Dynamics and Statistics of the Climate System Advance Access published October 12, 2016

Has natural variability a lagged influence on global temperature? A multi-horizon Granger causality analysis

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Abstract

At present the role of natural variability in influencing climate behaviour is widely discussed. The generally accepted view is that atmosphere-ocean coupled circulation patterns are able to amplify or reduce temperature increase from interannual to multidecadal time ranges, leaving the principal driving role to anthropogenic forcings. In this framework, the influence of these circulation patterns is considered synchronous with global temperature changes. Here we would like to investigate if there exists a lagged influence of these indices on temperature. In doing so, an extension of the Granger causality technique, which permits to test both direct and indirect causal influences, is applied. A lagged influence of natural variability is not evident in our analysis, if we except weak influences of some peculiar circulation indices in specific periods.

Keywords: Vector autoregressive models, Multi-horizon Granger causality, Global temperature, Natural variability.

1 Introduction

The search for the fundamental causes of the recent global warming has been extensively performed in the last decade by attribution studies through a dynamical approach using Global Climate Models (GCMs): see, for instance, Hegerl and Zwiers (2011). The results clearly indicate the necessity to consider anthropogenic forcings (and especially greenhouse gases radiative forcing - GHGRF) if one wants to recover the increase in global temperature (T) detected in the last half century (Bindoff et al., 2013).

However, several studies show how circulation patterns of the coupled atmosphere-ocean system can influence the evolution of temperature behaviour, possibly amplifying or reducing temperature increase over interannual or multidecadal time ranges (Hoerling et al., 2008; DelSole et al., 2011). Generally, in these studies the influences of these circulation patterns on global temperature appears to be synchronous (see, for instance, Hurrell, 1996; Comrie and McCabe, 2013).

Here we would like to investigate if there exists a lagged influence of these indices on global temperature. The motivation of our study relies on the evidence that these single oscillation patterns (here synthesized by indices based on Sea Surface Temperatures (SSTs)), usually show teleconnections with delayed effects on temperatures elsewhere, on very wide regions: see, for instance, previous studies about physically based relationships between Atlantic and Pacific SSTs (Saenko et al., 2004;Wang et al., 2011). Furthermore, the origins for the delayed increases in global surface temperature accompanying El Niño events have been explored, too (Trenberth et al., 2002). Finally, in other empirical attribution studies the values of circulation patterns have been also used as influence factors with a delayed effect on global temperature (Lean and Rind, 2008; Foster and Rahmstorf, 2011) even for indices here not considered, such as the North Atlantic Oscillation (Li et al., 2013).

In order to do so, we adopt a well-known technique for testing causality links, the so-called Granger causality analysis (Granger, 1969), which represents the appropriate statistical tool for identifying lagged influences. Recently this analysis method has been applied to several problems in climate science and extensively to the at-

tribution topic (Kaufmann and Stern, 1997; Diks and Mudelsee, 2000; Wang et al., 2004; Triacca, 2005; Modesale et al., 2006; Elsner, 2007; Kodra et al., 2010; Attanasio et al., 2012; Pasini et al., 2012; Triacca et al., 2013; Stern and Kaufmann, 2014; Pasini et al., 2015).

In particular, for the first time, we investigate both direct influences and indirect chains of causality, using a generalization of the notion of Granger causality proposed by Dufour and Renault (1998): the so-called multi-horizon causality. The results give new insight in the role of circulation patterns for temperature determination.

The paper is organized as follows. In Section 2 we introduce the notion of the multi-horizon causality. In Section 3 we describe the data and the used testing procedures. Section 4 presents the obtained results. Section 5 provides some concluding remarks.

2 Multi-horizon Granger causality

The notion of Granger causality was first introduced by Norbert Wiener (1956) and later reformulated and formalized by Clive Granger (1969). Conceptually, the idea of Granger causality is quite simple. Suppose that we have two variables, x, y, and a vector z of m auxiliary variables, and that we first attempt to forecast y_{t+1} using past terms of y and z. We then try to forecast y_{t+1} using past terms of y, x, and z. We say that x Granger causes y, if the second forecast is found to be more successful, according to standard cost functions. If the second prediction is better, then the past of x contains a useful information for forecasting y_{t+1} that is not in the past of y and z. Clearly, Granger causality is based on precedence and predictability.

