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The role of contagion in the transmission of financial stress

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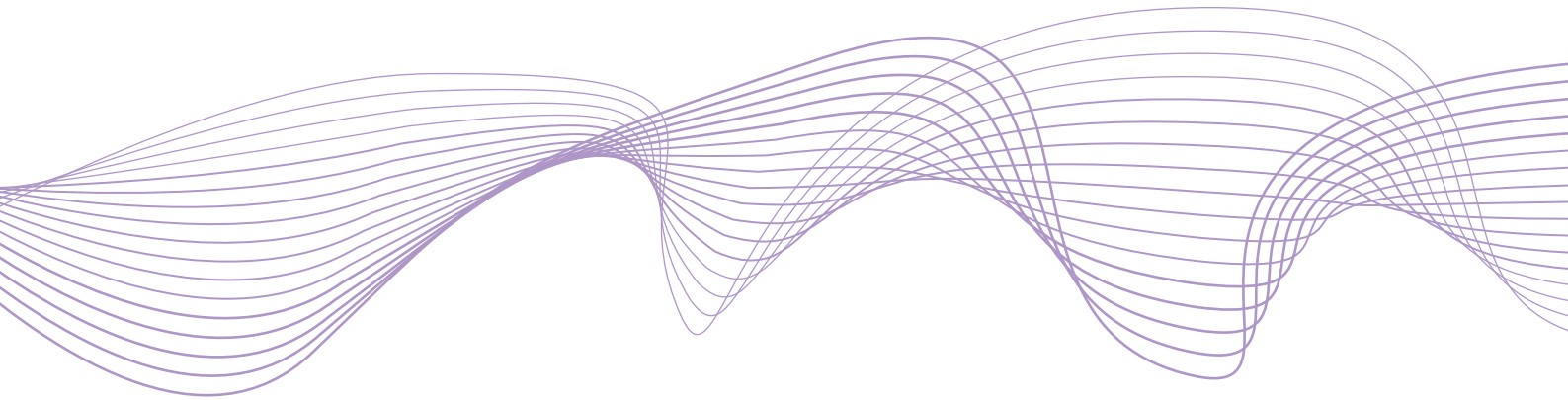

ESRB
European Systemic Risk Board
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Abstract

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.

JEL classification: E44, G01, C11, G21.

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

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 mechanisms of transferring risk from individual institutions to the financial markets at large lie at the heart of the financial system. Various important faults related to this process are highlighted by Acharya et al. (2012) that 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 Great Financial 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 that are outlined above and 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 and 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

[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).

other institutions, 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. Since the magnitude of contagion is determined by the spatial autoregressive coefficient in our model, testing the hypothesis that this parameter is statistically indifferent from zero can inform of 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-*à*-vis its own fundamentals. It is estimated that, on average an 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 component is disentangled from the remainder that can be interpreted as resulting from bank specific characteristics reflecting its 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 has 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. This suggests that the density of the banking network after the crisis decreased and that systemic risk due to contagion diminished.

The remainder of the paper proceeds as follows. Section 2 explains the econometric framework and provides the definitions and propositions that support the empirical results. Section 3 describes the data and estimation technique while section 4 discusses the main results and findings. Section 5 concludes.

2 Econometric Framework

I use a spatial autoregressive model to describe the dynamics of default probabilities between banks and in particular how financial stress can spillover from one institution to become systemically relevant. The first step to this exercise is to calculate the default probabilities that are unobserved in practice and should be estimated *a priori* with appropriate techniques. For this purpose I use Merton (1974) structural model and include an explanation of this approach in Appendix A [2].

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

[2]It should be stressed that such default probabilities are available through financial data providers albeit only for the largest financial institutions.

