Modelling Financial Contagion Using High Frequency Data

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Abstract

This paper develops a methodology for detecting and measuring contagion using high frequency data which disentangles continuous and discontinuous price movements. We demonstrate its finite sample properties using Monte-Carlo simulation, focusing on the empirically plausible parameter space. Decisions to extend the role of financial regulation around the world to the supervision of insurers post-GFC has been met with literature which supports both the systemic importance of insurers and contrasting evidence that insurers are rather the 'victims' of shocks transmitted via banks. We contribute to this debate by considering the time-varying evidence for contagion at both the firm level and the sectorial level impacts. A number of insurance companies exhibits bank-like characteristics. Our evidence for contagion effects from banks to the real economy, with similar impact from the insurers, supports the view that financial regulation on banks does need to be extended to the insurance sector.

Keywords: Factor model; Crisis transmission; Jumps; High frequency data.

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1 Introduction

Financial crises are characterized as 'fast and furious' (Kaminsky et al., 2003), and yet there is a surprisingly small literature using high frequency financial markets data to understand their transmission. Given the increasing availability of intra-day data for a variety of markets, and recent advances in applied financial econometrics demonstrating the improved estimation of volatility and price disruptions with high frequency data (see for example Andersen et al., 2007; Lahaye et al., 2011; Aït-Sahalia and Jacod, 2012), this leaves a void in the literature.

We extend the standard latent factor model approach to contagion used in lower frequency applications – such as Bekaert et al. (2014); Dungey and Renault (2018) – to the high frequency domain, building on the theoretical framework of Todorov and Bollerslev (2010) to disentangle the sensitivity to systematic diffusive and jump risks. The high frequency extension allows us to differentiate between the transmission of continuous and discontinuous components of the financial price process, allowing direct examination of the hypothesis that contagion may be transmitted mostly in the form of jumps; see Aït-Sahalia et al. (2015). While jumps occur in response to news shocks (Dungey et al., 2009; Andersen et al., 2007; Lahaye et al., 2011; Miao et al., 2013), the separate empirical identification of beta associated with jumps from continuous beta is relatively recent; see Todorov and Bollerslev (2010); Bollerslev et al. (2016); Alexeev et al. (2017).

Financial contagion is frequently modeled as "...a significant increase in market comovement after a shock" (Forbes and Rigobon, 2002, p.2223). However, no single definition of contagion is universally accepted. Forbes and Rigobon (2001) devotes an entire chapter to address its ambiguity and to establish a working definition of contagion. We follow Forbes and Rigobon (2001)'s definition of *shift-contagion*, as a shift in cross-asset linkages arising after a shock. This linkage can be measured by a number of different statistics, which in our case, are additional shocks transmitted from one firm to another, and the shock is taken as the Global Financial Crisis.

The difference in perception between contagion and spillover in the literature is tenuous (Rigobón, 2019). The former involves unusual transmissions of idiosyncratic shocks between

assets during crisis periods, whilst spillovers are contained in the usual relationships between assets – represented by beta in the CAPM framework. In the framework proposed in this paper, the idiosyncratic part of individual asset returns has both a continuous and discontinuous component, as per the model proposed by Todorov and Bollerslev (2010), and consequently, there are two potential channels of contagion – a continuous contagion effect and a discontinuous contagion effect. We demonstrate how to estimate these contagion effects, both continuous and discontinuous, during crisis periods. The discontinuous contagion effect is pertinent to jumps activity in the market, implying that unexpected news arrival are associated with potentially contagious events (as in Aït-Sahalia et al., 2015). The jump contagion parallels the lower frequency literature that tests for contagion during tail or outlier events; for example Favero and Giavazzi (2002); Bae et al. (2003); Boyson et al. (2010); Busetti and Harvey (2011).

We apply the framework to resolve the debate on the role of banks and insurers in promoting contagion during the Global Financial Crisis (GFC). We use high frequency data for the S&P 500 constituent stocks and focus on the period from 2003 to 2011. This period is characterised by stable market conditions during the first half of the sample, with the pronounced market turmoil surrounding the GFC in the second half. Decisions to extend the role of financial regulators around the world to the supervision of insurers post-GFC has been met with literature which supports both the systemic importance of insurers – such as Acharya and Richardson (2014) – and contrasting evidence that insurers are rather the 'victims' of shocks transmitted via banks, see Chen et al. (2014). Evidence for contagion effects from banks to insurers, with limited impact in the other direction, supports the view that financial regulation does not need to be extended to the insurance sector. We contribute to this debate by considering the time-varying evidence for contagion at a firm level from two key players in the recent crisis – AIG and Bank of America (BoA) – in the context of not only their impact on each other but also their sectorial level impact on the banking and insurance sectors and on the real economy firms in the S&P500. We examine all 500 firms included in the dataset,

but to be succinct, only present results for AIG and BoA as examples.[1]

Our first results confirm that estimates for the impact of discontinuous contagion are larger than for continuous contagion. The contagion impact from BoA to AIG is larger than the impact of contagion transmitted from AIG to BoA. At an industry level, although Bank of America has stronger linkages with both banking and insurance sectors prior to the crisis compared to AIG, the linkages originated from AIG to other banking and insurance firms demonstrate clear upward trends leading up to the GFC. Therefore, at the sectorial level, we find stronger evidence of contagion originated from AIG rather than from BoA. One potential explanation for this is that institutions such as AIG present a special case: McDonald and Paulson (2015) argue that AIG behaved more like a bank during this period. However, other evidence, in Ghosh and Hilliard (2012) strongly supports that shocks to AIG are quickly absorbed to other insurance companies, consistent with its role as a leader in that sector.

Furthermore, we estimate the contagion coefficients from AIG and BoA to the real economy firms in the sample. Results show that although the range of impacts across the firms is wider for the bank-originated contagion in both the continuous and discontinuous cases, it is not clear that the impact of contagion from the bank BoA is greater than the impact of contagion from the insurance company AIG. In both cases, the median and range of estimates is higher and wider in the pre-crisis period, but both drop and contract in the period after 2008. The impact on real economy firms from either source is, on average, not distinctly different from the two sources.

The remainder of this article is organized as follows. Section 2 proposes the estimation procedure of a simple bivariate contagion model using high frequency financial econometric techniques. We conduct Monte Carlo simulation in Section 3 to examine the finite sample performance of the estimation method. Section 4 studies the application to contagion from a number of key banks and insurance companies in the US during the period of 2003-2011 using the proposed model and provides evidence of the relative impact of discontinuous and continuous contagion effects. Section 5 concludes the paper.

[1] In Supplementary Appendix, we include results for Prudential Financial and Citigroup. Additional examples are available upon request.

2 Contagion Model in High Frequency

Our modeling framework combines the existing factor model approach to detecting contagion effects in the lower (generally daily or monthly) frequency data in Bekaert et al. (2005), Dungey and Martin (2007), Bekaert et al. (2014) with techniques in high frequency intra-day financial econometrics for measuring systematic risk in Todorov and Bollerslev (2010). Following the recent literature, we assume the return on any asset is a combination of a continuous Brownian process with time-varying volatility and a discrete jump component. We consider the high frequency intra-day log-return on asset j, r_j, which responds to both the continuous and jump movements in the market portfolio, r_0. The market return is common to all assets, but each individual asset also contains idiosyncratic Brownian and jump components. These assumptions lead to the following process for r_j:

$$r_{j,t} = \beta_j^c \, r_{0,t}^c + \beta_j^d \, r_{0,t}^d + f_{j,t}^c + f_{j,t}^d, \tag{1}$$

where $r_{0,t}^c$ and $r_{0,t}^d$ refer to the continuous and discontinuous (jump) components of the market return, respectively, and $f_{j,t}^c$ and $f_{j,t}^d$ represent the idiosyncratic continuous and discontinuous components of asset j. The market beta coefficients, β_j^c and β_j^d, capture the response of $r_{j,t}$ to market return $r_{0,t}$, which can be consistently estimated using the approach in Todorov and Bollerslev (2010) under some fairly general assumptions; for applications of this approach see Bollerslev et al. (2016) and Alexeev et al. (2017).

The latent factor model approach to detecting contagion augments a CAPM framework during crises with unanticipated transmissions between the idiosyncratic components of the model. These represent new channels of shock transmission which occur during crisis, as for example in Dungey and Martin (2007). The approach is supported by theoretical works in network finance where, during periods of stress, linkages between markets are either newly formed or broken, see Gai and Kapadia (2010) and Acemoglu et al. (2015). We adapt this approach to the high frequency environment by allowing asset j to be affected by movements from another asset, i, during periods of stress. We emphasise that these movements are not

part of the common component. Moreover, we allow both a continuous and jump aspect of this potential transmission. This can be captured by augmenting equation (1) with a contagion effect:

$$r_{j,t} = \beta_j^c \, r_{0,t}^c + \beta_j^d \, r_{0,t}^d + f_{j,t}^c + f_{j,t}^d + \delta_j^c \, f_{i,t}^c + \delta_j^d \, f_{i,t}^d, \tag{2}$$

where the loadings δ_j^c and δ_j^d denote the effect of the continuous and discontinuous movements in asset i on asset j, respectively. Note that the underlying assumption of equation (2) is that the direction of contagion effect is from asset i to asset j.[2]

If crises are transmitted through jump activities only, we would expect $\delta_j^c = 0$ and $\delta_j^d \neq 0$ during periods of stress. In the ideal case, the idiosyncratic components of any pair of asset returns are completely independent during non-crisis periods, and the linkage between asset returns occurs only due to the common market factor. We, however, acknowledge that this assumption may be too restrictive for an empirical application. Assets can be correlated via channels other than the market factor during non-crisis periods, such as industry factor, geographical factor, etc. To control for these effects, we estimate model (2) in non-crisis periods to establish the baseline values of δ^c and δ^d, which would, in turn, represent the existing linkage under non-crisis conditions. Thus, the deviations in δ^c and δ^d during crisis periods from the baseline values represent the continuous and discontinuous contagion effects. We estimate the two coefficients of interest δ_j^c and δ_j^d using an analog of Frisch-Waugh theorem (Frisch and Waugh, 1933; Lovell, 1963), by first partialling out the market influence $\beta_j^c r_{0,t}^c + \beta_j^d r_{0,t}^d$ from both $r_{j,t}$ and $r_{i,t}$, followed by estimation of the contagion effect from asset i to asset j in the second stage.

