The Role of Contagion in the Transmission of Financial Stress

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Abstract

The paper proposes a novel approach to understand contagion of financial distress in the banking system, which takes into account the spatial nature of the phenomena. We use a Bayesian spatial autoregressive model that treats the likelihood of default of each bank as endogenous, and dependant on the network formed by all the other banks. Identification is achieved by controlling for bank fundamentals and latent macrofinancial and bank specific shocks that have similar consequences to contagion and act as confounding factors. We find that peer effects account on average for approximately 50 per cent of total distress. Through the lens of a simulations exercise we study the importance of the structure of financial networks for financial stability shedding light on the empirical adherence of important theoretical prepositions that remain untested.

JEL classification: E44, G01, C11, G21.

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1 Introduction

Banking crises occur in waves and in each of these waves there are regional clusters, suggesting that contagion effects play a key role in triggering these events (see Reinhart and Rogoff (2009) and Peydro et al. (2015)). The mechanisms of transferring risk from individual entities to financial markets at large lie at the heart of the financial system. It is hard to discern how the losses from the US subprime mortgage lending, which constituted only a small portion of the overall US mortgage market have disrupted the global financial system without considering systemic interdependence. Contagion is a central element for understanding the events leading up to the crisis. It is defined by Peydro et al. (2015) as *a domino effect that a failure of one bank has on other banks and financial intermediaries*. Starting from this definition, this paper proposes a new framework to think about contagion that takes into account the spatial structure of the financial network formed by banks.

The empirical study of spillovers and contagion is one of the most demanding questions not yet fully understood in the literature. The difficulty in pinning down contagion stems from three key analytical challenges emphasized by Rigobon (2016) that motivate our approach. First, because contagion operates indirectly, working through feedback loops that amplify initial shocks to banks' fundamentals, whereby a bank's distress depends on its peers financial condition, it is essential to treat probabilities of failure as endogenous variables as noted by Danielsson et al. (2013), connected through banking links that vary over time. Second, measuring contagion relies on proper identification. Although contagion results in multiple financial institutions experiencing distress, this might also be caused by a coincident deterioration of bank fundamentals or an unfavourable economic environment. Disentangling these effects is tricky because the borders between contagion and macrofinancial fragility are blurred. Since contagion and macrofinancial shocks hit the economy simultaneously, identifying contagion in data is not trivial. Thirdly, heteroskedasticity and outliers are ubiquitous in financial data. As noted by Forbes and Rigobon (2002) failing to account for these features leads to misspecification and biased results and therefore a wrong interpretation of the magnitude of contagion.

Following the seminal contributions of Allen and Gale (2000) and Freixas et al. (2000) on the subject, much of the empirical literature on measuring contagion has focused on the question of whether contagion actually occurred during major episodes of crisis[1]. The apparently simple question is complicated by several statistical hurdles outlined above, that should be addressed to provide unbiased answers.

[1] Forbes (2012) and Peydro et al. (2015) provide a thorough review of the literature. Earlier literature is summarized by Dornbusch et al. (2000) and Rigobon (2002).

One strand of the literature focuses on cross-market correlations and tests for contagion by checking if correlations in equity returns increase significantly after a crisis. However, Forbes and Rigobon (2002), show that the increase in volatility in crisis episodes induces an upward bias in correlation coefficients. The authors highlight heteroskedasticity as the source of the bias, a feature of the data that non-parametric approaches are ill suited to deal with.

Another approach resorts to parametric models based on linear regressions. This strand of the literature studies connectedness in the banking system by jointly modelling time series in an endogenous setting through vector autoregressive models. Diebold and Yilmaz (2009, 2012); Demirer et al. (2018) and others, study spillovers between financial institutions through impulse responses analysis. The significance of one institution in explaining equity returns of others in the system is therefore regarded as the impact of a surprise to one series on others and the variance decomposition of equity returns of a given institution is used to compute a spillover index. This approach successfully establishes measures of connectedness. However, it critically relies on the specific identification scheme employed. Obtaining orthogonalized impulse responses is difficult because it involves establishing an order of exogeneity amongst institutions in the network. To address this problem, Diebold and Yilmaz (2009) report generalized impulse responses following the approach of Pesaran and Shin (1998). An alternative approach is also proposed by Rigobon (2016) and consists in identifying structural parameters by using the time-varying nature of the variance-covariance matrix of the residuals. Although Demirer et al. (2018) finds that global banks' equity connectedness has a strong geographic component, the spatial structure of the data is left unmodeled.

This paper proposes a different approach to measure contagion. We use a Bayesian spatial autoregressive (SAR) model, working on panel data, that takes into account the endogenous nature and spatial structure of banks' probabilities of default. We control for omitted variable bias by including time and bank fixed effects, while treating heteroskedasticity and outliers in the data, which are common concerns in the literature. The idea underlying this approach is to model distress of each individual bank in the system as a function of financial conditions of all other banks and its own fundamentals, while controlling for unobserved macrofinancial shocks and bank specific shocks which act as confounding factors.

Estimation is carried out with Bayesian techniques, whereby a panel of default probabilities of 50 US banks observed from 1990 to 2018 are explained by regressing these on the likelihood of default of other institutions, connected by an exogenous time dependent spatial weights matrix and a set of control variables that describe

banks' fundamentals. Unobserved macrofinancial and idiosyncratic shocks to banks are controlled for, in an effort to mitigating omitted variable bias. Contagion is identified by decomposing banks' probabilities of default into a component due to peer effects - given by spillovers of distress from other institutions, which we label contagion, from the component due to bank's fundamentals, including its liquidity position, solvency, leverage and risk.

The spatial weights matrix plays a key role in our exercise as it characterizes the banking network. We define it as a function of the variance-covariance structure of a VAR estimated on bank equity returns, which brings our paper closer to the literature that approaches contagion from a time series econometrics point of view (for instance Diebold and Yilmaz (2009)). However, unlike this strand of the literature, we refrain from performing structural analysis in a VAR setting and simply use the variance-covariance structure as a measure of inverse distance between banks. The main intuition for choosing this measure of distance between banks is that, if markets are efficient, prices reflect all available information and therefore reflect the level of interconnection between banks. Moreover, certain properties make the VAR variance-covariance matrix an attractive choice. First, it is symmetric, agreeing with the notion of distance. Second, it measures conditional covariance, purging some of the comovement in bank equity returns which are not due to true interconnection. Third, it is easily mapped into the licit space of spatial weights matrices with simple operations which are common in the spatial econometric literature.

Our approach has several advantages that make it particularly suitable to examine contagion of financial distress. First, studying contagion in a spatial econometric setting is appealing because it allows one to think about (and model) contagion as resulting from two main forces - i) interdependence, which is taken as exogenous in the model and ii) propagation, which is endogenous. It is the combination of these two forces that define the magnitude of contagion. Second, it provides a straightforward way of testing for the presence of contagion. Third, it exploits the information of both the cross-section and time-series dimension of the data. Hence, the model can examine how contagion evolves across time and also inform about the heterogeneity stemming from the different banks in the sample. Moreover, the panel structure of the data allows us to specify time and bank fixed effects that capture unobserved macrofinancial shocks and bank specific shocks, mitigating omitted variable bias. Fourth, Bayesian estimation provides flexibility to the model in dealing with outliers and heteroskedasticity, without having to specify a functional form for the former. It is also known to deal well with over-parametrization that arises when modelling variances and introducing time and bank fixed effects.

We find statistically significant and economically powerful spillovers of default probabilities within the banking system. Evidence suggests a banks' probability of default depends to a large extent, on peer effects stemming from the banking network vis-à-vis its own fundamentals. Amongst the principal characteristics of banks, profitability and risk stand out as the most important variables driving financial distress. Everything else equal, an increase of 1 per cent in Non-performing loans across the banking system induces a hike of 28 bps in the likelihood of default of each institution on average. This figure differs significantly across specifications but is statistically significant in every alternative.

Overall, we estimate that about 50 per cent of the average probability of default of banks is due to contagion. This result stems from the identification of the part of the probability of default that is due to the dynamics of the banking system. This parcel is disentangled from the remainder that can be interpreted as resulting from a bank's specific characteristics which are reflected in its balance sheet. We calculate the spillover resulting from idiosyncratic shocks to banks fundamentals, showing its consequences for financial stability and find significant heterogeneity amongst banks, revealing that some institutions are systemically more important than others. On average, an increase of 100bps in the probability of default of the most systemically important bank will have a knock-on effect of approximately 200bps on the remaining banks' probability of default. Whereas, the least influential bank's spillover is immaterial, amounting to less that halve per cent.

Consistent but parallel to the results of Gupta et al. (2017) that study the importance of interconnectivity in the banking system and its consequences for credit markets, We find that ρ, the amplifying parameter in our model, changes significantly when adding sequentially year fixed effects. In particular, year fixed effects that control for the Great Recession pull down significantly our estimates of ρ. This suggests that time fixed effects are key in purging the effect of macrofinancial shocks that should not be confounded with contagion.

In another direction, we contribute towards the discussion of the implications of the structure of financial networks to financial stability. We set up a simulation exercise whereby in each iteration a fictitious but empirically plausible financial network with a given level of interconnection and clustering is drawn and combined with real data to estimate contagion. The exercise sheds light on the empirical adherence of important theoretical prepositions in the literature that remain untested.

Although the theoretical guidance of the seminal contributions by Allen and Gale (2000) and Freixas et al. (2000) suggests diversification mitigates contagion, other authors argue otherwise (for example Blume et al. (2011, 2013)). Recently

Acemoglu et al. (2015) proposes that beyond a certain threshold, a more diversified network is a source of fragility since a negative shock to the financial system gets amplified more easily. From this point of view, diversified networks increase the likelihood of systemic distress undercutting the benefits of diversification. Through the lens of a spatial econometric model, contagion varies positively with interconnection. We find that the stronger the connections of each bank with the network, the more powerful contagion effects are. Results suggest however that high interconnection is necessary but not sufficient for the financial system to experience episodes of high contagion, which favours the idea that interconnection is a risk factor of financial contagion.

Are more clustered financial network less prone to contagion ? Allen et al. (2010b) suggest clustered financial networks, characterized by high levels of asset commonalities amongst banks, foster contagion. Our results are broadly consistent with this idea. We find that clustering results in a fat tailed distribution of the magnitude of contagion. When clustering is high, contagion will greatly depend on the level of interconnection. It is when clustering is high that we observe episodes of extreme levels of contagion.

The relationship between interconnection, clustering and financial stability is less straightforward. Results suggest that operating with high clustering and low interconnection delivers the best outcome from a financial stability point of view, characterized by high levels of stability and resilience. However, high clustering is also associated with the worse outcomes in terms of resilience. Overall, high clustering should be regarded as a risk factor associated with weakly resilient networks.

The remainder of the paper proceeds as follows. Section 2 reviews the literature on the main channels of contagion. Section 3 explains the econometric framework, describes the data, estimation technique, providing the definitions and propositions that support the empirical results. Section 4 discusses the main results and findings, including those related to the simulation exercise in section 4.5. Section 5 concludes.

2 The main channels of contagion

To begin the discussion, it is helpful to briefly describe the channels through which contagion materializes, that have been identified in the literature, and how the approach adopted throughout the paper brings them together in a common framework [2].

[2] Peydro et al. (2015) discusses each contagion channel in detail. Here we provide a summary of the most important forces at work.

The simplest channel of contagion is related to expectations. Because agents in the economy know that bank defaults are positively correlated, when a given bank experiences financial hardship, agents update their beliefs regarding the financial condition of the other banks. This channel is related to the birth of central banks in the nineteenth century and the establishment of deposit insurance funds to avoid bank runs. As shown by Diamond and Dybvig (1983) in the absence of deposit insurance, it is optimal for a depositor to run on a bank, even if the bank's solvency position is sound. In that sense, the belief that a bank's financial condition is weak may be a self-fulfilling prophecy. In well-developed financial systems contagion through expectations migrated to interbank markets since the risk of a bank run was mitigated by deposit insurance and convertibility suspension during times of financial distress. The short term, unsecured nature of lending activity in such money markets poses a significant threat to banks that over-rely on such a source of funding as was the case of several institutions during the recent crisis.

Another critical channel of contagion is conterparty risk, whereby distress of a bank in the system spills over to other banks that hold unsecured claims on it. Iyer and Peydró (2011) examine financial contagion stemming from the interbank market network, showing that contagion is greater for banks that hold larger amounts of unsecured wholesale funds. Moreover, the authors find that bank fundamentals play an important role in determining the magnitude of contagion, suggesting weaker banks are more exposed to contagion risk. Interconnections amongst institutions are found to be a key element of contagion dynamics, propagating a shock that is amplified by generating second round effects that go beyond the first (direct) effects. As emphasized by Peydro et al. (2015), counterparty risk is one of the major channels of contagion that played a central role in the banking crisis preceding the Great Recession. The significant increase in the derivatives market worldwide and in particular the ABS (asset backed securities) and CDS (credit default swaps) market that were closely related with subprime assets, increases the web of conterparty relationships between institutions, channelling distress that originated in a niche part of the overall financial system.

Contagion can also occur through liquidity. Diamond and Rajan (2001) show that contagion can happen not just because of bank runs and interconnectivity between institutions within the system but also because bank failures reduce overall liquidity in the financial system. Liquidity is critical for banks because one of their main functions is performing maturity transformation (ie, banks typically borrow short term and lend long term, Berger et al. (2012)). An inappropriate liquidity position of a bank can result in default because the duration of banks' assets tends

to be longer than the duration of its liabilities.

Liquidity risk materializes if the three sources of liquidity of banks (repo, securities and unsecured interbank markets) collapse simultaneously. Gorton and Metrick (2012) discuss the role played by the complexity of some assets held by banks in driving liquidity dry-ups (such as ABS and CDOs). Information asymmetry in the market of such assets generated by the difficulty in valuing these products explains why their markets might freeze, contaminating subsequently both the repo market and the unsecured interbank market. The repo market on one hand relies on these assets that are used as collateral. Whereas, the unsecured interbank market stalls via two main channels. First, banks hoarded liquidity to hedge liquidity shocks rather than lending in money markets. Second, monitoring costs increase if borrowers in money markets hold a significant amount of assets of difficult valuation.

As mentioned above, the repo market is one of the three sources of liquidity of financial institutions. It offers banks the possibility of obtaining liquidity provided that good collateral can be pledged. A repo or repurchase agreement is a form of short-term borrowing whereby the dealer sells an asset to a third party and buys it back at maturity, usually overnight. As shown by Brunnermeier et al. (2009) the dynamics of the repo market during a crisis may lead to two types of liquidity spirals. First, if lenders set the repo's price or haircut as a function of the collateral's risk or volatility, an endogenous shock resulting in greater asset volatility will lead to higher haircuts and thus a liquidity shortage. Second, in a similar way a decrease in asset prices will also have the effect of reducing liquidity, since funding through repo's is pegged to the market value of collateral. The liquidity shortage that results from these two mechanisms forces banks to sell assets to make up for the liquidity shortfall, imposing a downward pressure on asset prices in the economy. The feedback loop from liquidity shortages and depressed asset prices reinforce each other generating a liquidity spiral. Liquidity shortages have also been linked to fire sales that occur when banks are forced to sell their assets, usually at a discount. Acharya and Yorulmazer (2008) examine this phenomena and show how the interaction of liquidity shortages with fire sales may trigger systemic banking crisis.

