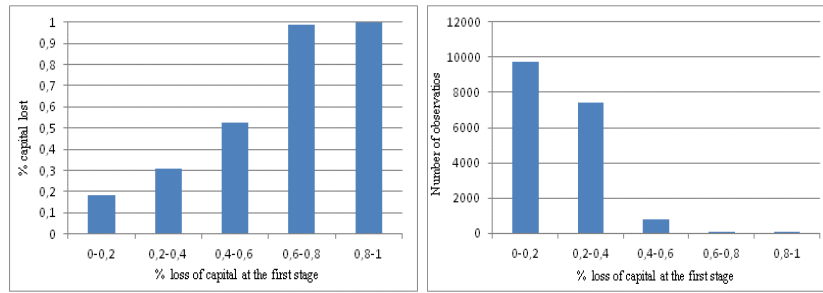
Table 5. 6: OLS regression analysis for Scenario 4 (Homogeneous banks with homogeneous exposures).

The table presents the regression results for Scenario 4. The dependent variable is CATEND measured as the total loss of capital due to contagion as percentage of total capital in the network. Explanatory variables are the constant term CATIN2, LEVIN, NOUTGOING, COUNT and P, the probability for a

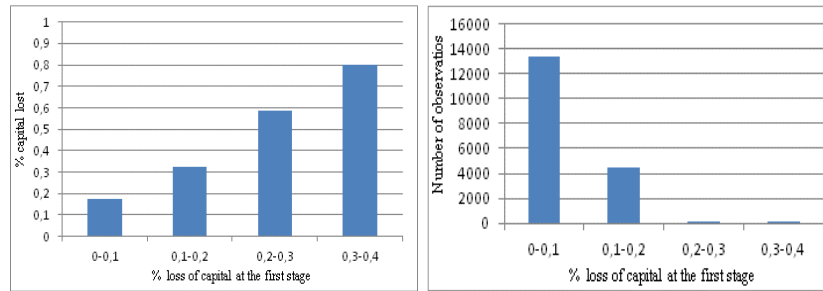
link to exist between two nodes.. Each cell displays the OLS standardized coefficients along with the corresponding *t*-statistics (shown in parentheses). The sample comprises of 18,000 realizations (simulated banking crises). \*, \*\* and \*\*\* denote significance at the 10, 5 and 1 percent level, respectively.

Finally, the number of rounds until no further bank defaults affects contagion risk in a statistically significant manner especially when small interbank networks are considered.

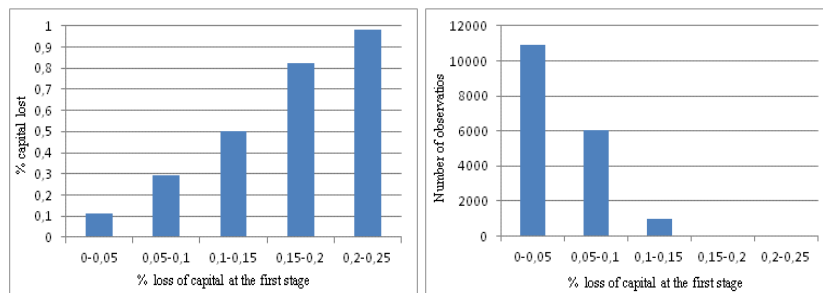
The main intuition behind these results is that increasing connectivity on a homogeneous interbank network can reduce the frequency of contagion in case the first bank that defaults is less leveraged as the interbank network has the dynamics to absorb more easily the shock and thus the initial shock is dissipated at a faster rate. This is the case for small network systems. As the size of the network increase and the system gets more leveraged, the stabilizing force of connectivity weakens and default cascades prevail.



