Comparison Analysis: Granger Causality and New Causality, and their applications to Motor Imagery

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Abstract—In this paper we first point out a fatal drawback that the widely used Granger causality (GC) needs to estimate the autoregressive model, which is equivalent to take a series of backward recursive operations which are infeasible in many irreversible chemical reaction models. Thus, New causality (NC) proposed by Hu et. al (2011) is theoretically shown to be more sensitive to reveal true causality than GC. We then apply GC and NC to motor imagery (MI) which is an important mental process in cognitive neuroscience and psychology and has received growing attention for a long time. We study causality flow during MI using scalp EEGs from 9 subjects in BCI competition IV held in 2008. We are interested in three regions: Cz (central area of the cerebral cortex), C3 (left area of the cerebral cortex) and C4 (right area of the cerebral cortex) which are considered to be optimal locations for recognizing MI states in literature. Our results show that i) there is strong directional connectivity from Cz to C3/C4 during left and right hand MIs based on GC and NC. ii) During left hand MI, there is directional connectivity from C4 to C3 based on GC and NC. iii) During right hand MI, there is strong directional connectivity from C3 to C4 which is much clearly revealed by NC than by GC, i.e., NC largely improves the classification rate. iv) NC is demonstrated to be much more sensitive to reveal causal influence between different brain regions than GC.

Index Terms—Granger causality, New causality, EEG, Motor imagery.

I. INTRODUCTION

GC is one of the most popular methods to detect the directional influence of system components because of its simplicity and easy implementation, and plays a key role in understanding systems behavior in many different areas, such as economics [1] and [2], climate studies [3] and [4], genetics [5], and neuroscience [6] and [7]. In neuroscience, GC has been mainly applied to disclose effective connectivity of brain regions, which is defined as the influence of one neuronal system exerts over another [8]. The basic idea of GC was originally conceived by Wiener in 1956 [9], and later formalized by Granger in 1969 [10] in the form of a linear regression model. The idea can be briefly described as follows: If the historical information of time series Y significantly improves the prediction accuracy of the future of time series X in a multivariate autoregressive (MVAR) model, then GC from time series Y to X can be identified. In classic GC, time-invariant MVAR models are used to fit the experimental data of the observed time series [11]. Although GC has tremendous applications in many areas, this success has also been accompanied by criticism from different perspectives [12] and [13]. The criticism of GC has most been centered around the philosophical debate on the relationship between GC and true causality. As its name implies, GC is not necessarily true causality [14]. For example, if both X and Y are driven by a common third process with different lags, one might still fail to reject the alternative hypothesis of GC and may produce misleading results. In addition, the pairwise bivariate GC has the drawback that it fails to reveal synergetic effects among variables [15]. In 2011, Hu et. al [16] pointed out shortcomings and/or limitations of GC by using several illustrative examples and showed that GC is only a causality definition in the sense of Granger and why GC does not reflect real causality at all, and meanwhile proposed NC defined as a causality from any time series Y to any time series X in the linear regression model of multivariate time series, which describes the proportion that Y occupies among all contributions to X and is shown to be more reasonable and understandable than GC to reveal real causality by those examples. NC is a natural extension of GC and overcomes GC’s shortcomings and/or limitations.

Brain-computer interface (BCI) is an emerging technology dealing with computer-aided control using exclusive brain activity, and has found applications across bioengineering fields and in neuroprosthetics [17]-[21]. The most commonly used experimental paradigm in this context is motor imagery (MI) [22], that is, the imagination of a motor action without any actual movement of limbs, which has clear practical significance and provides a new communication channel between the human brain and the computer [23]. MI is the mental simulation of a motor act that includes preparation for movement, passive observations of action and mental operations of motor representations implicitly or explicitly. The neuronal representations of MI have been studied intensively for years using brain imaging techniques, such as functional magnetic resonance imaging (fMRI), electroencephalogram (EEG) and positron emission tomography (PET) [6], [24]-[26]. MI as preparation for immediate movement likely involves activation of the motor executive brain regions. Recent findings based on fMRI suggest the existence of the causal connectivity of motor related core regions in fronto-parietal circuit [6] during MI. In EEG-based BCI research, C3, C4 and Cz are demonstrated to be optimal for recognizing MI states [27]. In [28] we...
discussed causal flows among C3, C4 and Cz during MI based on scalp EEG by applying GC and NC, and made some rough conclusions. In this study we will elaborate our findings.

In this paper, firstly we will describe GC and NC in time and frequency domains in time-invariant bivariate autoregressive models, and show the differences between GC and NC in a mathematical way. Given a jointly regression model in GC method one has to estimate the autoregressive model. By an illustrative example on one hand we give the exact formula for GC, which is only related to some coefficients and has nothing to do with the other coefficients, and thus GC cannot reveal true causality at all since true causality underlying the joint regression model with different time-invariant coefficients surely be different. On the other hand, we theoretically show that the estimation of the autoregressive model is equivalent to taking a series of backward recursive operations, which are infeasible however for many irreversible chemical reaction models. Thus, GC method cannot be applied at all. In this case GC value by forcibly estimating the auto regressive model (i.e., taking a series of backward recursive operations) inevitably cannot reveal true causality.

Then we apply GC and NC to analyze the data sets 2b from BCI competition IV held in 2008 [29] and provided by Graz University of Technology. The data includes 3 bipolar EEG channels (C3, C4 and Cz) and is from 9 subjects. We apply GC and NC to EEGs and find that i) there is strong directional connectivity from Cz to C3/C4 in both time domain and frequency domain (mu rhythm (8~12Hz)) during left and right hand MIs based on GC and NC where mu rhythm is an important property of EEG signals during MI [27]. ii) During left hand MI, there is directional connectivity from C4 to C3 based on GC and NC in both time domain and frequency domain. iii) During right hand MI, there is strong directional connectivity from C3 to C4 in both time domain and frequency domain. iv) For all these subjects being right-handedness, during right hand MI the portion (78%) of GC values from Cz to C3 being larger than that from C3 to Cz is less than the portion (85%) of NC values from Cz to C3 being larger than that from C3 to Cz. The portion (78%) of GC values from Cz to C4 being larger than that from C4 to Cz is much less than the portion (96%) of NC values from Cz to C4 being larger than that from C4 to Cz. So, the NC method identifies the causal influence from Cz to C3/C4 more clearly than GC method during right hand MI. These higher identification rates demonstrate that the NC method is much more sensitive than GC method to reveal true causal flow among different brain regions. v) For causality from C4 to C3 during left hand MI and causality from C3 to C4 during right hand MI, the portion of GC values having peaks in mu rhythm is 32%, however the portion of NC values having peaks in mu rhythm is 55%. This once again strongly demonstrates that the NC method is more sensitive than GC to reveal true directional causality flow in real EEG data. Therefore, the NC method may open a new window to study causality relationships and may have wide applications in economics and neuroscience.

