Econometric Modeling of Systemic Risk: Going Beyond Pairwise Comparison and Allowing for Nonlinearity

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Abstract

Financial instability and its destructive effects on the economy can lead to financial crises due to its contagion or spillover effects to other parts of the economy. Having an accurate measure of systemic risk gives central banks and policy makers the ability to take proper policies in order to stabilize financial markets. Much work is currently being undertaken on the feasibility of identifying and measuring systemic risk. In principle, there are two main schemes to measure interlinkages between financial institutions. One might wish to construct a mathematical model of financial market participant relations as a network/graph by using a combination of information extracted from financial statements like the market value of liabilities of counterparties, or an econometric model to estimate those relations based on financial series. In this paper, we develop a data-driven econometric framework that promotes an understanding of the relationship between financial institutions using a nonlinearly modified Granger-causality network. Unlike existing literature, it is not focused on a linear pairwise estimation. The method allows for nonlinearity and has predictive power over future economic activity through a time-varying network of relationships. Moreover, it can quantify the interlinkages between financial institutions. We also show how the model improve the measurement of systemic risk and explain the link between Granger-causality network and generalized variance decompositions network. We apply the method to the monthly returns of U.S. financial Institutions including banks, broker and insurance companies to identify the level of systemic risk in the financial sector and the contribution of each financial institution.

Keywords: Systemic risk; Risk Measurement; Financial Linkages and Contagion; Nonlinear Granger Causality; Directed Information Graphs

JEL classification: G1, G14, G21, G28, G31, C14, C51, D8, D85

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I. Introduction

Understanding the interconnection between the financial institutions is of great importance. In principle, there are two main approaches to measure such interconnections between financial institutions in the literature. One is based on a mathematical model of financial market participant relations as a graph using a combination of information extracted from financial statements like the market value of liabilities of counterparties, and the other one that is also the approach of this work is based on statistical analysis of financial series.

Most of the existing approaches in the literature are built on pairwise comparison or assuming linear relationship between the time series. For instance the authors in Billio et al. (2012) propose several measures of systemic risk to capture the connections between the monthly returns of different financial institutions (hedge funds, banks, brokers, and insurance companies) based on Granger-causality tests. They propose a definition of systemic risk as "any set of circumstances that threatens the stability of or public confidence in the financial system". This definition implies that the risk of such events is unlikely to be captured by any single metric that ignores the connections between the financial institutions. Billio et al. (2012) uses principle component analysis to estimate the number and importance of common factors driving the returns of financial institutions, and it uses *pairwise* Granger-causality tests to identify the network of Granger-causal relations among those institutions.

Another related work is Diebold and Yılmaz (2014). In this work, the authors propose a connectedness measure based on generalized variance decomposition (GVD) and consequently, define a weighted, directed network. However, the measure introduced in this work is limited to linear dynamical systems, more precisely, data-generating processes (DGPs). Moreover, as we will discuss later in Section III.B, their measure suffers from disregarding the entire network akin to pairwise analysis commonly used in the literature.

In Barigozzi and Hallin (2016), the authors focus on one particular network structure: the long-run variance decomposition network (LVDN). Similar to Diebold and Yılmaz (2014), the LVDN defines a weighted and directed graph where the weight that is associated with edge (i, j) represents the proportion of h-step-ahead forecast error variance of variable i which is accounted for by the innovations in variable j. LVDNs are characterized by the infinite vector moving average (VMA) that limits them to linear systems.

Connectedness measures based on correlation remain widespread, however, they measure only pairwise association and are mainly used for linear Gaussian dynamics. This makes them of limited value in financial-market contexts. Different approaches have been developed to relax these conditions. For example, equi-correlation approach in Engle and Kelly (2012) uses average correlations across all pairs. The CoVaR approach of Adrian and Brunnermeier (2008) measures the value-at-risk (VaR) of financial institutions conditional on other institutions experiencing financial distressand. The marginal expected shortfall (MES) approach of Acharya et al. (2010) measures the expected loss to each financial institution conditional on the entire set of institutions poor performance. Although these measures rely less on linear Gaussian methods and are certainly of

interest, they measure different things, and a general framework that can be used to capture the connectedness in different networks remains elusive. Introducing such measure is the main purpose of this work.

In this work, we develop a method that allows for nonlinearity of the data and does not depend on pairwise relationships among time series. We also show how the model improve the measurement of systemic risk and explain the connection between Granger-causality and variance decompositions method.

A. *Organization*

The rest of the paper is organized as follows. In Section II, we review the literature on graphical models, Granger causality, and introduce directed information graphs. In Section III, we study the causal network of linear models. Section IV studies the causal network of non-linear models. In Section V, we apply our non-linear method to learn the causal network of set of financial institutions and compare it with the linear regression method in the literature. Finally, we conclude in Section VI.

II.   Causal Network

In order to investigating the dynamic of systemic risk, it is important to measure the causal relationship between financial institutions. In this section, we propose a statistical approach to learn such causal interconnections using Granger causality Granger (1969).

A. *Graphical Models and Granger Causality*

Researchers from different fields have developed various graphical models suitable for their application of interest to encode interconnections among variables or processes. Markov Networks, Bayesian networks (BNs), and Dynamic Bayesian networks (DBNs) are three example of such graphical models that have been used extensively in the literature. In these particular graphical models, nodes represent random variables Koller and Friedman (2009); Murphy (2002).

Markov networks are undirected graphs that represent the conditional independence between the variables. On the other hand BNs and DBNs are directed acyclic graphs (DAGs) that encode conditional dependencies in a reduced factorization of the joint distribution.

Since the size of such graphical models depends on the time-homogeneity and the Markov order of the random processes. Therefore, in general, the graphs can grow with time. As an example, the DBN graph of a vector autoregressive (VAR) with m processes each of order L requires mL nodes Dahlhaus and Eichler (2003). As such they are not suitable for succinct visualization of relationships between the time series such as systemic risks.

In this work, we use directed information graphs (DIGs) to represent interconnections among the financial institutions in which each node represents a time series Quinn et al. (2015); Massey (1990). Below, we formally introduce this type of graphical models. We use an information-theoretical

generalization of the notion of Granger causality to determine the interconnection between time series. The basic idea in this framework was originally introduced by Wiener Wiener (1956), and later formalized by Granger Granger (1969). The idea reads as follows: "we say that X is causing Y if we are better able to predict the future of Y using all available information than if the information apart from the past of X had been used."

Granger formulated this framework for practical implementation using multivariate autoregressive (MVAR) models and linear regression. This version has been widely adopted in econometrics and other disciplines Granger (1963); Dufour and Taamouti (2010). More precisely, in order to identify the influence of X_t on Y_t in a MVAR comprises of three time series $\{X, Y, Z\}$, Granger's idea is to compare the performance of two linear regressions: the first predictor is non-nested that is it predicts Y_t given $\{X^{t-1}, Y^{t-1}, Z^{t-1}\}$, where X^{t-1} denotes the time series X up to time $t-1$ and the second predictor is nested that is it predicts Y_t given $\{Y^{t-1}, Z^{t-1}\}$. Clearly, the performance of the second predictor is bounded by the first predictor and if they have the same performance, then we say X does not cause Y. In this framework, since the dynamic between time series is linear, linear regression has been chosen. Next, we introduce directed information (DI), an information-theoretical measure that generalized Granger causality beyond linear models Quinn et al. (2011a).

DI has been used in many applications to infer causal relationships. For example, it has been used for analyzing neuroscience data Quinn et al. (2011b); Kim et al. (2011) and market data Etesami and Kiyavash.

B. Directed Information Graphs (DIGs)

In the rest of this section, we describe how the DI can capture the interconnections in causal[1] dynamical systems (linear or non-linear) and formally define DIGs.

Consider a dynamical system comprised of three time series $\{X, Y, Z\}$. To answer whether X has influence on Y or not over time horizon $[1, T]$, we compare the average performance of two particular predictors with predictions p and q over this time horizon. The first predictor uses the history of all three time series while the second one uses the history of all processes excluding process X. On average, the performance of the predictor with less information (the second one) is upper bounded by the performance of the predictor with more information (the first one). However, when the prediction of both predictors, i.e., p and q are close over time horizon $[1, T]$, then we declare that X does not cause Y in this time horizon; otherwise, X causes Y.

In order to measure the performance of a predictor, we consider a nonnegative loss function, $\ell(p, y)$, which defines the quality of the prediction. This loss function increases as the prediction p deviates more from the true outcome y. Although there are many candidate loss functions, e.g. the squared error loss, absolute loss, etc, for the purpose of this work we consider the logarithmic loss.

Moreover, in our setting, the prediction p lies in the space of probability measures over y. More precisely, we denote the past of all processes up to time $t1$ by $\mathcal{F}^{t-1}$ that is the σ-algebra generated

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by $\{X^{t-1}, Y^{t-1}, Z^{t-1}\}$, where X^{t-1} represents the time series X up to time $t-1$, and denote the past of all processes excluding process X, up to time $t-1$ by $\mathcal{F}^{t-1}_{-X}$.

The prediction of the first predictor that is non-nested at time t is given by $p_t := P(Y(t)|\mathcal{F}^{t-1})$ that is the conditional distribution of $Y(t)$ given the past of all processes and the second predictor which is nested is given by $q_t := P(Y_t|\mathcal{F}^{t-1}_{-X})$.

