Evidence of interdependence and contagion using a frequency domain framework

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A R T I C L E   I N F O

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This paper proposes a new measure of contagion, based on the frequency analysis of causality developed recently by Breitung and Candelon [Breitung, J., Candelon, B. 2006. Testing for short and long-run causality: a frequency domain approach, Journal of Econometrics, 12, 363–378]. This approach handles several of the statistical problems identified in the literature. It also permits clear differentiation between temporary and permanent shifts in cross-market linkages: the first case is contagion while the second one is simply a measure of interdependence among markets. With this new approach, we examine the “Tequila” and Asian crises and find evidence of contagion for both. During the Asian crisis, higher interdependence has also contributed to the diffusion of the crisis in Asia.

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1. Introduction

The international financial crises of the last decade have shown that financial shocks in one country can have rapid and large impacts in other countries. In recent years, numerous papers have examined the issue of whether contagion was responsible for this strong linkage among markets during periods of crisis. Measuring financial contagion however poses several problems.

One problem is that economists disagree on what contagion exactly is. The concept of contagion is inherited from the medical vocabulary and indicates the transmission of a contagious disease. The translation to an economic concept is not straightforward, as illustrated by the numerous definitions of contagion that can be found on the World Bank’s website. Several authors, among others Rigobon (2000) and Forbes and Rigobon (2002), define contagion as a significant and temporary increase in cross-market linkages after a shock.
Contagion can take place both across markets, for instance between the foreign exchange market and the stock market, and across countries. This concept of contagion is often labeled “shift-contagion”. Shift-contagion can be generated by multiple equilibria based on investor psychology, endogenous-liquidity shocks causing a portfolio reshuffling in exchange rate regimes (see Rigobon (2000) for a survey). For other authors, contagion is simply the cross-country or cross-market transmission of shocks, no matter whether the linkages are reinforced or not. These authors are generally concerned with the identification of the channels through which shocks are transmitted. The most important channels are the trade channel (Glick and Rose, 1998), the financial channel (Van Rickenghem and Weder, 2001), similarities between economies (Eichengreen, Rose and Wyplosz, 1996), policy coordination or geographical proximity (Bayoumi et al., 2003). In the literature, this approach of contagion is often referred to as “pure” or “fundamental based” contagion.

The remainder of this study will focus solely on the aforementioned “shift-contagion”. The term “contagion” is therefore used to describe a temporary and significant shift in cross-market linkages. It may occur that the shift in cross-market linkages after a shock is permanent rather than temporary. This paper will refer to this situation as a change in “market interdependence”. Therefore the terms “contagion” and “interdependence” describe two markedly different phenomena.

Measuring financial contagion also poses several statistical problems, as shown for instance by Forbes and Rigobon (2001) in several papers. A variety of econometric techniques have been used to measure contagion. An intuitive and widely used technique has been to test whether the correlation between two markets was significantly higher during the period following the crisis compared to the period preceding the crisis. For example, King and Wadhwani (1990) show that the cross-market correlation between the U.S., U.K and Japan has significantly increased after the U.S. stock market crash in 1987. Calvo and Reinhart (1995), Baig and Goldfajn (1998) use a similar approach to show the presence of shift-contagion after the 1994 Mexican peso crisis and the 1997 Asian crisis. Nevertheless, this intuitive approach has several shortcomings. First, correlation is a static measure, so it cannot account for the fact that linkages between markets can vary over time. Second, correlation automatically increases during periods of high volatility and during periods of globalization. Hence, it may turn out that a significant shift in the correlation coefficient after a crisis has nothing to do with contagion. Third, correlation is a symmetrical measure: an increase in the correlation between markets i and j does not give any information on the direction of the contagion (from i to j, from j to i, or both). Contagion, in fact, has a clearly asymmetric dimension. It is for all these reasons that several other approaches have been used to measure cross-market linkages: Forbes and Rigobon (2002) and Corsetti et al. (2002) use a principal component model and build a test robust to heteroscedasticity (i.e. volatility changes). Candelon et al. (2005) use the concept of common feature to measure time-varying linkages among markets.

The contribution of this paper consists of using existing causality tests in the frequency domain\footnote{see Geweke, 1982, Yao and Hosoya, 1998 and Breitung and Candelon, 2006.} to detect whether the strength of asset market linkages is altered by a financial crisis. This approach handles, in a unified framework, the problems identified above. It also permits a clear differentiation between temporary and permanent shifts in cross-market linkages, i.e. contagion and interdependence.