II. GC AND NC

Consider two stochastic time series which are assumed to be jointly stationary. Individually, under fairly general conditions, each time series admits an autoregressive representation

\[
\begin{align*}
X_{1,t} &= \sum_{j=1}^{m'} a_{1,j} X_{1,t-j} + \epsilon_{1,t} \\
X_{2,t} &= \sum_{j=1}^{m'} a_{2,j} X_{2,t-j} + \epsilon_{2,t}
\end{align*}
\]

and their joint representations are described as

\[
\begin{align*}
X_{1,t} &= \sum_{j=1}^{m} a_{11,j} X_{1,t-j} + \sum_{j=1}^{m} a_{12,j} X_{2,t-j} + \eta_{1,t} \\
X_{2,t} &= \sum_{j=1}^{m} a_{21,j} X_{1,t-j} + \sum_{j=1}^{m} a_{22,j} X_{2,t-j} + \eta_{2,t}
\end{align*}
\]

where \( t = 0, 1, \ldots, N \), the noise terms are uncorrelated over time (that is, let \( \theta_{k,t} = \eta_{1,t-k} \) and \( \xi_{k,t} = \eta_{2,t-k}, k = 1, 2, \ldots, m' \), then \( E[\theta_1 \theta_2] = E[\xi_1 \xi_2] = E[\xi_1 \eta_2] = 0 \) where \( E[\cdot] \) is the expectation value of a variable, \( i, j = 1, 2, \ldots, m', i \neq j \), \( \epsilon_i \) and \( \eta_i \) have zero means and variances of \( \sigma_{\epsilon_i}^2 \) and \( \sigma_{\eta_i}^2, i = 1, 2 \). The covariance between \( \eta_1 \) and \( \eta_2 \) is defined by \( \sigma_{\eta_1 \eta_2} = cov(\eta_1, \eta_2) \) [16]. For a practical system, a general approach for determining the order of the MVAR model is the AIC–Akaikes Information Criterion [30], [31], [32].

A. GC in Time Domain

Now consider the first equalities in (1) and (2), if \( \sigma_{\eta_1}^2 \) is less than \( \sigma_{\epsilon_1}^2 \) in some suitable sense \( X_2 \) is said to have a causal influence on \( X_1 \). In this case, the first equality in (2) is more accurate than in (1) to estimate \( X_1 \). Otherwise, if \( \sigma_{\eta_1}^2 = \sigma_{\epsilon_1}^2 \), \( X_2 \) is said to have no causal influence on \( X_1 \). In this case, two equalities are the same. Such kind of causal influence, called GC [33], [34], is defined by

\[
F_{X_2 \rightarrow X_1} = \ln \frac{\sigma_{\epsilon_1}^2}{\sigma_{\eta_1}^2} \tag{3}
\]

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

\[
F_{X_1 \rightarrow X_2} = \ln \frac{\sigma_{\epsilon_2}^2}{\sigma_{\eta_2}^2} \tag{4}
\]

B. NC in Time Domain

Based on the first equality in (2), we can see contributions to \( X_1,t \), which include \( \sum_{j=1}^{m} a_{11,j} X_{1,t-j} \) and \( \sum_{j=1}^{m} a_{12,j} X_{2,t-j} \) and the noise term \( \eta_{1,t} \) where the influence from \( \sum_{j=1}^{m} a_{11,j} X_{1,t-j} \) is causality from \( X_1 \)'s own past values. Each contribution plays an important role in determining \( X_1,t \). If \( \sum_{j=1}^{m} a_{12,j} X_{2,t-j} \) occupies a larger portion among all those contributions, then \( X_2 \) has stronger causality on \( X_1 \), or vice versa. Thus, a good definition for causality from \( X_2 \) to \( X_1 \) in the time domain should be able to describe what proportion
Given a joint regression model (2) of two jointly stationary variables $X_1$ and $X_2$, where $X_2$ occupies among all these contributions. So based on this general guideline NC from $X_2$ to $X_1$ is defined as [16]

$$n_{X_2 \rightarrow X_1} = \frac{\sum_{i=m}^{N} \left( \sum_{j=1}^{m} a_{12,j}X_{2,t-j} \right)^2}{\sum_{i=m}^{N} \left( \sum_{j=1}^{m} a_{1h,j}X_{h,t-j} \right)^2 + \sum_{i=m}^{N} \eta_i^2}$$
(5)

or

$$n_{X_2 \rightarrow X_1} = \frac{\sum_{i=m}^{N} \left( \sum_{j=1}^{m} a_{12,j}X_{2,t-j} \right)^2}{\sum_{i=m}^{N} \left( \sum_{j=1}^{m} a_{1h,j}X_{h,t-j} \right)^2 + \sum_{i=m}^{N} \eta_i^2}$$
(6)

Similarly, NC in time domain from $X_1$ to $X_2$ is defined by

$$n_{X_1 \rightarrow X_2} = \frac{\sum_{i=m}^{N} \left( \sum_{j=1}^{m} a_{21,j}X_{1,t-j} \right)^2}{\sum_{i=m}^{N} \left( \sum_{j=1}^{m} a_{2h,j}X_{h,t-j} \right)^2 + \sum_{i=m}^{N} \eta_i^2}$$
(7)

or

$$n_{X_1 \rightarrow X_2} = \frac{\sum_{i=m}^{N} \left( \sum_{j=1}^{m} a_{21,j}X_{1,t-j} \right)^2}{\sum_{i=m}^{N} \left( \sum_{j=1}^{m} a_{2h,j}X_{h,t-j} \right)^2 + \sum_{i=m}^{N} \eta_i^2}$$
(8)