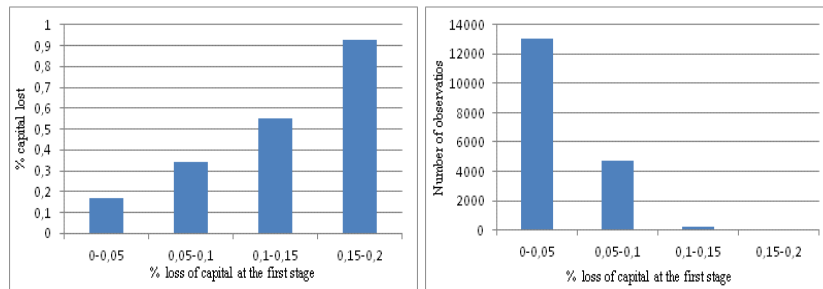
(a) N=20 banks



(b) N=50 banks



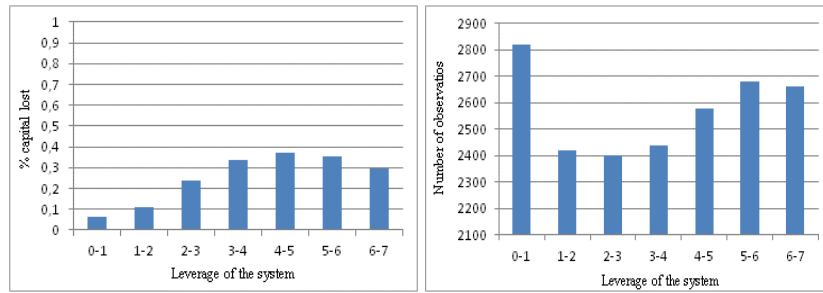
(c) N=80 banks



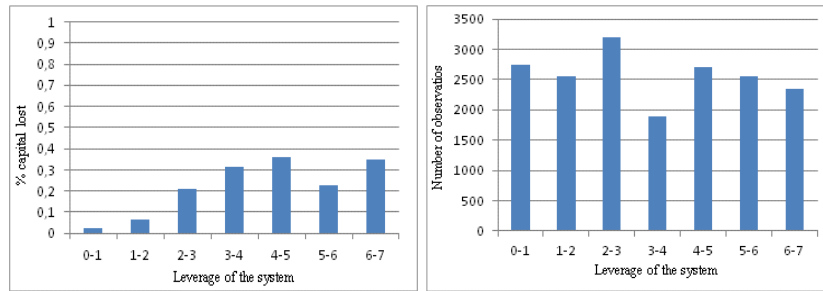
(d) N=100 banks

Figure 5. 9: Scenario 4: Homogeneous banks with homogeneous exposures (expressed as the total capital lost from the banking system due to the failure of at least one bank) as a function of the % initial loss of capital due to default of the first bank.

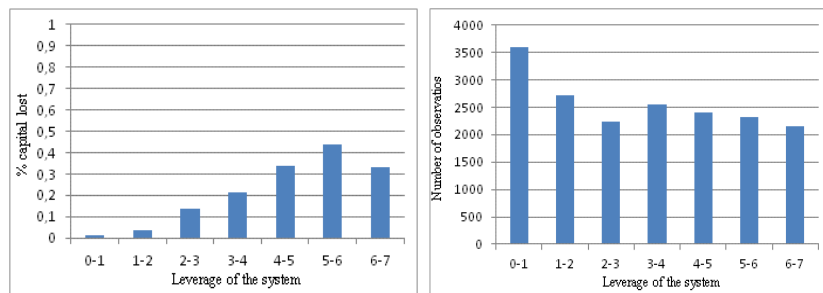
Panels (a)-(d) show the relation between the % initial loss of capital due to default of the first bank and the extent of contagion across interbank networks with different number of banks.



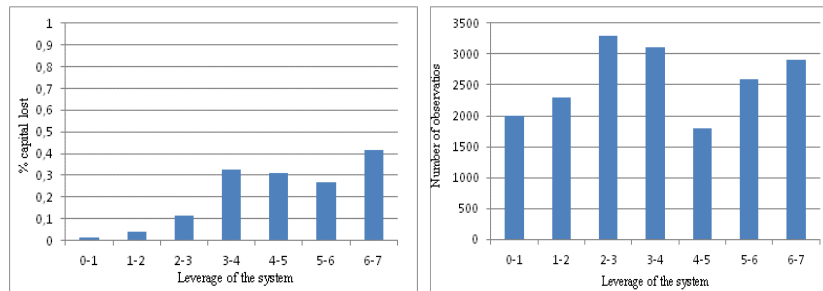
(a) N=20 banks



(b) N=50 banks



(c) N=80 banks



(d) N=100 banks

Figure 5. 10 : Scenario 4: Homogeneous banks with homogeneous exposures | Extent of contagion (expressed as the total capital lost from the banking system due to the failure of at least one bank) as a function of the leverage of the system.

Panels (a)-(d) show the relation between the leverage of the system and the extent of contagion across interbank networks with different number of banks.

Tables 5.7–5.10 depict robustness tests on all four scenarios based on random sampling. We have performed second run Monte Carlo simulations in order to examine whether the new results differ from the previous ones, thus checking how random sampling affects our main conclusions. We observe qualitatively similar results in all four cases to those from the first run providing evidence that our findings are stable across different simulation scenarios.