If z is empty, we deal with a bivariate Granger causality, otherwise with a multivariate Granger causality. The causal relationship between the variables x and y has often been investigated in a bivariate system ($m = 0$). However, it is well known that in a bivariate framework problems of spurious causality and of noncausality due to omission of a relevant variable can arise. These problems can be solved if a vector z of auxiliary variables is considered in the analysis.

It is important to note that, when $m > 0$, the original definition of Granger could

be insufficient to capture indirect causal links since it only deals with one step-ahead prediction. If a causal chain from x to y via z ($x \to z \to y$) exists, it is possible that x fails to cause y for horizon 1 and causes y at horizon $h > 1$. Thus an extension of Granger causality is necessary in multivariate models. Following Dufour and Renault (1998), we will use the following definitions of noncausality.

Let (y_t, x_t, z_t)' be a (3×1) vector time series. Consider the following information sets:

$$I_{yxz}(t) = \{y_t, x_t, z_t, y_{t-1}, x_{t-1}, z_{t-1}, \ldots\}$$

$$I_{yx}(t) = \{y_t, x_t, y_{t-1}, x_{t-1}, \ldots\}$$

$$I_{yz}(t) = \{y_t, z_t, y_{t-1}, z_{t-1}, \ldots\}$$

$$I_{y}(t) = \{y_t, y_{t-1}, \ldots\}$$

For any positive integer h (the prediction horizon) we denote with $P(y_{t+h}|I(t))$ the optimal linear forecast of the variable y_{t+h} based on the information set $I(t)$.

We say that:

(i) x does not cause y at horizon h given $I_{yx}(t)$ (denoted $x \overset{h}{\nrightarrow} y|I_{yx}(t)$) if

$$P(y_{t+h}|I_y(t)) = P(y_{t+h}|I_{yx}(t)) \; \forall t \in \mathbb{Z}$$

(i.bis) x does not cause y at horizon h given $I_{yxz}(t)$ (denoted $x \overset{h}{\nrightarrow} y|I_{yxz}(t)$) if

$$P(y_{t+h}|I_{yz}(t)) = P(y_{t+h}|I_{yxz}(t)) \; \forall t \in \mathbb{Z}$$

(ii) x does not cause y up to horizon h given $I_{yx}(t)$ (denoted $x \overset{(h)}{\nrightarrow} y|I_{yx}(t)$) if

$$x \overset{k}{\nrightarrow} y|I_{yx}(t) \text{ for } k = 1, \ldots, h$$

(ii.bis) x does not cause y up to horizon h given $I_{yxz}(t)$ (denoted $x \overset{(h)}{\nrightarrow} y|I_{yxz}(t)$) if

$$x \overset{k}{\nrightarrow} y|I_{yxz}(t) \text{ for } k = 1, \ldots, h$$

We observe that the conditions (i) and (ii) are equivalent (see Dufour and Renault (1998, Proposition 2.3)).

It is important to underline that the causal relationship between two variables x and y is not guaranteed to be conserved when a third variable z is considered in the analysis. In particular, we can have the following situations:

- Spurious causality. $x \xrightarrow{1} y|I_{yx}(t)$ and $x \not\xrightarrow{1} y|I_{yxz}(t)$
- Spurious noncausality. $x \not\xrightarrow{1} y|I_{yx}(t)$ and $x \xrightarrow{1} y|I_{yxz}(t)$

Another interesting pattern of causality is the following:

- Indirect causality. $x \not\xrightarrow{1} y|I_{yxz}(t)$ and $x \xrightarrow{2} y|I_{yxz}(t)$

It is possible to show that if $x \not\xrightarrow{1} y|I_{yxz}(t)$ and $x \xrightarrow{2} y|I_{yxz}(t)$, then there exists a causal chain from x to y via z, that is $x \xrightarrow{1} z|I_{yxz}(t)$ and $z \xrightarrow{1} y|I_{yxz}(t)$ (see Propositions 2.3 and 2.4 of Dufour and Renault (1998)). Thus we have called this pattern indirect causality.