Given a prediction p for an outcome $y \in \mathcal{Y}$, the log loss is defined as $\ell(p, y) := -\log p(y)$. This loss function has meaningful information-theoretical interpretations. The log loss is the Shannon code length, i.e., the number of bits required to efficiently represent a symbol y drawn from distribution p. Thus, it may be thought of the description length of y.

When the outcome y_t is revealed for Y_t, the two predictors incur losses $\ell(p_t, y_t)$ and $\ell(q_t, y_t)$, respectively. The reduction in the loss (description length of y_t), known as regret is defined as

$$r_t := \ell(q_t, y_t) - \ell(p_t, y_t) = \log \frac{p_t}{q_t} = \log \frac{P(Y_t = y_t|\mathcal{F}^{t-1})}{P(Y_t = y_t|\mathcal{F}^{t-1}_{-X})} \geq 0.$$

Note that the regrets are non-negative. The average regret over the time horizon $[1, T]$ given by $\frac{1}{T} \sum_{t=1}^{T} \mathbb{E}[r_t]$, where the expectation is taken over the joint distribution of X, Y, and Z is called *directed information* (DI). This will be our measure of causation and its value determines the strength of influence. If this quantity is close to zero, it indicates that the past values of time series X contain no significant information that would help in predicting the future of time series Y given the history of Y and Z. This definition may be generalized to more than 3 processes as follows,

Definition 1: *Consider a network of m time series $\underline{R} := \{R_1, ..., R_m\}$. We declare R_i influences R_j over time horizon $[1, T]$, if and only if*

$$I(R_i \to R_j || \underline{R}_{-\{i,j\}}) := \frac{1}{T} \sum_{t=1}^{T} \mathbb{E}\left[\log \frac{P(R_{j,t}|\mathcal{F}^{t-1})}{P(R_{j,t}|\mathcal{F}^{t-1}_{-\{i\}})}\right] > 0, \tag{1}$$

where $\underline{R}_{-\{i,j\}} := \underline{R} \backslash \{R_i, R_j\}$. $\mathcal{F}^{t-1}$ denotes the sigma algebra generated by $\underline{R}^{t-1} := \{R_1^{t-1}, ..., R_m^{t-1}\}$, and $\mathcal{F}^{t-1}_{-\{i\}}$ denotes the sigma algebra generated by $\{R_1^{t-1}, ..., R_m^{t-1}\} \backslash \{R_i^{t-1}\}$.

Definition 2: *Directed information graph (DIG) of a set of m processes $\underline{R} = \{R_1, ..., R_m\}$ is a weighted directed graph $G = (V, E, W)$, where nodes represent processes ($V = \underline{R}$) and arrow $(R_i, R_j) \in E$ denotes that R_i influences R_j with weight $I(R_i \to R_j || \underline{R}_{-\{i,j\}})$. Consequently, $(R_i, R_j) \notin E$ if and only if its corresponding weight is zero.*

Remark 1: *Pairwise comparison has been applied in the literature to identify the causal structure of time series Billio et al. (2012, 2010); Allen et al. (2010). Such comparison is not correct in general and fails to capture the true underlying network as we will see in the next example. For more details please see Quinn et al. (2015).*

Example 1: *As an example, consider a network of three times series $\{X, Y, Z\}$ with the following*

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linear model:

$$X_t = a_1 X_{t-1} + a_2 Z_{t-1} + \epsilon_{x_t},$$
$$Z_t = a_3 Z_{t-1} + \epsilon_{z_t}, \qquad (2)$$
$$Y_t = a_4 Y_{t-1} + a_5 Z_{t-1} + \epsilon_{y_t},$$

where ϵ_x, ϵ_y, and ϵ_z are three independent white noise processes, and $\{a_1, ..., a_5\}$ are non-zero coefficients of the model. Due to the functional relationships between these time series, we have that the causal network of this model is $X \leftarrow Z \rightarrow Y$, i.e., there is an arrow from Z to X and Z to Y because X_t and Y_t depend on Z_{t-1}, respectively. This can also be inferred using the DIs in (1), it is straight forward to show that

$$I(X \rightarrow Y || Z) = 0, \quad I(X \rightarrow Z || Y) = 0,$$
$$I(Y \rightarrow X || Z) = 0, \quad I(Y \rightarrow Z || X) = 0,$$
$$I(Z \rightarrow Y || X) > 0, \quad I(Z \rightarrow X || Y) > 0.$$

Notice that none of the above DIs are pairwise as they have conditioned on the remaining time series. However, considering the pairwise causal relationships, for instance between X and Y will give us

$$I(X \rightarrow Y) = \frac{1}{T} \sum_{t=1}^{T} \mathbb{E} \left[\log \frac{P(Y_t | Y^{t-1}, X^{t-1})}{P(Y_t | Y^{t-1})} \right] > 0.$$

Hence, looking into pairwise causal relationships, we obtain that X directly causes Y that is not the case in this example.

A causal model allows a factorization of the joint distribution in some specific ways. It was shown in (Quinn et al., 2015) that under a mild assumption, the joint distribution of a causal discrete-time dynamical system with m time series can be factorized as follows,

$$P_{\underline{R}} = \prod_{i=1}^{m} P_{R_i || \underline{R}_{B_i}}, \qquad (3)$$

where $B_i \subseteq -\{i\} := \{1, ..., m\} \setminus \{i\}$ is the minimal[2] set of processes that causes process R_i, i.e., parent set of node i in the corresponding DIG. Such factorization of the joint distribution is called minimal generative model. In Equation (3), $P(\cdot || \cdot)$ is called causal conditioning and defined as follows

$$P_{R_i || \underline{R}_{B_i}} := \prod_{t=1}^{T} P_{R_{i,t} | \underline{\mathcal{F}}_{B_i \cup \{i\}}^{t-1}},$$

and $\mathcal{F}_{B_i \cup \{i\}}^{t-1} = \sigma\{\underline{R}_{B_i \cup \{i\}}^{t-1}\}$.

It is important to emphasize that learning the causal network using DI does not require any specific model for the system. There are different methods that can estimate (1) given i.i.d. samples of the time series such as plug-in empirical estimator, k-nearest neighbor estimator, etc Jiao et al. (2013); Frenzel and Pompe (2007); Kraskov et al. (2004).

In general, estimating DI in (1) is a complicated task and has high sample complexity. However,
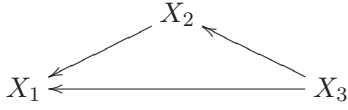
Figure 1. Corresponding DIG of the system in (4).

knowing some side information about the system can simplify the learning task. In the following section, we describe learning the causal network of linear systems. Later in Section IV, we discuss generalization to non-linear models.

C. Quantifying Causal Relationships

The purpose of this section is to justify that the DI introduced in (1) also quantifies the causal relationships in a network. We do so using a simple linear model and then generalize it to nonlinear systems.

Consider a network of three time series $\vec{X}_t = (X_{1,t}, X_{2,t}, X_{3,t})^T$ with the following dynamic

$$\vec{X}_t = \begin{pmatrix} 0 & 0.1 & 0.3 \\ 0 & 0 & -0.2 \\ 0 & 0 & 0 \end{pmatrix} \vec{X}_{t-1} + \vec{\epsilon}_t, \tag{4}$$

where $\vec{\epsilon}_t$ denotes a vector of exogenous noises that has normal distribution with mean zero and covariance matrix $\mathbf{I}$. Figure 1 shows the corresponding DIG of this network. Note that in this particular example that the relationships are linear, the support of the coefficient matrix also encodes the corresponding DIG of the network.

In order to compare the strength of causal relationships $X_2 \to X_1$ and $X_3 \to X_1$ over a time horizon $[1, T]$, we compare the performance of two linear predictors of $X_{1,t}$ over that time horizon. The first predictor (L_1) predicts $X_{1,t}$ using $\{X_1^{t-1}, X_3^{t-1}\}$ and the other predictor (L_2) uses $\{X_1^{t-1}, X_2^{t-1}\}$. If L_1 shows better performance compared to L_2, it implies that X_3 contains more relevant information about X_1 compared to X_2. In other words, X_3 has stronger influence on X_1 compared to X_2. To compare the performance of L_1 and L_2, we consider their mean squared errors over the time horizon $[1, T]$.

$$L_1: \quad e_1 := \frac{1}{T} \sum_{t=1}^{T} \min_{y_t \in \mathcal{A}_t} \mathbb{E}||X_{1,t} - y_t||^2, \quad \text{where} \quad \mathcal{A}_t := \text{span}\{X_1^{t-1}, X_3^{t-1}\},$$

$$L_2: \quad e_2 := \frac{1}{T} \sum_{t=1}^{T} \min_{z_t \in \mathcal{B}_t} \mathbb{E}||X_{1,t} - z_t||^2, \quad \text{where} \quad \mathcal{B}_t := \text{span}\{X_1^{t-1}, X_2^{t-1}\}.$$

It is easy to show that $e_1 = 1 + 0.1^2$ and $e_2 = 1 + 0.3^2$. Since $e_1 < e_2$, we infer that X_3 has stronger influence on X_1 compared to X_2.