	<b>N=20</b>	<b>N=50</b>	<b>N=80</b>	<b>N=100</b>
<b>CATIN 1</b>	0.044 (13.363)***	0.001 (0.389)	-0.004 (-1.135)	0.002 (0.579)
<b>CATIN2</b>	0.259 (10.095)***	0.163 (7.044)***	0.073 (3.301)***	0.050 (2.614)***
<b>LEVIN</b>	0.272 (10.899)***	0.255 (11.408)***	0.412 (19.261)***	0.402 (22.155)***
<b>NOUTGOING</b>	-0.213 (-10.339)**	-0.162 (-4.518)***	0.014 (-4.933)***	0.042 (1.328)
<b>COUNT</b>	0.569 (128.765)***	0.604 (147.789)***	0.523 (126.895)***	0.539 (131.678)***
<b>VARCAP</b>	-0.085 (-50.816)***	-0.075 (-57.790)***	-0.057 (-58.108)***	-0.054 (-64.019)***
<b>P</b>	0.021 (-5.089)***	0.141 (3.998)**	-0.006 (-0.171)	-0.012 (-0.405)
<b>Adjusted R<sup>2</sup></b>	0.786	0.785	0.768	0.789

Table 5. 7: Robustness tests: OLS regression analysis for Scenario 1(Heterogeneous banks with homogeneous exposures).

The table presents the regression results for Scenario1 applied on a second run of Monte Carlo simulations based on random sampling as robustness test. The dependent variable is CATEND measured as the total loss of capital due to contagion as percentage of total capital in the network. Explanatory variables are, CATIN1, CATIN2, LEVIN, NOUTGOING, COUNT, VARCAP and P, the probability for a link to exist between two nodes. Each cell displays the OLS standardized coefficients along with the corresponding *t*-statistics (shown in parentheses). The sample comprises of 18,000 realizations (simulated banking crises). \*, \*\* and \*\*\* denote significance at the 10, 5 and 1 percent level, respectively.

	<b>N=20</b>	<b>N=50</b>	<b>N=80</b>	<b>N=100</b>
<b>CATIN 1</b>	0.071 (23.366)***	0.001 (0.273)	0.008 (2.368)***	0.006 (1.730)*
<b>CATIN2</b>	0.207 (20.109)***	0.098 (8.941)***	0.101 (9.336)***	0.068 (6.721)***
<b>LEVIN</b>	0.602 (53.999)***	0.469 (38.008)***	0.313 (26.051)***	0.304 (26.494)***
<b>NOUTGOING</b>	-0.154 (-12.833)***	-0.096 (-4.023)***	-0.064 (-2.747)***	0.008 (0.391)
<b>COUNT</b>	0.459 (107.602)***	0.567 (131.004)***	0.590 (144.084)***	0.609 (156.107)***
<b>VARCAP</b>	-0.038 (-21.431)***	-0.067 (-41.713)***	-0.053 (-54.105)***	-0.051 (-40.597)***
<b>VARLOANS</b>	-0.220 (-42.365)***	-0.091 (-24.628)***	-0.018 (-2.107)**	-0.009 (-12.307)***
<b>P</b>	-0.223 (-18.398)***	-0.080 (-3.217)***	0.092 (3.779)***	0.061 (2.751)***
<b>Adjusted R<sup>2</sup></b>	0.817	0.770	0.772	0.800

Table 5. 8: Robustness tests: OLS regression analysis for Scenario 2 (Heterogeneous banks with heterogeneous exposures).

The table presents the regression results for Scenario2 applied on a second run of Monte Carlo simulations based on random sampling as robustness test. The dependent variable is CATEND measured as the total loss of capital due to contagion as percentage of total capital in the network. Explanatory variables are the constant term CATIN1, CATIN2, LEVIN, NOUTGOING, COUNT, VARCAP, VARLOANS and P, the probability for a link to exist between two nodes. Each cell displays the OLS standardized coefficients along with the corresponding *t*-statistics (shown in parentheses). The sample comprises of 18,000 realizations (simulated banking crises). \*, \*\* and \*\*\* denote significance at the 10, 5 and 1 percent level, respectively.



	<b>N=20</b>	<b>N=50</b>	<b>N=80</b>	<b>N=100</b>
<b>CATIN2</b>	0.200 (25.728)***	0.153 (18.410)***	0.127 (13.078)***	0.157 (21.173)***
<b>LEVIN</b>	0.282 (34.036)***	0.189 (22.195)***	0.329 (32.672)***	0.308 (37.777)***
<b>NOUTGOING</b>	-0.187 (-16.831)***	-0.168 (-11.145)***	-0.114 (-6.923)***	-0.145 (-11.721)***
<b>COUNT</b>	0.745 (190.987)***	0.773 (184.138)***	0.736 (164.477)***	0.765 (190.795)***
<b>VARLOANS</b>	-0.167 (-41.084)***	-0.137 (-33.586)***	-0.164 (-38.890)***	-0.196 (-46.316)***
<b>P</b>	-0.217 (-19.612)***	-0.226 (-14.785)***	-0.371 (-22.288)***	-0.323 (-25.206)***
<b>Adjusted R<sup>2</sup></b>	0.862	0.824	0.789	0.864

Table 5. 9: Robustness tests: OLS regression analysis for Scenario 3 (Homogeneous banks with heterogeneous exposures).

