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A Nonparametric Test for Granger-causality in Distribution with Application to Financial Contagion☆

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Abstract

This paper 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 introduced by Hong et al. (2009) and hence shares its main advantage, by checking a large number of lags with higher order lags discounted. Besides, our test is highly flexible as it can be used to check for Granger-causality in specific regions on the distribution supports, like the center or the tails. We prove that it converges asymptotically to a standard Gaussian distribution under the null hypothesis and thus it 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 for a set of European stock markets in order to analyse the spill-overs during the recent European crisis and to distinguish contagion from interdependence effects.

Keywords: Granger-causality, Distribution, Tails, Kernel-based test, Financial Spill-over.

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1. Introduction

Analysis of causal relationships holds an important part of the 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 in testing transmission effects between the whole distribution of random variables, recent literature has proposed some weak versions of this concept, as the causality in the frequency domain or for specific distribution moments. For instance, Granger-causality in mean (Granger, 1980, 1988) is widely used in macroeconomics.[1] Granger et al. (1986) also introduce the concept of Granger-causality in variance to test for causal effects in the second order moment between financial series.[2] A unified treatment of Granger-causality in the mean and the variance is formalized by Comte and Lieberman (2000).

More recently, some contributions have focused on the concept of Granger-causality in quantiles, an issue which is particularly important for non-Gaussian distributions that exhibit asymmetry, fat-tail characteristics and non-linearity (Lee and Yang, 2012; Jeong et al., 2012). Indeed, given these distributions, the dynamic in the tails can be rather different from the one in the center of the distribution. In this case, the information content of quantiles gives more insights on the distribution than the mean. Lee and Yang (2012) developed a parametric methodology for Granger-causality in quantiles which is based on the conditional predictive ability (CPA) framework of Giacomini and White (2006). Jeong et al. (2012) introduce a non-parametric approach to test for causality in quantiles and apply it to the detection of causal relations between the crude oil price, the USD/GBP exchange rate, and the gold price. A closely related but different concept is the Granger-causality in tail-event by Hong et al. (2009), a tail-event being identified as a situation where the value of a time

[1] See inter alii Sims (1972, 1980) who tests for Granger-causality in mean between money and income.

[2] This concept is further explored by Cheung and Ng (1996), Kanas and Kouretas (2002), Hafner and Herwartz (2004), to cite but a few.

series is lower than its Value-at-Risk at a specified risk level. Hence the test checks whether an extreme downside movement for a given time series has a predictive content for an extreme downside movement for another time series, and has many potential applications in risk management.

All the tests of causality in quantiles and tail-events share the same limit that statistical inference is exclusively performed at a particular 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), time series behavior of quantiles can vary considerably across the distribution because of long memory or non-stationarity. Hence, a Granger-causality test in quantiles or tail-events which does not consider a large number of quantiles simultaneously over the distribution support would be restrictive. Given that 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 distribution.

Very few papers developed testing procedures for Granger-causality in the whole distribution in a time series context. The only exceptions to our knowledge include Su and White (2007,2008,2012,2013), Bouezmarni et al. (2012) and Taamouti et al. (2012). For example, Su and White (2012) introduce a conditional independence specification test which can be used to test for Granger-causality in quantiles for a continuum values of quantile levels between $(0, 1)$. Bouezmarni et al. (2012) construct a nonparametric Granger-causality test in distribution based on conditional independence in the framework of copulas.[3] Our paper adds to this literature proposing a new methodology to test for Granger-causality in the whole distribution between two time series. Our testing procedure consists in dividing the distribution support of each series into a multivariate process of dynamic inter-quantile event variables, and by checking whether there is a spill-over effect between the two multivariate pro-

[3]See also Taamouti et al. (2012) for another approach from the copulas theory.

cesses, analyzing their cross-correlations structure. The test draws from the generalized portmanteau test for independence between multivariate processes in Bouhaddioui and Roy (2006).

It is worth mentioning that although our approach checks for the strong version of the Granger-causality concept (Granger, 1969), it is highly flexible as it can be used to test for causality in specific regions on the distribution supports, like the center or the tails (left or right).[4] 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 inter-quantile event variables should be defined so as to focus the analysis exclusively on this part of the distribution. This flexibility is one of the great advantage of our methodology compared to those based on copulas theory (Bouezmarni et al., 2012; Taamouti et al., 2012). It allows us to go beyond the simple rejection of the null hypothesis of Granger-causality for the whole distribution, as it provides us with the specific regions for which Granger-causality is rejected. Besides, our test statistic is a multivariate extension of the kernel-based nonparametric Granger-causality test in tail-event by Hong et al. (2009), and hence shares its main advantage: it checks for a large number of lags by discounting higher order lags. This characteristic is consistent with the stylized fact in empirical finance that recent events have much more influence in the current market trends than those older. In this line, our Granger-causality test in distribution is different from those available in the literature which check for causality uniformly for a limited number of lags.

Technically, we show that the test has a standard Gaussian distribution under the null hypothesis which is free of parameter estimation uncertainty. Monte Carlo simulations reveal indeed that the Gaussian distribution provides a good approximation of the distribution of our test statistic, even in small samples. Moreover, the test has power to reject the null hypothesis of causality in distribution stemming from different sources including linear and non-linear

[4]Note that Candelon et al. (2013) introduce a parametric test to check for Granger-causality in distribution tails, but the methodology does not apply for other regions of the distribution like the center.

causality in mean and causality in variance.

To illustrate the importance of this test for the empirical literature, we use it to better understand the spill-overs that have taken place within European stock markets during the recent crisis. Our Granger-causality test in distribution allows to consider asymmetry between markets (which is not possible using correlation), to take into account for break in volatility (as suggested by Forbes and Rigobon, 2002) and to distinguish between contagion and interdependence. Indeed, interdependence is a long run path and taking place in "normal periods" concerning hence the center of the distribution. On the contrary, contagion is detected by a short-run abrupt increase in the causal linkages taking place exclusively during crisis' period, i.e., in the tails of the distribution. As 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 on our results, we find weak (resp. strong) support for interdependence (resp. contagion) during the recent crisis. Interestingly, we observe a strong asymmetry between causal tests in the right and left tails: Whereas spill-overs are important in crisis periods, they are only weakly present in upswing times. Such a result constitutes an important feature for the European stock markets.

The paper is sketched as follows: the second Section presents the Granger-causality test in distribution. The properties of this test are analysed in Section 3 via a Monte-Carlo simulation experiment. Section 4 proposes the empirical application whereas 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. As this test is a multivariate extension of the Granger-causality test in tail-event introduced by Hong et al. (2009), we begin with the presentation of Hong et al. (2009) 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-event developed by Hong et al. (2009) checks whether an extreme downside risk from Y_t can be considered as a lagged indicator for an extreme downside risk for X_t. Hong et al. (2009) identify an extreme downside risk as a situation where X_t and Y_t are lower than their respective Value-at-Risk (VaR) at a prespecified level α. Recall that 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 portfolio of instruments is the maximum dollar loss within the α%-confidence interval (Jorion, 2007). For the two time series X_t and Y_t, we have

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

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

with $VaR_t^X\left(\theta_X^0\right)$ and $VaR_t^Y\left(\theta_Y^0\right)$ the VaR of X_t and Y_t respectively at time t, θ_X^0 and θ_Y^0 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

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

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

In the framework of Hong et al. (2009), an extreme downside risk occurs at time t for X_t, if the tail-event variable $Z_t^X\left(\theta_X^0\right)$ is equal to one, with

$$Z_t^X\left(\theta_X^0\right) = \begin{cases} 1 & \text{if} \qquad X_t < VaR_t^X\left(\theta_X^0\right) \\ 0 & \text{else.} \end{cases} \tag{5}$$