**Remark 1:** Given a joint regression model (2) of two jointly stationary variables $X_1$ and $X_2$. To derive GC value from $X_2$ to $X_1$ one has to estimate the noise variable $e_1$ in the autoregressive mode (1); that is, what is $e_1$? Is there any relationship between $e_1$ and $\eta(\eta_1$ and $\eta_2$)? Next we will show how $e_1$ is related to $\eta_1$ and $\eta_2$. We then rewrite the following simple joint regression model (9) of only one time delay:

$$\begin{align*}
X_{1,t} &= a_{11,1}X_{1,t-1} + a_{12,1}X_{2,t-1} + \eta_1,t \\
X_{2,t} &= a_{21,1}X_{1,t-1} + a_{22,1}X_{2,t-1} + \eta_2,t
\end{align*}$$
(9)

where we further assume $|a_{22,1}| < 1$. From (9) and by $n$ steps we can recursively obtain

$$\begin{align*}
X_{1,t} &= a_{11,1}X_{1,t-1} + a_{12,1}X_{2,t-1} + \eta_1,t \\
&= a_{11,1}X_{1,t-1} + a_{12,1}a_{21,1}X_{1,t-2} + a_{22,1}X_{2,t-2} + \eta_1,t + a_{22,1}a_{22,1}X_{2,t-3} + \eta_1,t \\
&= a_{11,1}X_{1,t-1} + a_{12,1}a_{21,1}X_{1,t-2} + a_{22,1}a_{22,1}X_{2,t-2} + a_{22,1}a_{22,1}a_{22,1}X_{2,t-3} + \eta_1,t \\
&\vdots
\end{align*}$$

where we can easily observe the fact that the more large $m^*$ is chosen, the more close to the real $X_{1,t}$, the right side of (13) is. Comparing (13) with $X_{1,t}$ in the autoregressive model.
clearly see that GC in (1) also involves the coefficients \(a_{11,1}\) and \(a_{12,1}\) and variance \(\sigma_{\eta_1}^2\) of the noise, of the linear regression model (9).

Therefore, we have theoretically shown that both GC and NC involve coefficients of the linear regression model. The authors in [35] said that NC is causal mechanism because it includes the coefficients of the linear regression model. This statement is not correct because GC in a mathematical way also involves the coefficients of the linear regression model. Thus, NC like GC and all GC-alike methods reveals causal effect (causal influence, causal flow, to name a few). In fact, causal mechanism represents a process and is a different concept from causal effect (see, e.g., the diagram of a simple causal mechanism in Figure 1 of [36]).

### C. GC in Frequency Domain

Granger causal influence from \(X_2\) to \(X_1\) in frequency domain is defined by \(I_{X_2}\rightarrow X_1(f)\)

\[-\ln (1 - \frac{(\sigma_{\eta_2}^2 - \frac{\sigma_{\eta_{12}}^2}{\sigma_{\eta_1}^2})}{|H_{12}(f)|^2}) \in [0, \infty)\]  

(19)

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

\[H_{12}(f) = \frac{1}{\det(A)}\pi_{12}(f),\]

\[H_{22}(f) = \frac{1}{\det(A)}\pi_{21}(f),\]

\[A = [\pi_{ij}]_{2 \times 2},\]

\[\pi_{kk}(f) = 1 - \sum_{j=1}^{m} \alpha_{kk,j} e^{-i2\pi f k}, k = 1, 2,\]

\[\pi_{hl}(f) = - \sum_{j=1}^{m} \alpha_{hl,j} e^{-i2\pi f k}, h, l = 1, 2, h \neq l.\]

Similarly, we define Granger causal influence from \(X_1\) to \(X_2\) by

\[I_{X_1}\rightarrow X_2(f) = -\ln (1 - \frac{(\sigma_{\eta_1}^2 - \frac{\sigma_{\eta_{12}}^2}{\sigma_{\eta_2}^2})}{|H_{21}(f)|^2}) \in [0, \infty)\]  

(20)

where \(S_{X_i, X_j}(f)\) is the spectrum of \(X_i, i = 1, 2.\)

### D. NC in Frequency Domain

Taking Fourier transformation on both sides of (2) leads to

\[
\begin{cases}
X_1(f) = a_{11}(f)X_1(f) + a_{12}(f)X_2(f) + \eta_1(f) \\
X_2(f) = a_{21}(f)X_1(f) + a_{22}(f)X_2(f) + \eta_2(f)
\end{cases}
\]

(21)

where \(a_{ij}(f) = \sum_{k=1}^{m} \alpha_{k,k} e^{-i2\pi f k}, i = \sqrt{-1}, l, j = 1, 2.\)

From (7), one can see that contributions to \(X_1(f)\) include \(a_{11}(f)X_1(f), a_{12}(f)X_2(f)\) and noise term \(\eta_1(f).\) So NC from \(X_2\) to \(X_1\) in frequency domain is defined as \(N_{X_2}\rightarrow X_1(f)\)

\[
|a_{12}(f)|^2 S_{X_2, X_2}(f) / |a_{11}(f)|^2 S_{X_1, X_1}(f) + |a_{12}(f)|^2 S_{X_2, X_2}(f) + \sigma_{\eta_2}^2
\]

(22)
Similarly, NC from $X_1$ to $X_2$ in frequency domain is defined as $N_{X_1 \to X_2}(f) = \frac{|a_{21}(f)|^2 S_{X_1X_2}(f)}{|a_{21}(f)|^2 S_{X_1X_1}(f) + |a_{22}(f)|^2 S_{X_2X_2}(f) + \sigma_n^2}$ (23)

