A Nonparametric Test for Granger Causality in Distribution With Application to Financial Contagion

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This article introduces a kernel-based nonparametric inferential procedure to test for Granger causality in distribution. This test is a multivariate extension of the kernel-based Granger causality test in tail event. The main advantage of this test is its ability to examine a large number of lags, with higher-order lags discounted. In addition, our test is highly flexible because it can be used to identify Granger causality in specific regions on the distribution supports, such as the center or tails. We prove that the test converges asymptotically to a standard Gaussian distribution under the null hypothesis and thus is free of parameter estimation uncertainty. Monte Carlo simulations illustrate the excellent small sample size and power properties of the test. This new test is applied to a set of European stock markets to analyze spillovers during the recent European crisis and to distinguish contagion from interdependence effects.

KEY WORDS: Financial spillover; Kernel-based test; Tails.

1. INTRODUCTION

Analysis of causal relationships is an important aspect of theoretical and empirical contributions in quantitative economics (see the special issues of the *Journal of Econometrics* in 1988 and 2006). Although the concept of causality as defined by Granger (1969) is broad and consists of testing transmission effects between the whole distribution of random variables, extensions of this concept have recently been proposed, such as causality in the frequency domain or for specific distribution moments. For instance, Granger causality in the mean (Granger 1980, 1988) is widely used in macroeconomics. For example, Sims (1972, 1980) test for Granger causality in the mean of money and income. Granger, Robins, and Engle (1986) also introduced the concept of Granger causality in variance to test for causal effects in the second-order moment between financial series. This concept was further explored by Cheung and Ng (1996), Kanas and Kouretas (2002), and Hafner and Herwartz (2008), among others. A unified treatment of Granger causality in the mean and variance is formalized by Comte and Lieberman (2000).

More recent contributions have focused on the concept of Granger causality in quantiles, a particularly important issue for non-Gaussian distributions that exhibit asymmetry, fat-tail characteristics, and nonlinearity (Lee and Yang 2012; Jeong, Härdle, and Song 2012). Indeed, for these distributions, the dynamic in the tails can differ substantially from that of the center of the distribution. In this case, the information content of the quantiles provides greater insight into the distribution than the content provided by the mean. Lee and Yang (2012) developed a parametric methodology for Granger causality in quantiles based on the conditional predictive ability (CPA) framework of Giacomini and White (2006). Jeong, Härdle, and Song (2012)

introduced a nonparametric approach to test for causality in quantiles and apply it to the detection of causal relations between the price of crude oil, the USD/GBP exchange rate, and the price of gold. A closely related but different concept is Granger causality in tail events by Hong, Liu, and Wang (2009). A tail event occurs when the value of a time series is lower than its value-at-risk at a specified risk level. Hence, the test determines if an extreme downside movement for a given time series has predictive content for an extreme downside movement for another time series, with numerous potential applications in risk management.

All tests of causality in quantiles and tail events share the limit that statistical inference is exclusively performed at a specific fixed level of the quantile. At this given level, the null hypothesis should not be rejected, while the opposite conclusion should hold for another quantile level. Indeed, as emphasized by Granger (2003) and Engle and Manganelli (2004), the time-series behavior of quantiles can vary considerably across the distribution because of long memory or nonstationarity. Hence, a Granger causality test in quantiles or tail events that does not consider a large number of quantiles simultaneously over the distribution support would be too restrictive. Because the predictive distribution of a time series is entirely determined by its

quantiles, testing for Granger causality for the range of quantiles over the distribution support is equivalent to testing for Granger causality in the distribution.

Testing procedures for Granger causality in the whole distribution in a time series context are developed only in Su and White (2007, 2008, 2012, 2014), Bouezmarni, Rombouts, and Taamouti (2012), and Taamouti, Bouezmarni, and El Ghouch (2014). For example, Su and White (2012) introduced a conditional independence specification test that can be used to test for Granger causality in quantiles over a continuum of values of quantile levels between (0, 1). Bouezmarni, Rombouts, and Taamouti (2012) constructed a nonparametric Granger causality test in distribution based on conditional independence in the framework of copulas. Taamouti, Bouezmarni, and El Ghouch (2014) also developed alternative Granger causality tests using the copulas theory. The present article adds to this literature by proposing a new nonparametric test for Granger causality in the whole distribution between two time series. To summarize, our testing procedure consists of dividing the distribution support of each series into a multivariate process of dynamic interquantile event variables. The test for causality in the distribution between the two series is enabled by an analysis of the cross-correlation structures of the multivariate processes and relies on the generalized portmanteau test for independence between multivariate processes developed by Bouhaddioui and Roy (2006).

Although our approach examines the strong version of the Granger causality concept (Granger 1969), it is sufficiently flexible that it can be used to test for causality in specific regions on the distribution supports, such as the center or the tails (left or right). While Candelon, Joëts, and Tokpavi (2013) introduced a parametric test to check for Granger causality in distribution tails, that methodology, in contrast to the tests developed in this article, does not apply to other regions of the distribution such as the center. For example, the test can be used to test for causality in the left-tail distribution for two time series. In this case, the multivariate process of interquantile event variables should be defined to focus the analysis exclusively on this part of the distribution. This flexibility constitutes a clear advantage of our methodology compared to those based on copulas theory (Bouezmarni, Rombouts, and Taamouti 2012; Taamouti, Bouezmarni, and El Ghouch 2014) and allows us to go beyond the simple rejection of the null hypothesis of Granger causality for the whole distribution because it identifies the specific regions for which Granger causality is rejected. Our test statistic is also a multivariate extension of the kernel-based nonparametric Granger causality test in tail events by Hong, Liu, and Wang (2009) and therefore shares its main advantage: the ability to examine a large number of lags by discounting higher-order lags. This characteristic is consistent with empirical evidence in finance that recent events have a greater influence on current market trends than older ones. Thus, our Granger causality test in distribution is different from those available in the literature that checks for causality uniformly for a limited number of lags.

Technically, we demonstrate that the test has a standard Gaussian distribution under the null hypothesis, which is free of parameter estimation uncertainty. Monte Carlo simulations confirm that the Gaussian distribution provides a good approximation of the distribution of our test statistic, even in small samples. Moreover, the test has the power to reject the null

hypothesis of causality in distribution stemming from different sources, including linear and nonlinear causality in the mean and causality in the variance.

To illustrate the importance of this test for the empirical literature, we explore the spillovers that have occurred within European stock markets during the recent crisis. Our Granger causality test in distribution allows us to consider asymmetry between markets (which is not possible using correlation), to take into account a break in volatility (as suggested by Forbes and Rigobon 2002) and to distinguish between contagion and interdependence. Indeed, interdependence is a long-run path that occurs during "normal periods" and therefore concerns the center of the distribution exclusively. By contrast, contagion is detected by a short-run abrupt increase in the causal linkages that occur during crisis periods, that is, only in the tails of the distribution. Because our test is designed to check for causality in specific regions of the distribution, it can be used to check for interdependence or contagion. Anticipating our results, we find weak (respectively, strong) support for interdependence (respectively, contagion) during the recent crisis. Interestingly, we observe a strong asymmetry between causal tests in the right and left tails: whereas spillovers are important in crisis periods, they are only weakly present during upswing periods. Such a result constitutes an important feature of European stock markets.

The article is organized as follows: the second section presents the Granger causality test in distribution. The properties of this test are analyzed in Section 3 via a Monte Carlo simulation experiment. Section 4 proposes the empirical application, and Section 5 concludes.

2. NONPARAMETRIC TEST FOR GRANGER CAUSALITY IN DISTRIBUTION

This section presents our kernel-based test for Granger causality in distribution between two time series. Because this test is a multivariate extension of the Granger causality test in tail events introduced by Hong, Liu, and Wang (2009), we first present their test and then introduce the new approach.

2.1 Granger Causality in Tail Event

For two time series X_t and Y_t, the Granger causality test in tail events developed by Hong, Liu, and Wang (2009) determines whether an extreme downside risk from Y_t can be considered a lagged indicator for an extreme downside risk for X_t. Hong, Liu, and Wang (2009) identified an extreme downside risk as a situation in which X_t and Y_t are lower than their respective value-at-risk (VaR) at a prespecified level α. VaR is a risk measure often used by financial analysts and risk managers to measure and monitor the risk of loss for a trading or investment portfolio. The VaR of an instrument or a portfolio of instruments is the maximum dollar loss within the $\alpha\%$-confidence interval (Jorion 2007). For the two time series X_t and Y_t, we have

$$\Pr\left[X_t < \text{VaR}_t^X\left(\theta_X^0\right) \big| \mathcal{F}_{t-1}^X\right] = \alpha, \tag{1}$$

$$\Pr\left[Y_t < \text{VaR}_t^Y\left(\theta_Y^0\right) \big| \mathcal{F}_{t-1}^Y\right] = \alpha, \tag{2}$$

where $\text{VaR}_t^X\left(\theta_X^0\right)$ and $\text{VaR}_t^Y\left(\theta_Y^0\right)$ are the VaR of X_t and Y_t, respectively, at time t; and θ_X^0 and θ_Y^0 are the true unknown

finite-dimensional parameters related to the specification of the VaR model for each variable. The information sets $\mathcal{F}_{t-1}^X$ and $\mathcal{F}_{t-1}^Y$ are defined as

$$\mathcal{F}_{t-1}^X = \{X_l, l \leq t-1\}, \tag{3}$$

$$\mathcal{F}_{t-1}^Y = \{Y_l, l \leq t-1\}. \tag{4}$$

In the framework of Hong, Liu, and Wang (2009), an extreme downside risk occurs at time t for X_t if the tail event variable $Z_t^X(\theta_X^0)$ is equal to one, with

$$Z_t^X(\theta_X^0) = \begin{cases} 1 & \text{if} \quad X_t < \text{VaR}_t^X(\theta_X^0) \\ 0 & \text{else.} \end{cases} \tag{5}$$

Similarly, an extreme downside risk for Y_t corresponds to $Z_t^Y(\theta_Y^0)$ taking a value of one, with

$$Z_t^Y(\theta_Y^0) = \begin{cases} 1 & \text{if} \quad Y_t < \text{VaR}_t^Y(\theta_Y^0) \\ 0 & \text{else.} \end{cases} \tag{6}$$