One other potential channel of contagion is related to information disclosure. Khan (2010) and others argue that fair value accounting is associated with an increase in contagion amongst banks. The mechanism is straightforward and works as follows. The book value of banks changes instantly in response to bad news, capital is therefore decreased and creditors revise upwards the risk premium surcharge, squeezing the bank's financial condition further. The authors find that the mechanism is stronger during periods of market illiquidity. While this argument is

coherent, there are three potential counterarguments to why this contagion channel's relevance might be negligible. First, rational investors price assets based on the present value of future cash flows rather than on book values. Moreover, the implicit assumption that investors remain optimistic in the absence of negative information disclosure underestimates the forward looking capacity of markets. Secondly, fair value accounting can be, to some degree, biased because valuation is carried out by the banks themselves based on internal models. Third, only a portion of financial institutions balance sheet is marked-to-market.

The recent crisis highlighted an important dimension of contagion, connected with nonbank financial institutions such as insurance companies, hedge funds, mutual funds and other entities broadly known as shadow banks such as special purpose vehicles and other off-balance sheet conduits. The latter offered banks the possibility of benefiting from regulatory and rating arbitrage thus becoming popular in the run up to the crisis (see Brunnermeier (2009)). As banks adopted an "originate-to-distribute" business model, whereby loans are underwritten, securitized and sold to other entities, the shadow banking system increased in size. Contagion lies in the relationship of banks with these third party entities. One lesson from the crisis was that, when investors started to sell-off liabilities belonging to special purpose vehicles that had been created by banks to issue asset backed securities, the banks themselves were forced to "bail-out" these entities, absorbing losses that originated off-balance sheet (see Hellwig (2009)).

The previous paragraphs discuss the most important channels of contagion highlighted in the literature. Figure 1 summarized the main forces at work.

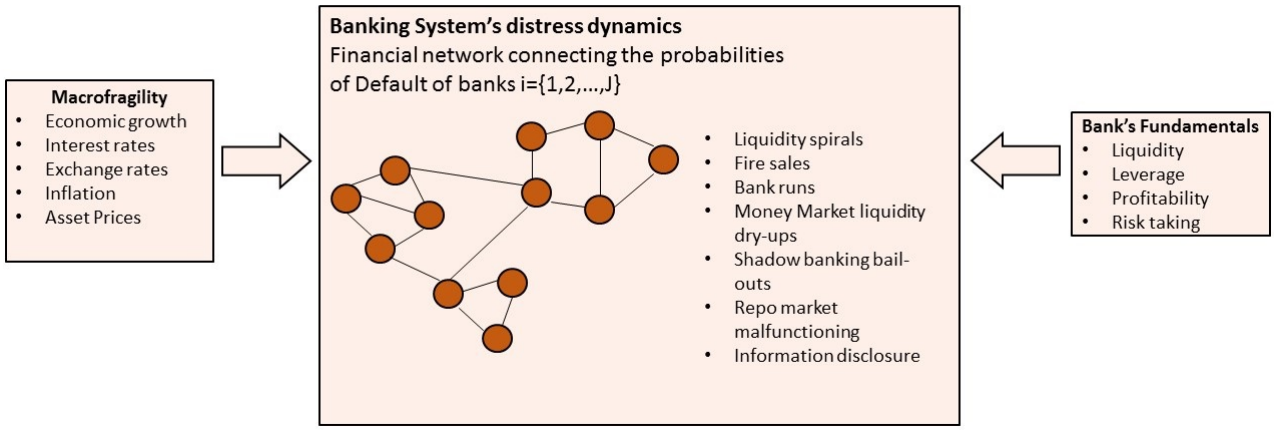


Figure 1: A blueprint of contagion - The main channels.

Although it is obvious that contagion channels interact and reinforce each other, it is challenging and beyond the scope of this paper to disentangle each mechanism separately. Instead, we focus on identifying overall contagion that results from the interaction of all these channels taken together, controlling for two main factors. First, macrofragility stemming from macrofinancial shock that vary over time. These include changes in the economy's financial conditions, risk-aversion, exchange rates, inflation expectations or asset prices, inter alia. These factors hit the banking system and lead to increases financial distress of each bank taken separately. However, they are orthogonal to contagion. Second, bank fundamentals such as liquidity, leverage, profitability or risk appetite are also controlled for.

Contagion in this paper is thus capturing any movement in default probabilities of banks due to peer effects, that is unrelated with macrofinancial shocks of any sort or movements in bank fundamentals, measured by the most relevant financial data on banks. The section that follows explains exactly how this problem is approached analytically.

3 Econometric Framework

A spatial autoregressive model is used to describe the dynamics of default probabilities of banks and in particular how financial strain can spillover from one institution to become systemically relevant. The first step to this exercise is to calculate the default probabilities that are unobserved in practice and should be estimated *a priori* with appropriate techniques. For this purpose we use Merton (1974) structural model [3].

The main idea underpinning the spatial model setup is that financial conditions of banks with strong economic relations are not independent but spatially correlated. In this context, three different types of interaction effects may explain the interdependence of default likelihoods of different banks: i) endogenous interaction effects, where financial conditions of a bank depend on the state of the banking system; ii) exogenous interaction effects, where the likelihood of default depends on the bank's fundamentals such as its liquidity, solvency position and profitability; iii) correlated effects, where similar unobserved common macrofinancial shocks hit banks at the same time resulting in a similar behaviour of their default probabilities. The model

[3]See Appendix A for an explanation the method employed. It should be stressed that such default probabilities are available through financial data providers such as Thomson Reuteurs or Bloomberg but only for the largest financial institutions.

can be written as follows

$$y_{it} = \rho \sum_{j=1, j\neq i}^{N_t} w_{ij,t} y_{jt} + X_{it}\beta + \mu_t + \phi_i + \varepsilon_{it}. \quad (1)$$

Where y_{it} denotes the probability of default observed across banks $i = \{1, ..., N\}$ at time $t = \{1, ..., T\}$, $\sum_{j=1,j\neq i}^{N_t} w_{ij,t} y_{jt}$ the spatially lagged dependent variable summarizing the endogenous interaction effects, where the interaction between banks is defined by a time-dependent spatial weights matrix W_t, with a generic element $w_{ij,t}$, whose construction is explained in the next section. Note that for each time period, a set of N_t probabilities of default are added to the regression. N_t represents the number of banks observed for each time period t that vary across time because some banks leave and others enter the panel. The banks whose dependent or independent variables include missing values are dropped for that specific time period t. X_{it} denotes the matrix of K independent variables, characterizing banks' fundamentals considered and ε_{it} stands for the disturbance terms of the different spatial units.

The parameter ρ is known as the spatial autoregressive coefficient, and is at centre stage in our exercise. It is endogenously determined and reflects the dependence between the default likelihood of a given bank with the set of default probabilities of all other banks. Hence, a higher ρ suggests greater amplification effects of financial stress from the network, hitting individual banks. β is a $K \times 1$ vector of parameters deemed random in a Bayesian setting and μ_t and ϕ_i control for time and bank fixed effects. The last terms μ_t and ϕ_i are key in ensuring that identification of actual contagion is archived. They certify that contagion is not being confused with macrofinancial and bank idiosyncratic shocks that have the same result as contagion but are conceptually independent from it. Time fixed effects μ_t control for macrofinancial shocks that hit the hole banking system at large and may cause default probabilities to jointly rise. Whereas, bank fixed effects ϕ_i control for isolated events that hit a single bank, at a time, that may lead to its default likelihood peaking for reasons lateral to contagion.

3.1 Direct and spillover effects of financial distress

One of the main advantages of using spatial econometric setup is that they offer the possibility of measuring direct and indirect (spillover) effects of the various explanatory variables on distress probabilities across banks. It is clear from the analytical expression 1 that a change in any of the covariates included in X_{it} will have a direct effect on bank i and potentially all other banks $j \neq i$ indirectly,

since financial distress of a bank explicitly depends on the likelihood of default of other banks. To measure the direct and indirect effects it becomes necessary to find the matrix of partial derivatives of the expected value of y_{it} with respect to each regressor. This can be done by writing model 1 in its reduced form as

$$y_{it} = (I - \rho W_t)^{-1}(X_{it}\beta + \mu_t + \phi_i) + (I - \rho W_t)^{-1}\varepsilon_{it}, \tag{2}$$

from which one can derive,

$$\frac{\partial E(y)}{\partial X_k} = (I - \rho W_t)^{-1}\beta_k. \tag{3}$$

Equation 3 measures the impact of a change in the k-th explanatory variable on the dependent variable (ie, probability of default) in the short term. Thus, summarizing the direct and indirect effects or externalities of a change to any covariate on financial stress. Analytically the direct effect is captured by the principal diagonal elements of expression 3, while the indirect effect is captured by off diagonal elements.

Building of the abovementioned setting, let us define contagion as follows,

DEFINITION 1 Consider a symmetric financial network of size N described at time t by the spatial weight matrix W_t. Then financial contagion is given by

$$\sum_{i \neq j}^{N} (I - \rho W_t)_{ij}^{-1} \tag{4}$$

Where I stands for the identity matrix of order N and ρ is the spatial autoregressive parameter, that measures amplification.

Hence, financial contagion is capturing the strength whereby a shock to a given bank's level of distress is propagated through the system and generating an externality that is unrelated with the financial condition of all other entities in the system. It is helpful to notice that the quantity described in 4 is the sum of the off-diagonal elements of a square matrix with ones on its principal diagonal. These off-diagonal elements reflect the impact of the bank represented row-wise by the node j on its counterparty depicted in column i - the indirect or multiplying effect.

The quantity expressed by 4 offers important insights about how distress propagates through the system. Let us write the infinite series of the matrix representation of 4 as

$$(I - \rho W)^{-1} = I + \rho W + \rho^2 W^2 + \rho^3 W^3 + ..., \tag{5}$$

which is simply the taylor expansion of 4. Each element of the infinite sum measures the propagation effect of a shock to the system. For instance, ρW the first round effect, $\rho^2 W^2$ the second-round effect of a shock to distress of a given bank and so forth.

Another important implication of Definition 1 is that if $\rho = 0$ or W_t is diagonal then contagion is non-existent. Thus, contagion may be viewed as a product of diversification or interconnection in the system, which is described by W, and an amplifying mechanism ρ.

PROPOSITION 1 Let ρ be the spatial autoregressive parameter, endogenously given by the spatial econometric model 1 measuring amplification. Then, under H_0, contagion does not exist.

$$H_0 : \rho = 0. \tag{6}$$

Proposition 1 provides a straightforward assessment of the presence of contagion. This can be easily done in a frequencist setting by looking at the t-test for ρ or in a Bayesian framework by examining the posterior distribution of ρ.

COROLLARY 1 Suppose the Data Generating Process of $\{y_1, y_2, ..., y_N\}$ is described by model 1 with $\rho = 0$. Then financial contagion is not present and the model becomes a standard panel regression.

It should be stressed however, that more interesting than assessing the existence of peer effects is to examine their magnitude. This can only be done by fully estimating the model. Before proceeding to estimation the following subsections explain how the financial network is constructed.

3.2 Charactering the network of US Banks W_t

Our approach relies on finding an appropriate measure of distance between banks that reflect their relationship and interconnectivity. This measure should ideally depict how intertwined the balance sheet of banks are. Hence, banks with strong commercial relationships or exposed to the same risks will be close, whereas banks with weak connections ought to be distant. Proximity is therefore not a measure of physical distance in our framework but takes the form of a social distance. It is also worth noting that, at this stage we are not concerned about causality. We are simply looking for a measure of interconnection between banks and not for a causal relation whereby one institution's condition impacts another.

Under the Efficient Market Hypothesis, equity prices and therefore the market value of each institution reflects all available information. Hence, contingent on this hypothesis, the extent to which the balance sheet of a pair of institutions is interconnected may be approximated by the degree of comovement of equity prices of such entities. The main intuition is that if two banks are exposed to common risks including potential claims on each other's balance sheet, their equity prices ought to commove and this can be captured by the covariance of equity prices of all entities.

To formalize this idea, we follow Diebold and Yilmaz (2009) and assume that the data generating process of R_t, the equity price returns of banks i=1,...,N, are well described by the following vector autoregressive process,

$$R_t = BR_{t-p} + \Sigma\varepsilon_t, \tag{7}$$

where, B is a matrix of autoregressive coefficients, Σ is the variance-covariance matrix of the VAR process, p is the number of lags and $\varepsilon_t \sim N(0,1)$.

Our main motivation for using the VAR specified in 7 is to find a suitable measure of distance. Therefore, the commonalities between our approach and that of Diebold and Yilmaz (2009) end in the estimation of model 7. Unlike the authors, which impose some structure on Σ, we simply use this quantity as a measure of inverse distance between banks (ie, the greater the covariance between a pair of entities the smaller the distance between them) [4]. The variance-covariance matrix Σ of the reduced form residuals of the VAR expressed in 7 are ideal measures of distance for two reasons. First, Σ is a symmetric matrix, satisfying the fundamental property of symmetry of distance [5]. Second, it is easy to convert Σ into a licit spatial weight matrix as discussed below.

DEFINITION 2 Let Σ_t be the Variance-Covariance matrix of the reduced form residuals of the process which describes Bank equity price returns R_t, for a set of banks $i = 1, ..., N$, estimated on a given sample $t = 1, ..., T$. The generic elements of the spatial weights matrix w for time T are given by

$$w_{ij} = exp\{D\Sigma D\}_{ij} \quad i \neq j, \tag{8}$$

and $w_{ij} = 0$ if $i = j$, since $d(x, x) = 0$. $D = diag(\Sigma)^{-1}$ is a diagonal matrix

[4] Although Diebold and Yilmaz (2009) only mentions the term "contagion" once, preferring the term "spillover", variance decompositions embed some form of causality necessary to solve the ambiguity inherent to the identification of structural shocks in the VAR.

[5] symmetry between any two points $x, y \in M$, where M is a metric space is satisfied if and only if $d(x, y) = d(y, x)$.

which include the variances of equity price returns for all banks.

The above definition describes the relation between the Variance Covariance matrix of the VAR describing bank equity price returns and the spatial weight matrix. Its calculation involves two transformations. First, Σ is normalized to the range $[0, 1]$ by pre and post multiplication of D. Second, we take the exponential of this quantity and thereby guarantee that off-diagonal entries of W_t are positive [6]. Whereas, diagonal elements of w_t are set to zero to exclude self-influence and be consistent with the notion of distance.

Next, we discuss how to allow the interdependency of banks in our model to vary over time.

DEFINITION 3 Let W_t be a block diagonal matrix, where each sub-matrix in the principal diagonal results from the row normalization of w_t - the spatial weights matrix describing the financial network at time t.

Definition 3 ensures that the row-wise sum of the spatial weight matrix W_t adds to 1, which is common practice in the literature. It's block diagonal structure allows the relations between entities to change over time. It is worth highlighting that each element w_t that compose the full fledged matrix W_t need not to have the same dimensions for each t. In fact, our dataset includes banks that are effectively 'dead', meaning that they were included in some time periods and disregarded in others. Hence, the generic element $w_{ij,t}$ in matrix W_t characterizes the distance between bank i and bank j at time period t. It results from the covariance structure between the equity returns of both banks, normalized to take values between 0 - no relationship; and 1 - strong relationship, as described.