3 Data and Methodology

Here we deal with the annual time series for the period $1866 - 2011$:

- Global temperature anomalies (T) from the version 4 of the Hadley Centre/Climatic Research Unit combined land and marine surface temperature global anomalies, HadCRUT4 (Morice et al., 2012): data retrieved from `http://www.cru.uea.ac.uk/data/`;
- Atlantic Multidecadal Oscillation, AMO (Enfield et al., 2001): data available at `www.esrl.noaa.gov/psd/data/timeseries/AMO`;
- Pacific Decadal Oscillation, PDO (Smith and Reynolds, 2004): data available at `ftp.ncdc.noaa.gov/pub/data/ersstv2/pdo.1854.latest.st`;
- Southern Oscillation Index (SOI), related to ENSO (Ropelewski and Jones, 1987; Allan et al., 1991; Konnen et al., 1998): data available at `www.cru.uea.\\ac.uk/cru/data/soi/soi.dat`;

- GHGRF: it is the radiative forcing of the main greenhouse gases (CO_2, CH_4, N_2O). In particular CO_2, CH_4 and N_2O concentrations (Hansen et al., 2007): data are available at http://data.giss.nasa.gov (since 1850); greenhouses gases main (CO_2+CH_4+N_2O) radiative forcing (GHGRF) has been calculated as in Ramaswamy et al. (2001).

3.1 Bivariate analysis

We are interested to study, separately, Granger causality from AMO, PDO or SOI to global temperature anomalies. First of all, a bivariate Granger causality analysis is performed by means of the following unrestricted VAR model:

$$\begin{bmatrix} y_t \\ x_t \end{bmatrix} = \begin{bmatrix} \mu_1 \\ \mu_2 \end{bmatrix} + \sum_{j=1}^{k} \begin{bmatrix} \alpha_{11,j} & \alpha_{12,j} \\ \alpha_{21,j} & \alpha_{22,j} \end{bmatrix} \begin{bmatrix} y_{t-j} \\ x_{t-j} \end{bmatrix} + \begin{bmatrix} \varepsilon_{1t} \\ \varepsilon_{2t} \end{bmatrix} \tag{1}$$

where $\mu = (\mu_1, \mu_2)'$ is a vector of constants, $\alpha_{il,j}$ are fixed coefficients and $\varepsilon_t = (\varepsilon_{1t}, \varepsilon_{2t})'$ is a bivariate white noise process. In this framework, the variable x does not cause y at horizon 1 given $I_{yx}(t)$ if and only if $\alpha_{12,j} = 0$, for $j = 1, \ldots, k$. Therefore the null hypothesis of noncausality is given by

$$H_0 : \alpha_{12,j} = 0 \qquad j = 1, \ldots, k.$$

In order to test this hypothesis, we use an out-of-sample approach (see Ashley, 1988). Our sample of observations $(y_t, x_t)_{t=1}^{N}$ is divided into a training set and a test set. The test set is composed by the last P observations, while the training sample consists of all previous $R = N - P$ observations. In particular we consider the unrestricted model (1) and the following restricted model

$$\begin{bmatrix} y_t \\ x_t \end{bmatrix} = \begin{bmatrix} \gamma_1 \\ \gamma_2 \end{bmatrix} + \sum_{j=1}^{k} \begin{bmatrix} \beta_{11,j} & 0 \\ \beta_{21,j} & \beta_{22,j} \end{bmatrix} \begin{bmatrix} y_{t-j} \\ x_{t-j} \end{bmatrix} + \begin{bmatrix} w_{1t} \\ w_{2t} \end{bmatrix} \tag{2}$$

Considering the Mean Squared Prediction Errors (MSPEs)

$$\text{MSPE}\left(\varepsilon_{1t}\right) = \frac{1}{P} \sum_{t=R+1}^{R+P} \varepsilon_{1t}^2$$

$$\text{MSPE}\left(w_{1t}\right) = \frac{1}{P} \sum_{t=R+1}^{R+P} w_{1t}^2$$

the null hypothesis of Granger noncausality becomes

$$H_0 : \mathrm{E}\left[\mathrm{MSPE}\left(w_{1t}\right)\right] - \mathrm{E}\left[\mathrm{MSPE}\left(\varepsilon_{1t}\right)\right] = 0$$

where E is the expectation value operator.

The alternative hypothesis is that the restricted model provides a bigger MSPE than the unrestricted model.

