Modelling Financial Contagion Using High Frequency Data

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Abstract

We develop a methodology for detecting and measuring contagion using high frequency data which combines advances in estimating beta for both continuous and discontinuous price movements and frameworks for modelling the transmission of shocks developed for lower frequency data. We propose a two-stage estimation procedure, and show its satisfactory finite sample properties, especially in the empirically plausible parameter space. The empirical application contributes to the current debate over the role of insurance companies in transmitting financial crises. Using data from two major US banks and two insurers we assess contagion to other financial sector and real economy firms using US equity market data over the period of 2003-2011. The results enable us to reconcile the existing evidence that large banks have stronger contagion effects on the insurers than is evident in reverse, but that at a sectoral level this is difficult to discern. By showing that the contagion effects of a source bank and source insurer on real economy firms are not distinctly different we contribute to the growing literature which supports that the role of banks and insurers should be of interest to regulators tasked with protecting the real economy from systemic problems in the financial sector.

Keywords: factor model, crisis transmission, jumps, high frequency data.

JEL: C10, C58, G01

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 which demonstrably improve estimation of volatility and price disruptions due to the unexpected arrival of information, see for example Andersen et al. (2007); Lahaye et al. (2011); Aït-Sahalia and Jacod (2012), this represents a gap in the literature.

This paper fills this gap with a model of contagion between assets detected using high frequency financial markets data. The existing paper in this literature of Aït-Sahalia et al. (2015) considers the transmission of shocks between assets via a self-exciting Hawkes process. The approach taken here extends the standard capital asset pricing model (CAPM) and latent factor model approach to contagion which is heavily utilised in the daily and lower frequency literature to the high frequency data domain; see Dungey et al. (2005) for an overview of how the factor framework nests other approaches taken in the literature. We draw from the advances in decomposing the CAPM model into a continuous and discontinuous component in Todorov and Bollerslev (2010) along with recent applications of this method which demonstrate that these two components have differential beta coefficients (see Alexeev et al., 2014; Todorov and Bollerslev, 2010; Bollerslev et al., 2015; Sayeed et al., 2015), and extend it to consider the differential impact of contagion sourced from the continuous and discontinuous sources. The discontinuous sources are analogous to the jump sources of contagion proposed in Aït-Sahalia et al. (2015).

Beta has been shown to vary with firm-specific information. In particular, earnings announcements for individual firms temporarily increase beta around the time of the news release (Patton and Verardo, 2012). As news release has been shown to be closely related to jumps (or discontinuities) in price movements for many assets (Dungey et al., 2009; Andersen et al., 2007; Lahaye et al., 2011; Miao et al., 2013), the logical extension is that one would expect that the beta associated with jump movements in stocks differs from that in continuous movements. More specifically, given the evidence in Patton and Verardo (2012), and that jumps are strongly associated with news surprises, we hypothesise that the influence of discontinuous movements is greater than that for continuous movements. The interrelationship between asset returns is likely to be influenced by transmission of the new information between them, whether it arrives via a continuous or discontinuous process. We hypothesise that jump based movements will have a larger impact than continuous based updating, based on the Patton and Verardo (2012) learning framework.

Contagion is frequently modelled as the disruption of the usual established relationships between assets by the transmission of a shock from one asset to another. The distinction

between contagion and spillovers is that the former involves unusual transmissions of idiosyncratic shocks between assets during periods of stress, whilst the spillovers are contained in the usual relationships between assets represented by the usual betas in the CAPM framework. In the framework proposed in this paper, the idiosyncratic part of individual asset returns have both a continuous and discontinuous component, as per the model proposed by Todorov and Bollerslev (2010), and consequently there are two potential avenues of contagion – a continuous contagion effect and a discontinuous contagion effect. The framework proposed here shows how we can estimate these contagion effects during periods of price disruption identified by jump activity in the market as a whole. Other mechanisms to select the periods on which to estimate the contagion effects could be considered, but this criteria respects the existing approach that jumps, representing unexpected news arrival, are associated with potentially contagious events in Aït-Sahalia et al. (2015), and is related to the lower frequency literature that tests for contagion during tail or outlier events; for example (Bae et al., 2003; Favero and Giavazzi, 2002; Boyson et al., 2010; Busetti and Harvey, 2011).

We apply the framework to the debate on the evidence for contagion during the period of 2003 to 2011 between banks and insurance companies. On one hand evidence supporting contagion effects from banks to insurers, but limited impact in the other direction, supports the view that financial regulation does not need to be extended to the insurance sector; see (Chen et al., 2014). Contrasting evidence is provided in Acharya and Richardson (2014) who support the extension of regulation to this sector. We contribute to this debate by considering the time-varying evidence for contagion at a firm level from a number of key US-listed banks and insurers, notably AIG, Bank of America, Goldman Sachs and Metlife, on other financial and real economy firms listed on the S&P500.

Our first results confirm that estimates for the impact of discontinuous contagion are higher than for continuous contagion, supporting the hypothesis that learning about information shocks sufficient to cause price disruption in an individual firm price is faster than for information which is incorporated via the usual continuous price process. We support the evidence that the contagion impact from these individual banks to selected other large individual banks and insurers is larger than the impact of contagion transmitted from the large insurers to this same group of banks. However, at an industry level we cannot discern a difference between the average impact of contagion from these four source firms to members of the banking or insurance industry. Thus at an individual level for large institutions we can obtain analytically different results, but not at an aggregate level. 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 industry. Reconciling these two discourses challenges the view of treating AIG differently for regulatory purposes. Our

final contribution to this debate is to estimate the contagion impact from AIG and Bank of America to the real economy firms in the sample, and find that although the range of impacts across the firms is wider for the bank-originated contagion in both the continuous and discontinuous case, it is not clear that the impact of contagion from banks is greater than the impact of contagion from insurance companies. In both cases the median and range of estimates is higher and wider in the pre-crisis period and both drops and contracts in the period after 2008. In sum the evidence supports that contagion between individual large banks and insurers do have differential impacts on each other, but this is both difficult to generalise across the entirety of the financial sector firms, and the impact on real economy firms from either source is on average not importantly different. If the aim of financial regulation is to protect the real economy firms from financial sector distress the evidence presented here supports the view that insurers are a potentially important component of the transmission of stress, and thus are rightly in the purview of the regulatory authorities.

The remainder of this article is organized as follows. Section 2 proposes the estimation procedure of a simple multi-factor 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 provides 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, providing evidence of the relative impact of discontinuous and continuous contagion effects. Section 5 concludes the paper.

2 Contagion Model in High Frequency

Our modeling framework draws parallels from the existing factor model approach to detecting contagion effects in the lower (generally daily) frequency data in Bekaert et al. (2014), Dungey and Martin (2007), Bekaert et al. (2005) and new techniques in high frequency 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 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 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 provided by Todorov and Bollerslev (2010) under some fairly general assumptions; for applications of this approach see Alexeev et al. (2014); Sayeed et al. (2015) and Bollerslev et al. (2015).

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 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 that during periods of stress, asset j may be affected by movements from another asset, i, which are not part of the common component – and 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. If crises are transmitted through jump activities only, then we will have $\delta_j^c = 0$ and $\delta_j^d \neq 0$ during periods of stress. 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 $r_{i,t}$, and then estimating 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 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. 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:

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

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

To simplify the notation, 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 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

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

We usually set $\tau \geq 2$, as the continuous Brownian component does not have any impact for high values of τ. The estimators provided by Todorov and Bollerslev (2010) are the discrete-time sample counterparts of equations (7) and (8) as in practice we observe discrete prices and returns. 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 can differ with the chosen window size.

For discretely observed asset returns $r_{i,t} = p_{i,t} - p_{i,t-1}$, $i = 0, 1, \ldots, N$, $t = 1, 2, \ldots, T$, 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. 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. The equivalent representation of the continuous beta (7) in a discrete sample is

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

where $\mathbb{1}$ is the indicator function. 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.

2.2 Test for Jumps

We identify jumps using the test proposed by 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 Lee and Mykland (2008) test detects the existence of jumps by examining the extreme values in $|r_{i,t}|/\sigma_{i,t}$. In order to construct the test statistic, we estimate the spot volatility $\sigma_{i,t}$ using the 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 $K = \sqrt{M \times 252}$ is suggested by 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

$$LM_{i,t} = |r_{i,t}| \,/\hat{\sigma}_{i,t}, \qquad t = K+1, \ldots, T, \tag{13}$$

will then have a standard Gumbel distribution under the null hypothesis of no jump at time t. The critical value has the following form

$$LM_{crit} = \frac{\zeta}{c\sqrt{2\ln M}} + \frac{\sqrt{2\ln M}}{c} - \frac{\ln(4\pi) + \ln(\ln M))}{2c\sqrt{2\ln M}}. \tag{14}$$

where $c = \sqrt{2/\pi}$ and $\zeta = -\ln(-\ln(1-\alpha))$, α denotes the daily significance level.[1] 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 price 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 biggest advantage 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. Hence we expect that the LM test will provide appropriate detection of the jumps which exist in the asset prices.

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.

