The Role of Contagion in the Transmission of Financial Stress

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This version: 6th of July 2018

Abstract

I examine the relevance of contagion in explaining financial distress in the US banking system by identifying the component of bank level probabilities that is due to contagion. Identification is achieved after controlling for macrofinancial and bank specific shocks that have similar consequences to contagion. I use a Bayesian spatial autoregressive model that allows for time-dependent network interactions, and find that bank default likelihoods depend, to a large extent, on peer effects that account on average for approximately 35 per cent of total distress. Furthermore, I find evidence of significant heterogeneity amongst banks and some institutions to be systemically more important that others, in terms of peer effects. Through the lens of a simulations exercise I study the importance of the structure of financial networks for financial stability. Evidence suggests diversification is negatively related to financial contagion. Whereas, clustering increases the vulnerability of the financial system to low frequency, high severity events such as the ones observed during financial crisis.

JEL classification: E44, G01, C11, G21.

Keywords: Systemic Risk, Contagion, Spatial Econometrics, Bayesian Methods.

*This paper is an extended version of "The role of contagion in the transmission of financial stress" Working Paper Series No.81 European Systemic Risk Board, European Central Bank, shortlisted for the 2018 Ieke Van Den Burg Prize. I wish to thank Dimitris Korobilis and John Tsoukalas for their comments and advise. I am also grateful to Ioannis Tsafos, Sisir Ramanan and Christiana Sintou, to the participants of the 50th Money, Macro and Finance Annual Conference, the 2nd Workshop on Financial Economics and Network Science and the 6th ECB/CBRT Conference in "Modelling Macro-Finance interaction", for their helpful comments. Any errors are my own. Correspondence: University of Glasgow, Adam Smith Business School, 11 Southpark terrace, Glasgow, UK e-mail: m.herculano.1@research.gla.ac.uk.

1 Introduction

Financial crises tend to occur in waves and in each of these waves there are regional clusters, suggesting that contagion effects play a major role in triggering these events (see Reinhart and Rogoff (2009) and Peydro et al. (2015)). The financial system plays a key role in the unfolding of financial crisis, particularly in the mechanisms of transferring risk from individual institutions to the financial markets at large. Various important related faults highlighted by Acharya et al. (2012) explain how the US sub-prime mortgage crisis, a small piece of the overall system, turned into a worldwide phenomenon. 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"*, adding that " *(...) contagion either precipitates the failure or increases the probability of failure and, more generally, increases the fragility of the banking system.* ". Starting from this definition, this paper proposes a new framework to think about contagion in the banking system that addresses three main statistical challenges identified in the literature and outlined below.

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). 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 banks' exposure to common shocks such as imprudent lending standards. In this case, the consequent write-offs of non-performing loans (and not contagion) could be the source of a crisis. Therefore, disentangling both effects is tricky as the borders between contagion and macrofinancial fragility are blurred. Since in practice contagion and macrofinancial shocks hit the economy simultaneously, identifying contagion in data is not trivial. Failing to account for these factors results in omitted variable bias and thus wrong answers. Thirdly, heteroskedasticity and outliers are ubiquitous in financial data. As noted by Forbes and Rigobon (2002) failing to account for these features will result in misspecification and biased results that lead the misinterpretation 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. 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. Forbes and Rigobon (2002), however show that the increase in volatility in crisis episodes induces an upward bias in correlation coefficients. The author highlights 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. Vector Autoregressive models employed by Constâncio (2012) examine evidence of contagion by jointly modelling time series in an endogenous setting, while controlling for global and idiosyncratic factors. Contagion is measured through impulse responses and regarded as the impact of a surprise to one series on others. This approach is plagued by the difficulty of identifying the structural parameters of the model. The standard cholesky identification scheme is not suitable in this context because it involves establishing an order of exogeneity amongst variables in the model. An alternative approach is however proposed by Rigobon (2016) and consists in identifying the structural parameters by using the time-varying nature of the variance-covariance matrix of the residuals. Along the same lines, latent factor and GARCH models are used to capture contagion in an attempt to model heteroskedasticity, a common problem of previous methods (see Celik (2012); Dungey et al. (2015)). The main critique to these methods is that they are not designed to deal with endogeneity and omitted variable problems.

This paper proposes a different approach to measure contagion. I 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, controls 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 the financial conditions of all other banks and its own fundamentals, while controlling for unobserved macrofinancial shocks and bank specific shocks. Estimation is carried out with Bayesian techniques, whereby the panel of default probabilities observed since 1990 for up to 1311 US banks available are explained by regressing these on the likelihood of default of other institutions,

[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).

connected by an exogenous time dependent weights matrix and a set of control variables that describe banks' fundamentals and also unobserved macrofinancial and idiosyncratic shocks to banks, 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 I label contagion, from another component due to bank's fundamentals, including its liquidity position, solvency, leverage and non-performing loans.

This 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, by using panel data 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, its panel structure 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.

I 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-a-vis its own fundamentals. It is estimated that, on average 100bps increase in probabilities of default across the banking system will lead to an increase of institution level distress by 39 bps. Amongst the principal characteristics of banks, non-performing loans (NPLs) stand out as the most important covariate driving financial distress. Everything else equal, an increase of 100bps in NPLs across the banking system induces a hike of 79 bps in likelihood of default of each institution on average.

Overall, evidence suggests that contagion accounts for about 35 per cent of the probability of default of banks. This result stems from the identification of the part of the default probability of each bank that is due to the behaviour of the banking system taken together. This parcel is disentangled from the remainder that can

be interpreted as resulting from bank specific characteristics reflecting it's business model and balance sheet. I calculate the spillover resulting from idiosyncratic shocks to banks fundamentals, showing its consequences for financial stability. I also 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 spillover of 87bps on the remaining banks' probability of default. 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, I find that ρ, the amplifying parameter in our model, changes significantly when adding sequentially year fixed effects during the Great Recession, suggesting that the density of the banking network after the crisis decreased, reducing systemic risk due to contagion.

In another direction, we contribute towards the discussion of the implications of the structure of financial networks to financial stability, shedding light on the empirical adherence of three important theoretical propositions in the literature that remain untested. First, does diversification in financial networks mitigate contagion ? Although the theoretical guidance of the seminal contributions by Allen and Gale (2000) and Freixas et al. (2000) suggests so, 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. Second, is a 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. Third, are diversification and clustering in financial networks desirable from a financial stability viewpoint ?

We rewrite some of the most important propositions proposed by the above mentioned authors in such a way that they can be empirically tested. The hypothesis are examined through the lens of a simulation exercise in which hypothetical financial networks are combined with real data and generate different patterns of diversification, clustering and contagion. This exercise allow us to draw important results on the significance of the structure of financial networks in promoting financial stability. Three important results emerge from this exercise. First, broadly consistent with the theoretical guidance of Allen et al. (2010b) and Freixas et al. (2000), more diversified financial networks are less prone to contagion. Second, higher levels of clustering allow for greater diversification in a financial network. However, when

clustering is high, the system is vulnerable to low frequency high severity events and in the tails, contagion levels are significantly higher. Third, the optimal financial network from a financial stability point of view is one featuring high levels of diversification and clustering. Nevertheless, 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].

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

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

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 spi-

rals. 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.

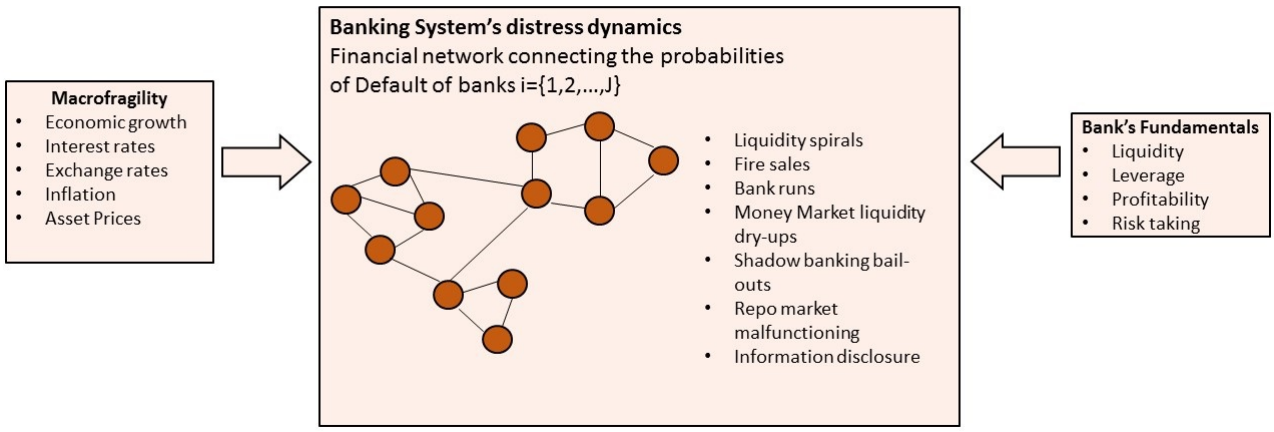


Figure 1: A blueprint of contagion - The main channels.

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

I use a spatial autoregressive model to describe the dynamics of default probabilities between banks and in particular how financial strain can spillover from one institution to become systemically relevant. Therefore, 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 I use Merton (1974) structural model and include an explanation of this approach in Appendix A [3].

The main idea underpinning the spatial panel 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 can be written as follows

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

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. Since spatial panel models can only be estimated on a balanced panel (see Data and Estimation section for more details), 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. ρ is known as the spatial autoregres-

[3] It should be stressed that such default probabilities are available through financial data providers such as Thomson Reuteurs or Bloomberg albeit only for the largest financial institutions.

sive 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 represents 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.

3.0.1 Direct and spillover effects of financial distress

One of the main advantages of using spatial panel models 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} wrt. the k-th explanatory variable X_k. By writing model 1 in its reduced form as

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

It is clear that

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

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.

At this point, we are in condition to establish what exactly is meant by contagion in this framework.

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

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

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

Hence, financial contagion is capturing the strength whereby a shock to banks fundamentals is propagated through the system and generating an externality that is unrelated with fundamentals. It is helpful to notice that the quantity described in 4 is a square matrix with ones on its principal diagonal. These reflect the direct effects of a shock to any covariate. Whereas, off diagonal elements of 4 reflect the impact of the bank represented row-wise by the node j on its conterparty depicted in column i. Furthermore, from the definition above, one can observe 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 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.

$$H_0 : \rho = 0. \tag{5}$$

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 characterized and the our approach to study the interactions between diversification and contagion.