An alternative to measure distances between financial institutions broadly used in the literature (see Gupta et al. (2017); Iyer and Peydró (2011)) consists in looking into the network of interbank market claims amongst institutions using detailed data on banks' counterparty risks. This approaches' merits are highlighted by Peydro et al. (2015) that also notes the main criticism to such an exercise. It ignores the channels of contagion other than those working through money market counterparty risks. In an efficient markets framework, prices are better devices for capturing all information on the connection between institutions, reflecting potential losses deriving from counterparty risk but also liquidity dry-ups and common exposures.

[6] the exponential transformation is popular in the spatial econometrics literature see LeSage (1999)

3.3 Interconnection, contagion and financial stability

The relationship between the structure of a financial networks and the magnitude of financial contagion is not straightforward and several conflicting views co-exist in the literature. The seminal contributions of Allen and Gale (2000) and Freixas et al. (2000) suggest that a more diversified network improves the resilience of the system to idiosyncratic shocks of individual banks. The main argument is intuitive. A more densely connected financial system is better hedged against the risk of an individual default, because a potential loss is shared between a larger number of creditors. In contrast to this view however, other authors argue that as interconnections heighten, the likelihood of a systemic crisis increases (see Blume et al. (2011, 2013)).

A synthesis of these two distinct lines of though is provided by Acemoglu et al. (2015). The author argues that it is the size of the shock to the system in times of crisis that defines whether a diversified network is desirable from a financial stability perspective. In the presence of small shocks, more diversified networks lead to a more robust financial system. However, in the event of large shocks, diversification facilitates contagion and fosters fragility. The intuition behind this result is that a small shock is better absorbed by a sufficiently diversified network that allows for risk sharing in a more efficient way. However, if the magnitude of the shock exceeds a certain threshold, the system's liquidity is insufficient to absolve the total loss and defaults emerge in cascade.

Another dimension of diversification is explored by Allen et al. (2010a) and has to do with the level of clustering in the network. The main idea is that asset commonalities amongst banks determines the extent of information contagion and hence the likelihood of default. In this view, the magnitude of contagion depends on the degree of clustering in the financial network. In the clustered financial network, where the risk is more concentrated, contagion is higher. Thus, diversification has two dimensions - interconnection and risk concentration or clustering.

We will empirically examine how the structure of the financial network - and in particular how interconnection and clustering, determine the magnitude of contagion. A spatial model is a good device to shed light on the relationship between interconnection and contagion since it allows for a clear identification of the forces at work. We will use this setting to test empirically the adherence of some theoretical results discussed so far. In particular, we ask how contagion changes when interconnection increases and how endogenous mechanisms captured by the amplification parameter ρ play a role in driving contagion. Since a definition of financial contagion is provided in Definition 1, the only element missing is a concise definition of interconnection and clustering. We follow the literature, in particular Acemoglu

et al. (2015) and define interconnection in a financial network using the concept of harmonic distance.

DEFINITION 4.1 Assume a financial network is defined by W, a square matrix describing the inverse distance between each node/bank in the network, where the generic element w_{ij} depicts the relationship between bank i and bank j. Then the harmonic distance of bank i to the network is given by

$$H(i) = \sum_{j \neq i} \frac{1}{w_{ij}}. \tag{9}$$

REMARK 1 Assume that the level of interconnection of bank k with the financial network is well described by the harmonic distance measure $H(k)$. Then the greater $H(k)$ the less interconnectedness in the network.

Definition 4.1 establishes a measure of interconnection, given by the inverse distance of banks with the banking system. Notice that greater levels of $H(.)$ suggest lower interconnection. Additionally, we measure the level of clustering in a financial network as follows.

DEFINITION 4.2 Let C_τ be a copula, describing the joint distribution of the elements in W and $\{f(w_{11}), f(w_{12}), ..., f(w_{JJ})\}$ be marginal distribution functions of each random variable w_{ij}. Then, function F denoting the joint distribution function of the financial network W is given by

$$F(w_{11}, w_{12}, ..., w_{JJ}) = C_\tau(f(w_{11}), f(w_{12}), ..., f(w_{JJ})). \tag{10}$$

Where τ is the copula's parameter and J stands for the number of banks in the network.

REMARK 2 Assume that a financial network structure is depicted by $F(w_{11}, w_{12}, ..., w_{JJ}) = C_\tau(f(w_{11}), f(w_{12}), ..., f(w_{JJ}))$. Then τ measures the level of concentration and greater levels of τ, indicate higher levels of clustering in the financial network.

A copula specifies a dependency structure between two or more random variables [7]. It is used in this paper to empirically model the definition of a clustered financial network put forward by Allen et al. (2010a). Intuitively, a clustered financial network is one where banks are not only connected but also hold identical portfolios. Thus,

[7] We discuss copula theory that is necessary for this paper in Appendix B and provide further references.

due to asset commonalities and more generally, common risk exposures, risk is more concentrated in a clustered network. On the contrary, an unclustered financial network is one where, none hold identical portfolios and consequently the risk is less concentrated. A copula allows us to empirically model tail dependency, which characterizes co-movement in interconnections.

Finally, to understand the impact of interconnection and clustering on contagion and its consequences for financial stability it is useful to define two desirable properties of financial networks

DEFINITION 5 Consider two financial networks defined by W and $\tilde{W}$. Conditional on the presence of financial contagion of magnitude c

[1] W is more stable than $\tilde{W}$ if $E[c|W] < E[c|\tilde{W}]$.

[2] W is more resilient than $\tilde{W}$ if $max\{c|W\} < max\{c|\tilde{W}\}$.

Stability and resilience are defined above as the empirical counterparts of the theoretical proposal of Acemoglu et al. (2015) and hereby imported to our empirical setting. They represent the expected and worst-case levels of contagion.

To study the impact of changes to the structure of the financial network W, we carry out a simulation exercise in order to examine the extent to which contagion changes in the presence of a more diversified vis-a-vis less diversified network. The routine is explained in detail in Appendix C. The main idea is to generate a large number of hypothetical financial network structures and examine how both contagion and financial stability are affected by interconnection and clustering. Next, we examine the following three prepositions in light of our spatial model.

H1. *Interconnection in financial networks mitigates contagion.*

H2. *A less clustered financial network is less prone to contagion.*

H3. *Interconnection in financial networks benefits financial stability.*

Before discussing the results an explanation of the data and estimation technique used is given in the next section.

3.4 Data and Estimation

A panel of the 50 largest US banks (by market value) are observed from 1990 until 2018, including those that have been de-listed to avoid exposure to survival bias. Daily data on stock market returns is used to construct the financial network. Probabilities of default are also calculated for a one year horizon, using daily data on stock market returns, market capitalization and total balance sheet liabilities.

The spatial regression specified in equation 1 relies on a set of measures of bank fundamentals meant to purge the effect of bank specific factors that describe a banks' financial condition. These include market value of each institution as a measure of size, loan to deposit ratio as a measure of liquidity. Earnings per share (EPS) and Return on Equity (ROE) are included to account for profitability, whereas leverage, Market-to-Book and Non-performing loans account for risk. A full description of the variables used in Table 1 appendix B

The criteria underpinning the choice of the specific regressors to include with a view of describing banks fundamentals was a compromise between the effort of including the most important indicators of a bank's financial position and not loosing too many observations due to missing values.

The general model used throughout the paper is known in the literature as spatial autoregressive - SAR model. It can be written in stacked matrix form as

$$y = \rho W_t y + X\beta + \varepsilon, \tag{11}$$

$$\varepsilon \sim N(0, \sigma^2 V) \tag{12}$$

Where X includes all K+2 regressors capturing bank fundamentals plus time and bank fixed effects. Potential heteroskedasticity is captured by V, a diagonal matrix modelling the dynamics of the variance, $V_{ii} = v_i, i = 1, ..., n, V_{ij} = 0, i \neq j$.

Estimating specification 11 in a Bayesian setting has the advantage of allowing the study of the full posterior distribution of the amplification parameter ρ, key to understand contagion dynamics and moreover, the quantity that allow us to gauge the spillover effect of a shift in each specific bank's fundamentals, given by $(I - \rho W_t)^{-1}$. Estimation through Bayesian methods also adds value by extending the basic spatial regression model to accommodate outliers and heteroskedasticity.

The likelihood of the SAR model specified above may be written as

$$p(\Psi|\beta, \sigma, \rho) = (2\pi\sigma^2)^{-n/2}|A|exp\Big[-\frac{1}{2\sigma^2}(Ay - X\beta)'(Ay - X\beta)\Big]. \tag{13}$$

Where $\Psi = \{y, X, W\}$ the data ; $A = (I_n - \rho W)$, thus $|A|$ is the determinant of A and n the number of observations. Bayesian estimation proceeds in the conventional way by specifying a prior $p(\theta)$ for each parameter included in $\theta = \{\beta, \sigma^2, \rho, V\}$ which is combined with the likelihood specified above to produce the posterior $p(\theta|\Psi)$ that may be found from a straightforward application of the Bayes' rule,

$$p(\theta|\Psi) \propto p(\Psi|\theta)p(\theta). \tag{14}$$

Hence, a first step to solve the model is to specify a set of priors for the parameters at hand. We set independent normal, inverse-gamma - NIG prior for β and σ^2, a uniform prior for ρ and a Chi-square for the n variance scalars v_i that give form to V.

$$p(\beta) \sim N(c, T), \tag{15}$$

$$p(\sigma^2) \sim IG(a, b), \tag{16}$$

$$p(\rho) \sim U(\lambda_{min}^{-1}, \lambda_{max}^{-1}), \tag{17}$$

$$p(r/v_i) \sim iid \quad \chi^2(r). \tag{18}$$

The choice of a NIG prior is motivated by its widespread use in the Bayesian Econometrics literature (see Koop (2003); Koop and Korobilis (2009)). We set a=b=c=0 and assign a very large prior variance for β (large T) such that our priors are uninformative. This is due to the fact that we do not wish to include any prior information about our parameters and rather remain agnostic.

The uniform prior for ρ is adopted by LeSage (1999) and is the sensible option given that we also want to remain agnostic *a priori* about what values should the dependence parameter take. It is possible and straightforward however in this framework to restrict ρ to a given interval such as $[-1, 1]$ or $[0, 1]$. This option might be tempting given that a negative ρ in this exercise might seem counter-intuitive. However, since this parameter plays an important role in our model, adopting a diffuse prior whereby the upper and lower limits of ρ are defined by $\{\lambda_{min}, \lambda_{max}\}$, which represent the minimum and maximum eigenvalues of the spatial weights matrix is more prudent and gives more flexibility to the model.

The modelling strategy to extend the SAR model to allow for heteroscedasticity was introduced by Geweke (1993). The set of scalars v_i are included to capture dynamics of the variance of the errors ε of unknown form. The prior for these is controlled by one single parameter r, representing the degrees of freedom of the χ^2 distribution. This allows us to estimate n variance terms v_i, by adding one single hyperparameter r to the model. The idea underlying such an approach is that changes to the key hyperparameter r can exert a significant impact on the prior of the parameters that it controls. This features gives the model more flexibility and can also boil down to the homoskedastic case where $V = I_n$ that happens if the hyperparameter r is assigned very high values.

Estimation proceeds in the standard way in a Bayesian setting by applying Bayes Theorem and combining the priors and likelihood following expression 14 to get the

posterior

$$p(\beta, \sigma^2, \rho, V|\Psi) \propto (\sigma^2 V)^{a^*+(k+2)/2+1}|A|exp\Big[-\frac{1}{2\sigma^2 V}[2b^* + (\beta - c^*)'(T^*)^{-1}(\beta - c^*)]\Big], \tag{19}$$

$$c^* = (X'X + T^{-1}(X'Ay + T^{-1}c), \tag{20}$$

$$T^* = (X'X + T^{-1})^{-1}, \tag{21}$$

$$a^* = a + n/2, \tag{22}$$

$$b^* = b + (c'T^{-1}c + y'A'Ay - c^*(T^*)^{-1}c^*)/2. \tag{23}$$

An important remark concerning the posterior is that, unlike standard normal linear models discussed in Koop (2003) that have conjugate NIG priors (ie, where the priors integrate with the likelihood to produce a posterior of the same family of distribution as the prior), the NIG priors in a spatial econometric framework do not result in a posterior of known form. Hence, the need to use a Markov Chain Monte Carlo (MCMC) sampler to find its distribution. The next section outlines the details of the MCMC used to estimate the model at hand.

3.4.1 The MCMC sampler for the heteroskedastic SAR model

Using an MCMC sampler is a common approach to deal with the hurdle of analysing complicated posteriors of unknown form. The main idea is to breakdown the problem of finding the full posterior density $p(\theta|\Psi)$ into smaller problems consisting of analysing the conditional distribution of each single parameter in θ. By sampling sequentially from these conditional distributions and bringing them together, one may approximate the full posterior.

We adopt the MCMC algorithm known as Metropolis-Hastings, named after the original authors seminal contribution. Hasting (1970) shows that given an initial value for the parameters θ_0 it is possible to construct a Markov-Chain up to state t with the correct equilibrium distribution, by sequentially drawing candidates θ^*, spanning the space of the parameter set, in such a way that a large number of samples of the posterior $p(\theta|\Psi)$ are generated. This algorithm is thus capable of sampling from conditional distributions for which the distribution form is unknown while the Gibbs sampler, an alternative MCMC routine, can only solve problems

where the conditional distributions of the parameters are of a known form.

The SAR model specified in 11-12 and 15-18 is a hybrid case since it involves conditional distributions of known form for the parameters $\{\beta, \sigma^2, V\}$ whereas the distribution of ρ is not known. We adopt the approach suggested by LeSage (1997) known as *Metropolis within Gibbs sampling*. Overall, this approach involves sampling for the parameter ρ through a Metropolis-Hastings routine, while using a Gibbs sampling from a normal and inverse gamma distributions for the parameters β and σ that result from the NIG priors used. To make this clear, the algorithm is summarized below. Starting from a set of arbitrary values β_0 σ_0^2, V_0 and ρ_0, the procedure involves sampling from the following conditional distributions.

1 Sample $p(\beta|\sigma_0^2, V_0, \rho_0)$ from $N(c^*, T^*)$, where the hyperparameters are calculated as

$$c^* = (X'V^{-1}X + \sigma^2 T^{-1})^{-1}(X'V^{-1}(I_n - \rho W)y + \sigma^2 T^{-1}c), \tag{24}$$

$$T^* = \sigma^2(X'V^{-1}X + \sigma^2 T^{-1})^{-1} \tag{25}$$

Keep the sampled draws β_1 and replace the existing β_0.

2 Sample $p(\sigma^2|\beta_1, V_0, \rho_0)$ from $IG(a^*, b^*)$, with hyperparameters calculated as shown below

$$a^* = a + n/2; \quad b^* = (2b + e'V^{-1}e)/2; \quad e = Ay - X\beta. \tag{26}$$

Keep the sampled draws σ_1^2 and replace the existing σ_0^2.

3 Sample for each diagonal element of V (v_i) conditional on all others, v_j with $i \neq j$. $p(\frac{e_i^2 + r}{v_i}|\beta_1, \sigma_1^2, v_j)$ from $\chi^2(r+1)$, where e_i represents the ith element of vector $e = Ay - X\beta$.

Keep the sampled draws V_1 and replace the existing V_0.