Analogous to the directed information graphs, we can generalize the above framework to nonlinear systems. Consider a network of m time series $\underline{R} = \{R_1, ..., R_m\}$ with corresponding DIG

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$G = (V, E, W)$. Suppose (R_i, R_j) and (R_k, R_j) belong to E, i.e., R_i and R_k both are parents of R_j. We say R_i has stronger influence on R_j compared to R_k over a time horizon $[1, T]$ if $P(R_{j,t}|\mathcal{F}_{-\{k\}}^{t-1})$ is a better predictor for $R_{j,t}$ compared to $P(R_{j,t}|\mathcal{F}_{-\{i\}}^{t-1})$ over that time horizon. In other words, R_i has stronger influence on R_j compared to R_k, if

$$\frac{1}{T} \sum_{t=1}^{T} \mathbb{E}\left[\log \frac{P(R_{j,t}|\mathcal{F}_{-\{k\}}^{t-1})}{P(R_{j,t}|\mathcal{F}_{-\{i\}}^{t-1})}\right] > 0.$$

The above inequality holds if and only if $I(R_i \to R_j || \underline{R}_{-\{i,j\}}) > I(R_k \to R_j || \underline{R}_{-\{k,j\}})$. Thus, the DI in (1) can quantify the causal relationships in a network. For instance, looking again at the system in (4), we obtain

$$I(X_2 \to X_1 || X_3) = \frac{1}{2} \log(1 + 0.1^2) < \frac{1}{2} \log(1 + 0.3^2) = I(X_3 \to X_1 || X_2).$$

III.   DIG of Linear Models

Herein, we study the causal network of linear systems. Consider a set of m stationary time series, and for simplicity assume they have zero mean, such that their relationships are captured by the following model:

$$\vec{R}_t = \sum_{k=1}^{p} \mathbf{A}_k \vec{R}_{t-k} + \vec{\epsilon}_t, \tag{5}$$

where $\vec{R}_t = (R_{1,t}, ..., R_{m,t})^T$, and $\mathbf{A}_k$s are $m \times m$ matrices. Moreover, we assume that the exogenous noises, i.e., $\epsilon_{i,t}$s are independent and also independent from $\{R_{j,t}\}$. For simplicity, we assume that the $\{\epsilon_{i,t}\}$ have mean zero. For the model in (5), it was shown in Etesami and Kiyavash that

$$I(R_i \to R_j || \underline{R}_{-\{i,j\}}) > 0,$$

if and only if $\sum_{k=1}^{p} |(\mathbf{A}_k)_{j,i}| > 0$, where $(\mathbf{A}_k)_{j,i}$ is the (j, i)th entry of matrix $\mathbf{A}_k$. Thus, to learn the corresponding causal network (DIG) of this model, instead of estimating the DIs in (1), we can check whether the corresponding coefficients are zero or not. To do so, we use the Bayesian information criterion (BIC) as the model-selection criterion to learn the parameter p Schwarz et al. (1978), and use F-tests to check the null hypotheses that the coefficients are zero Lomax and Hahs-Vaughn (2013).

Wiener filtering is another alternative approach that can estimate the coefficients and consequently learn the DIG Materassi and Salapaka (2012). The idea of this approach is to find the coefficients by solving the following optimization problem,

$$\{\hat{\mathbf{A}}_1, ..., \hat{\mathbf{A}}_p\} = \arg \min_{\mathbf{B}_1, ..., \mathbf{B}_p} \mathbb{E}\left[\frac{1}{T} \sum_{t=1}^{T} ||\vec{R}_t - \sum_{k=1}^{p} \mathbf{B}_k \vec{R}_{t-k}||^2\right].$$

This leads to a set of Yule-Walker equations that can be solved efficiently by Levinson-Durbin algorithm Musicus (1988).

A. DIG of GARCH models

The relationship between the coefficients of the linear model and the corresponding DIG can easily be extended to the financial data in which the variance of $\{\epsilon_{i,t}\}_{t=1}^{T}$ are no longer independent of $\{R_{i,t}\}$ but due to the heteroskedasticity, they are $\mathcal{F}_i^{t-1}$-measurable. More precisely, in financial data, the returns are modeled by GARCH that is given by

$$
\begin{aligned}
R_{i,t}|\mathcal{F}^{t-1} &\sim \mathcal{N}(\mu_{i,t}, \sigma_{i,t}^2), \\
\sigma_{i,t}^2 &= \alpha_0 + \sum_{k=1}^{q} \alpha_k (R_{i,t-k} - \mu_{i,t})^2 + \sum_{l=1}^{s} \beta_l \sigma_{i,t-l}^2,
\end{aligned}
\tag{6}
$$

where α_ks and β_ls are nonnegative constants.

PROPOSITION 1: *Consider a network of time series whose dynamic is given by (6). In this case, there is no arrow from R_j to R_i in its corresponding DIG, i.e., R_j does not cause R_i if and only if*

$$
\mathbb{E}[R_{i,t}|\mathcal{F}^{t-1}] = \mathbb{E}[R_{i,t}|\mathcal{F}_{-\{j\}}^{t-1}], \ \forall t.
\tag{7}
$$

Proof. See Appendix .A. □

Multivariate GARCH models are a a generalization of (6) in which the variance of $e_{i,t}$ is $\mathcal{F}^{t-1}$-measurable. In this case, not only $\mu_{i,t}$ but also $\sigma_{i,t}^2$ capture the interactions between the returns. More precisely, in multivariate GARCH, we have

$$
\begin{aligned}
\vec{R}_t|\mathcal{F}^{t-1} &\sim \mathcal{N}(\vec{\mu}_t, \mathbf{H}_t), \\
vech[\mathbf{H}_t] &= \Omega_0 + \sum_{k=1}^{q} \Omega_k vech[\vec{\epsilon}_{t-k} \vec{\epsilon}_{t-k}^T] + \sum_{l=1}^{p} \Gamma_l vech[\mathbf{H}_{t-l}],
\end{aligned}
$$

where $\vec{\mu}_t$ is an $m \times 1$ array, $\mathbf{H}_t$ is an $m \times m$ symmetric positive definite and $\mathcal{F}^{t-1}$-measurable matrix, and $\vec{\epsilon}_t = \vec{R}_t - \vec{\mu}_t$. Note that *vech* denotes the vector-half operator, which stacks the lower triangular elements of an $m \times m$ matrix as an $(m(m+1)/2) \times 1$ array.

PROPOSITION 2: *Consider a network of time series whose dynamic is captured by a multivariate GARCH model. In this case, there is no arrow from R_j to R_i in its corresponding DIG, i.e., R_j does not influence R_i if and only if both the condition in Proposition 1 and the following condition hold*

$$
\mathbb{E}[(R_{i,t} - \mu_{i,t})^2|\mathcal{F}^{t-1}] = \mathbb{E}[(R_{i,t} - \mu_{i,t})^2|\mathcal{F}_{-\{j\}}^{t-1}], \ \forall t.
\tag{8}
$$

Proof. See Appendix .B. □

Next example demonstrates the connection between the DIG of a multivariate GARCH and its corresponding parameters.

Example 2: *Consider the following multivariate GARCH(1,1) model*

$$\begin{pmatrix} R_{1,t} \\ R_{2,t} \end{pmatrix} = \begin{pmatrix} 0.2 & 0.3 \\ 0 & 0.2 \end{pmatrix} \begin{pmatrix} R_{1,t-1} \\ R_{2,t-1} \end{pmatrix} + \begin{pmatrix} \epsilon_{1,t} \\ \epsilon_{2,t} \end{pmatrix},$$

$$\begin{pmatrix} \sigma_{1,t}^2 \\ \rho_t \\ \sigma_{2,t}^2 \end{pmatrix} = \begin{pmatrix} 0 \\ 0.3 \\ 0.1 \end{pmatrix} + \begin{pmatrix} 0.2 & 0 & 0.3 \\ 0 & 0.2 & 0.7 \\ 0.1 & 0.4 & 0 \end{pmatrix} \begin{pmatrix} \epsilon_{1,t-1}^2 \\ \epsilon_{1,t-1}\epsilon_{2,t-1} \\ \epsilon_{2,t-1}^2 \end{pmatrix} + \begin{pmatrix} 0.3 & 0.5 & 0 \\ 0.1 & 0.2 & 0 \\ 0 & 0 & 0.4 \end{pmatrix} \begin{pmatrix} \sigma_{1,t-1}^2 \\ \rho_{t-1} \\ \sigma_{2,t-1}^2 \end{pmatrix}, \tag{9}$$

where $\rho_t = \mathbb{E}[\epsilon_{1,t}\epsilon_{2,t}]$. The corresponding DIG of this model is $R_1 \leftrightarrow R_2$. This is because R_2 influences R_1 through the mean and variance and R_1 influences R_2 only through the variance.

Remark 2: *Recall that as we mentioned in Remark 1 and Example 1, the pairwise Granger-causality calculation, in general, fails to identify the true causal network. It was proposed in Billio et al. (2012) that the returns of the ith institution linearly depend on the past returns of the jth institution, when*

$$\mathbb{E}[R_{i,t}|\mathcal{F}^{t-1}] = \mathbb{E}\left[R_{i,t}|R_{j,t-1}, R_{i,t-1}, \{R_{j,\tau} - \mu_{j,\tau}\}_{\tau=-\infty}^{t-2}, \{R_{i,\tau} - \mu_{i,\tau}\}_{\tau=-\infty}^{t-2}\right].$$

This test is obtained based on pairwise Granger-causality calculation and does not consider non-linear causation through the variance of $\{\epsilon_i\}$. For instance, if the returns of two institutions R_j and R_k cause the returns of the ith institution, then the above equality does not hold, because R_k cannot be removed from the conditioning.