The rest of the paper is organized as follows. Section 2 presents our new measure of contagion, as well as its empirical testing procedure. A simulation analysis is performed to analyze the robustness of the causality test in the frequency domain with respect to changes in volatility. In Section 3, we use our approach to test for the existence of contagion among several stock markets in Latin America and in Asia during the financial crises of 1994 and 1997. Section 4 concludes.

2. A new approach of contagion

2.1. Measuring contagion using causality in the frequency domain

The first feature of our approach is to propose a test of contagion based on causality measure rather than on contemporaneous correlation coefficients. This presents several advantages. First of all, as causality test is performed in a dynamic set-up (generally a Vector AutoRegression, VAR), it accounts for the propagation of shocks over time. Second, provided that the VAR is correctly specified, our approach is free from the
omitted variable problem encountered in papers using contemporaneous correlation. Third, causality allows for the asymmetric dimension of contagion.

The other important feature of our approach is to measure causality in the frequency domain. Such a framework allows us to discriminate between contagion and interdependence. To illustrate this point, assume that \( x_i \) and \( x_j \) are two asset returns in countries i and j, each return being composed of a permanent or long-run term (\( \beta \)) and a transitory or short-run term (\( \alpha \)). Stronger linkages between the two returns could be due either to a higher co-movement between the permanent components of the returns, or to a higher co-movement between their short-run components. There will be contagion only in the latter case; contagion is therefore measured by a stronger linkage among the short-run components of the two returns after a crisis. In the former case, as the shift in cross-market linkages is permanent, what is measured is not shift-contagion but a higher integration of markets. Simply computing correlations, even causality measures, without distinguishing between short- and long-run components will therefore only provide spurious measures of contagion.\(^2\) In a frequency domain approach however, each frequency corresponds to a particular component of the variable: components at low frequencies are more persistent than components at high frequencies. In particular, frequency 0 corresponds to a permanent component. Thanks to this frequency discrimination, we can isolate whether the increase in cross-market linkages is due to long-run (low frequency) or short-run (high frequency) components. Only the latter case corresponds to contagion.

2.2. Causality in the frequency domain: a test

Our new measure of contagion and interdependence is an application of the causality test in the frequency domain recently developed by Breitung and Candelon (2006). The usual definition of causality is due to Granger (1969) and is based on the forecast variance. To illustrate this, let us consider \( z_t = [x_t, y_t]' \) to be a two-dimensional vector of time series observed at \( t = 1, \ldots, T \). In our application, \( x_t \) and \( y_t \) will be equity returns in two different countries, with one of the two countries being the originating country where the crisis started. It is assumed that \( z_t \) has a finite order vector autoregressive (VAR) representation of the form:

\[
\Theta(L)z_t = \varepsilon_t, \tag{1}
\]

where \( \Theta(L) = I - \Theta_1 L - \cdots - \Theta_p L^p \) is a 2 x 2 lag polynomial with \( L^k z_t = z_{t-k} \). We assume that the error vector \( \varepsilon_t \) is \( \Sigma \), where \( \Sigma \) is positive definite. For ease of exposition, we do not include any deterministic terms in (1) although in empirical applications the model typically includes a constant. Here, \( y_t \) is Granger causal for \( x_t \) if the forecast variance of \( x_{t+1} \) conditional on \( x_t, x_{t-1}, \ldots \) is larger than forecast variance of \( x_{t+1} \) conditional on \( x_{t+1} \). In other words \( Y_t \) contains information to predict the one-step ahead value of \( x_t \).

The extension of this framework in the frequency domain has been proposed by Geweke (1982) and Hosoya (1991). Let \( G \) be the lower triangular matrix of the Cholesky decomposition \( G'G = \Sigma^{-1} \) such that \( E(\eta_t \eta_t') = I \) and \( \eta_t = Gz_t \). If system (1) is assumed to be stationary, the MA representation of the system is

\[
z_t = \phi(L)\varepsilon_t = \begin{bmatrix} \Phi_{11}(L) & \Phi_{12}(L)2 \\ \Phi_{21}(L) & \Phi_{22}(L) \end{bmatrix} \begin{bmatrix} \varepsilon_{1t} \\ \varepsilon_{2t} \end{bmatrix} = \Psi(L)\eta_t = \begin{bmatrix} \Psi_{11}(L) & \Psi_{12}(L)3 \\ \Psi_{21}(L) & \Psi_{22}(L) \end{bmatrix} \begin{bmatrix} \eta_{1t} \eta_{2t} \end{bmatrix}, \tag{2}
\]

where \( \phi(L) = \Theta(L)^{-1} \) and \( \Psi(L) = \phi(L)G^{-1} \).

