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Causality analysis of neural connectivity: New tool and limitations of spectral Granger causality ☆

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ABSTRACT

Granger causality (GC) is one of the most popular measures to reveal causality influence of time series based on the estimated linear regression model and has been widely applied in economics and neuroscience due to its simplicity, understandability and easy implementation. Especially, its counterpart in frequency domain, spectral GC, has recently received growing attention to study causal interactions of neurophysiological data in different frequency ranges. In this paper, on the one hand, for one equality in the linear regression model (frequency domain) we point out that all items at the right-hand side of the equality make contributions (thus have causal influence) to the unique item at the left-hand side of the equality, and thus a reasonable definition for causality from one variable to another variable (i.e., the unique item) should be able to describe what percentage the variable occupies among all these contributions. Along this line, we propose a new spectral causality definition. On the other hand, we point out that spectral GC has its inherent limitations because of the use of the transfer function of the linear regression model and as a result may not reveal real causality at all and lead to misinterpretation result. By one example we demonstrate that the results of spectral GC analysis are misleading but the results from our definition are much reasonable. So, our new tool may have wide potential applications in neuroscience.

1. Introduction

Given a set of time series, how to define causality influence among them has been a topic for over 2000 years and has yet to be completely resolved so far. In the literature one of the most popular definitions for causality is Granger causality (GC). The basic idea of GC was initially conceived by [1] and later formalized by Granger in the form of linear regression model [2]. It can be simply stated as follows: if the variance of the prediction error for the second time series at the present time is reduced by including past measurements from the first time series in the linear regression model, then the first time series can be said to have a causal (driving) influence on the second time series. Reversing the roles of the two time series one repeat the process to address the question of driving in the opposite direction. Due to its simplicity, understandability, easy implementation, GC has been widely used in economics. In recent years there has been growing interest to discuss causal interactions in neuroscience. For instance, with GC analysis Oya et al. [3] demonstrated causal interactions between auditory cortical fields in humans through intracranial evoked potentials to sound. Gow et al. [4] showed a consistent pattern of direct posterior superior temporal gyrus influences over sites distributed over the entire ventral pathway for words, non-words, and phonetically ambiguous items. Since frequency decompositions are often of particular interest for neurophysiological data, the original GC in time domain has been extended to spectral domain. Several spectral Granger or Granger-alike causality tools have been developed such as spectral GC [5], partial directed coherence (PDC) [6], relative power contribution (RPC) [7], directed transfer function (DTF) [8], short-time direct directed transfer function (SdDTF) [9], etc. The applications of these tools to neural data have yielded many promising results. For example, Brovelli et al. [10] applied spectral GC to identify causal influences from primary somatosensory cortex to motor cortex in the beta band (15–30 Hz) frequency during lever pressing by awake monkeys. Sato et al. [11] applied PDC to fMRI to discriminate physiological and nonphysiological components based on their frequency characteristics. Yamashita et al. [7] applied RPC to evaluate frequency-wise directed connectivity of BOLD signals. Kaminski and Liang [12] applied short-time DTF to show the predominant direction of influence from hippocampus

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to supramammilary nucleus (SUM) at the theta band (3.7–5.6 Hz) frequency. Korzeniewska et al. [9] used SdDTF to ECoG recordings to reveal frequency-dependent interactions, particularly in high gamma (>60 Hz) frequencies, between brain regions known to participate in the recorded language task.

The above spectral Granger or Granger-alike causality definitions are based on the transfer function matrix (or its inverse matrix) of the linear regression model. For two time series, all these definitions are equivalent to show whether one time series has causal influence on the other time series. The spectral GC definition can only be used to two time series. The other Granger-alike causality definitions can be applied to multi-dimensional time series. In this paper, we first propose a causality definition in frequency domain for the linear regression model and then take spectral GC as example and show shortcomings and/or limitations of spectral GC.