2.1 Estimation of Betas

We estimate the market betas of each individual asset i using the methodology of Todorov and Bollerslev (2010). It is assumed that the log-price of any asset $p_{i,t}$ follows a continuous-time jump diffusion process. Without loss of generality, for any asset i, $i = 1, 2, \ldots, N$, we

[2] In our empirical section, we use financial institutions that received the highest amounts of government bailout as the originators of contagion, or the "culprits".

have

$$r_{i,t} \equiv \mathrm{d}p_{i,t} = \gamma_{i,t}\,\mathrm{d}t + \sigma_{i,t}\,\mathrm{d}W_{i,t} + \kappa_{i,t}\,\mathrm{d}\mu_{i,t}, \qquad t \in (0, T], \tag{3}$$

where $\gamma_{i,t}$ is the drift term, $\sigma_{i,t}$ denotes the spot volatility, $W_{i,t}$ is a standard Brownian motion for asset i, $\kappa_{i,t}$ represents the size of jump at time t, and $\mu_{i,t}$ is a counting process for the discrete jump component. The jump measure $\mathrm{d}\mu_{i,t}$ is such that $\mathrm{d}\mu_{i,t} = 1$ if there is a jump in $p_{i,t}$ at time t, and $\mathrm{d}\mu_{i,t} = 0$ otherwise. We restrict our analysis to finite jump activity only.

The market return $r_{0,t}$ can be decomposed in a way similar to (3) as:

$$r_{0,t} = \gamma_{0,t}\,\mathrm{d}t + \sigma_{0,t}\,\mathrm{d}W_{0,t} + \kappa_{0,t}\,\mathrm{d}\mu_{0,t}. \tag{4}$$

Consider the simplest contagion model with one originating asset. For ease of notation, we refer to the originating asset as asset 1, and use subscript 1 to denote all of the parameters in its data generating process (DGP), and subscript j for the recipient assets hereafter, $j = 2, 3, \ldots, N$. Following the DGP of the market return (4), the factor representations for the origin of contagion, asset 1, and the recipient of contagion, asset j, are as follows:

$$r_{1,t} = \gamma_{1,t}\,\mathrm{d}t + \beta_1^c\,\sigma_{0,t}\,\mathrm{d}W_{0,t} + \beta_1^d\,\kappa_{0,t}\,\mathrm{d}\mu_{0,t} + e_{1,t}, \tag{5}$$

$$r_{j,t} = \gamma_{j,t}\,\mathrm{d}t + \beta_j^c\,\sigma_{0,t}\,\mathrm{d}W_{0,t} + \beta_j^d\,\kappa_{0,t}\,\mathrm{d}\mu_{0,t} + e_{j,t} + \delta_j^c\sigma_{1,t}\,\mathrm{d}W_{1,t} + \delta_j^d\kappa_{1,t}\,\mathrm{d}\mu_{1,t}, \tag{6}$$

where we use residual terms $e_{1,t} = \sigma_{1,t}\,\mathrm{d}W_{1,t} + \kappa_{1,t}\,\mathrm{d}\mu_{1,t}$, and $e_{j,t} = \sigma_{j,t}\,\mathrm{d}W_{j,t} + \kappa_{j,t}\,\mathrm{d}\mu_{j,t}$, $j = 2, 3, \ldots, N$, to denote collectively the idiosyncratic movements in each individual asset, including both the continuous and discontinuous components.

The beta coefficients in equation (5) and (6) can be represented as ratios of multi-power covariations of the continuous or discontinuous components between $r_{i,t}$ and $r_{0,t}$, $i = 1, 2, \ldots, N$. The continuous beta utilizes quadratic covariations of the continuous component,

$$\beta_i^c = \frac{[r_i^c, r_0^c]_t^2}{[r_0^c, r_0^c]_t^2}, \qquad \text{where} \quad [r_i^c, r_0^c]_t^2 = \beta_i^c \int_0^t \sigma_{0,s}^2 \mathrm{d}s. \tag{7}$$

Estimating the jump beta requires higher order power functions as shown by Todorov and

Bollerslev (2010). The covariations of the discontinuous component $[r_i^d, r_0^d]_t^{2\tau} = (\beta_i^d)^\tau \sum_{0<s\leq t} \kappa_{0,s}^{2\tau} = (\beta_i^d)^\tau [r_0^d, r_0^d]_t^{2\tau}$ lead to an expression for β_i^d as

$$\left|\beta_i^d\right| = \left(\frac{\left|\sum_{s\leq T} \text{sign}\{r_{i,s} r_{0,s}\} |r_{i,s} r_{0,s}|^\tau\right|}{\sum_{s\leq T} |r_{0,s}|^{2\tau}} \right)^{\frac{1}{\tau}}, \qquad \tau > 1. \tag{8}$$

We usually set $\tau \geq 2$, as the continuous Brownian component does not have any impact for high values of τ asymptotically. The estimators provided by Todorov and Bollerslev (2010) are the discrete-time sample counterparts of equations (7) and (8). For ease of notation, we still use T to denote the total number of observations in the discrete-time case, and hence $t = 1, 2, \ldots, T$. In empirical applications estimation is usually implemented with non-overlapping windows, in which case the value of T differs with the chosen window size.

For discretely observed asset returns, $r_{i,t} = p_{i,t} - p_{i,t-1}$, we set a threshold

$$\boldsymbol{u}_T = (\theta_0/T^\omega, \theta_1/T^\omega, \ldots, \theta_N/T^\omega),$$

with $\omega \in (0, \frac{1}{2})$ and θ_i a multiple of the estimated local volatility for asset i. Specifically, we use the bipower variation of Barndorff-Nielsen and Shephard (2004) to set θ_i,

$$\theta_i = 3 \left(\frac{\pi}{2} \sum_{s=1}^{T-1} |r_{i,s}|\, |r_{i,s+1}| \right)^{1/2}, \qquad i = 1, \ldots, N. \tag{9}$$

Those observations that satisfy the condition $|\mathbf{r}_t| = (|r_{0,t}|, |r_{1,t}|, \ldots, |r_{N,t}|) \leq \boldsymbol{u}_T$ are classified as continuous price movements, where the constant 3 in equation (9) implies that the price movements that are higher than three times of the estimated spot volatility are implicitly treated as jumps.[3] The equivalent representation of the continuous beta from (7) is a discrete sample counterpart

$$\hat{\beta}_i^c = \frac{\sum_{s=1}^T r_{i,s}\, r_{0,s}\, \mathbb{1}_{\{|\mathbf{r}_s| \leq \mathbf{u}_T\}}}{\sum_{s=1}^T (r_{0,s})^2\, \mathbb{1}_{\{|\mathbf{r}_s| \leq \mathbf{u}_T\}}}, \quad \text{for} \quad i = 1, \ldots, N, \tag{10}$$

[3] Here $\mathbf{r}_t$ and $\boldsymbol{u}_T$ are vector representations, with each element corresponding to individual asset ($i = 0, 1, ..., N$). Throughout the paper, we use bold font to denote vector representations.

where $\mathbb{1}$ is the indicator function. Similarly, the discrete-time estimator of β_i^d is

$$\hat{\beta}_i^d = \text{sign}\left\{\sum_{s=1}^{T} \text{sign}\{r_{i,s}\, r_{0,s}\}|r_{i,s}\, r_{0,s}|^{\tau}\right\}\left(\frac{\sum_{s=1}^{T} \text{sign}\{r_{i,s}\, r_{0,s}\}|r_{i,s}\, r_{0,s}|^{\tau}}{\sum_{s=1}^{T}(r_{0,s})^{2\tau}}\right)^{\frac{1}{\tau}}, \tag{11}$$

where $i = 1, \ldots, N$ and $\tau \geq 2$. Todorov and Bollerslev (2010) show that the estimators in (10) and (11) are consistent as the sampling frequency increases, and derive the corresponding central limit theorems for these estimators for statistical inference. For the finite sample properties of the proposed estimators see the simulation results in Alexeev et al. (2017).

2.2 Test for Jumps

We identify jumps using the test proposed in Lee and Mykland (2008) (henceforth LM), which has the advantage of pinpointing the within-day timing of the jumps. The intuition behind the LM test is rather straightforward. Under the assumption (3) that the asset price is a combination of a continuous Brownian component with time-varying volatility and discrete jumps, the standardized return, $|r_{i,t}|/\sigma_{i,t}$, should have a standard normal distribution if there is no jump. Therefore, the LM test relies on extreme values in $|r_{i,t}|/\sigma_{i,t}$ to detect the existence of jumps. In order to construct the test statistic, we estimate spot volatility, $\sigma_{i,t}$, using K observations prior to $r_{i,t}$,

$$\hat{\sigma}_{i,t} = \left(\frac{1}{K-2}\sum_{j=t-K+1}^{t-1}|r_{i,j}||r_{i,j-1}|\right)^{1/2}, \qquad t = K+1, \ldots, T, \tag{12}$$

where $K = \sqrt{M \times 252}$ as suggested in Lee and Mykland (2008), and M is the number of observations within one trading day. The estimator in (12) of the local volatility is robust to jumps (Barndorff-Nielsen and Shephard, 2004). The test statistic given by $LM_{i,t} = |r_{i,t}|/\hat{\sigma}_{i,t}$, $t = K+1, \ldots, T$, follows a standard Gumbel distribution under the null hypothesis of no jump at time t. The critical value has the following form

$$LM_{crit} = \frac{\zeta}{c\sqrt{2\ln M}} + \frac{\sqrt{2\ln M}}{c} - \frac{\ln(4\pi) + \ln(\ln M)}{2c\sqrt{2\ln M}}. \tag{13}$$

where $c = \sqrt{2/\pi}$ and $\zeta = -\ln(-\ln(1-\alpha))$, α denotes the daily significance level.[4] We conclude that there is a jump at the t-th observation if $|r_{i,t}| / \hat{\sigma}_{i,t} > LM_{crit}$. Following the literature, the size of the jump is estimated by $r_{i,t}$, as the jump will dominate the continuous diffusive component if it exists. Therefore, given a discretely observed return series $r_{i,t}$, $t = 1, 2, \ldots, T$, the decomposition between continuous and discontinuous price movements is implemented as $\hat{r}^c_{i,t} = r_{i,t}$ if there is no jump at time t, otherwise $\hat{r}^d_{i,t} = r_{i,t}$.

One of the main advantages of the Lee and Mykland (2008) approach over other existing jump tests is that, it is able to detect the exact timing of the jump at intra-day level, and thus the size and the number of jumps within each estimation window (i.e. one trading day or a month, etc.). This suits our purpose in estimating the contagion model (2) by separating the continuous and discontinuous components at the same frequency as the intra-day observations. In addition, Dumitru and Urga (2012) conduct a comprehensive Monte Carlo simulation and find that the LM test performs very well in terms of both size and power across sampling frequency, jump intensity and jump size.