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. The least square estimates of* ϕ_1 *can be obtained from an alternative regression*

$$y^*_t = (X^*_{1,t})'\phi_1 + \varepsilon^*_t,$$

where y^*_t *and* $X^*_{1,t} = \left(x^*_{11,t}, x^*_{12,t}, \ldots, x^*_{1k_1,t}\right)'$ *are the least square residuals obtained from auxiliary*

[1] 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 (14) instead of the original critical value given by Lee and Mykland (2008) in this paper.

regressions

$$y_t = X'_{2,t}\xi_y + y^*_t$$
$$x_{1i,t} = X'_{2,t}\xi_X + x^*_{1i,t}, \qquad i = 1, \ldots, k_1,$$

which are also estimated using least square methods.

The Frisch-Waugh Theorem provides a means of eliminating the effect of extra explanatory variables in the model. By projecting both y_t and $X_{1,t}$ onto $X_{2,t}$ and taking the orthogonal residuals, we effectively partial out the impact of $X_{2,t}$ 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.

The parameters of interests, δ^c_j and δ^d_j, exist in the DGP of asset j in equation (6), or the discrete-time counterpart,

$$r_{j,t} = \beta^c_j r^c_{0,t} + \beta^d_j r^d_{0,t} + \delta^c_j f^c_{1,t} + \delta^d_j f^d_{1,t} + e_{j,t}, \tag{15}$$

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.[2] More specifically, we classify any discrete-time market return as either continuous or discontinuous movement using the following criteria

$$r_{0,t} = \begin{cases} \hat{r}^c_{0,t} & \text{if } LM_{0,t} \leq LM_{crit} \\ \hat{r}^d_{0,t} & \text{if } LM_{0,t} > LM_{crit} \end{cases}. \tag{16}$$

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

$$\tilde{r}_{1,t} = r_{1,t} - \left(\hat{\beta}^c_1 \hat{r}^c_{0,t} + \hat{\beta}^d_1 \hat{r}^d_{0,t}\right), \text{ and } \tilde{r}_{j,t} = r_{j,t} - \left(\hat{\beta}^c_j \hat{r}^c_{0,t} + \hat{\beta}^d_j \hat{r}^d_{0,t}\right), \; j = 2, 3, \ldots, N. \tag{17}$$

We then reformulate equation (15) as follows to obtain the second stage estimation of the contagion coefficients,

$$\tilde{r}_{j,t} = \delta^c_j \tilde{r}^c_{1,t} + \delta^d_j \tilde{r}^d_{1,t} + \beta^d_j \kappa_{j,t} + \sigma_{j,t}\, dW_{j,t}, \qquad j = 2, 3, \ldots, N, \tag{18}$$

[2]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. In the current form we are able to break these effects into continuous and discontinuous components.

where the idiosyncratic Brownian and jump factors of asset j are contained in the residual $e_{j,t}$, and δ_j^c and δ_j^d 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

$$\hat{\delta}_j^c = \Big(\sum_{s=1}^{T} \tilde{r}_{1,s}\, \tilde{r}_{j,s}\, \mathbb{1}_{\{|\tilde{\mathbf{r}}_s| \le \tilde{\mathbf{u}}_T\}}\Big) / \Big(\sum_{s=1}^{T} (\tilde{r}_{1,s})^2\, \mathbb{1}_{\{|\tilde{\mathbf{r}}_s| \le \tilde{\mathbf{u}}_T\}}\Big), \tag{19}$$

$$\hat{\delta}_j^d = \text{sign}\left\{\sum_{s=1}^{T} \text{sign}\{\tilde{r}_{1,s}\, \tilde{r}_{j,s}\} |\tilde{r}_{1,s}\, \tilde{r}_{j,s}|^{\tau}\right\} \left(\frac{\sum_{s=1}^{T} \text{sign}\{\tilde{r}_{1,s}\, \tilde{r}_{j,s}\} |\tilde{r}_{1,s}\, \tilde{r}_{j,s}|^{\tau}}{\sum_{s=1}^{T} (\tilde{r}_{1,s})^{2\tau}}\right)^{\frac{1}{\tau}}. \tag{20}$$

It is straightforward to see that the estimators (19) and (20) 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 (19) and (10) and the usual least square estimates. The only difference between them is that we use a truncation threshold to discard the jump component. Thus the use of Frisch-Waugh Theorem can be 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 an examination of the finite sample performance of the Todorov and Bollerslev (2010) estimators, Alexeev et al. (2014) 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 in the existing empirical applications of this method that the estimated discontinuous beta is almost always higher than the continuous one (Todorov and Bollerslev, 2010; Alexeev et al., 2014; Bollerslev et al., 2015; Sayeed et al., 2015), there may be reason to be concerned 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. (2014).

In this section we investigate whether the values of the parameters in model (2) have any impact on the finite sample estimation of the contagion coefficients $\hat{\delta}^c$ and $\hat{\delta}^d$. 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 $\beta_1^c = 1$, $\beta_2^c = 1$, $\beta_1^d = 1.2$, $\beta_2^d = 1.2$; that is with the discontinuous beta higher than continuous beta reflecting the existing evidence. 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 estimation errors $\left(\hat{\delta}^c - \delta^c\right)$ and $\left(\hat{\delta}^d - \delta^d\right)$ are plotted in Figures 1–4 for the different parameterizations investigated.

Plots in the left column of Figure 1 depict the estimation errors in the estimated continuous contagion factor $\left(\hat{\delta}^c - \delta^c\right)$ for varying values of β_1^c, while keeping the other three beta coefficients fixed at the benchmark setting. The magnitude of the estimation error is very small overall; as β_1^c ranges from 0.5 to 1.5, the size of the estimation error is always bounded below 0.05.

The estimations of the discontinuous contagion parameters exhibit larger errors. The difference between the true values of β_1^c and β_1^d appears to contribute to the extent of estimation error in the discontinuous contagion parameter. This result is analogous to the finding that the estimation errors in both continuous and discontinuous beta coefficients become smaller as the difference between β_1^c and β_1^d gets smaller in Alexeev et al. (2014). The difference between the true values of the contagion parameters δ^c and δ^d appear also to be important in determining the estimation error in the discontinuous contagion. As shown in the right column of Figure 1, there is a clear pattern of increase in the estimation error $\left(\hat{\delta}^d - \delta^d\right)$ with an increase in $\left(\delta^c - \delta^d\right)$. Whilst the error can be quite substantial for some combinations of δ^c and δ^d, for example, $\left(\hat{\delta}^d - \delta^d\right) \approx 1$ when $\delta^c = 1.8$ and $\delta^d = 0.2$, this degree of difference 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, in particular it is bounded below 0.2 in the most extreme cases of $\beta_1^c = 0.5$ and $\beta_1^c = 1.5$ and even smaller as β_1^c becomes closer to β_1^d.

The main results from above carry over to our other experiments. Plots presented in Figures 2–4 provide three other scenarios: Plot 2 varies β_2^c, the loading on the continuous component for the asset in receipt of contagion; Plot 3 varies β_1^d, the loading on the discontinuous parameter in the asset propagating the shock; and plot 4 varies β_2^d, the loading on the discontinuous component for the asset in receipt of contagion. Each scenario demonstrates the same results: the estimation error in the continuous contagion effect is negligible, whereas the discontinuous contagion exhibit larger estimation error. This larger estimation error for the discontinuous effect is evidently affected by three factors: the difference between β_1^c and β_1^d, the difference between β_2^c and β_2^d, and lastly, the difference between δ^c and δ^d. The sign of the error changes given different values of δ^c and δ^d. In the majority of cases, the estimates of $\hat{\delta}^d$ exceed the true value δ^d, and underestimation of δ^d only occurs when δ^d is much higher than δ^c. These findings are consistent with those reported by Alexeev et al. (2014) in the estimation error of discontinuous beta.