The measure of causality suggested by Geweke (1982) and Hosoya (1991) is the following:

\[
M_{x \rightarrow y}(\omega) = \log \left[ 1 + \frac{|\Psi_{12}(e^{-i\omega})|^2}{|\Psi_{11}(e^{-i\omega})|^2} \right]. \tag{3}
\]

Several methods have been proposed to test for the null hypothesis of \( |\Psi_{12}(e^{-i\omega})| = 0 \), corresponding to the case where \( y \) does not cause \( x \) at frequency \( \omega \).
Breitung and Candelon (2006) propose a new and very simple approach to test for the null hypothesis of non-causality (i.e. $12(e^{-i\omega})| = 0^3$, using

$$\Psi_{12}(L) = -g_{22}\Theta_{12}(L)/|\Theta(L)|,$$

where $g_{22}$ is the lower diagonal element of $G^{-1}$ and $|\Theta(L)|$ is the determinant of $\Theta(L)$. It follows that $y$ does not cause $x$ at frequency $\omega$ if

$$|\Theta_{12}(e^{-i\omega})| = \left| \sum_{k=1}^{p} \theta_{12,k}\cos(k\omega) - \sum_{k=1}^{p} \theta_{12,k}\sin(k\omega) \right| = 0.$$

Their empirical procedure consists of testing for these linear restrictions. To simplify the notation, we let $\alpha_j = \theta_{11,j}$ and $\beta_j = \theta_{12,j}$

\begin{align*}
\dot{x}_t &= \alpha_1\dot{x}_{t-1} + \cdots + \alpha_p\dot{x}_{t-p} + \beta_1y_{t-1} + \cdots + \beta_py_{t-p} + \epsilon_{1t}, \\
\dot{y}_t &= -\dot{x}_{t-1} + 0.1y_{t-1} - 0.2y_{t-2} + 0.3y_{t-3} + \epsilon_{2t},
\end{align*}

where $\epsilon_{it} \sim N(0, \Sigma)$, $\Sigma = \begin{bmatrix} 0.5 & 0.2 \\ 0.2 & 0.5 \end{bmatrix}$ and $b_{\omega}(L) = 1 - 2\cos(\omega)L + L^2$. Therefore, at frequency $\omega$, $y$ is not a cause of $x$.

The hypothesis $M_{y \rightarrow x}(\omega) = 0$ is equivalent to the linear restriction

$$H_0 : \quad R(\omega)\beta = 0,$$

where $\beta = [\beta_1, \ldots, \beta_p]^\top$ and

$$R(\omega) = \begin{bmatrix} \cos(\omega) & \cos(2\omega) & \cdots & \cos(p\omega) \\ \sin(\omega) & \sin(2\omega) & \cdots & \sin(p\omega) \end{bmatrix}.$$

This restriction tests that (5) is an ordinary $F$ statistic and is asymptotically distributed as $F(2, T - 2p)$ for $\omega \in (0, \pi)$ such a method can be extended to higher dimensional systems or to cointegrated VARs (see Breitung and Candelon, 2007). Moreover, as indicated by Breitung and Candelon (2006, p.376), the comparison with the causality test in time domain is far from being straightforward.

### 2.3. Simulation study

It is well known that financial variables exhibit specific features such as conditional heteroscedasticity (ARCH) and extreme values (outliers). Before proceeding to the application of the causality test in the frequency domain to our dataset, we need to check the accuracy of the test in presence of these features.

For a simulation purpose, we should consider a data generating process ($DGP_0$) under the null hypothesis of no outlier and no ARCH. $DGP_0$ should have some specific properties. It first should be a bivariate dynamic model.$^5$

Without any loss of generality, we consider here a VAR(3)$.^6$ Moreover, we should be able to control the frequency at which the null hypothesis (non-causality) is not rejected. To this aim, we consider that $y_{t-1}$ is linked to $x_t$ via a

\begin{align*}
\dot{x}_t &= 0.1\dot{x}_{t-1} + 0.3 b_{\omega}(L)y_{t-1} + \epsilon_{1t} \\
y_{t} &= -\dot{x}_{t-1} + 0.1y_{t-1} - 0.2y_{t-2} + 0.3y_{t-3} + \epsilon_{2t},
\end{align*}

where $\epsilon_{it} \sim N(0, \Sigma)$, $\Sigma = \begin{bmatrix} 0.5 & 0.2 \\ 0.2 & 0.5 \end{bmatrix}$ and $b_{\omega}(L) = 1 - 2\cos(\omega)L + L^2$.