Hence, the time series Y_t does not Granger-cause (in downside risk or tail event at level α) the time series X_t if the following hypothesis holds

$$\mathbb{H}_0 : \mathbb{E}\left[Z_t^X(\theta_X^0) \middle| \mathcal{F}_{t-1}^{X\&Y}\right] = \mathbb{E}\left[Z_t^X(\theta_X^0) \middle| \mathcal{F}_{t-1}^X\right], \tag{7}$$

with

$$\mathcal{F}_{t-1}^{X\&Y} = \{(X_l, Y_l), \ l \leq t-1\}. \tag{8}$$

Under the null hypothesis and at the risk level α, spillovers of extreme downside movements from Y_t to X_t do not exist. Hong, Liu, and Wang (2009) proposed a nonparametric approach for testing for the null hypothesis in (7) based on the cross-spectrum of the estimated bivariate process of tail event variables $\{\widehat{Z}_t^X, \widehat{Z}_t^Y\}$, with components

$$\widehat{Z}_t^X \equiv Z_t^X(\widehat{\theta}_X), \quad \widehat{Z}_t^Y \equiv Z_t^Y(\widehat{\theta}_Y), \tag{9}$$

where $\widehat{\theta}_X$ and $\widehat{\theta}_Y$ are consistent estimators of the true unknown parameters θ_X^0 and θ_Y^0, respectively. To present their test statistic, let us define the sample cross-covariance function between the estimated tail event variables as

$$\widehat{C}(j) = \begin{cases} T^{-1} \sum_{t=1+j}^{T} \left(\widehat{Z}_t^X - \widehat{\alpha}_X\right)\left(\widehat{Z}_{t-j}^Y - \widehat{\alpha}_Y\right), \ 0 \leq j \leq T-1 \\ \\ T^{-1} \sum_{t=1-j}^{T} \left(\widehat{Z}_{t+j}^X - \widehat{\alpha}_X\right)\left(\widehat{Z}_t^Y - \widehat{\alpha}_Y\right), \ 1-T \leq j \leq 0, \end{cases} \tag{10}$$

where T is the sample length and $\widehat{\alpha}_X$ and $\widehat{\alpha}_Y$ are the sample mean of $\widehat{Z}_t^X$ and $\widehat{Z}_t^Y$, respectively. The sample cross-correlation function $\widehat{\rho}(j)$ is then equivalent to

$$\widehat{\rho}(j) = \frac{\widehat{C}(j)}{S_X S_Y}, \tag{11}$$

where S_X^2 and S_Y^2 are the sample variances of $\widehat{Z}_t^X$ and $\widehat{Z}_t^Y$, respectively. Using the cross-correlation function, the kernel estimator for the cross-spectral density of the bivariate process

of tail event variables corresponds to

$$\widehat{f}(\omega) = \frac{1}{2\pi} \sum_{j=1-T}^{T-1} \kappa(j\,M)\,\widehat{\rho}(j)\,e^{-ij\omega}, \tag{12}$$

where $\kappa(.)$ is a given kernel function and M is the truncation parameter. The truncation parameter M is a function of the sample size T such that $M \to \infty$ and $M/T \to 0$ as $T \to \infty$. The kernel is a symmetric function defined on the real line and taking value in $[-1, 1]$. It must be continuous at zero, with at most a finite number of discontinuity points such that

$$\kappa(0) = 1, \tag{13}$$

$$\int_{-\infty}^{\infty} \kappa^2(z)\,dz < \infty. \tag{14}$$

Under the null hypothesis of non-Granger causality in tail events from Y_t to X_t, the kernel estimator for the cross-spectral density is equal to

$$\widehat{f}_1^0(\omega) = \frac{1}{2\pi} \sum_{j=1-T}^{0} \kappa(j\,/M)\,\widehat{\rho}(j)\,e^{-ij\omega}. \tag{15}$$

This equation suggests that the distance between the two estimators $\widehat{f}(\omega)$ and $\widehat{f}_1^0(\omega)$ can be used to test for the null hypothesis. Hong, Liu, and Wang (2009) considered the following quadratic form

$$L^2\left(\widehat{f}, \widehat{f}_1^0\right) = 2\pi \int_{-\pi}^{\pi} \left|\widehat{f}(\omega) - \widehat{f}_1^0(\omega)\right|^2 d\omega, \tag{16}$$

which is equivalent to

$$L^2\left(\widehat{f}, \widehat{f}_1^0\right) = \sum_{j=1}^{T-1} \kappa^2(j\,/M)\,\widehat{\rho}^2(j). \tag{17}$$

The test statistic is a standardized version of the quadratic form given by

$$U_{Y \to X} = \left[T \sum_{j=1}^{T-1} \kappa^2(j\,/M)\,\widehat{\rho}^2(j) - C_T(M)\right] \Big/ D_T(M)^{\frac{1}{2}}, \tag{18}$$

and follows under the null hypothesis a standard Gaussian distribution, with $C_T(M)$ and $D_T(M)$ as the location and scale parameters

$$C_T(M) = \sum_{j=1}^{T-1} (1 - j\,/T)\,\kappa^2(j\,/M), \tag{19}$$

$$D_T(M) = 2 \sum_{j=1}^{T-1} (1 - j\,/T)(1 - (j+1)\,/T)\,\kappa^4(j\,/M). \tag{20}$$

2.2 Granger Causality in Distribution

In this section, we present our multivariate extension of the test of Hong, Liu, and Wang (2009); this extension permits the identification of Granger causality in the whole distribution between two time series.

2.2.1 Notations and the Null Hypothesis. The setting of our testing procedure is as follows. We consider a set $A = \{\alpha_1, \ldots, \alpha_{m+1}\}$ of $m + 1$ VaR risk levels that covers the distribution support of both variables X_t and Y_t, with $0 \le \alpha_1 < \cdots < \alpha_{m+1} \le 100\%$. For the first time series X_t, the corresponding VaRs at time t are $\text{VaR}_{t,s}^X(\theta_X^0, \alpha_s)$, $s = 1, \ldots, m + 1$, with

$$\text{VaR}_{t,1}^X(\theta_X^0, \alpha_1) < \cdots < \text{VaR}_{t,m+1}^X(\theta_X^0, \alpha_{m+1}), \qquad (21)$$

where the vector θ_X^0 is once again the true unknown finite-dimensional parameter related to the specification of the VaR model for X_t. We adopt the convention that $\text{VaR}_{t,s}^X(\theta_X^0, \alpha_s) = -\infty$ for $\alpha_s = 0\%$ and $\text{VaR}_{t,s}^X(\theta_X^0, \alpha_s) = \infty$ for $\alpha_s = 100\%$. We divide the distribution support of X_t into m disjoint regions, each related to the indicator or event variable

$$Z_{t,s}^X(\theta_X^0) = \begin{cases} 1 & \text{if} \quad X_t \ge \text{VaR}_{t,s}^X(\theta_X^0, \alpha_s) \text{ and } X_t \\ & \quad < \text{VaR}_{t,s+1}^X(\theta_X^0, \alpha_{s+1}) \\ \\ 0 & \text{else,} \end{cases} \qquad (22)$$

for $s = 1, \ldots, m$. For illustration, let $m + 1 = 5$, and suppose that the set $A = \{\alpha_1, \alpha_2, \alpha_3, \alpha_4, \alpha_5\} = \{0\%, 20\%, 40\%, 60\%, 80\%\}$. Figure 1 displays the support of X_t, along with the VaRs and the event variables defining the $m = 4$ distinct regions. We do not consider the event variable corresponding to the extreme $m + 1$ region identified by $X_t \ge \text{VaR}_{t,m+1}^X(\theta_X^0, \alpha_{m+1})$; this variable is implicitly defined by the first m event variables.

Now, let $H_t^X(\theta_X^0)$ be the vector of dimension $(m, 1)$ with components of the m event variables

$$H_t^X(\theta_X^0) = \left(Z_{t,1}^X(\theta_X^0), Z_{t,2}^X(\theta_X^0), \ldots, Z_{t,m}^X(\theta_X^0) \right)^T. \qquad (23)$$

We similarly define for the second time series Y_t these event variables collected in the vector $H_t^Y(\theta_Y^0)$, with

$$H_t^Y(\theta_Y^0) = \left(Z_{t,1}^Y(\theta_Y^0), Z_{t,2}^Y(\theta_Y^0), \ldots, Z_{t,m}^Y(\theta_Y^0) \right)^T. \qquad (24)$$

The time series Y_t does not Granger-cause the time series X_t in distribution if the following hypothesis holds

$$\mathbb{H}_0 : \mathbb{E}\left[H_t^X(\theta_X^0) \mid \mathcal{F}_{t-1}^{X\&Y} \right] = \mathbb{E}\left[H_t^X(\theta_X^0) \mid \mathcal{F}_{t-1}^X \right]. \qquad (25)$$

Therefore, Granger causality in the distribution from Y_t to X_t corresponds to Granger causality in the mean from $H_t^Y(\theta_Y^0)$ to $H_t^X(\theta_X^0)$. When the null hypothesis of noncausality in distribution holds, the event variables defined for the variable Y_t along

its distribution support do not have any predictive content for the dynamics of the same event variables over the distribution support of X_t.

Our null hypothesis is sufficiently flexible that it can be used to check for Granger causality in specific regions on the distribution supports, such as the center or tails (left or right), by restricting the set $A = \{\alpha_1, \ldots, \alpha_{m+1}\}$ of VaR levels to selected values. For instance, we can check for Granger causality in the left-tail distribution by setting A to $A = \{0\%, 1\%, 5\%, 10\%\}$. In this case, the rejection of the null hypothesis is of great importance in financial risk management because it indicates spillover effects from Y_t to X_t in the lower tail. Similarly Granger causality in the center of the distribution can be checked by setting, for example, $A = \{20\%, 40\%, 60\%, 80\%\}$. In the next subsection, we construct a nonparametric kernel-based test statistic to test for our general null hypothesis of noncausality (25) and analyze its asymptotic distribution.

2.2.2 Test Statistic and Asymptotic Distribution. The construction of this test statistic is closely related to the article of Bouhaddioui and Roy (2006), which proposes a generalized portmanteau test for the independence of two infinite-order vector auto regressive (VAR) series. Nevertheless, the asymptotic analysis differs because (i) we are not in a VaR framework, (ii) and the event variables we are considering, $Z_{t,s}^X(\theta_X^0)$ and $Z_{t,s}^Y(\theta_Y^0)$, are indicator variables, which are therefore not differentiable with respect to the unknown parameters θ_X^0 and θ_Y^0, respectively. To address this lack of differentiability, we consider several asymptotic results derived in Hong, Liu, and Wang (2009).

To present the test statistic for our general null hypothesis of noncausality, consider $\widehat{H}_t^X \equiv H_t^X(\widehat{\theta}_X)$ and $\widehat{H}_t^Y \equiv H_t^Y(\widehat{\theta}_Y)$ the estimated counterparts of the multivariate processes of event variables $H_t^X(\theta_X^0)$ and $H_t^Y(\theta_Y^0)$, respectively, with $\widehat{\theta}_X$ and $\widehat{\theta}_Y$ as $\sqrt{T}$ consistent estimators of the true unknown parameter vectors θ_X^0 and θ_Y^0. Let $\widehat{\Lambda}(j)$ denote the sample cross-covariance matrix between $\widehat{H}_t^X$ and $\widehat{H}_t^Y$, with

$$\widehat{\Lambda}(j) \equiv \begin{cases} T^{-1} \sum_{t=1+j}^{T} \left(\widehat{H}_t^X - \widehat{\Pi}_X \right) \left(\widehat{H}_{t-j}^Y - \widehat{\Pi}_Y \right)^T & 0 \le j \le T - 1 \\ \\ T^{-1} \sum_{t=1-j}^{T} \left(\widehat{H}_{t+j}^X - \widehat{\Pi}_X \right) \left(\widehat{H}_t^Y - \widehat{\Pi}_Y \right)^T & 1 - T \le j \le 0, \end{cases}$$
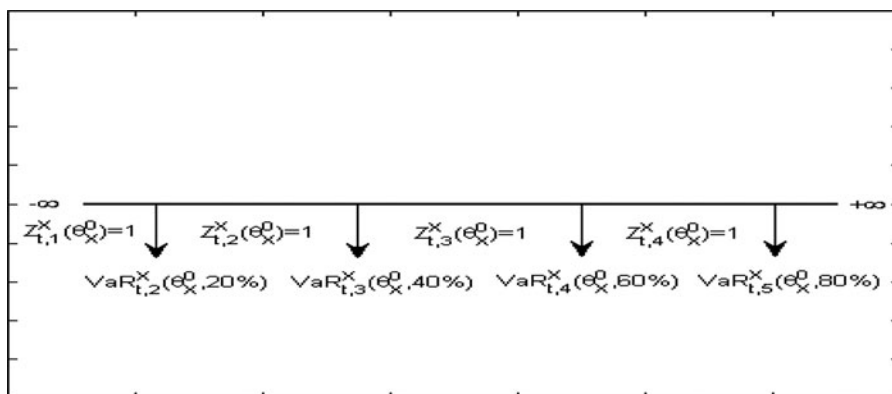$$(26)$$

Figure 1. Distribution support of X and localization of VaRs and event variables.

where the vector $\widehat{\Pi}_X$ (respectively, $\widehat{\Pi}_Y$) of length m is the sample mean of $\widehat{H}_t^X$ (respectively, $\widehat{H}_t^Y$). As in the univariate setting of Hong, Liu, and Wang (2009), we can replace $\widehat{\Pi}_X$ and $\widehat{\Pi}_Y$ by $\Pi_X = \mathbb{E}\left(H_t^X\left(\theta_X^0\right)\right)$ and $\Pi_Y = \mathbb{E}\left(H_t^Y\left(\theta_Y^0\right)\right)$, respectively, without affecting the asymptotic distribution of our test statistic. The corresponding sample cross-correlation matrix $\widehat{R}(j)$ equals

$$\widehat{R}(j) = \mathrm{D}\left(\widehat{\Sigma}_X\right)^{-1/2} \widehat{\Lambda}(j) \mathrm{D}\left(\widehat{\Sigma}_Y\right)^{-1/2}, \qquad (27)$$

where $\mathrm{D}(.)$ represents the diagonal form of a matrix and $\widehat{\Sigma}_X$ and $\widehat{\Sigma}_Y$ are the sample covariance matrices of $\widehat{H}_t^X$ and $\widehat{H}_t^Y$, respectively.