2.3 Estimation of the Contagion Effects

The Frisch-Waugh Theorem (or Frisch-Waugh-Lovell theorem) by Frisch and Waugh (1933) and further generalized by Lovell (1963) states a very desirable property of least square estimates in the linear regression framework. It breaks down a multiple regression into a two-step projection procedure while obtaining exactly the same coefficient estimates. Our approach of estimating the contagion coefficients employs a similar reasoning as the Frisch-Waugh Theorem, which we state below.

Theorem 1 (Frisch-Waugh Theorem) *Consider a multiple linear regression*

$$y_t = X'_{1,t}\phi_1 + X'_{2,t}\phi_2 + \varepsilon_t,$$

where $X_{1,t} = (x_{11,t}, x_{12,t}, \ldots, x_{1k_1,t})'$ *and* $X_{2,t} = (x_{21,t}, x_{22,t}, \ldots, x_{2k_2,t})'$ *are* $k_1 \times 1$ *and* $k_2 \times 1$ *vectors of explanatory variables, and* ϕ_1 *and* ϕ_2 *are the corresponding conformable vectors of coefficients.*

[4] Gilder et al. (2014) point out that there is an error in the original paper by Lee and Mykland (2008), where the constant 4 in the last term is omitted. Hence we use (13) instead of the original critical value given by Lee and Mykland (2008) in this paper.

The least square estimates of ϕ_2 can be obtained from an alternative regression

$$y_t^* = (X_{2,t}^*)'\phi_2 + \varepsilon_t^*,$$

where y_t^* and $X_{2,t}^* = \left(x_{21,t}^*, x_{22,t}^*, \ldots, x_{2k_2,t}^*\right)'$ are the least square residuals obtained from auxiliary regressions

$$\begin{aligned} y_t &= X_{1,t}'\xi_y + y_t^* \\ x_{2i,t} &= X_{1,t}'\xi_X + x_{2i,t}^*, \qquad i = 1, \ldots, k_2, \end{aligned}$$

which are also estimated using least square methods.

The Frisch-Waugh Theorem provides a means of eliminating the effects of extra explanatory variables in the model. By projecting both y_t and $X_{2,t}$ onto $X_{1,t}$ and taking the orthogonal residuals, we effectively partial out the impact of $X_{1,t}$ from them, which makes the second regression in Theorem 1 equivalent to the first. Although we do not estimate the contagion model using least square methods, the intuition behind Frisch-Waugh Theorem inspires us to use an analogous argument to control for the influence of common market movements on both the originating and recipient assets. In our case, the market continuous and jump factors are $X_{1,t}$, and the idiosyncratic contagion factors are $X_{2,t}$. After regressing the contagion recipient assets (y_t) and originating asset ($X_{2,t}$) on the market factors and taking the residual, we estimate the contagion coefficients using a second stage regression which involves only the residual series.

The parameters of interests, δ_j^c and δ_j^d, exist in the DGP of asset j in equation (6), or its discrete-time counterpart,

$$r_{j,t} = \beta_j^c r_{0,t}^c + \beta_j^d r_{0,t}^d + \delta_j^c f_{1,t}^c + \delta_j^d f_{1,t}^d + e_{j,t}, \tag{14}$$

in which the two market beta coefficients have been consistently estimated using the Todorov and Bollerslev (2010) approach, and the market jumps have been detected using the Lee and Mykland (2008) test.[5] More specifically, we classify any discrete-time market return as either

[5]This form can be seen to be directly analogous to the form for contagion modeling proposed in Dungey and Martin (2007) which proposes a factor specification describing the evolution of individual assets where contagion is due to the unexpected transmission of idiosyncratic shocks from the source asset to the target. The novelty of the current approach is that we are able to break these effects into continuous and discontinuous components.

continuous or discontinuous movement using the following criteria

$$r_{0,t} = \begin{cases} \hat{r}^c_{0,t} & \text{if } LM_{0,t} \leq LM_{crit}, \\ \hat{r}^d_{0,t} & \text{if } LM_{0,t} > LM_{crit}. \end{cases} \tag{15}$$

Combining the beta estimates and the detected jumps, we are able to extract the impact of market movements from individual assets as follows

$$\tilde{r}_{1,t} = r_{1,t} - \left(\hat{\beta}^c_1 \hat{r}^c_{0,t} + \hat{\beta}^d_1 \hat{r}^d_{0,t}\right), \text{ and } \tilde{r}_{j,t} = r_{j,t} - \left(\hat{\beta}^c_j \hat{r}^c_{0,t} + \hat{\beta}^d_j \hat{r}^d_{0,t}\right), \quad j = 2, 3, \ldots, N. \tag{16}$$

We proceed by reformulating equation (14) to obtain the second stage estimates of the contagion coefficients,

$$\tilde{r}_{j,t} = \delta^c_j \tilde{r}^c_{1,t} + \delta^d_j \tilde{r}^d_{1,t} + \beta^d_j \kappa_{j,t} + \sigma_{j,t}\, \mathrm{d}W_{j,t}, \qquad j = 2, 3, \ldots, N, \tag{17}$$

where the idiosyncratic Brownian and jump factors of asset j are contained in the residual $e_{j,t}$, and δ^c_j and δ^d_j can be estimated using the Todorov and Bollerslev (2010) approach with $\tilde{r}_{1,t}$ as the benchmark asset. Thus we have the following representation

$$\hat{\delta}^c_j = \frac{\sum_{s=1}^{T} \tilde{r}_{j,s}\, \tilde{r}_{1,s}\, \mathbb{1}_{\{|\tilde{\mathbf{r}}_s| \leq \tilde{\mathbf{u}}_T\}}}{\sum_{s=1}^{T} (\tilde{r}_{1,s})^2\, \mathbb{1}_{\{|\tilde{\mathbf{r}}_s| \leq \tilde{\mathbf{u}}_T\}}}, \tag{18}$$

$$\hat{\delta}^d_j = \text{sign}\left\{ \sum_{s=1}^{T} \text{sign}\{\tilde{r}_{j,s}\, \tilde{r}_{1,s}\} |\tilde{r}_{j,s}\, \tilde{r}_{1,s}|^{\tau} \right\} \left(\frac{\sum_{s=1}^{T} \text{sign}\{\tilde{r}_{j,s}\, \tilde{r}_{1,s}\} |\tilde{r}_{j,s}\, \tilde{r}_{1,s}|^{\tau}}{\sum_{s=1}^{T} (\tilde{r}_{1,s})^{2\tau}} \right)^{\frac{1}{\tau}}. \tag{19}$$

where $\tilde{\mathbf{r}}_s$ denotes the $N \times 1$ vector of filtered returns $(\tilde{r}_{1,t}, \tilde{r}_{2,t}, \ldots, \tilde{r}_{N,t})$, and $\tilde{\mathbf{u}}_T$ is the $N \times 1$ vector of thresholds re-estimated similar to equation (9), but with the filtered returns in (17) for $i = 1, ..., N$.

It is straightforward to see that the estimators (18) and (19) are direct analogs of the estimated betas given in equations (10) and (11). Further, there is a degree of correspondence between the estimates of the continuous coefficients in (18) and (10) and the least square estimates with the only difference of using a truncation threshold to discard the jump component

in (18). Thus, the use of Frisch-Waugh Theorem is justified and is expected to yield reasonable estimation results. In the following section, we conduct a Monte Carlo simulation to examine the finite sample performance of the estimated contagion coefficients.

3 Monte Carlo Simulation

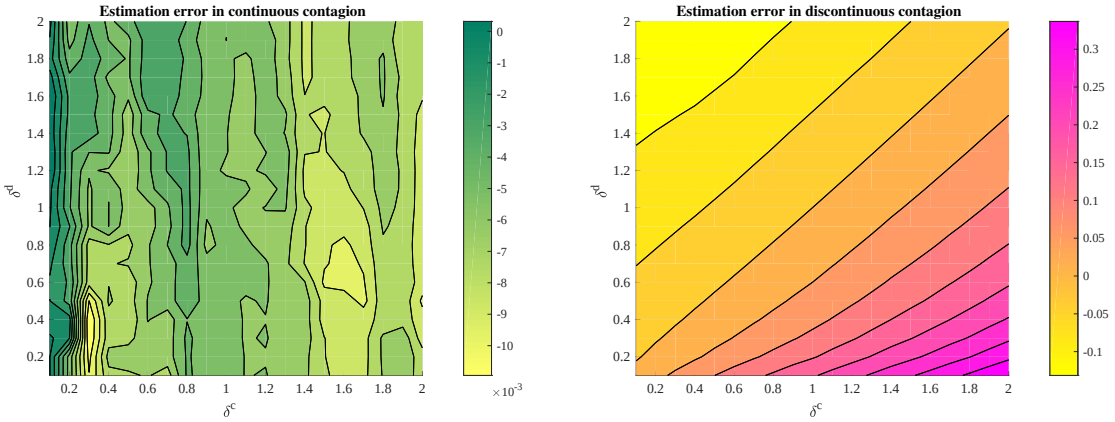
In this section we investigate whether parameter values in model (2) have any impact on the finite sample estimation of the contagion coefficients $\hat{\delta}^c$ and $\hat{\delta}^d$. In an examination of the finite sample performance of the Todorov and Bollerslev (2010) estimators, Alexeev et al. (2017) show that the estimation error in $\hat{\beta}_i^c$ is negligible across most sampling frequencies while the estimation error in $\hat{\beta}_i^d$ is largely affected by the difference between the true values of β_i^c and β_i^d. It is conceivable that the the estimated discontinuous beta is biased towards the true value of the continuous beta in finite sample, as the sample covariation between $r_{i,t}$ and $r_{0,t}$ is lowered by the smaller value of β_i^c. Given that estimated discontinuous beta is almost always higher than the continuous one in the existing empirical applications of this method (Todorov and Bollerslev, 2010; Bollerslev et al., 2016; Alexeev et al., 2017), there may be reasons for concern here, although in reality the span of the difference in the estimated applications is within the smallest range of bias shown in Alexeev et al. (2017).

