Evidence for Existence of Real Causality in the Case of Zero Granger Causality

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Abstract—Granger causality is one of the most popular measures to reveal causality influence of time series and has been widely applied in economics and neuroscience due to its simplicity and easy implementation. In this paper, we first introduce two key assumptions which are showed to be pre-required for defining Granger causality; that is, without the two assumptions, one cannot draw a conclusion that there is no real causality even if we derive zero Granger causality. We then discuss two cases for which Granger causality is zero but there exists real causality between two time series. Therefore, researchers must be caution in drawing any conclusion based on Granger causality value.

I. INTRODUCTION

Given a set of time series, how to define causality influence among them has been a topic for over two thousand years and has yet to be completely resolved. In the literature one of the most popular definitions for causality is Granger causality (Granger causality). Due to its simplicity and easy implementation, Granger causality has been widely used. The basic idea of Granger causality was originally conceived by Wiener [47] and later formalized by Granger in the form of linear regression model [20]. It can be simply stated as follows: if the variance ($\sigma_{\epsilon_1}^2$) of the prediction error for the first time series at the present time is not less than the variance ($\sigma_{\eta_1}^2$) of the prediction error by including past measurements from the second time series in the linear regression model, then the second time series can be said to have a causal (driving) influence on the first time series. Reversing the roles of the two time series one repeats the process to address the question of driving in the opposite direction. Granger causality value of $\ln(\sigma_{\epsilon_1}^2/\sigma_{\eta_1}^2)$ is defined to describe the strength of the causality which the second time series has on the first one [13]–[15], [20], [22], [35]. From Granger causality value, it is clear that (i) $\ln(\sigma_{\epsilon_1}^2/\sigma_{\eta_1}^2) = 0$ when there is no causal influence from the second time series to the first one and $\ln(\sigma_{\epsilon_1}^2/\sigma_{\eta_1}^2) > 0$ when there is; (ii) the larger the value of $\ln(\sigma_{\epsilon_1}^2/\sigma_{\eta_1}^2)$, the higher causal influence. In recent years there has been significant interest to discuss causal interactions between brain areas which are highly complex neural networks. For instance, with Granger causality analysis Freiwald et al. [14] revealed the

existence of both uni- and bi-directional influences between neural groups in the macaque inferotemporal cortex. Hesse et al. [22] analyzed EEG data from the Stroop task and disclosed that conflict situations generate dense webs of interactions directed from posterior to anterior cortical sites and the web of directed interactions occurs mainly 400 ms after the stimulus onset and lasts up to the end of the task. Roebroeck et al. [38] explored directed causal influences between neuronal populations in fMRI data. Oya et al. [35] demonstrated causal interactions between auditory cortical fields in humans through intracranial evoked potentials to sound. Gow et al. [19] applied Granger analysis to MRI constrained MEG and EEG data to explore the influence of lexical representation on the perception of ambiguous speech sounds. Gow et al. [18] showed a consistent pattern of direct posterior superior temporal gyrus influence over sites distributed over the entire ventral pathway for words, non-words, and phonetically ambiguous items.

In this paper, we point out that Granger causality in time domain cannot correctly reveal real causality between two time series in some cases. To do so, we introduce two obvious key assumptions for defining Granger causality. The two assumptions are based on the two estimated models (auto-regression model and joint regression model) of two time series and require checking re-estimation problem of realizations of the estimated joint regression model. Only when the two assumptions are satisfied, zero Granger causality does mean no real causality. Without the two assumptions, one cannot draw a conclusion that there is no real causality even if we derive zero Granger causality. Therefore, checking re-estimation problem of realizations of the estimated joint regression model is very important when researchers obtain zero Granger causality in the study of time series data. We then gave two cases for which Granger causality is zero but there exists real causality between two time series. Therefore, researchers must be caution in drawing any conclusion based on Granger causality value in time domain.

This paper is organized as follows. Causality analysis in time domain and frequency domain are discussed in Section II and Section III respectively. Several examples are provided in Section IV. Concluding remarks are given in Section V. The rest of this paper is organized as follows: Section II derives an equivalent linear programming (LP) formulation of KWTa, which is suitable for the neural network design. Section III introduces the neural network design procedure, architecture and properties. Some simulation results are presented in Section IV to show its performance. Finally, Section V concludes the paper.