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

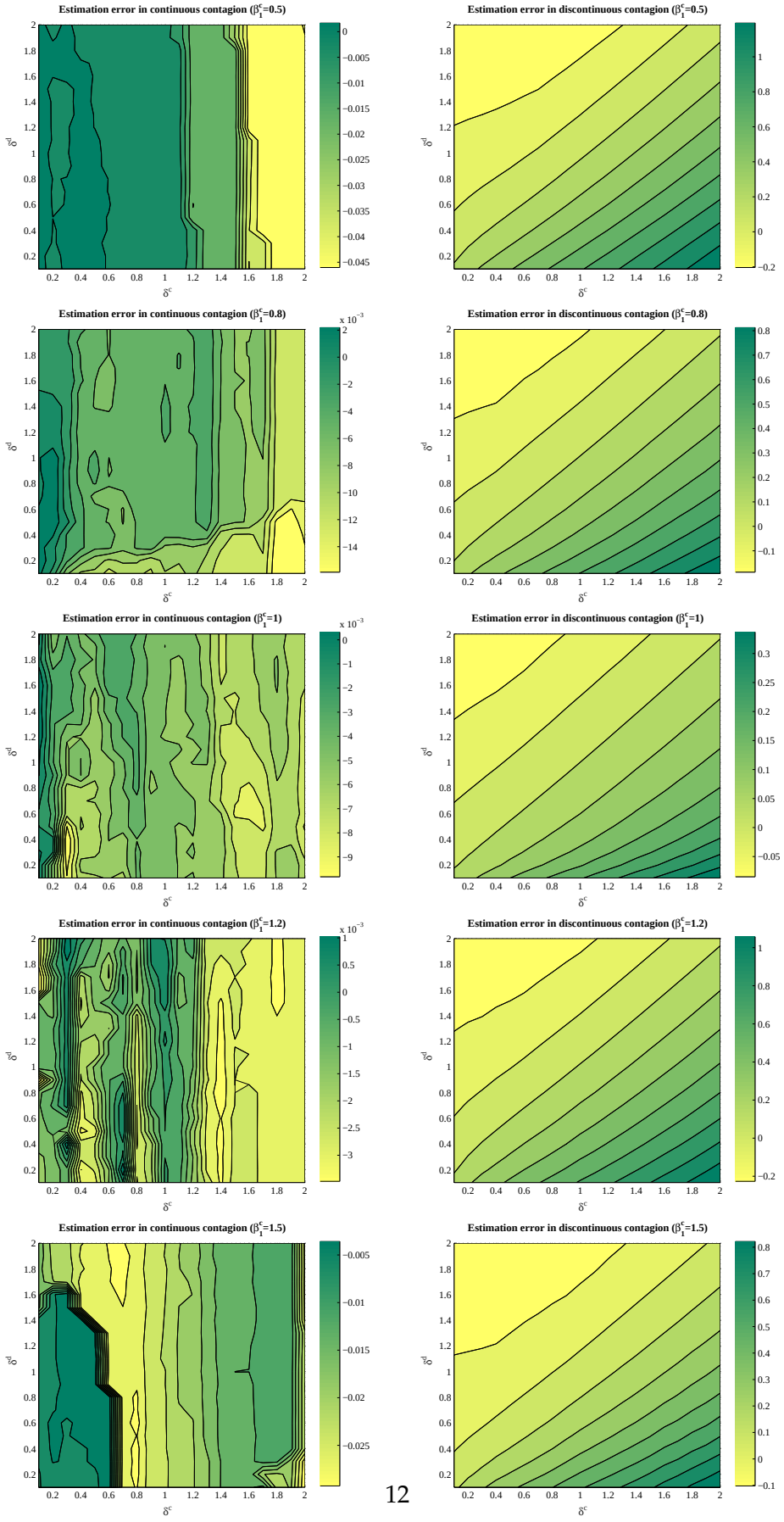


Figure 2: Estimation error in the second-stage contagion coefficients of $\hat{\delta}^c$ and $\hat{\delta}^d$ (parameters: $\beta_1^c = 1$, $\beta_1^d = 1.2$, $\beta_2^d = 1.2$)

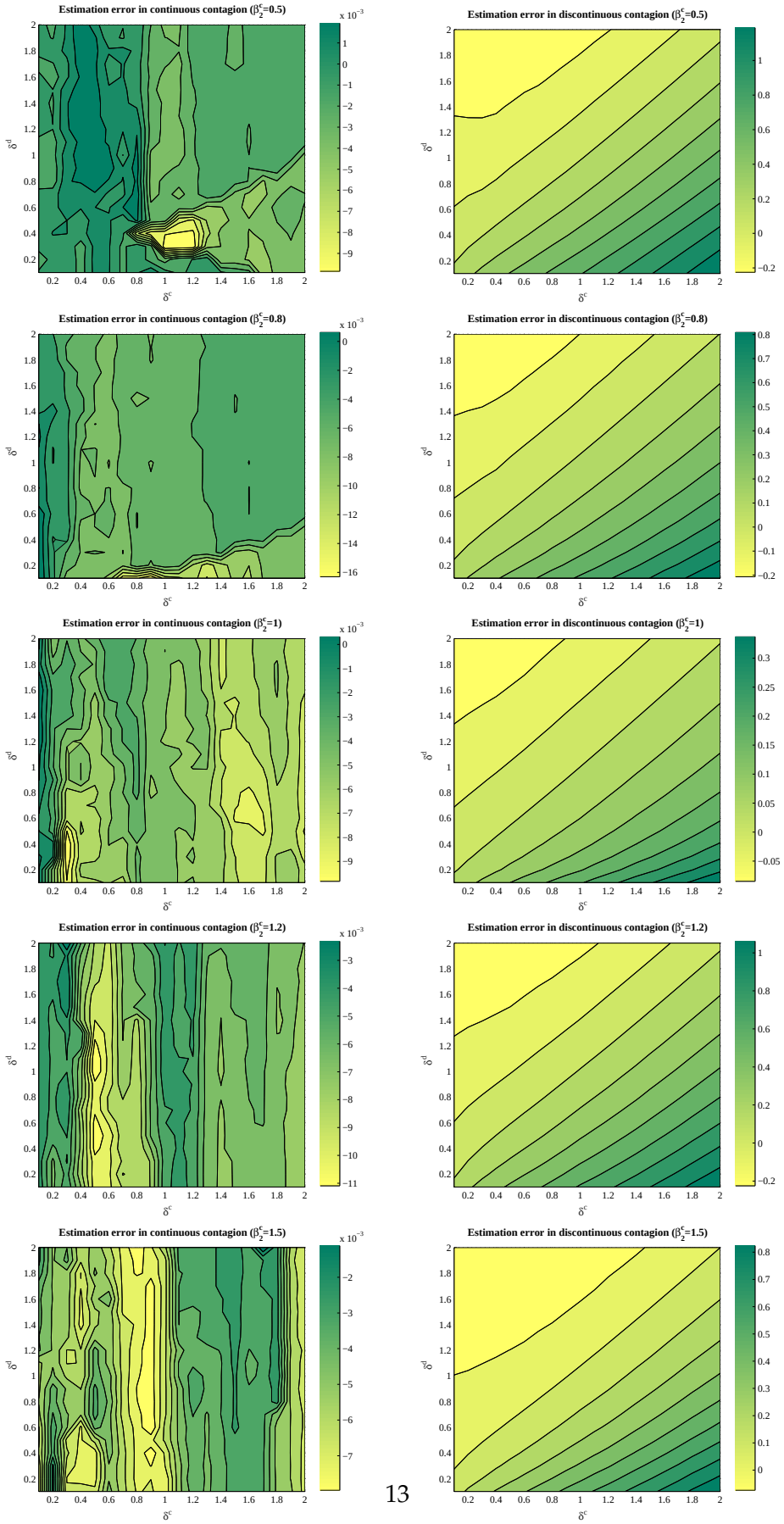


Figure 3: Estimation error in the second-stage contagion coefficients of $\hat{\delta}^c$ and $\hat{\delta}^d$ (parameters: $\beta_1^c = 1$, $\beta_2^c = 1$, $\beta_2^d = 1.2$)