Using the training set, the parameters of the models (1) and (2) are estimated by Ordinary Least Squared (OLS) and the P one-step-ahead forecast errors, for $t = R+1, \ldots, R+P$, are calculated as follows:

$$\hat{\varepsilon}_{1t} = y_t - \hat{\mu}_1 - \sum_{j=1}^{k} \hat{\alpha}_{11,j} y_{t-j} - \sum_{j=1}^{k} \hat{\alpha}_{12,j} x_{t-j}$$

$$\hat{w}_{1t} = y_t - \hat{\gamma}_1 - \sum_{j=1}^{k} \hat{\beta}_{11,j} y_{t-j}$$

Then we calculate the mean square prediction errors

$$\mathrm{MSPE}\left(\hat{\varepsilon}_{1t}\right) = \frac{1}{P} \sum_{t=R+1}^{R+P} \hat{\varepsilon}_{1t}^2$$

$$\mathrm{MSPE}\left(\hat{w}_{1t}\right) = \frac{1}{P} \sum_{t=R+1}^{R+P} \hat{w}_{1t}^2$$

In order to test the null hypothesis, we use two tests described in McCracken (2007): the MSE-t, commonly attributed to Diebold and Mariano (1995) or West (1996), and the MSE-REG tests, suggested by Granger and Newbold (1977).

Defining $d_t = \hat{\varepsilon}_{1t}^2 - \hat{w}_{1t}^2$, the MSE-t statistics is obtained by regressing d_t on a constant a on the test set, thus obtaining

$$\text{MSE-t} = \frac{\hat{a}}{se\left(\hat{a}\right)}$$

where $\hat{a}$ is the OLS estimate of a and $se\left(\hat{a}\right)$ is the $\hat{a}$'s standard error. Furthermore, in order to calculate MSE-REG statistics, we consider the following regression model:

$$\left(\hat{\varepsilon}_{1t} - \hat{w}_{1t}\right) = c\left(\hat{\varepsilon}_{1t} + \hat{w}_{1t}\right) + e_t$$

on the test set, where e_t is a white noise. The MSE-REG statistics can be thus evaluated by use of the t-statistics associated with the coefficient c, i.e.

$$\text{MSE-REG} = \frac{\hat{c}}{se\left(\hat{c}\right)}$$

where $\hat{c}$ is the OLS estimate of c and $se\left(\hat{c}\right)$ is the $\hat{c}$'s standard error.

In our case, we do not use the critical values described in McCracken (2007) because severale time series are not stationary (Kaufmann and Stern DI, 1997; Attanasio, 2012; Triacca et al., 2013). The critical values of the tests are calculated by means of the following bootstrap method (bootstrap based on residuals):

1. Calculate forecasts of the models (1) and (2) for the time series y_t using a forecast schemes.

2. Evaluate MSE-t and MSE-REG statistics.

3. Under the null hypothesis of non-causality, estimate the restricted model (2) employing the full sample and extract the estimates $\hat{\gamma}_j$, $\hat{\beta}_{lm,j}$ and the residuals $\hat{w}_t$.

4. Apply bootstrap procedure (resampling with replacement) on $\hat{w}_t$ and obtain the pseudo-residuals w_t^*.

5. Create the pseudo-data given by

$$\begin{bmatrix} y_t^* \\ x_t^* \end{bmatrix} = \begin{bmatrix} \hat{\gamma}_1 \\ \hat{\gamma}_2 \end{bmatrix} + \sum_{j=1}^{k} \begin{bmatrix} \hat{\gamma}_{11,j} & 0 \\ \hat{\gamma}_{21,j} & \hat{\gamma}_{22,j} \end{bmatrix} \begin{bmatrix} y_{t-j}^* \\ x_{t-j}^* \end{bmatrix} + \begin{bmatrix} w_{1t}^* \\ w_{2t}^* \end{bmatrix}$$

6. Using the pseudo-data, repeat the steps 1 and 2 calculating MSE-t and MSE-REG bootstrap statistics.

7. Repeat steps from 4 to 6 for M times (in our application we use a high value for M (10000), which should lead to avoid problems with the convergence of p-values).

8. Calculate the bootstrap p-values which is the proportion of the MSE-t (or MSE-REG) estimated bootstrap statistics that exceed the same statistic evaluated on the observed data.

The model order k, with $k \in \{1, 2, 3, 4\}$, of the unrestricted model (1) is selected by means of Akaike information criteria on the training set.