3.0.2 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 all 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 and takes the form of a social distance. As a first step in describing the financial networks, I calculate a Trailing Twelve Months (TTM) correlation matrix of stock prices for banks in our sample. I use daily frequency with a view of capturing quick co-movements in the market that may reflect changes in value of the underlying banks. Moreover, I only consider institutions whose stocks are active in the secondary market as an attempt to make sure that prices are an accurate measure of value as possible. A second step consists in converting this set of correlation matrices into weight matrices that provide the measure of social distance we need to estimate the model. To allow interconnections to vary over time in our sample, we use a block matrix that stores the weight matrix at each point in time in the principal diagonal.

$$W_t^* = \begin{bmatrix} \omega_1^* & 0 & 0 & \dots & 0 \\ 0 & \omega_2^* & 0 & \dots & 0 \\ \vdots & \vdots & \ddots & & 0 \\ 0 & 0 & 0 & \dots & \omega_t^* \end{bmatrix} \tag{6}$$

The weight matrix W_t^* describes the relationship between each bank in our panel, in each period of time $t = 1, ..., T$. It is worth highlighting that ω_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 ω_t^* characterizes the distance between bank i and bank j at time period t. It results from the correlation between the market prices of both banks, normalized to take values between 0 - no relationship; and 1 - strong relationship, as described below.

DEFINITION 2 Let Γ_t equal a transform of the correlation matrix Ω_t, where each generic element $\Gamma_{ij} = max(\Omega_{ij}, 0)$. Then the spatial weights matrix for time t

is given by

$$\omega_t^* = \Gamma_t - I_k, \tag{7}$$

Where k equals the number of banks present in the sample at time t.

DEFINITION 3 Let W_t^* be a diagonal block matrix, where each matrix in the principal diagonal ω_t stands for the spatial weights matrix describing the financial network at time t, as defined above. Then, the matrix resulting from the row-normalization of W_t^* is denoted W_t.

Definition 2 guarantees that its principal diagonal is in fact a vector of zeros, consistent with the literature on spatial econometrics (see LeSage (1999); Elhorst (2014)). Whereas, Definition 3 ensures that the weight matrix W_t that is the key input to the model assumes values between 0 and 1 and that its row-wise sum adds to 1.

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.

3.1 Diversification, 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 strengthen, 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.

In this section we study how the structure of the financial network - and in particular how diversified it is - determines the extent of contagion. A spatial panel model is a good device to shed light on the relationship between diversification 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 the theoretical guidance discussed so far. In particular, we ask how contagion reacts to an increase in diversification 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 diversification in its two dimensions - interconnection and clustering. We follow the literature, in particular Acemoglu et al. (2015) and define diversification 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 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 closeness of bank i to the network is given by

$$H(i) = \sum_{j \neq i} \frac{1}{w_{ij}}. \tag{8}$$

REMARK 1 Assume that the level of interconnection of bank k with the financial

network is well described by the harmonic closeness measure $H(k)$. Then the greater $H(k)$ the greater the level of diversification.

Definition 4.1 establishes a measure of diversification, given by the inverse distance of a bank with the banking system. Notice that greater levels of $H(.)$ suggest greater diversification. Additionally, we measure the level of clustering in a financial network in the following way

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

$$F(w_{11}, w_{12}, ..., w_{JJ}) = C_\tau(f(w_{11}), f(w_{12}), ..., f(w_{JJ})). \tag{9}$$

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. [4] 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, due to asset commonalities and common risk exposures, risk is more concentrated in a clustered network. In an opposite way, an unclustered financial network is one where, even if banks are interconnected, none hold identical portfolios and consequently the risk is less concentrated.

Finally, to understand the impact of diversification 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 $E[c|W] \leq E[c|\tilde{W}]$.

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

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

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 diversification. To be precise we will examine three controversial theoretical results put forward by Acemoglu et al. (2015); Allen and Gale (2000) and Allen et al. (2010a):

H1. *Diversification in financial networks mitigates contagion.*
H2. *A less clustered financial network is less prone to contagion.*
H3. *Diversification 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.

3.2 Data and Estimation

A panel of 1311 banks' 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, from a first stage calculation of correlation matrices of all quoted banks for each time period, using a rolling window of one year historical returns. 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 panel regression specified in equation 1 relies on a set of measures of bank fundamentals meant to purge from the probabilities of default 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) figures are included to account for profitability, whereas leverage, Market-to-Book values (Tobin's Q) and Non-performing loans' figures account for risk. The criteria underpinning the choice of the specific regressors to include with a view of depicting bank's fundamentals was a compromise between including the most important indicators of a bank's financial position and not loosing too many observations due

to missing values. This is mainly due to the fact that spatial panel models rely on balanced datasets to be estimated[5].

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

$$y = \rho W_t y + X^* \beta + \varepsilon, \tag{10}$$

$$\varepsilon \sim N(0, \sigma^2 V) \tag{11}$$

Where X^* includes all K+2 regressors capturing bank fundamentals plus time and bank fixed effects . The 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 10 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

$$p(\Psi|\beta, \sigma, \rho) = (2\pi\sigma^2)^{-n/2}|A|exp\Big[-\frac{1}{2\sigma^2}(Ay - X\beta)'(Ay - X\beta)\Big]. \tag{12}$$

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

$$p(\theta|\Psi) \propto p(\Psi|\theta)p(\theta). \tag{13}$$

Hence, a first step to solve the model is to specify a set of priors for the parameters at hand. I set an 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

[5]As noted by Elhorst (2014) in the event of missing observations, the accuracy of estimators is not guaranteed although there is some literature dealing with this problem (see Pfaffermayr (2009); Wang and fei Lee (2013)), a general approach is not available.

V.

$$p(\beta) \sim N(c, T), \tag{14}$$

$$p(\sigma^2) \sim IG(a, b), \tag{15}$$

$$p(\rho) \sim U(\lambda_{min}^{-1}, \lambda_{max}^{-1}), \tag{16}$$

$$p(r/v_i) \sim iid \quad \chi^2(r). \tag{17}$$

The choice of a NIG prior is motivated by its widespread use in the Bayesian Econometrics literature (see Koop (2003); Koop and Korobilis (2009)). I set a=b=c=0 and assign a very large prior variance for β (large T) and thus our priors are uninformative. This is due to the fact that I 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 I want also 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 leaves the data to speak for itself.

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 13 to get the

posterior

$$p(\beta, \sigma^2, \rho, V|\Psi) \propto (\sigma^2 V)^{a^*+(k+2)/2+1}|A|exp\Big[-\frac{1}{2\sigma^2 V}[2b^* + (\beta - c^*)'(T^*)^{-1}(\beta - c^*)]\Big], \tag{18}$$

$$c^* = (X'X + T^{-1}(X'Ay + T^{-1}c), \tag{19}$$

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

$$a^* = a + n/2, \tag{21}$$

$$b^* = b + (c'T^{-1}c + y'A'Ay - c^*(T^*)^{-1}c^*)/2. \tag{22}$$

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.

3.2.1 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.

I 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 10-11 and 14-17 I wish to estimate, 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. I will 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, I summarize the algorithm procedure step by step below. Starting from a set of arbitrary values β_0 σ_0^2, V_0 and ρ_0, I sample 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 from

$$c^* = (X'V^{-1}X + \sigma^2 T^{-1})^{-1}(X'V^{-1}(I_n - \rho W)y + \sigma^2 T^{-1}c), \tag{23}$$

$$T^* = \sigma^2(X'V^{-1}X + \sigma^2 T^{-1})^{-1} \tag{24}$$

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

$$a^* = a + n/2; \quad b^* = (2b + e'V^{-1}e)/2; \quad e = Ay - X\beta. \tag{25}$$

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)

$$p(\rho|\beta_1, \sigma_1^2, V_1) = |A|exp\Big[-\frac{1}{2\sigma^2 V}e'V^{-1}e\Big] \tag{26}$$

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 26 is unknown and thus 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, I 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 generate 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

$$\rho^c = \rho_{h-1} + c \times N(0,1). \tag{27}$$

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 26. The acceptance probability is given by

$$\psi(\rho^c, \rho_{h-1}) = min\Big[1, \frac{p(\rho_{h-1}|\beta,\sigma)}{p(\rho^c|\beta,\sigma)}\Big]. \tag{28}$$

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.

It can be seen from Figure 15 that the acceptance probability fluctuates between 0.4 and 0.6 for the first 3000 replicas, converging to 0.5 thereafter. This also provides a diagnostic tool to monitor our MCMC routine.

4 Discussion of the results

This section discusses empirical results focusing on the following questions. First, is there any evidence of contagion and if so, does its magnitude vary over time ? 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 ? Next, what is empirical relationship between diversification, contagion and financial stability ?

Before addressing the former 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 firm's asset value falling below its total liabilities within one year. Figure 12 describes the individual dynamics of these quantities since 1990 for the 9 largest institutions in the US as of 2018, measured by market capitalization. I see that literally every major financial institution suffered, to some extent, from 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 1311 US banks included in our sample. Unsurprisingly, the probability of default of a significant amount of institutions peaked in the run-up and unfolding 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 crisis. It can be seen that 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 iid[6], needed 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, I estimate a simple panel regression, reported in Table 3, column I and compare it with Maximum Likelihood SAR model estimates in column II. As I 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,

[6]independent and identically distributed

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 that accounts for heteroskedasticity, estimated with time and bank fixed effects is the benchmark regression and our workhorse throughout the paper. The results are reported in the column IV. It is important to emphasize that ρ, the parameter that measures contagion of distress between institutions, is significantly different from zero across specifications, suggesting that contagion is an important factor to take into consideration when thinking about the likelihood of default of each bank. Moreover, its magnitude varies across specifications but in all cases it is economically meaningful. In the baseline specification IV, ρ equals 0.429, meaning that on average, 100bps increase in probabilities of default across banks will increase the likelihood of default of each institution, taken individually by approximately 43 bps.

Another important aspect to highlight refers to the covariates included to account for bank fundamentals. I include leverage, that is estimated to increase a banks' likelihood of default by 24 bps. per unit. Profitability, measured by Return on Equity figures, that decreases distress by 18 bps, while size is estimated to discretely decrease the likelihood of default, everything else constant. Non-performing loans, measured as a percentage of total loans, are found to relate closely with bank level distress. Everything else equal, it is found that an increase of 1 per cent in this figure is estimated to lead to a 83 bps. increment of the likelihood of the default of a given institution. This result highlights 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.