The test statistic can thus be expressed as the following weighted quadratic form that accounts for the dependence between the current value of $\widehat{H}_t^X$ and the lagged values of $\widehat{H}_t^Y$

$$\widehat{\mathcal{T}} = \sum_{j=1}^{T-1} \kappa^2(j/M)\,\widehat{Q}(j), \qquad (28)$$

where $\kappa(.)$ is a kernel function, M is the truncation parameter, and $\widehat{Q}(j)$ is equal to

$$\widehat{Q}(j) = T \mathrm{vec}\left(\widehat{R}(j)\right)^T \left(\widehat{\Gamma}_X^{-1} \otimes \widehat{\Gamma}_Y^{-1}\right) \mathrm{vec}\left(\widehat{R}(j)\right), \qquad (29)$$

where $\widehat{\Gamma}_X$ and $\widehat{\Gamma}_Y$ are the sample correlation matrices of $\widehat{H}_t^X$ and $\widehat{H}_t^Y$, respectively. The restrictions on the truncation parameter M and the kernel function $\kappa(.)$ are the same as those considered by Hong, Liu, and Wang (2009) in their univariate setting (see Section 2.1). Most common kernels used in spectral analysis (Daniell, Parzen, Bartlett, truncated uniform) satisfy these restrictions. Moreover, as discussed by Hong, Liu, and Wang (2009), the choice of kernel is not important because they lead to comparable powers except for the uniform kernel, which does not discount higher-order lags. See Bouhaddioui and Roy (2006) for the same conclusion in a multivariate setting.

Following Bouhaddioui and Roy (2006), our test statistic is a centered and scaled version of the quadratic form in (28), that is,

$$V_{Y \to X} = \frac{\widehat{\mathcal{T}} - m^2 C_T(M)}{\left(m^2 D_T(M)\right)^{1/2}}, \qquad (30)$$

where $C_T(M)$ and $D_T(M)$ are as defined in (19) and (20), respectively. The above test statistic generalizes the one in Hong, Liu, and Wang (2009) in a multivariate setting. When m is equal to one, which corresponds to the univariate case in which each of the vectors $\widehat{H}_t^X$ and $\widehat{H}_t^Y$ has only one event variable, the test statistic $V_{Y \to X}$ in (30) is exactly equal to the test statistic in (18). The following proposition yields the asymptotic distribution of our test statistic.

Proposition 1. Suppose that the assumptions of Theorem 1 in Hong, Liu, and Wang (2009) hold. Then, under the null hypothesis of no Granger causality in distribution as stated in (25), we have

$$V_{Y \to X} = \frac{\widehat{\mathcal{T}} - m^2 C_T(M)}{\left(m^2 D_T(M)\right)^{1/2}} \longrightarrow^d \mathcal{N}(0, 1).$$

The assumptions of Theorem 1 in Hong, Liu, and Wang (2009) impose several regulatory conditions on the time se-

ries X_t and Y_t; on the VaR models used, including smoothness, moment conditions, and adequacy; on the kernel function $\kappa(.)$; and on the truncation parameter M. The latter should be equal to $M = cT^v$ with $0 < c < \infty$, $0 < v < 1/2$, $v < \min\left(\frac{2}{d-2}, \frac{3}{d-1}\right)$ if $d \equiv \max(d_X, d_Y) > 2$ and d_X (respectively, d_Y) is the dimension of the parameter θ_X (respectively, θ_Y). See Hong, Liu, and Wang (2009, pp. 275) for a complete discussion of these assumptions.

The proof of Proposition 1 proceeds as follows. Consider the following decomposition of our test statistic

$$V_{Y \to X} = \frac{\mathcal{T}^* - m^2 C_T(M)}{\left(m^2 D_T(M)\right)^{1/2}} + \frac{\widehat{\mathcal{T}} - \mathcal{T}^*}{\left(m^2 D_T(M)\right)^{1/2}}, \qquad (31)$$

where $\mathcal{T}^*$ is the pseudo version of the weighted quadratic form in (28) and (29) computed using the true correlation matrices Γ_X and Γ_Y, that is,

$$\mathcal{T}^* = \sum_{j=1}^{T-1} \kappa^2(j/M)\,\widehat{Q}^*(j), \qquad (32)$$

$$\widehat{Q}^*(j) = T \mathrm{vec}\left(\widehat{R}(j)\right)^T \left(\Gamma_X^{-1} \otimes \Gamma_Y^{-1}\right) \mathrm{vec}\left(\widehat{R}(j)\right). \qquad (33)$$

Under the decomposition in (31), the proof of Proposition 1 is given by the following two lemmas:

Lemma 1. Under the null hypothesis of no Granger causality in distribution and the assumptions of Theorem 1 in Hong, Liu, and Wang (2009), we have

$$\frac{\mathcal{T}^* - m^2 C_T(M)}{\left(m^2 D_T(M)\right)^{1/2}} \longrightarrow^d \mathcal{N}(0, 1). \qquad (34)$$

Lemma 2. Under the assumptions of Theorem 1 in Hong, Liu, and Wang (2009), we have

$$\frac{\widehat{\mathcal{T}} - \mathcal{T}^*}{\left(m^2 D_T(M)\right)^{1/2}} \longrightarrow^p 0. \qquad (35)$$

The proofs of these two Lemmas are reported in Appendix A.

3. SMALL SAMPLE PROPERTIES

In this section, we study the finite sample properties of our test via Monte Carlo simulation experiments. We analyze the size in the first part of the section, while the remainder of the section is devoted to an analysis of the power.

3.1 Empirical Size Analysis

We simulate the size of the nonparametric test of Granger causality in distribution assuming the following data-generating process (DGP) for the second time series Y_t:

$$\begin{cases} Y_t = 0.5 Y_{t-1} + u_{t,Y}, \\ u_{t,Y} = \sigma_{t,Y} v_{t,Y}, \\ \sigma_{t,Y}^2 = 0.1 + 0.9\sigma_{t-1,Y}^2 + 0.08 u_{t-1,Y}^2, \\ v_{t,Y} \overset{\text{iid}}{\sim} N(0, 1), \end{cases} \qquad (36)$$

which corresponds to an AR(1)-GARCH(1,1) model. We make the assumption that the first time series X_t follows the same process. Because the two processes are generated independently, there is no Granger causality in distribution between them. For a given value of sample size $T \in \{500, 1000, 2000\}$, for each simulation we compute our test statistic in (30) and make inferences using the asymptotic Gaussian distribution. For the computation of the test statistic, we need to specify a model to estimate the VaRs (at the risk level $\alpha_1, \ldots, \alpha_{m+1}$) and the m event variables for each variable X_t and Y_t. The $m + 1$ VaRs are computed using an AR(1)-GARCH(1,1) model estimated by quasi-maximum likelihood. The estimated values of the $m + 1$ VaRs at time t are

$$\text{VaR}_{t,s}^X = \widehat{\mu}_{t,X} + \widehat{\sigma}_{t,X} q \left(\widehat{v}_{t,X}, \alpha_s \right), \quad s = 1, \ldots, m + 1, \quad (37)$$

where $\widehat{\mu}_{t,X}$ and $\widehat{\sigma}_{t,X}$ are the fitted conditional mean and standard deviation at time t, respectively, and $q \left(\widehat{v}_{t,X}, \alpha_s \right)$ is the empirical quantile of order α_s of the estimated standardized innovations. We proceed similarly to compute the $m + 1$ VaRs and the corresponding m event variables for the second time series Y_t. Note that we set the parameter $m + 1$ to 14 and the set A to $A = \{\alpha_1, \alpha_2, \ldots, \alpha_{14}\} = \{0\%, 1\%, 5\%, 10\%, 20\%, \ldots, 90\%, 95\%, 99\%\}$, which covers regions in the tails and the center of the distribution support of each time series. For $\alpha_s = 0\%$, the VaR corresponds to $-\infty$. We must also make a choice about the kernel function to compute our test statistic. We consider the four different standard kernels, that is, the Daniell (DAN), the Parzen (PAR), the Bartlett (BAR), and the truncated uniform (TR) kernels. See Appendix B for the description of the four kernel functions.

Finally, for the choice of truncation parameter M, we use three different values: $M = [\ln(T)]$, $M = \left[1.5 T^{0.3}\right]$, and $M = \left[2 T^{0.3}\right]$, where $[.]$ is the integer portion of the argument. These rates lead to the values $M = 6, 10, 13$ for $T = 500$, $M = 7, 12, 16$ for $T = 1000$, and $M = 8, 15, 20$ for $T = 2000$. These values cover a range of lag orders for the sample sizes considered. Table 1 displays the empirical sizes of our test over 1000 simulations and for two different significance levels $\eta \in (5\%, 10\%)$. The results in Table 1 indicate that our test is adequately sized. Indeed, the rejection frequencies are close to the significance levels. Hence, the standard Gaussian distribution asymptotically provides a good approximation of the distribution of our test statistic. This result appears to hold regardless of the kernel function used and the value of the truncation parameter M.