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II. GRANGER CAUSALITY

In this section we first introduce the well-known Granger causality and conditional Granger causality. We begin with bivariate time series. Given two stochastic process $X_1(t)$ and $X_2(t)$ which are assumed to be jointly stationary. Their autoregressive representations are described as

$$\begin{cases} X_{1,t} = \sum_{j=1}^m \mathbf{a}_{11,j} X_{1,t-j} + \epsilon_{1,t} \\ X_{2,t} = \sum_{j=1}^m \mathbf{a}_{22,j} X_{2,t-j} + \epsilon_{2,t} \end{cases} \quad (1)$$

and their joint representations are described as

$$\begin{cases} X_{1,t} = \sum_{j=1}^m a_{11,j} X_{1,t-j} + \sum_{j=1}^m a_{12,j} X_{2,t-j} + \eta_{1,t} \\ X_{2,t} = \sum_{j=1}^m a_{21,j} X_{1,t-j} + \sum_{j=1}^m a_{22,j} X_{2,t-j} + \eta_{2,t} \end{cases} \quad (2)$$

where $t = 0, 1, \dots, N$, the noise terms are uncorrelated over time, ϵ_i and η_i have zero means and variances of $\sigma_{\epsilon_i}^2$ and $\sigma_{\eta_i}^2$, $i = 1, 2$. The covariance between η_1 and η_2 is defined by $\sigma_{\eta_1\eta_2} = \text{cov}(\eta_1, \eta_2)$.

Now consider the first equalities in (1) and (2). According to the original formulations in [20] and [47], if $\sigma_{\eta_1}^2$ is less than $\sigma_{\epsilon_1}^2$ in some suitable statistical sense, then X_2 is said to have a causal influence on X_1 . In this case, the first equality in (2) is more accurate than that in (1) to estimate X_1 . Otherwise, if $\sigma_{\eta_1}^2 = \sigma_{\epsilon_1}^2$, then X_2 is said to have no causal influence on X_1 . In this case, the two equalities are almost same. Such kind of causal influence called Granger causality (Granger causality) [13], [15], can be defined by

$$F_{X_2 \rightarrow X_1} = \ln \frac{\sigma_{\epsilon_1}^2}{\sigma_{\eta_1}^2}. \quad (3)$$

Obviously, $F_{X_2 \rightarrow X_1} = 0$ when there is no causal influence from X_2 to X_1 and $F_{X_2 \rightarrow X_1} > 0$ when there is. Similarly, The causal influence from X_1 to X_2 is defined by

$$F_{X_1 \rightarrow X_2} = \ln \frac{\sigma_{\epsilon_2}^2}{\sigma_{\eta_2}^2}. \quad (4)$$

To show whether the interaction between two time series is direct or is mediated by another recorded time series, conditional Granger causality [13], [16], was defined by

$$F_{X_2 \rightarrow X_1 | X_3} = \ln \frac{\sigma_{\epsilon_3}^2}{\sigma_{\eta_3}^2} \quad (5)$$

where $\sigma_{\epsilon_3}^2$ and $\sigma_{\eta_3}^2$ are variances of two noise terms, ϵ_3 and η_3 , of the following two joint autoregressive representations:

$$X_{1,t} = \sum_{j=1}^m \mathbf{a}_{11,j} X_{1,t-j} + \sum_{j=1}^m \mathbf{a}_{13,j} X_{3,t-j} + \epsilon_{3,t} \quad (6)$$

and

$$\begin{aligned} X_{1,t} &= \sum_{j=1}^m a_{11,j} X_{1,t-j} + \sum_{j=1}^m a_{12,j} X_{2,t-j} \\ &+ \sum_{j=1}^m a_{13,j} X_{3,t-j} + \eta_{3,t}. \end{aligned} \quad (7)$$

According to this definition, $F_{X_2 \rightarrow X_1 | X_3} = 0$ means that no further improvement in the prediction of X_1 can be expected by including past measurements of X_2 . On the other hand, when there is still a direct component from X_2 to X_1 , the past measurements of X_1, X_2, X_3 together result in better prediction of X_1 , leading to $\sigma_{\eta_3}^2 < \sigma_{\epsilon_3}^2$, and $F_{X_2 \rightarrow X_1 | X_3} > 0$.