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

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

Where y_{it} denotes the probability of default observed across banks $i = \{1, ..., N\}$ at time $t = \{1, ..., T\}$, $\sum_{j=1,j\neq i}^{N_t} w_{ij,t} y_{jt}$ the spatially lagged dependent variable summarizing the endogenous interaction effects, where the interaction between banks is defined by a time-dependent spatial weights matrix W_t, with a generic element $w_{ij,t}$, whose construction is explained in the next section. Note that for each time period, a set of N_t probabilities of default are added to the regression. N_t represents the number of banks observed for each time period t that vary across time because some banks leave and others enter the panel. 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 autoregressive coefficient, and is at centre stage in our exercise. It is endogenously determined and reflects the dependence between the default likelihood of a given bank with the set of default probabilities of all other banks. Hence, a higher ρ suggests greater amplification effects of financial stress from the network, hitting individual banks. β 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.

2.0.1 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 explana-

tory 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 writting 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 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 counterpart 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.

2.0.2 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. 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, at 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 the principal diagonal of the spatial weights matrix 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, a 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' counterpart 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 counterpart risks. In an efficient markets framework, prices are better devices for capturing all information on the connection between institutions, reflecting potential losses deriving from counterpart risk but also liquidity dry-ups and common exposures.

3 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[3].

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

$$y = \rho W_t y + X^* \beta + \varepsilon, \tag{8}$$

$$\varepsilon \sim N(0, \sigma^2 V) \tag{9}$$

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 - $V_{ii} = v_i, i = 1, ..., n, V_{ij} = 0, i \neq j$.

[3]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.

Estimating specification 8 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

$$p(\Psi|\beta, \sigma, \rho) = (2\pi\sigma^2)^{-n/2}|A|exp\Big[-\frac{1}{2\sigma^2}(Ay - X\beta)'(Ay - X\beta)\Big]. \tag{10}$$

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

$$p(\theta|\Psi) \propto p(\Psi|\theta)p(\theta). \tag{11}$$

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 V.

$$p(\beta) \sim N(c, T), \tag{12}$$
$$p(\sigma^2) \sim IG(a, b), \tag{13}$$
$$p(\rho) \sim U(\lambda_{min}^{-1}, \lambda_{max}^{-1}), \tag{14}$$
$$p(r/v_i) \sim iid \quad \chi^2(r). \tag{15}$$

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 11 to get the posterior

$$p(\beta, \sigma^2, \rho, V|\Psi) \propto (\sigma^2 V)^{a^*+(k+2)/2+1}|A|exp\Big[-\frac{1}{2\sigma^2 V}[2b^* + (\beta - c^*)'(T^*)^{-1}(\beta - c^*)]\Big], \tag{16}$$

$$c^* = (X'X + T^{-1}(X'Ay + T^{-1}c), \tag{17}$$

$$T^* = (X'X + T^{-1})^{-1}, \tag{18}$$

$$a^* = a + n/2, \tag{19}$$

$$b^* = b + (c'T^{-1}c + y'A'Ay - c^*(T^*)^{-1}c^*)/2. \tag{20}$$

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.

3.0.1 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 8-9 and 12-15 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 sampler for the 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

$$c^* = (X'V^{-1}X + \sigma^2 T^{-1})^{-1}(X'V^{-1}(I_n - \rho W)y + \sigma^2 T^{-1}c), \tag{21}$$

$$T^* = \sigma^2(X'V^{-1}X + \sigma^2 T^{-1})^{-1} \tag{22}$$

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

$$a^* = a + n/2; \quad b^* = (2b + e'V^{-1}e)/2; \quad e = Ay - X\beta. \tag{23}$$

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)

$$p(\rho|\beta_1, \sigma_1^2, V_1) = |A|exp\Big[-\frac{1}{2\sigma^2 V}e'V^{-1}e\Big] \tag{24}$$

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 24 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

$$\rho^c = \rho_{h-1} + c \times N(0,1). \tag{25}$$

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 24. The acceptance probability is given by

$$\psi(\rho^c, \rho_{h-1}) = min\Big[1, \frac{p(\rho_{h-1}|\beta, \sigma)}{p(\rho^c|\beta, \sigma)}\Big]. \tag{26}$$

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 8 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 of banks 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 ?