B. DIG of Moving-Average (MA) Models

The model in (5) may be represented as an infinite moving average (MA) or data-generating process (GDP), as long as $\vec{R}(t)$ is covariance-stationary, i.e., all the roots of $|\mathbf{I} - \sum_{k=1}^p \mathbf{A}_k z^k|$ fall outside the unit circle Pesaran and Shin (1998):

$$\vec{R}_t = \sum_{k=0}^{\infty} \mathbf{W}_k \vec{\epsilon}_{t-k}, \tag{10}$$

where $\mathbf{W}_k = 0$ for $k < 0$, $\mathbf{W}_0 = \mathbf{I}$, and $\mathbf{W}_k = \sum_{l=1}^p \mathbf{W}_{k-l}\mathbf{A}_l$. In this representation, $\{\epsilon_i\}$s are called shocks and if they are independent, they are also called orthogonal Diebold and Yılmaz (2014).

In this section, we study the causal structure of a MA model of finite order p. Consider a moving average model with orthogonal shocks given by

$$\vec{R}_t = \sum_{k=0}^{p} \mathbf{W}_k \vec{\epsilon}_{t-k}, \tag{11}$$

where $\mathbf{W}_i$s are $m \times m$ matrices such that $\mathbf{W}_0$ is non-singular with nonzero diagonals and without loss of generality, we can assume that $diag(\mathbf{W}_0)$ is the identity matrix. Equation (11) can be

written as $\vec{R}_t = \mathbf{W}_0\vec{\epsilon}_t + \mathcal{P}(L)\vec{\epsilon}_{t-1}$, where $\mathcal{P}(L) := \sum_{k=1}^{p} \mathbf{W}_k L^{k-1}$. Subsequently, we have

$$\mathbf{W}_0^{-1}\vec{R}_t = \vec{\epsilon}_t + \sum_{k=1}^{\infty} (-1)^{k-1} \left(\mathbf{W}_0^{-1}\mathcal{P}(L)\right)^k \mathbf{W}_0^{-1}\vec{R}_{t-k}. \tag{12}$$

This representation is equivalent to an infinite AR model. Hence using the result in Etesami and Kiyavash, we can conclude the following corollary.

COROLLARY 1: *Consider a MA model described by (11) with orthogonal shocks such that $\boldsymbol{W}_0$ is non-singular and diagonal. In this case, R_j does not influence R_i if and only if the corresponding coefficients of $\{R_{j,t-k}\}_{k>0}$ in R_i's equation are zero.*

In the interest of simplicity and space, we do not present the explicit form of these coefficients, but we show the importance of this result using a simple example.

Example 3: *Consider a MA(1) with dimension three such that $\boldsymbol{W}_0 = \boldsymbol{I}$, and*

$$\boldsymbol{W}_1 = \begin{pmatrix} 0.3 & 0 & 0.5 \\ 0.1 & 0.2 & 0.5 \\ 0 & 0.4 & 0.1 \end{pmatrix}, \quad \boldsymbol{W}_1^2 = \begin{pmatrix} 0.09 & 0.2 & 0.2 \\ 0.05 & 0.24 & 0.2 \\ 0.04 & 0.12 & 0.21 \end{pmatrix},$$

Using the expression in (12), we have $\vec{R}_t = \vec{\epsilon}_t + \sum_{k=1}^{\infty} (-1)^{k-1} \boldsymbol{W}_1^k \vec{R}_{t-k}$. Because, $\boldsymbol{W}_1^2$ has no nonzero entry, the causal network (DIG) of this model is a complete graph.

We studied the DIG of a MA model with orthogonal shocks. However, the shocks are rarely orthogonal in practice. To identify the causal structure of such systems, we can apply the whitening transformation to transform the shocks into a set of uncorrelated variables. More precisely, suppose $\mathbb{E}[\vec{\epsilon}_t \vec{\epsilon}_t^T] = \Sigma$, where the Cholesky decomposition of Σ is $\mathbf{V}\mathbf{V}^T$ Horn and Johnson (2012). Hence, $\mathbf{V}^{-1}\vec{\epsilon}_t$ is a vector of uncorrelated shocks. Using this fact, we can transform (11) with correlated shocks into

$$\vec{R}_t = \sum_{k=0}^{p} \tilde{\mathbf{W}}_k \vec{\tilde{\epsilon}}_{t-k}, \tag{13}$$

with uncorrelated shocks, where $\vec{\tilde{\epsilon}}_t := \mathbf{V}^{-1}\vec{\epsilon}_t$ and $\tilde{\mathbf{W}}_k := \mathbf{W}_k \mathbf{V}$.

Remark 3: *The authors in Diebold and Yılmaz (2014) applied the generalized variance decomposition (GVD) method to identify the population connectedness or in another word the causal structure of a MA model with correlated shocks. Using this method, they monitor and characterize the network of major U.S. financial institutions during 2007-2008 financial crisis. In this method, the weight of R_j's influence on R_i in (11) was defined to be proportional to*

$$d_{i,j} = \sum_{k=0}^{p} \left((\boldsymbol{W}_k \Sigma)_{i,j}\right)^2, \tag{14}$$

where $(\boldsymbol{A})_{i,j}$ denotes the (i,j)-th entry of matrix $\boldsymbol{A}$. Recall that $\mathbb{E}[\vec{\epsilon}_t \vec{\epsilon}_t^T] = \Sigma$. Applying the GVD

method to Example 3, where $\Sigma = \boldsymbol{I}$, we obtain that $d_{1,2} = d_{3,1} = 0$. That is R_2 does not influence R_1 and R_1 does not influence R_3. This result is not consistent with the Granger-causality concept since the corresponding causal network (DIG) of this example is a complete graph, i.e., every node has influence on any other node. Thus, GVD analysis of Diebold and Yılmaz (2014) is also seems to suffer from disregarding the entire network akin to pairwise analysis commonly used in traditional application of the Granger-causality.

IV.   DIG of Non-linear Models

DIG as defined in Definition 2 does not require any linearity assumptions on the model. But, similar to Billio et al. (2010), side information about the model class can simplify computation of (1). For instance, let us assume that $\underline{R}$ is a first-order Markov chain with transition probabilities:

$$P(\underline{Y}_t|\underline{R}^{t-1}) = P(\underline{R}_t|\underline{R}_{t-1}).$$

In this setup, $I(R_i \to R_j||\underline{R}_{-\{i,j\}}) = 0$ if and only if

$$P(R_{j,t}|\underline{R}_{t-1}) = P(R_{j,t}|\underline{R}_{-\{i\},t-1}), \forall t.$$

Recall that $\underline{R}_{-\{i\},t-1}$ denotes $\{R_{1,t-1}, ..., R_{m,t-1}\} \setminus \{R_{i,t-1}\}$. Furthermore, suppose that the transition probabilities are represented through a logistic function again as in Billio et al. (2010). More specifically, for any subset of processes $\mathcal{S} := \{R_{i_1}, ..., R_{i_s}\} \subseteq \underline{R}$, we have

$$P(R_{j,t}|R_{i_1,t-1}, ..., R_{i_s,t-1}) := \frac{\exp(\vec{\alpha}_\mathcal{S}^T \vec{U}_\mathcal{S})}{1 + \exp(\vec{\alpha}_\mathcal{S}^T \vec{U}_\mathcal{S})},$$

where $\vec{U}_\mathcal{S}^T := \bigotimes_{i \in \mathcal{S}}(1, R_{i,t-1}) = (1, R_{i_1,t-1}) \otimes (1, R_{i_2,t-1}) \otimes \cdots \otimes (1, R_{i_s,t-1})$, $\otimes$ denotes the Kronecker product, and $\vec{\alpha}_\mathcal{S}$ is a vector of dimension $2^s \times 1$. Under these assumptions, the causal discovery in the network reduces to the following statement: R_i does not influence R_j if and only if all the terms of $\vec{U}_{\underline{R}}$ depending on R_i are equal to zero. More precisely:

$$\vec{U}_{\underline{R}} = \vec{U}_{\underline{R}_{-\{i\}}} \otimes (1, R_{i,t-1}) = (\vec{U}_{\underline{R}_{-\{i\}}}, \vec{U}_{\underline{R}_{-\{i\}}} R_{i,t-1}).$$

Let $\vec{\alpha}_{\underline{R}}^T = (\vec{\alpha}_1^T, \vec{\alpha}_2^T)$, where $\vec{\alpha}_1$ and $\vec{\alpha}_2$ are the vectors of coefficients corresponding to $\vec{U}_{\underline{R}_{-\{i\}}}$ and $\vec{U}_{\underline{R}_{-\{i\}}} R_{i,t-1}$, respectively. Then $R_i \not\to R_j$ if and only if $\vec{\alpha}_2 = 0$.