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

$$Z_t^Y\left(\theta_Y^0\right) = \begin{cases} 1 & \text{if} \qquad Y_t < VaR_t^Y\left(\theta_Y^0\right) \\ 0 & \text{else.} \end{cases} \tag{6}$$

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

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

with

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

Under the null hypothesis and at the risk level α, it means that spill-overs of extreme downside movements from Y_t to X_t do not exist. Hong et al. (2009) propose 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 $\left\{\widehat{Z}_t^X, \widehat{Z}_t^Y\right\}$, with components

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

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

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

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

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

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

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

with $\kappa(.)$ a given kernel function and M the truncation parameter. The truncation parameter M is 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 $[-1, 1]$, 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-event 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 suggests using the distance between the two estimators $\widehat{f}(\omega)$ and $\widehat{f}_1^0(\omega)$ to test for the null hypothesis. Hong et al. (2009) consider 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 $D_T(M)$ 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 et al. (2009) which helps checking for 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 $A = \{\alpha_1, ..., \alpha_{m+1}\}$ of $m+1$ VaR risk levels which covers the distribution support of both variables X_t and Y_t, with $0\% <= \alpha_1 < ... < \alpha_{m+1} <= 100\%$. For the first time series X_t, the corresponding VaRs at time t are $VaR^X_{t,s}\left(\theta^0_X, \alpha_s\right)$, $s = 1, ..., m+1$, with

$$VaR^X_{t,1}\left(\theta^0_X, \alpha_1\right) < .... < VaR^X_{t,m+1}\left(\theta^0_X, \alpha_{m+1}\right), \tag{21}$$

where the vector θ^0_X 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 $VaR^X_{t,s}\left(\theta^0_X, \alpha_s\right) = -\infty$ for $\alpha_s = 0\%$ and $VaR^X_{t,s}\left(\theta^0_X, \alpha_s\right) = \infty$ for $\alpha_s = 100\%$.

We divide the distribution support of X_t into m disjoint regions, each related to the indicator or event variable

$$Z^X_{t,s}\left(\theta^0_X\right) = \begin{cases} 1 & \text{if} \qquad X_t \geq VaR^X_{t,s}\left(\theta^0_X, \alpha_s\right) \text{ and } X_t < VaR^X_{t,s+1}\left(\theta^0_X, \alpha_{s+1}\right) \\ 0 & \text{else,} \end{cases} \tag{22}$$

for $s = 1, ..., m$. 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 B.1 displays the support of X_t, along with the VaRs and the event variables defining the $m = 4$ distinct regions.[5]

Now, let $H^X_t\left(\theta^0_X\right)$ be the vector of dimension $(m, 1)$ with components the m event variables

$$H^X_t\left(\theta^0_X\right) = \left(Z^X_{t,1}\left(\theta^0_X\right), Z^X_{t,2}\left(\theta^0_X\right), ..., Z^X_{t,m}\left(\theta^0_X\right)\right). \tag{23}$$

[5]Remark that we do not consider the event variable corresponding to the extreme $m+1$ region identified by $X_t \geq VaR^X_{t,m+1}\left(\theta^0_X, \alpha_{m+1}\right)$. Indeed this variable is implicitly defined by the first m event variables.

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

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

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

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

Therefore, Granger-causality in distribution from Y_t to X_t corresponds to Granger-causality in mean from $H_t^Y\left(\theta_Y^0\right)$ to $H_t^X\left(\theta_X^0\right)$. When the null hypothesis of non causality in distribution holds, this means that 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.

Remark that our null hypothesis is flexible enough as it can be used to check for Granger-causality in specific regions on the distribution supports, like the center or the tails (left or right). This can be done by restricting the set $A = \{\alpha_1, ..., \alpha_{m+1}\}$ of VaR levels to some 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, as it suggests the existence of spill-over effects from Y_t to X_t that take place in the lower tail. Similarly Granger-causality in the center of the distribution can be checked by setting for example A to $A = \{20\%, 40\%, 60\%, 80\%\}$. In the next subsection, we construct a nonparametric kernel-based test statistic to test for our general null hypothesis in (25), and analyze its asymptotic distribution.

2.2.2. Test statistic and asymptotic distribution

Bouhaddioui and Roy (2006) introduce a generalized portmanteau test for the independence between two infinite order vector auto-regressive (VAR) series. Our test statistic relies for (25) on their work. However, the asymptotic analysis

differs because (i) we are not in a VAR framework, (ii) and the event variables $Z_{t,s}^{X}\left(\theta_{X}^{0}\right)$ and $Z_{t,s}^{Y}\left(\theta_{Y}^{0}\right)$ are indicator variables which are not differentiable with respect to the unknown parameters θ_{X}^{0} and θ_{Y}^{0}, respectively. The latter challenge is solved relying on some asymptotic results in Hong et al. (2009).

To present the test statistic, let $\widehat{H}_{t}^{X} \equiv H_{t}^{X}\left(\widehat{\theta}_{X}\right)$ and $\widehat{H}_{t}^{Y} \equiv H_{t}^{Y}\left(\widehat{\theta}_{Y}\right)$ be the estimated counterparts of the multivariate processes of event variables $H_{t}^{X}\left(\theta_{X}^{0}\right)$ and $H_{t}^{Y}\left(\theta_{Y}^{0}\right)$, respectively, with $\widehat{\theta}_{X}$ and $\widehat{\theta}_{Y}$ $\sqrt{T}$ consistent estimators of the true unknown parameter vectors θ_{X}^{0} and θ_{Y}^{0}. Denote $\widehat{\Lambda}\left(j\right)$ the sample cross-covariance matrix between $\widehat{H}_{t}^{X}$ and $\widehat{H}_{t}^{Y}$, with

$$\widehat{\Lambda}\left(j\right) \equiv \begin{cases} T^{-1}\sum\limits_{t=1+j}^{T}\left(\widehat{H}_{t}^{X}-\widehat{A}_{X}\right)\left(\widehat{H}_{t-j}^{Y}-\widehat{A}_{Y}\right)^{T} & 0 \leq j \leq T-1 \\ \\ T^{-1}\sum\limits_{t=1-j}^{T}\left(\widehat{H}_{t+j}^{X}-\widehat{A}_{X}\right)\left(\widehat{H}_{t}^{Y}-\widehat{A}_{Y}\right)^{T} & 1-T \leq j \leq 0, \end{cases} \tag{26}$$

where the vector $\widehat{A}_{X}$ (resp. $\widehat{A}_{Y}$) of length m is the sample mean of $\widehat{H}_{t}^{X}$ (resp. $\widehat{H}_{t}^{Y}$). The corresponding sample cross-correlation matrix $\widehat{R}\left(j\right)$ equals

$$\widehat{R}\left(j\right) = \mathrm{D}\left(\widehat{\Sigma}_{X}\right)^{-1/2}\widehat{\Lambda}\left(j\right)\mathrm{D}\left(\widehat{\Sigma}_{Y}\right)^{-1/2}, \tag{27}$$

where $\mathrm{D}\left(.\right)$ stands for the diagonal form of a matrix, $\widehat{\Sigma}_{X}$ and $\widehat{\Sigma}_{Y}$ the sample covariance matrices of $\widehat{H}_{t}^{X}$ and $\widehat{H}_{t}^{Y}$, respectively. We consider the following weighted quadratic form that accounts for the dependence between the current value of $\widehat{H}_{t}^{X}$ and lagged values of $\widehat{H}_{t}^{Y}$

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

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

$$\widehat{Q}\left(j\right) = Tvec\left(\widehat{R}\left(j\right)\right)^{T}\left(\widehat{\Gamma}_{X}^{-1}\otimes\widehat{\Gamma}_{Y}^{-1}\right)vec\left(\widehat{R}\left(j\right)\right), \tag{29}$$

with $\widehat{\Gamma}_{X}$ and $\widehat{\Gamma}_{Y}$ the sample correlation matrix of $\widehat{H}_{t}^{X}$ and $\widehat{H}_{t}^{Y}$, respectively. Following Bouhaddioui and Roy (2006), our test statistic is a centered and scaled version of the quadratic form in (28), i.e.,

$$V_{Y\rightarrow X} = \frac{\mathcal{T}-m^{2}C_{T}\left(M\right)}{\left(m^{2}D_{T}\left(M\right)\right)^{1/2}}, \tag{30}$$

with $C_T(M)$ and $D_T(M)$ as defined in (19) and (20) respectively. The above test statistic generalizes in a multivariate setting the one in Hong et al. (2009). Indeed when m is equal to one, which corresponds to the univariate case where 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 Hong et al. (2009) test statistic in (18). The following proposition gives the asymptotic distribution of our test statistic.