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 5 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 6, 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 picked up in the data are the savings and loans crisis in the beginning of the 1990s, the dot.com bubble in the early 2000s and more significantly the US subprime mortgage crisis that resulted in the Great Recession. Figure 7 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[4], 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 1, 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, Moran (1948) I statistic, another measure of spatial autocorrelation is also statisti-

[4]independent and identically distributed

cally 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 of this baseline regression 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 0.39, 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 39 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 100bps in this figure is estimated to lead to a 80 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 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 achieved 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 1. 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 100bps in NPLs across the banking system induces a hike of 118 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 for heteroskedasticity of unknown form by allowing the variance covariance matrix to vary, while remaining agnostic about its functional form.

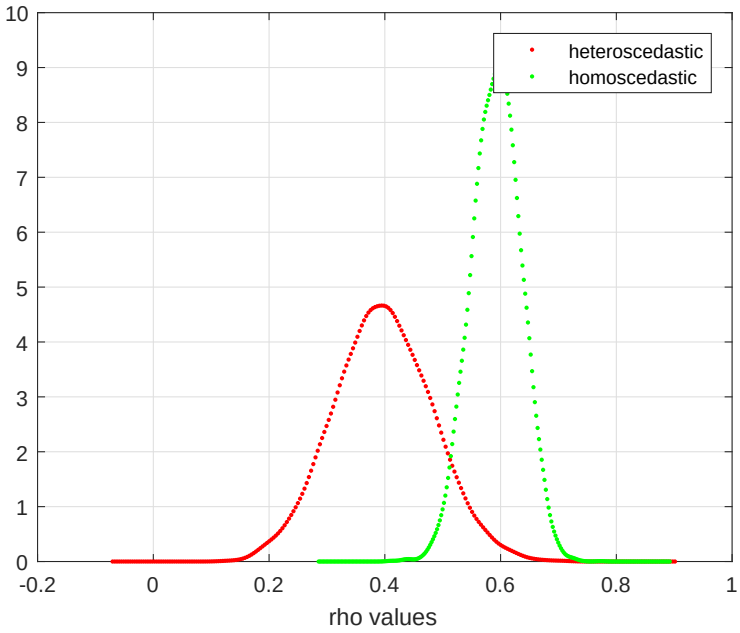


Figure 1: Full sample estimates of the posterior distribution of ρ

Figure 1 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. Albeit, it can also be seen in table 1 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.

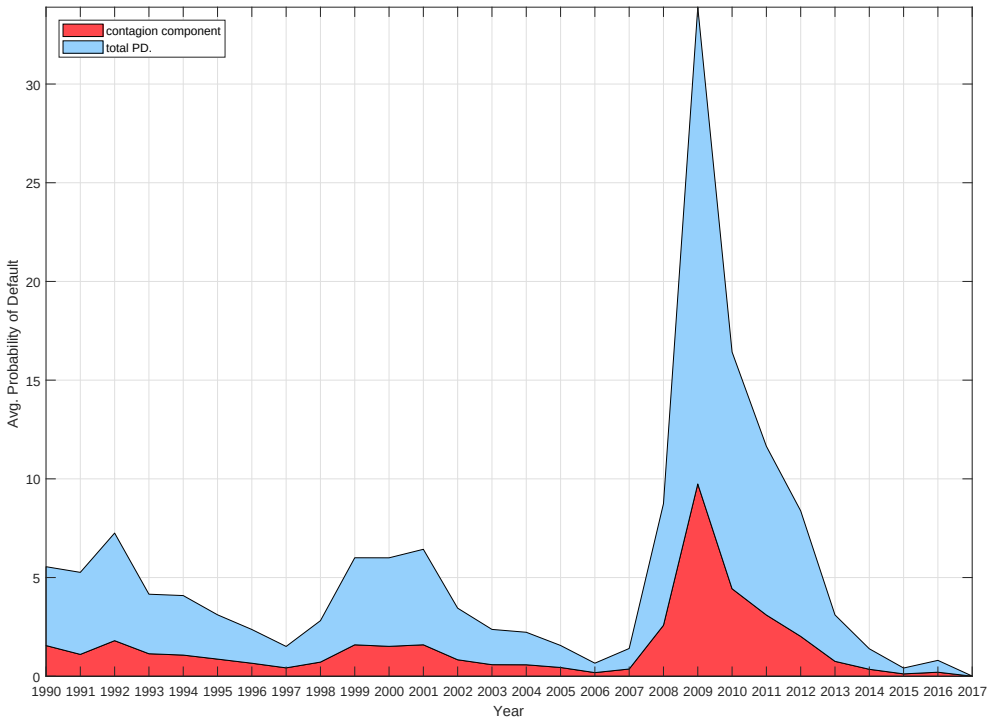
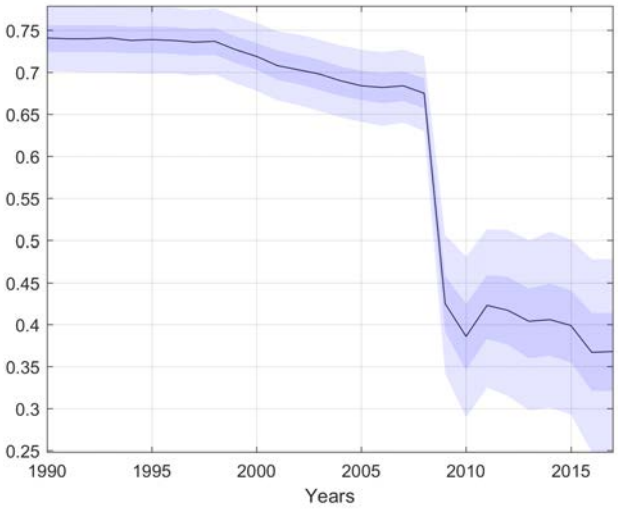