Another such non-linear models are Multiple chain Markov switching models (MCMS)-VAR Billio and Di Sanzo (2015), in which the relationship between time series $\underline{Y}_t$ is given by

$$Y_{i,t} = \mu_i(S_{i,t}) + \sum_{k=1}^{p} \sum_{j=1}^{m} (\mathbf{B}_k(S_{i,t}))_{i,j} Y_{j,t-k} + \epsilon_{i,t}, \text{ for } i \in \{1, ..., m\}, \tag{15}$$

and $\vec{\epsilon}_t := (\epsilon_{1,t}, ..., \epsilon_{m,t}) \sim \mathcal{N}(0, \Sigma(\vec{S}_t))$, where the mean, the lag matrices, and the covariance matrix of the error terms all depend on a latent random vector $\vec{S}_t$ known as the state of the system. $S_{i,t}$ represents the state variable associated with $Y_{i,t}$ that can take values from a finite set $\mathcal{S}$. The random sequence $\{\vec{S}_t\}$ is assumed to be a time-homogenous first-order Markov process with one-step ahead transition probability $P(\vec{S}_t | \underline{S}^{t-1}, \underline{Y}^{t-1}) = P(\vec{S}_t | \underline{S}_{t-1})$. Furthermore, we assume that given the past of the states, their presents are independent, i.e.,

$$P(\vec{S}_t | \underline{S}_{t-1}) = \prod_j P(S_{j,t} | \underline{S}_{t-1}).$$

Next result stresses a set of conditions under which by observing the time series $\underline{Y}_t$, we are able to identify the causal relationships between them.

PROPOSITION 3: *Consider a MCMS-VAR in which $\Sigma(\vec{S}_t)$ is diagonal for all $\vec{S}_t$. In this case, $I(Y_j \to Y_i || \underline{Y}_{-\{i,j\}}) = 0$ if*

- *$(\boldsymbol{B}_k(s_{i,t}))_{i,j} = 0$ for all realizations $s_{i,t}$,*
- *$(\Sigma(\vec{S}_t))_{i,i} = (\Sigma(S_{i,t}))_{i,i}$,*
- *$P(S_{k,t} | \underline{S}^{t-1}, \underline{S}_{-\{k\},t}) = P(S_{k,t} | S_{k,t-1})$ for every k.*

Proof. See Appendix .C. □

Note that the third condition in this proposition seems strong compared to the condition in Billio and Di Sanzo (2015). But notice that Billio and Di Sanzo (2015) studies the causal relationships between the time series given the state variables, which is not realistic as they are hidden. Below, we show a simple example in which Y_1 does not functionally depend on Y_2 and S_1 is statistically independent of S_2. However, in this example, observing the states leads to Y_2 has no influence on Y_1, but without observing the states we infer differently.

Example 4: *Consider a bivariate MCMS-VAR $\{Y_1, Y_2\}$ in which the states only take binary values and*

$$Y_{1,t} = b_{1,1}(S_{1,t})Y_{1,t-1} + 0.1\epsilon_{1,t},$$
$$Y_{2,t} = \mu_2(S_{2,t}) + 0.5Y_{1,t-1} + 0.1\epsilon_{2,t},$$

where $(\epsilon_{1,t}, \epsilon_{2,t}) \sim \mathcal{N}(0, I)$, $\mu_2(0) = 10$, $\mu_2(1) = -5$, $b_{1,1}(0) = 0.5$, and $b_{1,1}(1) = -0.5$. Moreover, the transition probabilities of the states are $P(S_{1,t}|S_{1,t-1}, S_{2,t-1}) = P(S_{1,t}|S_{1,t-1}) = 0.8$ whenever $S_{1,t} = S_{1,t-1}$, and $S_{2,t}$ equals to $S_{1,t-1}$ with probability 0.9. Based on Billio and Di Sanzo (2015), in this setup, $Y_{2,t-1}$ does not Granger-cause $Y_{1,t}$ given $Y_{1,t-1}, S_{1,t-1}$, i.e.,

$$P(Y_{1,t}|Y_{2,t-1}, Y_{1,t-1}, S_{1,t-1}) = P(Y_{1,t}|Y_{1,t-1}, S_{1,t-1}).$$

Note that in this example, $P(Y_{1,t}|Y_{2,t-1}, Y_{1,t-1}) \neq P(Y_{1,t}|Y_{1,t-1})$. This is because, $Y_{2,t-1}$ has information about $S_{2,t-1}$ which contains information about $S_{1,t-2}$.

V. Experimental Result

In we have introduced tools for identifying the causal structure in a network of time series. In this section, we put those tools to work and use them to identify and monitor the evolution of connectedness among major financial institutions during 2006-2016.

A. Data

We obtained the data for individual banks, broker/dealers, and insurers from ???, from which we selected the daily returns of all companies listed in Table I.

Banks				Insurances				Brokers			
1	FNMA US	16	BNS US	1	MET US	16	PFG US	1	MS US	16	WDR US
2	AXP US	17	STI US	2	ANTM US	17	LNC US	2	GS US	17	EV US
3	FMCC US	18	C US	3	AET US	18	AON US	3	BEN US	18	ITG UN
4	BAC US	19	MS US	4	CNA US	19	HUM US	4	MORN US	19	JNS US
5	WFC UN	20	SLM US	5	XL US	20	MMC US	5	LAZ US	20	SCHW US
6	JPM US	21	BBT US	6	SLF US	21	HIG US	6	ICE US	21	ETFC US
7	DB US	22	USB US	7	MFC US	22	CI US	7	AINV US	22	AMTD US
8	NTRS US	23	TD US	8	GNW US	23	ALL US	8	SEIC US		
9	RY US	24	HSBC US	9	PRU US	24	BRK/B US	9	FII US		
10	PNC US	25	BCS US	10	AIG US	25	CPYYY US	10	RDN US		
11	STT US	26	GS US	11	PGR US	26	AHL US	11	TROW US		
12	COF US	27	MS US	12	CB US			12	AMP US		
13	BMO US	28	CS US	13	BRK/A US			13	GHL US		
14	CM US			14	UNH US			14	AMG US		
15	RF UN			15	AFL US			15	RJF US		

Table I. List of companies in our experiment.

We calculated the causal network for different time periods that will be considered in the empirical analysis: 2006-2008, 2009-2011, 2011-2013, and 2013-2016.

B. Non-linearity Test

In this section, we applied a non-linearity test on the data to determine whether the underlying structure within the recorded data is linear or nonlinear. The non-linearity test applied in this section is based on nonlinear principle component analysis (PCA) Kruger et al. (2008). This test is based on two principles: the range of recorded data is divided into smaller disjunct regions; and accuracy bounds are determined for the sum of the discarded eigenvalues of each region. If this sum is within the accuracy bounds for each region, the process is assumed to be linear. Conversely, if at least one of these sums is outside, the process is assumed to be nonlinear.

More precisely, the second principle in this test requires computation of the correlation matrix for each of the disjunct regions. Since the elements of this matrix are obtained using a finite dataset, applying t-distribution and χ^2-distribution establish confidence bounds for both estimated mean

and variance, respectively. Subsequently, these confidence bounds can be utilized to determine thresholds for each element in the correlation matrix. Using these thresholds, the test calculates maximum and minimum eigenvalues relating to the discarded score variables, which in turn allows the determination of both a minimum and a maximum accuracy bound for the variance of the prediction error of the PCA model. This is because the variance of the prediction error is equal to the sum of the discarded eigenvalues. If this sum lies inside the accuracy bounds for each disjunct region, a linear PCA model is then appropriate over the entire region. Alternatively, if at least one of these sums is outside the accuracy bounds, the error variance of the PCA model residuals then differs significantly for this disjunct region and hence, a nonlinear model is required. For more details see Kruger et al. (2008).

We divided the operating region into 3 disjunct regions. The accuracy bounds for each disjuct region and also sum of the discarded eigenvalues were computed. These bounds were based on thresholds for each element of the correlation matrix corresponding to confidence level of 95%. Note that the processes were normalized with respect to the mean and variance of the regions for which the accuracy bounds were computed. Figure 2 shows the accuracy bounds and the sum of the discarded eigenvalues. As figures 2-(a) and 2-(b) illustrate, the recorded financial data is nonlinear.

(a) Region 1.   (b) Region 2.   (c) Region 3.
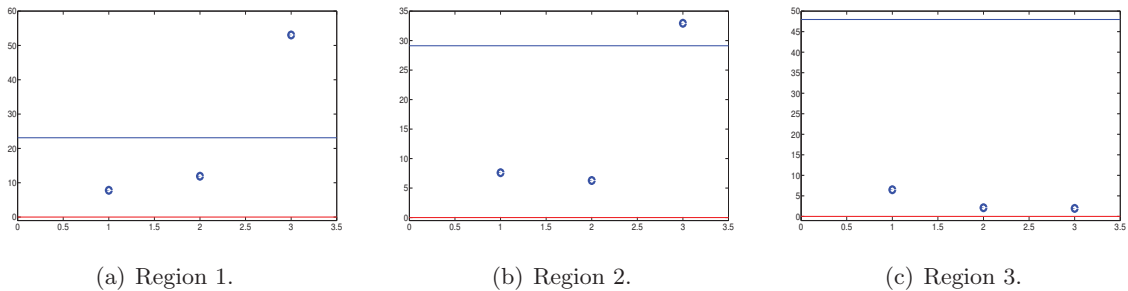
Figure 2. Benchmarking of the residual variances against accuracy bounds of each disjunct region.

