CHAPTER 9

Testing for Causality: A Survey of the Current Literature

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Chapter Outline

1 Introduction

Many tests of the Granger-type causality have been derived and implemented to test the direction of causality for time series (Granger, 1969; Geweke et al., 1983; Sims, 1972). These tests are based on investigating the validity of null hypotheses and are formulated as zero restrictions on the coefficients of the lags of a subset of the variables. The goal of this chapter is to provide a review survey on the most significant contributions to the literature in relevance to causality testing.

2 Bivariate Causality Tests in Time Series

2.1 Causality Tests Without Cointegration

With the aim of testing for the presence of bivariate causality, first, we need to introduce a Vector Autoregressive (VAR) model. In particular, we introduce a bivariate VAR model with

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two-period lag (actually, the number of appropriate lags must be determined through an informational optimization criterion, such as the Akaike criterion). We assume the presence of two endogenous variables y_t and x_t:

$$y_t = a_{10} + b_{11}y_{t-1} + b_{12}y_{t-2} + b_{13}x_{t-1} + b_{14}x_{t-2} + \varepsilon_{y_t}$$
$$x_t = a_{20} + b_{21}y_{t-1} + b_{22}y_{t-2} + b_{23}x_{t-1} + b_{24}x_{t-2} + \varepsilon_{x_t}$$

where ε_s are the error terms in the two equations. In matrix form, the above model yields:

$$\begin{pmatrix} y_t \\ x_t \end{pmatrix} = \begin{pmatrix} a_{10} \\ a_{20} \end{pmatrix} + \begin{pmatrix} b_{11} & b_{12} \\ b_{21} & b_{22} \end{pmatrix} \begin{pmatrix} y_{t-1} \\ x_{t-1} \end{pmatrix} + \begin{pmatrix} b_{13} & b_{14} \\ b_{23} & b_{24} \end{pmatrix} \begin{pmatrix} y_{t-2} \\ x_{t-2} \end{pmatrix} + \begin{pmatrix} \varepsilon_{y_t} \\ \varepsilon_{x_t} \end{pmatrix}$$

Needless to say, before any testing for causality, unit root testing should have identified whether the variables *x* and *y* should be in levels or in first differences.

The variable *x* does not cause the variable *y*, if the null hypothesis H_0: $b_{13} = b_{14} = 0$ cannot be rejected. Similarly, the variable *y* does not cause the variable *x*, if the null hypothesis H_0: $b_{21} = b_{22} = 0$ cannot be rejected. The causality tests are performed in both directions through a system of equations. To test the above null hypotheses, we employ Wald tests, in the same fashion as a restriction of the null hypothesis would be tested with the assistance of the *F*-test. If we reject the null hypothesis, then causality is established. The *F*-statistic is calculated as follows:

$$F = \left[\mathrm{SSE}_0 - \mathrm{SSE}\right] / \mathrm{SEE} \times \left[N - 2k - 1\right] / k$$

where SSE_0 denotes the sum of squared residuals for a model with restrictions ($b_{13} = b_{14} = b_{21} = b_{22} = 0$), SSE denotes the sum of squared residuals for the model without any restrictions, *k* is the number of lags, and *N* denotes the number of observations. Under the null hypothesis, the *F*-statistic follows an *F*-distribution with *k* degrees of freedom in the numerator and $N - 2k - 1$ degrees of freedom in the denominator (Box 9.1).

2.2 Causality Tests With Cointegration

In the case where the variables *y* and *x* are cointegrated, an Error Correction VAR (ECVAR) model should be employed. The ECVAR model can distinguish the long- and short-run relationship between the variables and can identify sources of causality that cannot be detected by the usual Granger causality test. In particular:

$$y_t = a_{10} + b_{11}\, y_{t-1} + b_{12}\, y_{t-2} + b_{13}\, x_{t-1} + b_{14}\, x_{t-2} + c_1\, z_{t-1} + \varepsilon_{y_t}$$
$$x_t = a_{20} + b_{21}\, y_{t-1} + b_{22}\, y_{t-2} + b_{23}\, x_{t-1} + b_{24}\, x_{t-2} + c_2\, z_{t-1} + \varepsilon_{x_t}$$

where *z*s represent the residuals from the cointegrating vector (i.e., the error-correction term). These terms also represent the speed of adjustment, namely the speed of returning

BOX 9.1 A Published Case Study

The STUDY: Sbaouelgi Jihène and Boulila Ghazi, 2013: The causality between income inequality and economic growth: empirical evidence from the Middle East and North Africa region. Asian Economic and Financial Review 3, 668–682.

Data

They use four indicators of income inequality: (1) the Gini index, (2) the openness rate, (3) the secondary school enrolment rate, and (4) the gross fixed capital formation as a percentage of GDP. Concerning economic growth, the growth rate of GDP per capita is employed, with the data sources being the Word Development Indicators database from the World Bank (2011). Nine MENA countries are used, spanning the period 1960–2011 on an annual basis.

Causality results

Table 9.1: Short-run Granger causality tests.

Countries and Variables	Null Hypothesis	
	Income Inequality → Growth	Growth → Income Inequality
Algeria		
GDP, Gini	0.435	0.210
GDP, GCF	1.442	0.854
GDP, Openness	1.001	0.664
GDP, School	0.331	0.008
Egypt		
GDP, Gini	0.526	0.052
GDP, GCF	14.565**	0.025
GDP, Openness	4.625	1.063
GDP, School	0.630	1.536
Jordan		
GDP, Gini	0.185	0.032
GDP, GCF	0.740	0.027
GDP, Openness	1.705	0.236
GDP, School	2.506	1.131
Mauritania		
GDP, Gini	0.277	0.407
GDP, GCF	0.119	0.017
GDP, Openness	1.204	1.454
GDP, School	2.172	8.596**

** $P \leq 0.05$. All variables are expressed in first differences in logs.

Causality conclusions

According to the findings in Table 9.1, the evidence illustrates that causation is present in the case of Egypt where it turns out to be unidirectional, running from income inequality to growth, while in the case of Mauritania it runs from growth to income inequality. These results hold at the 5% significance level.

to the equilibrium path when a shock hits the two variables. In this new case, causality can be tested both on short-run and long-run bases. In terms of the short-run analysis, the procedure is similar to the case shown in Section 1.1. In terms of the long-run analysis, it is the statistical significance of coefficients c_1 and c_2 that determines the presence of long-run causality. More specifically, if the coefficient c_1 turns out to be statistically significant, then the variable x causes the variable y in the long run. Similarly, if the coefficient c_2 turns out to be statistically significant, then that variable y causes the variable x in the long run as well.

Overall, the term "causality" does not mean that one variable causes the other variable, but it simply means that there is a correlation between the informational content of one variable and the previous values of another variable. Causality does not provide information, however, on the sign of the impact or how long it will last. However, Dufour and Renault (1998) are the first to present a theoretical framework, referred as long (or short) horizon noncausality, which allows researchers to disentangle potentially different causality relations over different forecast horizons. They provide definitions and a set of conditions which ensure the equivalence between standard Wiener–Granger type one-step-ahead noncausality and noncausality at any forecast period. Their methodology on the conditions on noncausality between two variables at a forecast horizon greater than one involves examining the statistical significance of multilinear zero restrictions on the coefficients of the VAR parameters. However, more details on this methodology are provided later in the text (Box 9.2).

BOX 9.2 A Published Case Study

The STUDY: Wadjamsse B. Djezou, 2014. The democracy and economic growth nexus: empirical evidence from Côte d'Ivoire. The European Journal of Comparative Economics 11, 251–266.

Data

Data are on an annual basis, spanning the period from 1960 to 2012. The economic performance is proxied by real GDP per capita, while data are obtained from the World Bank's World Development Indicators database. Data on democracy and regime durability were obtained from the Polity IV database. Regime durability indicates the number of years as the last substantive change in authority characteristics, that is, a measure of the durability of the regime's authority pattern. It is defined as a 3-point move in a country's democracy score. Democracy scores are based on the premise that a mature and internally coherent democracy is an institutional framework in which a political participation is unrestricted, that is, open and fully competitive, an executive recruitment is elective, and the constraints on the chief executive are substantial. Higher values represent stronger democracy with a range of −10 to 10.

Causality results

Table 9.2: Granger causality results.

	Source of Causation		
	Short-run Causality		
Dependent Variable	**Democracy Score**	**GDP**	**Long-run Causality**
Democracy score	–	0.426[0.65]	−3.52[0.00]***
GDP	0.068[0.93]	–	

The null hypothesis is of no causality. All variables are expressed in first differences. Figures in brackets denote *P*-values. *** $P \leq 0.01$.

Causality conclusions

The results of causality tests are reported in Table 9.2. They document that there is a long-run causality running from GDP to democracy as the estimated coefficient of the lagged error correction term is not only negative, but also statistically significant in the democracy equation. This is quite a fundamental result as it highlights the economic performance of the country that impacts its democratic status and not the reverse. In other words, past information on a country's economic performance does permit a better prediction of the level of democratization in that country. This suggests that poor countries should first of all eliminate poverty before discussing about political freedom (or election). Thus economic growth, through strong institutions, is a precondition for democratization.