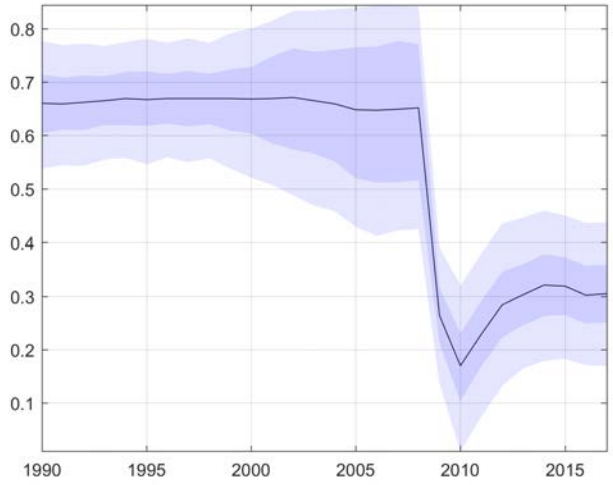
Figure 2: Decomposition of probabilities of default for US banks, 1990-2018. Red stacked line represents the proportion of the total observed probability of default due to contagion.

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 3: 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 3 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 varies between 0.10 and 0.25.

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 2 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 January, 2018 in column 1. Institutions in the table are sorted from largest to smallest in 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. The following columns (3-5) 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 17 bps. Whereas, a shock resulting in a 100bps hike of the probability of default of the most systemically significant bank will increase the probabilities of default across the board by a total of 107bps. Furthermore, evidence suggests that vulnerability to external shocks and systemic importance are not directly related to size. For instance, the bank showing 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 the most 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 30bps, of which 18bps 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 134bps. 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 173bps, of which 55bps 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 66bps of which 21bps 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 4 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 1.2, yielding a total effect that lies between 1 and 2.4, suggesting that shocks to bank fundamentals have powerful peer effects.