We simulate the market return according to (4) with jump intensity equal to 10, and the return of the two individual assets according to equations (5) and (6). The length of the time series is set to be $T = 1617$, which is equivalent to an average one month sample of 5-minute observations of the S&P500 stock returns. We use this as the benchmark scenario and test for robustness to other sampling frequencies. The size of the jump is drawn from a normal distribution $\mathcal{N}(0.1, 0.15)$. The values of the beta coefficients are kept fixed in the benchmark case, and then varied individually to examine whether these parameter values have any impact on the estimation error of $\hat{\delta}^c$ and $\hat{\delta}^d$. The initial setting is $\beta_1^c = 1$, $\beta_2^c = 1$, $\beta_1^d = 1.2$, $\beta_2^d = 1.2$, and reflects the existing evidence of higher discontinuous beta compared to the continuous beta. The values of δ^c and δ^d fall in the interval $(0, 2]$ with 0.1 increment. We use the modelling approach outlined in Section 2 to obtain the estimated contagion coefficients. The estima-

tion errors are defined as the differences between the estimated and the true coefficients, i.e. $\left(\hat{\delta}^c - \delta^c\right)$ and $\left(\hat{\delta}^d - \delta^d\right)$.

The left panel of Figure 1 depicts the estimation errors in the continuous contagion factor $\left(\hat{\delta}^c - \delta^c\right)$ while keeping all four beta coefficients fixed at the benchmark values. The magnitude of the estimation error is very small overall; as the true values of δ^c and δ^d range from 0.1 to 2, the size of the estimation error does not exceed 0.011 in absolute terms.

Figure 1: Estimation error in the second-stage contagion coefficients of $\hat{\delta}^c$ and $\hat{\delta}^d$ (benchmark case parameters: $\beta_1^c = \beta_2^c = 1$, $\beta_1^d = \beta_2^d = 1.2$).



On the other hand, the estimation of the discontinuous contagion parameters exhibits larger errors. The difference between the true values of the contagion parameters δ^c and δ^d appears to be the driving factor of estimation error in the discontinuous contagion. This result is analogous to the finding in Alexeev et al. (2017). As shown in the right panel of Figure 1, there is a clear pattern in the estimation error $\left(\hat{\delta}^d - \delta^d\right)$ which changes with the value of $\left(\delta^c - \delta^d\right)$. Whilst the error can be quite substantial for some combinations of δ^c and δ^d, for example, $\left(\hat{\delta}^d - \delta^d\right) \approx 0.38$ when $\delta^c = 2$ and $\delta^d = 0.1$, this degree of difference in the contagion parameters is well outside the differences estimated in the existing applications. In the more realistic case where $\delta^d > \delta^c$, the magnitude of the estimation error is relatively small. For the discontinuous contagion, $\left(\hat{\delta}^d - \delta^d\right) \approx -0.13$ in another extreme case of $\delta^c = 0.1$ and $\delta^d = 2$ is the maximum magnitude obtained in the upper-left half of the parameter region.

The main results from Figure 1 carry over to our other experiments. We vary the continuous betas in the range of [0.5, 1.5], and the discontinuous betas in the range of [0.5, 2.0],

and examine the estimation error in the contagion coefficients when $\delta^d > \delta^c$. Tables 1 and 2 tabulate the mean and maximum values of the estimation error across different sets of parameterizations, respectively. The overall conclusion from these two tables is that the estimation error in the continuous contagion effect is negligible, whereas the discontinuous contagion exhibit larger estimation error. In the majority of cases, we obtain negative estimation error, i.e. the true contagion coefficients are underestimated. The average estimation error shown in Table 1 are small in magnitude with the discontinuous contagion δ^d accurately estimated with an error margin in the range of (0.05, 0.15) on average.

Table 1: Mean estimation error in the second-stage contagion coefficients of $\hat{\delta}^c$ and $\hat{\delta}^d$ for different parameterizations

	Estimation error in δ^c				Estimation error in δ^d			
	β_1^c	β_2^c	β_1^d	β_2^d	β_1^c	β_2^c	β_1^d	β_2^d
0.5	-0.0043	-0.0003	-0.0050	-0.0182	-0.1425	-0.1465	-0.1507	-0.1409
0.8	-0.0034	-0.0048	-0.0082	-0.0024	-0.1161	-0.1223	-0.1176	-0.1170
1.0	-0.0042	-0.0042	-0.0061	-0.0051	-0.0490	-0.0490	-0.0491	-0.0491
1.2	-0.0012	-0.0060	-0.0025	-0.0025	-0.1385	-0.1380	-0.1383	-0.1383
1.5	-0.0164	-0.0052	-0.0065	-0.0053	-0.0798	-0.0675	-0.0759	-0.0755
1.8			-0.0134	-0.0087			-0.0630	-0.0598
2.0			-0.0170	-0.0014			-0.0888	-0.0776

We vary the β parameter value one at a time while keeping the other three fixed at the benchmark values. Specifically, the initial (benchmark) setting is $\beta_1^c = 1$, $\beta_2^c = 1$, $\beta_1^d = 1.2$, $\beta_2^d = 1.2$, and reflects the existing evidence of higher discontinuous beta compared to the continuous beta. The mean values of the estimation error tabulated here come from the parameter space where $\delta^d > \delta^c$.

Table 2: Maximum estimation error in the second-stage contagion coefficients of $\hat{\delta}^c$ and $\hat{\delta}^d$ for different parameterizations

	Estimation error in δ^c				Estimation error in δ^d			
	β_1^c	β_2^c	β_1^d	β_2^d	β_1^c	β_2^c	β_1^d	β_2^d
0.5	-0.0491	-0.0045	-0.0408	-0.0406	-0.3592	-0.3819	-0.3679	-0.3553
0.8	-0.0124	-0.0090	-0.0159	-0.0072	-0.2982	-0.3194	-0.2994	-0.2989
1.0	-0.0109	-0.0109	-0.0158	-0.0097	-0.1315	-0.1315	-0.1315	-0.1315
1.2	-0.0040	-0.0121	-0.0060	-0.0060	-0.3692	-0.3674	-0.3690	-0.3690
1.5	-0.0304	-0.0086	-0.0127	-0.0093	-0.2046	-0.1734	-0.1988	-0.1981
1.8			-0.0312	-0.0356			-0.1563	-0.1506
2.0			-0.0657	-0.0086			-0.2072	-0.1932

We vary the β parameter value one at a time while keeping the other three fixed at the benchmark values. Specifically, the initial (benchmark) setting is $\beta_1^c = 1$, $\beta_2^c = 1$, $\beta_1^d = 1.2$, $\beta_2^d = 1.2$, and reflects the existing evidence of higher discontinuous beta compared to the continuous beta. The mean values of the estimation error tabulated here come from the parameter space where $\delta^d > \delta^c$.

When we look at the maximum values of the estimation error in the parameter region $\delta^d > \delta^c$, Table 2 shows that the discontinuous contagion could be underestimated by 0.4 in some cases. However, these larger error occurred mostly in the case of $\delta^c = 0.1$ and $\delta^d = 2$. The underestimation of δ^d still prevails, as $\hat{\delta}^d$ is quite commonly biased towards δ^c. This indicates that in the empirical analysis, the true value of the discontinuous contagion coefficient is even higher than what is estimated from the data. In general, we find that larger estimation error in the discontinuous effect δ^d is closely linked to three factors: larger difference between β_1^c and β_1^d, larger difference between β_2^c and β_2^d, and lastly, larger difference between δ^c and δ^d.

We increase the sampling frequency to 1-min and 10-second in order to examine the behaviour of our estimation approach in larger samples. The continuous contagion parameter $\hat{\delta}^c$ displays only marginal estimation errors, most of which are of the magnitude 10^{-3} or lower. The overestimation of $\hat{\delta}^d$ is more severe when one of the beta coefficients takes a small value. However, in the parameter region $\delta^d > \delta^c$, the size of the estimation error is limited to 0.1 in the case of 1-minute sampling, and even smaller for 10-second sampling. The estimation error in $\hat{\delta}^d$ is below 0.05 in absolute value in most cases with 10-second sampling. The general trend is that as we sample more frequently, the estimation error in both continuous and discontinuous effects decrease to zero. In other words, our estimation procedure is able to locate the true values of the contagion coefficients as we sample more frequently.

It is not surprising that jump intensity affects the size of the estimation error. As the intensity increases, we obtain more accurate estimates of the beta coefficients, and of the jump component of market returns, both of which lead to smaller estimation errors in the contagion coefficients. Jump size has a similar influence on the estimated contagion effect as jump intensity. Larger-sized jumps are more easily detectable when holding other model parameters fixed. Hence, both the beta coefficients and the contagion coefficients can be estimated more accurately.

4 Empirical Application: Contagion in the US Financial Sector

The evidence for contagion during recession and financial crises periods is relatively well established with lower frequency data.[6] The 2007-2011 period includes the initial emergence of the GFC, usually regarded to mid-2007 and peaking in late 2008 during the few months after the bankruptcy of Lehman Brothers, the bailout of AIG and the announcement of the TARP (Troubled Asset Relief Program). Other highly volatile periods include mid-2010 during the Greek debt crisis, and late 2011 during the European sovereign debt crisis with the deterioration of economic conditions in the Eurozone as a whole. On August 5, 2011 Standard & Poor's downgraded America's credit rating for the first time in history, followed with short-selling ban by Greece on August 8, 2011, and other four EU countries on August 11, 2011. In this section we use the proposed high frequency contagion model and the two-stage estimation procedure to analyze the evidence for contagion between a number of key banks, insurance companies and major US firms during the period 2007 to 2011, the focal period of our investigation. We report the results of contagion effects from AIG and BoA in the paper, further examples are available in Supplementary Appendix or upon request.

The financial crisis of 2007-2009 is widely regarded as having emerged in the US mortgage backed securities market, and then spreading via the medium of the banking sector to other sectors of the US and international economy.[7] There is a significant debate as to whether insurance companies are major instigators of financial shocks which may be systemically risky, or like other firms, merely victims of shocks propagated via the banking sector; contrast for example, Chen et al. (2014) and Acharya and Richardson (2014). Chen et al. (2014) test for Granger causality on the systemic risk measure between banking and insurance industries, and conclude that banks have a much stronger influence on insurers, and the impact also lasts longer. On the other hand, they find that the effects of a shock from insurers on banks

[6] Evidence for transmission in US markets may be found for firms in Jorion and Zhang (2009) and Hertzel and Officer (2012), industry sectors in Dungey and Renault (2018), collateralized debt obligations (CDOs) in Longstaff (2010), for international transmission through equity markets in Bekaert et al. (2014) and the banking sector in Dungey and Gajurel (2015), and later in the European sovereign debt markets in Arghyrou and Kontonikas (2012), Caporin et al. (2018) and Broto and Pérez-Quirós (2015).