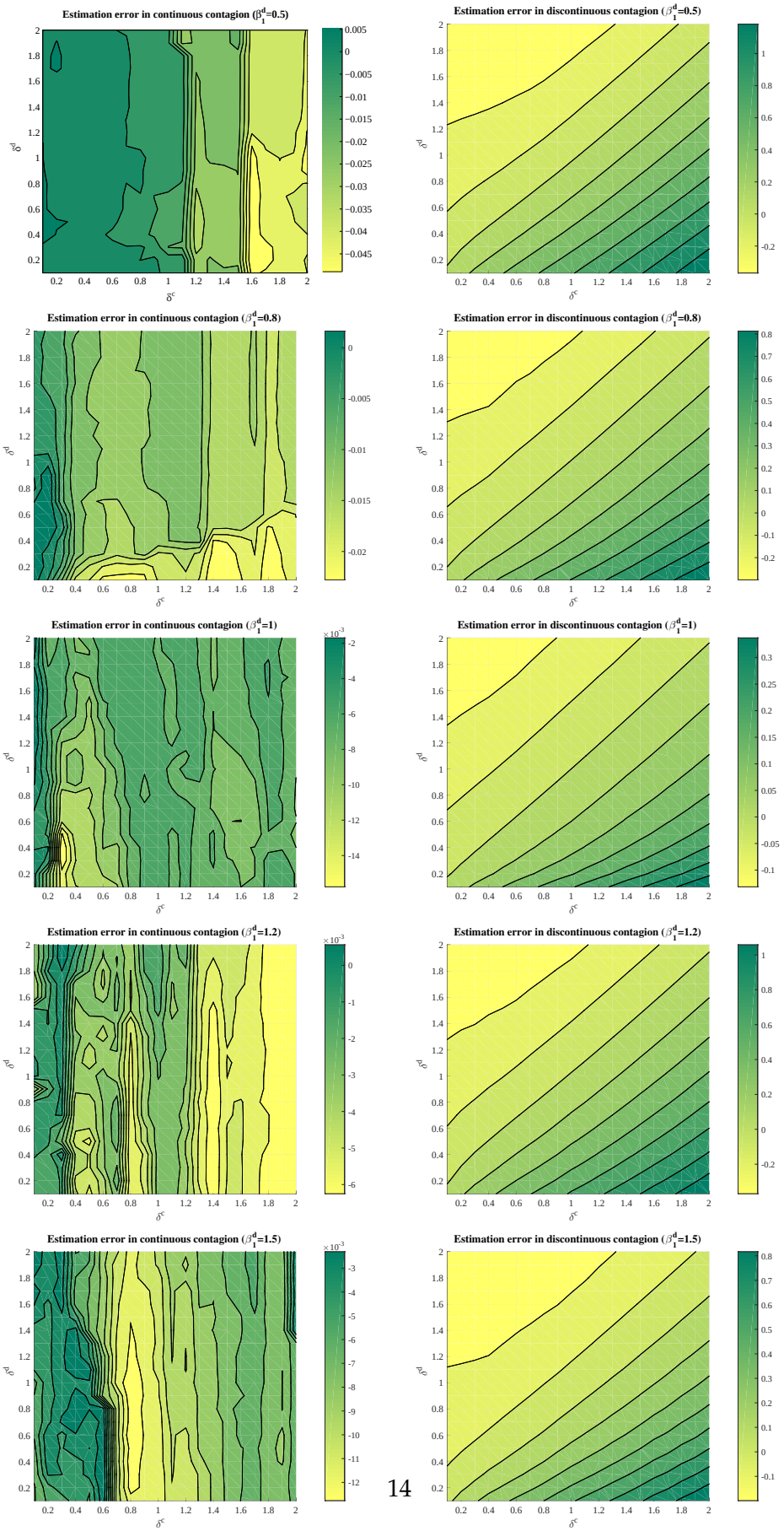
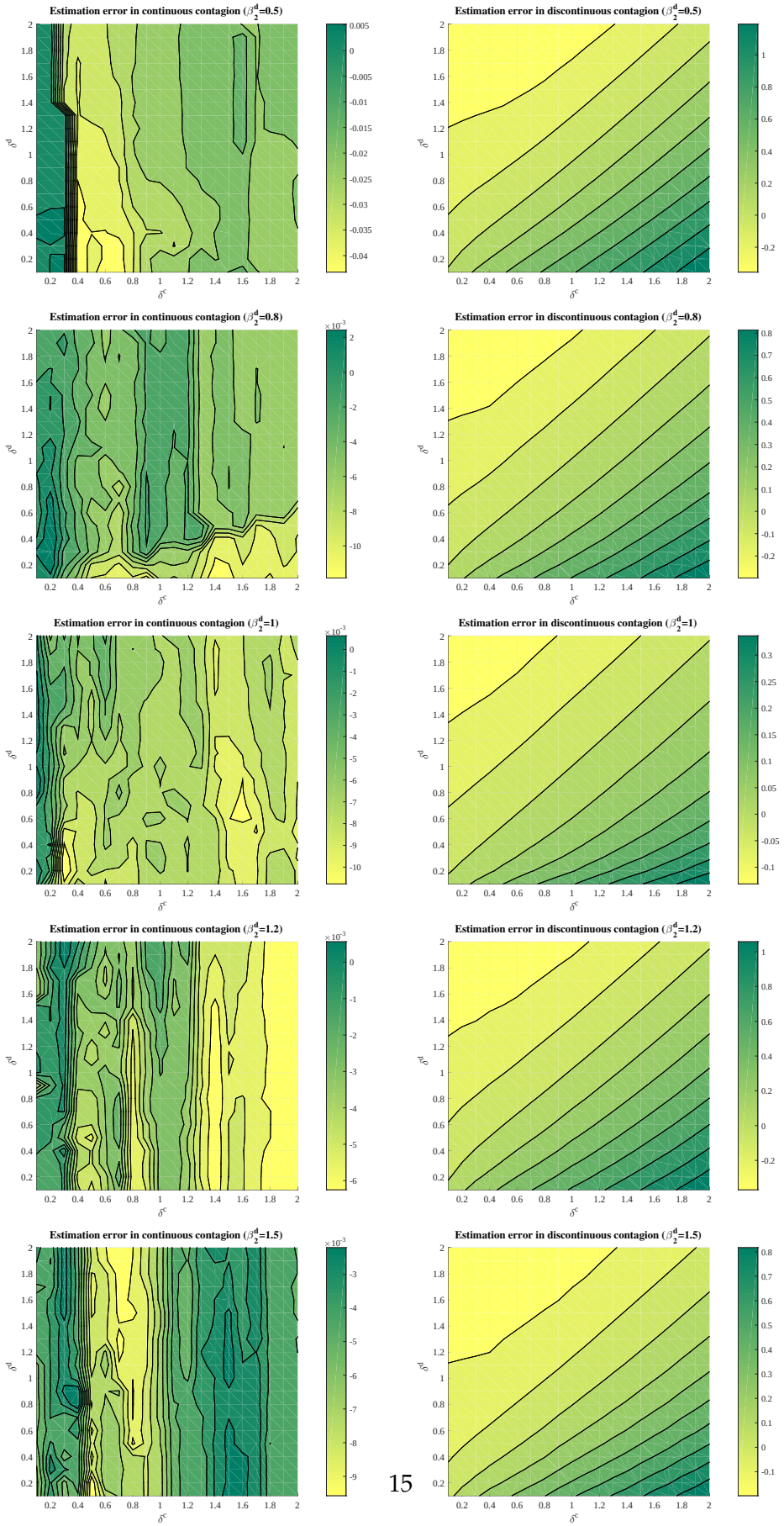


Figure 4: Estimation error in the second-stage contagion coefficients of $\hat{\delta}^c$ and $\hat{\delta}^d$ (parameters: $\beta_1^c = 1$, $\beta_2^c = 1$, $\beta_1^d = 1.2$)



We increase the sampling frequency to 1-minute and 10-second sampling in order to examine the asymptotic behaviour of the estimation approach proposed in Section 2. The continuous contagion parameter $\hat{\delta}^c$ displays only marginal estimation errors, most of which are of 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 more empirically realistic region of the parameter space that $\delta^d > \delta^c$, 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 asymptotically.

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 to Contagion in the US Financial Sector

The evidence for contagion during the financial crises of 2007-2013 is relatively well established with lower frequency data. 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 (2015), collateralized debt obligations (CDOs) in Longstaff (2010) and 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. (2014) and Broto and Perez-Quiros (2015).

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 2006 to 2008. 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. (Aalbers (2009) argues convincingly that the engagement of the banking sector in the housing market was critical to the spread of the crisis.)

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 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.[3] Harrington (2009) argues that although AIG was in the central stage during the GFC, insurance poses much less systemic risk than banking, and notes also that 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 investigate the debate of whether insurance companies are victims of the banks during the GFC. We estimate the contagion effects between insurance and banking stocks to examine this relationship. In particular we focus on the potential contagion effects from AIG and Bank of America, which were amongst the top 3 recipients of funding support from the Troubled Assets Relief Program (TARP) by mid-2009.[4] AIG was clearly a major focus of problems during the crisis period and its ongoing importance as reflected by its designation as a globally systemically important insurer (G-SII) by the Financial Stability Board. Bank of America has been central to ongoing assessments of systemically risky financial institutions in the US; it has been ranked within 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. (2010) since June 2008[5] – and since September 2008 it has frequently been ranked as the most systemically important – and ranked in the top 5 using the interconnectedness approach of Dungey et al. (2012), as well as being identified as a globally systemically important bank (G-SIB) by the Financial Stability Board. During 2007 Bank of America 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 long-running legal battles regarding settlements with other correspondents of the acquired institutions (including customers, shareholders and other counterparties).

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

[4]Citigroup received $50 billion, AIG received $40 billion, and Bank of America 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.

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

4.1 Data

The 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, obtained from the Thomson Reuters Tick History database via SIRCA, with intra-day returns and prices for the trading day from 9:30 am EST to 4:00 pm EST. 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.

The large dataset is used in its entirety to set the thresholds for the implementation; that is we use the characteristics of all 501 series to establish our threshold μ_T. This reduces the problem of different thresholds in examining different pairs of asset transmissions, and effectively means that we are able to simply produce contagion effects from any of the assets in the dataset to any other, and although we will not report on them individually in this paper they do allow us to form some impact effects for contagion from particular assets to the entirety of the market in a manner akin to that used in Patton and Verardo (2012) who estimate average effects on beta for news arrival across S&P500 stocks.

As Todorov and Bollerslev (2010) suggest, 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. After filtering out the market factor, we conduct the Lee and Mykland (2008) jump test on the asset chosen to be the source of the contagion to identify jumps still using a 10% significance level.

4.2 Are Insurers Victims of the Banks?

To illustrate our proposed model we estimate the contagion effects from AIG and Bank of America (BoA) to each other and Citigroup, Goldman Sachs (GS), ACE and Metlife. Each of these institutions is a major financial institution, with the three banks denoted as globally systemically important by the Financial Stability Board, and AIG and Metlife denoted as globally systemically important insurers. ACE is not only a large global insurer (although not denoted as globally systemically important at time of writing), but was also one of the three named firms in the investigation of concerns around industry practices regarding contingent commissions in 2004-2005 conducted by Eliot Sptizer; see Ghosh and Hilliard (2012) who estimate significant contagion from these firms to the remainder of the industry as a result of

this incident.