3 Multivariate Causality Tests in Time Series

3.1 Causality Without Cointegration

Next, we extend the bivariate causality tests into the multiple variable setting in the VAR scheme. For $t = 1,\ldots, T$, the n-variable VAR model can be represented as follows:

$$\begin{pmatrix} x_{1t} \\ x_{2t} \\ \vdots \\ x_{nt} \end{pmatrix} = \begin{pmatrix} a_{10} \\ a_{20} \\ \vdots \\ a_{n0} \end{pmatrix} + \begin{pmatrix} b_{11}(L) & \ldots & b_{1n}(L) \\ b_{21}(L) & \ldots & b_{2n}(L) \\ & \vdots & \\ b_{n1}(L) & \cdots & b_{nn}(L) \end{pmatrix} \begin{pmatrix} x_{1(t-1)} \\ x_{2(t-1)} \\ \vdots \\ x_{n(t-1)} \end{pmatrix} + \begin{pmatrix} \varepsilon_{1t} \\ \varepsilon_{2t} \\ \vdots \\ \varepsilon_{nt} \end{pmatrix}$$

where $(x_{1t},\ldots, x_{nt})$ is the n-variable vector stationary time series at time t, L is the backward operation, where $L(x_t) = x_{t-1}$, a_{i0} are intercept parameters, and $b_{ij}(L)$ are polynomials in the lag operator L:

$b_{ij}(L) = b_{ij}(1)L + b_{ij}(2)L^2 + \ldots + b_{ij}(p)L^p$ and $\varepsilon_t = (\varepsilon_{1t},\ldots, \varepsilon_{nt})'$ is the disturbance vector obeying the assumptions of the standard linear regression model.

Each equation in the VAR model is expected to have the same lag length for each variable and the regressors are identical in all equations. In other words, a uniform order p is chosen across all the lag polynomials $b_{ij}(L)$ in the VAR model according to a certain criterion, such as the Akaike's Information Criterion (AIC). To test causality, that is, with causality running from x_{2t} to x_{1t}, with the null hypothesis pertaining that the terms $b_{ij}(L)$ are significantly different from zero. Similarly, this applies for the remaining causality cases. First, we run regressions for each equation without any restrictions on the parameters, and we obtain the residual covariance matrix represented with the symbol Σ. Next, we run the regressions with the restriction imposed by the null hypothesis and obtain the restricted residual covariance matrix represented as Σ_0. Then, we get the likelihood ratio statistics suggested by Sims (1980):

$$(T-c)(\log|\Sigma_0|-\log|\Sigma|)$$

where T is the number of observations, c is the number of parameters estimated in each equation of the unrestricted system, and $\log|\Sigma_0|$ and $\log|\Sigma|$ are the natural logarithms of the determinant of restricted and unrestricted residual covariance matrix, respectively. This test statistic follows an asymptotic χ^2 distribution with the degrees of freedom being equal to the number of restrictions on the coefficients in the system.

3.2 Causality With Cointegration

In case of the presence of cointegration across the multiple variables, the causality test involves specifying a multivariate pth order vector error correction model (VECM) as follows:

$$\begin{pmatrix} x_{1t} \\ x_{2t} \\ \vdots \\ x_{nt} \end{pmatrix} = \begin{pmatrix} a_{10} \\ a_{20} \\ \vdots \\ a_{n0} \end{pmatrix} + \begin{pmatrix} b_{11}(L) & \dots & b_{1n}(L) \\ b_{21}(L) & \dots & b_{2n}(L) \\ & \vdots & \\ b_{n1}(L) & \dots & b_{nn}(L) \end{pmatrix} \begin{pmatrix} x_{1(t-1)} \\ x_{2(t-1)} \\ \vdots \\ x_{n(t-1)} \end{pmatrix} + \begin{pmatrix} c_1 z_{t-1} \\ c_2 z_{t-1} \\ \vdots \\ c_n z_{t-1} \end{pmatrix} + \begin{pmatrix} \varepsilon_{1t} \\ \varepsilon_{2t} \\ \vdots \\ \varepsilon_{nt} \end{pmatrix}$$

where again z_{t-1} is the lagged error-correction term derived from the long-run cointegrating relationship. The presence of short-run causality is investigated again through the employment of Wald testing in which if the null hypothesis is rejected, then causality is established. However, it is not easy to test for long-run causality just by looking at the significance of the error-correction term.

It is also worth mentioning here (this also applies in the bivariate case) that Pesaran and Pesaran (1997) recommended the cumulative sum of recursive residuals (CUSUM) and the CUSUM of square (CUSUMSQ) tests proposed by Brown et al. (1975) to assess the parameter constancy. Moreover, Hansen (1992) recommended three tests for parameter stability: the supremum F test (SupF), the Mean F test (MeanF), and L_C. These tests have the same null hypothesis that the parameters are stable. When the calculated probability values are greater than 0.05, then the null hypothesis is accepted (Box 9.3).

BOX 9.3 A Published Case Study

The STUDY: Dritsakis, N., Varelas, E., Adamopoulos, A., 2006. The main determinants of economic growth: an empirical investigation with Granger causality analysis for Greece. European Research Studies IX, 47–58.

Data

Annual data on real GDP (adjusted by the GDP deflator, real gross fixed capital formation, adjusted by the GDP deflator, and exports, measured by the real revenues of exports are obtained from the International Financial Statistics database. In addition, FDI data, measured by foreign direct investments adjusted by the GDP deflator, are also obtain from the World Bank database, spanning the period 1960–2002, while all variables measured in logarithms.

Granger causality results

Given the presence of cointegration, the analysis tests for Granger causality through the error-correction mechanism (Table 9.3).

Granger causality conclusions

There is a bidirectional causal relationship between foreign direct investments and real GDP, a unidirectional causal relationship between exports and real GDP, running from exports to real GDP, and finally, a unidirectional causal relationship between investments and real GDP.

Table 9.3: Granger causality results.

Dependent Variable	Testing Hypothesis	*F*-Test
GDP		
	Exports do not cause GDP Investments do not cause GDP FDI do not cause GDP	40.323[0.00] 32.894[0.00] 36.171[0.00]
Exports		
	GDP does not cause Exports	1.970[0.42]
Investments		
	GDP does not cause Investments	33.652[0.00]
FDI		
	GDP does not cause FDI	31.895[0.00]

Figures in brackets denote *P*-values. All variables are expressed in differences in logs. *** $P \leq 0.01$.

4 Alternative Causality Test Approaches in Time Series

Toda and Yamamoto (1995) and Dolado and Lutkepohl (1996) proposed a methodological approach that is applicable irrespective of the integration and cointegration properties of the system of the involved variables. This methodology involves using a modified Wald statistic for testing the significance of the parameters of the VAR model. The estimation of a VAR($s + d_{\max}$) guarantees the asymptotic χ^2 distribution of the Wald statistic, where s is the lag length in the system and $d_{\max}$ is the maximal order of integration in the model. As lagged dependent variables appear in each equation of the aforementioned causal models, their presence is expected to purge serial correlation among the error terms. The traditional F tests and its Wald test counterpart to determine whether some parameters of a stable VAR model are jointly zero, are not valid for nonstationary processes, as the test statistics do not have a standard distribution (Toda and Phillips, 1994).

Hill (2007) introduced a sequential multihorizon noncausality test. His testing approach is based on Wald type test statistics under the null hypothesis of joint zero parameter linear restrictions. He introduces a VAR framework of order p at horizon h. This framework yields:

$$Z_{t+h} = a + \sum_{k=1}^{p} 1_k^{(h)} Z_{t+1-k} + v_{t+h}$$

where Z is an m-vector stationary process with $m \geq 2$, $1_k^{(h)}$ are matrix-valued coefficients, v is a zero mean $m \times 1$ vector of a white noise process, and a is the intercept. Causality occurs at any horizon if it occurs at horizon 1. In the case that some or all variables are nonstationary, the above equation is extended in the Toda and Yamamoto (1995) model by adding d extra lags to the VAR process. Then, a Wald testing procedure of linear zero restrictions is employed to test for 1-step ahead noncausality. Hill (2007) has also developed a parametric bootstrap methodology that simulates small sample P-values.

Finally, we examine causality through the generalization proposed by Dufour et al. (2006). Given that certain studies have displayed that, in multivariate models, where a vector of auxiliary variables Z is used in addition to the variables of interest x_1 and x_2, it is possible that x_1 does not cause x_2 in the Granger sense (one period ahead), but can still help to predict x_2 several periods ahead. Such a generalization allows for the possibility of distinguishing between short-run and long-run causalities. The statistical procedure in Dufour et al. (2006) tested noncausality at various horizons in the context of finite-order VAR models. In such models, the noncausality restrictions at horizon 1 take the form of relatively simple zero restrictions on the coefficients of the VAR. However, at higher horizons, noncausality restrictions are generally nonlinear, taking the form of zero restrictions on multilinear forms in the coefficients of the VAR. When applying standard test statistics, such as Wald-type criteria, such forms can easily lead to asymptotically singular covariance matrices, so that the

standard asymptotic theory would not apply to such statistics. Consequently, they recommend simple tests that can be implemented only through linear regression methodologies. These tests are based on considering multiple-horizon VAR, that is, $(p; h)$-autoregressions, where the parameters of interest can be estimated by linear approaches. Restrictions of noncausality at various horizons may then be tested through simple Wald-type criteria after taking into account the fact that such autoregressions involve autocorrelated errors that are orthogonal to the regressors. The correction for the presence of autocorrelation in the errors may then be performed by using a heteroscedastic autocorrelation consistent covariance matrix estimator. Given the presence of a large number of parameters that could alleviate the unreliability of asymptotic approximations, the use of finite-sample procedures turns out to be crucial.