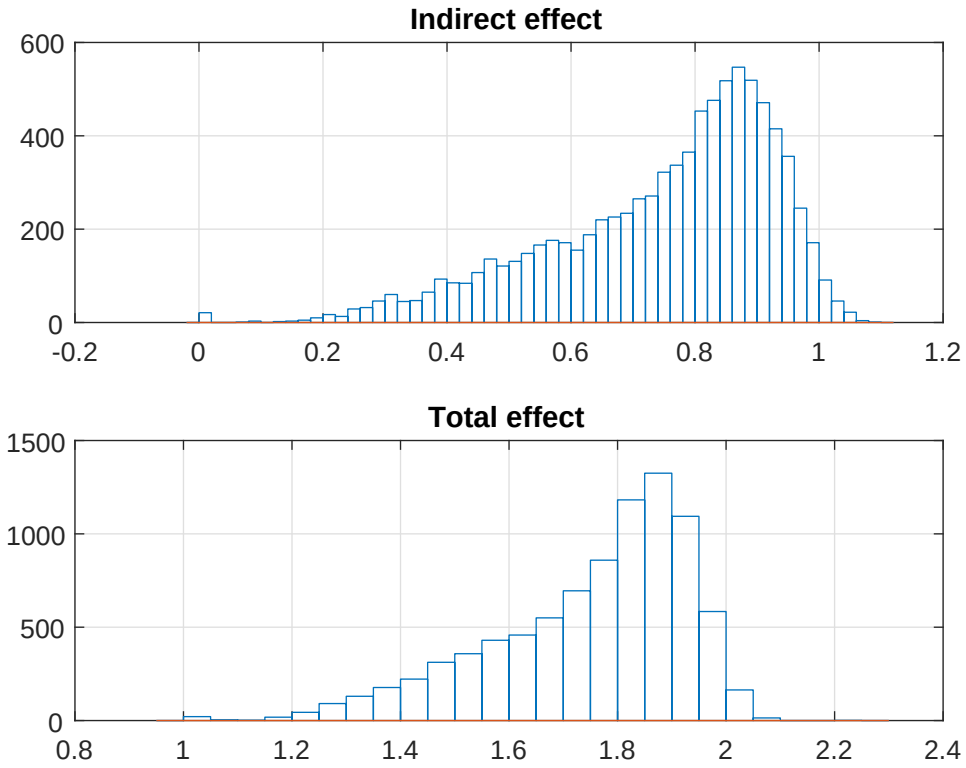


Figure 4: Posterior distribution of the direct, indirect and spillover effects estimated with from the heteroskedastic Bayesian spatial autoregressive model.

5 Conclusion

We have examined the evidence on contagion within the US banking system by studying a panel of default probabilities for a large number of 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 captured by a spatial econometric model that is estimated through Bayesian techniques. Several reasons make these models particularly insightful to study contagion. First, and most importantly, they allow for feedback effects between default likelihoods amongst banks, providing an analytical framework consistent with the definition of contagion put forward in the literature. Second, they handle panel data thus offering the possibility of purging unobserved bank specific and macrofinancial shocks that hit the economy together with contagion and yet are independent of it, mitigating omitted variable bias. Third, Bayesian techniques permit adequate mod-

elling of stochastic volatility, a major hurdle in the empirical literature in measuring contagion and to correct for the underlying bias.

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)[5] 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.

$$A = E + L \tag{27}$$

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 and estimates the market value and volatility of a firm's assets. Under the assumption that the value of liabilities are 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

$$E_t = max\{0, A_t - L\} \tag{28}$$

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 of 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.

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

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

$$E = AN(d_1) - Le^{-rT}N(d_2), \tag{29}$$

where

$$d_1 = \frac{ln(A/L) + (r + 0.5\sigma_A^2)T}{\sigma_A\sqrt{T}} \text{ and } d_2 = d_1 - \sigma_A\sqrt{T}. \tag{30}$$

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

$$\frac{dA}{A} = \mu_A dt + \sigma_A \varepsilon \sqrt{t} \tag{31}$$

where μ stands for average 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 asset 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

$$P(A_t < L) \tag{32}$$

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 32 Itô's general solution for the Stochastic Differential Equations written in equation 31 one gets

$$P(A_0 exp\{(\mu_A - \frac{\sigma_A}{2})t + \sigma_A\varepsilon\sqrt{t}\} \leq L) = P(\varepsilon \ \leq -d_1). \tag{33}$$

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

$$PD = 1 - N(d_1), \tag{34}$$

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

measure 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 [6] consists in estimating the market value of assets of a firm, therefore inferring how far each firm is from default.