2. Definition for causality in frequency domain

Consider the following general model:

$$\begin{cases} X_{1,t}=\sum_{j=1}^{m}a_{11,j}X_{1,t-j}+\sum_{j=1}^{m}a_{12,j}X_{2,t-j}+\cdots+\sum_{j=1}^{m}a_{1n,j}X_{n,t-j}+\eta_{1,t} \\ X_{2,t}=\sum_{j=1}^{m}a_{21,j}X_{1,t-j}+\sum_{j=1}^{m}a_{22,j}X_{2,t-j}+\cdots+\sum_{j=1}^{m}a_{2n,j}X_{n,t-j}+\eta_{2,t} \\ \vdots \\ X_{n,t}=\sum_{j=1}^{m}a_{n1,j}X_{1,t-j}+\sum_{j=1}^{m}a_{n2,j}X_{2,t-j}+\cdots+\sum_{j=1}^{m}a_{nn,j}X_{n,t-j}+\eta_{n,t} \end{cases} \quad (1)$$

where $X_i (i=1,\ldots,n)$ are n time series, $t=0,1,\ldots,N$, η_i has zero mean and variance of $\sigma^2_{\eta_i}$ and $\sigma_{\eta_i\eta_k}=\mathrm{cov}(\eta_i,\eta_k), i,\ k=1,\ldots,n$.

Taking Fourier transformation on both side of Eq. (1) leads to

$$\begin{cases} X_1(f)=a_{11}(f)X_1(f)+a_{12}(f)X_2(f)+\cdots+a_{1n}(f)X_n(f)+\eta_1(f) \\ X_2(f)=a_{21}(f)X_1(f)+a_{22}(f)X_2(f)+\cdots+a_{2n}(f)X_n(f)+\eta_2(f) \\ \vdots \\ X_n(f)=a_{n1}(f)X_1(f)+a_{n2}(f)X_2(f)+\cdots+a_{nn}(f)X_n(f)+\eta_n(f) \end{cases} \quad (2)$$

where

$$a_{lj}(f)=\sum_{k=1}^{m}a_{lj,k}e^{-i2\pi fk},\quad i=\sqrt{-1}, l,\ j=1,\ldots,n \quad (3)$$

From (2) one can see that contributions to $X_k(f)$ not only include $a_{k1}(f)X_1(f),\ldots,a_{kk-1}(f)X_{k-1}(f),a_{kk+1}(f)X_{k+1}(f),\ldots,a_{kn}(f)X_n(f)$ and the noise term $\eta_k(f)$, but also include $a_{kk}(f)X_k(f)$. Fig. 1 intuitively describes the contributions to $X_k(f)$. Each contribution plays an important role in determining $X_k(f)$. If $a_{ki}(f)X_i(f)$ occupies larger portion among all those contributions, then X_i has stronger causality on X_k, or vice versa. Thus, a good definition for causality from X_i to X_k in frequency domain should be able to describe what percentage X_i occupies among all these contributions. Motivated by this idea, a direct causality from X_i to X_k in frequency domain can be defined as follows:

$$N_{X_i\overset{\mathrm{D}}{\to}X_k}(f)=\frac{|a_{ki}(f)|\sqrt{S_{X_iX_i}(f)}}{\sqrt{|a_{k1}(f)|^2S_{X_1X_1}(f)+|a_{k2}(f)|^2S_{X_2X_2}(f)+\cdots+|a_{kn}(f)|^2S_{X_nX_n}(f)+\sigma^2_{\eta_k}}} \quad (4)$$

$i,k=1,\ldots,n, i\neq k$, where $S_{X_iX_l}(f)$ is the spectrum of $X_l, l=1,\ldots,n$.

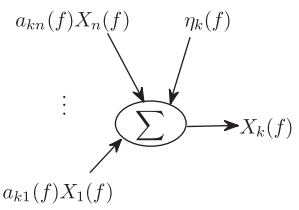

Fig. 1. Contributions to $X_k(f)$.