The concept of autoregression at horizon h and the relevant notations yield a VAR(p) process of the form:

$$Z_t = a_t + \sum_{k=1}^{p} 1_k Z_{t-k} + v_t$$

where $Z_t = (z_{1t}, z_{2t}, \ldots, z_{mt})'$ is a random vector, a_t is a deterministic trend, and v_t is a white noise process of order 2 with a nonsingular variance–covariance matrix Ω. The most common specification for a_t assumes that a_t is a constant vector, although other deterministic trends, such as seasonal dummies, could also be considered. The VAR(p) is an autoregression at horizon 1. This autoregressive form can be generalized to allow for projection at any horizon h given the information available at time t. Hence, the observation at time $t + h$ can be computed recursively from the above equation and yields:

$$Z_{t+h} = a_t^{(h)} + \sum_{k=1}^{p} 1_k^{(h)} Z_{t+1-k} + \sum_{j=0}^{h-1} d_j b_{t+h-j}$$

where $d_0 = I_m$ and $h < T$. The appropriate formulae for the coefficients $1_k^{(h)}$ and $a_t^{(h)}$ are given in Dufour and Renault (1998), and the d_j matrices are the impulse-response coefficients of the process. The above equation is called an autoregression of order p at horizon h or a $(p; h)$-autoregression. Within this framework, the hypothesis is that a variable z_{jt} does not cause another one, say z_{it}, at horizon h. The restrictions related to that hypothesis take the form:

$$H_0^{(h)} : 1_{ijk}^{(h)} = 0, k = 1, \ldots, p$$

Thus the null hypothesis takes the form of a set of zero restrictions on the coefficients of the matrix $1_k^{(h)}$. Under the null hypothesis of noncausality at horizon h from z_{jt} to z_{it}, the asymptotic distribution of the Wald statistic is $x^2(p)$. To get an appropriate distribution,

researchers need to take into account that the prediction error follows an MA($h - 1$) process. To that end, the approach uses the Newey–West procedure, which gives an automatically positive-semi-definite variance–covariance matrix. The provided Gaussian asymptotic distribution may not be very reliable in finite samples, especially if a VAR system is considered with a large number of variables and/or lags.

Due to autocorrelation, a larger horizon may also affect the size and the power of the test. An alternative to using the asymptotic chi-square distribution lies in using Monte Carlo test methodologies (Dufour, 2006) or bootstrap methodologies (Boxes 9.4 and 9.5).

BOX 9.4 Published Case Study on the Toda–Yamamoto Approach

The STUDY: Alimi, S.R., Ofonyelu, C.C., 2013. Toda-Yamamoto causality tests between money market interest rates and expected inflation: the Fisher hypothesis revisited. European Scientific Journal 9, 125–142.

Data

Annual time series data on nominal interest rates, inflation, and effective exchange rates for Nigeria are obtained from the Annual Report and Statements of Accounts published by the Central Bank of Nigeria, spanning the period 1970–2011. The analysis makes use of money market interest rates as nominal interests, as along with inflation can be used as proxies for expected inflation. In addition, the analysis employs the US six-month London Interbank Rate (USRATE), obtained from the World Economic Outlook Publication Report, as a proxy for the foreign interest rate. Finally, all variables are expressed in percentages.

Causality results

Given the uncertain results generated by the unit root tests, the Toda–Yamamoto results are reported in Table 9.4.

Causality conclusions

The findings illustrated in Table 9.4 show that there is only a unidirectional causality between inflation and the nominal interest rate, running from inflation to nominal interest rates, while there is no other causality evident, which provides empirical support to the Fisher hypothesis.

Table 9.4: Toda–Yamamoto causality results.

Null Hypothesis	χ^2 test	*P*-value	Results
Inflation does not cause interest rates	4.352	0.04	Reject the null
Interest rates do not cause inflation	0.162	0.69	Accept the null
Exchange rates do not cause interest rates	0.254	0.61	Accept the null
Interest rates do not cause exchange rates	1.583	0.21	Accept the null
US rate does not cause interest rates	0.884	0.35	Accept the null
Interest rates do not cause the US rate	0.086	0.77	Accept the null

BOX 9.5 Published Case Study on the Dufour, Pelletier, and Renault Causality Approach

The STUDY: Dufour, J.M., Bixi, J., 2016. Multiple Horizon Causality in Network Analysis: Measuring Volatility Interconnections in Financial Markets. Available at SSRN: https://ssrn.com/abstract=2745341.

Data

They study the crisis-sensitive volatility network in the US stock market. They are also interested in examining whether their volatility connectedness measures can reflect the underlying market systemic risk that plays an important role in the recent global financial crisis. Given that the volatility in stock markets is latent, they need a volatility proxy. The well-known VIX index, which has been widely accepted as a market volatility index by financial practitioners, is calculated from implied volatilities of the S&P 500 index options. It is sensitive to market turmoils. For each firm, they also exploit the information in their respective option contracts and thus, they use implied volatility, rather than using realized volatility estimated from stock intraday prices, for the quantities they are dealing which are more comparable to market indices. Volatility or implied volatility is sensitive to "terrifying news" in financial markets. The stock implied volatilities are inevitably contaminated by shocks in financial markets as risks are traded on markets. Nevertheless, implied volatility is still an excellent proxy to study the high-dimensional market volatility network.

Similar to the VIX index for the S&P 500 stock composite, in this paper the S&P 100 components implied volatilities are constructed with their respective at-the-money option contracts with 30-day maturity. This implied volatility measures the expected volatility of the underlying stock over the next 30 days. Therefore, they only consider the option contracts with 30-day maturity. The date range of the database is from 01/01/1996 to 08/31/2015. The companies whose IPO dates are after 01/01/2000 are dropped off, such that they can examine the two most important crises in the US stock market (i.e., the IT Bubble Burst and the Financial Crisis of 2007–09). The remaining full sample is from 20/08/1999 to 31/08/2015. There are missing values on some dates for some companies and, thus, they take linear interpolations to impute the missing values to get completed time series processes for estimations. They end up with 90 companies in the final sample, $N = 90$. The Industry Group classification for each node is from the North American Industry Groups database from MorningStar, LLC.

Causality results

This part reports only a fraction of the entire findings spectrum (mainly due to space availability issue). In particular, Table 9.5 reports the top 10 influential firms and their respective sector at different forecast horizons, $h = 1, 2, 3, 4, 5$. Given the forecast horizon h, they obtain the summary statistics of each row of the firm-wise causality table. The causality table is estimated by the full data sample (20/08/1999–31/08/2015). Nodes are the firms of selected S&P 100 components. For each firm i, they provide the median value of the entries, while for each given forecast horizon h, they sort the tickers by their median values and identify the top 10 influential firms. Moreover, Table 9.6 illustrates summary statistics of causality measures from each financial firm to other financial firms. The causality table is estimated by the full data sample (20/08/1999–31/08/2015). Nodes are the firms of selected S&P 100 components. For each

Table 9.5: Causality at different horizons.

	$h = 1$		$h = 2$		$h = 3$		$h = 4$		$h = 5$	
Rank	**Sector**	**Ticker**	**Sector**	**Ticker**	**Sector**	**Ticker**	**Sector**	**Ticker**	**Sector**	**Ticker**
1	F	BAC	T	CSCO	T	CSCO	T	CSCO	T	CSCO
2	C AAPL	C	AAPL	C	AAPL	C	AAPL	C	AAPL	
3	T	CSCO	F	C	F	AIG	F	AIG	F	AIG
4	F	C	F	AIG	F	C	F	C	F	C
5	F	BK	F	GS	F	GS	F	GS	F	GS
6	F	AIG	I	GE	I	GE	I	GE	I	GE
7	F	MET	F	MS	F	JPM	F	JPM	F	JPM
8	C	F	F	JPM	C	F	C	F	C	F
9	F	JPM	F	MET	T	IBM	T	IBM	T	IBM
10	F	MS	C	F	F	MET	T	EMC	T	EMC

B, Basic materials; *C*, consumer goods; *F*, financial; *H*, healthcare; *I*, industrial goods; *S*, services; *T*, technology; *U*, utilities.

Table 9.6: Summary statistics of causality measures from each financial firm to other financial firms.

Ticker	Median	Mean	Minimum	25%	75%	Maximum
BAC	0.42	1.21	0.00	0.04	1.66	5.33
MS	0.30	1.19	0.00	0.04	2.21	4.83
GS	0.25	0.43	0.00	0.00	0.82	1.70
BK	0.06	0.91	0.00	0.00	0.98	5.75
WFC	0.03	0.49	0.00	0.00	0.38	3.96
ALL	0.01	0.30	0.00	0.00	0.37	1.46
SPG	0.01	0.54	0.00	0.00	0.08	5.63
AXP	0.00	0.43	0.00	0.00	0.24	2.78
C	0.00	0.42	0.00	0.00	0.53	2.77
AIG	0.00	0.14	0.00	0.00	0.12	0.74
COF	0.00	0.39	0.00	0.00	0.12	4.63
JPM	0.00	0.10	0.00	0.00	0.15	0.51
MET	0.00	0.31	0.00	0.00	0.58	1.32
USB	0.00	0.11	0.00	0.00	0.03	0.55

financial firm *i*, it reports the minimum value, the maximum value, the mean value and the quantiles [25%, 50% (median), and 75%] of the entries in its "OUT" vector truncated within the financial sector. The reported values are 100 times of the raw values, and are kept with two digits. It sorts the tickers by their median values and identifies the top 3 influential firms in the financial sector.