In above Granger causality and conditional Granger causality, both of them emphasize that zero causality comes from no further improvement in the prediction of X_1 by including past measurements of X_2 . As such, in his early work [15] Geweke stated that Granger causality $F_{X_2 \rightarrow X_1} = 0 \Leftrightarrow \sigma_{\epsilon_1}^2 = \sigma_{\eta_1}^2 \Leftrightarrow a_{12,j} \equiv 0$. According to this statement, if $a_{12,j} \equiv 0$, then there is no causality (or Granger causality). If $a_{12,j} \neq 0$, then there is Granger causality. In this paper, we will focus on Granger causality (similar discussions for conditional Granger causality) and show that Granger causality being zero does not necessarily mean no real causality between two time series, in other words, there may still exist real causality between two time series in the case of zero Granger causality. To do so, we point out the following two obvious key assumptions for defining Granger causality from X_2 to X_1 .

Assumption 1: After we obtain two estimated Model (1) and Model (2) for two given time series, for any realization of the estimated Model (2) we estimate again and get the same Model (1) and Model (2) as before, that is, all coefficients keep unchanged.

Assumption 2: If $a_{12,j} \equiv 0$, then in the estimated Model (1) (or Model (2) in this case), $X_{1,t-j}$ has nothing to do with X_2 's past values.

Only in above assumptions, we can say $F_{X_2 \rightarrow X_1} = 0 \Leftrightarrow a_{12,j} \equiv 0$; that is, to determine whether or not there is causal influence from X_2 to X_1 we only need to check whether or not $a_{12,j} \equiv 0$. If $a_{12,j} \equiv 0$, then surely we can have $\sigma_{\epsilon_1}^2 = \sigma_{\eta_1}^2$ resulting in $F_{X_2 \rightarrow X_1} = 0$. As usual, all researchers directly calculate Granger causality based on the two estimated Model (1) and Model (2) for two given time series and do not check re-estimation problem of realizations of the estimated Model (2) at all. In the following, we will show that the situation may change without above assumptions. That is, without above assumptions, Granger causality $F_{X_2 \rightarrow X_1} = 0$ does not necessarily mean $a_{12,j} \equiv 0$ and thus there exists real causality from X_2 to X_1 .

III. TWO CASES FOR ZERO GRANGER CAUSALITY

In this section we will analyze the following two cases for Model (2): **Case A.** $\eta_{2,t} \equiv 0$. **Case B.** $\eta_{1,t} = \eta_{2,t}$. We will show $F_{X_2 \rightarrow X_1} = 0$ in these two cases and there exist real causalities for both of two cases. To do so, let's introduce spectral Granger causality as follows.

Spectral Granger causality ([8], [12], [13], [15], [16]): Given the bivariate Model (2), the Granger casual influence from X_2 to X_1 is defined by

$$\begin{aligned} I_{X_2 \rightarrow X_1}(f) &= -\log \left(1 - \frac{\left(\sigma_{\eta_2}^2 - \frac{\sigma_{\eta_1\eta_2}^2}{\sigma_{\eta_1}^2} \right) |H_{12}(f)|^2}{S_{X_1 X_1}} \right) \\ &\in [0, +\infty) \end{aligned} \quad (8)$$

where the transfer function is $H(f) = A^{-1}(f)$ whose components are

$$\begin{aligned} H_{11}(f) &= \frac{1}{\det(A)} \bar{a}_{22}(f), H_{12}(f) = -\frac{1}{\det(A)} \bar{a}_{12}(f), \\ H_{21}(f) &= -\frac{1}{\det(A)} \bar{a}_{21}(f), H_{22}(f) = \frac{1}{\det(A)} \bar{a}_{11}(f), \end{aligned} \quad (9)$$

$$A = [\bar{a}_{ij}]_{2 \times 2}, \bar{a}_{kk}(f) = 1 - \sum_{j=1}^m a_{kk,j} e^{-i2\pi f j}, k = 1, 2,$$

$$\bar{a}_{hl}(f) = - \sum_{j=1}^m a_{hl,j} e^{-i2\pi f j}, h, l = 1, 2, h \neq l.$$

For the above two cases and for any coefficients $a_{11,j}, a_{12,j}, a_{21,j}, a_{22,j}$, we can check $I_{X_2 \rightarrow X_1}(f) \equiv 0$ based on (8). On the other hand, noting the relationship (17.29) in [13] between time and frequency domains, we can easily see Granger causality $F_{X_2 \rightarrow X_1} = 0$ for both of Case A and Case B, that is, no Granger causality. Now one question is arising: Does there exist real causality for Case A or Case B? the answer is Yes. Let's discuss each case in detail as follows.