Remark 1. (i) It is easy to see that $0\le N_{X_i\overset{\mathrm{D}}{\to}X_k}(f)\le 1$. $N_{X_i\overset{\mathrm{D}}{\to}X_k}(f)\equiv 0$ if and only if $a_{ki}(f)\equiv 0$ which means all coefficients $a_{ki,1},\ldots,a_{ki,m}$ are zeros. $N_{X_i\overset{\mathrm{D}}{\to}X_k}(f)\equiv 1$ if and only if $\sigma^2_{\eta_k}=0$ and $a_{kj}(f)\equiv 0$, $j=1,\ldots,n, j\neq i$ which means there is no noise term η_k and all coefficients $a_{kj,1},\ldots,a_{kj,m}$ are zeros $j=1,\ldots,n, j\neq i$, that is, the kth equality in Model (2) can be written as

$$X_{k,t}=\sum_{j=1}^{m}a_{ki,j}X_{i,t-j}$$

from which one can see that X_k is completely driven by X_i's past values. (ii) Once Model (1) is evaluated based on the n time series $X_1,\ldots,X_n$, from (4) the direct bidirectional causalities between any two channels can be obtained. Based on this definition, the indirect causality from X_i to X_k via X_l may be defined as

$$N_{X_i\overset{\mathrm{ID}}{\to}X_k\ \text{via}\ X_l}(f)=N_{X_i\overset{\mathrm{D}}{\to}X_l}(f)\times N_{X_l\overset{\mathrm{D}}{\to}X_k}(f)$$

(iii) Given any route $R: X_i\to X'_{l_1}\to X'_{l_2}\to\cdots\to X'_{l_h}\to X_k$ where $\{X'_{l_1},\ldots,X'_{l_h}\}\subseteq\{X_1,\ldots,X_n\}-\{X_i,X_k\}$, the indirect causality from X_i to X_k via this route R may be defined as

$$N_{X_i\overset{\mathrm{ID}}{\to}X_k\ \text{via route}\ R}(f)=N_{X_i\overset{\mathrm{D}}{\to}X'_{l_1}}(f)\times\prod_{s=1}^{h-1}N_{X'_{l_s}\overset{\mathrm{D}}{\to}X'_{l_{s+1}}}(f)\times N_{X'_{l_h}\overset{\mathrm{D}}{\to}X_k}(f)$$

In the literature, there are several other measures to define Granger and Granger-alike causality in the frequency domain such as spectral GC [5,6], RPC [7], DTF [8], and SdDTF [9]. In the following, we will take spectral GC as an example and point out its shortcomings and/or limitations.

Given the bivariate model (1), the spectral Granger casual influence from X_2 to X_1 is defined by

$$I_{X_2\to X_1}(f)=-\log\left(1-\frac{\left(\sigma^2_{\eta_2}-\frac{\sigma^2_{\eta_1\eta_2}}{\sigma^2_{\eta_1}}\right)|H_{12}(f)|^2}{S_{X_1X_1}}\right) \quad (5)$$

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

$$H_{11}(f)=\frac{1}{\det(\mathbf{A})}\overline{a}_{22}(f),\quad H_{12}(f)=-\frac{1}{\det(\mathbf{A})}\overline{a}_{12}(f)$$

$$H_{21}(f)=-\frac{1}{\det(\mathbf{A})}\overline{a}_{21}(f),\quad H_{22}(f)=\frac{1}{\det(\mathbf{A})}\overline{a}_{11}(f)$$

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

$$k=1,2,\overline{a}_{hl}(f)=-\sum_{j=1}^{m}a_{hl,j}e^{-i2\pi fj} \quad (6)$$

$h,l=1,2,\ h\neq l$.

It is easy to see that when there is causality from X_2 to X_1, $I_{X_2\to X_1}(f)$ varies in $[0,+\infty)$. This definition has shortcomings and/or limitations as shown in the following remark.