The table presents the regression results for Scenario3 applied on a second run of Monte Carlo simulations based on random sampling as robustness test. The dependent variable is CATEND measured as the total loss of capital due to contagion as percentage of total capital in the network. Explanatory variables are the constant term CATIN2, LEVIN, NOUTGOING, COUNT, VARLOANS and P, the probability for a link to exist between two nodes. Each cell displays the OLS standardized coefficients along with the corresponding *t*-statistics (shown in parentheses). The sample comprises of 18,000 realizations (simulated banking crises). \*, \*\* and \*\*\* denote significance at the 10, 5 and 1 percent level, respectively.

	<b>N=20</b>	<b>N=50</b>	<b>N=80</b>	<b>N=100</b>
<b>CATIN2</b>	0.266 (25.631)***	0.196 (18.103)***	0.153 (13.994)***	0.126 (11.749)***
<b>LEVIN</b>	0.163 (17.098)***	0.247 (25.943)***	0.283 (29.111)***	0.357 (37.852)***
<b>NOUTGOING</b>	-0.306 (-19.180)***	-0.220 (-14.254)***	-0.164 (-10.529)***	-0.118 (-8.020)***
<b>COUNT</b>	0.616 (188.365)***	0.600 (161.885)***	0.609 (160.323)***	0.565 (145.072)***
<b>P</b>	-0.150 (-9.783)***	-0.256 (-17.542)***	-0.309 (-20.730)***	-0.371 (-26.650)***
<b>Adjusted R<sup>2</sup></b>	0.834	0.804	0.790	0.798

Table 5. 10: Robustness tests: OLS regression analysis for Scenario4 (Homogeneous banks with homogeneous exposures).

The table presents the regression results for Scenario4 applied on a second run of Monte Carlo simulations based on random sampling as robustness test. The dependent variable is CATEND measured as the total loss of capital due to contagion as percentage of total capital in the network. Explanatory variables are the constant term CATIN2, LEVIN, NOUTGOING, COUNT and P, the probability for a link to exist between two nodes.. Each cell displays the OLS standardized coefficients along with the corresponding *t*-statistics (shown in parentheses). The sample comprises of 18,000 realizations (simulated banking crises). \*, \*\* and \*\*\* denote significance at the 10, 5 and 1 percent level, respectively.

## 5.5. Conclusions

This paper investigates how complexity under a specific network structure, that has been extensively applied for the study of contagion in financial networks, affects interbank contagion. Similar to Leventides et al. (2019), we explore the interplay between heterogeneity, balance sheet composition in the spreading of contagion using four basic scenarios, under an Erdős-Rényi network structure using a wide range of link probabilities between any two banks.

Our findings indicate a non-monotonic relation between diversification and interbank contagion across the different sizes of interbank networks for all scenarios tested. While for small or medium interbank networks, connectivity can act as an absorbing barrier, such that interbank systems of these sizes can withstand the initial shock, for large network systems connectivity does not seem to provide an effective shield against capital losses. Our results, for the four scenarios tested, indicate that small and thus more concentrated interbank network systems are more prone to contagion. In these cases, we observe that the size of total capital losses is, in most cases, larger than that documented in medium and large sized systems, which is in line with the findings of Nier et al.(2007).

As far as heterogeneity is concerned, this enters in our experiments in the form of interbank claims and bank sizes. Our results clearly suggests that heterogeneity plays a significant role in the stability of the financial system. Similar to Leventides et al. (2019), we still find that when heterogeneity is introduced with respect to the size of each bank, the system's shock absorption capacity is enhanced. In addition, when we allow for heterogeneity on interbank exposures in our model, we observe additional resilience to the interbank network as an initial shock dissipates more easily than in the case of homogeneous interbank claims.

Finally, we should also justify the fact that we choose to work under an Erdős-Rényi network structure even if this network framework is not very realistic. In an such a network framework, where the probability of forming a link is homogeneous, the resulting network structure does not present marked heterogeneity. As we observed from all the four scenarios tested, the initial shock that hits the system seems to propagates into the system jeopardizing thus the stability of the entire system. This strengthens even more our arguments concerning the critical role that heterogeneity plays in the resilience of the financial system.