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$^3$ For a detailed exposition of the test, the reader should refer to the original paper.

$^4$ Note that $g_{22}$ is positive due to the assumption that $\Sigma$ is positive definite.

$^5$ In the paper, we do not consider higher dimensional systems.

$^6$ Other VAR($p$) processes with a lag order $p > 3$ can be considered instead, leading to the same results.
To investigate the consequences of misspecification for the causality test, we consider three other DGP (data generating process) possibilities possessing one feature usually attributed to financial series.

First, the presence of outliers, representing the crisis itself, might affect the causality test. Lütkepohl (1989) has demonstrated via simulation study that the performance of the Granger-causality test in the time domain is affected by the presence of structural breaks. Two cases are scrutinized here. In the first experiment, we consider DGP 1, which has the same definition as DGP 0 but with one outlier in the middle of the sample for both series. The size of the outlier corresponds to 20 times the variance of the process and thus represents a large shock. In DGP 2, two outliers of similar magnitude located towards the first and the last quarter of the sample are introduced.

Second, we should investigate the performance of our test in the presence of conditional heteroscedasticity. Under conditional heteroscedasticity, OLS estimators are still consistent but lose their efficiency, leading to size distortions for specification tests. As noticed by Rigobon (2000), heteroscedasticity is observable in financial series and leads to the under-acceptance of contagion. The aim of the simulation is to see how much conditional heteroscedasticity affects the causality test in the frequency domain. If so, an adequate correction (i.e., a White heteroscedastic consistent variance-covariance matrix) has to be employed. Conditional heteroscedasticity is introduced in DGP 0 via a multivariate constant conditional correlation GARCH (ccc-GARCH) à la Bollerslev (1990) such that DGP 3 as the following form:

\[ h_{i,t} = \omega_i + \alpha_i \varepsilon_{i,t-1}^2 + \beta_i h_{i,t-1} \] (6)

and \( H_t = D_t C H_t \), where \( D_t = \text{diag} \left( \sqrt{h_{i,t}} \right) \) and \( C = \begin{pmatrix} 1 & 0.5 \\ 0.5 & 1 \end{pmatrix} \).

The residuals are generated according to \( \varepsilon_t = u_t H_t \), where \( \varepsilon_t = (\varepsilon_{1t}, \varepsilon_{2t})' \) and \( u_t \) are independent \( N(0,1) \), and \( H_t \) comes from the Cholesky decomposition \( H_t = H_t H_t'^{-} \). We consider a parametrization \( (0.01, 0.2, 0.89) \) such that the unconditional variance equals one and thus is identical to the model without GARCH. The coefficients fit the models encountered in practice (see Candelon et al., 2005), i.e., with a steep news impact curves. Nevertheless, a modification of these coefficients would not affect the conclusion brought by these simulations.

For the Monte Carlo experiments, we compute the rejection frequencies based on 5,000 replications of the process with sample sizes \( T = 250 \), \( T = 500 \) and \( T = 1,000 \), and consider the 0.01 significance level. Table 1 indicates the results obtained.

---

Table 1: Empirical size analysis.

<table>
<thead>
<tr>
<th>( \omega )</th>
<th>( T = 250 )</th>
<th>( T = 500 )</th>
<th>( T = 1,000 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>( DGP_0 )</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( 3 \pi/4 )</td>
<td>0.011</td>
<td>0.011</td>
<td>0.010</td>
</tr>
<tr>
<td>( \pi/2 )</td>
<td>0.012</td>
<td>0.009</td>
<td>0.010</td>
</tr>
<tr>
<td>( \pi/4 )</td>
<td>0.009</td>
<td>0.011</td>
<td>0.010</td>
</tr>
<tr>
<td>( DGP_1 )</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( 3 \pi/4 )</td>
<td>0.295</td>
<td>0.339</td>
<td>0.265</td>
</tr>
<tr>
<td>( \pi/2 )</td>
<td>0.034</td>
<td>0.067</td>
<td>0.069</td>
</tr>
<tr>
<td>( \pi/4 )</td>
<td>0.614</td>
<td>0.535</td>
<td>0.504</td>
</tr>
<tr>
<td>( DGP_2 )</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( 3 \pi/4 )</td>
<td>0.465</td>
<td>0.649</td>
<td>0.670</td>
</tr>
<tr>
<td>( \pi/2 )</td>
<td>0.032</td>
<td>0.080</td>
<td>0.154</td>
</tr>
<tr>
<td>( \pi/4 )</td>
<td>0.923</td>
<td>0.740</td>
<td>0.693</td>
</tr>
<tr>
<td>( DGP_3 )</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( GARCH(0.01,0.2,0.79) )</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( 3 \pi/4 )</td>
<td>0.014</td>
<td>0.015</td>
<td>0.011</td>
</tr>
<tr>
<td>( \pi/2 )</td>
<td>0.015</td>
<td>0.015</td>
<td>0.010</td>
</tr>
<tr>
<td>( \pi/4 )</td>
<td>0.015</td>
<td>0.015</td>
<td>0.013</td>
</tr>
</tbody>
</table>

Note: Rejection frequencies of 5000 Monte Carlo replications of DGP 1 and DGP 2. The 0.01 significance level is used.