C.   Estimating the DIs

As we mentioned earlier, there are different methods that can be used to estimate (1) given i.i.d. samples of the time series. Plug-in empirical estimator and k-nearest neighbor estimator are such two methods Jiao et al. (2013); Frenzel and Pompe (2007); Kraskov et al. (2004). For our experimental results, we used k-nearest method to estimate the DIs since it shows relatively better performance compared to the other non-parametric estimators. To do so, we used the fact that

$$I(R_i \rightarrow R_j || \underline{R}_{-\{i,j\}}) = \frac{1}{T} \sum_{t=1}^{T} I(R_{j,t}; R_i^{t-1} | \underline{R}_{-\{i,j\}}^{t-1}, R_j^{t-1}),$$

where $I(X; Y|Z)$ denotes conditional mutual information between X and Y given Z Cover and Thomas (2012). Then, we estimated each of the above conditional mutual information using k-nearest method in Sricharan et al. (2011). Below, we describe the steps of k-nearest method to estimate $I(X; Y|Z)$.

Suppose that $N + M$ i.i.d. realizations $\{\mathbf{X}_1, ..., \mathbf{X}_{N+M}\}$ are available from $P_{X,Y,Z}$, where $\mathbf{X}_i$ denotes the ith realization of (X, Y, Z). The data sample is randomly divided into two subsets S_1 and S_2 of N and M points, respectively. In the first stage, an k-nearest density estimator $\widehat{P}_{X,Y,Z}$ at the N points of S_1 is estimated using the M realizations of S_2 as follows: Let $d(\mathbf{x}, \mathbf{y})$ denote the Euclidean distance between points $\mathbf{x}$ and $\mathbf{y}$ and $d_k(\mathbf{x})$ denotes the Euclidean distance between a point $\mathbf{x}$ and its k-th nearest neighbor among S_2. The k-nearest region is $S_k(\mathbf{x}) := \{\mathbf{y} : d(\mathbf{x}, \mathbf{y}) \leq d_k(\mathbf{x})\}$ and the volume of this region is $V_k(\mathbf{x}) := \int_{S_k(\mathbf{x})} dn$. The standard k-nearest density estimator Sricharan et al. (2011) is defined as

$$\widehat{P}_{X,Y,Z}(\mathbf{x}) := \frac{k-1}{MV_k(\mathbf{x})}.$$

Similarly, we obtain k-nearest density estimators $\widehat{P}_{X,Z}, \widehat{P}_{Y,Z}$, and $\widehat{P}_Z$. Subsequently, the N samples of S_1 is used to approximate the conditional mutual information:

$$\widehat{I}(X; Y|Z) := \frac{1}{N} \sum_{i \in S_1} \log \widehat{P}_{X,Y,Z}(\mathbf{X}_i) + \log \widehat{P}_Z(\mathbf{X}_i) - \log \widehat{P}_{X,Z}(\mathbf{X}_i) - \log \widehat{P}_{Y,Z}(\mathbf{X}_i).$$

For more details corresponding this estimator including its bias, variance, and confidence, please see Sricharan et al. (2011); Loftsgaarden et al. (1965).

D. DIG of the Financial Market

In this section, we learned the DIG of the aforementioned financial institutions by estimating the directed information quantities in (1). To do so, we divided the data into four sectors each of length almost 36 months, 2006-2008, 2009-2011, 2011-2013, and 2013-2016. We assumed that the DIG of the network did not change over each of these time periods. Furthermore, the data collected per working day are assumed to be i.i.d.. Hence, in this experiment the length of each time series was almost 36 and for each time instance we had nearly 19 independent realizations.

As we discussed in Section II.B, in order to identify the influence from node i on node j, we need to estimate $I(R_i \to R_j || \underline{R}_{-\{i,j\}})$, which in this experiment, required estimating a joint distribution of dimension 76. In general, without any knowledge about the underlying distribution, estimating such object requires a large amount of independent samples. Unfortunately, in this experiment, we had limited number of independent samples. Thus, we reduced the dimension by instead of conditioning on $\underline{R}_{-\{i,j\}}$ that is a set of size 74, we conditioned on a smaller subset $\underline{K}_{i,j}$ of $\underline{R}_{-\{i,j\}}$ with size 7. This set contained only those institutions with highest correlation with R_j. In another words, we ordered the institutions in $\underline{R}_{-\{i,j\}}$ based on their correlation value with R_j, and picked the first 7 of them. Afterward, we estimated $I(R_i \to R_j || \underline{K}_{i,j})$ to identify the connection between R_i and R_j.

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Figures 3 and 4 show the resulting graphs. Note that the type of institution causing the relationship is indicated by color: green for brokers, red for insurers, and blue for banks.
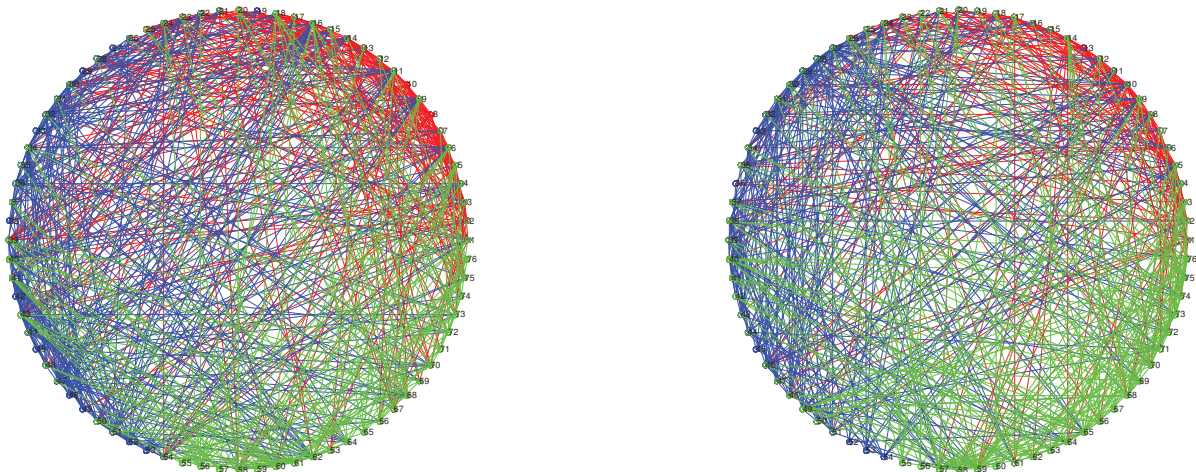
(a) January 2006 to December 2008   (b) January 2009 to December 2011
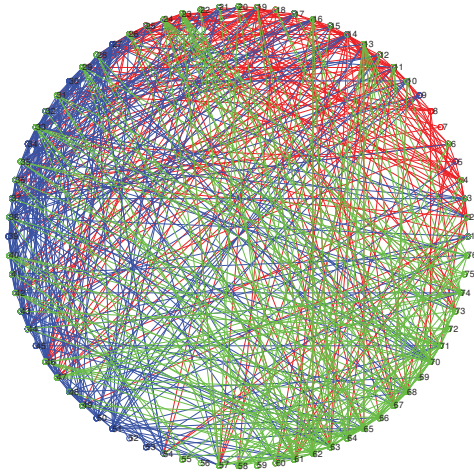
Figure 3. Recovered DIG of the daily returns of the financial companies in Table I. The type of institution causing the relationship is indicated by color: green for brokers, red for insurers, and blue for banks.

In order to compare our results with other methods in the literature, we also learned the causal network of these financial institutions by assuming linear relationships between the institutions and applying linear regression. Similarly, we reduced the dimension of the regressions by bounding the number of incoming arrows of each node to be a subset of size 20. More precisely, we picked 20 most correlated institutions with node i, let say $\{R_{j_1}, ..., R_{j_{18}}\}$ and obtained the parents of i by solving $\min_{a_j} \sum_t |R_{i,t} - \sum_{k=1}^{18} a_k R_{j_k, t-1}|^2$ The resulting graphs are depicted in Figures 5 and 6.

From these networks, we constructed the following network-based measures of systemic risk. We calculated the fraction of statistically significant Granger causality relationships among all pairs of financial institutions. This is known as the degree of Granger causality (DGC) and it is a measure of the risk of a system event Billio et al. (2012). Table II presents the DGC values and total number of connections of the DIGs and the networks obtain by linear regression.