[6] 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: Tables and Figures

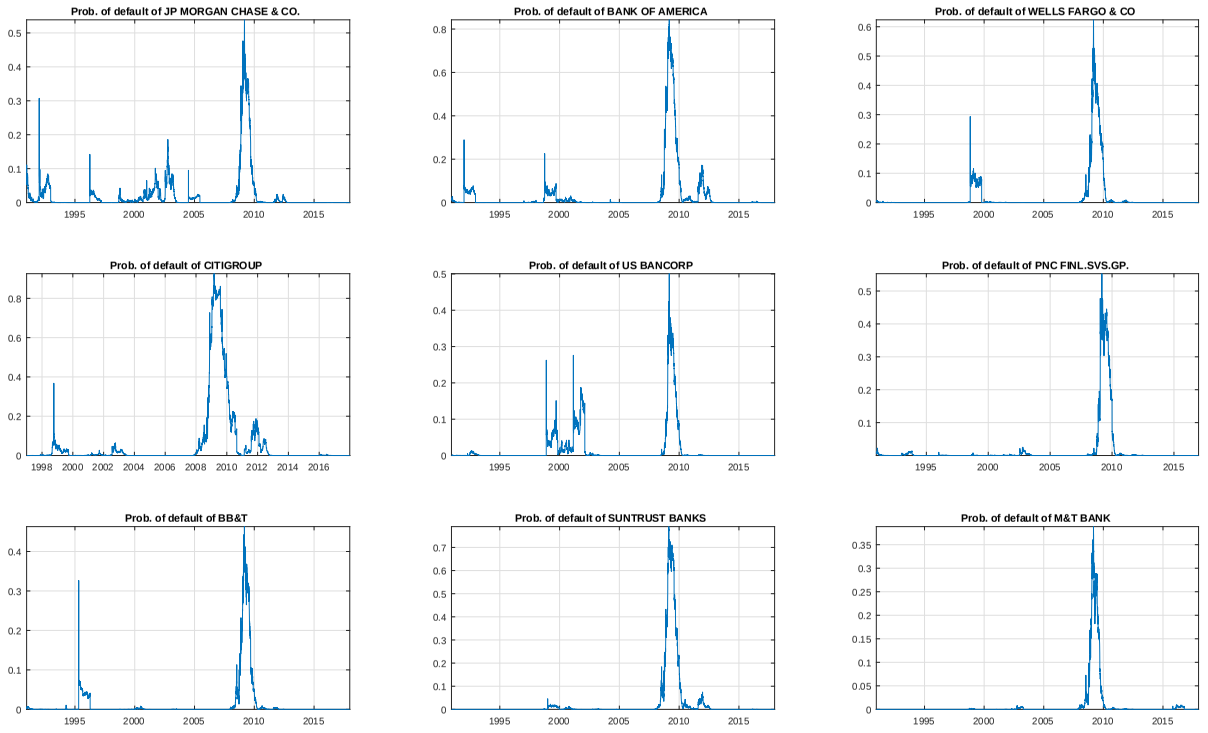


Figure 5: Time series Probability of Defaults of the 9 largest US banks - measured by Market Value - as of January 2018.