We plot the contagion coefficients for the exemplar recipient banks (first row) and insurance companies (second row) in Figure 5 for contagion from AIG, and Figure 6 for contagion from BoA. As with the relationship between the continuous and discontinuous betas in the existing empirical studies, the discontinuous contagion coefficient 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 is more contagious.

Consider first the question of whether the evidence for contagion from AIG on BoA differs to that for contagion from BoA to AIG, that is directly comparing Panel (a) of Figure 5 with that of Figure 6. The first aspect to note is that the impact parameters for the insurer on the bank are considerably smaller than those in the other direction - supporting the findings in Chen et al. (2014). The second result is that during the lead-up to the events of September 2008 the impact of the contagion effects from the insurer on the bank were *increasing* whilst those from the bank to the insurer were *decreasing*. This likely represents the increasing difficulties encountered by AIG during the build-up to its eventual rescue in October 2008. The most abrupt changes in both the continuous and discontinuous contagion coefficients for AIG to Bank of America 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 (see also Dungey et al., 2012).

In September 2008, the BoA dominates AIG as the contagion source, with a discontinuous contagion coefficient of over 2.6 on AIG, while the contagion 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).

We do not observe many abrupt changes in the contagion effects from BoA on the insurance companies in late 2008; compare the first row of Figure 5 with the second row of Figure 6. AIG is an exception – in Panel (a) of Figure 6 there are two peaks as previously noted. Panel (d) of Figure 6 indicates that there is some drop in the transmission to ACE, but in general the decline in contagion effects to the remaining insurers in the system are more consistent with a slow return to more normal conditions. Similarly, the contagion effects from BoA to other banks, Panels (b) and (c) of Figure 6 do not seem to have been as dramatically different during the periods of stress as was evident in the transmission to the banks from AIG in the same panels of Figure 5. It seems likely that this represents the systematic aspect of this crisis – these large banks were viewed as a sector that became systematically risky. One