[7] Aalbers (2009) argues convincingly that the engagement of the banking sector in the housing market was critical to the spread of the crisis.

are negligible. Based on this result, they suggest that regulators should focus on the banking rather than insurance activities.

The near-failure of AIG has led to a heated discussion about regulating non-bank financial institutions.[8] Harrington (2009) argues that although AIG was centre stage during the GFC, insurance poses much less systemic risk than banking, and the great majority of the funds used to rescue AIG ended up in the banking system directly. On the other hand, McDonald and Paulson (2015) argue that AIG had many features that made it similar to a bank, and that the calls on its funds can be considered akin to a bank run.

We use the proposed high frequency contagion model to estimate the contagion effects between insurance and banking stocks. We showcase our approach by focusing on estimation of contagion effects from AIG and Bank of America (BoA). These firms were amongst the top 3 recipients of funding support from the Troubled Assets Relief Program (TARP) by mid-2009.[9] AIG was clearly a major focus of problems during the crisis period, and its ongoing importance is reflected by its designation as a globally systemically important insurer (G-SII) by the Financial Stability Board.

BoA has been central to ongoing assessments of systemically risky financial institutions in the US. Since June 2008, BoA has been ranked among the top 3 most systemically risky financial institutions in the NYU-Stern Vlab project based on the marginal expected shortfall approach of Acharya et al. (2016)[10]. Since September 2008, it has frequently been ranked as the most systemically important – as well as being identified as a globally systemically important bank (G-SIB) by the Financial Stability Board. During 2007 BoA bought Countrywide Financial, one of the early dramatic failures due to the collapsing mortgage backed securities market, and Merrill Lynch in the days leading up to the collapse of Lehman Brothers in September 2008 – both of these acquisitions have involved the bank in lengthy legal battles regarding settlements with other correspondents of the acquired institutions (including

[8]For a detailed review on AIG's experience through the GFC see McDonald and Paulson (2015).

[9]Citigroup received $50 billion, AIG received $40 billion, and BoA received $35 billion by July 2009 (Harrington, 2009). Robustness checks for our results using Citigroup as the source of bank contagion and results are qualitatively unchanged.

[10]These rankings may be obtained from http://www.vlab.stern.nyu.edu, using the Systemic Risk Analysis tab and selecting MES for US financials.

customers, shareholders and other counterparties).

4.1 Data

Our data set consists of 5-minute observations on the equity prices of 501 stocks drawn from the historical constituents of the S&P500 index from January 2, 2003 to December 31, 2011, leading up to and surrounding the focal period of our study, the 2008 Global Financial Crisis. Intra-day returns and prices from 9:30 am to 4:00 pm EST are obtained from the Thomson Reuters Tick History database via SIRCA. We only select stocks which have sufficient data coverage over the nine years in order to have a more complete characterization of the contagion effects in the time series dimension. Details of the data downloading and cleaning process are documented in the web appendix of Dungey et al. (2012). We remove overnight returns, leading to 77 intra-day observations of 5-minute data on 2262 active trading days. We use the characteristics of all 501 series to establish our threshold vector $\boldsymbol{u}_T$ with parameter values $\tau = 2$ and $\omega = 0.49$.

As suggested in Todorov and Bollerslev (2010), daily estimates of the betas are quite noisy and fluctuate dramatically. Hence we use an estimation window of a month, and estimate the contagion model for 108 separate calendar months. We use the S&P500 index as the market portfolio in the first stage of estimating the continuous and discontinuous betas. The jump test by Lee and Mykland (2008) is performed on the S&P500 index at 10% significance level in the first stage.[11] After filtering out the market factor, we use the thresholding technique on the filtered returns to estimate the continuous and discontinuous contagion coefficients.

Table 3 presents descriptive summary statistics of coefficients estimates from equation (2), namely, the market betas, β_j^c and β_j^d (Panel A), and the contagion coefficients, δ_j^c and δ_j^d, for using AIG as the crisis originator (Panel B) and BoA as the crisis originator (Panel C). The estimates are pooled across all 108 distinct monthly periods and across all stocks (501 in case of β's and 500 in case of δ's). It is worth noting that the mean and median values of β_j^c and β_j^d

[11] We perform robustness checks by considering a range of significance levels when applying Lee and Mykland (2008) jump test. Specifically, we used $\alpha \in [0.20; 0.10; 0.05; 0.01; 0.005; 0.001]$. The results are presented in the Supplementary Appendix.

Table 3: Summary statistics of estimated coefficients. The summary statistics are based on β_j^c, β_j^d, δ_j^c, and δ_j^d estimates from equation (2) pooled across all 108 distinct monthly periods and across all stocks (501 in case of β's and 500 in case of δ's).

	Min	Mean	Median	Max	StDev	Skew	Kurt
Panel A: Market betas							
β_j^c	-1.1892	0.95292	0.9074	4.5064	0.4499	0.7229	4.7517
β_j^d	-2.7534	1.3121	1.2357	9.6477	0.54214	1.0606	8.6233
Panel B: for i =AIG							
δ_j^c	-0.7334	0.4039	0.3404	3.1472	0.2993	1.4469	6.4667
δ_j^d	-1.3774	0.6890	0.6187	4.6707	0.4391	1.3195	6.7836
Panel C: for i =BoA							
δ_j^c	-1.2951	0.5225	0.4691	3.2308	0.3224	1.3635	6.9834
δ_j^d	-2.7440	0.8768	0.8103	6.0564	0.4852	1.2180	7.2759

reported in Table 3 closely correspond to the benchmark levels used in our simulation exercise in Section 3. Evidently, after removing the common market factor in the first step, the linkages between individual assets, represented by δ's, are substantially smaller. For example, when using AIG as the originator, the median values of δ_j^c and δ_j^d are 0.35 and 0.62, respectively. Positive values of skewness are in line with the observations that medians are always smaller than the mean values. High values of kurtosis indicate concentration of most of our estimates around their mean values.

4.2 Evidence for High Frequency Contagion

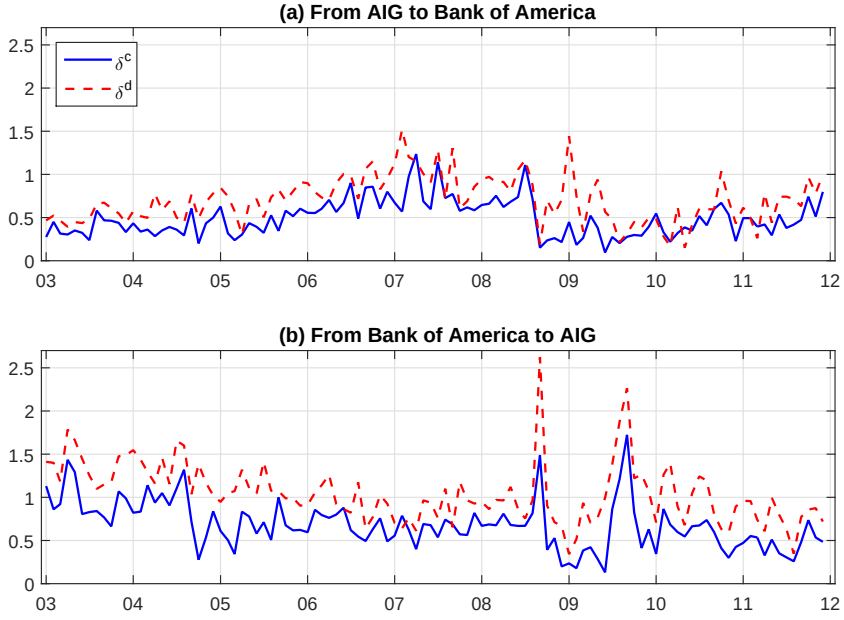
To illustrate our proposed model, we estimate the contagion effects from AIG and BoA to each other, and their average effect on all banking sector firms, insurance sector firms and real economy firms in the sample.[12]

Figure 2 presents the estimated coefficients δ^c and δ^d between AIG and BoA. As anticipated, the discontinuous coefficient δ^d is higher than the continuous counterpart most of the time. Along with the fact that jumps are larger in magnitude than continuous price movements, the results support the framework proposed in previous literature that jump activity

[12]We find similar results for Prudential Financial, Citigroup, among other banking and insurance firms. These results are presented in the Supplementary Appendix.

is more contagious (Aït-Sahalia et al., 2015).

Figure 2: The estimated contagion between AIG and Bank of America. The continuous contagion coefficients are represented by solid blue lines, and the discontinuous contagion coefficients are represented by dashed red lines.



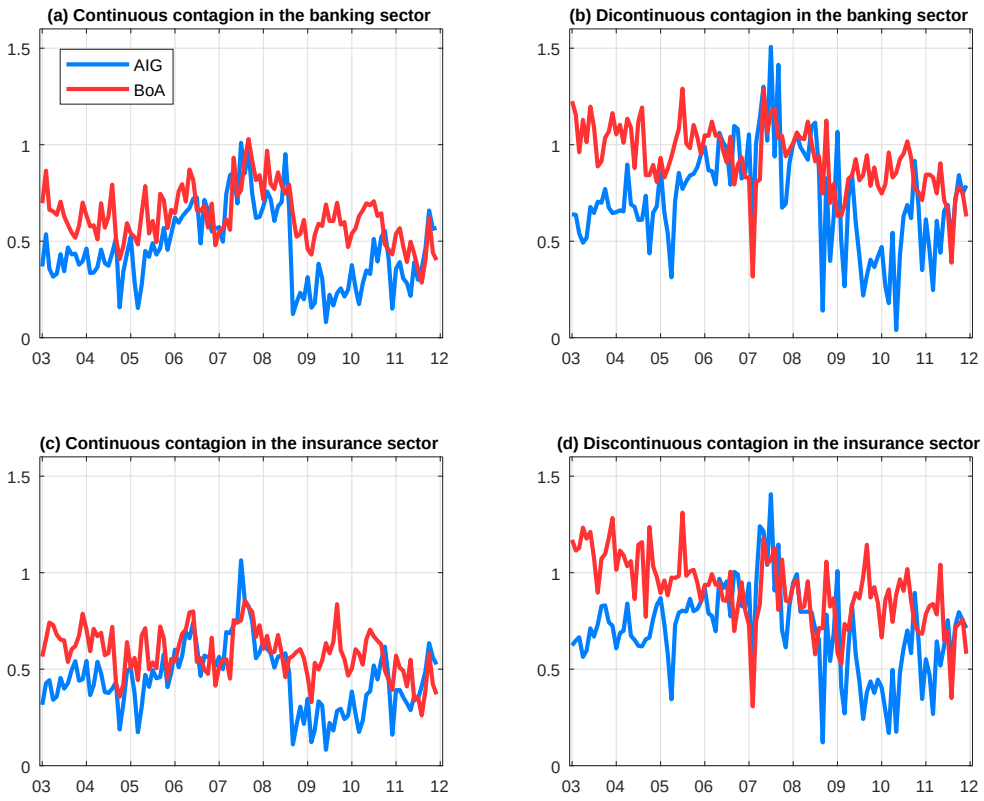
We use the sample prior to 2007 to establish the benchmark values of δ^c and δ^d during non-crisis periods. At the beginning of the sample, the impact parameters for the insurer AIG on the bank BoA are considerably smaller than those in the other direction. However, from 2005 onward, Panel (a) shows that the estimated contagion coefficients from the insurer on the bank were *increasing*, whilst Panel (b) shows that those from the bank to the insurer were *decreasing*. This likely represents the increasing difficulties encountered by AIG since 2005, which eventually leads to its rescue in October 2008. The most abrupt changes in both the continuous and discontinuous contagion coefficients for AIG to BoA occur in September 2008 (a drop in both coefficients from 1.1 in July 2008 to below 0.2 in September 2008) after which the rescue of AIG, effectively designating it "too big to fail", removed a substantial source of risk from the financial markets.