Non-performing loans (NPL) are found to be play a central role in determining banking distress. To examine this point further, I estimate a spatial Durbin model that differs from the SAR since it allows NPLs of banks to affect each other dynamically. This is archived by pre-multiplying non-performing loans by the spatial weight matrix, giving it a spatial structure, and including the new covariate in the regression. Results are reported in column V of Table 3. I find that, by allowing NPL to exhibit a spatial structure, the regressor becomes statistically more significant and the magnitude of the coefficient increases when compared to the baseline regression. Everything else equal, an average increase of NPLs across the banking system induces a hike of 127 bps. in the probability of default of a bank, taken individually. I also observe that the coefficients of other explanatory variables change significantly. In particular, the importance of leverage decreases while the

significance of the Tobin's Q or the Book-to-Market value of a bank, increases in explaining distress.

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 account heteroskedasticity of unknown form by allowing the variance covariance matrix to vary, while remaining agnostic about its functional form.

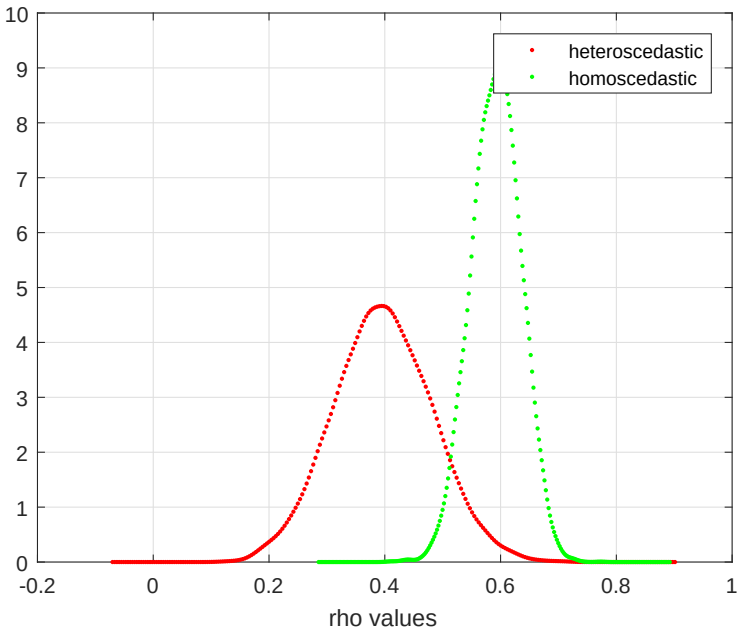


Figure 2: Full sample estimates of the posterior distribution of ρ

Figure 2 shows that, consistent with what is found in the literature, failing to account for heteroskedasticity induces a bias that leads to an overstatement of the magnitude of contagion. In our framework, the values for ρ decrease significantly. Nevertheless, it should be stressed that even accounting for this feature of the data, there is still strong evidence for the presence of contagion. It can also be seen from Table 3 that t-statistics for the coefficients of regressors reduce significantly when heteroskedasticity is controlled for.

4.2 Contagion across time

I 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, I decompose the probabilities of default into two components. The parcel due to contagion, that reflects peer effects and spillovers of distress from other institutions

is isolated from the component due to own fundamentals that include profitability, solvency, 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.

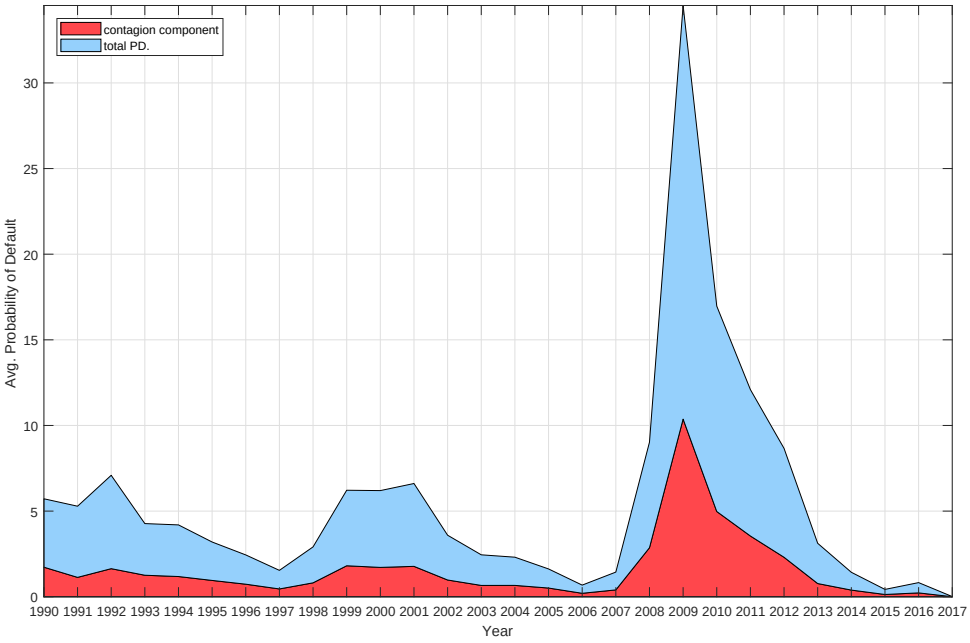
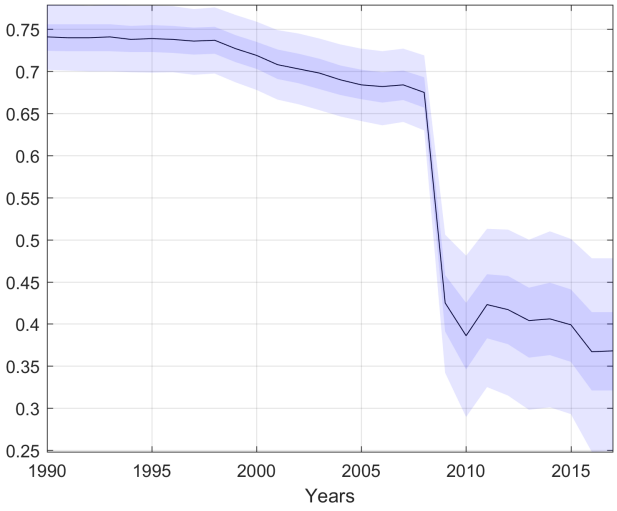


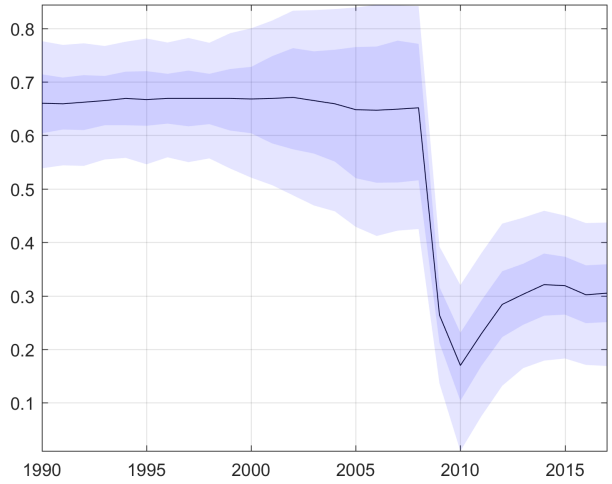
Figure 3: Decomposition of probabilities of default for US banks, 1990-2018. Red stacked line represents the proportion of the total observed probability of default due to contagion.

Figure 3 shows that contagion accounts for a significant part of the likelihood of the default of banks, on average 35 per cent throughout the full sample. This value increased during the great recession where bank probabilities of default surpassed 40 per cent on average.

I have discussed that contagion in our model is a product of two distinct forces. One one hand, interconnection between banks that is exogenous and defined by banking links. On the other hand, dependence measured by ρ, endogenously defined. I now examine how this parameter changes over time, considering the models with and without heteroskedasticity.



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



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

Figure 4: Posterior density of the parameter ρ estimated adding sequentially year fixed effects to the baseline Bayesian SAR with and without heteroskedastic errors. Colour bands highlight the posterior percentiles of the MCMC sampled draws.

Figure 4 shows that spatial dependence between banks decreases sharply in the aftermath of the crisis. Notwithstanding, ρ is still statistically significant throughout the sample, except for a brief period during the Great Recession. The hypothesis that heteroskedasticity biases estimates of the magnitude of contagion can also be confirmed. The average upward bias throughout time of ρ is about 0.10.

4.3 Contagion across banks

Beyond its time dynamics, the cross-sectional dimension of contagion matters because the domino effect that characterizes it may be triggered by a single chip. One of the advantages of using panel data is the possibility of exploring both time and cross-section to study the problem at hand. In this section I 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 other institution. It reports the average probability of default for each of the 50 largest institutions in the US banking system, measured by market value as of 2018 in column 1. Institutions in the Table are sorted from largest to smallest size. Column 2 quantifies the first term in our baseline regression 1, which I interpret as the probability of default of each bank due to contagion. Thus, this parcel indicates the vulnerability of each institution to external shocks to 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 columns express the direct, indirect or spillover and total effects of a shock to a given banks' fundamentals. These quantities give an idea of the externality or importance of each bank for overall financial stability.

I find significant heterogeneity amongst banks with respect to their peer effects. In particular, it is estimated that a shock to the least systemically important bank that causes its own probability of default to rise by 100bps. will cause a system-wide increase in the probabilities of default of 15bps, taken together. Whereas, a shock resulting in a 100bps. hike to the probability of default of the most systemically significant bank will increase the probabilities of default across the board by a total of 103bps. Furthermore, evidence suggests that vulnerability to external shocks and systemic importance are not directly related to size. In particular, the bank which shows the greatest knock-on effect on the system, and thus larger impact on financial stability doesn't belong to the top 10 largest institutions within those considered in the Table. On the other hand, the bank presenting greater vulnerability (ie, largest contagion component) is also not in 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 is financial stability compromised by a deterioration or improvement in the fundamentals of banks ? This is the question I look into through the lens of the two spatial econometric models that I have estimated in previous sections. Table 3 reports the main results estimated for the baseline specification - the Bayesian SAR model with time and bank fixed effects. The same variables are estimated with a Bayesian spatial Durbin model, that differs from the first in that it allows for network effects of non-performing loans, the covariate that is found to play an important role in driving financial distress. The reader may also find the values for the credibility set around these estimates (ie, the relevant percentiles of the posterior distribution of these variables).