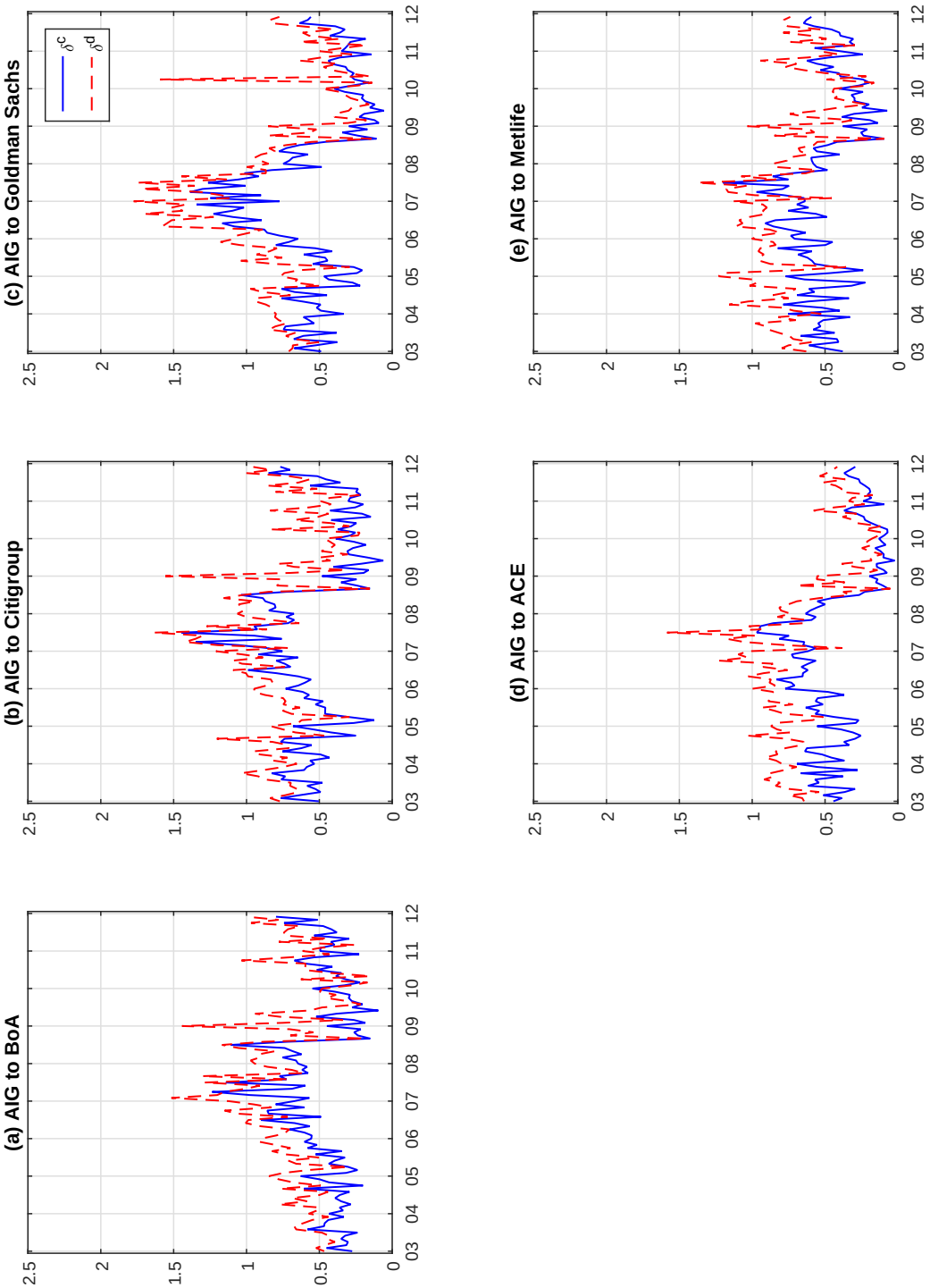


Figure 5: Contagion originated from AIG to banks and insurance companies

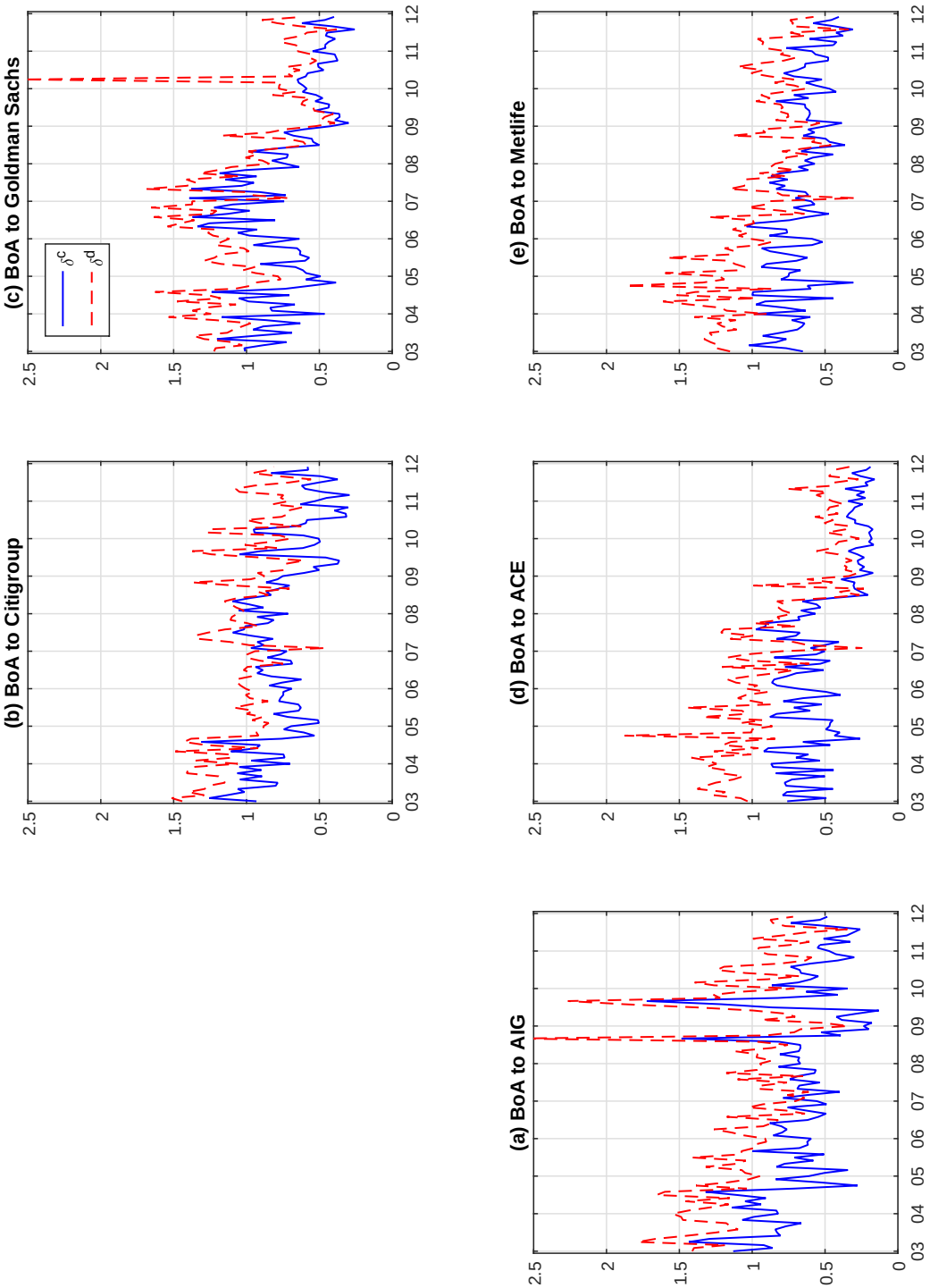


Figure 6: Contagion originated from BoA to banks and insurance companies

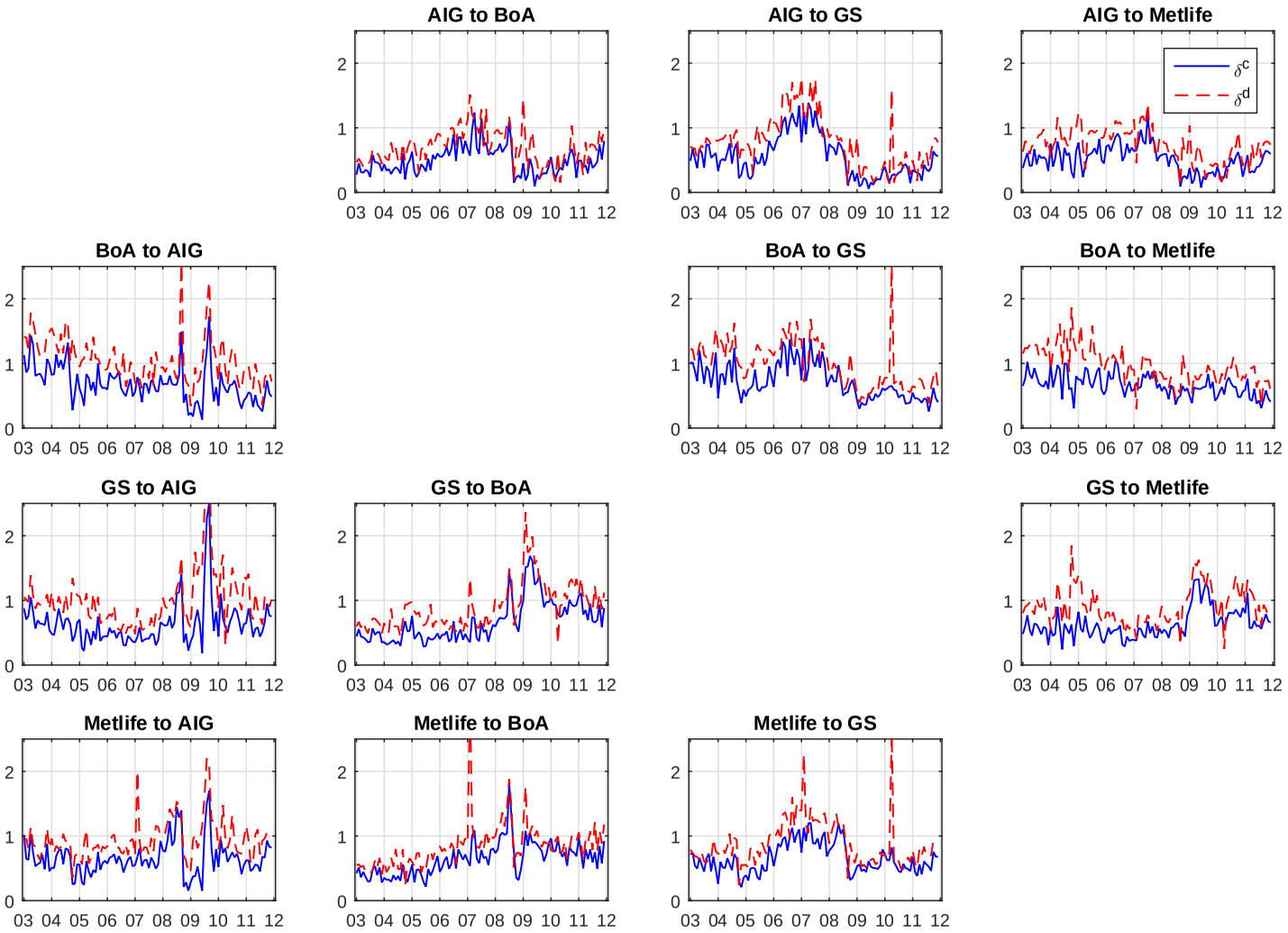
exception to this is in the relatively large estimate of discontinuous contagion from BoA to GS in April 2010, aligned with the escalation of the Greek debt crisis and revelations of Goldman Sachs' involvement in currency swaps for the Greek government. To explore this further we consider the evidence for contagion from GS, and another insurance company, Metlife, which is also a Globally Important Insurer.

Figure 7 provides the pairwise contagion results for AIG, BoA, GS and Metlife; the contrasting the contagion effects from source to target can be seen by comparing the corresponding panels on the two sides of the main diagonal in the figure. To benchmark, the first and second row of the figure report the contagion effects from AIG and BoA respectively, as discussed previously. The first two columns represent the contagion effects received by AIG and BoA, only the figures representing the contagion between these two assets themselves have been previously discussed. Contagion from GS is shown in the third row of Figure 7, and contagion to GS by the third column. GS was the heaviest user of the liquidity funds made available to the US banking sector in a bid to avert further systemic risk, was the major recipient of funds allocated to AIG via counterparty exposures and in the top two of largest counterparties for credit default swaps with AIG (McDonald and Paulson, 2015). The firm became visibly publicly unpopular, and was selected as one of the two case studies on poor practice in the investment banking sector by the US Senate (Senate, 2011). The contagion impact of GS on other institutions is clearly evident for AIG and BoA. In both cases there is a clear impact in September-October 2008 but the peak impact is felt in September 2009, when the first evidence of the Greek debt problems began to emerge with the first evidence of ratings watches and downgrades from Fitch and Moodys - the continuous and discontinuous contagion impacts on AIG are $\hat{\delta}^c = 2.5$ and $\hat{\delta}^d = 3.3$, with the discontinuous coefficient the highest impact recorded in our empirical evidence and beyond the vertical scale of the figures presented. The strong effects from Goldman Sachs to others are likely to represent its involvement in constructing debt instruments for Greece that have been subsequently widely criticized(for an overview see Featherstone, 2011). Notably, this is not as pronounced for Metlife.

Contagion effects to GS, represented in the third column of Figure 7 show, in contrast, that the effects from the other companies are concentrated in the discontinuous contagion component of the transmission process, and are contained to April 2010, the date when the Greek crisis became critical and uncertainty about a bailout peaked. It is evident from the Figure that at this point that there was substantial contagion from all firms represented to GS; potentially representing the counterparty and balance-sheet channels proposed for banking relationships in Kiyotaki and Moore (2002).

Contagion to Metlife, presented in column four of Figure 7, is considerably more muted than the other companies presented here. However, on occasion it has been associated with relatively large transmission of contagion effects presented in the fourth row of Figure 7.

Figure 7: Contagion between AIG, Bank of America, Goldman Sachs and Metlife



There are clear peaks in the transmission of discontinuous contagion from Metlife to GS and AIG in early 2010, consistent with our earlier analysis, but additionally there is a substantial flare in discontinuous contagion effects from Metlife to all other firms in the Figure – this date aligns with the February 2007 drop in Chinese markets which caused substantial drops in US markets. The contagion sourced from Metlife at this time may reflect its exposure to the Chinese economy through its direct presence in China via United Metlife in Shanghai; at the time its share price fell more than that of the other financial firms considered here, and did not rebound in the same manner.

Thus far we have examined the impact of these sources of contagion on a few key financial institutions. To obtain a more general view we extract the contagion impact of AIG, BoA, GS and Metlife 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 8. Panel (a) of Figure 8 provides the mean estimate of δ^c on banking sector stocks for each of the four source markets; Panel (b) provides the mean estimate of δ^c for the insurance companies in the sample. Panels (c) and (d) provide analogous plots for the mean δ^d estimates. 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 presented previously, there is no clear differentiation between the impact of contagion sourced from banks or insurers in any of the figures.

There is no clear evidence from these results that the average contagion impact from the source banks on the insurers outweighs the average contagion effect from the source insurers on the banks. While it seems that the insurers had smaller contagion impact in both continuous and discontinuous coefficients prior to the crisis, one of the banks, GS, also had similarly small impact, and in the run up to 2008 GS had a lower impact than the other firms. After the crisis the contagion impact from AIG on all banks in both continuous and discontinuous forms dropped to the least of the firms, but again the ranking of the other firms is mixed and changing. These mixed results are also evident in the average impact of the source firms on the insurance sector.