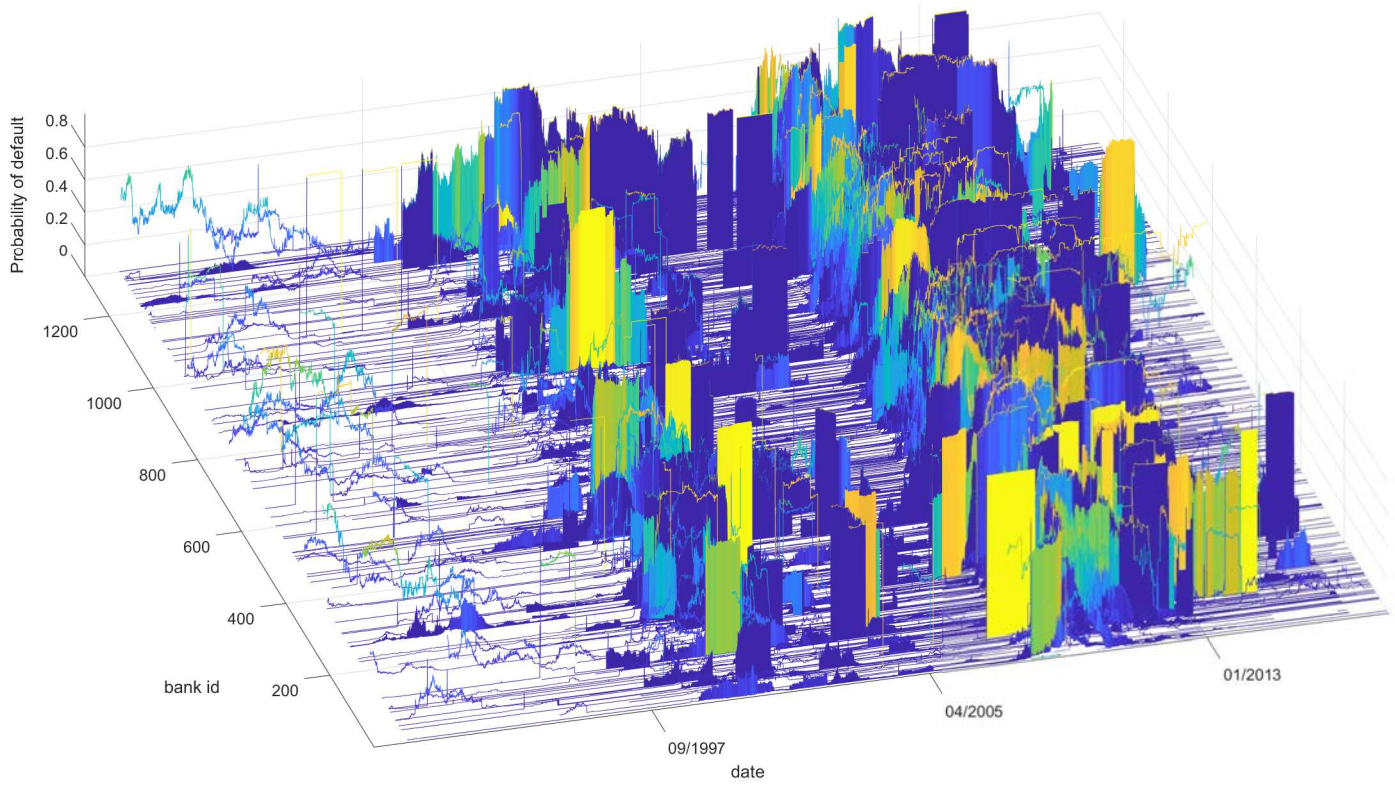
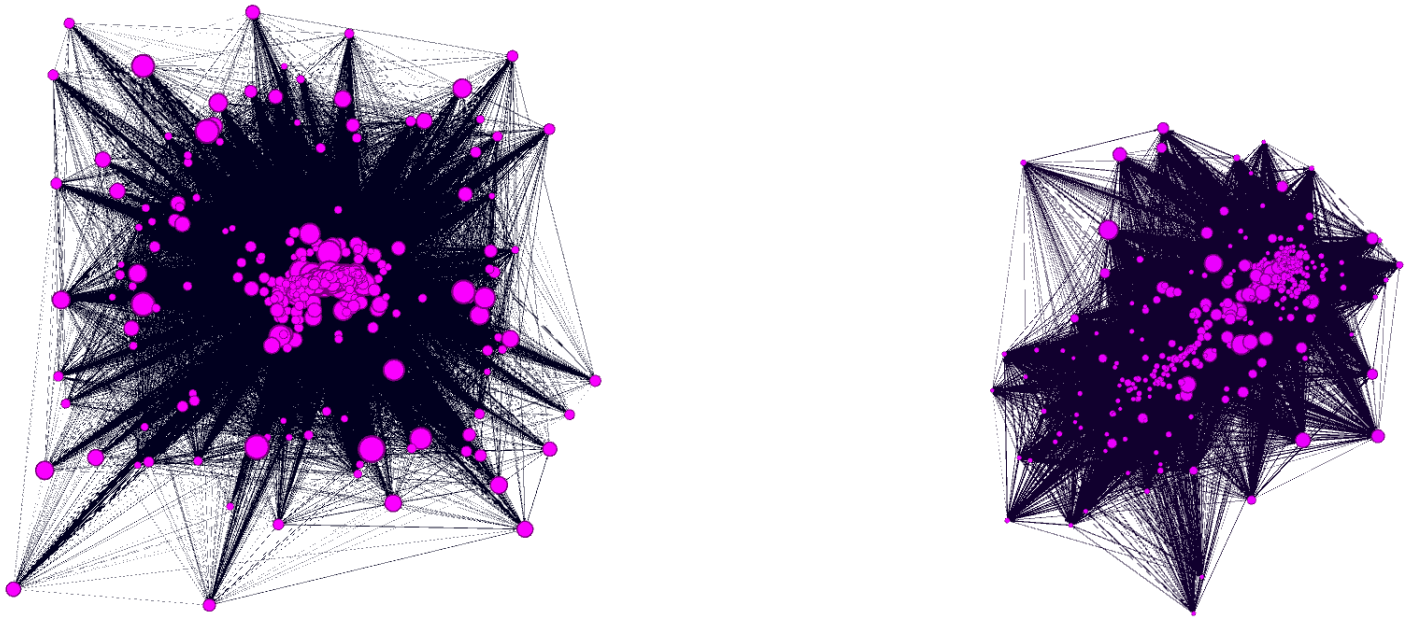