Causality conclusions

Table 9.5 reports the top 10 influential firms at different forecast horizons, $h = 1, 2, 3, 4, 5$, to take spillover effects into account. The firms and their orders in the list of top 10 influential firms are slightly different at different forecast horizons. For instance, in the case of only taking

direct effects into account ($h = 1$), the most influential financial firm is BAC and 7 out of 10 most influential firms belong to the financial sector; in the case of taking direct and indirect effects into account ($h = 5$), the most influential financial firm becomes AIG and only 4 out of 10 most influential firms is from the financial sector. The technology firms are actually influential. In the case of $h = 5$, 4 out 10 most influential firms belong to the technology sector and the top two influential firms come from the technology sector, if the Apple Inc. is considered as a technology firm. In short, measuring a static network that only characterizes direct effects in an economic network is far from enough to fully understand all interconnections and indirect effects. In contrast, directly measuring direct and indirect effects with the causality tables at different forecast horizons can provide "dynamic" pictures of interconnections in the S&P 100 network with different effect-radius. In many cases, what is truly important is firm's total effect (direct effect and indirect effect) rather than just its direct effect.

5 Asymmetric Causality

Given that a number of research studies consider that the impact of a positive shock is similar to the impact of a negative shock, this part of the survey considers the literature of the presence of an asymmetric structure in terms of causality. To this end, the literature considers the asymmetric causality behavior through the consideration of cumulative sums of positive and negative shocks. Hatemi-J (2012) considered such an asymmetric causality approach, along with a bootstrap simulation approach with leverage adjustments that generates the appropriate critical values. This methodological approach has the advantage that it is not necessarily based on datasets coming from a normal distribution.

Asymmetry here implies that positive and negative shocks could generate different causal effects. Let us consider again the bivariate model introduced in Section 1.1:

$$y_t = a_{10} + b_{11}\, y_{t-1} + b_{12}\, y_{t-2} + b_{13}\, x_{t-1} + b_{14}\, x_{t-2} + \varepsilon_{y_t}$$
$$x_t = a_{20} + b_{21}\, y_{t-1} + b_{22}\, y_{t-2} + b_{23}\, x_{t-1} + b_{24}\, x_{t-2} + \varepsilon_{x_t}$$

After estimating the above two equations and getting the corresponding residuals, we define as $\varepsilon^{+}_{y_t} = \max(\varepsilon_{y_t}, 0)$, $\varepsilon^{-}_{y_t} = \min(\varepsilon_{y_t}, 0)$, $\varepsilon^{+}_{x_t} = \max(\varepsilon_{x_t}, 0)$, and $\varepsilon^{+}_{x_t} = \min(\varepsilon_{x_t}, 0)$ their corresponding positive and negative shocks. Based on those definitions, the above system of equations yields:

$$y_t = a_{10} + \sum_{i=1}^{t} \varepsilon^{+}_{y_t} + \sum_{i=1}^{t} \varepsilon^{-}_{y_t}$$

and

$$x_t = a_{20} + \sum_{i=1}^{t} \varepsilon^{+}_{x_t} + \sum_{i=1}^{t} \varepsilon^{-}_{x_t}$$

Next, the positive and negative shocks of each variable can be defined in a cumulative form as:

$$y_t^+ = \sum_{i=1}^{t} \varepsilon_{y_i}^+$$
$$y_t^- = \sum_{i=1}^{t} \varepsilon_{y_i}^-$$
$$x_t^+ = \sum_{i=1}^{t} \varepsilon_{x_i}^+$$

and

$$x_t^- = \sum_{i=1}^{t} \varepsilon_{x_i}^-$$

In the next step, the analysis investigates the causal relationship between the above components. For example, in testing for causal relationship between positive cumulative shocks, the analysis assumes that $z_t^+ = (y_t^+, x_t^+)$ and that a VAR(p) model is used:

$$z_t^+ = \mathrm{b}_0 + \mathrm{B}_1 z_{t-1}^+ + \cdots + \mathrm{B}_p z_{t-1}^+ + v_t^+$$

where z_t^+ is the 2×1 vector of the variables, b_0 is the 2×1 vector of constant terms, and v_t^+ is the 2×1 vector of error terms. The matrix B_r is a 2×2 matrix of parameters for lag order r, with $r = 1, \ldots, p$. The analysis will test the null hypothesis that the kth element of z_t^+ does not Granger cause the ωth element of z_t^+ or in other words:

H_0: the row ω, column k element in B_r equals zero.

To test for causality, the analysis makes use of a Wald test by assuming that

$$Z = \left(z_1^+, \ldots, z_T^+\right) \text{being a} \, (n \times T) \text{ matrix}$$
$$D = (\mathrm{b}_0, \mathrm{B}_1, \ldots, \mathrm{B}_p) \text{being a} \, [n \times (1 + np)] \text{matrix}$$
$$\Lambda_t = \begin{pmatrix} 1 \\ z_t^+ \\ z_{t-1}^+ \\ \vdots \\ z_{t-p+1}^+ \end{pmatrix} \text{being a} \left[(1 + np) \times 1\right] \text{matrix}$$
$$\Lambda = \left(\Lambda_0, ..., \Lambda_{T-1}\right) \text{being a} \left[(1 + np) \times T\right] \text{matrix}$$
$$\delta = \left(v_1^+, \ldots, v_{\mathrm{T}}^+\right) \text{being a} \ (n \times T) \text{matrix}$$

The analysis defines the VAR(p) model as: $Z = D\Lambda + \delta$ and the null hypothesis of noncausality is H_0: $\mathrm{C}\beta = 0$ and is tested through the following Wald test:

$$(\mathrm{C}\beta)'[\mathrm{C}((\Lambda'\Lambda)^{-1} \times S_u)\mathrm{C}']^{-1}(\mathrm{C}\beta)$$

where $\beta = \text{vec}(D)$ and vec shows the column stacking operator, x represents the Kronecker product, and C is a $p \times n(1 + np)$ indicator matrix with all elements equal to one for restricted parameters and zeros for the remaining parameters. Finally, S_u is the variance–covariance matrix of the unrestricted VAR model (Box 9.6).

BOX 9.6 Published Case on Asymmetric Causality Case

The STUDY: Yildirim, S., Özdemir, B.K., Doğan, B., 2013. Financial development and economic growth nexus in emerging European economies: new evidence from asymmetric causality. International Journal of Economics and Financial Issues 3, 710–722.

Data

Given that both the stock and bond markets in the emerging European economies have not developed adequately, the analysis uses two different indicators, representing the activities and mediating dimension of the banking sector. These indicators are the ratio of M2 to GDP (denoted by FD1) and the liquid liabilities to GDP ratio (denoted by FD2). Economic growth is based on real GDP changes. Data are obtained from the International Financial Statistics database. Quarterly data are used for 10 countries: Bulgaria, Croatia, Hungary, Latvia, Lithuania, Poland, Romania, Russia, Turkey, and Ukraine. The time span is 1990–2012. Finally, all variables are expressed in natural logarithms.

Causality results

Due to space limits, a part of Table 9.7 is reported.

Table 9.7: Asymmetric causality results.

Bulgaria	Test Results
fdi^+ does not cause economic $growth^+$	1.072(6)
fdi^- does not cause economic $growth^-$	2.023(5)
fdi^- does not cause economic $growth^+$	1.368(5)
fdi^+ does not cause economic $growth^-$	35.132(5)
economic $growth^+$ does not cause fdi^+	4.187(6)
economic $growth^-$ does not cause fdi^-	22.716(5)
economic $growth^-$ does not cause fdi^+	6.420(5)
economic $growth^+$ does not cause fdi^-	8.704(5)

Critical values at 1% are: 38.042, 23.712, 21.700, 40.604, 50.993, 35.294, 31.789, 24.162, respectively.

Causality conclusions

The analysis uses the broad measure of the money stock to GDP ratio (M2 to GDP) as the financial development indicator in Table 9.7. This simple indicator helps measure the degree of monetization in an economy, and it is expected that the increases in M2 would be higher than GDP growth if financial deepening is occurring. At first glance, the impact of economic growth on financial development is more prominent. This pattern can be interpreted under the context of supply leading hypothesis. Further elaboration indicates that it is commonly observed that causality runs from negative growth shock to negative shocks in financial development. The feedback hypothesis or bidirectional causality does not exist.

6 Linear Panel Causality

We start with the Pooled Mean Group (PMG) estimator recommended by Pesaran et al. (1999). The PMG estimator is based on the Autoregressive Distributed Lag (ARDL) (p, q, q,…., q) model which is described as:

$$y_{it} = a_i + \sum_{j=1}^{p} \lambda_{ij} y_{i,t-j} + \sum_{j=0}^{q} \delta_{ij} x_{i,t-j} + v_{it}$$

where x_{it} is a vector of explanatory variables (regressors) for group i, a_i represents fixed effects, λ_{ij} are the coefficients of the lagged dependent variables, and δ_{ij} are coefficient vectors. We reparameterize the above equation and get:

$$\Delta y_{it} = \varphi_i y_{i,t-1} + \beta_i' x_{it} + \sum_{j=1}^{p-1} \lambda_{ij}^{*} \Delta y_{i,t-j} + \sum_{j=0}^{q-1} \delta_{ij}^{*} \Delta x_{i,t-j} + a_i + v_{it}$$
$$i = 1,2,\ldots,N, t = 1,2,\ldots,T$$

where $\varphi_i = -\left[1 - \Sigma_{j=1}^{p} \lambda_{ij}\right], \beta_i = \Sigma_{j=0}^{q} \delta_{ij}, \lambda_{ij}^{*} = -\Sigma_{m=j+1}^{p} \lambda_{im}$, for $j = 1,\ldots,p-1, \delta_{ij}^{*} = -\Sigma_{m=j+1}^{q} \delta_{im}$ for $j = 1,\ldots,q-1$.