4.3 Contagion Effect on the Real Economy

Plots in Figures 9 provide some evidence on the contagion effects from AIG and BoA to some examples of real economy firms: General Electric, IBM and Exxon Mobil. General Electric is more influenced by contagion effects from BoA than AIG before the crisis, while both decrease down to around 0.5 after mid-2007, and maintain this lower level. There is a substantial increase in the size of the contagion effects from the AIG to Exxon Mobil in 2006-2007, before this reduces considerably. Firms from the technology sector seem to be mostly insulated from contagion effects from the banking sector, and although the impact of contagion from AIG is relatively low, a pronounced drop in this effect is evident following the rescue program for AIG. The contagion effects from AIG and BoA to IBM drop to values close to zero after

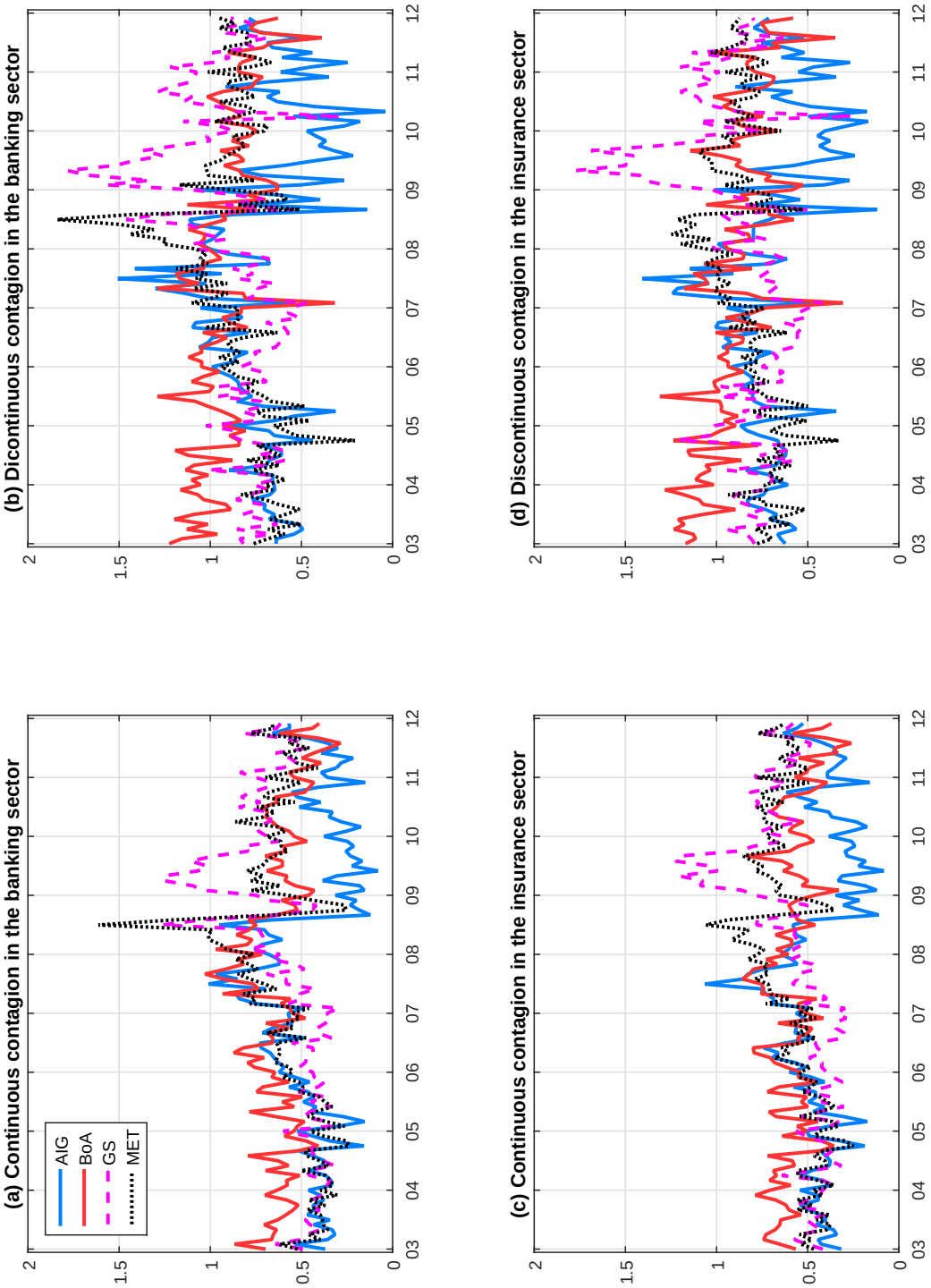


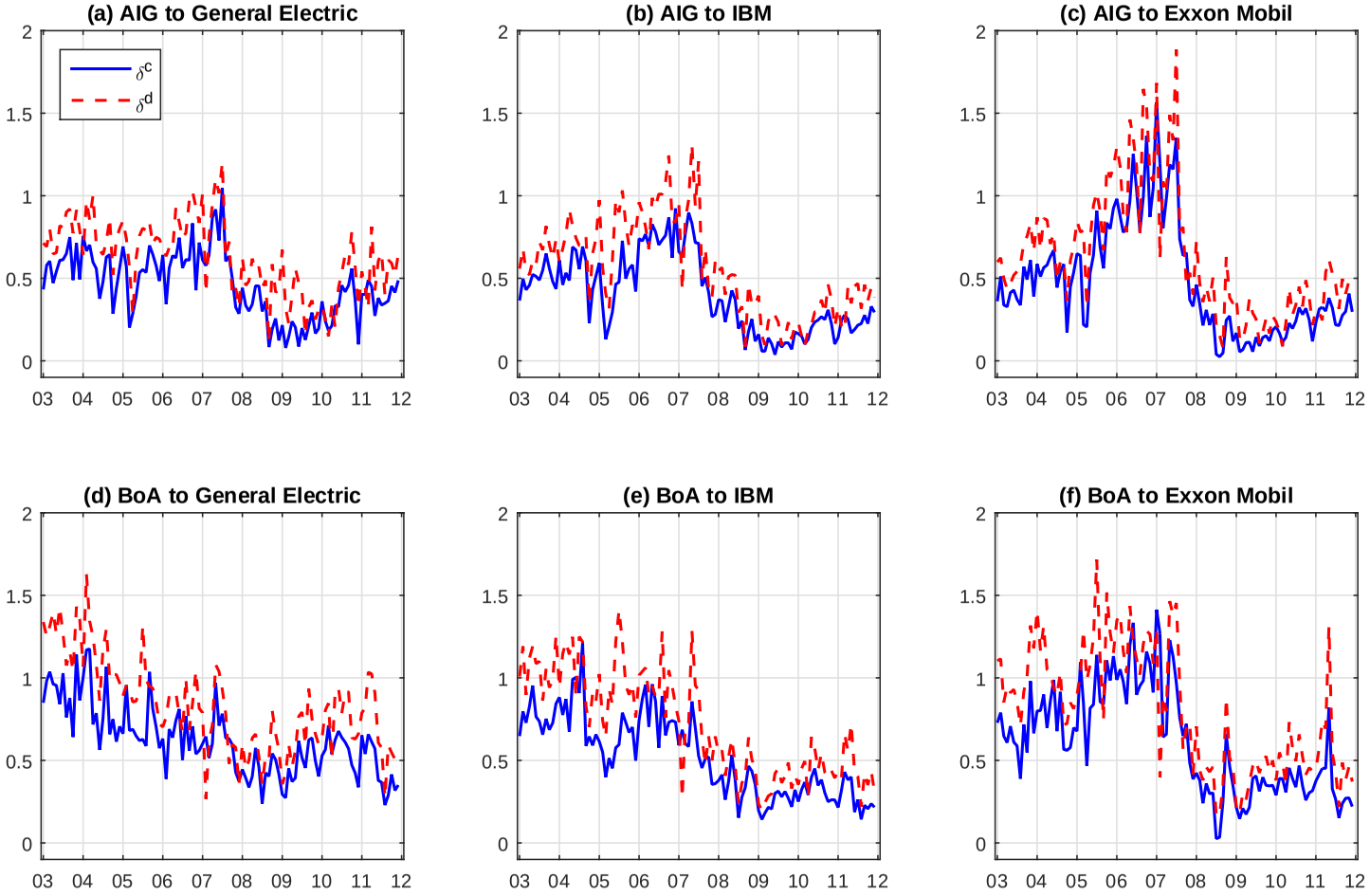
Figure 8: Contagion effects on insurance firms and banks (averaged within the sector)

2008. These stocks become the safe heaven for investors when most of the financial firms are struggling to recover. This disconnection between financial and real economy sectors may reflect the success of policies implemented by regulators and governments to rescue many financial institutions, in order to minimize the impact on the real economy. The reduction in the impact of contagion on Exxon Mobil from AIG, and to a lesser extent from BoA provides another example to support this hypothesis. However, the drop in the contagion effects from the financial sector firms to the real economy occurs in advance of the main US government interventions in the US - most are associated with drops after July 2007, aligned with the period in which the European Central Bank first intervened to support liquidity, easing the difficult credit conditions that prevailed at the time.