Remark 2: To understand the core difference between GC and NC and also to show which one (GC and NC) is better to reveal true causality (or trend of true causality because nobody knows what exactly is true causality for a given MVAR model), let’s reconsider the simple illustrative model (10) of [16], i.e.,

$$
\begin{align*}
X_{1,t} &= a_{12,1} X_{2,t-1} + \eta_{1,t} \\
X_{2,t} &= a_{21,1} X_{1,t-1} + \eta_{2,t}
\end{align*}
$$

from which we obtain

$$
X_{2,t-1} = a_{12,1} (a_{21,1} X_{1,t-2} + \eta_{2,t-1}) + \eta_{1,t} = a_{12,1} a_{21,1} X_{1,t-2} + a_{12,1} \eta_{2,t-1} + \eta_{1,t}
$$

where

$$
\epsilon_{1,t} = a_{12,1} \eta_{2,t-1} + \eta_{1,t}.
$$

GC value $F_{X_2 \to X_1} = \ln \frac{\sigma_n^2}{\sigma_{\eta_1}^2} = \ln \left( 1 - \frac{a_{12,1}^2 \sigma_{\eta_2}^2}{a_{12,1}^2 \sigma_{\eta_1}^2 + \sigma_{\eta_1}^2} \right) \in [0, \infty)$

which is equivalent to

$$
F_{X_2 \to X_1} = \frac{a_{12,1}^2 \sigma_{\eta_2}^2}{a_{12,1}^2 \sigma_{\eta_1}^2 + \sigma_{\eta_1}^2} = \frac{\sum_{t=1}^{N} (a_{12,1} \eta_{2,t-1})^2}{\sum_{t=1}^{N} (a_{12,1} \eta_{2,t-1})^2 + \sum_{t=1}^{N} \eta_{1,t}^2}
$$

$\in [0, 1]$

NC value

$$
\eta_{X_2 \to X_1} = \frac{\sum_{t=1}^{N} (a_{12,1} X_{2,t-1})^2}{\sum_{t=1}^{N} (a_{12,1} X_{2,t-1})^2 + \sum_{t=1}^{N} \eta_{2,t}^2} = \frac{\sum_{t=1}^{N} (a_{12,1} a_{21,1} X_{1,t-2} + a_{12,1} \eta_{2,t-1})^2}{\sum_{t=1}^{N} (a_{12,1} a_{21,1} X_{1,t-2} + a_{12,1} \eta_{2,t-1})^2 + \sum_{t=1}^{N} \eta_{2,t}^2}
$$

Comparing (28) with (29), we can make the following remarks:

i) both GC and NC are defined essentially based on the concept of proportion.

ii) GC describes the causal effect due to the reduction of the prediction error (the change from $\epsilon_{1,t} = a_{12,1} \eta_{2,t-1} + \eta_{1,t}$ to $\eta_{1,t}$). Or, equivalently, it describes what is the portion of $\eta_2$ in the two parts: $a_{12,1} \eta_{2,t-1}$ and $\eta_{1,t}$. In other words, it measures the extent to which the past of $X_2$ increases predictive power of the future of $X_1$ over and above the extent to which the past of $X_1$ already predicts its own future. Term $a_{12,1} a_{21,1} X_{1,t-2}$ is considered to be the predictive effect of past $X_1$ on its own future, does not make a direct contribution to GC.

iii) NC describes what is the portion of $X_2$ in the two parts: $a_{12,1} X_{2,t-1}$ and $\eta_{1,t}$.

iv) The core difference in GC and NC is that the term $a_{12,1} a_{21,1} X_{1,t-2}$ is ignored in GC, while it is included in NC. NC is only when $a_{12,1} a_{21,1} = 0$. When one tries to define a causality approach to reveal true causality from $X_2$ to $X_1$, the most basic question is: what is $X_2$? Note that both $a_{12,1} X_{2,t-1}$ and $\eta_{2,t-1}$ together produce $X_{2,t-1}$, that is, $X_2$ includes two pieces of information: $a_{21,1} X_1$ and $\eta_2$. Any one of them is insufficient to represent $X_2$. The complete information represented in $X_2$ must be considered to reveal true causality from $X_2$ to $X_1$. From this point of view, GC in (28) ignores a significant part of the information of $X_2$: $a_{21,1} X_{1,t-2}$ and thus may not be able to reveal true causality or trend of true causality from $X_2$ to $X_1$.

v) NC is much more suitable than GC to reveal true causality (or trend of true causality), as demonstrated by a number of illustrative examples in [16] which include experimental EEG data. Although we do not know exactly true causality from $X_2$ to $X_1$ in the first equality in (24), we definitely know the fact that $X_2$ has larger true causality on $X_1$, if $X_2$ occupies larger portion in $a_{12,1} X_{2,t-1}$ and $\eta_{1,t}$. NC in the first equality of (29) provides an effective way to reveal causality from $X_2$ to $X_1$ (for example, we can also define another causality approach replacing “square” by “absolute value” in NC formula), which is consistent with the above fact. However, GC in (28) does not reflect the above fact.

vi) To demonstrate that the term $a_{12,1} a_{21,1} X_{1,t-2}$ in above (25) may have an important influence on $X_{1,t}$ and thus cannot be ignored, let’s assume $a_{12,1} = 1, a_{21,1} = -0.9$, then model (24) becomes

$$
\begin{align*}
X_{1,t} &= X_{2,t-1} + \eta_{1,t} \\
X_{2,t} &= -0.9 X_{1,t-1} + \eta_{2,t}
\end{align*}
$$