4 Sample ρ from its unknown distributional form found in LeSage (1997),

$$p(\rho|\beta_1, \sigma_1^2, V_1) = |A|exp\Big[-\frac{1}{2\sigma^2 V}e'V^{-1}e\Big] \tag{27}$$

Some final remarks regarding the estimation approach are in order. First, steps [1-4] entail one pass-through our MCMC algorithm. While the conditional distributions for the parameters sampled in [1-3] are known, the distribution in equation 27 is unknown, hence the need to recur to the M-H for sampling within an otherwise straightforward Gibbs sampler. To find a way around the unknown conditional distribution of ρ, we follow LeSage and Pace (2009) that, based on the proposal of Holloway et al. (2002), suggests the use of a *tuned random walk procedure* to gen-

erate a candidate value for the parameter ρ. In each pass-through h of the MCMC routine, a candidate ρ^c is drawn from the following distribution

$$\rho^c = \rho_{h-1} + c \times N(0,1). \tag{28}$$

This candidate value can be seen as random deviate from the previously accepted value ρ_{h-1} adjusted by a tuning parameter c.

The candidate drawn ρ^c is then used as an input to a standard M-H algorithm whereby an acceptance probability is calculated by evaluating both the candidate draw and the draw accepted in the previous pass-through in the conditional distribution of ρ given in 27. The acceptance probability is given by

$$\psi(\rho^c, \rho_{h-1}) = min\Big[1, \frac{p(\rho_{h-1}|\beta,\sigma)}{p(\rho^c|\beta,\sigma)}\Big]. \tag{29}$$

The tuning parameter c is adjusted based on this acceptance probability. When $\psi(.)$ falls below 0.4 it is modified such that the new parameter $c^* = c/1.1$ whereas, if it increases above 0.6, $c^* = c \times 1.1$. The purpose of this technique is to ensure that the draws of ρ span the entire space of the conditional distribution.

Figure 15 provides some diagnostic of the M-H sampler. We observe that the acceptance probability adjustments vary significantly between 0.4 and 0.6 for the first 2000 replicas, stabilizing thereafter. The bottom panel of the same figure shows the movements of the M-H sampler for the parameter ρ. No pattern of autocorrelation seems to exist and draws seem to be independent.

4 Discussion of the results

This section discusses empirical results focusing on the following questions. First, is there any evidence of contagion ? In particular, how much of the likelihood of default is on average explained by contagion ? Second, is there evidence of heterogeneity between banks with respect to their spillover effect on other banks distress ? and third, what is the direct and indirect effect of a shock to a bank's fundamentals on other bank's distress ? Lastly, we investigate the empirical relationship between interconnection, contagion and financial stability.

Before addressing these questions, a brief description of bank level probabilities of default and the banking network calculated *a priori* is in order. The likelihood of default of each financial institution is given by the probability of the bank's asset value falling below its total liabilities within one year. Figure 11 describes the individual dynamics of these quantities since 1990 for the 9 largest institutions in

our sample as of 2018, measured by market capitalization. We observe that literally every major financial institution experienced varying degrees of financial distress during the Great Recession. This point is more obvious in Figure 13, which plots the joint behaviour of the probabilities of default for all US banks. Unsurprisingly, the probability of default of a significant amount of institutions peaked in the run-up and unfolding of US recessions since the 1990. The three main events that can be observed in the data were the savings and loans crisis in the beginning of the 1990s, the dot.com bubble and more significantly the US subprime mortgage crisis that resulted in the Great Recession. Figure 14 depicts the network of banks before and during the onset of the Great Recession. Interconnectivity fell as institutions decoupled from each other.

4.1 Baseline results

The starting point for any spatial econometric analysis is to question the assumption that regression errors are idenpendent and identically distributed, which is necessary to derive OLS estimates with desirable properties. Hence, a natural first step is to test whether the spatial dynamics of bank probabilities of default are statistically meaningful. To do so, we start by estimating a simple panel regression, reported in Table 3, column I and compare it with Maximum Likelihood SAR model estimates in column II. As mentioned earlier, if the data does not have a spatial structure then the SAR will boil down to the simple panel regression model estimated with a Pooled OLS. From a statistical viewpoint ρ, the spatial autoregressive coefficient, is significantly different from zero. Moreover, Moran I statistic, another measure of spatial autocorrelation is also statistically significant, pointing towards the conjecture that the data generating process is better described by a model that allows for a spatial structure.

The Bayesian spatial autoregressive model, estimated with time and bank fixed effects is reported in the column III. Column IV, which presents baseline results, accounts for heteroskedasticity. It is important to emphasize that ρ, the parameter that measures amplification of distress between institutions, is significantly different from zero across all specifications, which suggests that contagion is an important factor to take into consideration when thinking about the likelihood of default of each bank. Its magnitude varies across specifications but in all cases it is economically meaningful. In the SAR specification III, ρ equals 0.39. Whereas when heteroskedastic errors are modelled, the estimate of ρ increases to about 0.55.

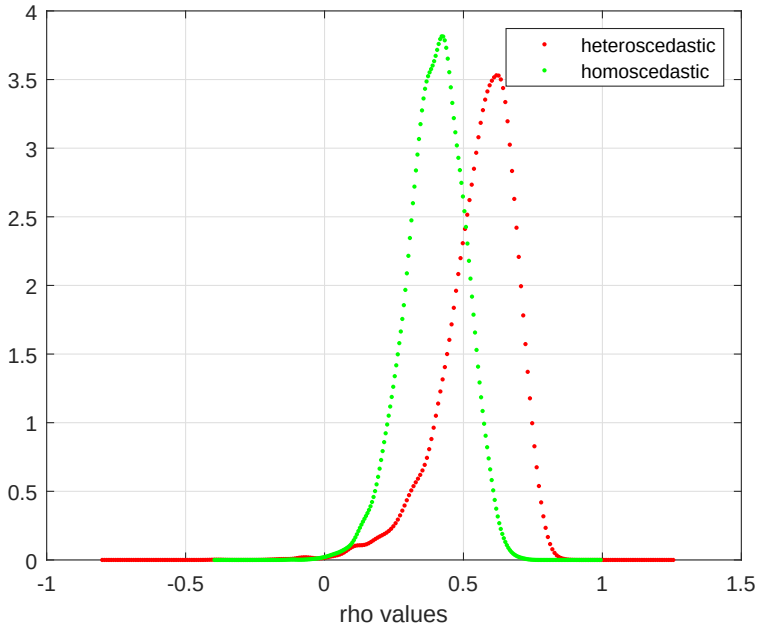
Another important aspect to highlight refers to the covariates included to account for bank fundamentals. These include Leverage, which refers to bank's total debt to

common equity. Profitability, measured as the return on Equity (ROE). Size, which is accounted for by including the stock market capitalization. Risk, which include Non-performing loans, measured as a percentage of total loans. Liquidity which is proxied by the loan-to-deposit ratio. Valuation relative to fundamentals, captured by the Market-to-Book value and Earnings Per Share.

Profitability and risk are found to be the most meaningful predictors of bank distress. Everything else equal, an increase of 1 per cent in Return of Equity is estimated to lead to a 25 bps decrement of the likelihood of the default of a given institution. Furthermore, Non-performing loans are estimated to increase the likelihood of default. On average, for each 1 percentage point increase in NPLs, the likelihood of distress increases by 28 bps. This magnitude is even larger for other specification highlighting the importance of loan delinquencies for the solvency of the banking system which was in the origin of the systemic banking crisis leading to the Great Recession. These figures do not include spillovers effects. We will discuss these in detail further in the paper.

As mentioned previously, the empirical literature on contagion emphasises the importance of taking into account heteroskedasticity when measuring contagion and spillovers. Our baseline model explicitly accounts for heteroskedasticity of unknown form, while remaining agnostic about its functional form.

Figure 2: Full sample estimates of the posterior distribution of ρ



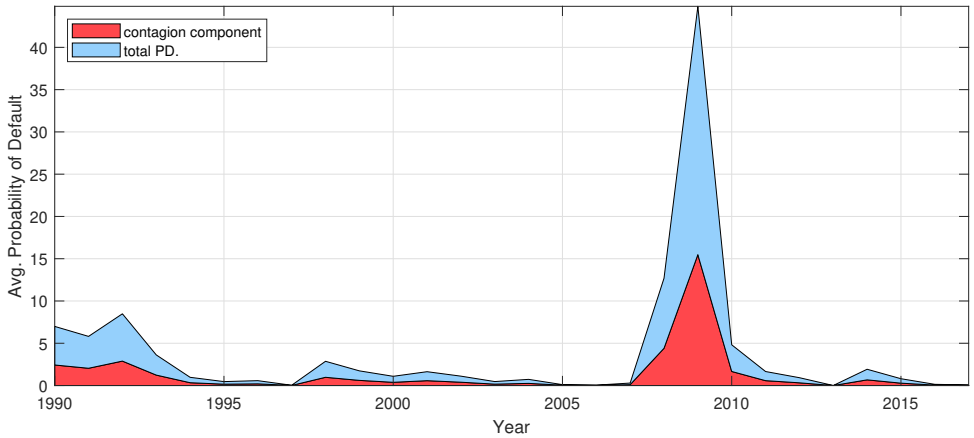
Notes: Posterior distribution of ρ as given by the homoskedastic Bayesian sar model vis-à-vis the same specification taking into account heteroskedasticity.

Figure 2 shows that, the estimate of the magnitude of contagion greater when heteroskedasticity in taken into consideration. However, some caution is needed in interpreting this result since the size of and sign of the bias is ultimately an empirical question and different samples may lead to different answers. Therefore, this result can not be generalized. Nevertheless, in our setup the values of ρ increase significantly. It should be stressed that even if the reality of stochastic volatility is disregarded, there is still strong evidence for the presence of contagion.

4.2 Contagion across time

We now focus on answering the main question of how much of the probabilities of default of banks does contagion account for, on average. To address this point, we decompose the probabilities of default into two components. The parcel due to contagion, that reflects peer effects or spillovers of distress from other institutions is isolated from the component due to own fundamentals that include profitability, valuation, size, liquidity and risk. To mitigate omitted variable bias, estimation controls for time and bank fixed effects. These additional regressors ensure that macrofinancial shocks and bank idiosyncratic shocks do not bias our estimates of contagion.

Figure 3: Contagion component of average probability of default of the US banking system over the period 1990-2018.

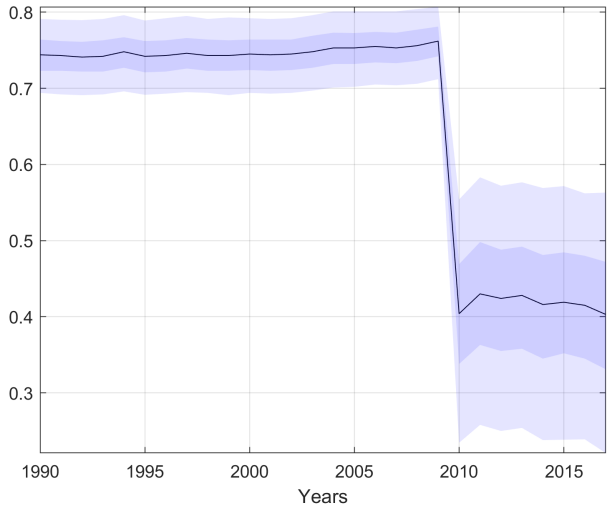


Notes: Decomposition of probabilities of default for US banks, 1990-2018. Red stacked line represents the proportion of the total observed probability of default due to contagion.

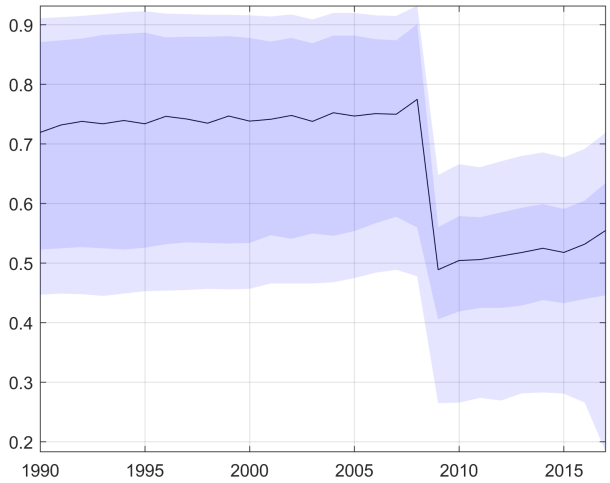
Figure 3 shows that contagion accounts for a significant part of the likelihood of default of banks, on average 53 per cent throughout the full sample. We have discussed that contagion in our model is a product of two distinct forces. On one hand, interconnection between banks that is exogenous and defined by banking links that

vary over time. On the other hand, amplification measured by ρ that is endogenously defined. We now examine how this parameter changes when adding sequentially year fixed-effects, considering the models with and without heteroskedasticity.

Figure 4: Time-varying spatial autocorrelation in the US banking system 1990-2018.



(a) Time-varying posterior distribution of ρ - Homoskedastic



(b) Time-varying posterior distribution of ρ - Heteroskedastic

Notes: Posterior density of the parameter ρ estimated on the full sample, adding sequentially year fixed effects to the baseline Bayesian SAR with and without heteroskedastic errors. Colour bands highlight the 5th, 25th, 75th and 95th percentiles of the posterior distribution of ρ.

Two main messages can be taken from Figure 4. First, when accounting for heteroskedasticity our estimates of the magnitude of contagion increase. Comparing

both panels in the Figure, one can observe a significant difference in the size of ρ. Second, time fixed effects are effective in capturing unobserved macrofinancial shocks. Notoriously, the "crisis" dummies seem to be picking up the effect of the joint uprise of probabilities of default during 2008-2009 which would bias our estimates of ρ if left unaccounted. More importantly, we see that even controlling for these factors contagion is still sizeable and statistically significant.

4.3 Contagion across banks

Modelling contagion with a spatial model offers the advantages of being able to explore the time dimension but also the cross-section dimension of contagion. In this section we examine the significance of each bank in our sample in driving other banks distress.

Table 4 investigates the contribution of each bank in inducing system wide distress and the vulnerability of each bank in the event of a shock to another institution. Column 1 reports the average probability of default for each of the 50 largest institutions in the US banking system since 1990. Institutions in the Table are sorted from largest to smallest in terms of size, as measured by market value as of January 2018 [8]. Column 2 quantifies the first term in our baseline regression 1, which can be interpreted as the probability of default of each bank due to contagion. Therefore, this term indicates the vulnerability of each institution to an external shocks hitting other banks. It is worth noting that, given that column 1 reports observed probabilities of default and column 2 presents the parcel of fitted probabilities due to contagion, as implied by the model, it is possible to observe higher levels of contagion contributions than actual probabilities of default. This will happen when financial fragility as proxied by a bank's fundamentals is actually contributing negatively to its probability of default. In other words, such a bank will exhibit above average levels of fundamentals. Whereas, the following column expresses the indirect or spillover effect of a one standard deviation shock to a given banks' fundamentals. These quantities give an idea of the externality or importance of each bank for overall financial stability.

Results suggest significant heterogeneity with respect to both the systemic importance of each bank and its vulnerability to external shocks. This is due to heterogeneity in the behavior of each entity's probability of default over time and its relevance in the banking network. Furthermore, evidence suggests that vulnerability to external shocks and systemic importance are not directly related to size. In particular, banks which appear to have a knock-on effect on the system, and thus

[8] We consider that the market value of banks that are effectively 'dead' is the last observed.

larger impact on financial stability do not necessarily belong to the top 10 largest institutions within those considered in the Table. On the other hand, banks presenting greater vulnerability (ie, largest contagion component) are also not necessarily the top 10 club of largest institutions. Overall, results show a significant heterogeneity of banks with respect to their systemic importance and vulnerability.