Proposition 1. *Suppose that Assumptions of Theorem 1 in Hong et al. (2009) hold. Then under the null hypothesis of no Granger-causality in distribution as stated in (25), we have*

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

Assumptions of Theorem 1 in Hong et al. (2009) impose some regulatory conditions on the time series X_t and Y_t, on the VaR models used including smoothness, moment conditions and adequacy, on the kernel function $\kappa(.)$, and also 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 (resp. d_Y) is the dimension of the parameter θ_X (resp. θ_Y). See Hong et al. (2009, pp. 275) for a complete discussion on these assumptions.

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

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

with $\mathcal{T}^*$ the pseudo version of the weighted quadratic form in (28-29) computed using the true correlation matrices Γ_X and Γ_Y, i.e.,

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

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

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

Lemma 2. *Under Assumptions of Theorem 1 in Hong et al. (2009), we have*

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

Lemma 3. *Under Assumptions of Theorem 1 in Hong et al. (2009), we have*

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

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 and the remaining one is devoted to the 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 first time series X_t:

$$\begin{cases} X_t = \sigma_t v_t, \\ \sigma_t^2 = 0.1 + 0.5\sigma_{t-1}^2 + 0.2X_{t-1}^2, \\ v_t \sim m.d.s.\ (0,1), \end{cases}$$

which corresponds to a GARCH(1,1) model. We make the assumption that the second time series Y_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 the sample size $T \in \{500, 1.000, 2.000\}$, and for each simulation, we compute our test statistic in (30) and make inference 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 $\alpha_1, ..., \alpha_{m+1}$) and the m event variables for each variable X_t and Y_t. The $m+1$ VaRs are computed using a GARCH(1,1) model estimated by quasi-maximum likelihood. The estimated values of the $m+1$ VaRs at time t are

$$VaR_{t,s}^{X} = \widehat{\sigma}_{t,X} q\left(\widehat{v}_t, \alpha_s\right), \quad s = 1, ..., m+1, \tag{36}$$

where $\widehat{\sigma}_{t,X}$ is the fitted volatility at time t, and $q\left(\widehat{v}_t, \alpha_s\right)$ 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 $A = \{\alpha_1, \alpha_2, ..., \alpha_{14}\} = \{0\%, 1\%, 5\%, 10\%, 20\%, ..., 90\%, 95\%, 99\%\}$, which covers regions in the tails and the center of the distribution support of each time series.[6] We also need to make a choice about the kernel function in order to compute our test statistic. We consider the four different usual kernels, i.e. the Daniell (DAN), the Parzen (PAR), the Bartlett (BAR) and the Truncated uniform (TR) one.

Lastly for the choice of the truncation parameter M, we use three different values: $M = [\ln(T)]$, $M = \left[1.5T^{0.3}\right]$ and $M = \left[2T^{0.3}\right]$, with [.] the integer part of the argument. These rates lead to the values $M = 6, 10, 13$ for $T = 500$, $M = 7, 12, 16$ for $T = 1.000$, and $M = 8, 15, 20$ for $T = 2.000$. These values cover a range of lag orders for the sample sizes considered. Table B.1 displays the empirical sizes of our test over 500 simulations and for two different nominal risk levels $\eta \in (5\%, 10\%)$. Results in Table B.1 show that our test is well-sized. Indeed, the rejection frequencies are close to the nominal risk levels. Hence, the standard Gaussian distribution provides asymptotically a good approximation of the distribution of our test statistic. This result seems 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. Since causality in distribution springs from causality in moments such as mean or variance, we assume different DGPs which correspond to these cases. The first DGP assumes the existence of a linear Granger-causality in mean in order to generate data under

[6] Recall that for $\alpha_s = 0\%$, the VaR corresponds to $-\infty$.

the alternative hypothesis:

$$\begin{cases} Y_t = 0.5Y_{t-1} + u_{t,Y}, \\ u_{t,Y} = \sigma_{t,Y} v_{t,Y}, \\ \sigma^2_{t,Y} = 0.1 + 0.5\sigma^2_{t-1,Y} + 0.2u^2_{t-1,Y}, \end{cases} \tag{37}$$

$$\begin{cases} X_t = 0.5X_{t-1} + 0.3Y_{t-1} + u_{t,X}, \\ u_{t,X} = \sigma_{t,X} v_{t,X}, \\ \sigma^2_{t,X} = 0.1 + 0.5\sigma^2_{t-1,X} + 0.2u^2_{t-1,X}, \end{cases} \tag{38}$$

where both $v_{t,Y}$ and $v_{t,X}$ are martingale difference sequences with mean 0 and variance 1. The empirical powers of our test are computed over 500 simulations for $T \in \{500, 1.000, 2.000\}$. As in the analysis of the size, we consider three values of the truncation parameter M, and two nominal risk levels $\eta = 5\%$, 10%. The results are reported in Table B.2, only for the Daniell kernel to save space.[7] For comparison we also display in Table B.2 results for the Granger-causality test in mean. In order to have a fair comparison, we do not use the usual parametric Granger-causality test in mean derived from a vector autoregressive model. We consider instead the kernel-based non-parametric Granger-causality test in mean introduced by Hong (1996). Results in Table B.2 show that our kernel-based nonparametric test for Granger-causality in distribution has appealing power properties. For instance, the rejection frequencies of the null hypothesis for $(T, M) = (500, 6)$ are equal to 93.6% and 95.6% for $\eta = 5\%$ and 10%, respectively. For $T = 1.000, 2.000$ the powers are equal to one. The rejection frequencies of the Granger-causality test in mean are always equal to 100% and hence are slightly higher than the ones of our Granger-causality test in distribution for the smallest sample. This result is expected as the assumed causality in distribution springs from causality in mean.

To stress the relevance of our testing approach, we consider a second representation of the DGPs under the alternative hypothesis, assuming causality in

[7] Results for the other kernels are similar and available from the authors upon request. The only exception occurs for the uniform kernel which has a relatively low power, because of its uniform weighting which does not discount higher order lags.

distribution stemming from a non-linear form of causality in mean. Precisely, we generate data for the time series Y_t using the specification in (37), and the second time series is generated as follows

$$\begin{cases} X_t = 0.5X_{t-1} + 0.3Y_{t-1}^2 + u_{t,X}, \\ u_{t,X} = \sigma_{t,X} v_{t,X}, \\ \sigma_{t,X}^2 = 0.1 + 0.5\sigma_{t-1,X}^2 + 0.2u_{t-1,X}^2. \end{cases} \tag{39}$$

Table B.3 reports the rejection frequencies over 500 simulations. The presentation is similar to Table B.2. We observe that while the Granger-cauality test in mean fails to reject the null hypothesis for most of the simulations, our test still exhibits good power in detecting this non-linear form of causality. For illustration the rejection frequency of the null hypothesis for $(T, M) = (500, 6)$ is equal to 75.2% for $\eta = 5\%$, while it is only equal to 18.2% for the causality test in mean in the same configuration. Remark that for our test, the power drops as the truncation parameter M increases. Moreover, the power increases as the sample size increases and converges to 100%.