We close this subsection remembering that the problem of having to test for multi-horizon noncausality does not emerge in bivariate models. In fact, it is possible to show that x does not cause y at horizon 1 given $I_{yx}(t)$ if and only if x does not cause y at any horizon h given $I_{yx}(t)$. The reason is that in a bivariate system any causal effect of x on y must flow directly from x to y: a causal chain cannot exist.

3.2 Trivariate analysis

In this subsection, in order to investigate the various patterns of causality (spurious causality, spurious noncausality, indirect causality), we introduce the formalism for a trivariate analysis. In this case the unrestricted VAR(k) model becomes

$$\begin{bmatrix} y_t \\ x_t \\ z_t \end{bmatrix} = \begin{bmatrix} c_1 \\ c_2 \\ c_3 \end{bmatrix} + \sum_{j=1}^{k} \begin{bmatrix} \phi_{11,j} & \phi_{12,j} & \phi_{13,j} \\ \phi_{21,j} & \phi_{22,j} & \phi_{23,j} \\ \phi_{31,j} & \phi_{32,j} & \phi_{33,j} \end{bmatrix} \begin{bmatrix} y_{t-j} \\ x_{t-j} \\ z_{t-j} \end{bmatrix} + \begin{bmatrix} u_{1t} \\ u_{2t} \\ u_{3t} \end{bmatrix} \tag{3}$$

The variable x does not cause y at horizon 1 given $I_{yxz}(t)$ if and only if $\phi_{12,j} = 0$, for $j = 1, \ldots, k$. However, in this situation the causality may be indirect through z if $\phi_{13,j}$ and $\phi_{32,j}$ are not zero for some j. In this case, when x does not cause y at horizon 1 given $I_{yxz}(t)$, the existence of an indirect causality through z implies that x causes y at horizon 2 given $I_{yxz}(t)$. Giles (2002) proposed a sequential procedure that provides information on the horizon at which the causality, if any, arises. Here we use this procedure. First we test the null hypothesis

$$H_0^{(1)} : \phi_{12,1} = \ldots = \phi_{12,k} = 0$$

If this hypothesis is rejected, then $x \xrightarrow{1} y|I_{yxz}(t)$: we call this "direct causality". Otherwise the following conditional null hypotheses are tested

$$H_0^{(2)} : \phi_{32,1} = \ldots = \phi_{32,k} = 0 | \phi_{12,1} = \ldots = \phi_{12,k} = 0$$

$$H_0^{(3)} : \phi_{13,1} = \ldots = \phi_{13,k} = 0 | \phi_{12,1} = \ldots = \phi_{12,k} = 0$$

If $H_0^{(2)}$ and $H_0^{(3)}$ are rejected, then $x \xrightarrow{1} z|I_{yxz}(t)$, $z \xrightarrow{1} y|I_{yxz}(t)$ and $x \xrightarrow{1} y|I_{yxz}(t)$. Thus we conclude that $x \xrightarrow{2} y|I_{yxz}(t)$. While we accept $x \xrightarrow{2} y|I_{yxz}(t)$ when we accept one or both of the hypotheses $H_0^{(2)}$ and $H_0^{(3)}$.

Even in this case, in order to test the null hypotheses $H_0^{(1)}$, $H_0^{(2)}$ and $H_0^{(3)}$ we use the out-of-sample approach. The hypothesis $H_0^{(1)}$ is tested using the unrestricted model (3) and the following restricted model

$$\begin{bmatrix} y_t \\ x_t \\ z_t \end{bmatrix} = \begin{bmatrix} a_1 \\ a_2 \\ a_3 \end{bmatrix} + \sum_{j=1}^{k} \begin{bmatrix} \theta_{11,j} & 0 & \theta_{13,j} \\ \theta_{21,j} & \theta_{22,j} & \theta_{23,j} \\ \theta_{31,j} & \theta_{32,j} & \theta_{33,j} \end{bmatrix} \begin{bmatrix} y_{t-j} \\ x_{t-j} \\ z_{t-j} \end{bmatrix} + \begin{bmatrix} v_{1t} \\ v_{2t} \\ v_{3t} \end{bmatrix} \tag{4}$$

Estimating the parameters of these two models by means of OLS, we can obtain the P one-step-ahead forecast errors of the first equation of the two models and the mean square prediction errors MSPE $(\hat{u}_{1t})$ and MSPE $(\hat{v}_{1t})$, respectively. If MSPE $(\hat{v}_{1t}) >$ MSPE $(\hat{u}_{1t})$ and this difference is statistical significant, then the null hypothesis $H_0^{(1)}$ is rejected and we conclude that $x \xrightarrow{1} y|I_{yxz}(t)$.