I find that the most relevant bank characteristics that influence their likelihood of default are non-performing loans and profitability as measured by Return on Equity. In particular, an increase in 1 unit in the Return on Equity of a bank is found to reduce distress by approximately 33bps, of which 14bps are accounted for positive spillovers from the system. Whereas, an increase in 1 percentage point in delinquency rates, as measured by NPLs, are estimated to increase financial distress by 149bps. on average. Estimates from the spatial Durbin model are more significant. According to this specification, 1 percentage point increase in NPLs of a given bank will increase its likelihood of defaulting by as much as 209bps, of which 82bps are due to network effects. The Tobin's Q as measured by the book-to-value ratio of an individual bank is only significant for the spatial Durbin model specification. The same is true for liquidity, proxied by the Loan-to-Deposit ratio and size. A change of 1 unit in the Book-to-value ratio results in an improvement in financial conditions of 89bps of which 35bps are due to peer effects, while liquidity and size have statistically significant but very modest and economically uninteresting effects on the likelihood of default.

Figure 5 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 left skewed and concentrated between 0 and 1, yielding a total effect that lies between 1 and 2, suggesting that shocks to bank fundamentals have powerful peer effects.

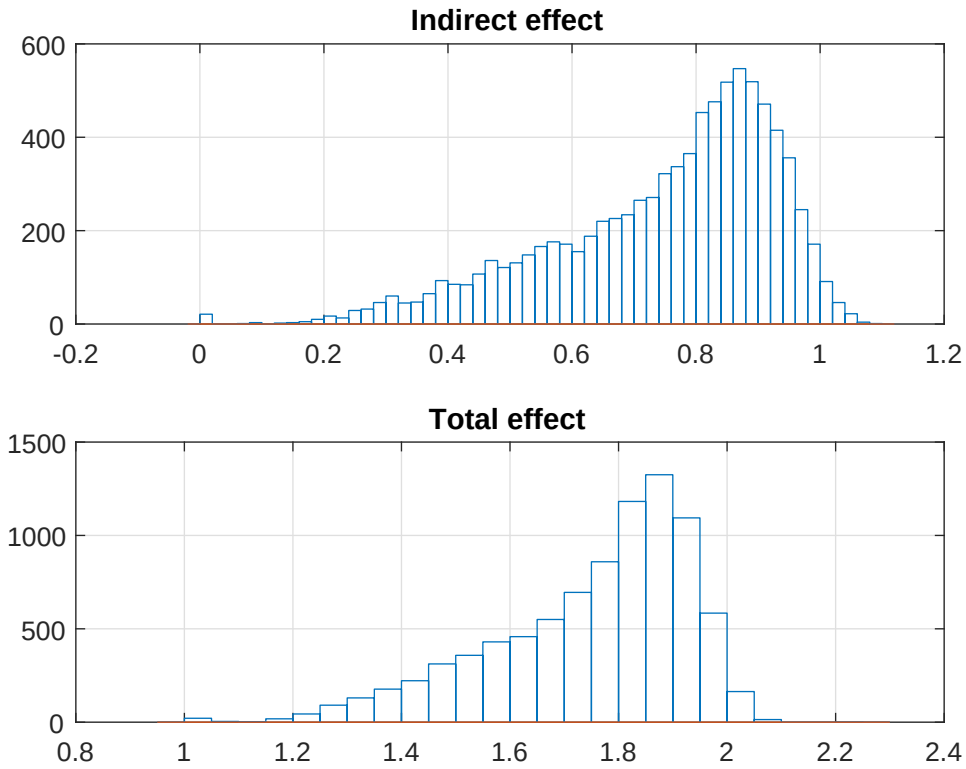


Figure 5: Posterior distribution of the direct, indirect and spillover effects estimated with from the heteroskedastic Bayesian spatial autoregressive model.

4.5 Diversification, contagion and financial stability

To empirically examine the extent to which diversification in financial networks represents heightened contagion risk, we design a Monte Carlo experiment and draw a hypothetical financial network in each iteration (see Appendix D for details). Each pseudo financial network $\tilde{W}$ is built from randomly drawing each of its element $\tilde{w}_{ij}$, characterizing the distance between a pair of banks $\{i, j\}$. One out of three candidate distributions shown below are chosen to describe the distance between banks

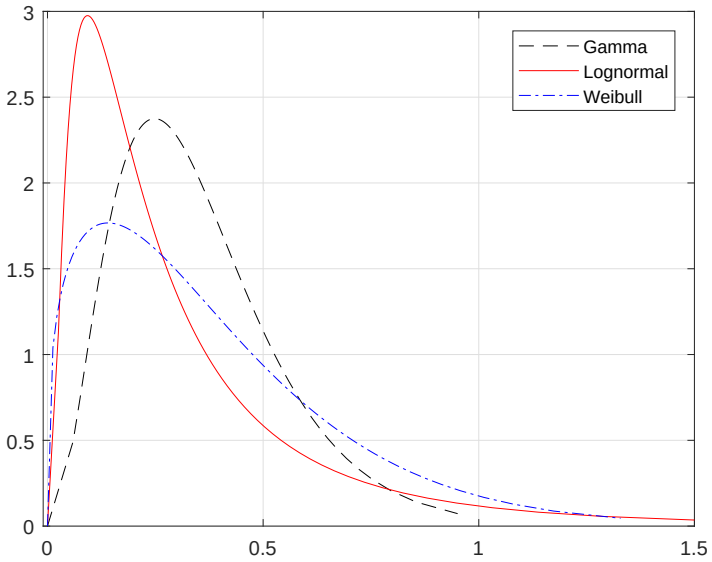


Figure 6: Three candidate distributions with different tail configurations to describe distance between a pair of banks $\{i, j\}$.

The three distributions are parametrized through the method of moments and represent three different types of tail configurations. It can be seen than, while the Gamma distribution has a lighter tail, the Weibull and the Lognormal distributions present fatter tails. The intuition underlying the choice of different tail configurations for the distribution of distances between banks is to distinguish between close-knit financial networks and networks where the distance between banks taken together is more heterogeneous. The joint behaviour of the full matrix $\tilde{W}$ composed by random draws w_{ij} is then modelled with a Gaussian copula with two different dependence parameters. A value of $\rho = 0.1$ is set to represent low clustering in the network. Whereas, $\rho = 0.9$ is chosen to model a scenario of high clustering, where banks are heavily exposed to common shocks.

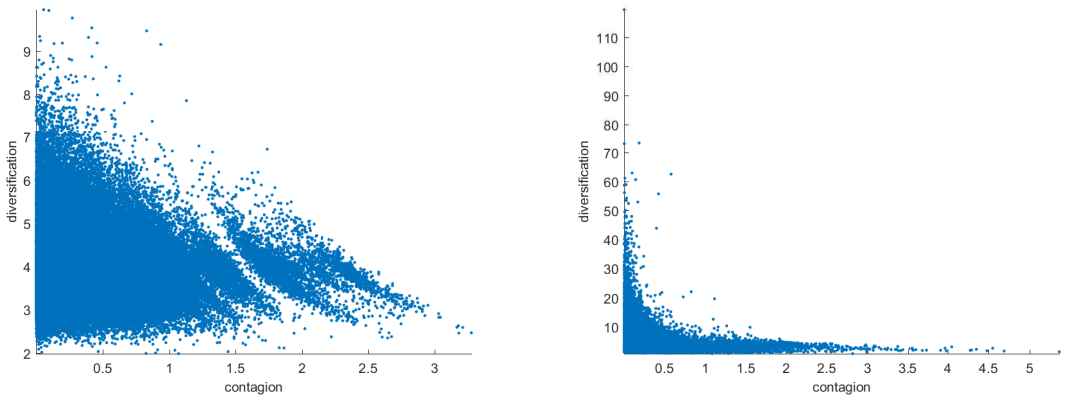
Turning to the results of the simulation exercise, we examine whether empirical evidence validates each of the three hypothesis suggested in section 3.1.

H1. *Diversification in financial network mitigates contagion.*

First, is a diversified financial network less prone to episodes of contagion ? Simulation results suggest so. Figure 7 shows the relationship between diversification and contagion as implied by simulation outcomes. Diversification, measured as the harmonic distance between a pair of banks is negatively related to contagion. This result is robust to different choices of distributions to describe the distance between each pair of banks and the two different copula specifications considered (see Figures

16 and 17). An important aspect of this result is that even though contagion is indeed lower for more diversified financial networks and it is when diversification is at its lowest that contagion is more powerful, it can be observed that low diversification does not imply necessarily high contagion. Figure 7 (a) shows that for the majority of financial networks where diversification is low, contagion is also moderate. This is consistent with the view that low diversification is a risk factor that contributes to the vulnerability of the financial system to contagion, as opposed to the idea that diversification is a necessary and sufficient condition for contagion.

Another aspect that can be observed from Figures 7, 16 and 17 is that the magnitude of contagion changes significantly according to both the distribution of the distance between banks and the level of clustering in the network. For instance, the use of a Lognormal distribution together with a Gumbel copula yield contagion levels that exceed 40 fold.



(a) Low clustering

(b) High clustering

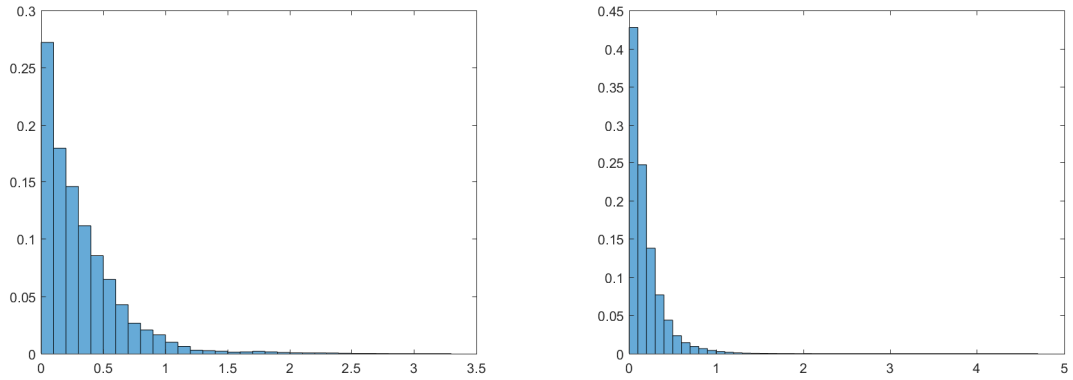
Figure 7: Simulation results. Relationship between diversification and contagion where $\tilde{W}$ is drawn from a Gamma distribution and a Gaussian copula is fitted to the data with $\rho = 0.1$ and $\rho = 0.9$, representing low and high degrees of clustering, respectively.