Lastly, we generate data under the alternative hypothesis, assuming Granger-causality in variance. Formally, we suppose once again that Y_t has the specification in (37), and X_t is generated as

$$\begin{cases} X_t = 0.5X_{t-1} + u_{t,X}, \\ u_{t,X} = \sigma_{t,X} v_{t,X}, \\ \sigma_{t,X}^2 = 0.1 + 0.5\sigma_{t-1,X}^2 + 0.2u_{t-1,X}^2 + 0.7Y_{t-1}^2. \end{cases} \tag{40}$$

Results displayed in Table B.4 are qualitatively similar to the ones in Table B.3. Our causality test in distribution has good powers in rejecting the null hypothesis, while the causality test in mean exhibits low powers. Overall the reported values are lowers to the ones in Tables B.2 and B.3. This pattern can be explained by the fact that (i) causality in variance takes place mainly in the tails, (ii) and the dynamics of the tails are more difficult to fit due to the lack of data.

4. Empirical part

Recent financial crises have all been characterized by quick and large regional spill-overs of negative financial shocks. For example, consecutively to the Greek distress (2009), South European countries have been contaminated, facing skyrocketing refinancing rates. Besides it has impacted North European states in an opposite way. Considered as safe harbors for investors, they were able to refinance their debt on markets at lower rates. It is obvious that the degree of globalization within 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. And as Southern European countries used foreign capital markets to finance their domestic investments and boost their growth, they have been highly subject to financial instability.

It is of major importance for empirical studies to evaluate the importance of these spill-overs. Theoretically it relies on the crisis-contingent theories, which explain the increase in market cross-correlation after a shock issued in an origin country as resulting from multiple equilibria based on investor psychology; endogenous liquidity shocks causing a portfolio recomposition; and/or political disturbances affecting the exchange rate regime.[8] [9] The presence of spill-overs during a crisis can be thus tested empirically by a significant and transitory increase in cross-correlation between markets. (See inter alia King and Wadhwani, 1990, Calvo and Reinhart, 1995 and Baig and Goldfajn, 1998). Nevertheless, this intuitive approach, which presents the advantage of simplicity as it avoids the identification of the transmission channels, presents many shortcomings:

First, Forbes and Rigobon (2002) show that an increase in correlation can be exclusively driven by an higher volatility during crisis periods. In such a case, it could not be attributed to a stronger economic interdependence. To correct for this potential bias, they thus propose to use a modified version of

[8] see Rigobon (2000) for a survey.

[9] On contrary, according to the non-crisis-contingent theories, the propagation of shocks does not lead to a shift from a good to a bad equilibrium, but the increase in cross-correlation is the continuation of linkages (trade and/or financial) existing before the crisis.

the correlation[10] and test for its temporary increase during crisis period.

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 such a reason, Bodart and Candelon (2009) prefer to consider an indicator of causality to measure spill-overs. It is thus possible to evaluate asymmetrical spill-overs, which can then move from i to j, j to i or in both directions. Besides, using Granger-causality approach requires the estimation of multivariate dynamic models which are less prone to potential misspecification issues.

It is, more or less, feasible to tackle both these shortcomings in a classical framework. Nevertheless, even if comparing causality between pre- and crisis periods allows to evaluate spill-overs, it does not permit to separate interdependence and contagion. Interdependence deals with the long run structural links between markets. It thus provides information on the extend to which markets are integrated. Therefore, interdependence should be analysed without considering extreme positive or negative events. On the contrary, contagion deals with short-run abrupt increases in the causal linkages and takes place exclusively during crisis' period. Thus, testing for contagion requires to exclusively focus on the extremal left tail of the distribution, as it is performed in extreme value theory (see Hartman et al., 2004). Our Granger-causality test in distribution allows to tackle all these issues. Indeed, it offers an asymmetric measure of spill-overs, based on a dynamic representation. Besides, it is possible to investigate if causality has increased for the whole distribution but also for specific percentiles of the distribution, in particular those located at the left tail or right tails, corresponding to extreme events.

As an illustration, we analyse the recent European crisis and consider a set of 12 European daily stock market indices (Austria, Belgium, Finland, France, Germany, Greece, Ireland, Italy, Luxemburg, the Netherlands, Portugal and Spain) downloaded from datastream ranging from January 1, 2007 to May 6,

[10] In fact, they are using the unconditional correlation instead of the conditional one.

2011 (i.e. $T = 1.134$ observations). The first empirical illustration consists in testing for interdependence. It is performed implementing the pairwise Granger-causality test for the whole distribution but removing crisis's periods, i.e. the right and left tails. Then, in a second analysis, we repeat this analysis for the left tail in order to test for contagion during crisis. This part refers to the EVT approach of spill-overs and extend Hartmann et al (2004). Similarly, the test is conducted for the right tail, i.e. upswing period. We can then compare the strength of contagion during crises vs boom periods and check in which periods contagion is the most significant.

4.1. The general design of the Granger-causality test in distribution to test for spill-over

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 levels α_s $s = 1, ..., m+1$, which cover its distribution support. As for the Monte Carlo simulations, we consider the following set for the VaR levels $A = \{0\%, 1\%, 5\%, 10\%, ..., 90\%, 95\%, 99\%\}$ with $m + 1 = 14$. To compute the VaRs, we use a semi-parametric model. Formally, we suppose that each index returns series $R_{i,t}$ $i = 1, ..., 12$, follows an $AR(m)$-$GARCH(p, q)$ model, with:

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

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

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

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

$$VaR_{t,s}^{i} = \sum\nolimits_{j=1}^{m} \widehat{\phi}_{i,j} R_{i,t-j} + \widehat{\sigma}_{t,i} q\left(\widehat{v}_{i,t}, \alpha_s\right), \quad s = 1, ..., m+1, \tag{44}$$

with $\widehat{\sigma}_{t,i}$ the fitted volatility at time t for the index number i, and $q\left(\widehat{v}_{i,t}, \alpha_s\right)$ the empirical quantile of order α_s of the estimated standardized innovations $\widehat{v}_{i,t}$. Table B.5 displays the estimation results of the AR(m)-GARCH(p, q) models for the indices. As shown through the Ljung-Box test applied to the residuals and their squares, the retained specifications succesfully 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 inter-quantiles event variables, and compute for each couple (i, j) of indices our kernel-based non parametric 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 $\left[1.5T^{0.3}\right]$ which leads to the value of $M = 12$ for the whole sample of length $T = 1.134$.

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 from the distribution the extreme events. The new set A of VaRs risk levels is equal to $A = \{20\%, 30\%, ..., 70\%, 80\%\}$ with $m + 1 = 7$. Table B.6 displays the results of the test. The reported values are the p-values in percentage. Hence, for a nominal risk 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%. Test statistics corresponding to the rejection of the null hypothesis of no causality are put in bold. The last column labelled "Sum" provides for a given index in row, the number of time it is Granger-caused by the others. Similarly, the last row labelled "Sum" provides for a given stock market index the number of time it Granger-causes others stock market indices. Lastly, the entry corresponding to the last row and last column gives the total number of significant Granger-causality cases for our set of indices. It turns out that interdependence 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. Looking at the countries results, we observe that the Austrian and the French stock markets are the most

integrated ones, as they are affected by 3 other European markets, respectively. On the contrary, Greece, Ireland, Italy, Luxemburg and Netherlands appear as independent from the other markets. It is interesting to notice that the causal matrix is not symmetric: France which is among the most caused markets does not affect any market. It hence supports our choice for causality as a measure of spill-over rather than correlation. The most causal markets are Netherlands, Greece and Portugal. The presence of these two last countries is interesting as 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.