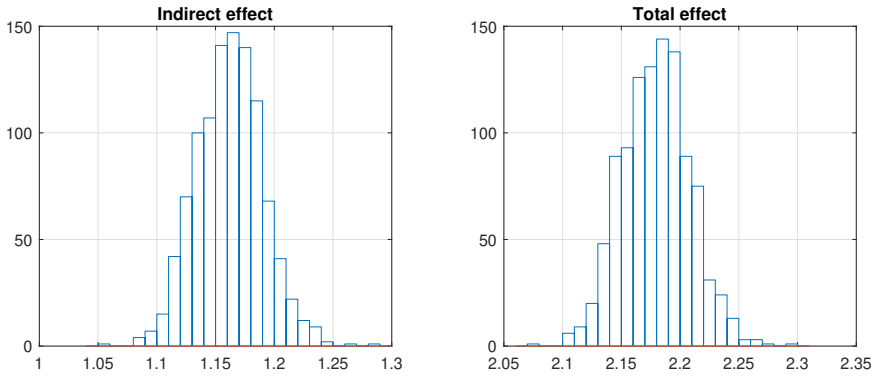
4.4 Spillover of shocks to banks fundamentals

How are bank's financial condition compromised by a deterioration in their fundamentals ? We examine this point through the lens of our spatial econometric model estimated in previous sections. Table 5 reports the main results estimated for the baseline specification - the Bayesian SAR model with time and bank fixed effects and an heteroskedastic error term. The values of the credibility set around these estimates (ie, the relevant percentiles of the posterior distribution of these estimates) are also reported in the table.

We find that the most relevant bank characteristics that influence the likelihood of default are non-performing loans and profitability as measured by Return on Equity. In particular, an increase in 1 percentage point in the Return on Equity of a bank is found to reduce distress by approximately 60 basis points (bps), of which 34bps accounts for positive spillovers that pass-through neighbouring banks and back to itself. Whereas, an increase in 1 percentage point in delinquency rates, as measured by NPLs, are estimated to increase financial distress by 65 bps, on average of which 36 bps account for spillover effects. Although non-performing loans are statistically significant predictors of the probability of default of banks, it should be noted that the estimate of the magnitude of their effect on distress is subject to significant model uncertainty as this estimate changes materially across specifications. Amongst other potential drivers of distress included in the model, neither appear to significantly predict probabilities of failure.

Figure 5 below, summarizes the magnitude of the multiplying effect through which a one unit shock to the k-th explanatory variable gets amplified, resulting in a spillover that equals such multipliers times β_k. The distribution of the multiplying effect is discretely left skewed and concentrated between 1.1 and 1.25, yielding a total effect that lies between 2.05 and 2.3, suggesting that shocks to bank fundamentals have powerful peer effects.

Figure 5: Spillover magnitude of a shock to financial distress in the US banking system.



Notes: Posterior distribution of the indirect and total effect in the baseline specification.

4.5 Interconnection, contagion and financial stability

To empirically examine the extent to which clustering and interconnection in financial networks represent heightened contagion risk, we design a Monte Carlo experiment and draw a hypothetical financial network in each iteration. Each pseudo financial network $\tilde{W}$ results from taking random draws from a lognormal distribution, which are then mapped into the space of licit spatial weight matrix (see Appendix D for details). The random numbers used to generate $\tilde{W}$ are drawn from a Gaussian copula with a given fixed parameter τ, which as argued, defines the level of clustering in the network. In an effort to guarantee that $\tilde{W}$ is empirically plausible, although artificial, we parametrize the distribution generating each replica with $\bar{\Sigma}$ and V_{Σ}, the mean and standard deviation of the posterior distribution of the Variance-Covariance matrix of the VAR specified in 7, which is based on observed interconnections.

Therefore, the artificial financial network $\tilde{W}$ composed by random draws $\tilde{w_{ij}}$ generated by this procedure depends on the distribution of W which is assumed to follow an lognormal distribution and the level of clustering which is controlled by the copula family used and its parameter τ. This exercise allows us to measure in each draw

- Interconnection - as the harmonic distance $H(k)$ set forth in Definition 4.1;
- Contagion $\tilde{c}$ - which results from re-estimating the model for each draw $\tilde{W}$;
- Stability and Resilience - which are mere statistics that can be obtained for each pair $\{\tilde{W}, \tilde{c}\}$.

The simulation exercise is carried out for the case in which the artificial financial

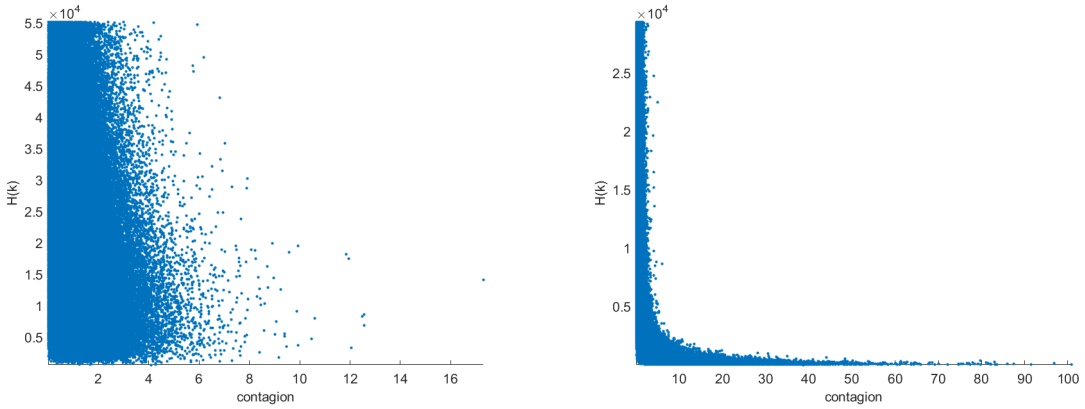
network is set to a low clustering regime (ie, the value of ρ is set to 0.1 to represent low clustering) and a high clustering regime (where $\rho = 0.9$). Turning to the results of the simulation exercise, we examine whether the evidence validates each of the three hypothesis discussed in section 3.3

H1. *Interconnection in financial network mitigates contagion.*

We start by examining the relationship between interconnection and contagion in light of our simulation exercise. Figure 6 shows that interconnection, measured as the harmonic distance between each bank and the network is positively related to contagion. Greater distance between a given bank and the network and therefore lower interconnection is associated with lower contagion.

The intuition is straightforward. Banks with weaker ties to their peers are less influential in terms of the spillover effect they exert on the system. However, it is important to note that this result only holds on average. For a given level of interconnection, one may observe very different levels of contagion. For instance, in panel A of figure 6 it is possible to observe that for an harmonic distance of 1000, there are institutions with levels of contagion lower than 1. Whereas spillovers from other institutions at the same level of harmonic distance are greater than 4. Overall, high levels of interconnection do not necessarily imply high contagion. For the majority of replicas where interconnection is high, contagion is moderate. This is consistent with the view that high interconnection is a risk factor that contributes to the vulnerability of the financial system to contagion, as opposed to the idea that interconnection is a necessary and sufficient condition for contagion.

Figure 6: Simulation results - Relationship between interconnection and contagion.



(a) Low clustering (b) High clustering

Notes: $\tilde{W}$ is drawn from a lognormal distribution. Replicas from a Gaussian copula parametrized with $\tau = 0.1$ and $\tau = 0.9$ represent low and high degrees of clustering, respectively. H(k) stands for the harmonic distance between institution k and the network. Figures zoom in the 90th percentile of contagion.

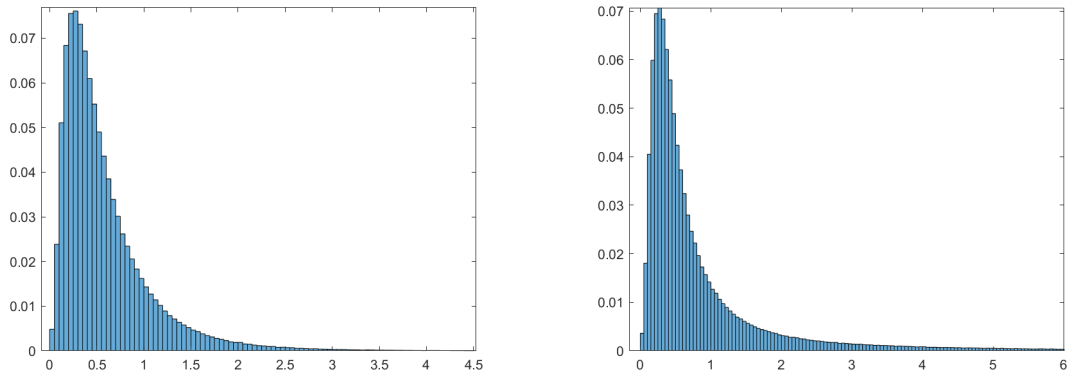
The result holds for different choices regarding the level of clustering considered. However, it can be seen that the negative relationship between harmonic distance and contagion is more pronounced in financial networks characterized by high levels of clustering. We discuss how clustering may foster contagion next.

H2. *A less clustered financial network is less prone to contagion.*

The degree of clustering in a financial network refers to the level of commonality of bank balance sheets or the extent to which banks, taken together, are exposed to common risks (see Allen et al. (2010a)). Simulation results reported in Figure 6 suggest that a high level of clustering promote greater levels of density in the financial network, leading to greater contagion and the more frequent observation of extreme values. This can also be confirmed in Figure 7 which shows that, although the average contagion level is lower, the distribution of the magnitude of contagion is fat tailed for high clustered networks. Whereas, low clustered networks feature less extreme contagion in the tail but average contagion is higher.

The intuition behind these results is that, given a high level of commonality of bank returns, the arrival of bad news is priced-in by markets, penalizing all banks simultaneously and thereby amplifying the shock to the system. In this state of nature the distribution of the magnitude of contagion will be fat tailed compared to a scenario of low clustering.

Figure 7: Simulation results - Distribution of the magnitude of contagion



(a) Low clustering (b) High clustering

Notes: Histograms describe the distribution of the magnitude of contagion for the set of financial networks $\tilde{W}$ draw with $\rho = 0.1$ and $\rho = 0.9$, representing low and high degrees of clustering, respectively.

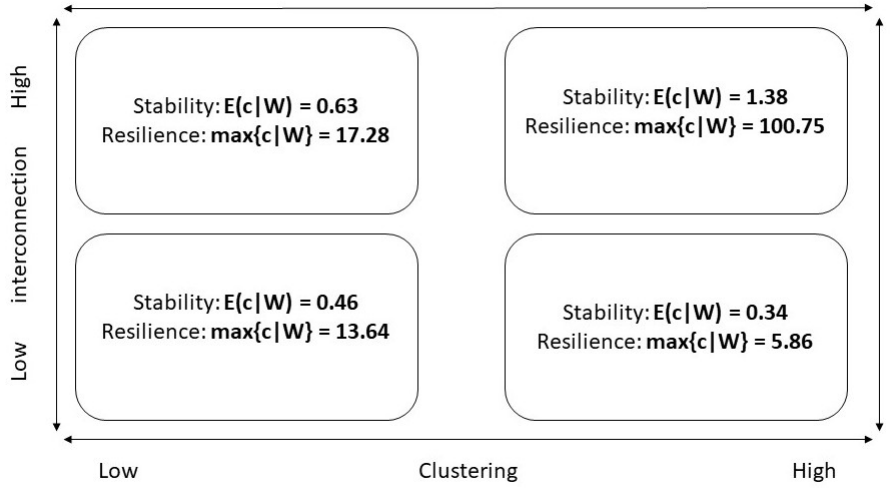
H3. *A more diversified financial network benefits financial stability.*

To study financial stability implications of the level of interconnection and clustering in the system, we assess four different types of financial networks based on the criteria set forth in Definition 5, proposed in section 3. The main results are summarized in Figure 8. The diagram shows the two criteria of financial stability of a network as a function of interconnection and clustering levels. In interpreting these results, it is worth highlighting that, in accordance with Definition 5, greater levels of $E(c|W))$ and $max\{c|W\}$ signify lower states of stability and resilience, respectively. This is because $E(c|W)$ measures the expected value of the magnitude of contagion and $max\{c|W\}$ the worst-case contagion level. Hence, the greater these quantities, the lower financial stability.

Stability is given by the expected value of contagion for each draw, averaged across draws, while resilience is given by the maximum value of contagion in the system for a set of draws. Hence, the results shown below summarize the average value of stability and resilience across simulations. We see that the optimal financial network, for which stability and resilience are greater is one in which interconnection is low and clustering is high. However, the set of financial networks exhibiting high clustering are only optimal from a financial stability viewpoint if interconnection is low at all times. If interconnection is high in a highly clustered network, the financial system is not resilient, and exposed to powerful low frequency, high severity events. In particular, high clustering and high interconnection, when combined, produce contagion episodes, with a magnitude that largely exceeds those observed in all

other states studied in this paper.

Figure 8: Box matrix chart showing contagion levels for each state of diversification and clustering



Notes: Low and high levels of interconnection correspond to the first and last quartile of the simulation results with respect to the harmonic distance of each observation. Whereas, low and high levels of clustering correspond to the simulations obtained by setting the copula parameter ρ to 0.1 and 0.9, respectively.

As a second best, financial networks that exhibit low interconnection and low clustering show intermediate levels of contagion and resilience. Whereas, the least stable and resilient networks are those in which both interconnection and clustering are high.

5 Conclusion

This paper advocates that there is a spatial element to financial contagion that should be taken into account when examining spillover of financial distress in the banking system. We examine the evidence of contagion within the banking system by studying a panel of default probabilities for a large number of US banks observed from 1990 to 2018. The panel structure of the data allow us to explore the dynamics of contagion across time, shedding light on the importance of contagion to the build up of financial distress. It also offers the possibility of looking into the cross-sectional heterogeneities within the banking system. Contagion is captured by a spatial econometric model that is estimated through Bayesian techniques. Several reasons make these models particularly insightful to study contagion. First, and most importantly, they allow for feedback effects between default likelihoods amongst banks, providing an analytical framework consistent with the definition of contagion put forward in the literature. Second, they handle panel data thus offering the possibility of purging unobserved bank specific and macrofinancial shocks that hit the economy together with contagion and yet are independent of it, mitigating omitted variable bias. Third, Bayesian techniques permit adequate modelling of stochastic volatility, a major hurdle in the empirical literature in measuring contagion and to correct for the underlying bias.

This framework is used to empirically test three important prepositions about financial networks. Through a simulation exercise, we find that interconnection positively affects contagion risk and that clustering leads to a fatter tailed distribution of the magnitude of contagion. Financial networks where both clustering is high and interconnection is low exhibit the highest levels of resilience and stability. Notwithstanding, the interaction of high interconnection and high clustering in a financial network exposed the system to low frequency, high severity events such as the ones felt in the wake of the crisis. One of the most impressive features of the recent financial crisis was the powerful worldwide spillover induced by the US subprime mortgage crisis. We have shown that such events can be captured by economic models provided that heavy tails and the joint behavior of distress are taken into account.