Pesaran et al. (1999) assumed that the ARDL (p, q, q,…., q) model is stable, if the roots of the following equation:

$$1 - \sum_{j=1}^{p} \lambda_{ij} = 0$$

lies outside the unit circle. This assumption ensures that $\varphi_i < 0$ and, thus, there is a long-run relationship between y_{it} and x_{it} defined by:

$$y_{it} = -[\beta_i / \varphi_i x_{it} + \eta_{it}]$$

where η_{it} is a stationary process and the long-run coefficients $\theta_i = -\beta_i/\varphi_i = \theta$ are the same across the group. Therefore when a cointegrating relationship between x_{it} and y_{it} is established, namely as:

$$y_{it} = a_{0i} + a_{1i} x_{it} + a_{2i} z_{it} + v_{it}$$

with z representing another control variable, the ARDL (1,1,1) equation yields:

$$y_{it} = a_{0i} + a_{1i} x_{it} + a_{2i} x_{i,t-1} + a_{3i} z_{it} + a_{4i} z_{i,t-1} + \lambda_i y_{i,t-1} + v_{it}$$

and

$$x_{it} = b_{0i} + b_{1i}y_{it} + b_{2i}y_{i,t-1} + b_{3i}z_{it} + b_{4i}z_{i,t-1} + \mu_i y_{i,t-1}u_{it}$$

while the error-correction equations yield:

$$\Delta y_{it} = \varphi_1(y_{i,t-1} - \theta_1 x_{it} - \theta_2 z_{it}) - a_{2i}\Delta x_{it} - a_{3i}\Delta z_{it} + v_{it}$$

and

$$\Delta x_{it} = \varphi_2(x_{i,t-1} - \theta_3 y_{it} - \theta_4 z_{it}) - a_{4i}\Delta y_{it} - a_{5i}\Delta z_{it} + u_{it}$$

We can evaluate the null hypothesis of non-causality as $a_{2i} = 0$ and $b_{2i} = 0$.

We can also analyze causal empirical relationships by using the panel data causality testing methodology, developed by Holtz-Eakin et al. (1988). This methodology is closely related to an approach proposed by Anderson and Hsiao (1981). The test involves estimation of error-correction equations as below:

$$\Delta \ln y_{it} = b_1(\ln y_{i,t-1} - c_1\Delta \ln x_{it}) + a_{1i} + v_{1it}$$
$$\Delta \ln x_{it} = b_2(\ln x_{i,t-1} - c_2\Delta \ln y_{it}) + a_{2i} + v_{2it}$$

where a denote the time effects or alternatively the time trend. The parameters b_1 and b_2 denote the error-correction terms. The question of whether or not x causes y can be tested through the hypothesis:

$b_1 = c_1 = 0, H_0 : x$ does not Granger cause y in the long run

$b_2 = c_2 = 0, H_0 : y$ does not Granger cause x the long run.

The rejection of the first null hypothesis and the acceptance of the second null hypothesis is interpreted as causality from x to y, while the rejection of the second null hypothesis and the acceptance of the first null hypothesis are interpreted as causality in the reverse direction. If both hypotheses are rejected, then there is no feedback between the two variables. Assuming that the residuals of the level equation are serially uncorrelated, the values of y lagging two periods or more can be used as instruments in the first-differenced equation. The estimation equation and moment conditions can be estimated by first-differenced General Method of Moments (GMM), which was developed by Arellano and Bond (1991). However, conventional GMM estimations exhibit a major drawback if the explanatory variables display persistence over time. By persistence, we mean that the mean reversion process of the explanatory variable is very slow. In this case, their lagged levels may be rather poor instruments for their differences. Therefore researchers should use the system GMM estimator that was introduced by Blundell and Bond (1998), which combines the regression equation in first differences, instrumented with the lagged levels of the regressors, with the regression equation in the levels, instrumented with lagged

the differences of the regressors. However, a drawback of the approach is that it ignores the heterogeneity problem in the cross-sections.

The above problem led to the development of the panel causality test by Hurlin and Venet (2001). They make use of a panel Granger model where for each individual i and for all $t = 1,\ldots, T$ the model yields:

$$y_{it} = \sum_{k=1}^{r} \gamma i^{k} y_{i,t-1} + \sum_{k=0}^{r} \beta_{i}^{k} x_{i,t-k} + v_{it}$$

where the autoregressive coefficients (k) and the regression coefficients slopes $i(k)$ are assumed to be constant for all $k = 1,\ldots, r$. The autoregressive coefficients are also assumed to be the same for all units, while the regression coefficients slopes can vary across individuals. Due to this, there are four types of causality relationships proposed by Hurlin and Venet (2001) to take account of the heterogeneity in the underlying processes. The testing procedure involves the combination of various sets of tests that make use of the F-test and when the null hypothesis of the first case is rejected, we could then proceed to the second case and even further, depending on the results. But when the null hypothesis for any case could not be rejected, the tests can then be ended. The four causality tests are therefore stated below. The Homogenous Noncausality (HNC) case is the first test, which implies testing whether a particular variable is not causing another one in all the cross-sections of the samples. If the computed F-statistic is significant, then the HNC hypothesis is rejected, that is, implying that there is causality in at least one member of the panel, and then we continue testing the HNC. In contrast, if the HNC hypothesis is accepted, then there is not any causality relationship across any member of the panel and the testing process will not proceed further. The second case is testing the Homogenous Causality (HC) hypothesis and we test whether one variable is causing another across all entities, that is, countries of the sample. If the F-statistic is not significant, then the HC hypothesis is accepted, implying that there is causality across all members of the panel and further testing will not be necessary. But when the HC hypothesis is rejected, then there is no causality relationship in at least one member of the panel. In the third case, we consider that when the HC hypothesis is rejected, there is no HC and then we move on to the heterogeneity tests to determine which of the members of the panel exhibits a causal relationship. Heterogeneous Causality (HEC) is the third case and the implication of this is that there are causal relationships that exist in at least one individual, and causality could rise to a maximum of N individuals. Finally, the fourth case, the Heterogeneous Noncausality (HENC) hypothesis implies that for not less than one individual, and for at most $N - 1$ individuals, there is causality across them. The rejection of the HENC hypothesis implies that the statistic is significant; therefore, there is causality for the individual under consideration. The second test is tested the joint hypothesis of no causality for a subgroup of individuals in the panel. In this case, the slope coefficients of all lags across the individuals of the subgroups are constrained to zero. If the F-statistic is significant, this implies the rejection

of the HENC hypothesis for the subgroup under consideration and we can conclude that causality exists for this subgroup of panel members (Babajide, 2010).

Next, we consider the panel causality test introduced by Dumitrescu and Hurlin (2012). This test is a simple version of the Granger (1969) noncausality test for heterogeneous panel data models with fixed coefficients, while it takes into consideration two dimensions of heterogeneity: the heterogeneity of the regression model used to test the Granger causality and the heterogeneity of the causality relationships. We consider the following linear model:

$$y_{it} = a_i + \sum_{k=1}^{K} \gamma i^k y_{i,t-k} + \sum_{k=1}^{K} \beta_i^k x_{i,t-k} + v_{it}$$

where x and y are two stationary variables observed for N individuals in T periods. Also, $\beta_i = \left(\beta_i^1, \ldots, \beta_i^k\right)'$ and the individual effects a_i are assumed to be fixed in the time dimension. We assume that the lag orders of K are identical for all cross-section units of the panel. We also allow the autoregressive parameters γ_i^k and the regression coefficients β_i^k to vary across groups. Under the null hypothesis, it is assumed that there is no causality relationship for any of the units of the panel. This assumption is called the HNC hypothesis, which is defined as:

$$H_0 : \beta_i = 0, \quad \text{for} \quad i = 1, \ldots, N$$

The alternative is specified as the HENC hypothesis. Under this hypothesis, we allow for two subgroups of cross-section units. There is a causality relationship from x to y for the first one, but it is not necessarily based on the same regression model. For the second subgroup, there is no causality relationship from x to y. We consider a heterogeneous panel data model with fixed coefficients (in time) for this group. This alternative hypothesis is described by the following two parts:

$$H_1 : \beta_i = 0 \quad \text{for } i = 1, \ldots, N_1$$

and

$$\beta_i \neq 0 \quad \text{for } i = N_1 + 1, \ldots, N$$

It is also assumed that β_i may vary across groups and there are $N_1 < N$ individual processes with no causality from x to y. N_1 is unknown, but it provides the condition $0 \leq N_1/N < 1$. The following average statistic HNC $W_{N,T}^{\text{HNC}}$ is proposed, which is related with the null HNC hypothesis, as follows:

$$W_{N,T}^{\text{HNC}} = 1/N \sum_{i=1}^{N} W_{i,T}$$

where $W_{i,T}$ denotes the individual Wald statistics for the ith cross-section unit corresponding to the individual test: H_0: $\beta_i = 0$.

We define $Z_i = [e, Y_i, X_i]$ as a $(T \times 2K + 1)$ matrix, where e denotes a $(T, 1)$ vector and $Y_i = \left[y_i^1, \ldots, y_i^k\right]$, $X_{i=}\left[x_i^1, \ldots, x_i^k\right]$. $\theta_i = (\alpha_i\ \gamma_i'\ \beta_i')$ is the vector of parameters of the model. In addition, $R = [0, I_K]$ is a $(K, 2K + 1)$ matrix. For each $i = 1, \ldots, N$, the Wald statistic $W_{i,T}$ corresponding to the individual test H_0: $\beta_i = 0$ is defined as:

$$W_{it} = \theta_i' R' [\sigma_i^2 R (Z_i' Z_i)^{-1} R'] R \theta_i$$

Under the null hypothesis of noncausality, each individual Wald statistic converges to a chi-squared distribution with K degrees of freedom for $T \to \infty$.