We further assume $\eta_1$ and $\eta_2$ are two noises with normal distribution with zero mean and variance of 1. From (30) we can get

$$
X_{1,t+1} = -0.9 X_{1,t-2} + \eta_{1,t} + \eta_{2,t-1}.
$$

From model (30) we generate one realization of 2000 sample points and plot five variables in Figure 1, from which one can see that generally speaking, the amplitudes of $X_{2,t-1}$ are larger than that of $\eta_{1,t}$ and the amplitudes of $-0.9 X_{1,t-2}$ are larger than that of $\eta_{1,t} + \eta_{2,t-1}$. In this case from (31) we calculate

$$
\frac{\sum_{t=2}^{N} (-0.9 X_{1,t-2})^2}{\sum_{t=2}^{N} (-0.9 X_{1,t-2})^2 + \sum_{t=1}^{N} (\eta_{1,t} + \eta_{2,t-1})^2} = 0.81
$$

which implies $X_{1,t}$ is mainly determined by $-0.9 X_{1,t-2}$, that is, $-0.9 X_{1,t-2}$ has a major influence on $X_{1,t}$ and thus the influence from $\eta_{1,t} + \eta_{2,t-1}$ is rather small. Since $-X_{1,t-2}$ is from $X_{2,t-1}$, this implies $X_{2,t-1}$ has a major influence (i.e., causal influence) on $X_{1,t}$. In fact, we can also calculate NC value $\eta_{X_2 \to X_1} = 0.91$ which demonstrates that $X_2$ has a major causal influence on $X_1$ and the influence of $\eta_1$ on $X_1$ is rather small. However, GC is based on the rather small term
\( \epsilon_{1,t} = \eta_{1,t} + \eta_{2,t-1} \) to calculate the proportion in (28) and get the equivalent GC value \( F_{X_2 \rightarrow X_1} = 1/(1+1) = 1/2 \) which is far away from 1. Obviously, this result is not reasonable.

\[ \begin{align*}
-0.9X_{1,t-2}, X_{2,t}, \eta_{1,t}, \eta_{2,t-1}, \eta_{1,t} + \eta_{2,t-1}.
\end{align*} \]

Fig. 1. Trajectories for \(-0.9X_{1,t-2}, X_{2,t}, \eta_{1,t}, \eta_{2,t-1}, \eta_{1,t} + \eta_{2,t-1}\).

vii) We can view model (24) as two neurons with connection weights of \( a_{12,1} \) and \( a_{21,1} \) having the structure shown in Fig. 2 where neuron 1 is consist of substance \( X_1 \) and neuron 2 is consist of substance \( X_2 \). \( X_1 \) and \( X_2 \) are two totally different substances. In Fig. 2, the left and right arrows represent information flows from substances \( X_2 \) and \( X_1 \) respectively, the thickness of the arrow represents the strength of the flow which is controlled by \( a_{12,1} \) or \( a_{21,1} \). For example, if \( a_{12,1} = 1 \), substance \( X_2 \) in neuron 2 is fully transferred to neuron 1. It is well known that, in the field of chemical industry, chemical reaction is represented by chemical equation which typically has a unidirectional arrow to represent irreversible reaction [37]. If we view model (24) as two chemical reaction equations with regression time-dependance and further assume the chemical reactions are irreversible, then we can only have unidirectional arrow reaction equations \( a_{12,1}X_{2,t-1} + \eta_{1,t} \rightarrow X_{1,t} \) and \( a_{21,1}X_{1,t-1} + \eta_{2,t} \rightarrow X_{2,t} \), and we cannot have unidirectional arrow reaction equations \( X_{1,t} \rightarrow a_{12,1}X_{2,t-1} + \eta_{1,t} \) and \( X_{2,t} \rightarrow a_{21,1}X_{1,t-1} + \eta_{2,t} \). Thus, the backward recursive operation in (25) cannot be applied, that is, substance \( X_2 \) at time \( t - 1 \) cannot be backward decomposed into substance \( X_1 \) at time \( t - 2 \) and \( \eta_2 \) at time \( t - 1 \). However, given a jointly regression model (9) which describes two irreversible chemical reactions, as shown in Remark 1, the estimation of the auto regressive model for \( X_{1,t} \) is actually a process by taking a series of backward recursive operations. Therefore, the estimation of the auto regressive model for \( X_{1,t} \) is not feasible in practice, and as a result GC method cannot be applied at all. In this case GC value \( F_{X_2 \rightarrow X_1} \) by forcibly estimating the auto regressive model for \( X_{1,t} \) (i.e., taking a series of backward recursive operations) inevitably cannot reveal true causality from \( X_2 \) to \( X_1 \). In fact, to reveal true causality from \( X_2 \) to \( X_1 \) in model (24) which describes two irreversible chemical reactions, we should consider two contributions: \( X_{2,t-1} \) and \( \eta_{1,t} \) which consist of the whole information of \( X_{1,t} \) as shown in NC method in (29). We should not decompose \( X_{2,t-1} \) into \( a_{21,1}X_{1,t-2} \) and \( \eta_{2,t-1} \) since this process is irreversible, and then consider two contributions: \( \eta_{1,t} \) and \( a_{12,1}\eta_{2,t-1} \) which consist of the whole information of \( \epsilon_{1,t} \) in (26) as shown in GC method in (28). This fact once again demonstrates that NC method better reveals true causality than GC method.

**Remark 3:** i) For model (9) we have GC value from \( X_2 \) to \( X_1 \) as in (16) or (17) from which one can see that \( F_{X_2 \rightarrow X_1} \) has nothing to do with \( a_{11,1} \) and \( a_{12,1} \) and only involves partial information \( (a_{12,1}, a_{22,1}, \sigma_{\eta_1}^2 \text{ and } \sigma_{\eta_2}^2) \) of model (9). In fact, true causality from \( X_2 \) to \( X_1 \) should be surely different for different time invariant parameters \( a_{11,1} \) and \( a_{12,1} \) because the linear regression model is actually changed in this case. In this way, we clearly demonstrate that GC may not be necessarily reflect true causality of model (9) at all.

ii) In this study, we will further demonstrate that NC is more sensitive than GC to reveal true causality influence based on EEG during MI states.