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 returns, reflecting the extent to which banks, taken together, are exposed to common risks (see Allen et al. (2010a)). Simulation results reported in Figure 8 suggest that a high level of clustering in a financial network allows for greater diversification but can also lead to greater contagion when diversification is low. As such, the distribution of the magnitude of contagion is fat tailed for high clustered

networks albeit, average contagion level is lower. 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.



(a) Low clustering

(b) High clustering

Figure 8: Simulation results. 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 the financial stability implication of diversification, 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 9. The diagram shows the two criteria of financial stability of a network as a function of diversification and clustering levels. In interpreting the results in this diagram it is worth highlighting that, in accordance with Definition 5, higher 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 in the network. Hence, the greater these quantities, the lower financial stability.

Stability of a financial network 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 higher is such that both clustering and diversification are high. However, we observed that the set of financial networks exhibiting high clustering are only optimal from a financial stability viewpoint if diversification is high at all times. If diversification is low in a highly clustered network, the financial system is not resilient, and exposed to powerful low frequency, high severity events.

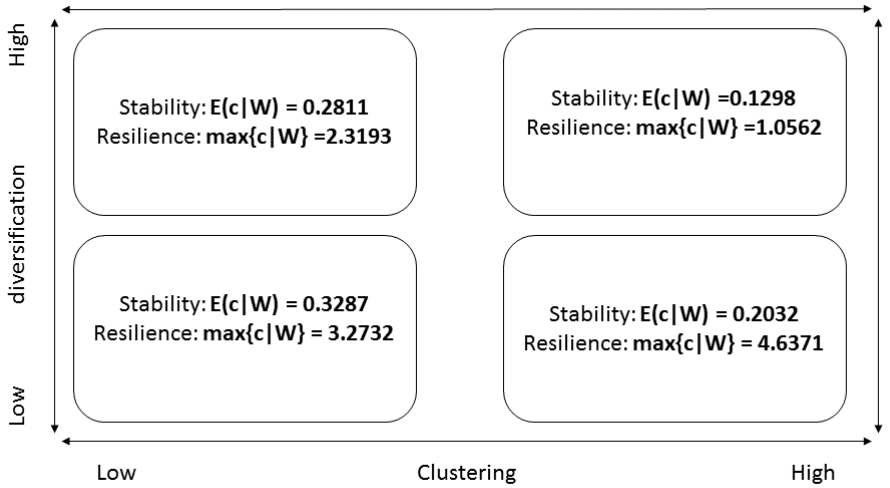


Figure 9: Box matrix chart showing contagion levels for each state of diversification and clustering

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

5 Conclusion

We have examine the evidence on 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 throughout the business cycle. Moreover, it offers the possibility of looking into the cross-sectional heterogeneities within the banking system. Contagion is captures 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.

We rewrite some of the most important theoretical propositions in such a way that they can be empirically tested. Through a simulation exercise, we find that diversification mitigates contagion risk and that clustering leads to a fatter tailed distribution of the magnitude of contagion. Financial networks where both clustering and diversification are high exhibit the highest levels of resilience and stability. Notwithstanding, the interaction of low diversification 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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Appendix A: 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)[7] 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.

$$A = E + L \tag{29}$$

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

$$E_t = max\{0, A_t - L\} \tag{30}$$

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.

Hence, the value of equity may be written, for a given horizon T, as a function

[7]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 and Lopez (2010) 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.

of assets A, liabilities L and a risk free interest rate r as follows

$$E = AN(d_1) - Le^{-rT}N(d_2), \tag{31}$$

where

$$d_1 = \frac{ln(A/L) + (r + 0.5\sigma_A^2)T}{\sigma_A\sqrt{T}} \text{ and } d_2 = d_1 - \sigma_A\sqrt{T}. \tag{32}$$

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

$$\frac{dA}{A} = \mu_A dt + \sigma_A \varepsilon \sqrt{t} \tag{33}$$

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

$$P(A_t < L) \tag{34}$$

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 34 Itô's general solution for the Stochastic Differential Equations written in equation 33 one gets

$$P(A_0 exp\{(\mu_A - \frac{\sigma_A}{2})t + \sigma_A \varepsilon \sqrt{t}\} \leq L) = P(\varepsilon \ \leq -d_1). \tag{35}$$

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

$$PD = 1 - N(d_1), \tag{36}$$

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 [8] consists in estimating the market value of assets of a firm, therefore inferring how far each firm is from default.

[8] 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.)

Appendix B: 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 I focus only on copula theory that is necessary for this article [9].

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 $C : [0, 1]^d \rightarrow [0, 1]$ 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

$$H(X, Y) = C(F(X), F(Y)), \forall X, Y \in \Re^2 \tag{37}$$

The theorem can easily be generalized for d random variables, in which case the copula has d-dimensions[10]. 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.

[9] 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. I found Nelsen (2005); McNeil et al. (2005) and Embrechts (2009) very insightful.

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

Let us first consider the Gaussian copula C_{τ}^{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

$$C_{\rho}^{gauss}(X,Y) = \int_{-\inf}^{\Phi^{-1}(X)} \int_{-\inf}^{\Phi^{-1}(y)} \frac{1}{2\pi(1-\rho^2)^{1/2}} exp\left\{ -\frac{s^2 - 2\rho st + t^2}{2(1-\rho^2)} \right\} \tag{38}$$

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

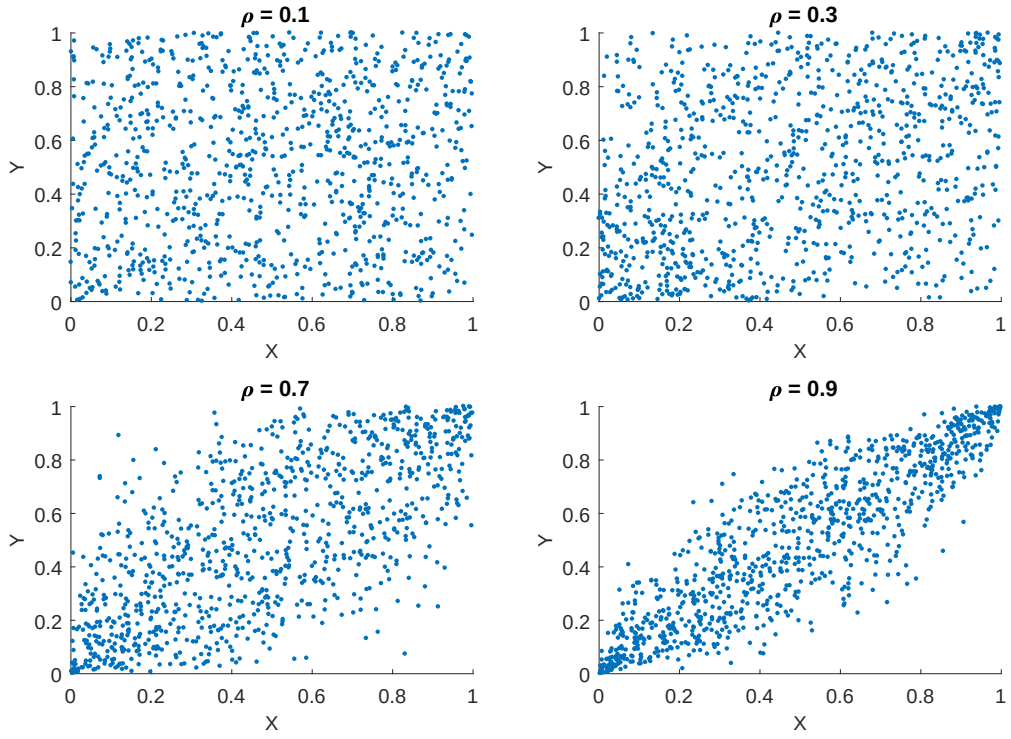


Figure 10: 5000 draws from a random vector (X,Y) generated with a Gaussian copula with marginals which are uniformly distributed between 0 and 1 and different levels of dependency ρ.

Setting $\rho = 0$, 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 $\rho = 0.9$ result in high clustering of the generated random variables.

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 ρ 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

$$C_\theta^{gum}(X,Y) = exp\Bigg\{ -\Big((-lnX)^\theta + (-lnY)^\theta\Big)^{1/\theta}\Bigg\}, \quad 1 \leq \theta < \inf, \quad \forall X, Y \in [0,1]. \tag{39}$$

The parameter θ is as a measure of association between X and Y. Just as ρ ,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

$$\tau(X,Y) = P[(X-\tilde{X})(Y-\tilde{Y}) > 0] - P[(X-\tilde{X})(Y-\tilde{Y}) < 0]. \tag{40}$$

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

$$\tau^{gum}(X,Y) = 1 - \frac{1}{\theta} \tag{41}$$

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[11].

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

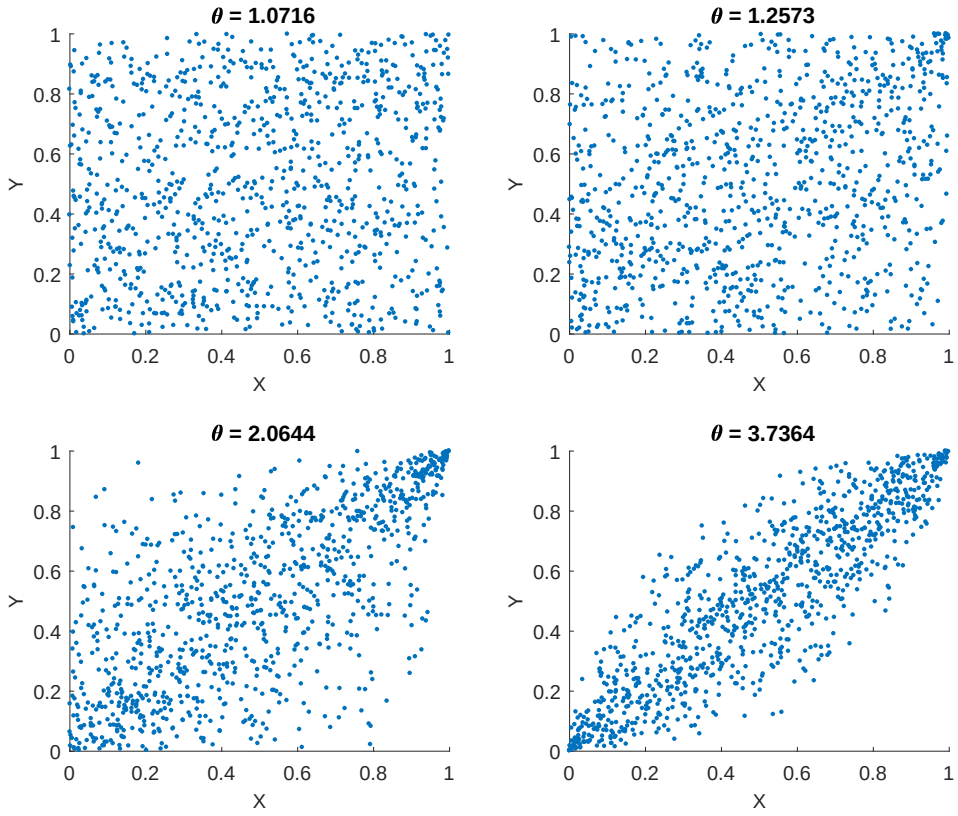


Figure 11: 5000 draws random vector (X,Y) generated with a gumbel copula with marginals which are uniformly distributed between 0 and 1 and different levels of dependency θ.