3.2 Empirical Power Analysis

We now simulate the empirical power of our test. Because causality in distribution springs from causality in moments such as mean or variance, we assume different DGPs corresponding to these cases. The first DGP assumes the existence of a linear Granger causality in the mean to generate data under the alternative hypothesis. Hence, we assume that the second time series Y_t has the DGP in (36) and that the first time series X_t is the

Table 1. Empirical sizes of the Granger causality test in distribution

T	M	η	DAN	BAR	PAR	TR
500	6	5%	5.90	6.00	5.60	5.60
		10%	11.20	10.60	10.70	10.30
	10	5%	5.50	5.40	5.70	4.50
		10%	10.20	9.70	10.70	11.40
	13	5%	5.50	5.40	5.00	6.00
		10%	9.90	10.00	10.20	10.70
1000	7	5%	5.30	5.40	4.70	6.10
		10%	9.60	9.50	10.80	12.10
	12	5%	5.70	6.00	5.00	6.00
		10%	11.90	10.50	9.70	10.10
	16	5%	6.00	5.90	5.50	5.90
		10%	10.60	11.20	10.30	10.60
2000	8	5%	4.40	4.30	5.70	5.50
		10%	10.30	10.80	10.00	9.80
	15	5%	5.40	5.30	4.70	5.10
		10%	8.60	9.20	10.20	8.90
	20	5%	5.90	5.60	4.80	4.70
		10%	9.30	9.40	9.60	9.10

NOTES: The table displays the empirical sizes (in %) of the Granger causality test in distribution. Rejection frequencies are reported over 1000 simulations for two significance levels η, where T is the sample size and M is the truncation parameter. DAN, BAR, PAR, and TR refer to the Daniell, Bartlett, Parzen, and truncated uniform kernels, respectively.

following

$$\begin{cases} X_t = 0.5 X_{t-1} + 0.4 Y_{t-1} + u_{t,X}, \\ u_{t,X} = \sigma_{t,X} v_{t,X}, \\ \sigma_{t,X}^2 = 0.1 + 0.9 \sigma_{t-1,X}^2 + 0.08 u_{t-1,X}^2, \\ v_{t,X} \overset{\text{iid}}{\sim} N(0, 1). \end{cases} \quad (38)$$

The empirical powers of our test are computed over 1000 simulations for $T \in \{500, 1000, 2000\}$. As in the analysis of size, we consider three values of the truncation parameter M, and two significance levels $\eta = 5\%, 10\%$. The results for the four kernels used in the analysis of size are reported in Table 2. For comparison, Table 2 also displays the results of the Granger causality test in mean in parentheses. To ensure an appropriate comparison, we do not use the usual parametric Granger causality test in mean derived from a vector autoregressive model but consider instead the kernel-based nonparametric Granger causality test in mean introduced by Hong (1996). The results in Table 2 indicate that our kernel-based nonparametric test for Granger causality in distribution has appealing power properties. For instance, with the Daniell kernel, the rejection frequencies of the null hypothesis for $(T, M) = (500, 6)$ equal 84.40% and 89.80% for $\eta = 5\%$ and 10%, respectively. For $T = 2000$, the powers are, in most cases, equal to one. The rejection frequencies of the Granger causality test in mean are always equal to or close to 100% and hence are higher than the ones obtained by applying our Granger causality test in distribution for the smallest sample size. This result is expected because the assumed causality in distribution springs from causality in the mean. In all configurations, the uniform kernel leads to the smallest powers because its uniform weighting scheme does not discount higher-order lags. Moreover, we observe as Hong, Liu, and Wang (2009) did

Table 2. Empirical powers of the Granger causality test in distribution: DGP1

T	M	η	DAN	BAR	PAR	TR
	6	5%	84.40 (99.80)	87.20 (99.80)	91.20 (99.80)	55.40 (99.80)
		10%	89.80 (99.80)	91.60 (99.80)	94.20 (99.80)	64.00 (99.80)
500	10	5%	73.20 (99.80)	78.60 (99.80)	85.60 (99.80)	42.80 (99.80)
		10%	79.60 (99.80)	84.60 (99.80)	90.20 (99.80)	50.20 (99.80)
	13	5%	67.80 (99.80)	73.80 (99.80)	79.60 (99.80)	35.00 (99.80)
		10%	74.60 (99.80)	80.20 (99.80)	86.00 (99.80)	44.80 (99.80)
	7	5%	99.40 (100.00)	99.60 (100.00)	99.60 (100.00)	90.80 (100.00)
		10%	99.60 (100.00)	99.60 (100.00)	100.00 (100.00)	95.20 (100.00)
1000	12	5%	98.00 (100.00)	99.20 (100.00)	99.20 (100.00)	79.80 (100.00)
		10%	98.80 (100.00)	99.40 (100.00)	99.60 (100.00)	87.60 (100.00)
	16	5%	96.00 (100.00)	97.80 (100.00)	99.20 (100.00)	71.60 (100.00)
		10%	96.80 (100.00)	99.00 (100.00)	99.20 (100.00)	78.40 (100.00)
	8	5%	100.00 (100.00)	100.00 (100.00)	100.00 (100.00)	100.00 (100.00)
		10%	100.00 (100.00)	100.00 (100.00)	100.00 (100.00)	100.00 (100.00)
2000	15	5%	100.00 (100.00)	100.00 (100.00)	100.00 (100.00)	99.60 (100.00)
		10%	100.00 (100.00)	100.00 (100.00)	100.00 (100.00)	99.60 (100.00)
	20	5%	100.00 (100.00)	100.00 (100.00)	100.00 (100.00)	99.00 (100.00)
		10%	100.00 (100.00)	100.00 (100.00)	100.00 (100.00)	99.40 (100.00)

NOTES: The table displays the empirical powers (in %) of the Granger causality test in distribution. Rejection frequencies are reported over 1000 simulations for two significance levels η, where T is the sample size and M is the truncation parameter. For comparison, we also report (in parentheses) the rejection frequencies of the kernel-based nonparametric test in mean. DAN, BAR, PAR, and TR refer to the Daniell, Bartlett, Parzen, and truncated uniform kernels, respectively. Data are generated under the alternative hypothesis assuming linear Granger causality in mean.

Table 3. Empirical powers of the Granger causality test in distribution: DGP2

T	M	η	DAN	BAR	PAR	TR
	6	5%	100.00 (48.60)	100.00 (49.20)	100.00 (50.80)	98.20 (40.80)
		10%	100.00 (52.80)	100.00 (52.40)	100.00 (53.20)	98.80 (47.40)
500	10	5%	99.60 (48.20)	99.80 (48.20)	100.00 (48.60)	94.60 (42.00)
		10%	99.80 (51.20)	100.00 (52.00)	100.00 (51.80)	97.40 (47.60)
	13	5%	99.20 (47.60)	99.60 (49.20)	99.80 (48.00)	90.00 (41.20)
		10%	99.60 (51.20)	99.80 (52.20)	100.00 (52.00)	93.40 (48.60)
	7	5%	100.00 (51.00)	100.00 (50.20)	100.00 (50.00)	100.00 (45.20)
		10%	100.00 (54.00)	100.00 (53.20)	100.00 (52.80)	100.00 (49.40)
1000	12	5%	100.00 (49.40)	100.00 (50.80)	100.00 (50.80)	100.00 (48.20)
		10%	100.00 (54.40)	100.00 (54.40)	100.00 (52.80)	100.00 (53.80)
	16	5%	100.00 (50.60)	100.00 (50.60)	100.00 (50.80)	100.00 (48.20)
		10%	100.00 (53.80)	100.00 (54.80)	100.00 (53.40)	99.60 (55.40)
	8	5%	100.00 (52.20)	100.00 (52.20)	100.00 (51.00)	100.00 (52.20)
		10%	100.00 (56.60)	100.00 (56.00)	100.00 (55.00)	100.00 (56.60)
2000	15	5%	100.00 (55.40)	100.00 (54.80)	100.00 (52.60)	100.00 (55.60)
		10%	100.00 (60.40)	100.00 (59.80)	100.00 (56.60)	100.00 (60.60)
	20	5%	100.00 (57.40)	100.00 (56.60)	100.00 (53.60)	100.00 (56.20)
		10%	100.00 (62.20)	100.00 (61.80)	100.00 (57.60)	100.00 (61.80)

NOTES: The table displays the empirical powers (in %) of the Granger causality test in distribution. Rejection frequencies are reported over 1000 simulations for two significance levels η, where T is the sample size and M is the truncation parameter. For comparison, we also report (in parentheses) the rejection frequencies of the kernel-based nonparametric test in mean. DAN, BAR, PAR, and TR refer to the Daniell, Bartlett, Parzen, and truncated uniform kernels, respectively. Data are generated under the alternative hypothesis assuming nonlinear Granger causality in mean.

that the rejection frequencies of the null hypothesis decrease with the truncation parameter M.

To stress the relevance of our testing approach, we consider a second type of alternative DGP that assumes causality in distribution stemming from a nonlinear form of causality in the mean. Precisely, we generate data for the time series Y_t using the specification in (36), and the first time series is generated as follows:

$$\begin{cases} X_t = 0.5X_{t-1} + 0.3Y_{t-1}^2 + u_{t,X}, \\ u_{t,X} = \sigma_{t,X} v_{t,X}, \\ \sigma_{t,X}^2 = 0.1 + 0.9\sigma_{t-1,X}^2 + 0.08u_{t-1,X}^2, \\ v_{t,X} \stackrel{iid}{\sim} N(0,1). \end{cases} \quad (39)$$

Table 3 reports the rejection frequencies over 1000 simulations. The presentation is similar to that in Table 2. We observe that our test continues to exhibit good power in detecting this nonlinear form of causality. Indeed, the rejection frequencies are in all cases close to or even equal to one, even when considering the uniform kernel. By contrast, the Granger causality test in mean fails to reject the null hypothesis for approximately half of the simulations, and the rejection frequencies do not seem to increase significantly with the sample size. For illustration, the rejection frequency of the null hypothesis for $(T, M) = (500, 6)$

amounts to 100% for the Daniell kernel and $\eta = 5\%$, while it is only equal to 48.60% for the causality test in mean.

Finally, we generate data under the alternative hypothesis, assuming Granger causality in variance. Formally, we suppose once again that Y_t has the specification in (36), and X_t is generated as

$$\begin{cases} X_t = 0.5X_{t-1} + u_{t,X}, \\ u_{t,X} = \sigma_{t,X} v_{t,X}, \\ \sigma_{t,X}^2 = 0.1 + 0.8\sigma_{t-1,X}^2 + 0.08u_{t-1,X}^2 + 0.8u_{t-1,Y}^2, \\ v_{t,X} \stackrel{iid}{\sim} N(0,1). \end{cases} \quad (40)$$

Frequency rejections are displayed in Table 4 and are qualitatively similar to those reported in Table 3. Our causality test in distribution performs quite well in rejecting the null hypothesis, while the causality test in mean is less robust and rejects the null in few cases. Finally, we observe that the rejection frequencies are lower than those reported in Tables 2 and 3. This result occurs because (i) causality in variance occurs mainly in the tails and (ii) the dynamics of the tails are more difficult to fit due to the lack of data.

For the DGP in (40), we slightly decrease the persistence of volatility for the process X_t, which is equal to $\alpha_X + \beta_X = 0.08 + 0.8 = 0.88$. This calibration differs from those considered in previous simulations, in which the persistence was set to

Table 4. Empirical powers of the Granger causality test in distribution: DGP3

T	M	η	DAN	BAR	PAR	TR
	6	5%	35.2 (17.80)	33.60 (16.40)	27.80 (14.40)	36.60 (18.40)
		10%	46.60 (22.00)	45.40 (20.20)	38.00 (17.00)	45.00 (25.40)
500	10	5%	39.60 (20.80)	39.60 (20.20)	34.60 (17.80)	34.00 (25.40)
		10%	50.60 (24.60)	50.60 (22.80)	46.80 (21.60)	43.00 (31.40)
	13	5%	40.20 (21.20)	40.80 (20.40)	38.40 (19.40)	33.00 (25.80)
		10%	51.00 (27.20)	51.00 (25.20)	48.60 (22.60)	42.00 (31.20)
	7	5%	48.20 (20.40)	46.20 (17.00)	41.20 (13.40)	43.00 (25.80)
		10%	57.60 (23.60)	57.80 (21.40)	51.40 (16.00)	57.00 (31.40)
1000	12	5%	52.00 (25.80)	52.60 (23.60)	49.00 (18.80)	36.20 (30.80)
		10%	61.00 (31.40)	61.20 (29.40)	58.60 (23.20)	48.20 (35.80)
	16	5%	49.60 (28.80)	52.00 (27.20)	52.80 (22.40)	31.80 (30.20)
		10%	60.20 (34.60)	62.60 (33.00)	60.60 (27.80)	42.40 (37.80)
	8	5%	78.60 (25.00)	77.80 (22.20)	72.20 (18.60)	69.20 (27.60)
		10%	85.60 (31.80)	83.80 (29.00)	79.60 (24.80)	79.40 (33.40)
2000	15	5%	79.20 (28.80)	80.80 (28.40)	78.80 (24.60)	59.40 (33.60)
		10%	85.60 (36.00)	87.20 (33.40)	85.80 (31.00)	71.60 (38.80)
	20	5%	77.80 (31.60)	81.20 (29.20)	79.80 (27.00)	52.80 (38.60)
		10%	85.00 (38.40)	86.00 (36.20)	86.80 (33.00)	64.20 (35.80)

NOTES: The table displays the empirical powers (in %) of the Granger causality test in distribution. Rejection frequencies are reported over 1000 simulations for two significance levels η, where T is the sample size and M is the truncation parameter. For comparison, we also report (in parentheses) the rejection frequencies of the kernel-based nonparametric test in mean. DAN, BAR, PAR, and TR refer to the Daniell, Bartlett, Parzen, and truncated uniform kernels, respectively. Data are generated under the alternative hypothesis assuming Granger causality in variance.