The main contribution of this paper is to propose an alternative framework to study contagion that deals with the major analytical challenges identified in the literature. Results suggest contagion substantially contributes to the build up of distress in the banking system, accounting for a statistically powerful and economically meaningful portion of default probabilities of banks. The significant heterogeneity with respect to institution level spillovers suggests that some institutions

are systemically more relevant that other and such importance is not proportional to size.

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A Estimation Methods

A.1 Estimating Banks Probabilities of Default

To understand the importance of contagion it is necessary to establish a benchmark that can serve the purpose of reflecting the level of stress in the financial system at each point in time.

I adopt the methodology proposed by Merton (1974)[9] for assessing the default probability of an entity. Robert Merton proposed a structural credit risk model that models a firm's equity as a call option on its assets. This method allows for a firm's equity to be valued with the option pricing formulae of Black and Scholes (1973). From an accounting viewpoint, the book value of a firm's assets is forcedly equal to the value of its equity plus liabilities.

$$A = E + L \tag{30}$$

Although the book value of assets and liabilities are observable, as they are reported periodically by firms, their market values are not. One only observes market prices that reflect a firms equity at a high frequency basis. There are no market value for assets and liabilities. Merton uses the Black and Scholes option pricing formulae to relate the market value of equity to assets and liabilities in a common framework to estimate the market value and volatility of a firm's assets. Under the assumption that the value of liabilities is fixed a priori for a given horizon T, a firm's total equity value can be regarded as the payoff of a call option given below

$$E_t = max\{0, A_t - L\} \tag{31}$$

Note that the firm defaults when the market value of assets falls below a non stochastic default threshold defined by the value of the firm's liabilities at a given horizon. One obvious shortcoming of this approach is that it ignores the structure and maturity liabilities. To address this issue this paper follows the rule of thumb and estimates this input by assuming that total liabilities equal total short-term liabilities plus one half of long term liabilities. The value of L is sometimes referred to as the default threshold.

[9]Some popular alternatives in the literature include Altman (1968) Z-Score, that has been applied to banks by Cihak et al. (2012) and is published regularly by the World Bank. In a different context, Jiménez et al. (2013) uses the log odds ratio of a bank's NPL ratio. However, this measure only captures credit risk. Another approach adopted by Giglio (2011) and others consists in gauging the default likelihood of large institutions implicit in Bond yields and CDS instruments.

Hence, the value of equity may be written, for a given horizon T, as a function of assets A, liabilities L and a risk free interest rate r as follows

$$E = AN(d_1) - Le^{-rT}N(d_2), \tag{32}$$

where

$$d_1 = \frac{ln(A/L) + (r + 0.5\sigma_A^2)T}{\sigma_A\sqrt{T}} \text{ and } d_2 = d_1 - \sigma_A\sqrt{T}. \tag{33}$$

This expression results from a straightforward application of the Black and Scholes formulae. It assumes that assets follow a Geometric Brownian Motion described by the stochastic differential equation below

$$\frac{dA}{A} = \mu_A dt + \sigma_A \varepsilon \sqrt{t} \tag{34}$$

where μ stands for asset return, σ_A is equal to the standard deviation of the asset return, and ε is a random variable following a standard normal distribution. The probability of default arises from the likelihood that the value of the assets falls below the default threshold, given by the value of debt payments L_t, at a given time horizon. Formally, the likelihood of default occurrence is given by

$$P(A_t < L) \tag{35}$$

Thus, uncertainty associated to the value of the assets relative to promised payments is what drives defaults. In other words, as noted by Gray et al. (2007) "*Balance sheet risk is the key to understanding credit risk and crisis probabilities* ". Plugging into Equation 35 Itô's general solution for the Stochastic Differential Equations written in equation 34 one gets

$$P(A_0 exp\{(\mu_A - \frac{\sigma_A}{2})t + \sigma_A\varepsilon\sqrt{t}\} \leq L) = P(\varepsilon \ \leq -d_1). \tag{36}$$

Thus, the probability of default of each institution is found simply by evaluating

$$PD = 1 - N(d_1), \tag{37}$$

Where $N(.)$ is the cumulative Normal distribution function and d_1 is commonly referred to as distance to default.

This approach is superior to other alternatives in two important ways. First, it is broadly applicable to any institution, provided that its market price is a reliable

signal of intrinsic value. Thus, a larger number of institutions can be considered without having to restrict our sample to those that issue CDS. Second, by relying on market variables, the measure of stress obtained reflects all available information on a given entity, rather than over-relying on balance sheet data solely that is not accurate in producing real time signals of financial stress. Adopting this approach relies however on the working assumption that the markets are efficient and thus prices reflect all available and relevant information of a given entity.

Like any other enterprise, banks fund their assets by resorting to debt or issuing equity. Although the market value of a bank's assets is an important measure of its financial health, this quantity is not observable. The merit of the Merton model [10] consists in estimating the market value of assets of a firm, therefore inferring how far each firm is from default.

[10] Also known as Moody's KMV model due to its widespread use in developing ratings of financial securities (see Gupton et al. (2007) for more details.)

A.2 A brief introduction to Copulas

A copula is a mathematical object that allows us to model the dependency between two or more random variables. In this section we focus only on copula theory that is necessary for this article [11].

Consider two random variables, X and Y denoting the distance between bank i and bank j for any two pair of banks $\{i, j\}$ with $i \neq j$. To specify the joint distribution of X and Y (ie, to define $F(X, Y) = P[X \leq x, Y \leq y]$) it is necessary to know the individual distribution functions $F(X)$ and $F(Y)$ but also the dependency structure between X and Y that describes how both variables behave jointly. This last element is characterized with a copula. In a more formal way a copula is defined as follows

DEFINITION B.1 A d-dimensional copula is a mathematical object mapping $C : [0, 1]^d \to [0, 1]$ through a determined density function.

Exactly how the joint distribution of X and Y is formed from a copula and their marginals is determined by Sklar's theorem, written below for the bivariate case.

Theorem B.1 Let C be a copula and $\{F(X), F(Y)\}$ be marginal distribution functions of X and Y. Then, function H denoting the joint distribution function of X and Y is given by

$$H(X, Y) = C(F(X), F(Y)), \forall X, Y \in \Re^2 \tag{38}$$

The theorem can easily be generalized for d random variables, in which case the copula has d-dimensions[12]. The simplest example of a Copula satisfying Definition B.1 is the independence Copula, according to which $C^I(u_1, u_2) = u_1 u_2$. Notice that the independence Copula simply prescribes that the joint distribution is the product of the marginals.

There are a variety of Copulas to choose from. The suitability of each different copula is dependent by the application at hand. We wish to model the pairwise distances between all banks in our sample. Thus, we will only consider a set of copulas that can be generalized to a multi-dimensional case. We look more closely at two copulas in particular: the Gaussian copula and the Gumbel copula. Our discussion is restricted to the bivariate case d=2. However, both the Gaussian and the Gumbel copulas allow for higher dimensions.

[11] It is out of the scope of this paper to provide a thorough review of the extensive literature on the topic. The interested reader is encouraged to see reference manuscripts. We found Nelsen (2005); McNeil et al. (2005) and Embrechts (2009) very insightful.

[12] For a proof of the theorem see McNeil et al. (2005).

Let us first consider the Gaussian copula C_p^{gauss}, a member of a general class of elliptical copulas. It does not have a closed form expression but it can be defined as an integral as follows

$$C_p^{gauss}(X,Y) = \int_{-\inf}^{\Phi^{-1}(X)} \int_{-\inf}^{\Phi^{-1}(y)} \frac{1}{2\pi(1-p^2)^{1/2}} exp\left\{ -\frac{s^2 - 2pst + t^2}{2(1-p^2)} \right\} \tag{39}$$

For all X,Y $\in$ [0,1], $|p| \leq 1$, where $\Phi(.)$ denotes the univariate standard normal distribution function. The parameter p determines the level of dependency in the Gaussian copula as can be seen in the Figure below [13].

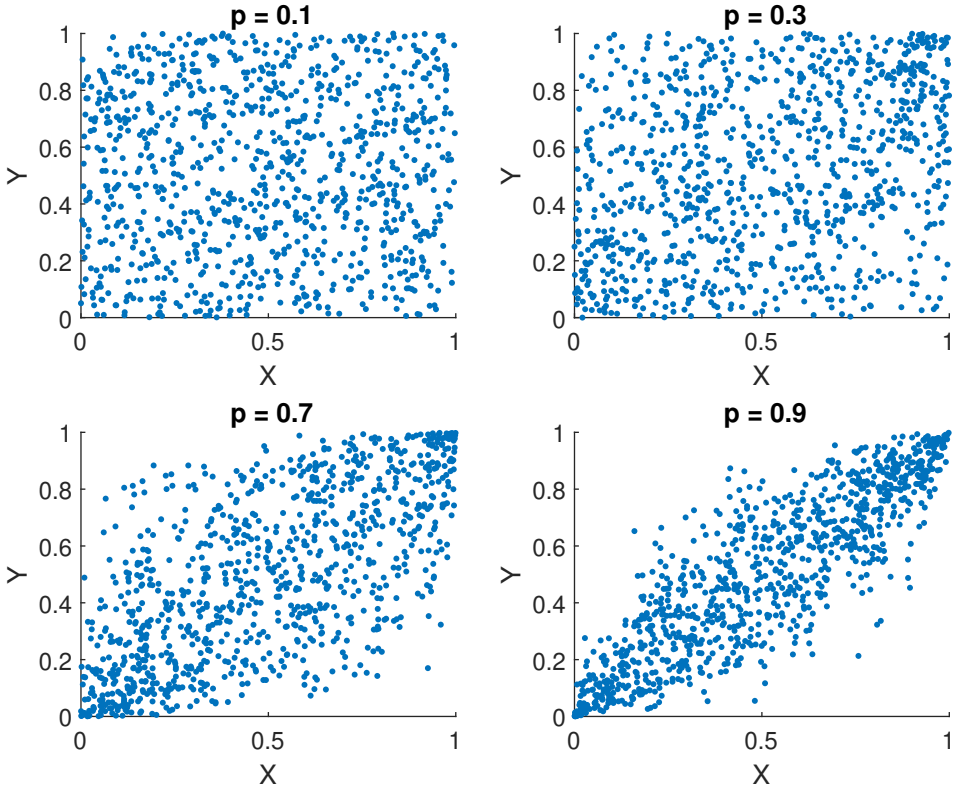


Figure 9: 1000 draws from a random vector (X,Y) generated with a Gaussian copula with marginals which are uniformly distributed between 0 and 1 and different levels of dependency p.

Setting $p = 0$, for instance, makes the marginal distributions independent and thus, the Gaussian copula breaks down into a simple independence copula. Whereas, high levels of dependence, for instance $p = 0.9$ result in high clustering of the generated random variables.

[13] In this paper ρ is used to denote the spatial autoregressive coefficient in the SAR model. Therefore, to avoid confusion we use p to denote linear correlation coefficient.

The Gaussian copula became popular in the aftermath of the great financial crisis. It came to the attention of the general public and was dubbed as *"the formula that killed Wall-Street"* in the web-article of Salmon (2008) due to its misuse in pricing derivatives by investment banks. Its popularity owes to its simplicity and straightforward implementation. It is calibrated with one single parameter p that describes the level of association between two random variables.

The second copula we consider is the Gumbel copula C_θ^{gum}, written below in its bivariate form

$$C_\theta^{gum}(X,Y) = exp\Bigg\{ -\Big((-lnX)^\theta + (-lnY)^\theta\Big)^{1/\theta}\Bigg\}, \quad 1 \leq \theta < \inf, \quad \forall X, Y \in [0,1]. \tag{40}$$

The parameter θ is as a measure of association between X and Y. Just as p ,the linear correlation coefficient is used to parametrize the Gaussian copula, θ is associated to the Kendall's rank correlation, albeit in a less trivial form. Define Kendall's rank correlation between two random variables X and Y as

$$\tau(X,Y) = P[(X-\tilde{X})(Y-\tilde{Y}) > 0] - P[(X-\tilde{X})(Y-\tilde{Y}) < 0]. \tag{41}$$

Where the pair $(\tilde{X}, \tilde{Y})$ denote two random variables independent of (X,Y) but with the same distribution function. This measure indicates the likelihood of X and Y moving in tandem. The Kendall's tau is related to the parameter of the Gumbel copula in the following way

$$\tau^{gum}(X,Y) = 1 - \frac{1}{\theta} \tag{42}$$

Moreover, a relationship between Kendal's tau and the spearman correlation coefficient is suggested by Fang et al. (2002) allowing us to generate a set of random numbers from a gumbel copula parametrized from a single correlation coefficient[14].

[14]Theorem 3.2 in Fang et al. (2002) suggests that $\tau = \frac{2}{\pi} arcsin(p)$

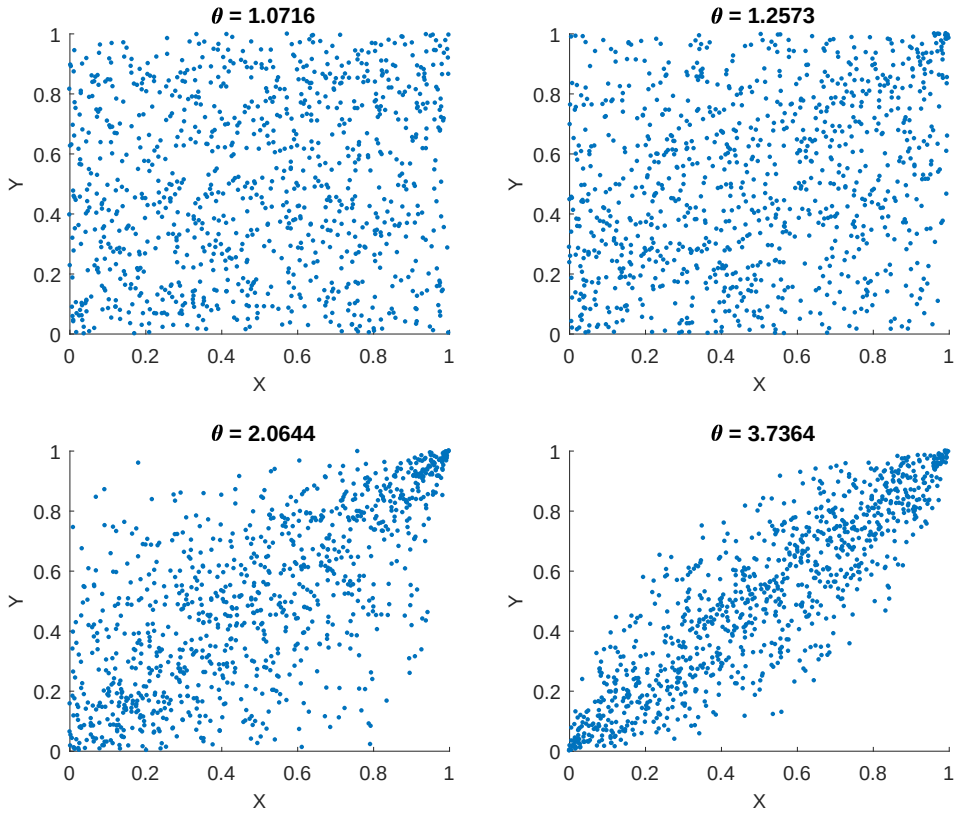


Figure 10: 1000 draws random vector (X,Y) generated with a gumbel copula with marginals which are uniformly distributed between 0 and 1 and different levels of dependency θ.