Figure 11 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 ρ plotted in Figure 10, 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.

Appendix C: An inventory of candidate distributions and copulas

	Gamma	**Lognormal**	**Weibull**
scale	θ	μ	θ
location	α	σ	τ
$f(X)$	$\frac{(X/\theta)^{\alpha}e^{-X/\theta}}{x\Gamma\alpha}$	$\frac{\phi(z)}{\sigma X}, \quad z=\frac{lnX-\mu}{\sigma}$	$-\frac{\alpha(-X/\theta)^{\alpha}e^{-(-X/\theta)^{\alpha}}}{X}, \quad x>0$
$F(X)$	$\Gamma(\alpha; X/\theta)$	$\Phi(z)$	$exp\{-(-X/\theta)^{\alpha}\}$
$E(X^k)$	$\frac{\theta^k\Gamma(\alpha+k)}{\Gamma(\alpha)}, \quad k>-\alpha$	$exp\{k\mu+1/2k^2\sigma^2\}$	$(-1)^k\theta^k\Gamma(1+k/\alpha), \quad k>-\alpha$

Table 1: Description of the three distributions used to model distances between each pair of banks i, j.

	$C(X,Y)$	parameters
Gaussian	$\int_{-\inf}^{\Phi^{-1}(X)}\int_{-\inf}^{\Phi^{-1}(y)}\frac{1}{2\pi(1-\rho^2)^{1/2}}exp\left\{-\frac{s^2-2\rho st+t^2}{2(1-\rho^2)}\right\}$	$-1\leq\rho\leq 1$
Gumbel	$exp\left\{-\left((-lnX)^{\theta}+(-lnY)^{\theta}\right)^{1/\theta}\right\}$	$1\leq\theta<\inf$

Table 2: Description of the two copulas used to model financial networks.

Appendix D: 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. [12]

2 Through the inverse transform method, convert the dependent uniform random vectors into an appropriate distribution, parametrized using the Method of Moments. [13].

3 Construct the pseudo - spatial weights matrix $\tilde{W}$ for the draw and estimate the model below

$$y = \rho\tilde{W}y + X\beta + \varepsilon. \tag{42}$$

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

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

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

[13] We consider a light tailed, medium tailed and heavy tail alternative distribution (see Appendix C).

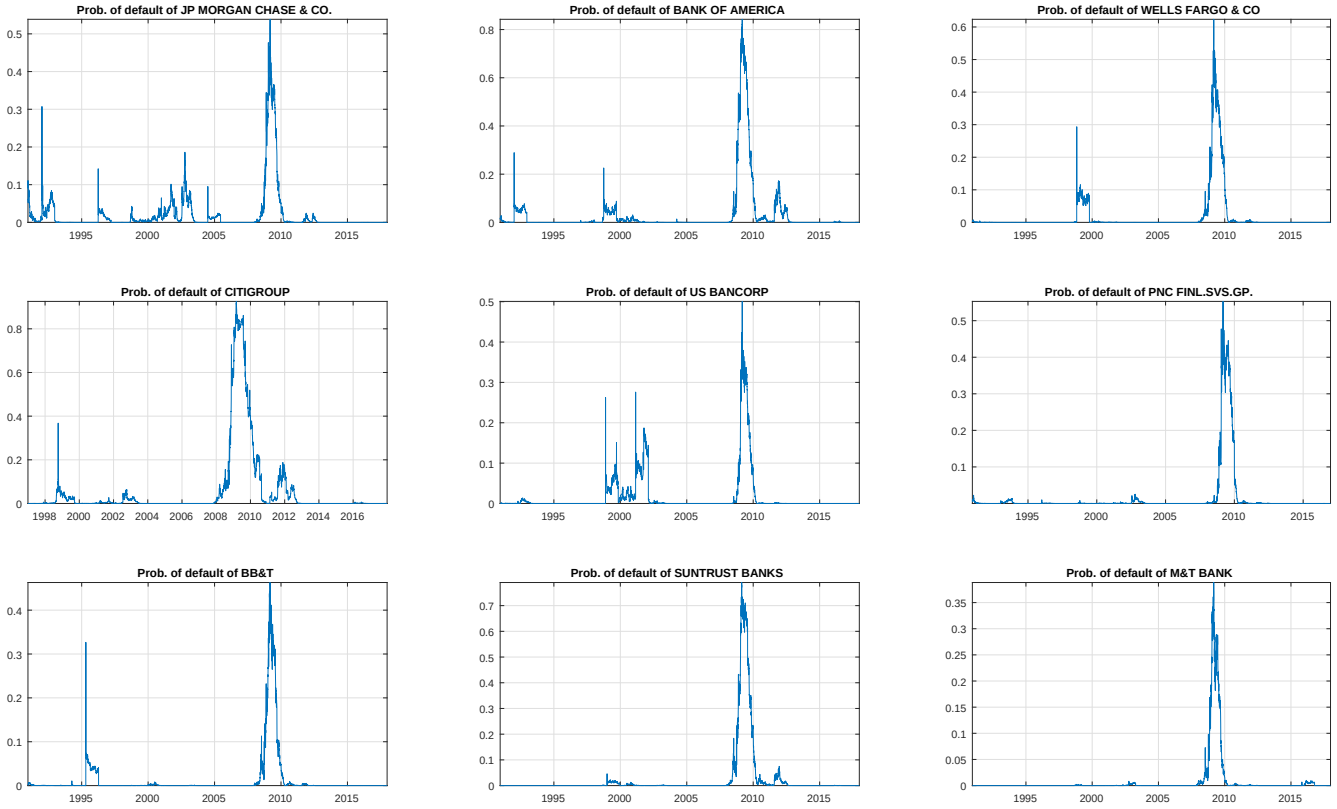


Figure 12: Time series Probability of Defaults of the 9 largest US banks - measured by Market Value - as of January 2018.

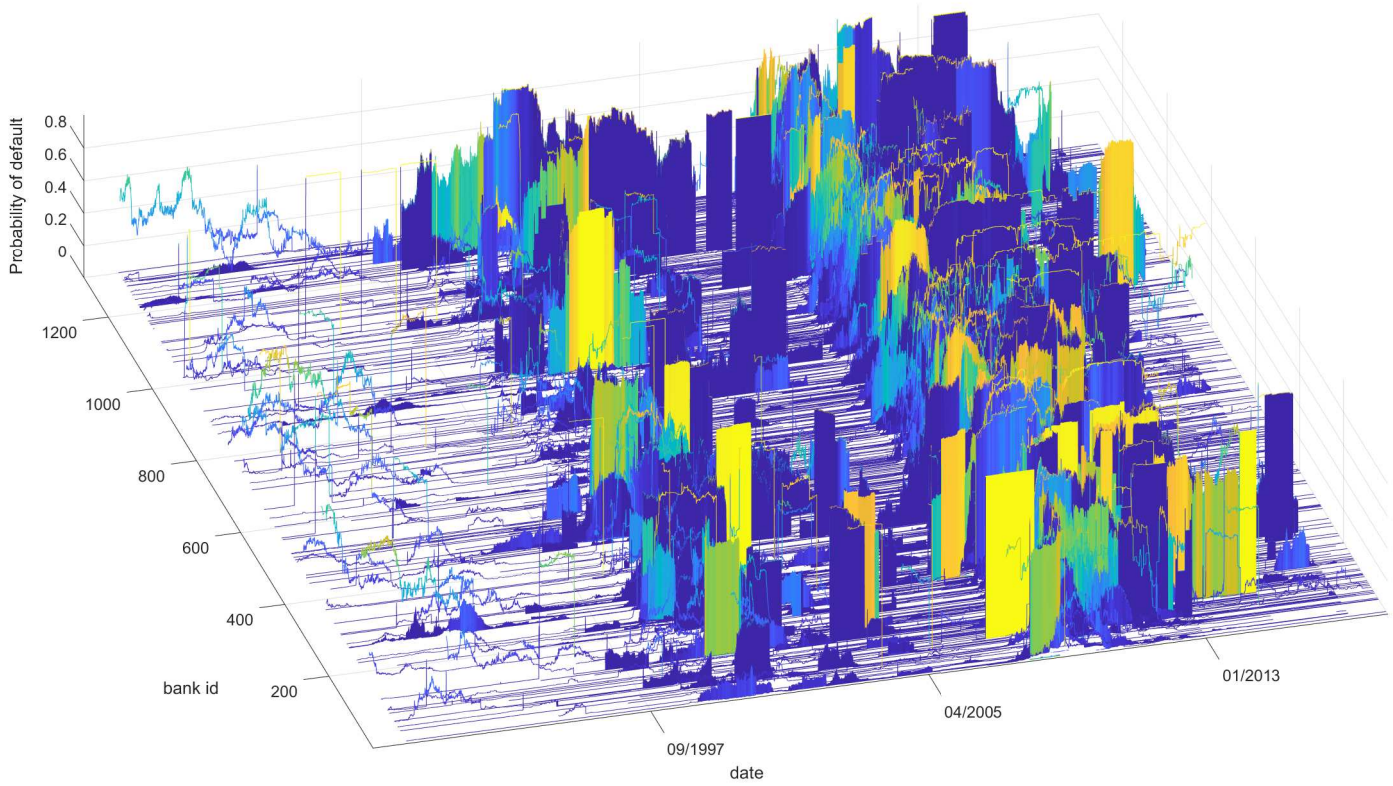
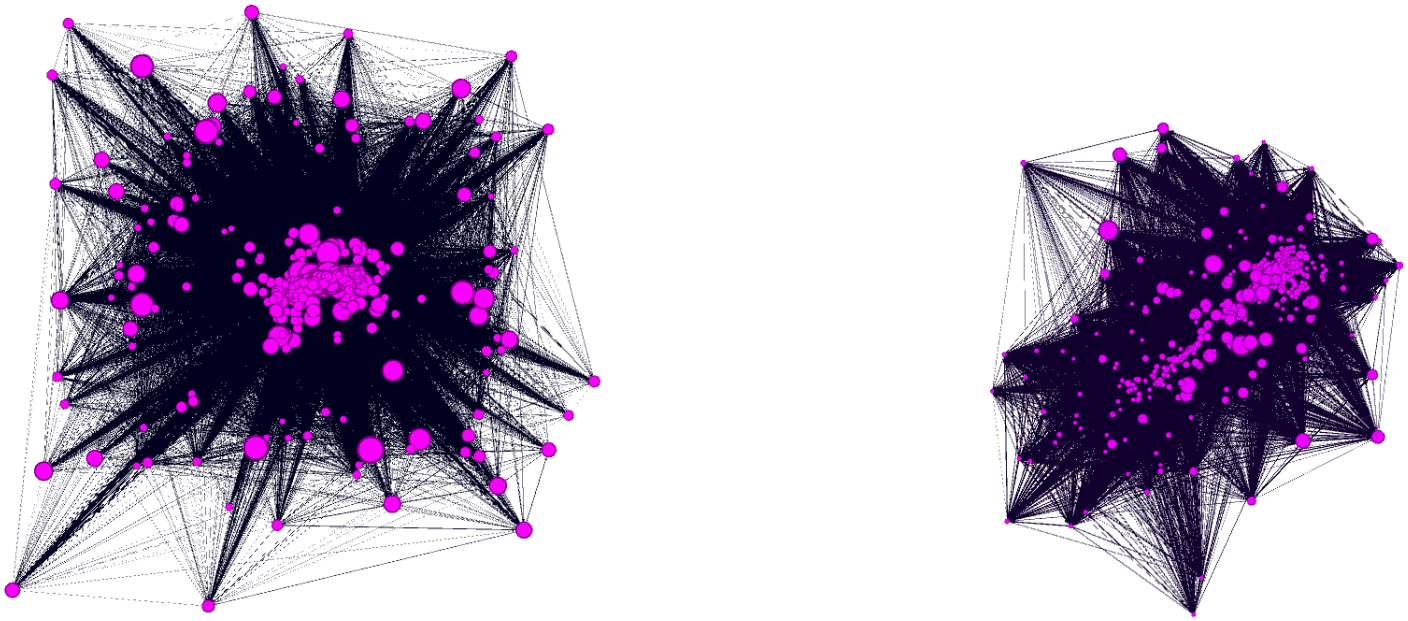