Otherwise, indirect causality must be investigated. In this case we examine indirect Granger causality by means of the chain

$$x \xrightarrow{1} z|I_{yxz}(t) \text{ and } z \xrightarrow{1} y|I_{yxz}(t)$$

Thus we consider the VAR model (3) imposing $\phi_{12,j} = 0$, for $j = 1, \ldots, k$

$$\begin{bmatrix} y_t \\ x_t \\ z_t \end{bmatrix} = \begin{bmatrix} c_1 \\ c_2 \\ c_3 \end{bmatrix} + \sum_{j=1}^{k} \begin{bmatrix} \phi_{11,j} & 0 & \phi_{13,j} \\ \phi_{21,j} & \phi_{22,j} & \phi_{23,j} \\ \phi_{31,j} & \phi_{32,j} & \phi_{33,j} \end{bmatrix} \begin{bmatrix} y_{t-j} \\ x_{t-j} \\ z_{t-j} \end{bmatrix} + \begin{bmatrix} u_{1t} \\ u_{2t} \\ u_{3t} \end{bmatrix} \tag{5}$$

The null hypothesis $H_0^{(2)}$ can be tested considering the previous model (5) and the following model

$$\begin{bmatrix} y_t \\ x_t \\ z_t \end{bmatrix} = \begin{bmatrix} a_1 \\ a_2 \\ a_3 \end{bmatrix} + \sum_{j=1}^{k} \begin{bmatrix} \theta_{11,j} & 0 & \theta_{13,j} \\ \theta_{21,j} & \theta_{22,j} & \theta_{23,j} \\ \theta_{31,j} & 0 & \theta_{33,j} \end{bmatrix} \begin{bmatrix} y_{t-j} \\ x_{t-j} \\ z_{t-j} \end{bmatrix} + \begin{bmatrix} v_{1t} \\ v_{2t} \\ v_{3t} \end{bmatrix} \tag{6}$$

Estimating the model parameters *via* OLS, we obtain the P one-step-ahead forecast errors

$$\hat{u}_{3t} = z_t - \hat{c}_3 - \sum_{j=1}^{k} \hat{\phi}_{31,j} x_{t-j} - \sum_{j=1}^{k} \hat{\phi}_{32,j} y_{t-j} - \sum_{j=1}^{k} \hat{\phi}_{33,j} z_{t-j}$$

$$\hat{v}_{3t} = z_t - \hat{a}_3 - \sum_{j=1}^{k} \hat{\theta}_{31,j} x_{t-j} - \sum_{j=1}^{k} \hat{\theta}_{33,j} z_{t-j}$$

We calculate the mean square prediction errors MSPE $(\hat{u}_{3t})$ and MSPE $(\hat{v}_{3t})$ and test the null hypothesis $H_0^{(2)}$ employing the MSE-t and MSE-REG tests. The critical

values are always calculated by means of previous bootstrap method. Finally, the null hypothesis $H_0^{(3)}$ can be tested using the same procedure described for $H_0^{(2)}$.

It is important to underline that, assuming a $100\alpha_1\%$ significance level for a test of $H_0^{(1)}$ and a $100\alpha_2\%$ significance level for a test of $H_0^{(2)}$ and $H_0^{(3)}$, the overall size is bounded by $\alpha = \alpha_1 + 2\alpha_2$.

The model order k, with $k \in \{1, 2, 3, 4\}$, of the unrestricted VAR model in equation (3) is selected, considering the training set, by means of Akaike information criteria.

Finally, it is worthwhile to stress that in our study the forecasts are calculated by means of the fixed scheme. Under this scheme, each one-step-ahead forecast is generated using parameters that are estimated only once using data from 1 to R.