Figure 10 exhibits four sets of random numbers generated with a gumbel copula that is parametrized with four different values for θ that are calculated as counterparts of the four levels of p plotted in Figure 9, as suggested by Fang et al. (2002). The gumbel copula belongs to a set of Extreme Value copulas, designed for the purpose of capturing extreme events.

A.3 Simulating the impact of the Financial Network's structure on Financial Stability

A Monte Carlo experiment is set-up as follows:

1 Generate a sequence of dependent uniform random numbers u between 0 and 1 using a copula with a given tail configuration [15].

2 Through the inverse transform method, convert the dependent uniform random matrices into (artificial) spatial weight matrices using an eligible distribution function [16]

$$\tilde{W} \sim F(\bar{W}, V_W), \tag{43}$$

with mean $\bar{W}$ and standard deviation V_W, which were obtained by running the Bayesian VAR in 7. $\bar{\Sigma}$ and V_Σ, which are used to compute the mean and standard deviation of W are the mean and the standard deviation of the posterior of the Variance-Covariance matrix of the VAR describing equity price returns. The relationship between both quantities is defined in Definition 2.

3 Estimate the model below

$$y = \rho\tilde{W}y + X\beta + \varepsilon. \tag{44}$$

4 compute the measures
i) contagion: $c(\tilde{W}) = \sum_{i\neq k}^{N}(I - \rho W)^{-1}$ and
ii) interconnection: $H(\tilde{W}) = \sum_{i\neq j}\frac{1}{w_{ij}}$. defined above.

5 Repeat [1-3] a large number of times and store the most relevant quantities.

[15] See Appendix B for more details on Copulas and the two alternative specifications considered in this exercise.

[16] Any distribution function defined in $\mathbb{R}_0^+$ is licit since that is the interval for which distances make sense. We use a lognormal distribution.

B Additional Tables and Figures

B.1 Data Description

code	Variable	Description	Source
MV	Market Value	Share price multiplied by the number of ordinary shares in issue in millions of dollars.	Eikon/Datastream
MTBV (Worldscope item 03501)	Market-to-Book Value	Defined as the market value of the ordinary equity divided by the balance sheet value of the ordinary equity in the company.	Eikon/Datastream
EPS	Earnings Per Share (EPS)	Latest annualised rate that may reflect the last financial year or be derived from an aggregation of interim period earnings.	Eikon/Datastream
WC08301	Return on Equity (ROE)	Latest annualised rate that may reflect the last financial year or be derived from an aggregation of interim period earnings.	Eikon/Datastream
WC08301	Total Debt % common equity	Leverage Ratio, Annual & Interim Item; Field 08231	Eikon/Datastream
WC15061	Non Performing Loans	Non-Performing Loans / Loans-Total * 100 (Other Ratio, Annual Item; Field 15061)	Eikon/Datastream
LTD	Loan to Deposit Ratio	Loan/Deposits ratio	Eikon/Datastream
WC08231	Leverage	Total Debt % common equity, Leverage Ratio, Annual & Interim Item; Field 08231	Eikon/Datastream
WC03255	Total Debt	Represents all interest bearing and capitalized lease obligations. It is the sum of long and short term debt.	Worldscope
WC03251	Short term debt	Represents all interest bearing financial obligations, excluding amounts due within one year. It is shown net of premium or discount.	Worldscope

Table 1: Variable description and data sources.

	Prob. of Default			Leverage			EPS			Market-to-Book value			Return on Equity			NPL			Loan-to-Deposit ratio			Market Value		
Year	1Q	Mean	3Q	1Q	Mean	3Q	1Q	Mean	3Q	1Q	Mean	3Q	1Q	Mean	3Q	1Q	Mean	3Q	1Q	Mean	3Q	1Q	Mean	3Q
1990	0.00	4.39	0.85	89.32	206.47	233.33	0.28	0.94	1.35	0.55	0.95	1.14	11.46	10.57	16.08	1.17	2.43	2.34	73.47	78.41	87.33	205.11	857.16	1043.70
1991	0.00	3.62	1.06	101.96	179.83	205.27	0.41	0.86	1.02	0.60	1.00	1.20	7.99	9.83	15.27	1.41	2.40	3.08	71.74	75.84	82.85	253.72	1138.04	1660.64
1992	0.00	5.39	4.25	93.56	179.03	196.51	0.40	0.95	1.31	0.89	1.25	1.45	12.62	13.28	17.23	0.98	2.25	2.23	66.59	75.29	86.10	282.10	2029.65	2803.48
1993	0.00	2.36	0.55	81.78	215.02	318.32	0.58	1.23	1.45	1.06	1.39	1.66	14.86	15.94	18.41	0.70	1.36	1.38	73.68	79.98	91.16	372.64	2507.93	3418.32
1994	0.00	0.62	0.09	102.24	236.77	297.71	0.63	1.38	1.51	1.04	1.36	1.63	13.35	16.01	18.75	0.51	0.94	1.13	76.02	85.77	99.51	390.36	2597.00	3280.64
1995	0.00	0.29	0.02	90.12	218.28	331.31	0.64	1.35	1.63	1.11	1.36	1.58	13.95	15.91	18.91	0.51	0.91	1.22	76.15	88.38	100.89	486.69	3085.53	3712.97
1996	0.00	0.35	0.01	71.08	211.11	333.41	0.72	1.49	1.63	1.40	1.76	2.07	13.11	15.77	17.50	0.44	0.77	0.88	79.24	90.19	104.25	601.29	4781.19	5549.37
1997	0.00	0.02	0.02	98.43	255.17	372.19	0.80	2.06	2.15	1.79	2.37	2.82	13.93	16.44	18.42	0.47	0.70	0.76	84.68	92.31	103.26	938.64	8681.57	10094.83
1998	0.01	1.85	0.63	91.81	230.33	322.54	0.89	2.25	2.14	1.86	2.60	3.06	14.32	15.79	17.57	0.38	0.55	0.69	77.66	91.51	99.34	976.63	11611.29	12385.32
1999	0.03	1.10	1.12	128.03	273.55	374.12	0.78	2.35	2.29	1.72	2.57	2.97	15.10	17.15	19.86	0.45	0.61	0.66	82.53	95.69	106.23	691.69	16890.25	14134.69
2000	0.03	0.71	1.13	121.12	231.69	317.38	0.86	2.74	2.57	1.28	1.82	2.22	12.84	15.61	18.87	0.39	0.65	0.79	83.93	95.65	104.14	513.87	16275.50	10151.82
2001	0.02	1.42	0.72	102.99	207.59	287.39	0.84	2.74	2.74	1.58	2.04	2.31	10.53	13.42	16.50	0.59	1.00	1.00	75.75	88.59	101.14	620.15	18030.32	10416.35
2002	0.00	0.70	0.46	114.57	204.81	271.62	0.86	2.76	2.64	1.60	1.99	2.29	12.57	14.78	18.55	0.52	1.01	1.00	79.13	87.52	100.19	750.02	16565.14	10357.42
2003	0.01	0.30	0.29	94.74	210.95	282.13	1.16	3.15	3.42	1.63	2.02	2.32	12.99	14.76	17.74	0.39	0.77	0.88	79.46	88.70	100.13	879.79	17449.40	10624.89
2004	0.00	0.46	0.00	102.55	203.86	279.78	1.26	3.45	3.69	1.79	2.21	2.65	12.40	14.80	17.49	0.31	0.54	0.61	81.95	91.62	103.16	1039.34	21259.51	11586.29
2005	0.00	0.07	0.00	110.57	192.78	256.96	1.62	3.80	3.99	1.62	2.09	2.53	11.51	14.39	16.88	0.25	0.48	0.56	79.98	91.35	103.12	1172.12	22473.71	13907.74
2006	0.00	0.03	0.00	100.16	182.16	212.54	1.62	3.96	3.66	1.62	2.00	2.24	10.24	13.70	17.02	0.18	0.46	0.57	85.04	95.28	104.08	1373.22	23288.93	13898.60
2007	0.00	0.20	0.08	126.14	223.31	284.33	1.55	4.08	4.41	1.36	1.79	2.09	6.29	9.87	13.64	0.44	0.83	1.06	90.34	100.45	110.98	1389.61	23597.62	12469.02
2008	2.15	8.73	12.76	130.32	245.02	325.37	0.92	2.25	3.41	0.97	1.38	1.77	-6.57	-3.63	8.21	0.81	1.80	2.36	89.14	100.88	112.05	1058.93	15524.84	8616.31
2009	8.93	29.93	51.38	89.40	163.20	214.98	0.04	1.05	1.56	0.53	0.92	1.20	-6.39	-1.97	8.46	1.74	3.39	4.17	80.66	89.35	95.30	795.80	12069.86	5684.79
2010	0.14	3.17	4.00	60.23	136.62	170.15	0.00	1.52	1.83	0.87	1.19	1.44	0.34	4.12	9.89	1.63	2.80	3.61	76.44	85.99	95.06	1408.28	17714.31	8712.51
2011	0.08	1.07	0.98	63.15	120.05	145.65	0.33	1.83	2.30	0.83	1.11	1.31	3.87	7.63	10.76	1.07	2.38	3.06	77.42	84.97	94.53	1570.22	16198.74	7366.97
2012	0.03	0.61	0.60	51.06	107.51	124.65	0.73	2.19	2.87	0.83	1.12	1.40	6.12	9.62	11.94	0.96	1.92	2.31	74.58	83.68	92.10	1672.47	16438.55	8639.48
2013	0.00	0.00	0.00	49.22	104.87	137.80	1.01	2.62	3.42	0.98	1.32	1.66	6.57	9.07	11.59	0.78	1.82	2.57	78.63	85.45	91.99	2261.92	21983.21	11001.22
2014	0.00	1.25	0.00	48.04	103.09	139.81	1.07	2.68	3.81	1.07	1.39	1.60	6.45	9.00	10.85	0.54	1.60	2.16	78.79	86.43	94.00	2854.47	25579.61	11907.34
2015	0.00	0.84	0.01	45.22	101.07	138.61	1.38	2.88	3.33	1.02	1.39	1.65	6.61	9.13	11.26	0.51	1.39	1.93	79.29	87.27	95.12	3402.46	27174.74	13000.56
2016	0.00	0.09	0.09	43.12	100.04	135.68	1.47	3.20	3.91	0.99	1.29	1.53	6.74	8.92	10.75	0.55	1.37	1.84	76.54	87.05	94.18	3479.07	25860.98	12259.93
2017	0.00	0.04	0.00	56.82	101.96	146.38	1.62	2.89	3.81	1.28	1.72	2.12	6.69	9.08	11.53	0.58	1.41	1.86	76.56	85.37	91.24	4496.03	41405.75	19701.06

Table 2: **Descriptive Statistics.** Mean, first and third quartiles of the distribution of each variable used, for each year for the full sample considered - 1990-2018. EPS and NPL abbreviate Earnings per Share and Non-Performing Loans, respectively.

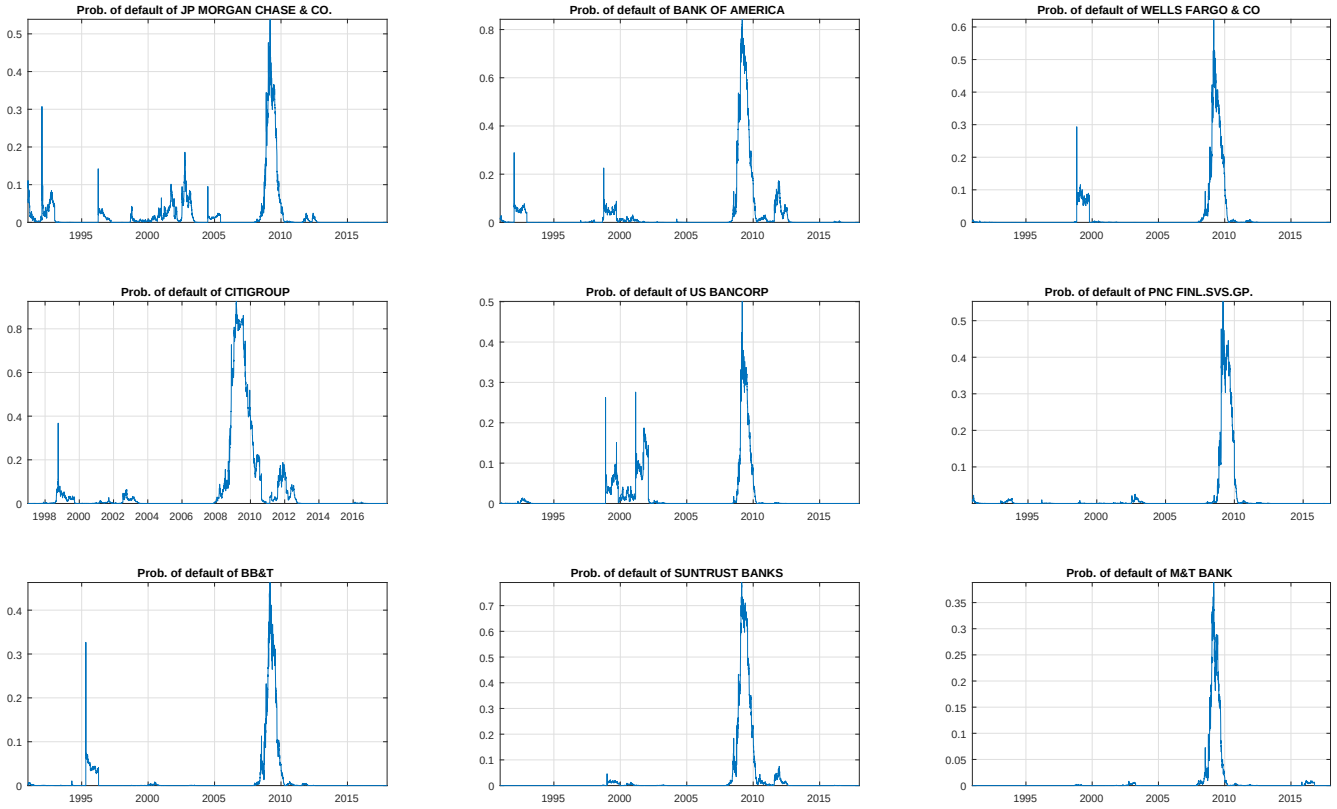


Figure 11: Time series Probability of Defaults of the 9 largest US banks - measured by Market Value - as of January 2018.

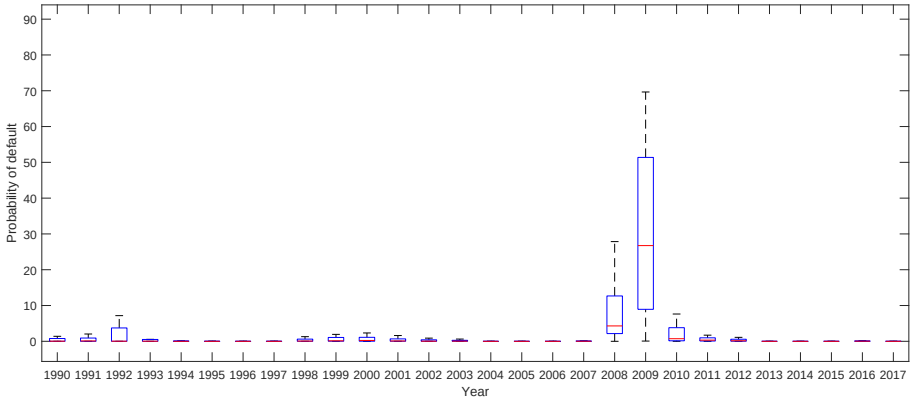


Figure 12: Box Plot of the distribution of Probabilities of default across banks for each year in the sample considered.