Figure 13: Joint behaviour of default likelihoods of all US banks included in the sample from 1990 until 2018. Periods of significant probability of default ($\geq$75%) are highlighted in yellow.



(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		V	
	POLS	**t-stat.**	**SAR (ML)**	**t-stat.**	**BSAR (Homosk)**	**t-stat.**	**BSAR (Heterosk)**	**t-stat.**	**BSDM (Heterosk)**	**t-stat.**
Leverage	0.329	15.4	0.306	14.56	0.305	14.25	0.241	2.27	0.057	1.18
Earnings per Share	0.003	1.85	0.003	1.89	0.003	1.855	0.000	0.27	0.002	1.34
Tobin's Q	-0.533	-3.7	-0.467	-3.36	-0.458	-3.28	0.0244	0.242	-0.449	-3.92
Return on Equity	-0.130	-26.	-0.123	-25.5	-0.123	-25.4	-0.175	-8.10	-0.182	-4.15
Non-performing Loans % total loans (NPL)	0.973	21.1	0.912	20.0	0.916	20.10	0.795	6.74		
W*NPL									1.186	7.81
Loan to Deposit ratio	0.016	3.46	0.015	3.31	0.016	0.638	0.002	0.63	0.006	1.68
size (market value)	-0.012	-1.7	-0.012	-1.68	-0.01	-1.31	-0.00	-1.3	-0.00	-2.7
ρ			0.603	122	0.593	4.79	0.396	4.79	0.377	3.75
Sigma squared	109.0		106.2		108.1		118.3		121.4	
Moran I's statistic			22.35							
R-squared	0.324		0.33		0.331		0.283		0.25	
Year FE	Y		Y		Y		Y		Y	
Bank FE	N		N		Y		Y		Y	
No. obs	8735		8735		8735		8735		8735	

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	Direct effect	Spillover	Total effect
JP MORGAN CHASE & CO.	2.20	0.40	1.00	0.61	1.61
BANK OF AMERICA	3.12	0.61	1.00	0.75	1.75
WELLS FARGO & CO	1.68	1.39	1.00	0.65	1.65
CITIGROUP	5.56	1.38	1.00	0.82	1.82
US BANCORP	1.59	1.76	1.00	0.86	1.86
PNC FINL.SVS.GP.	1.43	0.75	1.00	0.83	1.83
BB&T	1.26	0.60	1.00	0.77	1.78
SUNTRUST BANKS	2.46	0.84	1.00	0.43	1.43
M&T BANK	0.82	0.40	1.00	0.66	1.66
KEYCORP	3.37	0.39	1.00	0.67	1.67
FIFTH THIRD BANCORP	3.18	0.83	1.00	0.68	1.69
CITIZENS FINANCIAL GROUP	0.03	1.12	1.00	0.47	1.47
REGIONS FINL.NEW	4.36	0.77	1.00	0.80	1.80
CREDICORP	0.52	0.99	1.00	0.67	1.68
HUNTINGTON BCSH.	3.84	0.40	1.00	0.66	1.66
COMERICA	1.63	0.78	1.00	0.75	1.75
FIRST REPUBLIC BANK	0.03	0.69	1.00	0.79	1.79
SVB FINANCIAL GROUP	2.79	1.61	1.00	0.68	1.68
ZIONS BANCORP.	2.90	1.23	1.00	0.50	1.50
EAST WEST BANCORP	3.57	1.14	1.00	0.75	1.75
SIGNATURE BANK	0.93	0.64	1.00	0.54	1.55
FIRST HORIZON NATIONAL	2.16	0.91	1.00	0.64	1.64
PACWEST BANCORP	3.75	0.79	1.00	0.80	1.80
PEOPLES UNITED FINANCIAL	4.24	0.77	1.00	0.71	1.71
NEW YORK COMMUNITY BANC.	1.15	0.76	1.00	0.67	1.67
BANK OF THE OZARKS	0.62	0.63	1.00	0.80	1.80
BOK FINL.	2.65	1.16	1.00	0.64	1.64
CULLEN FO.BANKERS	0.59	1.32	1.00	0.50	1.50
WESTERN ALL.BANCORP.	8.16	1.17	1.00	0.59	1.59
COMMERCE BCSH.	0.28	1.32	1.00	0.24	1.25
SYNOVUS FINANCIAL	4.72	1.67	1.00	0.87	1.87
STERLING BANCORP	1.05	2.09	1.00	0.17	1.17
WEBSTER FINANCIAL	2.24	1.62	1.00	0.67	1.67
PINNACLE FINANCIAL PTNS.	3.40	0.70	1.00	0.71	1.71
SLM	4.09	1.75	1.00	0.87	1.87
PROSPERITY BCSH.	0.67	0.81	1.00	0.70	1.70
WINTRUST FINANCIAL	1.84	1.71	1.00	0.84	1.84
UMPQUA HOLDINGS	6.13	1.53	1.00	0.36	1.37
FNB	7.13	0.68	1.00	0.72	1.73
FIRST CTZN.BCSH.A	0.39	0.91	1.00	0.68	1.69
TEXAS CAPITAL BANCSHARES	1.05	0.80	1.00	0.52	1.52
BANKUNITED	0.02	0.66	1.00	0.69	1.70
INVESTORS BANCORP	6.02	0.40	1.00	0.72	1.72
HANCOCK HOLDING	1.65	0.65	1.00	0.81	1.81
TFS FINANCIAL	0.03	1.61	1.00	0.65	1.65
IBERIABANK	0.38	0.38	1.00	0.64	1.64
HOME BANCSHARES	0.83	0.46	1.00	0.65	1.65
ASSOCIATED BANC-CORP	1.79	5.68	1.00	0.62	1.66
CHEMICAL FINL.	0.84	1.32	1.00	0.77	1.77
MB FINANCIAL	2.63	0.41	1.00	0.41	1.42

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		BSDM FE	95% credibility set	
Direct effect						
Leverage	24**	-1	50	6	-8	23
Earnings per Share	0	0	0	0	0	1
Tobin's Q	2	-24	30	-45***	-75	-14
Return on Equity	-18***	-22	-11	-18***	-30	-8
NPL	79***	44	107			
W*NPL		0	0	119***	69	149
Loan to Deposit ratio	0	-1	2	1*	0	2
size (market value)	0	-1	0	-1***	-2	0
Indirect effect						
Leverage	17	2	42	3	-2	10
Earnings per Share	0	0	0	0	0	0
Tobin's Q	1	-16	15	-21**	-46	-6
Return on Equity	-12***	-22	-5	-9*	-20	-2
NPL	54**	22	108			
W*NPL		0	0	55**	21	105
Loan to Deposit ratio	0	0	1	0	0	1
size (market value)	0	-1	0	0*	-1	0
Total effect						
Leverage	41	-1	100	9	-11	36
Earnings per Share	0	0	1	0	0	1
Tobin's Q	4	-50	50	-66	-125	-19
Return on Equity	-30	-47	-17	-27	-52	-10
NPL	134	72	229			
W*NPL		0	0	173	101	258
Loan to Deposit ratio	0	-1	3	1	0	3
size (market value)	-1	-3	1	-1	-3	0

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 t-statistics.