4 Results

In this framework, we analyze which pattern of variability (among those ones considered here) are able to have a lagged influence on global temperature T, which is our y variable, by means of Granger causality. Following the same approach used in previous papers (Attanasio et al., 2012; Pasini et al., 2012), the out-of-sample tests are performed on five test sets which span the following periods: 1941-2011, 1951-2011, 1961-2011, 1971-2011, 1981-2011. The bivariate results obtained by our analysis are very clear: if we take PDO as x variable in equation (1), the null hypothesis of Granger non-causality on T is often rejected (with only two exceptions) at a 5% significance level (Table 1). Otherwise there is a clear general evidence of Granger causality from AMO or SOI to global temperature. In fact the null hypothesis of non-causality is always rejected at 5% level (Tables 2 and 3).

In the trivariate case, we consider $z =$ GHGRF in order to study the robustness of the bivariate results. Previous studies (Attanasio et al. 2012; Pasini et al. 2013; Stern and Kaufmann 2014) have shown that GHGRF Granger causes global temperature.

Here we test both direct and indirect causality of circulation patterns on T. In the first case, we test the MSPEs coming from the unrestricted and restricted models (3) and (4). If no direct causality is found, the possibility of an indirect causality

through a causality chain is explored. The chains are formed by a first causal link between the oscillation pattern considered and GHGRF: a causal link between these variables is physically possible because of the influence of an oceanic release of GHGs in the atmosphere. The second link is obviously between GHGRF and T. If these two causal links should be both verified in a statistical significant manner by means of Granger tests, then an indirect causality arises even if no direct causality exists between a circulation pattern and T.

When PDO is considered as x in equation (3), we never find direct or indirect Granger causality (Table 4 and Table 5).

The other results are very impressive. In particular the bivariate outcomes of SOI and AMO are not statistically robust. In fact there is no direct Granger causality (with only one exception), at 5% significant level, from SOI to T in our test sets (Table 6). Even the causal chains are always interrupted (Table 7). However, it is worthwhile to note that in the fourth test set the empirical evidence that does not support the null hypothesis $H_0^{(1)}$ is not too strong. In fact the p-value is just slightly smaller than 0.05.

When x = AMO, the null hypothesis of non-causality is never rejected and we do not find a direct causality link (Table 8). In all test sets, the causal chains are not completed (Table 9).

In summary, we find direct Granger causality just for SOI on the fourth test set. Thus, the apparent causality from AMO and SOI to T, which we found in a bivariate framework, generally disappears when the most influent context causal variable - GHGRF - is inserted in the information set, even considering a possible causal chain through this variable. Nevertheless, weak signals of natural variability influence can be still recognized in single time intervals, namely the weaker influence of SOI in the more recent decades.

Finally, we should point out:

- the p-values of the MSE-REG test are very similar to those of the MSE-t test;
- using BIC (Bayesian Information Criterion) to select the VAR orders, the results of very low causality found employing AIC are further strengthened.

These results are available from the authors upon request.

5 Conclusions

In this paper we have analysed the causal role of the climate natural variability, here exemplified by three circulation patterns, on the behaviour of global temperature. In particular, once accepted the idea that a synchronous relationship exists between these patterns and global temperature, we have investigated the presence of possible lagged influences.

After a first evidence of strong causality for AMO and SOI, this has revealed itself as a spurious causality due to omission of variables in the information set considered. Once completed this set with data about greenhouse gases, the causality between natural variability and global temperature disappeared almost completely, even in the framework of the original analysis performed here about the role of possible indirect links.

In general, a lagged causal link from the indices of natural variability considered here to global temperature is not evident in our analysis, if we exclude some cases of weak influences in specific periods.

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Table caption

Table 1: Results of bivariate Granger noncausality tests from PDO to T.

Test set	Model Order	p-value
$[1941-2011]$	1	0.0071*
$[1951-2011]$	1	0.0093*
$[1961-2011]$	1	0.1383
$[1971-2011]$	1	0.3560
$[1981-2011]$	2	*nc*

nc indicates that the MSPE of the unrestricted model (1) is bigger than the MSPE of the restricted model (2), so that the MSE-t test is not calculated. * indicates that the null hypothesis is rejected at 5% significance level.

Table 2: Results of bivariate Granger noncausality tests from AMO to T.

Test set	Model Order	p-value
$[1941-2011]$	1	0.0001*
$[1951-2011]$	1	0.0020*
$[1961-2011]$	1	0.0017*
$[1971-2011]$	1	0.0010*
$[1981-2011]$	4	0.0043*

* indicates that the null hypothesis is rejected at 5% significance level.

Table 3: Results of bivariate Granger noncausality tests from SOI to T.