4.3. Testing for contagion

Contagion is apprehended implementing our Granger-causality test in left-tail distribution. The set A of VaRs risk levels is now set as $A = \{0\%, 1\%, 5\%, 10\%\}$ with $m + 1 = 4$. Table B.7 displays the outcomes of the tests. The number of causal pairs increases to 35.6% of the cases supporting hence the presence of contagion. We observe that the most causal markets are Portugal, Italy, Netherlands, Greece and Ireland, and except Netherlands, this group includes all the countries in turmoils (Portugal, Italy, Greece and Ireland), around which the crisis was build. On the other side, the most caused markets are Austria, Belgium, Italy, France, Luxembourg and Greece. Remark the predominant role of Italy and Greece in the system, which cause and are caused in many cases.

The Granger-causality test is now repeated for the left-tail distribution with $A = \{90\%, 95\%, 99\%, 100\%\}$, i.e., $m + 1 = 4$ and results are reported in Table B.8. It appears that "positive" contagion is only supported in 7.5% of the cases and concerns mainly Spain as spill-overs driver, and Luxembourg, Germany and Belgium as spill-overs receivers. The asymmetry between causal tests in the right and left tail is striking. Whereas spill-overs are important in crisis periods, they are only weakly present in upswing times. Such a feature highlights that European stock markets integration is strongly vulnerable to negative, and to a lesser extend positive, shocks. European policy makers should acknowledge it

and set up structural measures to limit it.

5. Conclusion

A kernel-based non-parametric test for Granger-causality in distribution between two time series is proposed in this paper. The test checks for spill-overs between the multivariate processes of dynamics inter-quantile event variables associated to each variable. Beyond the existing approaches our testing methodology has two main advantages. First, it can be used to test for Granger-causality in specific regions of the distributions, like the center or the tails (left and right). Second, it checks for a large number of lags discounting higher order lags, and hence is consistent against causality which carries over long distributional lags.

We show that the test has a standard Gaussian distribution under the null hypothesis which is free of parameter estimation uncertainty. We run a Monte Carlo simulations exercise which shows that the Gaussian distribution is valid in small samples. The test also has very appealing power properties in various settings including linear and non-linear causality in mean and causality in variance.

In an empirical part we implement our testing procedure to 12 European daily stock market indices to analyze spill-overs during the recent European crisis. As our test is designed to check for causality in specific regions of the distribution (center or tails), it can be used to test for both the presence of interdependence and contagion. Indeed interdependence can be checked through Granger-causality in the center of the distribution, as interdependence is a long run path that takes place in normal periods. On the contrary, contagion refers to a short-run abrupt increase in the causal linkages taking place exclusively during crisis period, and can be tested via Granger-causality in distribution's tails.

The empirical results indicate that European stock market integration is far from being achieved, because we observe very weak evidence supporting interdependence. On contrary, our results support the presence of contagion, with a strong asymmetry between contagion in the right and left tails. More precisely,

contagion is important in crisis periods, whereas it is weak in upswing times. Such a result constitutes an important feature for the European stock markets, and policy makers should acknowledge it in designing structural measures for financial stability.

Appendix A. Proof of LEMMAS

Appendix A.1. Proof of LEMMA 2

Lemma 2: Under Assumptions of Theorem 1 in Hong et al. (2009), we have

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

Proof: Consider the pseudo version of the weighted quadratic form $\mathcal{T}^*$ defined as

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

$$\widehat{Q}^*(j) = T vec\left(\widehat{R}(j)\right)^T \left(\Gamma_X^{-1} \otimes \Gamma_Y^{-1}\right) vec\left(\widehat{R}(j)\right), \tag{A.3}$$

where Γ_X (resp. Γ_Y) is the correlation matrix of the true unknown multivariate process of event variables $H_t^X\left(\theta_X^0\right)$ (resp. $H_t^Y\left(\theta_Y^0\right)$). Recall that $H_t^X\left(\theta_X^0\right)$ is defined as

$$H_t^X\left(\theta_X^0\right) = \left(Z_{t,1}^X\left(\theta_X^0\right), ..., Z_{t,m}^X\left(\theta_X^0\right)\right), \tag{A.4}$$

where the event variables $Z_{t,s}^X\left(\theta_X^0\right)$, $s = 1, ..., m$, are related to distinct regions on the distribution support of X_t. Hence, they are mutually independent, and the associated correlation matrix Γ_X is equal to the identity matrix. The same reasoning applies for $H_t^Y\left(\theta_Y^0\right)$, with the consequence that Γ_Y is also equal to the identity matrix. Hence, the pseudo weighted quadratic form $\mathcal{T}^*$ defined in (A.2-A.3) takes the expression

$$\mathcal{T}^* = T\sum_{j=1}^{T-1} \kappa^2(j/M)\, vec\left(\widehat{R}(j)\right)^T vec\left(\widehat{R}(j)\right). \tag{A.5}$$

Since $\widehat{R}(j)$ is defined as the cross-correlation matrix at lag-order j between $\widehat{H}_t^X = \left(\widehat{Z}_{t,1}^X, ..., \widehat{Z}_{t,m}^X\right)$ and $\widehat{H}_t^Y = \left(\widehat{Z}_{t,1}^Y, ..., \widehat{Z}_{t,m}^Y\right)$, its components are given by

the correlations between $\widehat{Z}_{t,k}^{X}$ and $\widehat{Z}_{t-j,p}^{Y}$ with $k=1,...m$ and $p=1,...,m$. Let us denote $\widehat{\rho}_{k,p}(j)$ such correlation, i.e.,

$$\widehat{\rho}_{k,p}(j)=\operatorname{corr}\left(\widehat{Z}_{t,k}^{X},\widehat{Z}_{t-j,p}^{Y}\right). \tag{A.6}$$

With the definition of the vec operator, it is easy to see that $vec\left(\widehat{R}(j)\right)$ has m^2 components given by $\widehat{\rho}_{k,p}(j)$, $k=1,...m$ and $p=1,...,m$. Consequently, the pseudo quadratic form $\mathcal{T}^{*}$ in (A.5) becomes

$$\begin{aligned}\mathcal{T}^{*} &= T\sum_{j=1}^{T-1}\kappa^{2}(j/M)\sum_{k=1}^{m}\sum_{p=1}^{m}\widehat{\rho}_{k,p}^{2}(j),\\ &= \sum_{k=1}^{m}\sum_{p=1}^{m}\left(T\sum_{j=1}^{T-1}\kappa^{2}(j/M)\,\widehat{\rho}_{k,p}^{2}(j)\right).\end{aligned} \tag{A.7}$$

For a given value of the couple (k,p), the quadratic form in the bracket of (A.7) corresponds to the uncentered and unscaled test statistic in Hong et al. (2009) for the Granger-causality from $\widehat{Z}_{t,p}^{Y}$ to $\widehat{Z}_{t,k}^{X}$, and these statistics are obviously independent. We deduce from this that under Assumptions of Theorem 1 in Hong et al. (2009)

$$T\sum_{j=1}^{T-1}\kappa^{2}(j/M)\,\widehat{\rho}_{k,p}^{2}(j)\longrightarrow^{d}\mathcal{N}\left(C_{T}(M),D_{T}(M)\right). \tag{A.8}$$