**III. EXPERIMENTAL METHOD**

In this section, we first describe our EEG data for analysis. The data sets 2b from BCI competition IV held in 2008 were used and provided by Graz University of Technology [29]. These data sets consist of EEG data from 9 subjects. The subjects were right-handed, had normal or corrected-to-normal vision and were paid for participating in the experiments [29]. All volunteers were sitting in an armchair, watching a flat screen monitor placed approximately 1 m away at eye level. For each subject 5 sessions were provided, whereby the first three sessions were training data, and the last two sessions were evaluation data. We only analyzed the training data because the classification result of the evaluation data was unknown. Thus, there are a total of \( 9 \times 3 = 27 \) sessions for our analysis. The cue-based screening paradigm consists of two classes, namely left hand MI (Class 1) and right hand MI (Class 2). Each session contains some trails of Class 1 and some trails of Class 2. For each session we extract all the trails data of MI. Then put the left hand MI trail data together and average the trials data as one time series data of Class 1. For the right hand MI trial data, we do the same preprocessing to get one time series data of Class 2. Each class data consists of 3 channels (C3, Cz and C4) data. In this study, we analyze the data as follows: firstly, we detect the effective connectivity in the time domain by calculating GC and NC between Cz and C3/C4 and between C3 and C4. Secondly, we calculate GC and NC in the frequency domain between Cz and C3/C4 and between C3 and C4, and choose three subjects as representatives to show our results. For GC and NC results in time and frequency domains we make comparison to conclude that NC is much more sensitive than GC to reveal real causality based on EEG data during MI states. For all analyzed EEGs, after applying AIC to determine the choices of \( m \), we then average these values and get \( m = 12 \) which is chosen as the order of the estimated MVAR models for all analyzed EEGs.
IV. SIMULATION RESULTS

A. Effective Connectivity Analysis in Time Domain

For the MI data including Class 1 and Class 2, we extract channels (C3, C4 and Cz) data as three time series. For each class, we first apply GC to calculate $F_{C3 \rightarrow Cz}$, $F_{Cz \rightarrow C3}$, $F_{C4 \rightarrow Cz}$, $F_{Cz \rightarrow C4}$, and meanwhile we apply NC to calculate $n_{C3 \rightarrow Cz}$, $n_{Cz \rightarrow C3}$, $n_{C4 \rightarrow Cz}$, $n_{Cz \rightarrow C4}$. The results are shown in Fig.3 (Class 1) and Fig.4 (Class 2) where each session has one causality value for each method and as a result there are totally 27 points for each curve because of 27 sessions being involved, and summarized in Table I and Table II. From Table I and Table II one can see that there is strong directional connectivity from Cz to C3/C4 in time domain during left and right hand MIs based on GC and NC where strong directional causal flow is statistically significant discussed later.

We note that i) from Table I causality influence phenomenon (93%) revealed by NC ($n_{Cz \rightarrow C3} > n_{C3 \rightarrow Cz}$) is more clear than that (89%) revealed by NC ($n_{Cz \rightarrow C4} > n_{C4 \rightarrow Cz}$) during left hand MI. This phenomenon demonstrates more circuits of directional causal connectivity from Cz to C3 than that from Cz to C4 during left hand MI; ii) from Table II causality influence phenomenon (96%) revealed by NC ($n_{Cz \rightarrow C4} > n_{C4 \rightarrow Cz}$) is more clear than that (85%) revealed by NC ($n_{Cz \rightarrow C3} > n_{C3 \rightarrow Cz}$) during right hand MI. This phenomenon demonstrates more circuits of directional causal connectivity from Cz to C4 during right hand than that from C3 to C4 during right hand MI; iii) the portion (93% from Table I) revealed by NC ($n_{Cz \rightarrow C3} > n_{C3 \rightarrow Cz}$) during left hand MI is less than that (96% from Table II) revealed by NC ($n_{Cz \rightarrow C4} > n_{C4 \rightarrow Cz}$) during right hand MI. This phenomenon demonstrates more circuits of directional causal connectivity among Cz, C3 and C4 regions during right hand MI than left hand MI, and implies the influence of brain asymmetry of right-handedness on effective connectivity networks by noting that all subjects are right-handed. This result is consistent with the finding revealed by fMRI [6].

It is interesting to observe that NC values in red lines in Fig.3 and Fig.4 are rather small for most of 27 sessions during left hand and right hand MI compared to NC values in blue lines, that means causal flows from C3/C4 to Cz are always rather small for most of 27 sessions no matter left hand MI or right hand MI compared to causal flows from Cz to C3/C4. In other word, for most of 27 sessions no matter left hand MI or right hand MI, there is always unilateral causal flow from Cz to C3/C4. However, GC values in red lines do not have such a property; that is, for most of 27 sessions no matter left hand MI or right hand MI, there are always lateral causal flows between Cz and C3/C4. For all these subjects being right-handedness, from Table II during right hand MI we find that i) the portion (78%) of GC values from Cz to C3 being larger than that from C3 to Cz is less than the portion (85%) of NC values from Cz to C3 being larger than that from C3 to Cz; ii) the portion (78%) of GC values from Cz to C4 being larger than that from C4 to Cz is much less than the portion (96%) of NC values from Cz to C4 being larger than that from C4 to Cz. So, NC method identifies the causal influence from Cz to C3/C4 more clearly than GC method during right hand MI. These higher identification rates demonstrate that the NC method is much more sensitive than GC method to reveal true causal influence among different brain regions.

![Fig. 3. (a), (b) GC between Cz and C3/C4 in time domain during left hand MI. (c), (d) NC between Cz and C3/C4 in time domain during left hand MI.]

**Table I**

<table>
<thead>
<tr>
<th>Causality Methods</th>
<th>Cz → C3 &gt; C3 → Cz</th>
<th>Cz → C4 &gt; C4 → Cz</th>
</tr>
</thead>
<tbody>
<tr>
<td>GC</td>
<td>0.89</td>
<td>0.93</td>
</tr>
<tr>
<td>NC</td>
<td>0.93</td>
<td>0.89</td>
</tr>
</tbody>
</table>

**Table II**

<table>
<thead>
<tr>
<th>Causality Methods</th>
<th>Cz → C3 &gt; C3 → Cz</th>
<th>Cz → C4 &gt; C4 → Cz</th>
</tr>
</thead>
<tbody>
<tr>
<td>GC</td>
<td>0.78</td>
<td>0.78</td>
</tr>
<tr>
<td>NC</td>
<td>0.85</td>
<td>0.96</td>
</tr>
</tbody>
</table>

Next, we apply GC to calculate $F_{C3 \rightarrow C4}$, $F_{C4 \rightarrow C3}$, and apply NC to calculate $n_{C3 \rightarrow C4}$, $n_{C4 \rightarrow C3}$. The results are shown in Fig.5 and summarized in Table III.