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We consider such a large outlier to give the maximum penalty at the size of the causality test.

Simulations with different GARCH parameters are available from the authors upon request.
It turns out that the size of the test is clearly affected by the presence of one or two large outliers (20 times the variance of the process). In the presence of outliers, the null of non-contagion is too often rejected, and therefore it would fallaciously lead to support for causality. This experiment indicates us that a particular care for outliers has to be done in empirical studies. The simplest advice consists in removing them before performing the causality analysis.

The presence of ccc-GARCH is also investigated via the simulation of $DGP_3$. It turns out that, contrary to the outlier case, the rejection frequency is higher than the nominal size and lies around 6%. The test is thus slightly oversized in the presence of ccc-GARCH. To go deeper, the empirical power of the causality test in the frequency domain in the presence of ccc-GARCH is analysed, by simulating 5000 times $DGP_0$ and $DGP_3$. We consider a sample size of 250 observations for two particular frequencies ($\pi/2$ and $\pi/4$). Rejection frequency is plotted in Fig. 1. It turns out that in the presence of ccc-GARCH, the empirical power has the same shape as in the presence of i.i.d. white noise residuals. A leakage problem, as well as a decrease in the power for frequencies close to 0, are observed. We nevertheless notice that the power of the causality test is always lower in the presence of ccc-GARCH. This experiment indicates that the causality test in the frequency domain is not strongly affected by the presence of ccc-GARCH. Therefore, an empirical study can deal with series exhibiting GARCH process.

To summarize, the causality test in the frequency domain is not sensitive to volatility clusters (ccc-GARCH) but should not be applied on series exhibiting outliers.

### 3. Empirical analysis

The approach developed in the two previous sections is here used to test whether contagion occurred during two famous periods of international financial crisis, the Mexican “Tequila” crisis of 1994 and the Asian “flu” crisis of 1997.

Contagion is examined at the stock market level. We use daily equity data for a sample of eleven emerging countries from Asia and Latin America. The Asian sample includes Hong Kong, Indonesia, Malaysia, the Philippines, South Korea, Taiwan and Thailand; the Latin American sample consists of Argentina, Brazil, Chile, Mexico, and Venezuela, which are the largest economies in the region. All the data are retrieved from Datastream. Datastream stock market indices are all expressed in US dollars, which is usual practice in many studies (see for instance Forbes and Rigobon, 2000; Bekaert et al., 2003; Bae and et al., 2000). Equity market returns are computed through log-differentiation.

For our empirical investigation, we follow Forbes and Rigobon (2001) and calculate two-day rolling over returns ($R_{2,t}$) in order to account for differences in time zones and official holidays among the different countries in the sample. As shown in the simulation part, a proper application of the causality test in the frequency domain necessitates a particular care of outliers. Otherwise, we will fallaciously overestimate the

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9 This choice of emerging countries is usual in studies concerned with recent episodes of financial contagion. See for instance Bekaert, Harvey and Ng (2003), Forbes and Rigobon (2000) and Kaminsky and Reinhart (2001).

10 In some papers, stock market indices are measured in local currency instead of dollars. Bae et al. (2000) and Forbes and Rigobon (2000) find that the choice of the currency denomination does not tend to significantly alter their results.
presence of causality. Therefore, we decided to remove all outliers. In the presence of ccc-GARCH, the most standard and simple approach to detect outliers is the Median Absolute Deviation (MAD) procedure.\footnote{For a more detailed description of the procedure, see Hotta and Tsay (1988).}

According to this procedure, an observation is classified as an outlier if:

\[
R_{2,t} > \gamma \beta \text{med}\left( |R_{2,t} - \text{med}(R_{2,t})| \right),
\]

where \( \text{med} \) is the median operator, \( \beta \) is a constant equal to \((1/q_{0.75})\) where \( q_{0.75} \) is the 75th fractile of the sample distribution of \( R_{2,t} \). The parameter \( y \) is fixed arbitrarily, but a value of 2 or 3 is commonly used in practice. In this paper, \( y \) is set equal to 3.