Remark that the event variables in Hong et al. (2009) are related to one-sided regions, whereas we have to deal with two-sided regions in our framework. However in their proof of Theorem 1, it is easy to see that all of the technical results remain valid even in the case of two-sided regions. For this, it suffices to replace $Z_{1t}(\theta_1)$ and $Z_{2t}(\theta_2)$ by $Z_{t,k}^{X}(\theta_X)$ and $Z_{t,p}^{Y}(\theta_Y)$, with θ_X and θ_Y any vector in the parameter spaces Θ_X and Θ_Y, respectively. Moreover, the martingale difference sequence $W_{1t}(\theta_1)$ and $W_{2t}(\theta_2)$ in the proof of Theorem 1 in Hong et al. (2009) must be replaced by $W_{t,k}^{X}(\theta_X)$ and $W_{t,p}^{Y}(\theta_Y)$ respectively in order to deal with two-sided regions, with

$$\begin{aligned}W_{t,k}^{X}(\theta_X) &= Z_{t,k}^{X}(\theta_X)-Z_{t,k}^{X}\left(\theta_X^{0}\right)-\mathbb{E}\left(Z_{t,k}^{X}(\theta_X)\,\middle|\,\mathcal{F}_{t-1}^{X}\right)+\mathbb{E}\left(Z_{t,k}^{X}\left(\theta_X^{0}\right)\,\middle|\,\mathcal{F}_{t-1}^{X}\right)\\ &= Z_{t,k}^{X}(\theta_X)-Z_{t,k}^{X}\left(\theta_X^{0}\right)-\left[F_X\left(VaR_{t,k+1}^{X}(\theta_X)\right)-F_X\left(VaR_{t,k}^{X}(\theta_X)\right)\right]+\\ &\quad+\left[F_X\left(VaR_{t,k+1}^{X}\left(\theta_X^{0}\right)\right)-F_X\left(VaR_{t,k}^{X}\left(\theta_X^{0}\right)\right)\right],\end{aligned}$$

$$
\begin{aligned}
W_{t,p}^{Y}\left(\theta_{Y}\right) &= Z_{t,p}^{Y}\left(\theta_{Y}\right)-Z_{t,p}^{Y}\left(\theta_{Y}^{0}\right)-\mathbb{E}\left(Z_{t,p}^{Y}\left(\theta_{Y}\right)\left|\mathcal{F}_{t-1}^{Y}\right.\right)+\mathbb{E}\left(Z_{t,p}^{Y}\left(\theta_{Y}^{0}\right)\left|\mathcal{F}_{t-1}^{Y}\right.\right) \\
&= Z_{t,p}^{Y}\left(\theta_{Y}\right)-Z_{t,p}^{Y}\left(\theta_{Y}^{0}\right)-\left[F_{Y}\left(VaR_{t,p+1}^{Y}\left(\theta_{Y}\right)\right)-F_{Y}\left(VaR_{t,p}^{Y}\left(\theta_{Y}\right)\right)\right]+ \\
&\quad +\left[F_{Y}\left(VaR_{t,p+1}^{Y}\left(\theta_{Y}^{0}\right)\right)-F_{Y}\left(VaR_{t,p}^{Y}\left(\theta_{Y}^{0}\right)\right)\right],
\end{aligned}
$$

with $F_X(.)$ and $F_Y(.)$ the conditional cumulative distribution functions of X and Y respectively. Hence, we can conclude using (A.8) that the pseudo quadratic form $\mathcal{T}^*$ in (A.7) has the limiting distribution

$$
\mathcal{T}^{*}\longrightarrow^{d}\mathcal{N}\left(m^{2}C_{T}\left(M\right),m^{2}D_{T}\left(M\right)\right). \tag{A.9}
$$

and this completes the proof of LEMMA 2.

Appendix A.2. Proof of LEMMA 3

Lemma 3: Under Assumptions of Theorem 1 in Hong et al. (2009), we have

$$
\frac{\mathcal{T}-\mathcal{T}^{*}}{\left(m^{2}D_{T}\left(M\right)\right)^{1/2}}\longrightarrow^{p}0. \tag{A.10}
$$

Proof: The proof of Lemma 3 proceeds by combining elements in the proofs of Proposition 3.2 in Bouhaddioui and Roy (2006) and Theorems A.1 and A.3 in Hong et al. (2009). Formally, given that

$$
D_{T}\left(M\right)=M\int_{0}^{\infty}\kappa^{4}\left(z\right)dz\left[1+o\left(1\right)\right], \tag{A.11}
$$

as $M\rightarrow\infty$, the proof of Lemma 3 can be established showing that

$$
\mathcal{T}-\mathcal{T}^{*}=O_{p}\left(M/T^{1/2}\right). \tag{A.12}
$$

Based on Lemma 4.1 in El Himdi and Roy (1997), the quadratic forms $\mathcal{T}$ and $\mathcal{T}^*$ can be rewritten in term of cross-covariances as

$$
\mathcal{T}=T\sum_{j=1}^{T-1}\kappa^{2}\left(\frac{j}{M}\right)vec\left(\widehat{\Lambda}\left(j\right)\right)^{T}\left(\widehat{\Sigma}_{X}^{-1}\otimes\widehat{\Sigma}_{Y}^{-1}\right)vec\left(\widehat{\Lambda}\left(j\right)\right), \tag{A.13}
$$

$$
\mathcal{T}^{*}=T\sum_{j=1}^{T-1}\kappa^{2}\left(\frac{j}{M}\right)vec\left(\widehat{\Lambda}\left(j\right)\right)^{T}\left(\Sigma_{X}^{-1}\otimes\Sigma_{Y}^{-1}\right)vec\left(\widehat{\Lambda}\left(j\right)\right), \tag{A.14}
$$

with $\widehat{\Lambda}\left(j\right)$ the sample cross-covariance matrix at lag-order j, Σ_X and Σ_Y the covariance matrices of the true multivariate processes of event variables $H_t^X\left(\theta_X^0\right)$

and $H_t^Y\left(\theta_Y^0\right)$, and $\widehat{\Sigma}_X$ and $\widehat{\Sigma}_Y$ their sample counterparts given by the covariance matrices of $\widehat{H}_t^X \equiv H_t^X\left(\widehat{\theta}_X\right)$ and $\widehat{H}_t^Y \equiv H_t^Y\left(\widehat{\theta}_Y\right)$, respectively. It follows that

$$\mathcal{T}-\mathcal{T}^* = T\sum_{j=1}^{T-1}\kappa^2\left(\frac{j}{M}\right) vec\left(\widehat{\Lambda}\left(j\right)\right)^T\left\{\widehat{\Sigma}_X^{-1}\otimes\widehat{\Sigma}_Y^{-1}-\Sigma_X^{-1}\otimes\Sigma_Y^{-1}\right\} vec\left(\widehat{\Lambda}\left(j\right)\right). \tag{A.15}$$

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 $\widehat{Z}_{t,k}^X$, $k=1,...,m$. Let $\widehat{C}_{k,p}$ be a typical element of $\widehat{\Sigma}_X$ with

$$\widehat{C}_{k,p} = \operatorname{cov}\left(\widehat{Z}_{t,k}^X, \widehat{Z}_{t,p}^X\right). \tag{A.16}$$

Let $C_{k,p}^0$ be the true value of $\widehat{C}_{k,p}$, i.e., the covariance between the true event variables $Z_{t,k}^X\left(\theta_X^0\right)$ and $Z_{t,p}^X\left(\theta_X^0\right)$. 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

$$\widehat{C}_{k,p}-C_{k,p}^0 = \widehat{M}_1\left(\widehat{\theta}_X\right)+\widehat{M}_2\left(\widehat{\theta}_X\right)+\widehat{M}_3\left(\widehat{\theta}_X\right), \tag{A.17}$$

with

$$\widehat{M}_1\left(\widehat{\theta}_X\right) = T^{-1}\sum_{t=1}^{T}\left[\widehat{Z}_{t,k}^X - Z_{t,k}^X\left(\theta_X^0\right)\right]\left[Z_{t,p}^X\left(\theta_X^0\right)-\pi_p^X\right] \tag{A.18}$$

$$\widehat{M}_2\left(\widehat{\theta}_X\right) = T^{-1}\sum_{t=1}^{T}\left[Z_{t,k}^X\left(\theta_X^0\right)-\pi_k^X\right]\left[\widehat{Z}_{t,p}^X - Z_{t,p}^X\left(\theta_X^0\right)\right] \tag{A.19}$$

$$\widehat{M}_3\left(\widehat{\theta}_X\right) = T^{-1}\sum_{t=1}^{T}\left[\widehat{Z}_{t,k}^X - Z_{t,k}^X\left(\theta_X^0\right)\right]\left[\widehat{Z}_{t,p}^X - Z_{t,p}^X\left(\theta_X^0\right)\right], \tag{A.20}$$