**Table III**

<table>
<thead>
<tr>
<th>Causality</th>
<th>class 1</th>
<th>class 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>$F_{C3 \rightarrow C4}$ &gt; $F_{C4 \rightarrow C3}$</td>
<td>0.37</td>
<td>0.60</td>
</tr>
<tr>
<td>$F_{C4 \rightarrow C3}$ &gt; $F_{C3 \rightarrow C4}$</td>
<td>0.64</td>
<td>0.41</td>
</tr>
<tr>
<td>$n_{C3 \rightarrow C4}$ &gt; $n_{C4 \rightarrow C3}$</td>
<td>0.37</td>
<td>0.81</td>
</tr>
<tr>
<td>$n_{C4 \rightarrow C3}$ &gt; $n_{C3 \rightarrow C4}$</td>
<td>0.64</td>
<td>0.19</td>
</tr>
</tbody>
</table>

From Fig.5 and Table III, one can see that i) during left hand MI, there is strong directional connectivity from C4 to C3 based on GC and NC, more exactly, the portion of causality values from C4 to C3 being larger than that from C3 to C4 is 64%. ii) During right hand MI, there is strong directional connectivity from C3 to C4, more exactly, the portion of
B. Effective Connectivity Analysis in Frequency Domain

In frequency domain, we will apply GC (19) and NC (22) to calculate spectral causality. We calculate the causality between Cz and C3/C4, and between C3 and C4. We choose three subjects as representatives to show how to analyze the MI data. For each subject, we also show the power spectra of Cz, C3, C4 of Class 1 and Class 2. We use Matlab function pwelch to calculate the power spectrum with window size 128, frequency resolution 0.05 Hz, and maximum frequency 20 Hz.

Subject 1 (i.e., Subject101 in Table IV): The power spectra of Cz, C3 and C4 are shown in Fig.6 where there are clearly peaks in mu rhythm (8 ~ 12 Hz) of the power spectra during left hand and right hand MI. The feature is a basic characteristic of EEG signals during MI [27].

GC and NC between Cz and C3/C4 in the frequency domain during left hand MI and during right hand MI are shown in Fig.7 and Fig.8, respectively. From Fig.7 and Fig.8 one can see that i) in mu rhythm there always exist \( I_{Cz\rightarrow C3} > I_{C3\rightarrow Cz}, \) \( I_{Cz\rightarrow C4} > I_{C4\rightarrow Cz} \),

causality values from C3 to C4 being larger than that from C4 to C3 is 60% by GC method, and 81% by NC method. We also note that during right hand MI, the portion (60%) of GC values from C3 to C4 being larger than that from C4 to C3 is much less than the portion (81%) of NC values from C3 to C4 being larger than that from C4 to C3. So, NC method identifies the causal influence from C3 to C4 during right hand MI more clearly than GC method (that is, NC method largely improves the classification rate). Therefore, in time domain, we once again demonstrate that NC method is much more sensitive than GC method to reveal causal influence among different brain regions during MI states.
$N_{\text{Cz} \rightarrow \text{C3}} > N_{\text{C3} \rightarrow \text{Cz}}, N_{\text{Cz} \rightarrow \text{C4}} > N_{\text{C4} \rightarrow \text{Cz}}.$

These results are consistent with the findings obtained in time domain, that is, there is strong causal influence from Cz to C3/C4 during left hand and right hand MI. i) The peaks always appear in mu rhythm (8 \sim 12 Hz) for all NC results (blue curves) which are consistent with the peaks of the power spectra in Fig. 6, but they do not appear in GC results except for (a) of Fig. 7. This means NC method can better reveal real causal influence from Cz to C3/C4 than GC method in the frequency domain.

GC and NC in the frequency domain between C3 and C4 are shown in Fig. 9 from which one can see that i) the causality of C4 on C3 in mu rhythm is larger than that of C3 on C4 during left hand MI, but the results are reversed during right hand MI. This consequence is consistent with the conclusion got in time domain. ii) During right hand MI, the peaks in mu rhythm (8 \sim 12 Hz) appear for NC results in (d), which are consistent with peaks of the power spectra in Fig. 6, but they do not appear for GC results in (b). This also demonstrates that NC method is better than GC method to reveal real causal influence between C3 and C4 during this right hand MI in the frequency domain.

**Subject 2 (i.e., Subject303 in Table IV):** The power spectra of Cz, C3 and C4 are shown in Fig. 10 where there are clearly peaks in about 10 Hz of during left hand and right hand MI for all three channels. It indicates that the subject performed well during MI, that is, the basic feature in mu rhythm (8 \sim 12 Hz) is very obvious for all channels.

GC and NC between Cz and C3/C4 in the frequency domain during left hand MI and during right hand MI are shown in Fig. 11 and Fig. 12, respectively. GC and NC in the frequency domain between C3 and C4 are shown in Fig. 13. Based on Figs. 11-13 we can have similar discussions as in Subject 1.
omitted here for brevity.

Subject 3 (i.e., Subject501 in Table IV): The power spectra of Cz, C3 and C4 are shown in Fig.14 where there are clearly peaks in about 11 Hz of during left hand and right hand MI for all three channels. It implies that the subject performed well during MI.

GC and NC between Cz and C3/C4 in the frequency domain during left hand MI and during right hand MI are shown in Fig.15 and Fig.16, respectively. GC and NC in the frequency domain between C3 and C4 are shown in Fig.17. Based on Figs.15~17 we can have similar discussions as in Subject 1 omitted here for brevity.

By the above analysis from three representative subjects, we can make the following conclusions: i) there exists obvious causal influence from Cz to C3/C4 in mu rhythm which is stronger than that from C3/C4 to Cz during left hand and right hand MI, that is, Cz region has causal influences on C3 and C4 regions during MI. ii) There exists clear causal influence from C4 to C3 in mu rhythm which is larger than that from C3 to C4 during left hand MI, but the result is reversed during right hand MI. Thus, different MI can lead to different directional causal influence. iii) NC method can better reveal real causal influence among Cz, C3 and C4 three regions in mu rhythm than GC method during MI.