Each outlier is then replaced by a 10-day average centered around the abnormal observation using:

\[
R_{10x,t} = \frac{1}{9} \sum_{i=-4}^{4} R_{2k,t}
\]

Our empirical work uses bivariate models (as in (1)) composed of the return in the country that is considered to be the source of the crisis (the originating country) and the return in another country, either in Latin America or in Asia. Contagion can therefore occur between countries in a similar or different geographical regions.

For the Tequila crisis of 1994, as the crisis was triggered by the devaluation of the Mexican peso in December 19th, 1994, the originating country is undoubtedly Mexico. Regarding the East Asian crisis, the choice of the country where the crisis originated is not so obvious: in some papers, it is considered that the crisis started with the Thai Baht devaluation on July 2, 1997 while other papers consider that the crisis was triggered by the sharp decline in the Hong Kong stock market in mid-October 1997. In this paper, we consider separately both countries as the originating country.

We estimate each model over a pre- and a post-crisis periods. In order to make our results comparable with those from earlier studies, we take the chronology of the crises from previous studies, more precisely from Forbes and Rigobon (2002). Following Forbes and Rigobon (2002), we fix the Tequila crisis as lasting from December 16th, 1994 (when the exchange rate regime was abandoned) to January 2nd, 1995. Regarding the Asian crisis, taking Hong Kong as the originating country, the crisis period goes from October 16th, 1997 (when the Hong Kong stock market crashed) through November 3rd 1997; alternatively, when we take Thailand as the origin of the crisis, the crisis period goes from July 2nd, 1997 (when the Thai Baht is devaluated) through

\begin{table}[h]
\centering
\caption{Optimal lag length.}
\begin{tabular}{lcccc}
\hline
Origin & Tequila crisis & Asian flu & Asian flu \\
& Mexico & Thailand & Hong-Kong \\
\hline
Pre-crisis & Post-crisis & Pre-crisis & Post-crisis & Pre-crisis & Post-crisis \\
\hline
Argentina & 14 & 8 & 17 & 7 & 20 & 6 \\
Brazil & 11 & 7 & 17 & 4 & 16 & 4 \\
Chile & 13 & 9 & 16 & 9 & 14 & 6 \\
Venezuela & 13 & 3 & 18 & 3 & 20 & 3 \\
Mexico & - & - & 15 & 3 & 20 & 4 \\
Indonesia & 11 & 5 & 17 & 2 & 17 & 4 \\
Korea & 16 & 10 & 16 & 2 & 13 & 8 \\
Malaysia & 13 & 13 & 16 & 2 & 29 & 4 \\
Philippines & 13 & 7 & 16 & 4 & 21 & 4 \\
Taiwan & 13 & 4 & 18 & 4 & 22 & 9 \\
Hong-Kong & 14 & 16 & 17 & 4 & - & - \\
Thailand & 13 & 19 & - & - & 20 & 5 \\
\hline
\end{tabular}
\end{table}

Note: Lag length have been selected using the Akaike information criterion (AIC). For The Tequila crisis, the pre-crisis period is 1/01/1993–16/12/1994 and the post-crisis period 2/01/1995–29/12/1995. For the Asian flu, if Thailand is considered as the originating country, the pre-crisis period is 1/01/1996–02/07/1997 and the post-crisis period is 28/07/1997–31/12/1998. If Hong-Kong is the originating country, the pre-crisis period is 1/01/1996–16/10/1997 and the post-crisis period is 03/11/1997–31/12/1998.
July 28th, 1997 (when Thailand calls the IMF). In line with Forbes and Rigobon (2002), we fix the beginning of the period preceding the crisis on January 1st, 1993 for the Mexican Peso crisis, and on January 1st, 1996 for the Asian crisis, no matter whether the originating country is Hong-Kong or Thailand. Finally, we define the period following the crisis as starting on the last day of the crisis period through the end of the year following the crisis. Precisely, the estimation periods are the following ones: (i) Tequila crisis: pre-crisis = 1/01/1993 to 16/12/1994; post-crisis = 2/01/1995 to 29/12/1995; (ii) Asian crisis (Hong-Kong = originating country): pre-crisis = 1/01/1996 to 16/10/1997; post-crisis = 03/11/1997 to 31/12/1998; (iii) Asian crisis (Thailand = originating country): pre-crisis = 1/01/1996 to 02/07/1997; post-crisis = 28/07/1997 to 31/12/1998.