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 $\pi_k^X = \mathbb{E}\left(Z_{t,k}^X\left(\theta_X^0\right)\right)$ and $\pi_p^X = \mathbb{E}\left(Z_{t,p}^X\left(\theta_X^0\right)\right)$. Using the following result in the proof of theorem A.3 in Hong et al. (2009)

$$\sup_{\theta_X\in\Theta_X}\left|\widehat{M}_1\left(\theta_X\right)\right| = O_p\left(T^{-1/2}\right), \tag{A.21}$$

with θ_X any $\sqrt{T}$-consistent estimator of θ_X^0 in the space Θ_X, we have for the first term

$$\widehat{M}_1\left(\widehat{\theta}_X\right) = O_p\left(T^{-1/2}\right). \tag{A.22}$$

Similar arguments apply for the last two terms, with the consequence that

$$\widehat{M}_2\left(\widehat{\theta}_X\right) = O_p\left(T^{-1/2}\right), \tag{A.23}$$

$$\widehat{M}_3\left(\widehat{\theta}_X\right) = O_p\left(T^{-1/2}\right). \tag{A.24}$$

We deduce that

$$\widehat{C}_{k,p} - C^0_{k,p} = O_p\left(T^{-1/2}\right), \tag{A.25}$$

and

$$\widehat{\Sigma}_X - \Sigma_X = O_p\left(T^{-1/2}\right). \tag{A.26}$$

Using the same reasoning for the elements of $\widehat{\Sigma}_Y$ we have that

$$\widehat{\Sigma}_Y - \Sigma_Y = O_p\left(T^{-1/2}\right), \tag{A.27}$$

and

$$\widehat{\Sigma}_X^{-1} \otimes \widehat{\Sigma}_Y^{-1} - \Sigma_X^{-1} \otimes \Sigma_Y^{-1} = O_p\left(T^{-1/2}\right). \tag{A.28}$$

Hence equation (A.15) becomes

$$\begin{aligned} \mathcal{T} - \mathcal{T}^* &= T\sum_{j=1}^{T-1} \kappa^2\left(\frac{j}{M}\right) vec\left(\widehat{\Lambda}(j)\right)^T O_p\left(T^{-1/2}\right) vec\left(\widehat{\Lambda}(j)\right) \qquad \text{(A.29)} \\ &= O_p\left(T^{1/2}\right)\sum_{j=1}^{T-1} \kappa^2\left(\frac{j}{M}\right) vec\left(\widehat{\Lambda}(j)\right)^T vec\left(\widehat{\Lambda}(j)\right). \end{aligned}$$

The rest of the proof proceeds by showing that

$$\mathcal{B}(T) = \sum_{j=1}^{T-1} \kappa^2\left(\frac{j}{M}\right) vec\left(\widehat{\Lambda}(j)\right)^T vec\left(\widehat{\Lambda}(j)\right) = O_p(M/T). \tag{A.30}$$

We decompose $\mathcal{B}(T)$ into two parts

$$\mathcal{B}(T) = \mathcal{B}_1(T) + \mathcal{B}_2(T), \tag{A.31}$$

with

$$\mathcal{B}_1(T) = \sum_{j=1}^{T-1} \kappa^2\left(\frac{j}{M}\right)\left\{vec\left(\widehat{\Lambda}(j)\right)^T vec\left(\widehat{\Lambda}(j)\right) - vec(\Lambda(j))^T vec(\Lambda(j))\right\}, \tag{A.32}$$

$$\mathcal{B}_2(T) = \sum_{j=1}^{T-1} \kappa^2\left(\frac{j}{M}\right) vec\left(\Lambda(j)\right)^T vec\left(\Lambda(j)\right), \tag{A.33}$$

where $\Lambda(j)$ is the cross-covariance matrix at lag-order j of the true event variables $H_t^X\left(\theta_X^0\right)$ and $H_t^Y\left(\theta_Y^0\right)$. Let us first consider $\mathcal{B}_1(T)$ in (A.32). Since $\widehat{\Lambda}(j)$ is defined as the estimated cross-covariance matrix at lag-order j between

$$\widehat{H}_t^X = \left(\widehat{Z}_{t,1}^X, \widehat{Z}_{t,2}^X, ..., \widehat{Z}_{t,m}^X\right), \tag{A.34}$$

and

$$\widehat{H}_t^Y = \left(\widehat{Z}_{t,1}^Y, \widehat{Z}_{t,2}^Y, ..., \widehat{Z}_{t,m}^Y\right), \tag{A.35}$$

its components are given by the covariances between $\widehat{Z}_{t,k}^X$ and $\widehat{Z}_{t-j,p}^Y$, $k = 1, ..., m$, $p = 1, ..., m$. Denote $\widehat{C}_{k,p}(j)$ such covariances. Using the definition of the vec operator we thus have

$$\mathcal{B}_1(T) = \sum_{j=1}^{T-1} \kappa^2\left(\frac{j}{M}\right) \sum_{k=1}^{m}\sum_{p=1}^{m} \left\{\widehat{C}_{k,p}^2(j) - C_{k,p}^2(j)\right\}, \tag{A.36}$$

which can be rewritten as

$$\begin{aligned}
\mathcal{B}_1(T) &= \sum_{j=1}^{T-1} \kappa^2\left(\frac{j}{M}\right) \sum_{k=1}^{m}\sum_{p=1}^{m} \left\{\left(\widehat{C}_{k,p}(j) - C_{k,p}(j)\right)^2 + \left(\widehat{C}_{k,p}(j) - C_{k,p}(j)\right) C_{k,p}(j)\right\} \\
&= \sum_{k=1}^{m}\sum_{p=1}^{m} \left\{\widehat{Q}_1 + \widehat{Q}_2\right\},
\end{aligned} \tag{A.37}$$

with

$$\widehat{Q}_1 = \sum_{j=1}^{T-1} \kappa^2\left(\frac{j}{M}\right) \left(\widehat{C}_{k,p}(j) - C_{k,p}(j)\right)^2, \tag{A.38}$$

$$\widehat{Q}_2 = \sum_{j=1}^{T-1} \kappa^2\left(\frac{j}{M}\right) \left(\widehat{C}_{k,p}(j) - C_{k,p}(j)\right) C_{k,p}(j). \tag{A.39}$$

Using the results of Theorem A.1 in Hong et al. (2009), that is

$$\widehat{Q}_1 = O_p\left(M^{1/2}/T\right), \tag{A.40}$$

$$\widehat{Q}_2 = O_p\left(M^{1/2}/T\right), \tag{A.41}$$

we have

$$\mathcal{B}_1(T) = O_p\left(M^{1/2}/T\right). \tag{A.42}$$

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

$$\mathcal{B}_2(T) = \sum_{j=1}^{T-1} \kappa^2\left(\frac{j}{M}\right) vec(\Lambda(j))^T vec(\Lambda(j)) = O_p(M/T). \tag{A.43}$$

We deduce from (A.42) and (A.43) that

$$\mathcal{B}(T) = O_p(M/T), \tag{A.44}$$

and

$$\mathcal{T} - \mathcal{T}^* = O_p\left(T^{1/2}\right) O_p(M/T) = O_p\left(M/T^{1/2}\right). \tag{A.45}$$

This completes the proof of Lemma 3.