$\alpha_X + \beta_X = 0.98$. We decrease the persistence of volatility because if the volatility of the process X_t is highly persistent, the current volatility $\sigma_{t,X}^2$ must be mainly driven by its past values rather than $u_{t-1,Y}^2$, lowering the effect of causality. To provide further insight on this point, Figure 2 displays the power curve: we fix the parameter α_X to 0.08 and consider different values of β_X, with $\beta_X = 0.5, 0.6, 0.7, 0.8, 0.9$. We observe indeed that the rejection frequencies decrease with persistence.
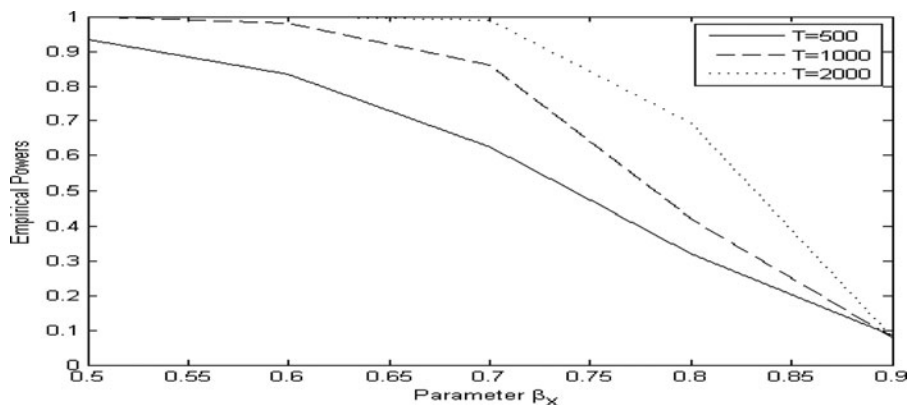
4. EMPIRICAL PART

Recent financial crises have been characterized by rapid, large regional spillovers of negative financial shocks. For example, consecutive to the Greek distress, southern European countries have been contaminated and face skyrocketing refinancing rates. Northern European states have been impacted in an opposite manner. Considered safe harbors for investors, these countries were able to refinance their debt on markets at lower rates. It is obvious that the degree of globalization within the European Union as well as the low degree of fiscal federalism has fostered the speed as well as the amplitude of the transmission mechanism of such a shock. Because southern European countries used foreign capital markets to finance their domestic investments and boost their growth, they have been highly subject to financial instability.

Empirical studies must evaluate the importance of these spillovers. Theoretically it relies on crisis-contingent theories, which explain the increase in market cross-correlation after a shock issued in an origin country as the result of multiple equilibria based on investor psychology; endogenous liquidity shocks causing a portfolio recomposition; and/or political disturbances affecting the exchange rate regime. By contrast, according to noncrisis-contingent theories, the propagation of shocks does not lead to a shift from a good to a bad equilibrium; the increase in cross-correlation is the continuation of linkages (trade and/or financial) that existed before the crisis. The presence of spillovers during a crisis can thus be tested empirically by a significant and transitory increase in cross-correlation between markets (see inter alia King and Wadhwani 1990; Calvo and Reinhart 1995; Baig and Goldfajn 1998). Nevertheless, this intuitive approach, which has the advantage of simplicity because it avoids the identification of transmission channels, presents many shortcomings.

First, Forbes and Rigobon (2002) demonstrated that an increase in correlation can be exclusively driven by higher volatility during crisis periods. In such a case, the increase in correlation could not be attributed to stronger economic interdependence. To correct for this potential bias, they propose the use of the unconditional correlation rather than the conditional one and test for its temporary increase during crisis periods.

Figure 2. Rejection frequencies when considering DGP3 and several values of β_X.

Second, correlation is a symmetrical measure: an increase in the correlation between markets i and j does not provide any information on the direction of the contagion (from i to j, from j to i, or both). For this reason, Bodart and Candelon (2009) preferred to consider an indicator of causality to measure spillovers. It is thus possible to evaluate asymmetrical spillovers that can then move from i to j, j to i, or in both directions. In addition, using the Granger causality approach requires the estimation of multivariate dynamic models, which are less prone to potential misspecification issues.

Addressing both these shortcomings in a classical framework is relatively feasible. However, although comparing causality between precrisis and crisis periods permits the evaluation of spillovers, it does not permit the separation of interdependence from contagion. Interdependence addresses the long-run structural causality between markets and thus provides information on the extent to which markets are integrated. Therefore, interdependence should be tested independently of extreme positive or negative events. By contrast, contagion addresses short-run abrupt increases in causal linkages and occurs exclusively during crisis periods. Thus, testing for contagion requires an exclusive focus on the extreme left tail of the distribution, as in extreme value theory (see Hartmann et al. 2004). Considering the whole distribution to evaluate contagion would hence alter the conclusions. Our new causality test allows all these issues to be addressed because causality can be tested for the whole distribution as well as for specific percentiles of the distribution.

As an illustration, we analyze the recent European crisis considering a set of 12 European daily stock market indices (Austria, Belgium, Finland, France, Germany, Greece, Ireland, Italy, Luxemburg, the Netherlands, Portugal and Spain), yielding 132 pairwise systems. Data are downloaded from Datastream ranging from January 1, 2007 to May 6, 2011 (i.e., $T = 1134$ observations). The first empirical illustration consists of testing for interdependence, which is performed by implementing the pairwise Granger causality for the center of the distribution, that is, removing extreme events located on the right and left tails. A large share of rejection of the noncausality null hypothesis would support the hypothesis of interdependence. Then, in a second analysis, we implement the causality test exclusively for the left tail to test for contagion during crisis. Such a hypothesis would be supported if we observed a higher percentage of noncausality rejection than that previously obtained when considering the center of the distribution. Similarly, the test is conducted for the right tail, that is, the upswing period. We can then compare the strength of contagion during crises and boom periods and determine which period contagion is the most significant.

4.1 The General Design of the Granger Causality Test in Distribution to Test for Spillover

To implement the Granger causality test in distribution in our empirical illustration, we first need to compute for each index $m + 1$ series of VaRs corresponding to $m + 1$ risk level α_s $s = 1, \dots, m + 1$, which cover its distribution support. As for the Monte Carlo simulations, we consider the following set for the VaR levels $A = \{0\%, 1\%, 5\%, 10\%, \dots, 90\%, 95\%, 99\%\}$

with $m + 1 = 14$. To compute the VaRs, we use a semiparametric model. Formally, we suppose that each index returns series $R_{i,t}$ $i = 1, \dots, 12$, following an AR(m)-GARCH(p, q) model, with

$$R_{i,t} = \sum_{j=1}^{m} \phi_{i,j} R_{i,t-j} + \varepsilon_{i,t}, \tag{41}$$

$$\varepsilon_{i,t} = \sigma_{i,t} v_{i,t}, \tag{42}$$

$$\sigma_{i,t}^2 = \kappa_i + \sum_{j=1}^{q} \gamma_{i,j} \varepsilon_{i,t-j}^2 + \sum_{j=1}^{p} \beta_{i,j} \sigma_{i,t-j}^2, \tag{43}$$

and $v_{i,t}$ is an iid innovation with mean zero and unit variance. The choice for an AR(m)-GARCH(p, q) is consistent with the Forbes and Rigobon (2002) correction and accounts for a volatility increase that biases the causality analysis. For each index, this model is estimated using the quasi-maximum likelihood method. Hence, the $m + 1$ series of VaRs are obtained as

$$\text{VaR}_{t,s}^i = \sum_{j=1}^{m} \widehat{\phi}_{i,j} R_{i,t-j} + \widehat{\sigma}_{i,t} q\left(\widehat{v}_{i,t}, \alpha_s\right), \quad s = 1, \dots, m + 1, \tag{44}$$

where $\widehat{\sigma}_{i,t}$ is the fitted volatility at time t for the index number i and $q\left(\widehat{v}_{i,t}, \alpha_s\right)$ is the empirical quantile of order α_s of the estimated standardized innovations $\widehat{v}_{i,t}$. Table 5 displays the estimation results of the AR(m)-GARCH(p, q) models for the indices. As shown by the Ljung–Box test applied to the residuals and their squares, the retained specifications successfully capture the dependence in the first two moments.

With the fitted series of VaRs at hand, we calculate for each index the multivariate process of dynamic interquantiles events variables and compute for each couple (i, j) of indices our kernel-based nonparametric test statistic $V_{j \to i}$ as defined in (30). For the computation, we use the Daniell kernel and set the truncation parameter M to $\left[1.5 T^{0.3}\right]$, which yields a value of $M = 12$ for the whole sample of length $T = 1134$.

Table 5. Estimation results of the AR-GARCH models

Index	$\phi_{i,1}$	κ_i	$\gamma_{i,1}$	$\gamma_{i,2}$	$\gamma_{i,3}$	$\beta_{i,1}$	$\text{LB}_{\widehat{v}_{i,t}}$ (6)	$\text{LB}_{\widehat{v}_{i,t}^2}$ (6)
AT		0.000 (4.148)	0.150 (7.752)			0.839 (44.371)	5.217	1.840
BEL		0.000 (5.597)	0.137 (10.559)			0.839 (63.863)	4.738	2.857
FI		0.000 (3.590)	0.081 (8.170)			0.907 (75.696)	5.750	0.758
FR		0.000 (4.310)	0.127 (8.340)			0.857 (50.623)	3.445	7.400
GER		0.000 (4.568)	0.117 (7.773)			0.863 (50.254)	3.717	6.317
GRE	0.072 (2.184)	0.000 (3.057)	0.116 (7.549)			0.881 (62.744)	10.665	8.400
IE		0.000 (3.792)	0.131 (6.687)			0.853 (40.945)	7.161	1.985
IT		0.000 (4.670)	0.000 (0.000)	0.179 (4.831)		0.797 (34.446)	3.523	5.840
LU		0.000 (3.289)	0.081 (9.318)			0.912 (96.818)	2.789	1.073
NL		0.000 (4.574)	0.129 (8.913)			0.854 (56.663)	4.441	3.166
PT		0.000 (4.185)	0.183 (8.490)			0.800 (39.230)	5.986	1.622
ES		0.000 (5.356)	0.058 (1.787)	0.034 (0.799)	0.160 (4.377)	0.708 (21.312)	3.374	9.580

NOTES: For each index, the table displays the estimation results of the AR-GARCH model in Equations (41)–(43). We report the parameter estimates followed in brackets by the student statistics. The two last columns give the results of the Ljung–Box test applied to the series of the standardized innovations $\widehat{v}_{i,t}$ and its square, respectively, with 6 as the number of lags. The critical value for the rejection of the null hypothesis at the 5% significance level is 12.59.