In September 2008, the BoA dominates AIG as the contagion source, with a discontinuous contagion coefficient of over 2.6 on AIG, and substantially exceeds its usual range prior to 2007, while the contagion coefficient from AIG to BoA is only around 0.2. One year later, the

discontinuous contagion from BoA reaches another peak of 2.3, in September 2009, compared with 0.45 from AIG to BoA. The highest discontinuous contagion from AIG to BoA occurs in February 2007, aligned with the Chinese stock market wobble, and may represent initial concerns about AIG, who reported more than $11 billion's loss in unrealized credit default swap (CDS) during 2007 (McDonald and Paulson, 2015).

To obtain a more general view, we extract the contagion impact of AIG (blue line) and BoA (red line) on all 18 insurers and 23 banks from our sample, and plot the mean estimates for the contagion impact parameters within each sub-sector in Figure 3. Panel (a) of Figure 3 provides the mean estimate of δ^c on banking sector stocks for the two potential originators, AIG and BoA; Panel (b) provides the mean estimate of δ^d for the banking stocks in the sample. Panels (c) and (d) provide analogous plots for the estimates in the insurance sector. It is evident from the plots that although the discontinuous impact coefficients are generally higher than the continuous ones, as seen in the individual results.

Figure 3: Contagion effects on insurance firms and banks (averaged within the sub-sector)



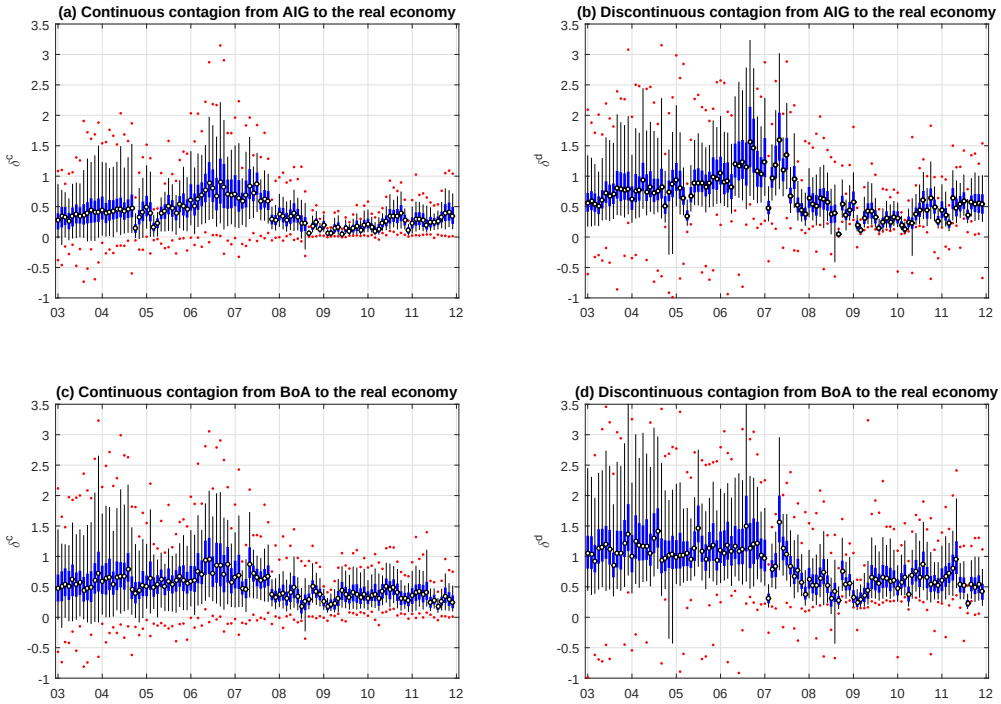
Prior to 2007, Bank of America has stronger linkages with both banking and insurance sectors compared to AIG, as shown across all panels in Figure 3. These linkages are mostly preserved during the crisis period, with no abrupt changes in their magnitude observed in the average estimated contagion coefficients originating from BoA. On the other hand, AIG exhibits weaker linkages with the banking and insurance sectors prior to 2007, but the upward trends leading up to the GFC is clearly observed. The peak values of the average contagion coefficients originating from AIG occur in mid-2007 in all cases considered in Figure 3. In particular, in the case of contagion from AIG to the banking sector, the average values of δ^c and δ^d remain at high levels from mid-2007 to late-2008. Therefore, at the sectorial level, we find stronger evidence of contagion originated from AIG rather than from BoA. The peak occurs in July 2007, correspond to the initial emergence of AIG's huge loss in security lending. AIG expanded its securities lending rapidly in the run-up to 2008. At the end of 2003, the firm had less than $30 billion in securities lending outstanding. At the peak in 2007Q3, AIG had securities lending outstanding of $88.4 billion. AIG consistently lent more than 15 percent of its domestic life insurance assets: in 2007, for example, this number was 19 percent (McDonald and Paulson, 2015).

4.3 Contagion Effects on the Real Economy

Evidence on the effect of contagion sourced from AIG and BoA to the non-financial sectors is provided in Figure 4, where red dots display the minimum and maximum values of the estimated δ^c or δ^d, whiskers are the 2.5^{th} and 97.5^{th} percentiles, boxes show the interquartile ranges, and black dots are the medians. It is immediately apparent that not only is the median estimate of the continuous contagion coefficient δ^c lower than the discontinuous contagion coefficient δ^d, the range of estimates is also smaller. This is consistent with smaller standard deviations of the estimated δ^c compared with δ^d, as reported in Table 3.

We first examine the continuous contagion effects originating from AIG and BoA depicted in Panels (a) and (c) of Figure 4, respectively. In the pre-crisis period, the continuous contagion estimates to the real economy sector show wide ranges and slight upward trends in values.

Figure 4: Contagion originated from AIG and Bank of America to real economy firms



Since the beginning of the crisis in 2007, the estimated δ^c does not exhibit any abnormalities compared to the benchmark established using the pre-2007 sample. Although the median values are high in 2007, the ranges of the estimates are, in fact, becoming smaller. This is consistent with a degree of disconnection between the financial sector and the real economy as a result of the crisis, and, potentially, the policy interventions designed to protect the real economy by calming the conditions in the financial sector.

Similar patterns are observed for the discontinuous contagion coefficients with a few extreme estimates corresponding to significant events, as discussed at the beginning of this section. The pattern observed in the individual firm results shown in previous figures for discontinuous contagion from AIG and BoA is largely preserved in the overall real economy results. Prior to the peak of the crisis, the discontinuous contagion coefficient from AIG is increasing and has a relatively wide range, while that for the contagion coefficient estimates from BoA is stable, but also with a wide range. After 2007, the impact and range of the discontinuous contagion coefficients from both AIG and BoA on the rest of the economy is

slightly lower, and with smaller variation, although this variation remains greater than that associated with continuous contagion.

There appears to be little difference between the impact of AIG or BoA in terms of continuous contagion effects on the real economy firms as a whole. While pre-crisis benchmark of contagion coefficients from AIG is lower than that from BoA in general, these coefficients increase and become higher than those from BoA at the peak. Contagion effects from AIG and BoA are similar in terms of the average effects in the post-2007 period – although the BoA effects have a slightly wider range. In this respect, these examples suggest that these two institutions had an equivalent role to play in transmitting the financial sector shocks to the real economy.

The evidence in our empirical examples support and reconcile some contradictory findings in the literature. First of all, the impact of contagion between the two financial institutions supports that contagion from the bank (BoA) has larger impact on the insurance company AIG than vice versa, consistent with the evidence in Chen et al. (2014) and Harrington (2009). However, we find stronger evidence of contagion originated from AIG rather than from BoA. While banks and insurers have the most impact on each other, their impact on the real economy sector from either source is similar. We acknowledge that we have not explored the impact of contagion from all possible insurers or banks in the sector. However, our findings contribute to understanding the sources of contagion. On one hand, if we assume that AIG is structurally distinct from other insurers, then the results support the hypothesis of McDonald and Paulson (2015) that AIG is fundamentally behaving similarly to a bank. On the other hand, the evidence of Ghosh and Hilliard (2012) finds strong support for the direct transmission of shocks from AIG to other firms in the insurance business, suggesting that there are strong commonalities between AIG and other insurers, so that AIG is, in fact, representative of the entire insurance sector.

In summary, the empirical evidence stemming from our proposed framework finds that, while contagion effects from BoA to AIG may be larger than the impact of contagion from AIG to BoA, there is limited evidence of this on an industry-wide scale. The impact of shocks from

either part of the financial sector is similar for real economy firms. Insurance and banking both transmit financial sector shocks to the real economy.

5 Conclusion

This paper proposes a new approach to detecting and measuring the impact of contagion using high frequency financial data. Despite the increasing evidence that high frequency data provides a number of advantages in measuring volatility and extreme events (such as jumps), there is very limited literature applying it to detection of crisis conditions.[13] Combining the established latent factor approach based on CAPM framework to detecting contagion common in the lower frequency literature, typified for example by Bekaert et al. (2014) and Dungey and Martin (2007), and the recent high frequency CAPM decomposition into continuous and discontinuous components of Todorov and Bollerslev (2010), we build a framework to detect the presence of contagion effects and estimate their impacts. The finite sample properties of the estimation approach in detecting the contagion parameters are demonstrated to be similar to those for estimating the beta parameters in Todorov and Bollerslev (2010) as shown in Alexeev et al. (2017).