In Table 2, we report the optimal lag length of each bivariate system for the different sub-periods, having used the AIC information criteria. It is well known that this information criterion slightly overestimates the optimal lag length. By taking the highest dimension of the dynamic structure, we build a conservative causality test, rejecting as often as possible the causality hypothesis as well as the contagion one.

Our results are presented in Tables 3 and 4. We report the results of the non-causality test at 1% (Table 3) and 5% (Table 4) empirical size for the pre- and post-crisis period. Panel A contents the results at high frequencies. In Panel B, Yes (resp. No) indicates that the null of no causality is rejected (resp. not rejected) in the neighbourhood of $\omega = 0$ considered here as $\omega = [0,0.1]$. A Yes supports the existence of interdependence.

<table>
<thead>
<tr>
<th>Origin</th>
<th>Tequila crisis</th>
<th>Asian flu</th>
<th>Asian flu</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td>Pre-crisis</td>
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<tr>
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<tr>
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</table>

Note: In Panel A, Yes (resp. No) indicates that the null of no causality is rejected (resp. not rejected) for at least a frequency $\omega = [2\pi/3, \pi]$. Cases in bold indicate when causality is not rejected at $\omega = [2\pi/3, \pi]$ for the post-crisis period, whereas it is rejected for the pre-crisis period; contagion is thus supported. In Panel B, Yes (resp. No) indicates that the null of no causality is rejected (resp. not rejected) in the neighbourhood of $\omega = 0$ considered here as $\omega = [0,0.1]$. A Yes supports the existence of interdependence.

Figures representing the test statistics at each frequency $\omega = [0,\pi]$ are not reported to save space, but can be checked in an appendix at http://www.personeel.unimaas.nl/b.candelon/bc.htm.
ω ∈ [2π/3, π]. This frequency range is somewhat arbitrary, but it is acknowledged that the transmission of shocks among equity markets is very fast (it can spread from one equity market to the other equity markets during the same day) and generally does not exceed half a week. For instance, using impulse response analysis, Baig and Goldfjan (1988) find that during the Asian crisis, the impact on neighboring markets of shocks originating from Thailand’s stock market disappeared after about 4 days. When non causality is not rejected (resp. rejected), a “No” (resp. “Yes”) is reported. As explained before, there is evidence of shift-contagion if non causality is rejected at high frequencies (i.e. “Yes”) for the post-crisis period, whereas it is rejected for the pre-crisis period (i.e. “No”). Similarly, in Panel B of Tables 3 and 4, we report the results of the causality tests at frequencies around 0 (we consider \( \omega \in [0,0.1] \)). A rejection (resp. non rejection) is indicated by “No” (resp. “Yes”). A “Yes” supports the existence of economic integration.

For each system, we eliminated outliers using the MAD algorithm. We can then assume that the residuals are free from autocorrelation and outliers, and thus that the models are correctly specified.

### 3.1. Contagion in Latin America

Evidence for contagion after the Mexican crisis is found in three countries, namely Argentina (at 5%), Brazil and Chile (at 1%). For these countries, there is at least one range of frequencies within the high frequencies

\[ \omega = [2\pi/3, \pi] \] 

... and the frequency \( (\omega) \) is obtained via \( \omega = \frac{2\pi}{cp} \).

---

The correspondence between the component periodicity \( (cp) \) and the frequency \( (\omega) \) is obtained via \( \frac{2\pi}{cp} = \omega \).

Results are available from the authors upon request.

---

### Table 4

Evidence of contagion and interdependence (at a nominal size of 5%).

<table>
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<tr>
<th>Origin</th>
<th>Tequila crisis</th>
<th>Asian flu</th>
<th>Asian flu</th>
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<td><strong>Panel A: Evidence of contagion</strong></td>
<td><strong>Panel B: Evidence of interdependence</strong></td>
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<td></td>
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<tr>
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<td>–</td>
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</tbody>
</table>