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## **6. Application of complex network analysis for systemic risk monitoring and policy formulation /Policy insights from interbank networks**

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### **6.1 Introduction**

Representing the banking system as a network is arguably more realistic than to model it as a representative bank, as traditional macro finance models do. For this reason, network analysis can be utilized by policymakers and regulators in developing effective policies on financial stability. During the last decade, there has been a growing interest from financial stability experts at central banks and supervisory authorities in the analysis of financial interconnectedness of financial institutions by means of network representation and analysis. One of the early promoters of the introduction of network theory to financial systems was the Bank of England's chief economist Andrew Haldane. In his speech at the Financial Student Association in Amsterdam on 28 April of 2009, Haldane argued that the crisis presented policymakers and regulators with a large body of evidence and strong incentives to change the way financial markets are understood. For Haldane the shift needed was clear; a better understanding of the complexity of the financial system and the application of some of the lessons from other disciplines – such as ecology, epidemiology, biology and engineering – to the financial sphere.

### **6.2 The Basel Process of Capital Regulation**

The Basel Committee on Banking Supervision (BCBS) has its origins in the financial market turmoil that dates back in the 1980s. In response to disruptions in the global financial markets, representatives from central banks of the G10<sup>11</sup> countries met in Basel, Switzerland to issue guidelines relating to capital and risk management activities of global banking institutions. Since it was established, the BCBS has formulated the Basel I, Basel II, and Basel III accords.

In 1988, the Basel Committee on Banking Supervision (BCBS) produced the first Basel Accord, also known as Basel I (Goodhart, 2011). This document established the first global minimum capital requirements for international banks, in order to improve the stability of the financial sector and maintain confidence in bank solvency. Focusing primarily on credit risk, the intended goal was to define how much capital banks should hold to remain safe. This pure microprudential set of rules was later enforced by law in many countries in 1992 and amended in 1996 to incorporate market risk regulation.

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<sup>11</sup>The committee was expanded in 2009 and 2014 to 28 jurisdictions consisting of central banks and authorities with formal responsibility for the supervision of banking business. Currently, committee members come from Brazil, Canada, Germany, Australia, Argentina, China, France, India, Saudi Arabia, the Netherlands, Russia, Hong Kong, Japan, Italy, Korea, Mexico, Singapore, Spain, Luxembourg, Turkey, Switzerland, Sweden, South Africa, the United Kingdom, the United States, Indonesia and Belgium.

In Basel I, that is, the 1988 Basel Accord weighed the capital owned by a bank against the credit risk it faced. Basel I defined the bank capital ratio and set the ball rolling for solvency monitoring and reporting. Assets of financial institutions were classified and grouped into five risk categories according to credit risk, carrying risk weights 0%, 10%, 20%, 50% and 100%. The 1988 Basel Accord has also set the minimum of 8% of regulatory capital for banks, measured in terms of credit risk-weighted assets

In June 1999, the Basel Committee issued a proposal for a new capital adequacy framework to replace the 1988 Basel Accord. This led to revised Basel framework "International Convergence of Capital Measurement and Capital Standards" (Basel II) which published in 2004. The fundamental goal of the Basel Committee was to further strengthen the soundness and stability of the international banking system. The new Basel Accord comprised three pillars. The first Pillar encompassed the calculation of capital requirements on the basis of bank risks (credit, market and operational risk). Further focal points were the specification of basic principles for qualitative banking supervision and risk management in banks (pillar II), and the introduction of supervisory disclosure requirements in order to strengthen market discipline (pillar III).

The need for extending the Basel II framework appeared quickly after its implementation in 2008 given the new sources of regulatory concerns triggered by the Global Financial Crisis. Thus, in December 2010, the Basel Committee on Banking Supervision (BCBS) published the first version of the new Basel framework "A global regulatory framework for more resilient banks and banking systems " (Basel III). Basel III revises and strengthens the three pillars established by Basel II, and extends it in several areas. The new Basel framework set new standards that targeted both the microprudential and macroprudential levels through multiple individual measures. Increased quality, quantity and transparency of regulatory capital and the introduction of capital buffers are some of the main highlights. Basel III also introduced higher capital requirements for particularly risky products and reduced leverage ratios in an attempt to prevent excessive bank leverage. Under this new framework, more details have been specified on the supervisory treatment of systemically important banks. The definition of "*Global Systemically Important Banks (G-SIBs)*," (Basel Committee on Banking Supervision, 2011) does include, for the first time, the concept of interconnectedness, thereby measured as the aggregate value of assets and liabilities each bank has with respect to other banking institutions. Basel III was initially agreed upon by the members of the Basel Committee on Banking Supervision in November 2010, and was scheduled to be introduced from 2013 until 2015; however, implementation was extended repeatedly to 31 March 2019 and then again until 1 January 2022.