The methodology is illustrated with an empirical application to detecting the extent of contagion from Bank of America and AIG to other S&P500 firms since the GFC started in 2007. Distinguishing discontinuous contagion from continuous contagion empirically confirms that the former generally has a higher impact coefficient than the latter. This is consistent with the common interpretation of jumps that unanticipated news arrival is absorbed relatively quickly. We show that the impact of contagion from BoA to AIG outweighs the reverse impact. However, the industry-wide average contagion effects of AIG on banking and insurance companies in the sample outweigh the contagion effects of BoA on the same firms. The overall contagion effects on the real economy from either source is similar. It is difficult to discern a substantial difference between the median and interquartile range impacts. The results support the view that both of these financial institutions played a role in transmitting

[13] See Aït-Sahalia et al. (2015) on contagion, and Black et al. (2012) on detecting jumps during periods of stress.

financial sector turmoil to the firms of the real economy during the period surrounding the Global Financial Crisis.

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A Supplementary Appendix

A.1 Additional examples

Evidence on the effect of contagion from four major Systemically Important Financial Institutions (SIFIs) are presented in Figures A.1–A.4. The boxplots in the figures show the dynamics of the continuous and jump contagion effects for the two insurers and the two banks, namely, AIG, Prudential Financial, Bank of America, and Citigroup. Red dots represent the minimum and maximum values of the estimated δ^c or δ^d, with whiskers denoting the 2.5^{th} and 97.5^{th} percentiles, vertical blue lines represent interquartile ranges, and black dots are the medians.

Comparing the two insurers, AIG and Prudential, the overall movements in the estimated contagion coefficients are quite similar, especially in the period leading up to the crisis in 2007. We have experimented with other insurers and obtained similar results. These commonalities among insurance firms supports the role of AIG as a representative of the entire insurance sector. The main distinction of AIG from other insurance firms is that immediately after late-2008, the continuous contagion coefficients from AIG (Panels (a) and (c) in Figure A.1) contract to a much narrower range than those from Prudential (Panels (a) and (c) in Figure A.2). This is consistent with a degree of disconnection between AIG and the rest of the economy as a result of the policy interventions designed to protect the real economy. Such effects are also observed in the discontinuous contagion coefficients from AIG (Panels (b) and (d) of Figure A.1).

Figures A.3 and A.4 contrast the estimated contagion coefficients from BoA and Citigroup, the two banks who received the highest amount of government bailout in TARP. Prior to the crisis, BoA generally shows stronger linkage with the financial sector, as well as the real economy, than Citigroup. The overall tendency in both δ^c and δ^d estimates is very similar. There are a few sharp drops in the discontinuous contagion coefficient δ^d in early 2007 for AIG and BoA, whereas Prudential and Citigroup do not display such abrupt changes.

Figure A.1: Contagion originated from AIG to real economy (top panels) and financial sector firms (bottom panels).

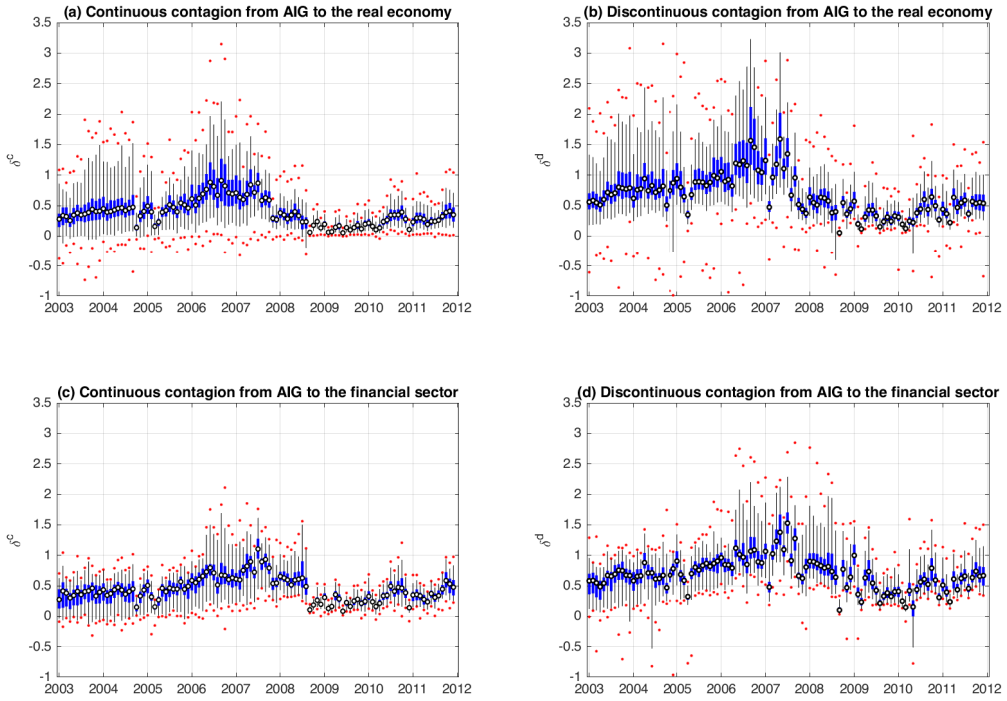


Figure A.2: Contagion originated from Prudential Financial to real economy (top panels) and financial sector firms (bottom panels).

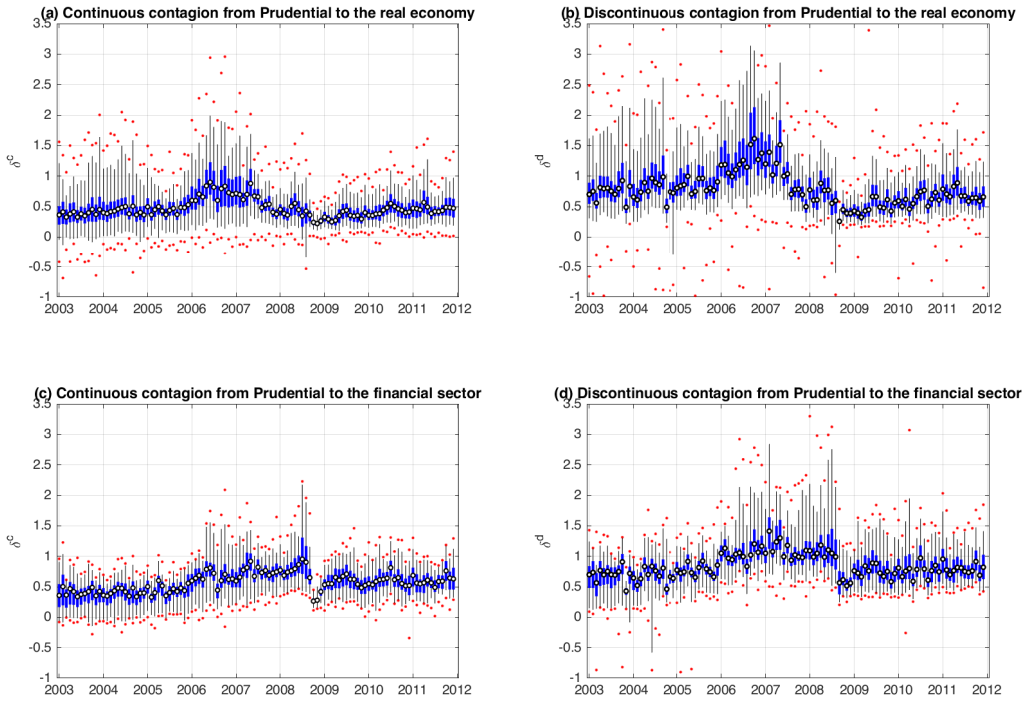


Figure A.3: Contagion originated from BoA to real economy (top panels) and financial sector firms (bottom panels).

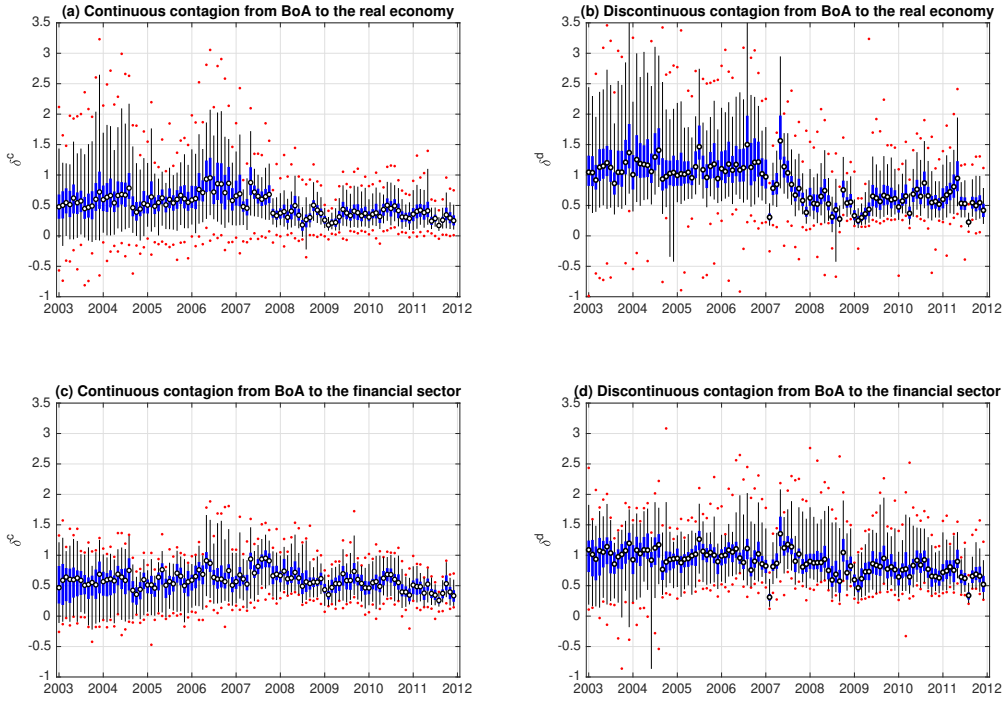
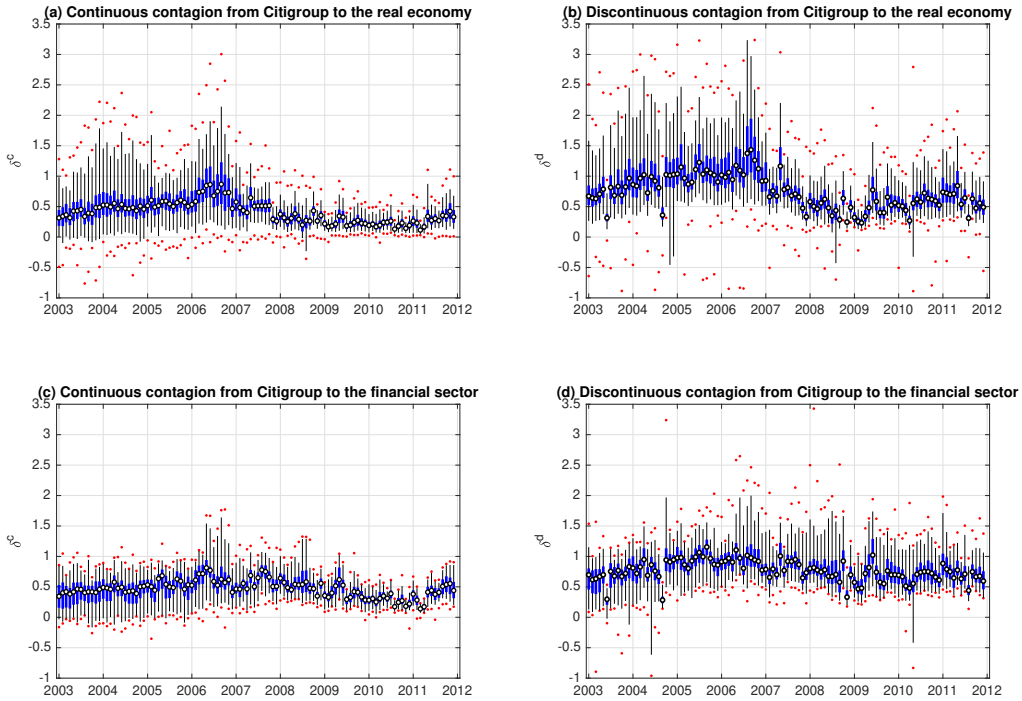