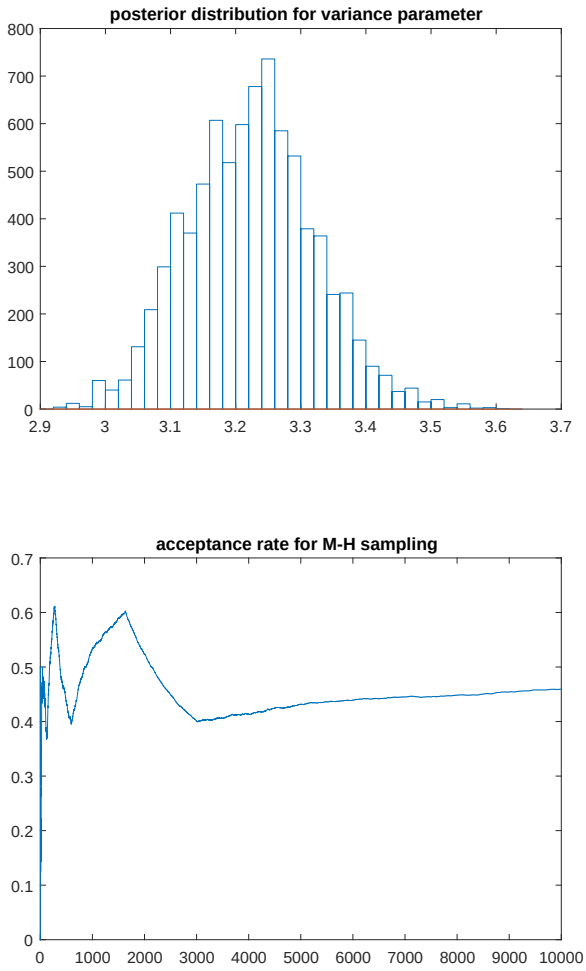
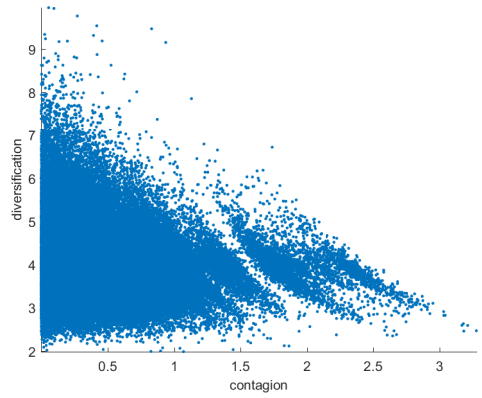
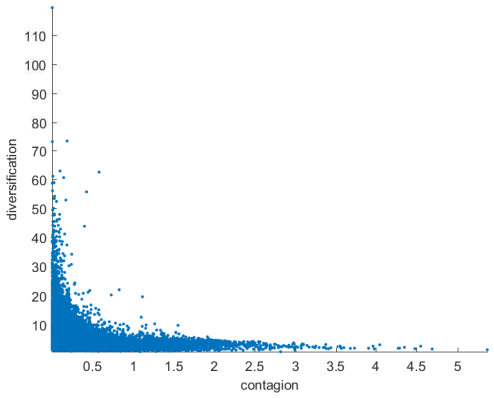


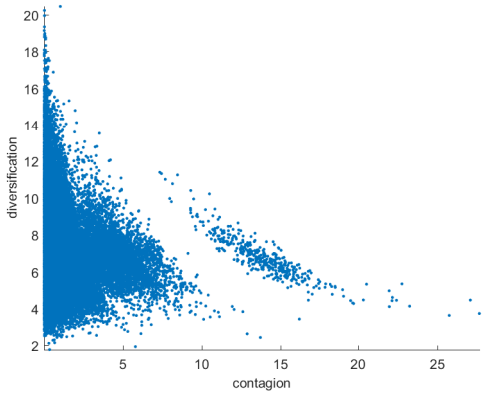
Figure 15: Model Estimation Diagnostics. Posterior distributions for the variance parameter and Metropolis Hastings algorithm acceptance rate.



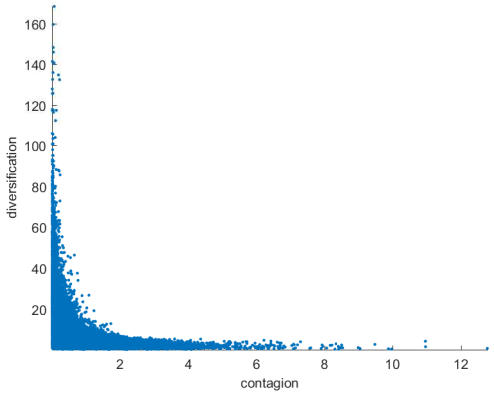
(a) Gamma - Low clustering



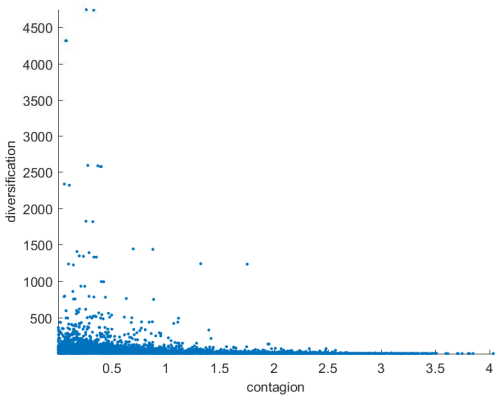
(b) Gamma - High clustering



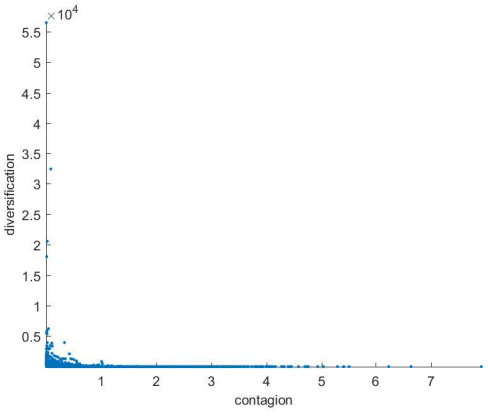
(c) Lognormal - Low clustering



(d) Lognormal - High clustering

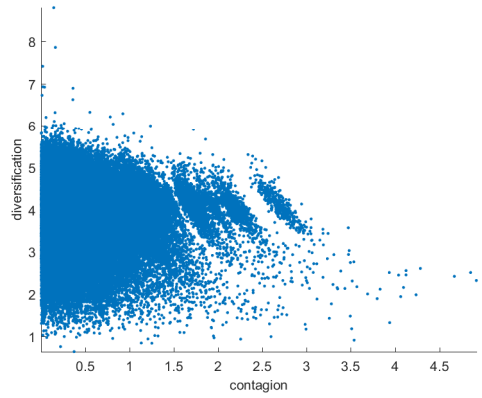


(e) Weibull - Low clustering

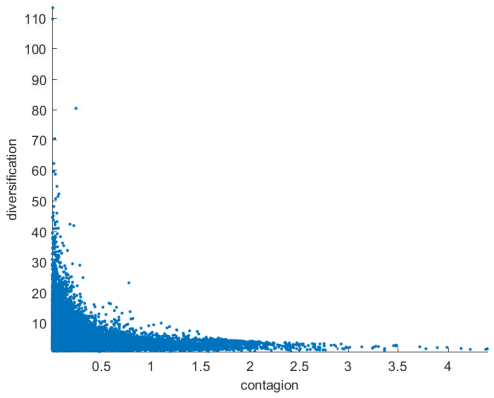


(f) Weibull - High clustering

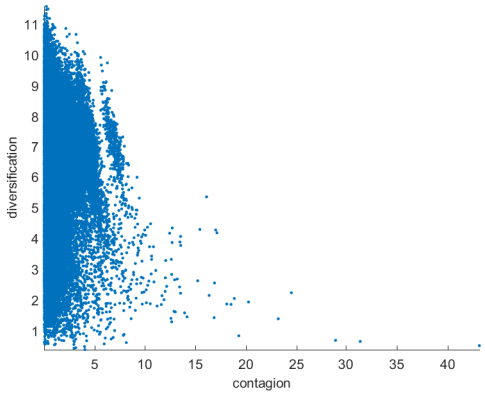
Figure 16: Simulation results. Relationship between diversification and contagion where $\tilde{W}$ is drawn from three candidate distance distributions - Gamma, Lognormal and Weibull and a Gaussian copula is fitted to the data with $\rho = 0.1$ and $\rho = 0.9$, representing low and high degrees of clustering, respectively.



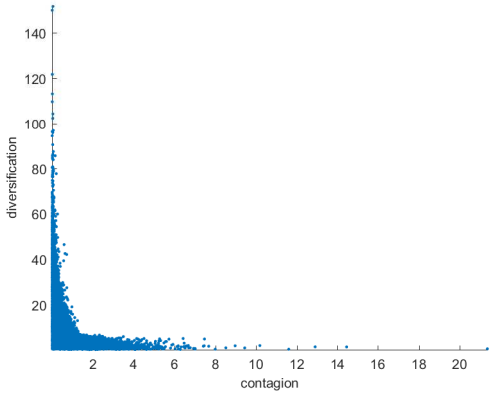
(a) Gamma - Low clustering



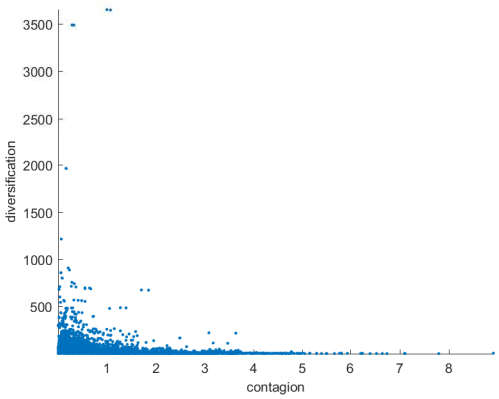
(b) Gamma - High clustering



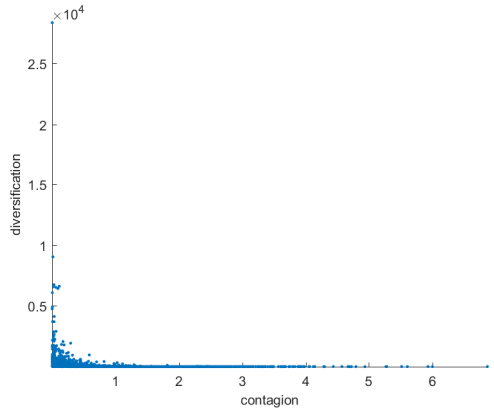
(c) Lognormal - Low clustering



(d) Lognormal - High clustering



(e) Weibull - Low clustering



(f) Weibull - High clustering

Figure 17: Simulation results. Relationship between diversification and contagion where $\tilde{W}$ is drawn from three candidate distance distributions - Gamma, Lognormal and Weibull and a Gumbel copula, representing low and high degrees of clustering, respectively.