### 6.3 Policy applications

One of the many lessons that emerged from the Global Financial Crisis (GFC) of 2007 is that banks were overseen mainly on individual basis without sufficient consideration for systemic risk. Filling this gap will require a new set of macro-prudential tools to regulate and supervise institutions based on their size and their interconnectedness or complexity. The findings presented in this Thesis have significant implications on banking supervision and policy conduct for central banks and supervisory authorities.

The role of supervisory authorities is critical to preserve financial stability in banking system and limit the contagion of financial stress of a financial institution to others and, thus to avert adverse effects on the proper functioning of the financial system and economy. However, the a priori evaluation and measurement of financial contagion risk is a challenging task since its estimation entails great uncertainty. After all, most regulators and policymakers believe that systemic events can be only identified after the fact. Since the primary role of supervisory authorities is to identify, measure and reduce systemic risk, the identification of such a risk clearly needs to be under a probabilistic framework which can predict the level of overall systemic risk in different scenarios. Many indicators for measuring systemic risk has been proposed over the last decade. Two of them are the Acharya et al.'s (2011) marginal expected shortfall (MES) which estimates a financial institution's loss conditional on the banking system being in distress and the Adrian and Brunnermeier's (2010) Conditional Value-at-Risk (CoVaR) which evaluates systemic losses conditional on each financial institution being in distress. These measures usually take into account the size, the probability of bank default, and even the correlation of each bank. However, these measures do not take into account the reciprocal web of exposures linking financial institutions and the systemic importance of each bank in the banking system.

Based on this gap, researchers and regulators make use of network models in assessing contagion risk. The network models usually consider the interbank network as propagation channel for financial contagion. For this reason, there can be used mathematical models which predict financial contagion based on measurable variables such as the leverage or the density of the network. As pointed out by Billio et al. (2012) when we think about systemic and contagion risk we might focus on the "four L's"; these being leverage, liquidity, losses and linkages. Our research proposes such models that give the ability on the supervisor to quantify the possibility of contagion given various measurable variables that has at his disposal. Our analysis shows that although the risk of contagion is low frequency event, interbank exposures may constitute a devastating channel of contagion at turbulent periods through which problems affecting one bank may spread to other banks. As systemic risk evolves over time, regulatory policies should include not only a frequent monitoring of interbank exposures but also a regular assessment of the interbank market structure,

such as the overall leverage of the system, the nature of the interconnectedness and the heterogeneity across bank sizes and interbank exposures.

Central bank, acting as the supervisor, will have the ability to estimate the dependent variable (contagion effect) taking into account risk indicators (directly measurable variables) that are employed in our analysis such as the leverage of the network, the size or the heterogeneity of the system. The supervisor can monitor these measurements frequently and check if the stability of the system improves or deteriorates. Our numerical simulations suggest that attention should be given to the structure and the size of interbank loans between banks. The crucial thing is to limit systemic risk and the contagion effect by preventing banks from failing in the first place, placing particular emphasis on the systemic banks, being these banks with few connections but large risk exposure each or these banks with many connections and low risk exposure each. Specifically, our results show that once the initial shock spread in the system, the extent to which the propagation will stop is primarily associated with the network structure of interbank exposure in the system and the total capital adequacy of the system. Capital adequacy of the system plays a prominent role whether the interbank network system withstands an initial shock or incur contagious breakdowns with detrimental consequences to the entire economy.

Furthermore, the supervisor can also compare different network structures as far as contagion is concerned. However, this is somehow a passive intervention of the supervisor where he can ring the alarm bells for immediate actions, should the conditions deteriorate. However, the supervisor can also apply active risk management techniques. Parameters' boundaries concerning contagion and systemic risk can be set, ranging from low to medium or high risk. These boundaries can be interpreted as limits to the independent variables that supervisor can measure. Every time the supervisor observes that contagion has entered into a danger zone or has the propensity to enter, he will have to take corrective actions with regards to the variables mentioned above. If for example, the supervisor observes that the leverage of the system is heading into dangerously levels resulting in increased contagion levels, he will intervene aiming to limit the leverage of the system imposing borrowing restrictions to critical banks<sup>12</sup> or imposing increases on the capital buffers that are required to hold. Furthermore, if the increased contagion levels are due to low connectivity across banks or to put it differently, there is increased loan portfolio concentration in particular regions of the network, the regulator will give incentives for increased connectivity and thus risk sharing across banks in an attempt to lower the probability of contagious defaults.