Table 6. Results of bilateral tests of Granger causality in the center of the distribution

	AT	BEL	FI	FR	GER	GRE	IE	IT	LU	NL	PT	ES	SUM
AT	100.0	**2.3**	27.1	14.0	27.0	38.9	79.1	**2.7**	48.2	**1.0**	19.7	29.6	3
BEL	**0.5**	100.0	27.6	19.9	64.4	53.9	**4.2**	5.3	18.3	31.5	85.6	6.4	2
FI	80.9	98.3	100.0	10.4	90.8	22.6	21.0	70.2	60.1	79.8	21.8	**3.7**	1
FR	42.0	28.0	38.6	100.0	8.2	**0.0**	39.4	10.2	**2.6**	58.3	**0.1**	44.7	3
GER	95.9	26.1	27.4	8.6	100.0	**0.2**	36.9	98.4	5.1	16.3	14.0	98.4	1
GRE	54.1	47.3	28.7	50.7	60.6	100.0	28.3	30.3	55.6	92.1	72.8	70.2	0
IE	42.4	59.6	5.2	11.8	15.7	90.6	100.0	15.9	25.4	85.6	23.4	42.6	0
IT	65.0	42.6	80.9	72.9	9.6	46.2	77.3	100.0	45.3	44.0	46.4	91.9	0
LU	37.6	97.7	65.3	7.2	12.0	91.6	29.7	30.7	100.0	95.0	48.3	11.0	0
NL	94.9	99.3	75.4	15.5	23.7	73.7	35.6	16.1	53.1	100.0	25.8	43.0	0
PT	99.1	86.9	84.4	40.3	**0.2**	9.2	21.2	73.2	42.7	**1.3**	100.0	69.2	2
ES	58.2	71.7	96.6	61.5	64.7	34.9	81.2	9.7	64.6	71.9	**4.0**	100.0	1
SUM	1	1	0	0	1	2	1	1	1	2	2	1	13

NOTES: Each entry of the table gives the *p*-value (in %) of the test of causality in the center of the distribution from the index *j* in the column toward the index *i* in the row. Entries with the rejection of the null hypothesis at the 5% significance level are emphasized in bold. The last column, labeled "Sum," indicates the number of times a given index in a row is Granger-caused by the others. Similarly, the last row, labeled "Sum," indicates the number of times a given index in a column Granger-causes the other indices. The entry corresponding to the last row and the last column gives the total number of significant Granger causalities in the system. The tests are performed over the period ranging from January 1, 2007, to May 6, 2011, with a total of $T = 1134$ observations.

4.2 Testing for Interdependence

To test for interdependence, we follow the general design of the pairwise test of Granger causality in distribution as described above, except that we remove the extreme events from the distribution. The new set A of VaRs risk levels is equal to $A = \{20\%, 30\%, \ldots, 70\%, 80\%\}$ with $m + 1 = 7$. Table 6 displays the results of the test. The reported values are the *p*-values in percentages. Hence, for a significance level of 5%, we reject the null hypothesis of no causality from index *j* to index *i* when the reported value is lower than 5%. *p*-Values corresponding to the rejection of the null hypothesis of no causality are shown in bold. The last column, labeled "Sum," indicates the number of times a given index in a row is Granger-caused by the others. Similarly, the last row labeled "Sum" indicates the number of times a given stock market index Granger-causes other stock market indices. Finally, the entry corresponding to the last row and last column reports the total number of rejections of the

null of no causality for our set of countries. Thus, interdependence (defined as causality in the center of the distribution) is supported in only 9.8% of the cases (13 cases out of 132). This result indicates that European stock market integration is far from being achieved. Among the country results, we observe that the Austrian and French stock markets are the most integrated because they are each affected by three other European markets. By contrast, Greece, Ireland, Italy, Luxembourg, and the Netherlands appear to be independent from the other markets. Interestingly, the causal matrix is not symmetric: France, which is among the most caused markets, does not affect any market. This result supports our choice of causality rather than correlation as a measure of spillover. The most causal markets are the Netherlands, Greece, and Portugal. The identification of these two last countries is interesting because they were among the main drivers of the European crisis. Their causal importance, which can be qualified as systemic for the rest of Europe, should have constituted a signal of alarm at the edge of the crisis.

Table 7. Results of bilateral tests of Granger causality in the left-tail distribution

	AT	BEL	FI	FR	GER	GRE	IE	IT	LU	NL	PT	ES	SUM
AT	100.0	13.2	**4.6**	37.1	92.6	**0.0**	61.1	**0.9**	**0.1**	**0.0**	**0.0**	**1.2**	7
BEL	**0.3**	100.0	23.9	**0.3**	62.9	**0.0**	**0.0**	**0.0**	64.7	**0.0**	**0.0**	10.0	7
FI	76.8	26.8	100.0	71.3	93.5	25.3	41.8	69.8	8.1	21.9	**0.3**	84.3	1
FR	**0.0**	41.0	57.0	100.0	16.2	**2.5**	**2.3**	**0.2**	79.8	34.7	**0.0**	73.1	5
GER	57.5	43.0	94.5	36.9	100.0	43.3	10.4	**0.2**	41.7	44.1	**0.5**	59.1	2
GRE	14.2	**0.0**	**0.0**	16.1	82.7	100.0	45.9	9.9	96.0	**4.4**	**0.0**	68.2	4
IE	60.1	61.3	31.4	5.9	94.1	72.0	100.0	**1.7**	27.8	50.1	20.3	59.8	1
IT	**0.0**	**0.0**	56.4	9.9	35.5	**0.5**	**0.0**	100.0	83.9	**0.0**	**0.0**	41.3	6
LU	5.5	**0.0**	16.0	90.0	88.2	36.5	**0.4**	79.5	100.0	**0.8**	3.8	**1.4**	5
NL	8.5	11.1	**0.0**	83.1	9.9	6.4	**1.3**	14.0	96.6	100.0	**0.0**	64.8	3
PT	74.1	15.5	79.1	**4.5**	46.9	**4.0**	61.4	79.5	28.1	**2.0**	100.0	21.1	3
ES	36.1	38.4	67.7	**0.0**	9.6	82.5	20.3	**0.0**	91.2	15.6	**1.2**	100.0	3
SUM	3	3	3	3	0	5	5	6	1	6	10	2	47

NOTES: Each entry of the table gives the *p*-value (in %) of the test of causality in the left-tail distribution from the index *j* in the column toward the index *i* in the row. Entries with the rejection of the null hypothesis at the 5% significance level are indicated in bold. The last column, labeled "Sum," indicates the number of times a given index in a row is Granger-caused by the others. Similarly, the last row, labeled "Sum," indicates the number of times a given index in column Granger-causes the other indices. The entry corresponding to the last row and the last column gives the total number of significant Granger causalities in the system. The tests are performed over the period ranging from January 1, 2007, to May 6, 2011, with a total of $T = 1134$ observations.

Table 8. Results of bilateral tests of Granger causality in the right-tail distribution

	AT	BEL	FI	FR	GER	GRE	IE	IT	LU	NL	PT	ES	SUM
AT	100.0	7.7	13.5	69.5	13.8	**2.6**	90.1	85.4	84.5	83.5	90.3	34.2	1
BEL	39.2	100.0	**2.7**	68.5	76.7	12.3	10.3	15.5	32.8	58.3	47.9	**3.6**	2
FI	19.5	80.2	100.0	72.7	5.8	8.0	71.4	58.0	16.1	58.2	42.1	**2.1**	1
FR	35.4	28.5	18.7	100.0	17.5	47.6	31.2	42.0	56.1	6.4	31.8	6.4	0
GER	10.1	36.2	5.5	8.7	100.0	56.0	59.3	16.7	94.3	**1.5**	38.0	**4.0**	2
GRE	86.5	11.5	34.8	30.3	14.1	100.0	21.5	62.6	7.5	76.9	85.5	56.4	0
IE	29.2	6.9	47.8	67.5	22.4	10.9	100.0	59.4	82.3	94.7	74.5	85.1	0
IT	29.3	21.8	20.3	47.3	42.9	12.1	21.9	100.0	33.8	14.0	11.6	10.1	0
LU	31.7	90.3	24.9	**2.6**	10.5	73.8	77.4	**1.0**	100.0	10.7	23.5	**4.1**	3
NL	14.1	42.5	28.6	10.7	66.6	64.9	16.4	45.2	91.2	100.0	44.1	36.9	0
PT	62.3	56.5	6.0	47.3	14.8	30.7	**2.7**	99.3	61.7	48.6	100.0	86.2	1
ES	73.8	85.7	13.4	39.2	7.3	59.0	62.4	56.5	31.9	77.7	54.1	100.0	0
SUM	0	0	1	1	0	1	1	1	0	1	0	4	10

NOTES: Each entry of the table gives the p-value (in %) of the test of causality in the right-tail distribution from the index j in a column toward the index i in a row. Entries with the rejection of the null hypothesis at the 5% significance level are indicated in bold. The last column, labeled "Sum," indicates the number of times a given index in a row is Granger-caused by the others. Similarly, the last row, labeled "Sum," indicates the number of times a given index in a column Granger-causes the other indices. The entry corresponding to the last row and the last column gives the total number of significant Granger causalities in the system. The tests are performed over the period ranging from January 1, 2007, to May 6, 2011, with a total of $T = 1134$ observations.

4.3 Testing for Contagion

As explained previously, contagion is apprehended by implementing our Granger causality test in the left-tail distribution. The set A of VaRs risk levels is now set as $A = \{0\%, 1\%, 5\%, 10\%\}$ with $m + 1 = 4$. Table 7 displays the outcomes of the tests. The pairs for which we find a rejection of the null of no causality in the left tail of the distribution amount to 35.6% of the cases (47 rejections over 132 cases). This result is clearly higher than that obtained considering the center of the distribution, hence supporting the presence of contagion. Moreover, we observe that the most causal markets are Portugal, Italy, the Netherlands, Greece, and Ireland, and except for the Netherlands, this group includes all countries in turmoil (Portugal, Italy, Greece, and Ireland) around which the crisis was built. By contrast, the most caused markets are Austria, Belgium, Italy, France, Luxembourg, and Greece. Remark the predominant role in the system of Italy and Greece, which cause and are caused in many cases.

The Granger causality test is now repeated for the right-tail distribution with $A = \{90\%, 95\%, 99\%, 100\%\}$, that is, $m + 1 = 4$. The results are reported in Table 8. Contagion in positive periods is only supported in 7.5% of the cases (10 rejections over 132 cases) and concerns mainly Spain as the driver of spillover and Luxembourg, Germany, and Belgium as spillover receivers. The huge difference in causal links for the right (7.5%) and left tails (35.6%) is striking. Whereas spillovers are important in crisis periods, they are only weakly present during upswing periods. This feature emphasizes the substantial vulnerability of European stock markets to negative shocks. European policy makers should acknowledge this vulnerability and implement structural measures to limit it.

5. CONCLUSION

A kernel-based nonparametric test for Granger causality in distribution between two time series is proposed in this article.

The test checks for spillovers between the multivariate processes of dynamic interquantile event variables are associated with each variable. Our testing approach has two main advantages over existing approaches. First, it can be used to test for Granger causality in specific regions of the distributions, such as the center or the tails (left and right). Second, it checks for a large number of lags by discounting higher-order lags and hence is consistent against causality, which carries over long distributional lags.

We demonstrate that the test has a standard Gaussian distribution under the null hypothesis, which is free of parameter estimation uncertainty. A Monte Carlo simulation exercise revealed that the Gaussian distribution is valid in small samples. The test also has very appealing power properties in various settings, including linear and nonlinear causality in mean and causality in variance.

In the empirical section, we implement our testing procedure for 12 European daily stock market indices to analyze spillover during the recent European crisis. Because our test is designed to check for causality in specific regions of the distribution (center or tails), it can be used to test for the presence of interdependence as well as contagion. Indeed, interdependence can be determined through Granger causality in the center of the distribution because interdependence is a long-term path that occurs during normal periods. By contrast, contagion refers to a short-term, abrupt increase in the causal linkages occurring exclusively during crisis periods and can be tested via the Granger causality in the distribution's tails.

The empirical results indicate that European stock market integration is far from achieved because we observe few cases of an interdependent pairwise relationship. By contrast, our results support the presence of contagion, with a strong difference between contagion in the right and left tails. More precisely, contagion is frequent among countries during crisis periods and comparatively infrequent during upswing periods. This result reveals an important feature of the European stock markets, and policy makers should acknowledge

this feature in designing structural measures for financial stability.