Case A. $\eta_{2,t} \equiv 0$. Let's consider the following very simple model:

$$\begin{cases} X_{1,t} = -0.99X_{2,t-1} + \eta_{1,t} \\ X_{2,t} = -X_{2,t-2} \end{cases} \quad (10)$$

where η_1 is a white noise process with zero mean and variance $\sigma_{\eta_1}^2 = 1$. Since $\eta_{2,t} \equiv 0$ we have $F_{X_2 \rightarrow X_1} = 0$. No Granger causality! However, by observing the first equality of (10), real causal influence from X_2 to X_1 indeed exists. Moreover, for one realization (called realization A) of Model (10) with X_2 's initial conditions being $X_{2,1} = 6.0869, X_{2,2} = -1.3372$, we plot trajectories $X_{1,t}$ and $\eta_{1,t}$ in Figure 1A and 1B from which one can clearly see that X_1 has larger amplitudes compared with η_1 . So, what causes such kind of larger amplitudes for X_1 ? Of course, it is X_2 because there is no X_1 's past value at all in Model (10). For another realization (called realization B) of Model (10) with X_2 's initial conditions being $X_{2,1} = X_{2,2} = 0$ and $\eta_{1,t}$ being same as in realization A, we can see $X_{1,t} = \eta_{1,t}$ in this case. So, what causes the big change of amplitudes of X_1 from realization A to realization B? Of course, it is due to the change of X_2 (i.e., the change of the initial conditions $X_{2,1}$ and $X_{2,2}$). Realization A and realization B further support the existence of causal influence from X_2 to X_1 . Now one may ask: now that there exists real causal influence from X_2 to X_1 , why we get Granger causality $F_{X_2 \rightarrow X_1} = 0$? The reason is as follows: for any realization of Model (10) when we estimate autoregression model, we get

$$\begin{cases} X_{1,t} = \sum_{j=1}^m a_j X_{1,t-j} + \epsilon_{1,t} \\ X_{2,t} = -X_{2,t-2} \end{cases} \quad (11)$$

where $\sigma_{\epsilon_1}^2 = \sigma_{\eta_1}^2 = 1$ (resulting in $F_{X_2 \rightarrow X_1} = \ln \frac{\sigma_{\epsilon_1}^2}{\sigma_{\eta_1}^2} = 0$), which means no any improvement when using the joint regression Model (10). Moreover, for different realization, one gets different coefficient a_j , which means that a_j is

related to initial conditions of X_2 . Now we rewrite Model (11) as

$$\begin{cases} X_{1,t} = \sum_{j=1}^m a_j X_{1,t-j} + 0 \times X_{2,t-1} + \epsilon_{1,t} \\ X_{2,t} = -X_{2,t-2}. \end{cases} \quad (12)$$

Could we say X_2 has no causal influence on X_1 based on Model (12) because the coefficient of X_2 in the first equality is zero? No, absolutely no. Note that a_j keep changed for different realizations. So, above Assumption 1 is violated and Model (12) is different from Model (2) which satisfies the assumption and then is used to define Granger causality. Hence, from another aspect, Model (12) further confirms the fact that there exists causal influence from X_2 to X_1 .

Fig. 1. A) Trajectory $X_{1,t}$ of one realization of above Model (10). B) Trajectory $\eta_{1,t}$ of the same realization as in A).

Case B. $\eta_{1,t} = \eta_{2,t}$.

Let's consider the following very simple model:

$$\begin{cases} X_{1,t} = -0.99X_{2,t-1} + \eta_{1,t} \\ X_{2,t} = \eta_{1,t} \end{cases} \quad (13)$$

where η_1 is a white noise process with zero mean and variance $\sigma_{\eta_1}^2 = 1$. Since $\eta_{2,t} = \eta_{1,t}$ one can obtain Granger causality $F_{X_2 \rightarrow X_1} = 0$. No Granger causality! However, if we rewrite Model (13) as

$$\begin{cases} X_{1,t} = -0.99X_{2,t-1} + X_{2,t} \\ X_{2,t} = \eta_{1,t}, \end{cases} \quad (14)$$

then one can clearly see that X_1 is completely determined by X_2 ; that is, $X_{1,t}$ is determined by two parts: $-0.99X_{2,t-1}$ and $X_{2,t}$. The first part is called to have causal influence on X_1 from X_2 's past value. The second is called to