Our results also suggest that it would be wiser and more prudent policy to set capital requirements from a system-wide angle rather than imposing a common threshold to all financial institutions. In other words, capital requirements should be set to each

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<sup>12</sup>This is in line with the latest reforms in banking regulation. An example of this kind of policy reform include the new standard that the BCBS introduced in April 2014, setting a lower large exposures limit for exposures between G-SIBs of 15% of Tier 1 capital as opposed to the 25% limit applied to other counterparties.

bank according to its systemic importance within the system. This notion is in line with the suggestions of Haldane and May (2011) and Alter et al. (2015).

Our suggested analysis is easily explainable, reproducible and can be carried out for all banks in a banking system for a certain point in time. Repeating this exercise periodically for a range of parameters concerning contagion and systemic risk makes it possible to judge how the stability of the financial system evolves over time. This could give regulators important information on how e.g. certain regulatory actions affect the stability of the financial system.

Finally, let us emphasize that regulators should review periodically the parameters of their model they have decided to work with, respond quickly to fast-evolving market conditions and adapt their policies. Early regulatory intervention is of crucial importance, since it paves the way to tackling the undesirable developments contributing to contagion or systemic collapse.

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## 7. Closing Discussion

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The crisis that began in the US in 2007 and developed into a full-blown international banking crisis with the collapse of Lehman Brothers in September 2008 will be remembered in history as the worst financial crisis since the Great Depression. The initial crisis, which originated in the U.S. subprime mortgage-backed securities and collateralized debt obligations markets, affected an ever-widening group of market participants around the world through a complex web of interlinkages. The initial shock spread rapidly through beyond the United States' borders, thus contaminating the global financial system. This distress has led to the default of a large number of banks and jeopardized the existence of many other financial institutions. In an effort to ease the aftermaths of that financial crisis, governments were forced to bail-out large, complex and highly interconnected financial intermediaries as they feared the unforeseeable consequences of their default. Ten years later, one of the many lessons the crisis taught us is the recognition of the importance of interconnectedness as a key dimension of systemic risk. Financial institutions need to be controlled not only in relation to their propensity to undertake high risk due to the protection they enjoy on their liability side, but also in relation to the risk they transmit to other institutions with which they are connected to by a complex web of exposures. For regulators and central bankers the crucial thing about systemic and contagion risk is to measure the systemic importance of individual institutions and whether this importance is translated to their size, leverage or their interconnection with other institutions.

This thesis is the result of an effort to develop a better understanding of systemic risk and to analyze the interplay of several crucial drivers on interbank contagion, such as bank capital ratios, leverage, interconnectedness and homogeneity across banks' sizes. Making use of the tools that network theory provides, we consider network models and try to address the following questions: Does heterogeneity, leverage and interconnectedness matter for systemic risk and the propagation of contagion? If so, in what respect? As pointed out by Billio et al. (2012) when we think about systemic and contagion risk we might focus on the "four L's"; these being leverage, liquidity, losses and linkages.

After reviewing the recent literature on interbank contagion and providing some basic concept from network theory, we start our study by giving a different definition to contagion related to that, that is observed in the recent literature. Thus, unlike most papers in the recent literature (Nier et al., 2007; Gai and Kapadia, 2010; Chinazzi et al., 2015; Amini et al., 2016) we define the term contagion as the situation in which the initial failure of a bank leads to the failure of at least one other bank, while the extent of contagion is measured by the total capital loss in the banking system due to the failure of at least one bank. In other words, we are mostly interested in detecting the magnitude of capital losses in the banking network rather than the number of banks that were adversely affected. We examine, in chapter 4, via a comprehensive



network model the knock-on effects an initial default can bring into the interbank network. Due to lack of data, we generate large number of banking systems via a network structure framework and balance sheet allocation. In each realization, we construct interbank networks of various sizes and test four scenarios by varying the equity size of banks and the interbank exposure structure across creditor banks. Furthermore, we assume that the network of interbank claims and obligations forms randomly. This assumption enable us to capture all possible scenarios that may appear in real-world situations. Our findings show that heterogeneity in bank sizes and interbank exposures matters a great deal in the stability of the financial system, as its absorption capacity is enhanced. Also, the level of interconnectedness hugely impacts on the system's resilience, especially in smaller and highly interconnected interbank networks. We provide also evidence that highly leveraged banks form the main channel through which financial shocks propagate within the system and such effect is more pronounced in large interbank networks than in smaller ones.