Next, a third panel causality approach has been developed by Kònya (2006) which allows accounting for both cross-sectional dependence and heterogeneity issues. It is based on Seemingly Unrelated Regressions (SUR) systems and Wald tests with specific bootstrap critical values. This particular methodology enables testing for Granger causality on each individual panel member separately, by taking into account the potential contemporaneous correlation across entities (e.g., countries, regions, sectors, firms, banks) involved in the sample. This panel causality approach examines the relationship between y and x and can be studied using the following bivariate finite order vector autoregressive (VAR) model as before:

$$y_{it} = a_{1i} + \sum_{s=1}^{ly_1} b_{1is} y_{i,t-s} + \sum_{s=1}^{lx_1} c_{1is} x_{i,t-s} + v_{1it}$$

and

$$x_{it} = a_{2i} + \sum_{s=1}^{ly_2} b_{2is} y_{1,t-s} + \sum_{s=1}^{lx_2} c_{2is} x_{i,t-s} + v_{2it}$$

where the index i ($i = 1, \ldots, N$) denotes one entity, that is, country, the index t ($t = 1, \ldots, T$) the period, s is the lag, and ly_1, lx_1, ly_2, and lx_2 denote the lag lengths. The error terms, v_{1it} and v_{2it} are considered to be white noises (i.e., they have zero means, constant variances, and are individually serially uncorrelated) and may be correlated with each other for a given entity, that is, country.

In one entity, that is, country i, there is one-way Granger causality running from x to y, if in the first equation not all c_{1i} are zero, but in the second equation all b_{2i} are zero. There is one-way Granger causality from y to x, if in the first equation all c_{1i} are zero, but in the second equation not all b_{2i} are zero. There is two-way Granger causality between y and x, if neither all b_{2i} nor all γ_{1i} are zero. Finally, there is no Granger causality between y and x, if all b_{2i} and γ_{1i} are zero. The OLS estimators of the parameters are consistent and asymptotically efficient. This suggests that the $2N$ equations in the system can be estimated one-by-one, in any preferred order, while the entire system for all entities, that is, countries, can be estimated

by the SUR procedure to take into account contemporaneous correlations within the systems (in the presence of contemporaneous correlation, the SUR estimator is more efficient than the OLS estimator). Following Kònya (2006), specific bootstrap Wald critical values are used to implement Granger causality. This methodological procedure has several advantages: (1) it does not assume that the panel is homogeneous, so it is possible to test for Granger-causality on each individual panel member separately, (2) this approach does not require pretesting for unit roots and cointegration, though it still requires the specification of the lag structure, and (3) this panel Granger causality approach allows researchers to detect for how many and for which members of the panel there exists one-way Granger causality, two-way Granger causality, or no Granger causality. However, Konya's testing procedure has a drawback for the panel datasets, if the number of cross-sections (N) is not reasonably smaller than time periods (T), because the SUR estimator is only feasible for panels with large T and small N.

The approaches described above can control for heterogeneity, but they are not capable of accounting for cross-sectional dependence. To overcome this problem, Emirmahmutoglu and Kose (2011) developed a panel causality methodology that accounts for cross-country heterogeneity irrespective of whether the variables of interest are nonstationary or cointegrated. In addition to this flexibility, because the critical values for panel statistics are derived from bootstrap distributions, this methodology also considers the cross-section dependency. This approach considers the following VAR model for each cross-section:

$$y_{it} = a_i + \mathrm{B}_{1i} y_{i,t-1} + \ldots + \mathrm{B}_{pi} y_{i,t-pi} + \ldots + \mathrm{A}_{p+d,i} y_{i,t-pi-di} + v_{it}$$

where y_{it} is a vector of endogenous variables, a_i denotes the p dimensional vector of fixed effects, p_i is the optimal lag(s), and d_i is the maximum integration degree of the variables. The null hypothesis of no-Granger causality against the alternative hypothesis of Granger causality is tested by imposing zero restrictions on the first p parameters. The so-called modified Wald statistic has the asymptotic chi-square distribution with p degrees of freedom. To test the Granger noncausality hypothesis for the panel, the Fisher statistic is developed and defined as:

$$\lambda = -2\sum_{i=1}^{N} \ln(\pi_{\mathrm{i}})$$

where π_i is the probability corresponding to the individual modified Wald statistic. The Fisher statistic has an asymptotic chi-square distribution with $2N$ degrees of freedom. However, the limit distribution of the Fisher test statistic is no longer valid in the presence of cross-section dependency. To accommodate for cross-section dependency in the panel, Emirmahmutoglu and Kose (2011) suggest obtaining an empirical distribution of the panel statistic using the bootstrap methodology. For more details on the bootstrap methodology, the reader should refer to Emirmahmutoglu and Kose (2011) (Boxes 9.7 and 9.8).

BOX 9.7 Published Case in Panel Granger Causality by Pesaran et al. (1999)

The STUDY: Costantini, V., Martini, C., 2009. Causality Between Energy Consumption and Economic Growth: a Multi-sectoral Analysis Using Non-stationary Cointegrated Panel Data. Working Paper, No. 102/09, Department of Economics, University of Rome III.

Data

They obtained information on 71 countries, divided into two groups: OECD, with 26 countries, and NO-OECD, with 45 countries. The countries included in the OECD group are quite homogeneous, whereas those in the NO-OECD group are quite heterogeneous, both with respect to their development level and their policy settings. The dataset combines several sources. For the energy sectors, they obtained data from the IEA publications on OECD and NO-OECD energy balances, containing annual data on energy final consumption for the entire economy and for its main sectors, that is, industry, commerce and public services, transport, and residential sectors, all expressed in terms of kg of oil equivalent. All information on economic performance across the different sectors is taken from the World Bank dataset on World Development Indicators. More specifically, they consider gross domestic product, the value added of the industry and services, household's final consumption expenditures, all considered in terms of per capita constant 2000 US$. For the transport sector, they employed GDP as the economic dimension. Data on energy prices are provided by IEA statistics on energy prices and taxes (quarterly) for OECD countries, only for the period 1978–2005. They obtained data for the whole energy sector, along with the four specific end use sectors and considered four different energy prices: total energy price, total industry price, total household price, and total gasoline price (all expressed in terms of constant 2000 US$ per ton). They use the total industry price both for the industrial and the service sector even though many contributions affirm that the best price variable for services is the cost of electricity. In this dataset, electricity prices are often missing or are not complete throughout the time period, thus, consistently reducing the number of observations. Although they are aware that they could specify energy sectors with prices even for NO-OECD countries through the general Consumer Price Index as a proxy of energy prices, but they opted to adopt sector-specific energy prices to obtain more accurate estimates of price elasticities, as CPI does not account homogenously for energy services across all countries. For bivariate models, data availability allows considering the period 1970–2005 for the full sample and the NO-OECD sample, whereas for the case of OECD countries, the time series cover the period 1960–2005.

For multivariate models including energy prices, they have a reduced sample with only OECD countries, spanning the period 1978–2005. Considering the wide divergence among countries, both in the energy sectors and in economic performance, they considered per capita levels and thus they transformed all data into natural logarithms, given the high variance in levels between developed and developing countries.

Panel Granger causality results

Having estimated the VECM across all sectors and distinct subsamples, the analysis performs simple Wald *F*-tests on the significance of the coefficients, evaluating three different Granger causality relationships: a short-run causality, testing the significance of the coefficients related

Table 9.8: Panel causality tests.

	Short-run	Long-run	Strong Causality		
Dependent Variable	**GDP**	**EC**	**ECT**	**GDP-ECT**	**EC-ECT**
GDP	—	3.15*	−1.75	—	4.00
EC	4.06**	—	−9.67***	18.53***	—

EC, Energy consumption; *ECT*, error correction term. * Significant at 10% level. ** Significant at 5% level. *** Significant at 1% level. The heteroschedasticity of the error terms is corrected by using White robust standard errors both in periods (White period system robust covariances) and in cross-sections (coefficient covariance method: White cross-section system robust). The method for iteration control for GLS and GMM weighting specifications is to iterate weights and coefficients sequentially to convergence. To correct for possible autocorrelation we use the Newey–West estimator of the weighting matrix in the GMM criterion.

to the lagged economic and energy variables, a long-run causality related to the coefficient for the error-correction term, and a strong causality to test whether the sources of causation are jointly significant. The results of the VECM with two simultaneous equations for the analysis of the causal relationships between energy consumption and economic growth are reported in Table 9.8. This Table reports results in terms of the Wald *F*-test on the coefficients. Again, due to space limitations, only a part of this table is reported (the full sample results).

Granger causality conclusions

When the bivariate VECM model is performed on the whole economy, a bidirectional short-run causality and a unidirectional long-run relationship, where the economic growth is the driver of energy consumption and not vice versa is established. In addition, the negative sign of the estimated speed of adjustment coefficients are in accordance with the convergence toward long-run equilibrium. The larger the value of that coefficient, the stronger is the response of the variable to the previous period's deviation from long-run equilibrium, if any.

BOX 9.8 Panel Granger Causality on Published Case by Dumitrescu and Hurlin (2012)

The STUDY: Zeren, F., Ari, A., 2013. Trade openness and economic growth: a panel causality test. International Journal of Business and Social Science, 4 317–324.

Data

Their study investigates causality between trade openness and economic growth in the case of the G7 countries (i.e., Germany, France, Canada, Japan, Italy, US, and UK), spanning the period 1970–2011. Economic growth is measured using per capita GDP with constant 2000 US$, and trade openness is measured exports plus imports as a share of GDP. The data used in the paper are obtained from the World Development Indicators database provided by the World Bank. Both variables are expressed in their natural logarithms.