have instantaneous causal (see the concept of instantaneous causality in [13]) influence on X_1 from X_2 's current value. Usually, causal influence from X_2 to X_1 is talking about the first kind of causal influence. Thus, real causal influence from X_2 (i.e., X_2 's past value) to X_1 indeed exists by noting that X_2 does carry predictive information about X_1 (i.e., the term $0.99X_{2,t-1}$). On the other hand, in the following, from other aspects, we will show the existence of causal influence from X_2 to X_1 . For one realization of Model (13) we plot trajectories $X_{1,t}$ and $\eta_{1,t}$ in Figure 2A and 2B from which one can clearly see that X_1 has larger amplitudes compared with η_1 . What causes such kind of larger amplitudes for X_1 ?

Fig. 2. A) Trajectory $X_{1,t}$ of one realization of above Model (13). B) Trajectory $\eta_{1,t}$ of the same realization as in A).

Of course, it is X_2 because there is no X_1 's past value at all in Model (13). Now one question is arising: now that there exists real causal influence from X_2 to X_1 , why we get Granger causality $F_{X_2 \rightarrow X_1} = 0$? The reason is as follows: for any realization of Model (13) when we estimate autoregression model for X_1 , we get

$$X_{1,t} = \sum_{j=1}^m a_j X_{1,t-j} + \epsilon_{1,t} \quad (15)$$

where $\sigma_{\epsilon_1}^2 = \sigma_{\eta_1}^2 = 1$ (resulting in $F_{X_2 \rightarrow X_1} = \ln \frac{\sigma_{\epsilon_1}^2}{\sigma_{\eta_1}^2} = 0$), which means no any improvement when using the joint regression Model (13). Now we rewrite Model (15) as

$$X_{1,t} = \sum_{j=1}^m a_j X_{1,t-j} + 0 \times X_{2,t-1} + \epsilon_{1,t}. \quad (16)$$

Could we say X_2 has no causal influence on X_1 based on Model (16) because the coefficient of X_2 is zero? No,

absolutely no. Note that $X_{1,t-j}$ at the right hand side of Model (16) is closely related to X_2 's past values based on the relation in (13). Assumption 2 is violated. Thus, Model (16) is different from the first equality in Model (2). This further confirms the fact that there exists real causal influence from X_2 to X_1 for Model (13).

IV. DISCUSSIONS

In preceding section, we gave two cases under which Granger causality is zero, but real causality may still exist. The main reason lies in the fact that both of the two cases violate Assumptions 1 and 2 respectively. This again confirms that Assumptions 1 and 2 are pre-required for introducing Granger causality. Otherwise, without the two assumptions, Granger causality may lead to misinterpretation results. Therefore, when researchers conduct Granger causality analysis for time series in economics and neuroscience, the first step is to check whether or not the estimated Model (1) and Model (2) satisfy the two assumptions. Only when the two assumptions are satisfied, then one can use Granger causality to reveal whether or not there is causality influence between two series. If one does not check the assumptions, one cannot make a judgement to say there is no real causality between two time series in the case of zero Granger causality.

V. CONCLUSIONS

In human cognitive neuroscience, researchers are now not limited to only the study of the putative functions of particular brain regions, but moving toward how different brain regions (such as visual cortex, parietal or frontal regions, amygdala, or hippocampus) may causally influence on each other. These causal influences with different strengths and directionalities may reflect the changing functional demands on the network to support cognitive and perceptual tasks. Granger causality, one of the most popular causality measures, was initially widely used in economics and has recently received growing attention in neuroscience to reveal causal interactions for neurophysiological data. Especially in frequency domain several variant forms of Granger causality have been developed to study causal influences for neurophysiological data in different frequency decompositions during cognitive and perceptual tasks.

In our previous work [25] we proposed new spectral causality, pointed out shortcomings and/or limitations of spectral Granger causality, and showed advantages our new spectral causality over spectral Granger causality. In this paper, we discussed Granger causality in time domain. We first mentioned two assumptions which are showed to be pre-required for defining Granger causality; that is, without the two assumptions, one cannot draw a conclusion that there is no real causality even if we derive zero Granger causality. We then gave two cases for which Granger causality is zero but there exists real causality between two time series. Therefore, researchers must be caution in drawing any conclusion based on Granger causality value in time and frequency domains.

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