Appendix B. Tables and Figures

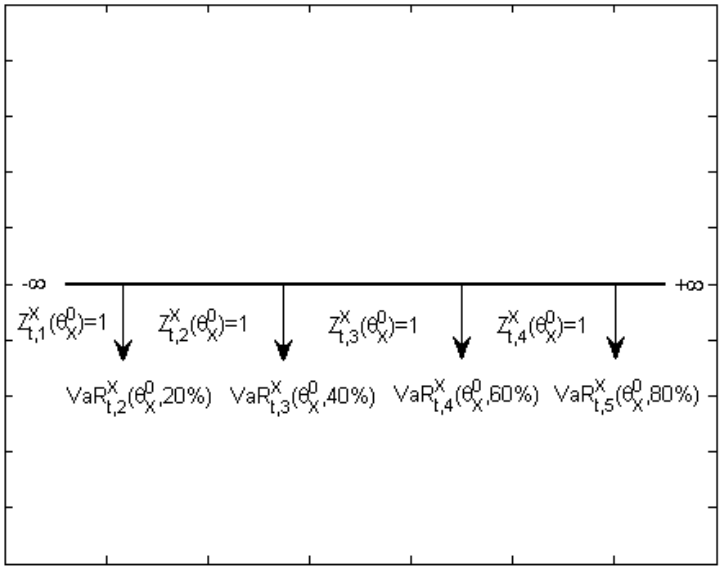

Figure B.1: Distribution support of X and localization of $VaRs$ and event variables

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

T	M	η	DAN	BAR	PAR	TR
500	6	5%	5.80	5.80	5.60	4.40
		10%	10.40	10.40	11.40	9.80
	10	5%	5.20	5.60	5.80	5.60
		10%	10.00	9.60	10.20	11.60
	13	5%	5.60	4.80	5.20	5.40
		10%	10.40	10.60	10.20	11.80
1000	7	5%	5.00	4.20	4.40	5.20
		10%	9.00	9.40	8.40	10.80
	12	5%	5.00	5.20	5.00	6.00
		10%	9.40	10.00	9.00	11.80
	16	5%	5.40	5.60	6.20	6.20
		10%	10.00	10.20	10.00	11.40
2000	8	5%	6.60	7.00	6.60	5.20
		10%	11.80	11.80	13.40	8.80
	15	5%	5.60	6.20	6.40	6.20
		10%	10.20	11.20	11.60	11.60
	20	5%	5.00	5.00	6.60	4.80
		10%	10.60	10.80	11.20	9.60

Notes: The table displays the empirical sizes (in %) of the Granger-causality test in distribution. Rejection frequencies are reported over 500 **simulations for two nominal risk levels η, with T the sample size and M the truncation parameter. DAN, BAR, PAR and TR refer to the Daniell, the Bartlett, the Parzen, and the truncated kernels, respectively.**

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

T	M	η	Distribution	Mean
500	6	5%	93.60	100.00
		10%	95.60	100.00
	10	5%	91.80	100.00
		10%	93.20	100.00
	13	5%	90.40	100.00
		10%	92.40	100.00
1000	7	5%	100.00	100.00
		10%	100.00	100.00
	12	5%	100.00	100.00
		10%	100.00	100.00
	16	5%	100.00	100.00
		10%	100.00	100.00
2000	8	5%	100.00	100.00
		10%	100.00	100.00
	15	5%	100.00	100.00
		10%	100.00	100.00
	20	5%	100.00	100.00
		10%	100.00	100.00

Notes: The table displays the empirical powers (in %) of the Granger-causality test in distribution. Rejection frequencies are reported over 500 simulations for two nominal risk levels η, with T the sample size and M the truncation parameter. For comparison, we also report the rejection frequencies of the kernel-based nonparametric test in mean. For both test, results are reported for the Daniell kernel. Data are generated under the alternative hypothesis assuming Granger-causality in mean.

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

T	M	η	Distribution	Mean
500	6	5%	75.20	18.20
		10%	82.80	21.40
	10	5%	68.20	16.20
		10%	76.60	20.60
	13	5%	60.20	15.80
		10%	70.60	20.60
1000	7	5%	97.80	20.60
		10%	98.40	21.60
	12	5%	95.80	19.20
		10%	97.00	23.20
	16	5%	93.00	19.20
		10%	95.80	22.60
2000	8	5%	100.00	24.20
		10%	100.00	28.60
	15	5%	100.00	19.60
		10%	100.00	24.40
	20	5%	100.00	17.60
		10%	100.00	21.80

Notes: The table displays the empirical powers (in %) of the Granger-causality test in distribution. Rejection frequencies are reported over 500 simulations for two nominal risk levels η, with T the sample size and M the truncation parameter. For comparison, we also report the rejection frequencies of the kernel-based nonparametric test in mean. For both test, results are reported for the Daniell kernel. Data are generated under the alternative hypothesis assuming nonlinear Granger-causality in mean.

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

T	M	η	Distribution	Mean
500	6	5%	51.40	18.80
		10%	61.40	23.00
	10	5%	50.20	20.40
		10%	60.40	25.80
	13	5%	48.20	21.40
		10%	57.80	25.80
1000	7	5%	79.80	17.80
		10%	86.80	21.60
	12	5%	72.40	22.60
		10%	81.60	27.40
	16	5%	67.00	21.80
		10%	76.20	29.00
2000	8	5%	99.20	15.40
		10%	99.40	19.40
	15	5%	96.60	18.80
		10%	97.60	23.40
	20	5%	93.00	19.00
		10%	95.80	24.40

Notes: The table displays the empirical powers (in %) of the Granger-causality test in distribution. Rejection frequencies are reported over 500 simulations for two nominal risk levels η, with T the sample size and M the truncation parameter. For comparison, we also report the rejection frequencies of the kernel-based nonparametric test in mean. For both test, results are reported for the Daniell kernel. Data are generated under the alternative hypothesis assuming Granger-causality in variance.

Table B.5: Estimation results of AR-GARCH models

Index	$\phi_{i,1}$	κ_i	$\gamma_{i,1}$	$\gamma_{i,2}$	$\gamma_{i,3}$	$\beta_{i,1}$	$LB_{\widehat{v}_{i,t}}(6)$	$LB_{\widehat{v}^2_{i,t}}(6)$
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 parameters estimates followed in brackets by the student statistics. The two last columns give the results of the Ljung-Box test applied to the serie of the standardized innovations $\widehat{v}_{i,t}$ and its square, respectively, with 6 the number of lags. The critical value for the rejection of the null hypothesis at the 5% nominal risk level is equal to 12.59.

Table B.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 column towards the index i in row. Entries with the rejection of the null hypothesis at the 5% nominal risk level are emphasized in bold. The last column labelled "Sum" provides for a given index in row, the number of time it is Granger-caused by the others. Similarly, the last row labelled "Sum" provides for a given index in column, the number of time it Granger-causes others indices. The entry corresponding to the last row and the last column gives the total number of significant Granger-causality 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 B.7: Results of bilateral tests of Granger causality in 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 left-tail distribution from the index j in column towards the index i in row. Entries with the rejection of the null hypothesis at the 5% nominal risk level are emphasized in bold. The last column labelled "Sum" provides for a given index in row, the number of time it is Granger-caused by the others. Similarly, the last row labelled "Sum" provides for a given index in column, the number of time it Granger-causes others indices. The entry corresponding to the last row and the last column gives the total number of significant Granger-causality 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 B.8: Results of bilateral tests of Granger causality in 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 right-tail distribution from the index j in column towards the index i in row. Entries with the rejection of the null hypothesis at the 5% nominal risk level are emphasized in bold. The last column labelled "Sum" provides for a given index in row, the number of time it is Granger-caused by the others. Similarly, the last row labelled "Sum" provides for a given index in column, the number of time it Granger-causes others indices. The entry corresponding to the last row and the last column gives the total number of significant Granger-causality 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.

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