Note: In Panel A, Yes (resp. No) indicates that the null of no causality is rejected (resp. not rejected) for at least a frequency \( \omega \in [2\pi/3, \pi] \). Cases in bold indicate when causality is not rejected at \( \omega = [2\pi/3, \pi] \) for the post-crisis period, whereas it is rejected for the pre-crisis period; contagion is thus supported. In Panel B, Yes (resp. No) indicates that the null of no causality is rejected (resp. not rejected) in the neighbourhood of \( \omega = 0 \) considered here as \( \omega \in [0,0.1] \). A Yes supports the existence of interdependence.
window defined above ($\omega \in [2.2, 2.8]$ for Chile, $\omega \in [2.2, 2.8]$ for Brazil and $\omega \in [2.2, 2.2]$ for Argentina), at which causality is not rejected for the post-crisis period, whereas it is rejected for the pre-crisis period. This indicates that the linkages between these countries and Mexico did indeed increase after the Tequila crisis, supporting the idea of “shift-contagion”. In the case of Venezuela, causality at high frequencies is rejected for the post-crisis period, which suggests that the country was not contagiously affected by the Tequila crisis. Our results partly differ from those reported by Forbes and Rigobon (2002): while these authors conclude that there has been no shift-contagion in Latin America during the Tequila crisis, we find however that there has been shift-contagion from Mexico to at least three Latin American countries. It turns out however that causality test performed in the time domain as it is usually done, matches Forbes and Rigobon’s (2002) conclusions. It is also worth noting that long-run interdependence between Mexico and the other Latin American countries has not increased after the crisis. Instead, we observe that when there was interdependence before the crisis, it has disappeared after the crisis (see Argentina at 1% and Venezuela 5%).

Panel A of Tables 3 and 4 shows that the Asian crisis had almost no spillover effect in Latin America, whichever the selected originating country. We can only detect support for contagion in Argentina at 5%, if Hong-Kong is the originating country. This result indicates that contagion occurs mainly within a region, rather than across regions, as it has already been documented in Glick and Rose (1999) and in Kaminsky and Reinhart (2000).

3.2. Contagion in Asian countries

Regarding the Asian flu and its impact in Asia, shift-contagion is also detected. When the Thai Baht devaluation is assumed to be at the origin of the crisis, our analysis provides evidence for contagion from Thailand to Indonesia (at 1%), Taiwan and the Philippines (at 5%). Alternatively, if we consider that the Asian crisis was triggered by the crash of the Hong Kong stock market, our causality test indicates contagion from Hong Kong to the Philippines (at 1%) and Malaysia (at 5%). It is interesting to point out that, with the exception of the Philippines, the set of countries contagiously affected by the Asian flu differs whether the originating country is Thailand or Hong Kong. It also appears from Panel B of Tables 3 and 4 that for the set of countries affected by contagion, higher long-run interdependence with the originating country is also detected after the crisis. This result suggests that both shift-contagion and higher interdependence among equity markets contributed to the transmission of the crisis from Hong Kong or Thailand to the other Asian countries. This feature distinguishes the Asian flu from the Tequila crisis, for which shift-contagion was not associated with higher interdependence.

With respect to spillovers of the Tequila crisis in Asia, it is found that apart from the Philippines (at 5%), the contagion to Asian countries was weak. This result suggests once again that contagion is mainly a regional phenomenon.

4. Conclusion

The international financial crises of the last decade have shown that financial shocks in one country can have rapid and large impacts in other countries. This phenomenon revived the literature on contagion, with a surge of papers investigating whether contagion is responsible for this strong linkage among markets during periods of crisis. Measuring financial contagion is not an easy task, because of both conceptual and statistical problems.

In this paper, contagion is defined as a temporary and significant increase in cross-market linkages after a shock. We then propose a new measure of contagion using the causality test in the frequency domain proposed by Breitung and Candelon (2006). This approach has two main advantages over existing methods of measuring contagion. First, it provides an elegant way to deal with several of the statistical problems identified in the literature in a unified framework. Second, it permits clearly differentiation between temporary and permanent shifts in cross-market linkages: the first case is contagion, while the second is simply a measure of interdependence among markets.

15 The standard Granger-causality test corresponds to a causality test performed on the whole range of frequencies. Results of this test for the different systems are available from authors upon request.
With this new approach, we test for the existence of contagion among several stock markets in Latin America and Asia during the international financial crises of 1994 and 1997. Our paper provides three main results. While several studies using a time series framework reject the existence of contagion, we find support for contagion during the two crises. In addition, our approach highlights that during the Asian crisis, both contagion and higher interdependence were responsible for the stronger linkages across markets. Such a feature is not observed during the Tequila crisis. Finally, it appears that the spillover effects of these crises have been geographically confined to the region where the shock occurred. This supports the view that contagion is more regional than global, as already suggested by Glick and Rose (1999) and Kaminsky and Reinhart (2000). These three results suggest that causality in the frequency domain is a proper framework for studying contagion.

In this paper, we confine our analysis to bivariate models neglecting a possible third country effect (a shock originating in country \( i \) could affect country \( j \) indirectly via country \( k \)). Fruitful extension of our approach would consist in considering higher dimensional systems integrating indirect transmission effect.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.ememar.2008.11.003.

References