Figure A.4: Contagion originated from Citigroup to real economy (top panels) and financial sector firms (bottom panels).



A.2 Robustness checks of using different levels of significance in LM test

Lee and Mykland (2008) jump test is known to over-detect jumps in the presence of microstructure noise if the sampling frequency is less than 15 minutes (Lee and Mykland, 2012). To check the robustness of our contagion estimates due to potential spurious jump detection, we implement the Lee and Mykland (2008) jump test using a range of significance levels, $\alpha \in$ [0.001; 0.005; 0.01; 0.05; 0.10; 0.20]. The results are presented in Figures A.5–A.8, where we plot the average values of the estimated contagion coefficients using different levels of significance α for AIG, BoA, Prudential and Citigroup as the shock originator, respectively. The results are almost indistinguishable from each other, with only small discrepancies. Very similar set of results are available for the median values of the estimated contagion coefficients (omitted here for brevity).

Figure A.5: Contagion originating from AIG to real economy (top panels) and financial sector firms (bottom panels) at different levels of significance in LM jump test.

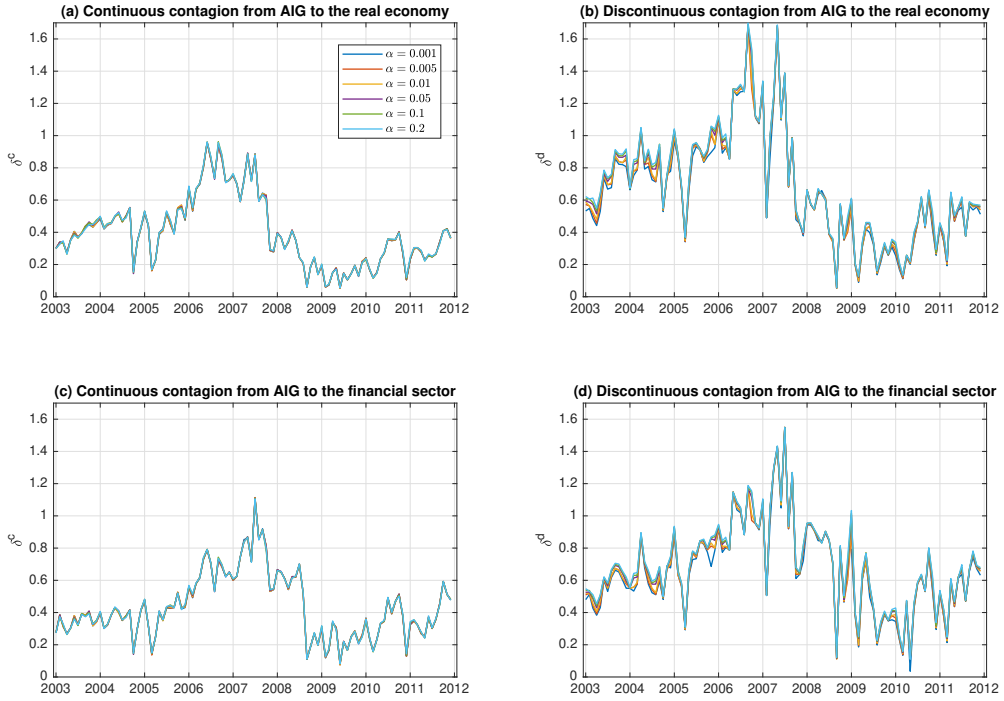


Figure A.6: Contagion originating from Prudential Financial to real economy (top panels) and financial sector firms (bottom panels) at different levels of significance in LM jump test.

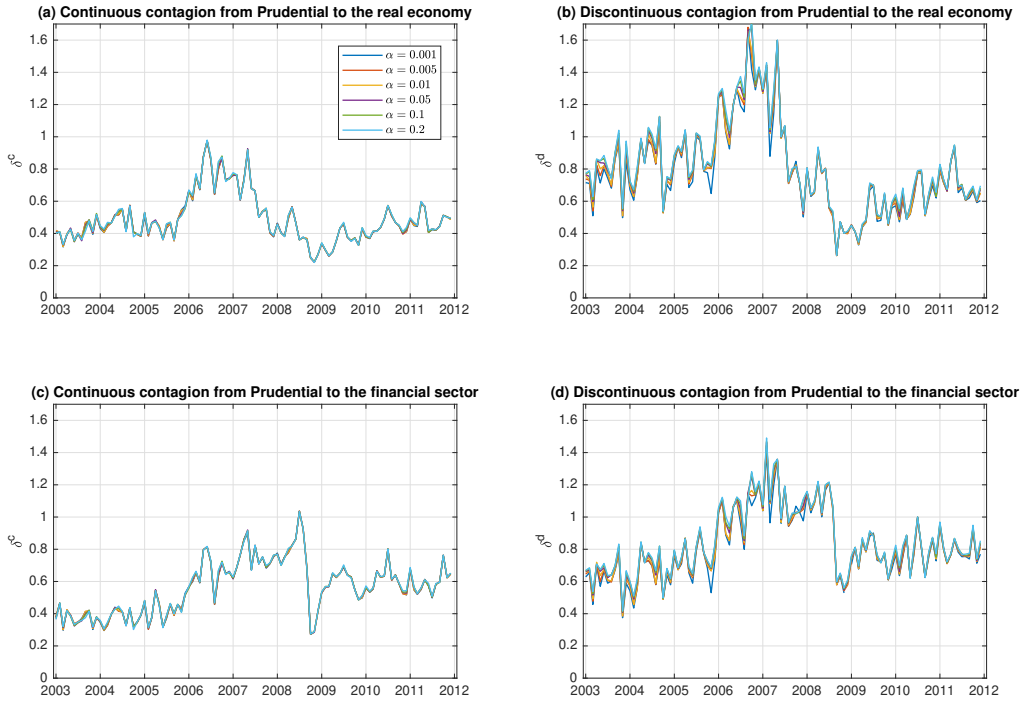


Figure A.7: Contagion originating from Bank of America to real economy (top panels) and financial sector firms (bottom panels) at different levels of significance in LM jump test.

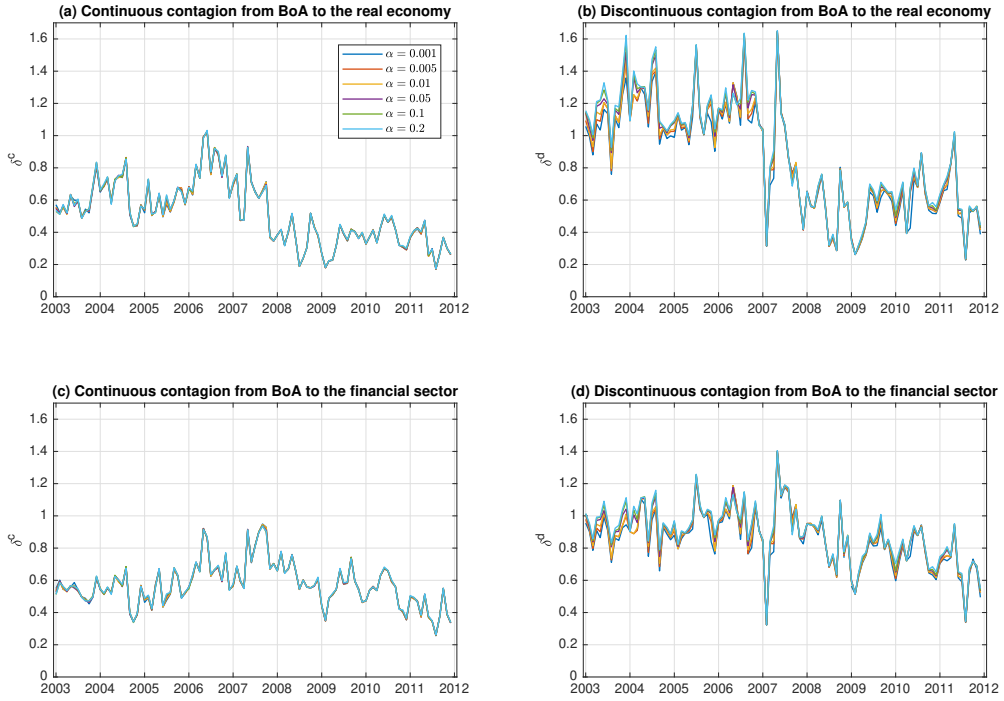


Figure A.8: Contagion originating from Citigroup to real economy (top panels) and financial sector firms (bottom panels) at different levels of significance in LM jump test.