Panel Granger results

Table 9.9: Panel Granger causality results.

Tests Openness	Trade Openness → Economic Growth	Economic Growth → Trade
$W_{N,T}$	6.811*	5.937*
$Z_{N,T}$	12.351*	9.519*
$\hat{Z}_{N,T}$	3.411*	2.588*

* Significant at 5%.

Panel Granger causality conclusions

When the test statistics in Table 9.9 are compared to the bootstrap critical values in Table 9.9 by Dumitrescu and Hurlin (2012), it is inferred that these test statistics are statistically significant. Thus a bidirectional causality relationship exists between trade openness and economic growth. For the case of the G7 countries there exists a bidirectional causality relationship.

7 Nonlinear and Nonparametric Causality

This section describes the available nonlinear tests for exploring Granger causality. There are two main facts justifying the employment of such nonlinear testing procedures. First, standard linear Granger causality tests have extremely low power in detecting certain kinds of nonlinear relationships (Brock, 1991; Gurgul and Lach, 2009). Second, as the traditional linear approach is based on testing the statistical significance of suitable parameters only in terms of the mean equation, causality in higher-order structures (i.e., causality in variance) cannot be explored (Diks and Degoede, 2001). The application of nonlinear causality approaches may be a solution to this problem, because it allows exploring complex dynamic links between the variables of interest.

First, we refer to the approach proposed by Diks and Panchenko (2006). To this end, we define for $t = 1, 2,\ldots$, the $L_x + L_y + 1$-dimensional vector $W_t = [x_{t-L_x}^{L_x}, y_{t-L_y}^{L_y}, Y_t)$. The null hypothesis that x_t does not Granger cause y_t may be written in terms of density functions:

$$f_{x,y,z}(x,y,z) = f_{x,z}(x,z)\, f_{z|x,y}(z \mid x,y) = f_{x,z}(x,z)\, f_{z|y}(z \mid y)$$

where $f_x(z)$ stands for the probability density function of the random vector X at point z, $x = x_{t-L_x}^{L_x} y = y_{t-L_y}^{L_y}$, $z = y_t$ for $t = 1, 2,\ldots$ Next, we can define the correlation integral $C_W(\varepsilon)$ for the multivariate random vector W by the following expression:

$$C_W(\varepsilon) = \mathrm{P}[\| W_1 - W_2 \|] \le \varepsilon = \iint I(\| s_1 - s_2 \|) \le \varepsilon\; f_W(s_1)\, f_W(s_2)\, ds_2\, ds_1$$

where W_1 and W_2 are independent with distributions in the equivalence class of the distribution of W, I denotes the indicator function (equal to one, if the condition in brackets holds true, otherwise is equal to zero), $\|x\| = \sup\{|x_i|: i = 1,..., d_W\}$ denotes the supremum norm (d_W is the dimension of the sample space W) and $\varepsilon > 0$. Hiemstra and Jones (1994) claimed that the null hypothesis in Granger's causality test implies that for every $\varepsilon > 0$:

$$C_{x,y,z}(\varepsilon)/C_{x,y}(\varepsilon) = C_{y,z}(\varepsilon)/C_{y}(\varepsilon)$$

They recommend calculating sample versions of correlation integrals and then testing whether the left-hand- and right-hand-side ratios differ significantly or not. They propose the use of the following formula as an estimator of the correlation integral:

$$C_{W,n}(\varepsilon) = 2/[n(n-1)] \times \sum_{i<j} \sum I_{ij}^{W}$$

where $I_{ij}^{W} = I\left(\left\|W_i - W_j\right\|\right) < \varepsilon$. In terms of the expected value and density functions, the authors managed to test their recommendation based on the asymptotic theory of the F-test statistic. Furthermore, they present some advice concerning the proper way of choosing the bandwidth according to the sample size. This adaptation is helpful in reducing the bias of the test, which is one of the serious problems which arise for long time series. The performance of the modified test is also based on the same lags for each pair of time series analyzed. They generate the following statistic for testing for causality:

$$T(\varepsilon) = (n-1)/[n(n-2)] \times \sum_{i} \dot{f}_{x,y,z}(x_i, y_i, z_i)\dot{f}_{y}(y_i) - \dot{f}_{x,y}(x_i, y_i)\dot{f}_{y,z}(y_i, z_i)$$

where n is the sample size, and $\dot{f}_w(w)$ is a local density estimator of a d_w. They prove that under strong mixing (strong mixing refers to the condition introduced by Rosenblatt (1971)) and has to do with the central limit theorem for "weakly dependent" random variables. It has received considerable importance in terms of the probability theory, due to its tractability in the derivation of the asymptotic properties for various functions in relevance to dependent random variables, for example, members of the important class of linear stochastic processes are strongly strong mixing, provided they are based on innovation random variables which have Lebesgue-integrable characteristic functions, while certain AR(1) processes do not represent strong mixing cases, the above test statistic satisfies:

$$\sqrt{n}[T(\varepsilon) - q]/S_n \to N(0,1)$$

where $\to$ denotes convergence in distribution, q denotes lags, and S_n is the estimator of the asymptotic variance of T. We can follow Diks and Panchenko's suggestion to implement a

one-tailed version of the test, rejecting the null hypothesis, if the left-hand-side of the above expression is too large. As the statistic diverges to positive infinity under the alternative hypothesis, a calculated statistic greater than 1.28 implies the rejection of the null hypothesis at the 10% level of significance. In their test, the value of the bandwidth plays an important role in making a decision on nonlinear causality. Since a bandwidth value smaller (larger) than one generally results in larger (smaller) *P*-values (Bekiros and Diks, 2008), usually, the bandwidth value is equal to one.

This part examines nonlinear causality through the Smooth Transition Autoregressive (STAR) modeling approach. To this end, the following univariate STAR model is described based on the methodological approach proposed by Teräsvirta (1994):

$$y_t = a_{10} + b_1 w_{\mathrm{t}} + (b_{20} + b_2 w_{\mathrm{t}}) F(y_{t-d}) + u_t$$

where $w_t = (y_{t-1}, \ldots, y_{t-p})$, $u_t \to N(0, \sigma_u^2)$ and a $F(y_{t-d})$ denotes a transition function, while d is an unknown delay parameter. There are two choices for the transition function that are based on the following logistic function:

$$F(y_{t-d}) = \left[1 + \exp(-\gamma_{\mathrm{L}}(y_{t-d} - c_{\mathrm{L}}))\right]^{-1} \quad \text{with } \gamma_{\mathrm{L}} > 0$$

and the following exponential function:

$$F(y_{t-d}) = \left[1 + \exp(-\gamma_{\mathrm{E}}\left(y_{t-d} - c_{\mathrm{E}}\right)^2\right]^{-1} \quad \text{with } \gamma_{\mathrm{E}} > 0$$

The above transition functions yield logistic STAR (LSTAR) and exponential STAR (ESTAR) models, respectively. In the case of LSTAR, two regimes can be considered, depending on the small and large values of the transition variable relative to the threshold parameter, c_{L}. This type of models can be appropriate to model business cycle asymmetries where expansion and contraction periods have different dynamics. In other words, the LSTR specification accounts for asymmetric realizations, in the sense that the two regimes are associated with small and large values of the transition variable relative to the threshold value. In contrast, the regimes in ESTAR models are subject to small and large absolute values of the transition function relative to c_{E}. The ESTAR transition function is symmetric around the threshold parameter, while the values close to c_{E} differ. The STAR-based test of Granger causality can be performed through the additive smooth transition regression model, presented with reference to Skalin and Teräsvirta (1996):

$$y_t = a_{10} + b_1 w_t + (b_{20} + b_2 w_t) F(y_{t-d}) + \delta_1 v_t + (\delta_{20} + \delta_2 v_t G(x_{t-e}) + u_t$$

where $v_t = (x_{t-1},\ldots, x_{t-q})'$ and $G(.)$ shows the transition function, while e is an unknown delay parameter. The noncausality hypothesis is H_0: $G0$ and $\delta_{1i} = 0$, and $i = 1,\ldots, q$.

In case that nonlinear or threshold cointegration has been detected, we may test the transmissions using threshold error-correction modeling approaches (TECM). The TECM can be presented as follows (Enders and Siklos, 2001):

$$\Delta Y_t = a + \rho_1 Z^+_{t-1} + \rho_2 Z^-_{t-1} + \sum_{i=1}^{n_1} \delta_i \Delta y_{t-i} + \sum_{i=1}^{n_2} \theta_i \Delta x_{t-i} + \varepsilon_t$$

where $Y_t = (y_t, x_t)'$, $Z^+_{t-1} = h_t \hat{g}_{t-1}$, $Z^-_{t-1} = (1-h_t)\hat{g}_{t-1}$, such that $h_t = 1$, if $\hat{g}_{t-1} \geq \psi$, $h_t = 0$, if $\hat{g}_{t-1} < \psi$ and ε is a white noise disturbance. Through the system, the Granger causality tests are examined by testing whether all the coefficients of Δy_{t-i} and Δx_{t-i} are statistically different from zero, based on a standard F-test and if the ρ coefficients of the error-correction are also significant. Granger causality tests are very sensitive to the selection of the lag length, and thus the appropriate lag lengths can be determined through the Akaike criterion.