Test set	Model Order	p-value
$[1941-2011]$	2	0.0019*
$[1951-2011]$	2	0.0015*
$[1961-2011]$	2	0.0010*
$[1971-2011]$	2	0.0005*
$[1981-2011]$	2	0.0010*

* indicates that the null hypothesis is rejected at 5% significance level.

Table 4: Results of direct Granger noncausality tests from PDO to T in the trivariate system given by PDO, T and GHGRF.

Test set	Model Order	p-value
$[1941-2011]$	2	0.2564
$[1951-2011]$	2	0.0987
$[1961-2011]$	2	*nc*
$[1971-2011]$	2	*nc*
$[1981-2011]$	2	*nc*

nc indicates that the MSPE, for the equation of T, of the unrestricted model (3) is bigger than the MSPE of the restrected model (4), so that the MSE-t test is not calculated. * indicates that the null hypothesis is rejected at 5% significance level.

Table 5: Results of indirect Granger noncausality tests from PDO to T via GHGRF, in the trivariate system given by PDO, T and GHGRF.

Test set	Model Order	PDO $\overset{1}{\nrightarrow}$ GHGRF (p-value)	GHGRF $\overset{1}{\nrightarrow}$ T (p-value)
$[1941-2011]$	2	*nc*	0.0035*
$[1951-2011]$	2	0.1460	0.0065*
$[1961-2011]$	2	*nc*	0.0070*
$[1971-2011]$	2	*nc*	0.0082*
$[1981-2011]$	2	0.3577	0.0088*

nc indicates that the MSPE, for the equation of GHGRF, of the unrestricted model (5) is bigger than the MSPE of the restricted model (6), so that the MSE-t test is not calculated. * indicates that each null hypotheses is rejected at 2.5% significance level.

Table 6: Results of direct Granger causality from SOI to T in the trivariate system given by SOI, T and GHGRF.

Test set	Model Order	p-value
$[1941-2011]$	2	0.2429
$[1951-2011]$	2	0.3208
$[1961-2011]$	2	0.2191
$[1971-2011]$	2	0.0428*
$[1981-2011]$	3	0.1110

Table 7: Results of indirect Granger causality from SOI to T via GHGRF, in the trivariate system given by SOI, T and GHGRF.

Test set	Model Order	SOI $\overset{1}{\nrightarrow}$ GHGRF (p-value)	GHGRF $\overset{1}{\nrightarrow}$ T (p-value)
$[1941-2011]$	2	0.1651	0.0045*
$[1951-2011]$	2	*nc*	0.0071*
$[1961-2011]$	2	*nc*	0.0066*
$[1971-2011]$	2	–	–
$[1981-2011]$	3	*nc*	0.0289

nc indicates that the MSPE, for the equation of GHGRF, of the unrestricted model (5) is bigger than the MSPE of the restricted model (6), so that the MSE-t test is not calculated. * indicates that each null hypotheses is rejected at 2.5% significance level.

Table 8: Results of direct Granger causality from AMO to T in the trivariate system given by AMO, T and GHGRF.

Test set	Model Order	p-value
$[1941-2011]$	2	0.1720
$[1951-2011]$	2	0.2400
$[1961-2011]$	2	*nc*
$[1971-2011]$	2	0.1344
$[1981-2011]$	3	0.3461

nc indicates that the MSE, for the equation of T, of the unrestricted model (3) is bigger than the MSE of the restrected model (4), so the MSE-t test is not calculated. * indicates that the null hypothesis is rejected at 5% significance level.

Table 9: Results of indirect Granger causality from AMO to T via GHGRF, in the trivariate system given by AMO, T and GHGRF.

Test set	Model Order	AMO $\overset{1}{\nrightarrow}$ GHGRF (p-value)	GHGRF $\overset{1}{\nrightarrow}$ T (p-value)
$[1941-2011]$	2	*nc*	0.0047
$[1951-2011]$	2	*nc*	0.0081*
$[1961-2011]$	2	*nc*	0.0075*
$[1971-2011]$	2	0.9622	0.0097*
$[1981-2011]$	3	0.0754	0.0309

nc indicates that the MSPE, for the equation of GHGRF, of the unrestricted model (5) is bigger than the MSPE of the restricted model (6), so that the MSE-t test is not calculated. * indicates that each null hypotheses is rejected at 2.5% significance level.