DIGs		
2006-2008	0.1225	698
2009-2011	0.1114	635
2011-2013	0.1065	607
2013-2016	0.0930	530

Linear Models		
2006-2008	0.1453	828
2009-2011	0.1288	734
2011-2013	0.1174	669
2013-2016	0.1216	693

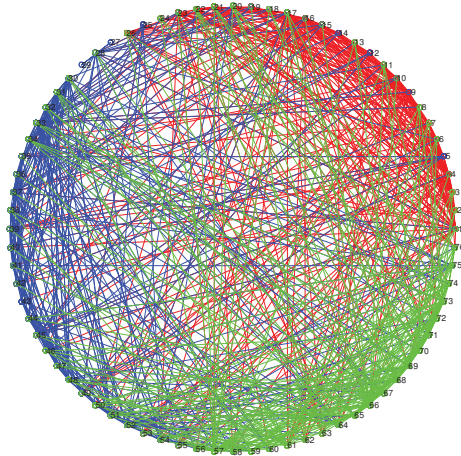**Table II.** DGC values and total number of connections.
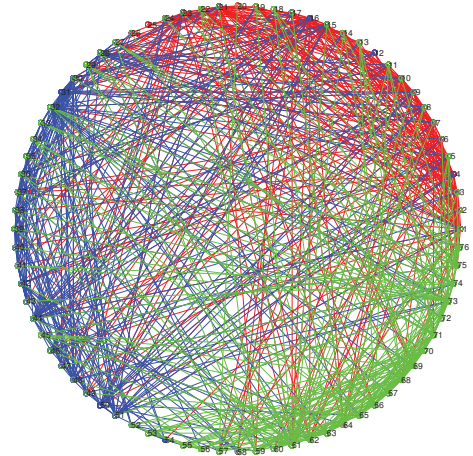
17
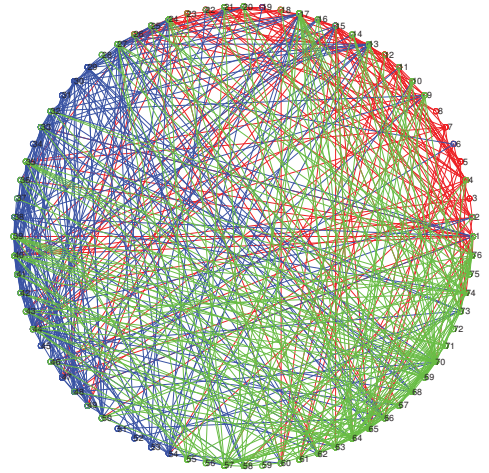
(a) January 2011 to December 2013   (b) January 2013 to June 2016

Figure 4. Recovered DIG of the daily returns of the financial companies in Table I. The type of institution causing the relationship is indicated by color: green for brokers, red for insurers, and blue for banks.
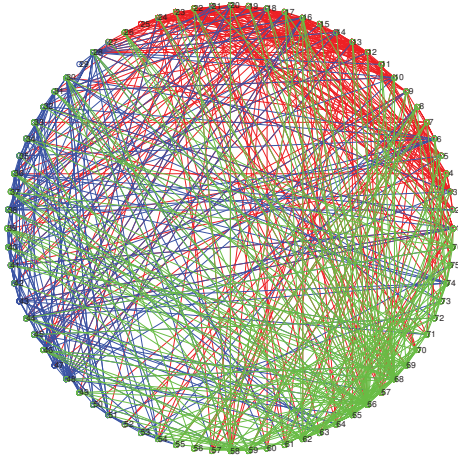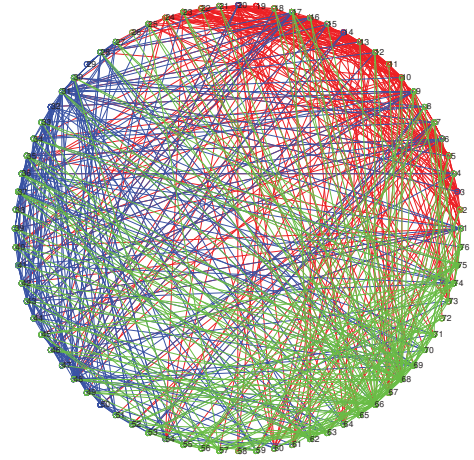

(a) January 2006 to December 2008   (b) January 2009 to December 2011

Figure 5. Recovered network of the daily returns of the financial companies in Table I using linear regression. The type of institution causing the relationship is indicated by color: green for brokers, red for insurers, and blue for banks.

In order to assess the systemic importance of single institutions, we computed the number of financial institutions that are caused by institution i and also the number of financial institutions

(a) January 2011 to December 2013

(b) January 2013 to June 2016

Figure 6. Recovered network of the daily returns of the financial companies in Table I using linear regression. The type of institution causing the relationship is indicated by color: green for brokers, red for insurers, and blue for banks.

that are causing institution i. Figure 7 demonstrates the average number of out-degree and in-degree distributions of the DIGs. Correspondingly, Figure 8 shows these quantities for the networks obtain by linear regression.

Tables III and IV represent the average number of connections between the sectors e.g., 0.1719 fraction of connections are from Banks to Insurances during 2006-2008 in the DIG.

	2006-2008			2009-2011			2011-2013			2013-2016		
	Ins.	Ba.	Br.	Ins.	Ba.	Br.	Ins.	Ba.	Br.	Ins.	Ba.	Br.
Insurance	.1390	.1719	.1074	.1291	.1575	.1213	.1054	.1301	.1104	.1075	.1151	.1340
Bank	.1361	.1332	.0702	.0866	.1402	.1039	.1417	.1631	.1021	.0774	.1830	.1302
Broker	.0774	.1017	.0630	.0740	.929	.0945	.0906	.0873	.0692	.0774	.0774	.0981

Table III. Average number of connections between different sectors in the DIGs.

	2006-2008			2009-2011			2011-2013			2013-2016		
	Ins.	Ba.	Br.	Ins.	Ba.	Br.	Ins.	Ba.	Br.	Ins.	Ba.	Br.
Insurance	.1896	.0688	.0737	.1785	.1076	.0640	.2033	.0792	.1016	.2107	.0851	.0678
Bank	.0906	.1872	.0809	.1322	.1431	.0899	.1136	.1226	.1001	.1010	.1515	.1053
Broker	.0857	.1063	.1171	.0790	.0708	.1349	.1226	.0673	.0897	.1082	.0895	.0808

Table IV. Average number of connections between different sectors in the networks obtained using regression.

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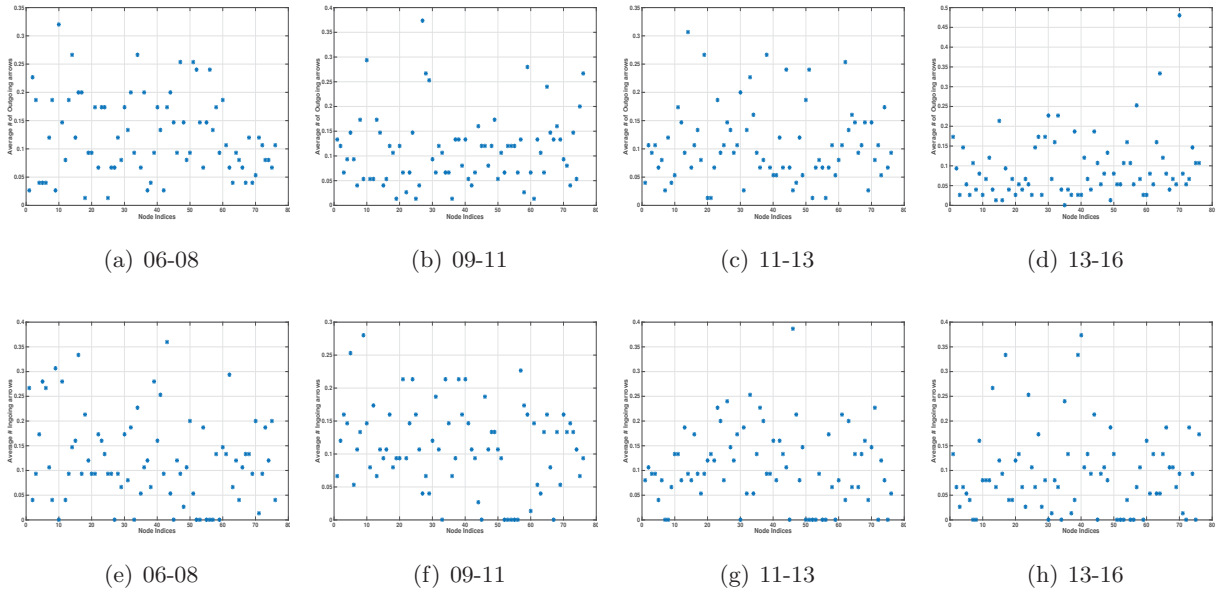
Figure 7. Out and In degree distributions of the DIGs obtained in Section V.D.
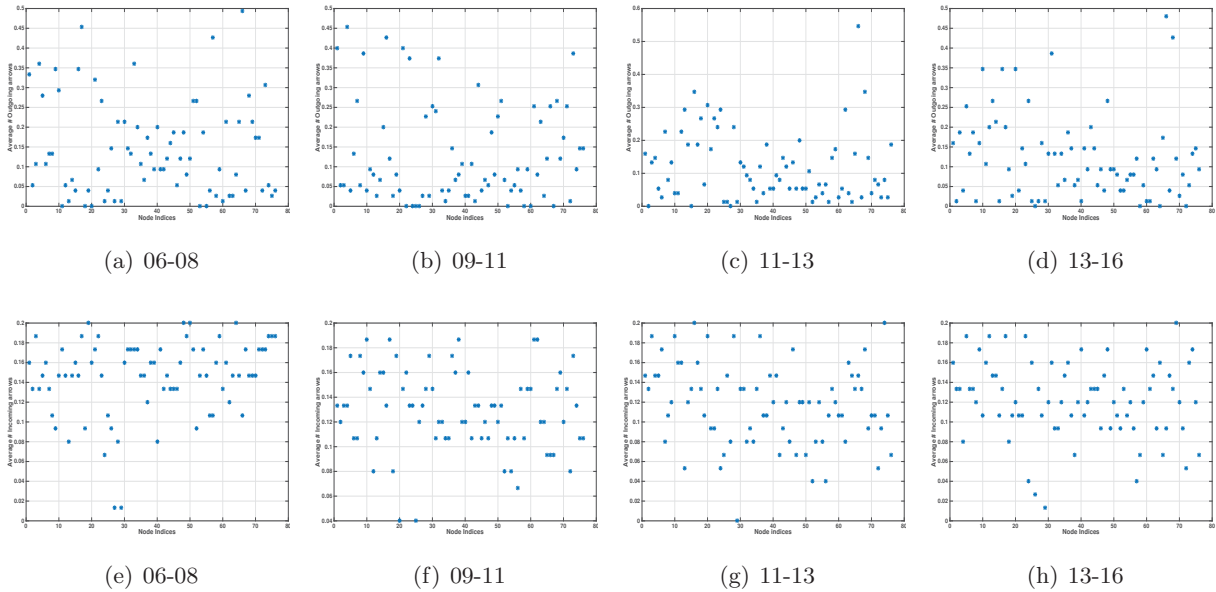
Figure 8. Out and In degree distributions of the networks obtained using linear regression.