APPENDIX A: PROOF OF LEMMAS

A.1 Proof of Lemma 2

Lemma 2. Under the null hypothesis of no Granger causality in distribution and the assumptions of Theorem 1 in Hong, Liu, and Wang (2009), we have

$$\frac{\mathcal{T}^* - m^2 C_T(M)}{\left(m^2 D_T(M)\right)^{1/2}} \longrightarrow^d \mathcal{N}(0, 1).$$

Proof. Consider the following decomposition

$$\frac{\mathcal{T}^* - m^2 C_T(M)}{\left(m^2 D_T(M)\right)^{1/2}} = \frac{\mathcal{T} - m^2 C_T(M)}{\left(m^2 D_T(M)\right)^{1/2}} + \frac{\mathcal{T}^* - \mathcal{T}}{\left(m^2 D_T(M)\right)^{1/2}},$$

where $\mathcal{T}$ is the test statistic involving the cross-correlation matrix $R(j)$ of the true processes of event variables $H_t^X(\theta_X^0)$ and $H_t^Y(\theta_Y^0)$ and their respective correlation matrices Γ_X and Γ_Y, that is,

$$\mathcal{T} = \sum_{j=1}^{T-1} \kappa^2(j/M) Q(j),$$

$$Q(j) = T \text{vec}(R(j))^T \left(\Gamma_X^{-1} \otimes \Gamma_Y^{-1}\right) \text{vec}(R(j)).$$

With this decomposition, the proof of Lemma 2 is given by the following two Lemmas:

Lemma 2.1. Under the assumptions of Theorem 1 in Hong, Liu, and Wang (2009), we have

$$\mathcal{T}^* - \mathcal{T} = o_p\left(M^{1/2}\right).$$

Lemma 2.2. Under the null hypothesis of no Granger causality in distribution, we have

$$\frac{\mathcal{T} - m^2 C_T(M)}{\left(m^2 D_T(M)\right)^{1/2}} \longrightarrow^d \mathcal{N}(0, 1).$$

A.1.1 Proof of Lemma 2.1. The following notations are required. Let $b_t^{(1)} = \Sigma_X^{-1/2}\left(H_t^X(\theta_X^0) - \Pi_X\right)$ and $b_t^{(2)} = \Sigma_Y^{-1/2}\left(H_t^Y(\theta_Y^0) - \Pi_Y\right)$, be the centered and scaled multivariate processes of event variables, with $\Pi_X = \mathbb{E}\left(H_t^X(\theta_X^0)\right)$ and $\Pi_Y = \mathbb{E}\left(H_t^Y(\theta_Y^0)\right)$, Σ_X and Σ_Y being the covariance matrices of $H_t^X(\theta_X^0)$ and $H_t^Y(\theta_Y^0)$, respectively. We denote $\widehat{b}_t^{(1)} = \Sigma_X^{-1/2}\left(\widehat{H}_t^X - \Pi_X\right)$ and $\widehat{b}_t^{(2)} = \Sigma_Y^{-1/2}\left(\widehat{H}_t^Y - \Pi_Y\right)$ their analogs based on the estimated processes of multivariate event variables $\widehat{H}_t^X$ and $\widehat{H}_t^Y$. We denote $C_{\widehat{b}}(j)$ the sample cross-covariance matrix at lag-order j between $\widehat{b}_t^{(1)}$ and $\widehat{b}_t^{(2)}$, with $C_{\widehat{b}}(j) = \Sigma_X^{-1/2}\widehat{\Lambda}(j)\Sigma_Y^{-1/2}$, and $\widehat{\Lambda}(j)$ is the cross-covariance matrix at lag-order j between $\widehat{H}_t^X$ and $\widehat{H}_t^Y$. Finally, $C_b(j)$ is the pseudo version of $C_{\widehat{b}}(j)$, with $C_b(j) = \Sigma_X^{-1/2}\Lambda(j)\Sigma_Y^{-1/2}$, and $\Lambda(j)$ is the cross-covariance matrix at lag-order j between $H_t^X(\theta_X^0)$ and $H_t^Y(\theta_Y^0)$.

With these notations and based on Bouhaddioui and Roy (2006, proof of Lemma 2, pp. 538), $\mathcal{T} - \mathcal{T}^*$ can be rewritten as $\mathcal{T} - \mathcal{T}^* = A_T^{(1)} + A_T^{(2)}$, with

$$A_T^{(1)} = T \sum_{j=1}^{T-1} \kappa^2(j/M) \|\text{vec}(C_{\widehat{b}}(j)) - \text{vec}(C_b(j))\|^2,$$

$$A_T^{(2)} = 2T \sum_{j=1}^{T-1} \kappa^2(j/M) \langle \text{vec}(C_b(j)), \text{vec}(C_{\widehat{b}}(j)) - \text{vec}(C_b(j))\rangle,$$

where $\|x\|$ is the Euclidean norm and $\langle x, y \rangle = x^T y$ is the inner product. Hence, the rest of the proof proceeds by showing that $A_T^{(1)} = o_p\left(M^{1/2}\right)$ and $A_T^{(2)} = o_p\left(M^{1/2}\right)$.

For the first term $A_T^{(1)}$, using the property $\text{vec}(ABC) = \left(C^T \otimes A\right)\text{vec}(B)$, we have

$$A_T^{(1)} = T \sum_{j=1}^{T-1} \kappa^2(j/M) \left\|\left(\Sigma_Y^{-1/2} \otimes \Sigma_X^{-1/2}\right)\left(\text{vec}(\widehat{\Lambda}(j)) - \text{vec}(\Lambda(j))\right)\right\|^2,$$

and we obtain

$$A_T^{(1)} \le T \sum_{j=1}^{T-1} \kappa^2(j/M) \left\|\Sigma_Y^{-1/2} \otimes \Sigma_X^{-1/2}\right\|_F^2 \left\|\text{vec}(\widehat{\Lambda}(j)) - \text{vec}(\Lambda(j))\right\|^2,$$

with $\|A\|_F^2 = \text{trace}(A^T A)$ the square of the Frobenius norm of the matrix A. For the rest of the proof, it suffices to show that $L_T^{(1)} = o_p\left(M^{1/2}/T\right)$, with

$$L_T^{(1)} = \sum_{j=1}^{T-1} \kappa^2(j/M) \left\|\text{vec}(\widehat{\Lambda}(j)) - \text{vec}(\Lambda(j))\right\|^2.$$

Note that we have

$$L_T^{(1)} = \sum_{u,v=1}^{m,m} \sum_{j=1}^{T-1} \kappa^2(j/M)\left(\widehat{C}_{u,v}(j) - C_{u,v}(j)\right)^2,$$

where $\widehat{C}_{u,v}(j)$ and $C_{u,v}(j)$ are the (u, v) elements of the matrices $\widehat{\Lambda}(j)$ and $\Lambda(j)$, respectively. Hence, using the result of Proposition A.1. in Hong, Liu, and Wang (2009, pp. 282), that is, $\sum_{j=1}^{T-1} \kappa^2(j/M)\left(\widehat{C}_{u,v}(j) - C_{u,v}(j)\right)^2 = o_p\left(M^{1/2}/T\right)$, we conclude that $L_T^{(1)} = o_p\left(M^{1/2}/T\right)$, and $A_T^{(1)} = o_p\left(M^{1/2}\right)$.

For the second term $A_T^{(2)}$, we have

$$A_T^{(2)} = 2T \sum_{j=1}^{T-1} \kappa^2(j/M)\left\{\text{vec}(C_b(j))^T\left(\text{vec}(C_{\widehat{b}}(j)) - \text{vec}(C_b(j))\right)\right\},$$

and using once again the property $\text{vec}(ABC) = \left(C^T \otimes A\right)\text{vec}(B)$, and the property $(A \otimes B)(C \otimes D) = (AC) \otimes (BD)$, simple calculus give

$$A_T^{(2)} = 2T \sum_{j=1}^{T-1} \kappa^2(j/M)\left\{\text{vec}(\Lambda(j))^T P\left(\text{vec}(\widehat{\Lambda}(j)) - \text{vec}(\Lambda(j))\right)\right\},$$

with $P = \Sigma_Y^{-1} \otimes \Sigma_X^{-1}$. Let $\widehat{C}_s(j)$, $s = 1, \ldots, m^2$ be the elements of $\text{vec}(\widehat{\Lambda}(j))$, and $C_s(j)$, $s = 1, \ldots, m^2$ the elements of $\text{vec}(\Lambda(j))$. Then $A_T^{(2)}$ is equal to

$$A_T^{(2)} = 2T \sum_{j=1}^{T-1} \kappa^2(j/M)\left\{\sum_{s,r=1}^{m^2,m^2} C_s(j)\left(\widehat{C}_r(j) - C_r(j)\right) P_{sr}\right\},$$

where P_{sr} is the (s, r) element of the matrix P, and we have

$$A_T^{(2)} = 2T \sum_{s,r=1}^{m^2,m^2} P_{sr} \sum_{j=1}^{T-1} \kappa^2(j/M)\left\{C_s(j)\left(\widehat{C}_r(j) - C_r(j)\right)\right\}.$$

Based on the result of Proposition A.2. in Hong, Liu, and Wang (2009, pp. 282), that is,

$$\sum_{j=1}^{T-1} \kappa^2(j/M)\left\{C_s(j)\left(\widehat{C}_r(j) - C_r(j)\right)\right\} = o_p\left(M^{1/2}/T\right), \quad \text{(A.1)}$$

we conclude that $A_T^{(2)} = o_p\left(M^{1/2}\right)$, and this completes the proof of Lemma 2.1.

Note that Proposition A.2. in Hong, Liu, and Wang (2009) is about the following quadratic form $\sum_{j=1}^{T-1} \kappa^2(j/M)\left\{C_r(j)\left(\widehat{C}_r(j) - C_r(j)\right)\right\}$, in which the expression in the brace involves the product of two terms that have the same index. This differs from our expression in (A.1) with the two terms of

the product having different indices. However, by going through their proof of Proposition A.2., results do not change considering different indices.

A.1.2 Proof of Lemma 2.2. The result of Lemma 2.2. is obtained from a simple modification of the proof of Lemma 1 in Bouhaddioui and Roy (2006, pp. 529), putting $b_t = (b_t^{(1)}, b_t^{(2)})^T$, with $b_t^{(1)} \equiv \Sigma_X^{-1/2}(H_t^X(\theta_X^0) - \Pi_X), b_t^{(2)} \equiv \Sigma_Y^{-1/2}(H_t^Y(\theta_Y^0) - \Pi_Y)$. Note that the asymptotic normality result of Lemma 1 in Bouhaddioui and Roy (2006) is obtained under two assumptions: (i), $b_t^{(1)}$ and $b_t^{(2)}$ are multivariate iid sequences, respectively, (ii) and the two processes are independent. In our framework, the assumption (i) is satisfied because $b_t^{(1)}$ and $b_t^{(2)}$ are the centered and scaled versions of the true multivariate processes of interquantile event variables $H_t^X(\theta_X^0)$ and $H_t^Y(\theta_Y^0)$, and both $H_t^X(\theta_X^0)$ and $H_t^Y(\theta_Y^0)$ are multivariate iid sequence. Indeed, the m components of $H_t^X(\theta_X^0)$ (or $H_t^Y(\theta_Y^0)$) although cross-sectionally dependent, do not have lag-dependence between them. Note that the absence of lag-dependence is the usual hypothesis that is checked when validating value-at-risk models through backtesting procedures. Finally, for the second assumption (ii), we do not need here the full assumption of independence between $b_t^{(1)}$ and $b_t^{(2)}$, but only that $b_t^{(1)}$ is independent of $\{b_s^{(2)}, s < t\}$, which is satisfied under our null hypothesis of no Granger causality from Y_t to X_t. This supposes that we must allow here for the reverse possibility that X_t Granger-causes Y_t. However, the normality result of our Lemma 2.2. remains valid, if by going through their proof of Lemma 1, we consider only positive lags ($j > 0$), excluding the cases $j \leq 0$, which include instantaneous causality and causality from X_t to Y_t.