Figure 6: Joint behaviour of default likelihoods of all US banks included in the sample from 1990 until 2018. Periods of significant probability of default ($\geq$75%) are highlighted in yellow.



(a) US banking system network in 2006

(b) US banking system network in 2009

Figure 7: 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 1: **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 2: **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 3: **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.

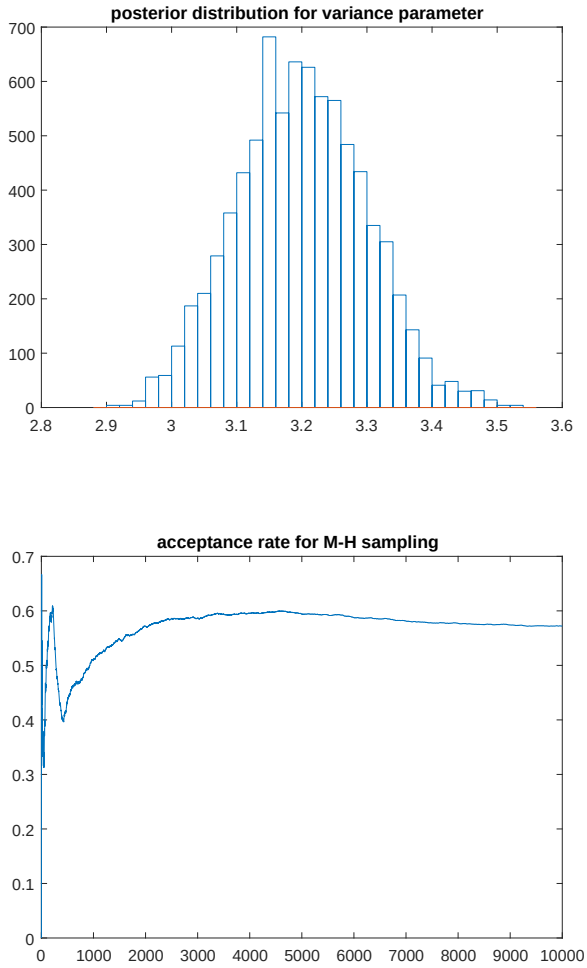


Figure 8: Model Estimation Diagnostics. Posterior distributions for the variance parameter and Metropolis Hastings algorithm acceptance rate.

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