VI.   Conclusion

In this work, we developed a data-driven econometric framework to understand the relationship between financial institutions using a non-linearly modified Granger-causality. Unlike existing literature, it is not focused on a linear pairwise estimation. The proposed method allows for nonlinearity and it does not suffer from pairwise comparison to identify the causal relationships

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between financial institutions. We also show how the model improve the measurement of systemic risk and explain the link between Granger-causality and variance decomposition. We apply the model to the monthly returns of U.S. financial Institutions including banks, broker, and insurance companies to identify the level of systemic risk in the financial sector and the contribution of each financial institution.

Appendix A.   Proof of Proposition 1

Note that in this model, since the variance of each $e_{i,t}$ is $\mathcal{F}_i^{t-1}$-measurable, the only term that contains the effect of the other returns on the i-th return is $\mu_{i,t}$. Hence, if (7) holds, then $\mu_{i,t}$ is independent of R_j. This implies the result. Moreover, when $\mu_{i,t} = \sum_{k=1}^{p} \sum_{l=1}^{m} a_{i,l}^{(k)} R_{l,t-k}$, using the result in Etesami and Kiyavash, we declare R_j affects R_i if and only if $\sum_{k=1}^{p} \sum_{l=1}^{m} |a_{i,l}^{(k)}| > 0$, where $a_{i,l}^{(k)}$ denotes the (j,l)-th entry of matrix $\mathbf{A}_k$ in (5).

Appendix B.   Proof of Proposition 2

First, we need to show that if there is no arrow from R_j to R_i in the corresponding DIG, then (7) and (8) hold. This case is straight forward, since when $I(R_j \rightarrow R_i || \underline{R}_{-\{i,j\}}) = 0$, then for all t, $R_{i,t}$ is independent of R_j given $\mathcal{F}_{-\{j\}}^{t-1}$. This concludes both (7) and (8).

To show the converse, we use the fact that in multivariate GARCH model, $\vec{R}_t | \mathcal{F}^{t-1}$ is a multivariate Gaussian random process. Thus, if the corresponding mean and variance of $R_{i,t}$ do not contain any influence of R_j^{t-1} given the rest of the network, then $R_{i,t}$ is independent of R_j^{t-1} given $\underline{R}_{-\{j\}}^{t-1}$. This holds if both conditions in (7) and (8) that are corresponding to the mean and the variance, respectively, are satisfied.

Appendix C.   Proof of Proposition 3

Suppose the conditions in Proposition 3 hold. We show that $I(Y_j \rightarrow Y_i || \underline{Y}_{-\{i,j\}}) = 0$.

$$P(Y_{i,t} | \underline{Y}^{t-1}) = \sum_{S_{i,t}} P(Y_{i,t} | \underline{Y}^{t-1}, S_{i,t}) P(S_{i,t} | \underline{Y}^{t-1})$$

$$= \sum_{S_{i,t}} P(Y_{i,t} | \underline{Y}_{-\{j\}}^{t-1}, S_{i,t}) P(S_{i,t} | \underline{Y}^{t-1}).$$

The second equality holds because given $S_{i,t}$, $Y_{i,t}$ is a linear function of $(\mu_i(S_{i,t}), \vec{Y}_{t-p}, ..., \vec{Y}_{t-1})$ plus the error term $\epsilon_{i,t}$. From the first and second conditions in Proposition 3, we have the coefficients corresponding to Y_j are zero and also the error term is independent of Y_j. Thus, $Y_{i,t}$ is independent of Y_j^{t-1} given $\underline{Y}_{-\{j\}}^{t-1}, S_{i,t}$.

If we show $P(S_{i,t} | \underline{Y}^{t-1}) = P(S_{i,t} | \underline{Y}_{-\{j\}}^{t-1})$, using the above equality, we obtain that $P(Y_{i,t} | \underline{Y}^{t-1}) =$

$P(Y_{i,t}|\underline{Y}^{t-1}_{-\{j\}})$ for all t. This implies $I(Y_j \to Y_i || \underline{Y}_{-\{i,j\}}) = 0$. To do so, we have

$$P(S_{i,t}|\underline{Y}^{t-1}) = \sum_{S_{i,t-1}} P(S_{i,t}|\underline{Y}^{t-1}, S_{i,t-1})P(S_{i,t-1}|\underline{Y}^{t-1})$$

$$= \sum_{S_{i,t-1}} P(S_{i,t}|\underline{Y}^{t-1}_{-\{j\}}, S_{i,t-1})P(S_{i,t-1}|\underline{Y}^{t-1})$$

$$= \sum_{S_{i,t-1}} P(S_{i,t}|\underline{Y}^{t-1}_{-\{j\}}, S_{i,t-1})P(S_{i,t-1}|\underline{Y}^{t-1}_{-\{j\}}) = P(S_{i,t}|\underline{Y}^{t-1}_{-\{j\}}).$$

The second equality is due to condition three and the fact that $\vec{S}_t$ is conditionally independent of $\underline{Y}_{t-1}$ given $\underline{S}_{t-1}$. The third equality is due to the following

$$P(S_{i,t-1}|\underline{Y}^{t-1}) = P\left(S_{i,t-1}|\underline{Y}^{t-2}, Y_{i,t-1}, \underline{Y}_{-\{i,j\},t-1}, Y_{j,t-1}\right)$$

$$= P\left(S_{i,t-1}|\underline{Y}^{t-2}, F_i(\underline{Y}^{t-2}_{-\{j\}}, S_{i,t-1}), \underline{Y}_{-\{i,j\},t-1}, F_j(\underline{Y}^{t-2}, S_{j,t-1})\right)$$

$$= P\left(S_{i,t-1}|\underline{Y}^{t-2}, F_i(\underline{Y}^{t-2}_{-\{j\}}, S_{i,t-1}), \underline{Y}_{-\{i,j\},t-1}\right),$$

where F_js represent the functional dependency between time series given in (15), i.e., $Y_{m,t-1} := F_m(\underline{Y}^{t-2}, S_{m,t-1})$. The above equality holds due to the third condition that states are mutually independent and the fact that all the Y_j's coefficients are zero in Y_i's equation. Same reasoning implies

$$P\left(S_{i,t-1}|\underline{Y}^{t-2}, F_i(\underline{Y}^{t-2}_{-\{j\}}, S_{i,t-1}), \underline{Y}_{-\{i,j\},t-1}\right)$$

$$= P\left(S_{i,t-1}|\underline{Y}^{t-3}, F_i(\underline{Y}^{t-2}_{-\{j\}}, S_{i,t-1}), Y_{i,t-2}, \underline{Y}^{t-1}_{-\{i,j\},t-2}, Y_{j,t-2}\right)$$

$$= P\left(S_{i,t-1}|\underline{Y}^{t-3}, F_i(\underline{Y}^{t-2}_{-\{j\}}, S_{i,t-1}), F_i(\underline{Y}^{t-3}_{-\{j\}}, S_{i,t-2}), \underline{Y}^{t-1}_{-\{i,j\},t-2}, F_j(\underline{Y}^{t-3}, S_{j,t-2})\right)$$

$$= P\left(S_{i,t-1}|\underline{Y}^{t-3}, F_i(\underline{Y}^{t-2}_{-\{j\}}, S_{i,t-1}), F_i(\underline{Y}^{t-3}_{-\{j\}}, S_{i,t-2}), \underline{Y}^{t-1}_{-\{i,j\},t-2}\right)$$

$$\vdots$$

$$= P\left(S_{i,t-1}|F_i(\underline{Y}^{t-2}_{-\{j\}}, S_{i,t-1}), F_i(\underline{Y}^{t-3}_{-\{j\}}, S_{i,t-2}), ..., \underline{Y}^{t-1}_{-\{i,j\}}\right) = P\left(S_{i,t-1}|\underline{Y}^{t-1}_{-\{j\}}\right).$$

Recall that $\underline{Y}^t_{\mathcal{K},t'}$ denotes the time series with index set $\mathcal{K}$ from time t' up to time t.

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Notes

[1] In causal systems, given the full past of the system, the present of the processes become independent. In other words, there are no simulations relationships between the time series.

[2] Minimal in terms of its cardinality.

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