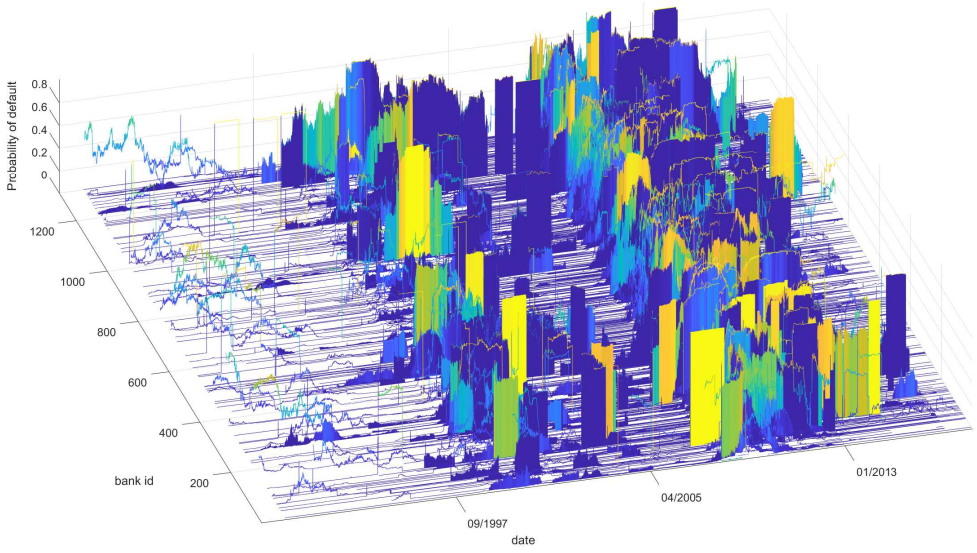
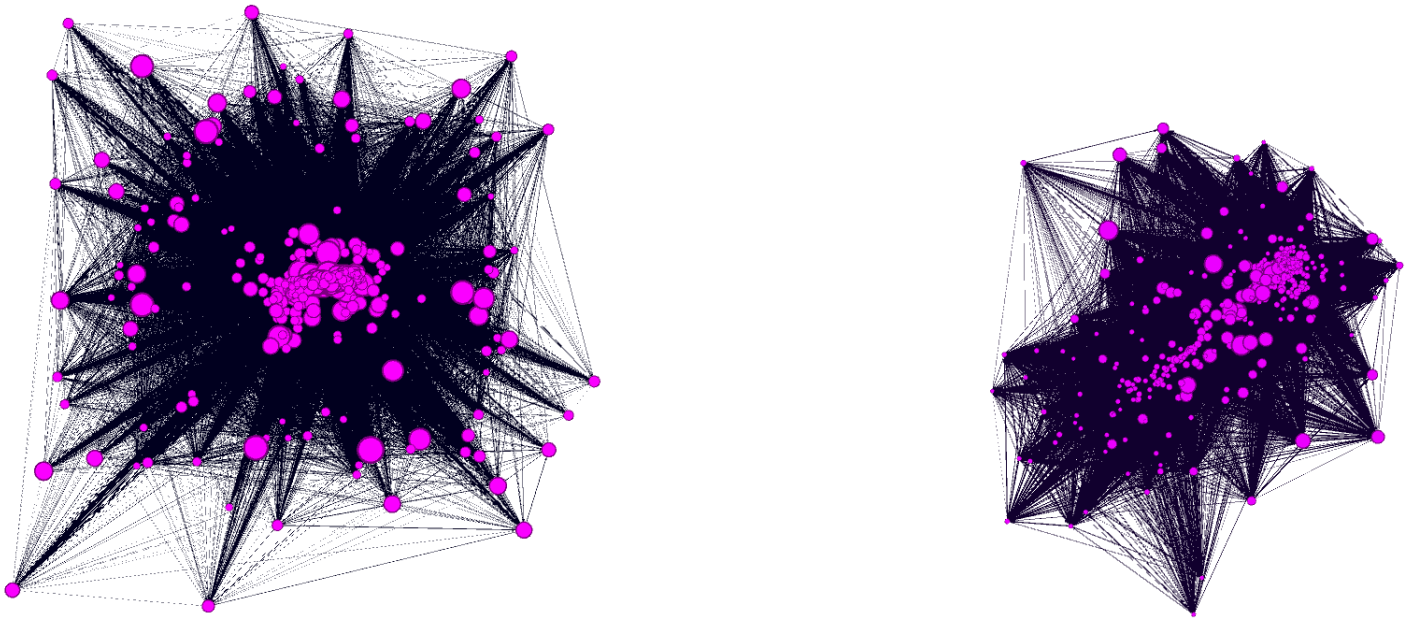


Figure 13: Joint behaviour of default likelihoods of all US banks included in the sample from 1990 until 2018. Periods of significant probability of default ($\geq$75%) are highlighted in yellow.



(a) US banking system network in 2006

(b) US banking system network in 2009

Figure 14: Financial Network depicting the relationship between banks in the US before and after the crisis. The length of the edges indicate the strength of the relation, while the size of the nodes show the strength of connections of each institution with the system. Average covariance went down from 0.5764 to 0.3682.

Determinants	I		II		III		IV	
	POLS	**t-stat.**	**SAR (ML)**	**t-stat.**	**BSAR (Homosk)**	**t-stat.**	**BSAR (Heterosk)**	**t-stat.**
Leverage	0	0	-0.0525	-0.25	-0.0948	-0.33	0.084	0.70
Earnings per Share	-0.07	-1.11	-0.062	-0.99	-0.1113	-1.2	-0.035	-0.7
Market-to-Book Value	-0.1644	-0.45	-0.0157	-0.04	-0.1579	-0.35	0.245	1.137
Return on Equity	-0.3269	-11.48	-0.305	-11.16	-0.3175	-10.69	-0.253	-7.28
Non-performing Loans % total loans (NPL)	0.5086	21.1	0.6349	4.06	0.625	3.428	0.281	1.96
Loan to Deposit ratio	0.0041	0.32	0.02	1.81	0.0217	1.08	0.007	0.80
size (market value)	0.0045	0.8	0.0037	0.67	0.0036	0.420	-0.002	-0.75
ρ			0.472	73.87	0.3856	3.67	0.545	4.03
Sigma squared	41.54		39.75		40.58		47.49	
Moran I's statistic			35.25					
R-squared	0.524		0.520		0.556		0.481	
Year FE	Y		Y		Y		Y	
Bank FE	N		N		Y		Y	
No. obs	1045		1045		1045		1045	

Table 3: **Drivers of US bank default likelihood.** Maximum Likelihood(ML) and Bayesian estimates of different specifications of the Spatial Autoregressive (SAR) models of interest. For the sake of model comparison and contrary to Bayesian convention, t-statistics are calculated from the posterior mean and standard deviation of the sampled MCMC draws for the parameters.

Bank id.	PD	ρWy	Spillover
JP MORGAN CHASE & CO.	2.19	1.320	5.931
WELLS FARGO & CO	1.67	1.385	7.281
BANK OF AMERICA	3.10	1.385	11.482
CITIGROUP	5.54	1.350	17.896
US BANCORP	1.58	1.345	5.553
PNC FINL.SVS.GP.	1.48	1.397	7.580
BB&T	1.25	1.346	5.393
SUNTRUST BANKS	2.45	1.407	11.585
M&T BANK	0.82	1.355	4.088
KEYCORP	3.35	1.381	13.541
FIFTH THIRD BANCORP	3.16	1.389	14.647
CITIZENS FINANCIAL GROUP	0.03	0.118	0.055
REGIONS FINL.NEW	4.50	1.418	15.492
CREDICORP	0.54	1.430	1.086
FIRST REPUBLIC BANK	0.03	0.249	0.053
HUNTINGTON BCSH.	3.82	1.387	16.240
COMERICA	1.62	1.353	6.819
SVB FINANCIAL GROUP	2.74	1.449	7.627
ZIONS BANCORP.	2.88	1.406	12.967
EAST WEST BANCORP	3.55	1.509	12.078
SIGNATURE BANK	0.93	1.646	2.931
NEW YORK COMMUNITY BANC.	1.19	1.144	3.619
PEOPLES UNITED FINANCIAL	4.23	1.121	19.558
BANK OF THE OZARKS	0.62	1.351	1.929
PACWEST BANCORP	3.97	1.601	10.929
CULLEN FO.BANKERS	0.61	1.261	2.049
COMMERCE BCSH.	0.28	1.223	1.600
BOK FINL.	2.60	1.199	6.026
HUDSON CITY BANC. DEAD - DELIST.02/11/15	1.42	1.513	3.306
WESTERN ALL.BANCORP.	8.12	1.933	19.620
SYNOVUS FINANCIAL	4.69	1.344	15.909
CITY NATIONAL DEAD - DELIST.02/11/15	1.26	1.461	4.247
WEBSTER FINANCIAL	2.23	1.384	9.477
PRIVATEBANCORP DEAD - DELIST.23/06/17	3.08	1.591	8.319
PROSPERITY BCSH.	0.70	1.324	2.455
TFS FINANCIAL	0.03	2.081	0.056
FIRST HORIZON NATIONAL	2.15	1.323	8.307
INVESTORS BANCORP	6.54	1.850	16.690
FNB	7.09	1.210	20.763
PINNACLE FINANCIAL PTNS.	3.32	1.468	7.399
UMPQUA HOLDINGS	6.04	1.332	15.092
WINTRUST FINANCIAL	1.83	1.389	7.164
POPULAR	5.95	1.287	18.631
IBERIABANK	0.38	1.151	1.107
TEXAS CAPITAL BANCSHARES	1.05	1.577	3.309
FIRST CTZN.BCSH.A	0.40	1.344	1.336
HANCOCK HOLDING	1.63	1.219	4.791
BANKUNITED	0.02	0.238	0.031

Table 4: **System-wide Direct and Spillover effect of a shock to the 50 largest banks probability of default.** Where $\rho * y$ measure the dependence between each bank and the banking system and the Direct/Indirect/Total effects are estimated performing the calculation of $(I - \rho W)^{-1}$ (see methodology section)

	BSAR FE	95% credibility set	
Direct effect			
Leverage	0.09	-0.230	0.406
Earnings per Share	-0.04	-0.182	0.074
Market to Book value	0.25	-0.355	0.858
Return on Equity	-0.26***	-0.359	-0.174
NPL	0.29*	-0.095	0.774
Loan to Deposit ratio	0.01	-0.015	0.034
size (market value)	0	-0.012	0.008
Indirect effect			
Leverage	0.12	-0.185	0.561
Earnings per Share	-0.05	-0.242	0.058
Market to Book value	0.31	-0.249	1.097
Return on Equity	-0.34**	-0.746	-0.060
NPL	0.36	0.002	0.985
Loan to Deposit ratio	0.01	-0.013	0.043
size (market value)	0	-0.016	0.006
Total effect			
Leverage	0.21	-0.537	1.123
Earnings per Share	-0.09	-0.542	0.173
Market to Book value	0.56	-0.838	2.223
Return on Equity	-0.6***	-1.258	-0.259
NPL	0.65*	-0.205	1.880
Loan to Deposit ratio	0.02	-0.037	0.091
size (market value)	-0.01	-0.033	0.018

Table 5: **Direct and Spillover effects of the different covariates included in the model.** Figures show the variation of the probability of default in basis points, given an increase in 1 unit of each covariate taken individually. Once again, p-values are computed by dividing the mean by the standard deviation of posterior estimates found via MCMC routine. *, **, *** denote coefficients significant at 10 %, 5 % and 1 % levels according to their (pseudo) t-statistics.

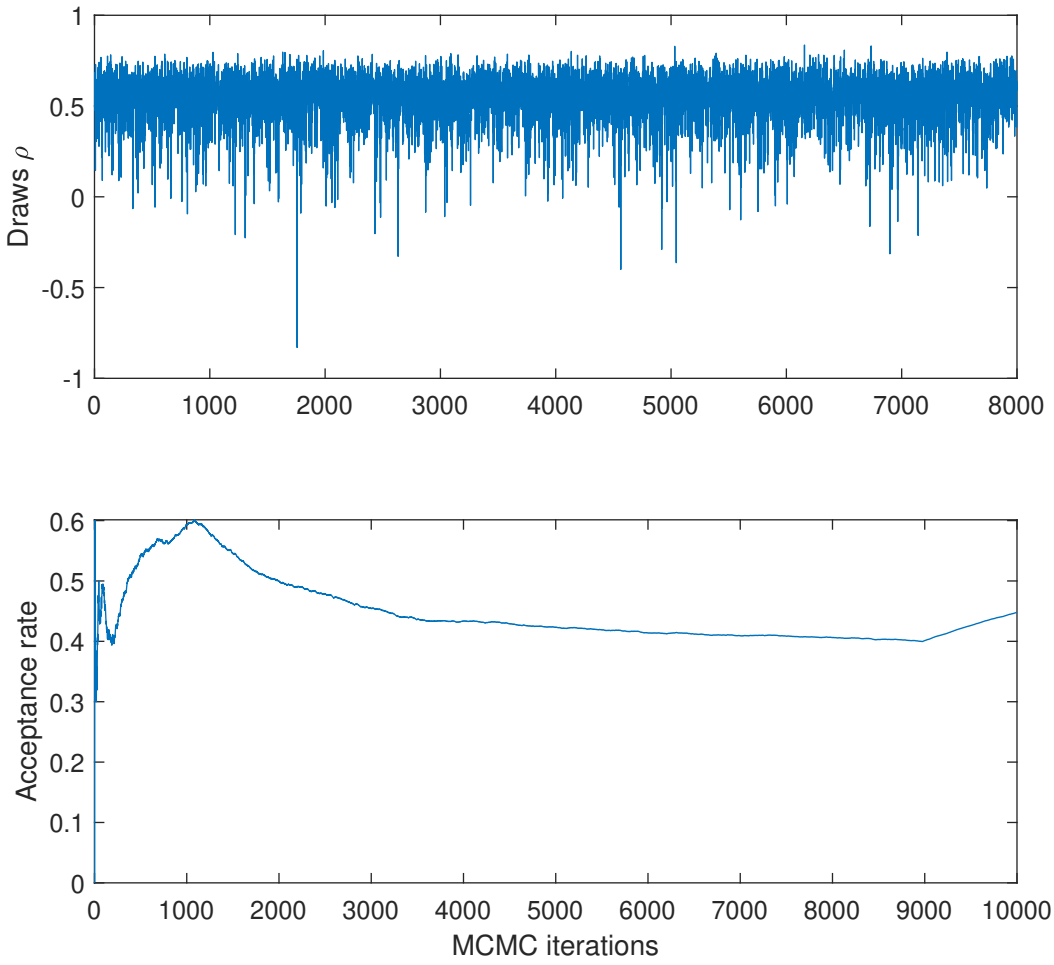


Figure 15: Model Estimation Diagnostics. Metropolis Hastings acceptance rate and draws for ρ.