In chapter 5, we extend the model developed in the previous chapter to include a wide variety of network topologies and provide a better understanding of the relation between network structure, banks' characteristics and interbank contagion. While the focus of the previous chapter is on the various factors that affect interbank contagion such as bank capital ratios, leverage, interconnectedness and homogeneity across banks' sizes, the model lacks flexibility as far as the variability of the networks links is concerned. In order to circumvent this problem, we introduce the Erdős-Rényi probabilistic network model in our study to provide a wider vicinity of scenarios concerning the network structure of the interbank system and study how homogeneity within the interbank network affects the propagation of financial distress from one institution to the other parts of the system through bilateral exposures. The introduction of the Erdős-Rényi probabilistic network model provides us with a wider vicinity of scenarios concerning the network structure of the interbank system. Under this framework, we build up multiple scenarios of various network structures that include a satisfactory number of cases via Monte Carlo simulations. In every single network that we construct, we investigate the dynamics of cascading defaults from an initial random shock that hits the system. Erdős-Rényi random graph model which is one of the earliest theoretical network models was introduced by Erdős and Rényi (1960). In this random graph, each possible link between any two nodes can occur with a certain independent and identical probability-the Erdős and Rényi probability. The Erdős and Rényi (1960) random graph model is a model in which has been extensively applied for the study of contagion in financial networks, e.g. Iori et al. (2006), Nier et al. (2007), Gai and Kapadia (2010), May and Arinaminpathy (2010) and Amini et al. (2016). Using the Erdős-Rényi network structure, the degree distribution or the connectivity among banks can vary with respect to the chosen probability  $p$ . Thus, each random network generated with the same parameters  $N$ ,  $p$  looks slightly different. Not only the detailed wiring network graph changes between realizations, but so does the number of links. Random graphs or Erdős-Rényi graphs are useful for modeling, analysis, and solving of structural and algorithmic problems

arising in mathematics, theoretical computer science, statistical mechanics, natural sciences, and even in social sciences.

Similar to the next chapter, we explore the interplay between heterogeneity, balance sheet composition in the spreading of contagion using four basic scenarios, under an Erdős-Rényi network structure using a wide range of link probabilities between any two banks. Our findings indicate a non-monotonic relation between diversification and interbank contagion across the different sizes of interbank networks for all scenarios tested. While for small or medium interbank networks, connectivity can act as an absorbing barrier, such that interbank systems of these sizes can withstand the initial shock, for large network systems connectivity does not seem to provide an effective shield against capital losses. Our results, for the four scenarios tested, indicate that small and thus more concentrated interbank network systems are more prone to contagion. In these cases, we observe that the size of total capital losses is, in most cases, larger than that documented in medium and large sized systems, which is in line with the findings of Nier et al.(2007). As far as heterogeneity is concerned which enters in our experiments in the form of interbank claims and bank sizes. Our results clearly suggests that heterogeneity plays a significant role in the stability of the financial system. Similar to the previous chapter, we still find that when heterogeneity is introduced with respect to the size of each bank, the system's shock absorption capacity is enhanced. In addition, when we allow for heterogeneity on interbank exposures in our model, we observe additional resilience to the interbank network as an initial shock dissipates more easily than in the case of homogeneous interbank claims. Finally, we should also justify the fact that we choose to work under an Erdős-Rényi network structure even if this network framework is not very realistic. In an such a network framework, where the probability of forming a link is homogeneous, the resulting network structure does not present marked heterogeneity. As we observed from all the four scenarios tested, the initial shock that hits the system seems to propagates into the system jeopardizing thus the stability of the entire system. This strengthens even more our arguments concerning the critical role that heterogeneity plays in the resilience of the financial system.

From a regulative perspective, our study provide, in chapter 6, insights for the measurement of systemic risk under a network context and give the ability to the supervisor to quantify the possibility of contagion given various measurable variables that has at his disposal. Our analysis shows that although the risk of contagion is low frequency event, interbank exposures may constitute a devastating channel of contagion at turbulent periods through which problems affecting one bank may spread to other banks. As systemic risk evolves over time, regulatory policies should include not only a frequent monitoring of interbank exposures but also a regular assessment of the interbank market structure, such as the overall leverage of the system, the nature of the interconnectedness and the heterogeneity and homogeneity across bank sizes and interbank exposures. The supervisor can also compare different network structures as far as contagion is concerned. However, this is somehow a passive intervention of the supervisor where he can ring the alarm bells for immediate

actions, should the conditions deteriorate. However, the supervisor can also apply active risk management techniques. Parameters' boundaries concerning contagion and systemic risk can be set ranging from low to medium or high risk. These boundaries can be interpreted as limits to the independent variables that supervisor can measure. Every time the supervisor observes that contagion has entered into a danger zone or has the propensity to enter, he will have to take corrective actions with regards to the variables mentioned above.

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