Further evidence on the effect of contagion sourced from AIG and BoA to the non-financial sectors is provided in Figure 10, where red dots display the minimum and maximum values of the estimated δ^c or δ^d, whiskers are the 2.5^{th} to 97.5^{th} percentile, boxes show the interquartile range, and black dots are the median. It is immediately apparent that not only is the median estimate of the continuous contagion coefficient lower than the discontinuous contagion coefficient, the range of estimates is also smaller. In the pre-crisis period, the continuous contagion estimates show a much wider range and slightly higher median than post 2008 for both AIG and BoA to the real economy in Panels (a) and (c) of Figure 10. 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. 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 contagion 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. Of particular interest is that there appears to be little to choose between the impact of AIG or BoA in terms of continuous contagion effects on the real economy firms as a whole. In terms of the discontinuous component of contagion, whilst the initial pre-crisis impact of contagion from AIG is lower than that from BoA in general, the impact from AIG increases to be generally above that from BoA at the peak, and there is little to choose between them in terms of the median effects in the post 2007 period - although the BoA effects have a slightly wider range. In this respect, these examples suggest that these 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 apparently contradictory findings in the literature. On the one hand the impact of contagion from the specific

Figure 9: Contagion originated from AIG and Bank of America to real economy firms



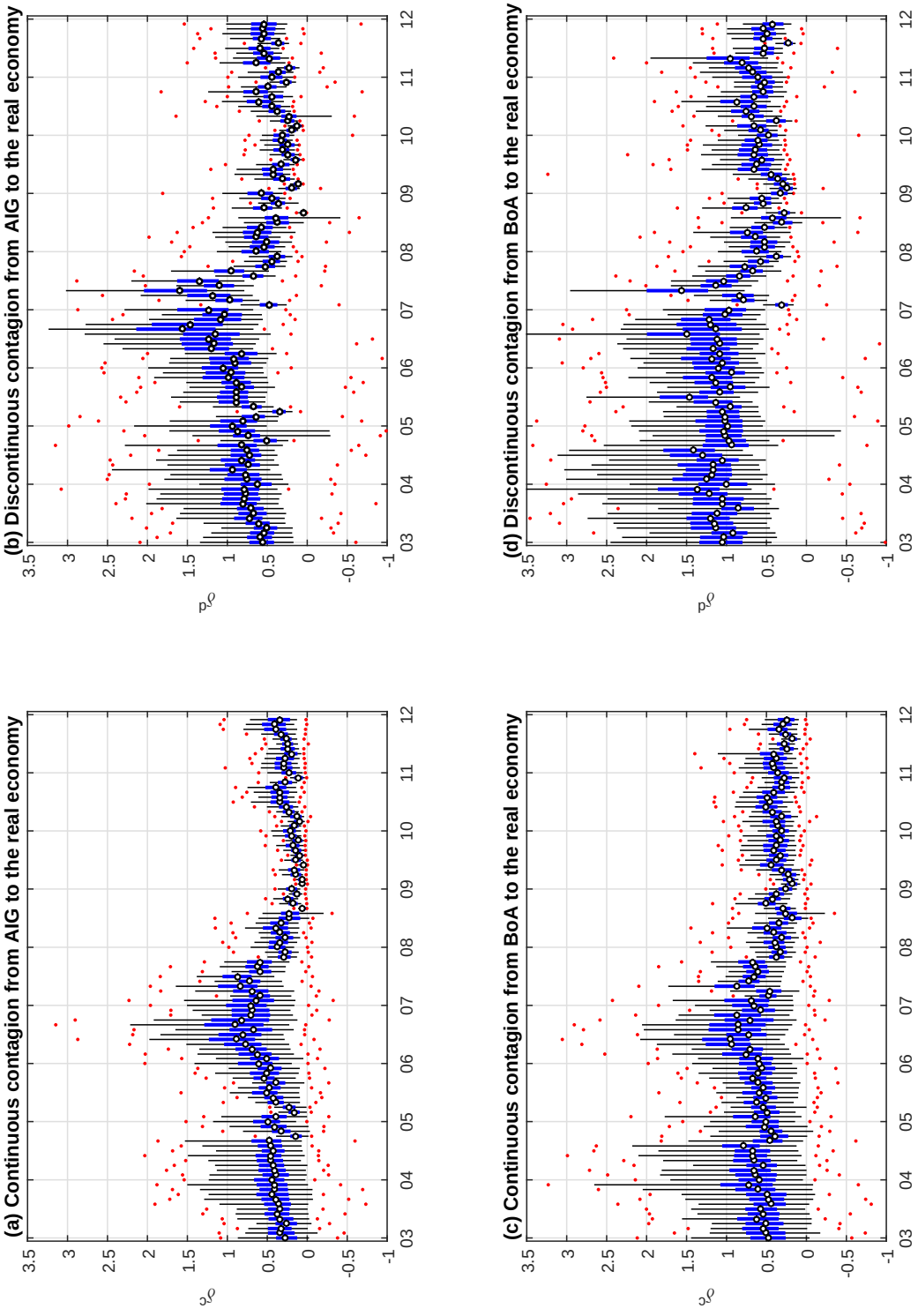


Figure 10: Contagion originated from AIG and Bank of America to real economy firms

banks and insurance companies investigated here, BoA, GS, AIG and Metlife, shows that the estimated effects support that contagion from banks has larger impact on insurance companies for these cases, consistent with the evidence in Chen et al. (2014) and Harrington (2009). However, when we consider sector level effects, it is difficult to differentiate the average impact on banks or insurers from these sources, consistent with Acharya and Richardson (2014). Further, and not inconsistent with the finding with these papers, while we may debate whether banks or insurers have most impact on each other, the effects on the real economy from contagion from either source are similar. While we acknowledge that we have not explored the impact of contagion from all possible insurers or banks in the sector, these results do provoke some interesting results. On one hand, if we believe that AIG is somehow different to 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 challenges to AIG to other firms in the insurance business, suggesting that there are strong commonalities between AIG and other firms, so that AIG is not behaving as some form of pseudo-bank but is in fact indicative of the entire insurance sector.

In summary, the empirical evidence presented from this framework finds that while contagion effects from banks to insurers may be larger than the impact of contagion from insurers to banks in individual cases, there is limited evidence of this on an industry-wide scale, and further, the impact of shocks from either part of the financial sector is similar for real economy firms. Insurance and banking both have important roles to play in transmitting financial sector shocks (and potentially systemic risk) to the real economy. If the aim of financial regulation is to defuse the potential transmission of this risk to the real economy, both sectors of the financial industry are important.

5 Conclusion

This paper contributes a new methodological 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 a relatively small literature applying it to detection of crisis conditions; see Aït-Sahalia et al. (2015) on contagion, Dungey, Holloway, Yalama and Yao (2015) and Dungey, Matei and Treepongkaruna (2015) on the changing nature of high frequency data during crises, and Black et al. (2012) on detecting jumps during periods of stress. Combining the established latent factor approach based on CAPM 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 small 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 by Alexeev et al. (2014).

The methodology is illustrated with an empirical application to detecting the extent of contagion from major US banks and insurance companies to other S&P500 firms. Contagion effects from Bank of America and Goldman Sachs, and from the insurance giants AIG and Metlife are estimated for a specific selection of firms from the banking, insurance and real economy sectors of the US economy. Distinguishing discontinuous contagion from continuous contagion empirically confirms that the former generally has a higher impact coefficient than the latter - consistent with the idea that more unanticipated news arrival is absorbed more quickly. The results allow us to contribute to the debate on whether financial regulators should be concerned about the potential for transmission of financial shocks via insurance companies. Our findings support that the impact of contagion from the banks on insurers outweighs the impact of insurers on banks in particular cases, consistent with the small existing empirical literature. However, when we consider industry-wide averages, the contagion impact coefficients of our source banks (BoA and GS) on all the insurance companies in the sample is not clearly larger than the contagion impact coefficients of our source insurers (AIG and Metlife) on all banks in the sample; the results are quite mixed and change over the sample period. Further, when we consider the real economy firms, the effects of the source banks has a somewhat higher range of effect on the real economy firms than that from the source insurers, but it is difficult to discern a substantial difference between the median and interquartile range impacts. The results support the view that both the source banks and the source insurance companies play a role in transmitting financial sector turmoil to the firms of the real economy. If the aim of regulators is to reduce the impact of financial sector risk on the real economy then oversight of the activities of both arms of the financial sector seems warranted.

Acknowledgement

We are grateful for comments from Vitali Alexeev, Alexandre Dmitriev, Petko Kalev, Jia Li, Jing Tian, Lars Winkelmann, Farshid Vahid, Jacob Wong and seminar participants at Tasmanian School of Business and Economics, University of Tasmania; Department of Econometrics and Business Statistics, Monash University; School of Business and Economics, Free University of Berlin. This research is supported by funding from ARC DP130100168. This research was partly written while Dungey was a Hallsworth Fellow at the University of Manchester, and she is grateful for their hospitality.

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