Next, we may test for Granger noncausality through the nonparametric test proposed by Nishiyama et al. (2011). The test statistic is constructed based on moment conditions for causality in the mean. To apply the test, the Nadaraya–Watson nonparametric estimator of moments is needed. Let $z(x_t,y_t)$ be a sample of T observations on dependent random variables in $\mathbb{R}\times\mathbb{R}$, with a joint distribution function F. Suppose now we need to test Granger noncausality in the mean from x_{t-1} to y_t. This corresponds to testing the null hypothesis:

$$H_0 : \Pr\{E[u_t \mid Z_{t-1}] = 0\} = 1$$

against the alternative hypothesis:

$$H_1 : \Pr\{E[u_t \mid Z_{t-1} = 0]\} < 1$$

where $\underline{u}_t = y_t - E[y_t|y_{t-1}]$. If the null hypothesis is true, then past changes in x_t cannot affect the conditional mean of y_t. The authors have illustrated that the above null and alternative hypotheses can be rewritten in terms of unconditional moment restrictions:

$$H_0 : \Pr\{E[u_t f(y_{t-1})h(Z_{t-1})] = 0\} < 1$$

against the alternative hypothesis:

$$H_1 : \Pr\{E[u_t f(y_{t-1})h(Z_{t-1})] = 0\} < 1$$

where $h(z)$ is any function in the Hilbert space $s_r^{\perp}$ that is orthogonal to the Hilbert L_2 space: $s_r = \{s(.)|E[s(y_{t-1})^2] < \infty$. As $E[u_t f(y_{t-1})\, h(Z_{t-1})]$ is unknown, we can use a nonparametric approach to estimate it. According to these authors, and through the Nadaraya–Watson methodology, we can estimate this conditional mean. To test the null hypothesis against the alternative hypothesis, the authors use the following test statistic:

$$\hat{S}_T = \sum_{i=1}^{k_T} w_i \alpha_i^2$$

where $\alpha_{\mathrm{i}} = 1/\sqrt{T}\,\Sigma u_t \dot{f}(y_{t-1})\dot{h}(Z_{t-1})$, while w_i is a nonnegative weighting function, such as $w_i = 0.9^i$. The above test statistic depends on the sample size. The authors have illustrated that, under the null hypothesis, the statistic converges in distribution to:

$$\sum_{i=1}^{\infty} w_i \varepsilon_i^2, \text{as} T \to \infty, \quad \text{where } \varepsilon_{\mathrm{i}} \text{ are i.i.d.} N(0,1).$$

In other words, for a given summable positive sequence of weights $\{w_i\}$, the test statistic is pivotal and is asymptotically distributed as an infinite sum of weighted chi-squares. The main advantage of this test is that the simulation is very simple and the critical values are not dependent on the data.

Finally, we can also test whether past changes in x_t can affect the conditional distribution of y_t. The null hypothesis is defined when the distribution of y_t is conditional on its own past and past changes in x_t are equal to the distribution of y_t conditional on their own past only, almost everywhere. This is similar to test the conditional independence between y_t and past changes in x_t conditionally on the past y_t. According to Florens and Mouchart (1982) Florens and Mouchart (1982) and Florens and Fougère (1996), this is also a test of Granger noncausality in distribution, as opposed to the tests of Granger noncausality in mean mentioned above. This allows researchers to capture the dependence due to both low and high-order moments and quantiles. Furthermore, Granger causality tests provide useful information on whether knowledge of past changes in x_t can improve short-run forecast of movements in y_t. A new nonparametric test statistic is considered, proposed by Bouezmarni and Taamouti (2014). The test is based on a comparison of the conditional distribution functions using an L_2 metric. In case researchers are interested in testing Granger noncausality in the distribution from x_{t-1} to y_t, the following null hypothesis is tested:

$$H_0 : \Pr\{E[y_t \mid y_{t-1,} x_{t-1}] = F(y_t \mid y_{t-1})]\} = 1$$

against the alternative hypothesis:

$$H_1 : \Pr\{E[y_t \mid y_{t-1}, x_{t-1}] = F(y_t \mid y_{t-1})]\} < 1$$

where $F(y_t|y_{t-1}, x_{t-1})$ is the conditional distribution function of y_t given y_{t-1} and x_{t-1}, and $F(y_t|y_{t-1})$ is the conditional distribution function of y_t given only y_{t-1}. If the null hypothesis is true, then the past changes in x_t cannot affect the conditional distribution of y_t. As $F(y_t|y_{t-1}, x_{t-1})$ and $F(y_{t+1}|y_t)$ are unknown, a nonparametric approach is used to estimate them. If we denote $\bar{U}_{t-1} = (y_{t-1}, x_{t-1})'$ and $\bar{u} = (y, x)'$ then the Nadaraya–Watson estimator of the conditional distribution of y_t given y_{t-1} and x_{t-1} is defined as:

$$\hat{C}_{h_1}(y_t \mid \bar{u}) = \left[\sum_{t=2}^{T} K_{h_1}(\bar{u} - \bar{U}_{t-1}) I_{A_{y_t}}(y_t)\right] \Big/ \left[\sum_{t=2}^{T} K_{h_1}(\bar{u} - \bar{U}_{t-1})\right]$$

where $K_{h_1}(.) = h_1^{-2} K(./h_1)$, $K(.)$ stands for a kernel function, $h_1 = h_{1,T}$ is a bandwidth parameter, and $I_{A_{y_t}}(.)$ is an indicator function, which is defined on the set $A_{y_t} = [y_t, +\infty]$. Similarly, the Nadaraya–Watson estimator of the conditional distribution of y_t given y_{t-1} is defined as:

$$\hat{C}_{h_2}(y_t \mid y) = \left[\sum_{t=2}^{T} K^*_{h2}(y - y_{t-1}) I_{A_{y_t}}(y_t)\right] \Big/ \left[\sum_{t=2}^{T} K^*_{h_2}(y - y_{t-1})\right]$$

where $K^*_{h_2}(.) = h_2^{-1} K^*(./h_2)$, for $K^*(.)$ a different kernel function, $h_2 = h_{2,T}$ is a different bandwidth parameter. To test the null hypothesis against the alternative hypothesis, the authors recommend the following test statistic:

$$\hat{A} = (1/T) \sum_{t=2}^{T} \left\{ \hat{C}_{h_1}\left(y_t | \bar{u}\right) - \hat{C}_{h2}\left(y_t | y\right) \right\}^2 w\left(\bar{U}_{t-1}\right)$$

where $w(.)$ is a nonnegative weighting function of the data $\bar{U}_{t-1}$ for $2 \le t \le T$. The test statistic $\hat{A}$ is close to zero, if conditionally on y_{t-1}, the variables y_t and x_{t-1} are independent, and it diverges in the opposite case. The authors have established the asymptotic distribution of the nonparametric test statistic. They show that the test is asymptotically pivotal under the null hypothesis and follows a normal distribution. As the distribution of the test statistic is valid only asymptotically, for finite samples they suggest using a local bootstrap version of the test statistic. The simple resampling from the empirical distribution will not conserve the conditional dependence structure in the data, thus, it is important to use the local

smoothed bootstrap suggested by Paparoditis and Politis (2000). The latter improves quite a lot the finite sample properties (size and power) of the test. As optimal bandwidths are not available, they have considered the bandwidths $h_1 = c_1 T^{-1/4.75}$ and $h_2 = c_2 T^{-1/4.25}$ for various values of c_1 and c_2. The empirical power of the test also performs quite well (Boxes 9.9 and 9.10).

BOX 9.9 Published Paper on Nonparametric Causality

The STUDY: Muhtaseb, B.M.A., Daoud, H.E., 2017. Tourism and economic growth in Jordan: evidence from linear and nonlinear frameworks. International Journal of Economics and Financial Issues 7, 214–223.

Data

Data for Jordan are on a quarterly basis, spanning the period 1998–2015, and are on real GDP in US$ and at constant 2005 prices and on real international tourism receipts expressed in constant US$. GDP data were obtained from the Central Bank of Jordan, while those on the tourism variable from the World Travel and Tourism Council.

Granger causality results and conclusions

Table 9.10 presents the results from the nonlinear causality testing between tourism and economic growth. The DP test indicates that there is a bidirectional nonlinear causality.

Table 9.10: The DP test.

Null Hypothesis	Statistic	*P*-value
International tourism receipts does not cause economic growth	1.71*	0.0015
Economic growth does not cause international tourism receipt	1.43*	0.0023

* Statistically significant at 1%.

BOX 9.10 Published Paper on Noncausality Through the Nonparametric Test Proposed by Nishiyama et al. (2011)

The STUDY: Bekiros, S., Gupta, R., 2015. Predicting Stock Returns and Volatility Using Consumption Aggregate Wealth Ratios: A Nonlinear Approach. Working Paper, No. 2015-05, Department of Economics, University of Pretoria.

Data

The value adjusted CRSP index (CRSP-VW), obtained from the Center for Research in Security Prices, is deflated by the personal consumption expenditure chain type price deflator (2009 = 100) to provide real stock prices. Stock returns are computed as the real log returns (rcrspr), and its volatility (rcrspv) as the squared values of the returns. The data span ranges from 1952 to 2013, it is on a quarterly basis and is obtained from Sydney Ludvigson's website. Their goal is to determine whether the consumption aggregate wealth ratio can cause both real stock returns and their volatility.

Causality results and conclusions

The results are reported in Table 9.11. As it can be seen, the consumption-based wealth ratio is found to cause both real stock returns and their volatility.

Table 9.11: Nonlinear causality test.

Dependent Variable	Test Statistic
Real stock returns	77.29**
Volatility of real stock returns	47.54**

** Significant at 5%.

8 Conclusion

This chapter surveyed all the relevant tests in causal analysis provided in the relevant literature, while emphasized inferences on linear time series and panel causality, on asymmetric causality and nonparametric causality. Moreover, the presentation of each test was accompanied by an empirical application that had been already published in the literature to depict the practical application of those testing methodologies in a clearer way. We do hope that readers will appreciate it.

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