A.2 Proof of Lemma 3

Lemma 3. Under the assumptions of Theorem 1 in Hong, Liu, and Wang (2009), we have

$$\frac{\widehat{\mathcal{T}} - \mathcal{T}^*}{\left(m^2 D_T(M)\right)^{1/2}} \xrightarrow{p} 0.$$

Proof. The proof of Lemma 3 proceeds by combining elements in the proofs of Proposition 3 in Bouhaddioui and Roy (2006), Theorems A.1. and A.3. in Hong, Liu, and Wang (2009). Formally, given that

$$D_T(M) = M \int_0^\infty \kappa^4(z)\,dz\,[1 + o(1)],$$

as $M \to \infty$, the proof of Lemma 3 can be established by showing that $\widehat{\mathcal{T}} - \mathcal{T}^* = O_p(M/T^{1/2})$. Based on Lemma 4.1 in El Himdi and Roy (1997), the quadratic forms $\widehat{\mathcal{T}}$ and $\mathcal{T}^*$ can be rewritten in term of cross-covariances as

$$\widehat{\mathcal{T}} = T \sum_{j=1}^{T-1} \kappa^2(j/M) \mathrm{vec}\left(\widehat{\Lambda}(j)\right)^T \left(\widehat{\Sigma}_X^{-1} \otimes \widehat{\Sigma}_Y^{-1}\right) \mathrm{vec}\left(\widehat{\Lambda}(j)\right),$$

$$\mathcal{T}^* = T \sum_{j=1}^{T-1} \kappa^2(j/M) \mathrm{vec}\left(\widehat{\Lambda}(j)\right)^T \left(\Sigma_X^{-1} \otimes \Sigma_Y^{-1}\right) \mathrm{vec}\left(\widehat{\Lambda}(j)\right),$$

where $\widehat{\Lambda}(j)$ is the sample cross-covariance matrix at lag-order j, Σ_X and Σ_Y are the covariance matrices of the true multivariate processes of event variables $H_t^X(\theta_X^0)$ and $H_t^Y(\theta_Y^0)$, and $\widehat{\Sigma}_X$ and $\widehat{\Sigma}_Y$ are their sample counterparts given by the covariance matrices of $\widehat{H}_t^X \equiv H_t^X(\widehat{\theta}_X)$ and $\widehat{H}_t^Y \equiv H_t^Y(\widehat{\theta}_Y)$, respectively. It follows that

$$\widehat{\mathcal{T}} - \mathcal{T}^* = T \sum_{j=1}^{T-1} \kappa^2(j/M) \mathrm{vec}\left(\widehat{\Lambda}(j)\right)^T \left\{\widehat{\Sigma}_X^{-1} \otimes \widehat{\Sigma}_Y^{-1} - \Sigma_X^{-1} \otimes \Sigma_Y^{-1}\right\}$$

$$\mathrm{vec}\left(\widehat{\Lambda}(j)\right). \tag{A.2}$$

Now, let us study the asymptotic behavior of $\widehat{\Sigma}_X$. The components of this matrix are given by the covariance between the estimated event variables $\widehat{Z}_{t,k}^X, k = 1, \ldots, m$. Let $\widehat{C}_{k,p}$ be a typical element of $\widehat{\Sigma}_X$ with $\widehat{C}_{k,p} = \mathrm{cov}\left(\widehat{Z}_{t,k}^X, \widehat{Z}_{t,p}^X\right)$. Let $C_{k,p}^0$ be the true value of $\widehat{C}_{k,p}$, that is, the covariance between the true event variables $Z_{t,k}^X(\theta_X^0)$ and $Z_{t,p}^X(\theta_X^0)$. Note that $C_{k,p}^0$ is a typical element of Σ_X. The difference between $\widehat{C}_{k,p}$ and $C_{k,p}^0$ can be decomposed as follows:

$$\widehat{C}_{k,p} - C_{k,p}^0 = \widehat{M}_1\left(\widehat{\theta}_X^0\right) + \widehat{M}_2\left(\widehat{\theta}_X^0\right) + \widehat{M}_3\left(\widehat{\theta}_X^0\right),$$

with

$$\widehat{M}_1\left(\widehat{\theta}_X^0\right) = T^{-1} \sum_{t=1}^{T} \left[\widehat{Z}_{t,k}^X - Z_{t,k}^X(\theta_X^0)\right]\left[Z_{t,p}^X(\theta_X^0) - \pi_p^X\right]$$

$$\widehat{M}_2\left(\widehat{\theta}_X^0\right) = T^{-1} \sum_{t=1}^{T} \left[Z_{t,k}^X(\theta_X^0) - \pi_k^X\right]\left[\widehat{Z}_{t,p}^X - Z_{t,p}^X(\theta_X^0)\right]$$

$$\widehat{M}_3\left(\widehat{\theta}_X^0\right) = T^{-1} \sum_{t=1}^{T} \left[\widehat{Z}_{t,k}^X - Z_{t,k}^X(\theta_X^0)\right]\left[\widehat{Z}_{t,p}^X - Z_{t,p}^X(\theta_X^0)\right],$$

where we replace the sample means $\widehat{\pi}_k^X$ and $\widehat{\pi}_p^X$ of $\widehat{Z}_{t,k}$ and $\widehat{Z}_{t,p}$ by their true respective values $\pi_k^X = \mathbb{E}\left(Z_{t,k}^X(\theta_X^0)\right)$ and $\pi_p^X = \mathbb{E}\left(Z_{t,p}^X(\theta_X^0)\right)$. Using the following result in the proof of Theorem A.3. in Hong, Liu, and Wang (2009, pp. 286), that is, $\sup_{\theta_X \in \Theta_X} \left|\widehat{M}_1(\theta_X)\right| = O_p\left(T^{-1/2}\right)$, where θ_X is any $\sqrt{T}$-consistent estimator of θ_X^0 in the space Θ_X, we have for the first term $\widehat{M}_1\left(\widehat{\theta}_X^0\right) = O_p\left(T^{-1/2}\right)$. Similar arguments apply for the last two terms, with the consequence that $\widehat{M}_2\left(\widehat{\theta}_X^0\right) = O_p\left(T^{-1/2}\right)$, $\widehat{M}_3\left(\widehat{\theta}_X^0\right) = O_p\left(T^{-1/2}\right)$. We deduce that $\widehat{C}_{k,p} - C_{k,p}^0 = O_p\left(T^{-1/2}\right)$ and $\widehat{\Sigma}_X - \Sigma_X = O_p\left(T^{-1/2}\right)$.

Using the same reasoning for the elements of $\widehat{\Sigma}_Y$, we have $\widehat{\Sigma}_Y - \Sigma_Y = O_p\left(T^{-1/2}\right)$ and

$$\widehat{\Sigma}_X^{-1} \otimes \widehat{\Sigma}_Y^{-1} - \Sigma_X^{-1} \otimes \Sigma_Y^{-1} = O_p\left(T^{-1/2}\right).$$

Hence Equation (A.2) becomes

$$\widehat{\mathcal{T}} - \mathcal{T}^* = T \sum_{j=1}^{T-1} \kappa^2(j/M) \mathrm{vec}\left(\widehat{\Lambda}(j)\right)^T O_p\left(T^{-1/2}\right) \mathrm{vec}\left(\widehat{\Lambda}(j)\right) \tag{A.3}$$

$$= O_p\left(T^{1/2}\right) \sum_{j=1}^{T-1} \kappa^2(j/M) \mathrm{vec}\left(\widehat{\Lambda}(j)\right)^T \mathrm{vec}\left(\widehat{\Lambda}(j)\right).$$

The rest of the proof proceeds by showing that

$$\mathcal{B}(T) = \sum_{j=1}^{T-1} \kappa^2(j/M) \mathrm{vec}\left(\widehat{\Lambda}(j)\right)^T \mathrm{vec}\left(\widehat{\Lambda}(j)\right) = O_p(M/T).$$

We decompose $\mathcal{B}(T)$ into two parts: $\mathcal{B}(T) = \mathcal{B}_1(T) + \mathcal{B}_2(T)$, with

$$\mathcal{B}_1(T) = \sum_{j=1}^{T-1} \kappa^2(j/M) \left\{\mathrm{vec}\left(\widehat{\Lambda}(j)\right)^T \mathrm{vec}\left(\widehat{\Lambda}(j)\right) - \mathrm{vec}(\Lambda(j))^T\right.$$

$$\left. \mathrm{vec}(\Lambda(j))\right\},$$

$$\mathcal{B}_2(T) = \sum_{j=1}^{T-1} \kappa^2(j/M) \mathrm{vec}(\Lambda(j))^T \mathrm{vec}(\Lambda(j)),$$

where $\Lambda(j)$ is the cross-covariance matrix at lag-order j of the true event variables $H_t^X(\theta_X^0)$ and $H_t^Y(\theta_Y^0)$. Let us first consider $\mathcal{B}_1(T)$. We have

$$\mathcal{B}_1(T) = \sum_{j=1}^{T-1} \kappa^2\left(\frac{j}{M}\right) \sum_{u,v=1}^{m,m} \left\{\widehat{C}_{u,v}^2(j) - C_{u,v}^2(j)\right\},$$

with $\widehat{C}_{u,v}$ and $C_{u,v}$ the (u, v) element of the matrices $\widehat{\Lambda}(j)$ and $\Lambda(j)$, respectively. Using the result of Theorem A.1. in Hong, Liu, and

Wang (2009, pp. 282), that is, $\sum_{j=1}^{T-1} \kappa^2(j/M)\left(\widehat{C}_{u,v}^2(j) - C_{u,v}^2(j)\right) = o_p\left(M^{1/2}/T\right)$, we obtain $\mathcal{B}_1(T) = O_p\left(M^{1/2}/T\right)$.

For the second term $\mathcal{B}_2(T)$, using the Markov inequality, we have

$$\mathcal{B}_2(T) = \sum_{j=1}^{T-1} \kappa^2\left(\frac{j}{M}\right) \text{vec}(\Lambda(j))^T \text{vec}(\Lambda(j)) = O_p(M/T).$$

(A.4)

We deduce that $\mathcal{B}(T) = \mathcal{B}_1(T) + \mathcal{B}_2(T) = O_p(M/T)$ and $\widehat{\mathcal{T}} - \mathcal{T}^* = O_p\left(T^{1/2}\right) O_p(M/T) = O_p\left(M/T^{1/2}\right)$. This completes the proof of Lemma 3.

APPENDIX B: DESCRIPTION OF THE KERNEL FUNCTIONS

The four usual kernels are defined as follows:

- the Daniell (DAN) kernel

$$\kappa(z) = \frac{\sin(\pi z)}{\pi z}, \quad z \in \mathbb{R},$$

(B.1)

- the Parzen (PAR) kernel

$$\kappa(z) = \begin{cases} 1 - 6z^2 + 6|z|^3 & \text{if} & |z| \le 0.5 \\ 2(1 - |z|)^3 & \text{if} & 0.5 < |z| \le 1 \\ 0 & \text{otherwise,} \end{cases}$$

(B.2)

- the Bartlett (BAR) kernel

$$\kappa(z) = \begin{cases} 1 - |z| & \text{if} & |z| \le 1 \\ 0 & \text{otherwise,} \end{cases}$$

(B.3)

- the truncated uniform (TR) kernel

$$\kappa(z) = \begin{cases} 1 & \text{if} & |z| \le 1 \\ 0 & \text{otherwise.} \end{cases}$$

(B.4)

ACKNOWLEDGMENTS

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