In our experiment analysis, there are 9 subjects (27 training sessions data sets). Similar to the process as in above three subjects, we dealt with all the data sets and summarize the results in Table IV. It is noted that mu rhythm (8~12Hz) is a well-known neurophysiological phenomenon in EEG during MI [27], so, we marked the data as “good” if the power spectra of three channels have peak(s) of obvious mu rhythm. Otherwise, we marked the data as “bad”. In this way, we have totally 5 bad data sets and 22 good data sets (see the column of

Fig. 12. Subject 2: (a), (b) GC between Cz and C3/C4 in the frequency domain during right hand MI. (c), (d) NC between Cz and C3/C4 in the frequency domain during right hand MI. One can clearly see that the peaks in about 10Hz appear for NC results, but do not appear for GC results.

Fig. 13. Subject 2: (a), (b) GC between C3 and C4 in the frequency domain. (c), (d) NC between C3 and C4 in the frequency domain.

Fig. 14. Subject 3: (a) The power spectra of Cz, C3, and C4 during left hand MI. (b) The power spectra of Cz, C3, and C4 during right hand MI. From (a) and (b), one can clearly see the peaks in the mu (8~12Hz) band for all three channels.
power spectrum in Table IV). In our analysis in the frequency domain, we only handled these 22 good data sets for which we have three kinds of results in \( \mu \) rhythm as follows:

Case 1) calculate GC and NC between Cz and C3/C4 during left hand and right hand MI. If the causality value from Cz to C3/C4 is larger than that from C3/C4 to Cz, then the result of the data is viewed as "good", otherwise, it is viewed as "bad".

Case 2) Calculate GC and NC between C3 and C4 during left hand MI. If the causality value from C4 to C3 is larger than that from C3 to C4, then the result of the data is viewed as "good", otherwise, it is viewed as "bad".

Case 3) Calculate GC and NC between C3 and C4 during right hand MI. If the causality value from C3 to C4 is larger than that from C4 to C3, then the result of the data is viewed as "good", otherwise, it is viewed as "bad".

Results in Case 1, Case 2 and Case 3 are reported in Table IV. According to these results, we have 17 data sets of "good" results in Case 1, 15 data sets of "good" results in Case 2 and 13 data sets of "good" results in Case 3. So, the portion of spectral causality values from Cz to C3/C4 being larger than that from C3/C4 to Cz based on GC and NC during left hand and right hand MI is \( 17/22 = 77\% \) where 22 is the number of "good" data set summarized in Table IV (we do not remind this again in the remaining part of the paper), the portion of spectral causality values from C4 to C3 being larger than that from C3 to C4 based on GC and NC during left hand MI is \( 15/22 = 68\% \), and the portion of spectral causality values from C3 to C4 being larger than from C4 to C3 based on GC and NC during right hand MI is \( 13/22 = 59\% \).

To demonstrate the advantage of NC over GC in the frequency domain, we further mark whether there are peaks in \( \mu \) rhythm for the results in Case 1, Case 2 and Case 3 of Table IV because the peak in \( \mu \) rhythm is the most basic property of EEG signal during MI states [27]. The corresponding results are summarized in Table V (where \( \leftrightarrow \) means bidirectional causal flows) and Table VI. From Table V one can see that there always exist peaks in \( \mu \) rhythm for all 17 good data set in Case 1 of Table IV by NC method. So, for the results in Case 1 of Table V, during left hand and right hand MI the portion of spectral NC value from Cz to C3/C4 being larger than that from C3/C4 to Cz and meanwhile having peaks in \( \mu \) rhythm is \( 17 \times 4/(22 \times 4) = 77\% \) where 4 represents the four cases in Table V. However, the corresponding portion based on GC is only \( (10 + 11 + 9 + 11)/(22 \times 4) = 53\% \) from Table V. From Table VI one can see that the portion of spectral causality values between C3 and C4 have peaks in \( \mu \) rhythm by NC method is \( (12 + 12)/(22 \times 2) = 54\% \) where 2 represents the two cases in Table VI. However the corresponding portion by GC method is \( (6 + 8)/(22 \times 2) = 32\% \). Thus, NC method is much clearer to reveal the causal influence among Cz, C3 and C4 than GC method in the frequency domain.

C. Significance test for NC results

In this subsection, we will take analysis of significance test only for NC results in time domain by using surrogate data for subject 1. Significance test analysis for GC results is similar and omitted here. When we calculate NC from channel 2 to channel 1, we only consider surrogate data for channel 2 by randomly shuffling channel 2. In the frequency domain, we calculate NC values from one surrogate data to one raw data, the surrogate data results have very small values and do not have any peak in \( \mu \) rhythm discussed later in this section. Similar analysis can be done for subjects 2 and 3 and omitted here for space reason.

In time domain, during left hand MI we randomly shuffle C4 by 100 times, each time we calculate NC from surrogate data of C4 to C3 and thus obtain 100 NC values for which histogram is shown in Fig.18(a) where the red circle is NC
value from raw C4 to raw C3. From Fig. 18(a) one can see that NC value from raw C4 to raw C3 is larger than all NC values from surrogate data to raw C3, and thus is statistically significant, that is, C4 has significant causal influence on C3. Similarly we plot histogram for NC values from Cz to C3 in Fig.18(b) and histogram for NC values from Cz to C4 in Fig.18(c) where surrogate data are obtained by shuffling Cz. From Fig.18(b) and Fig.18(c) one can see that Cz has significant causal influence on C3/C4.

During right hand MI, similar to Fig. 18, we plot histograms in Fig. 19 where surrogate data are obtained by shuffling C3 in (a), and shuffling Cz in (b) and (c). From Fig. 19 one can see that NC value from raw C3 to raw C4 or from raw Cz to raw C3/C4 is statistically significant. It is not undesired form of bias.

In the frequency domain, during left MI we shuffle C4 and get one surrogate data and calculate NC from C4 (surrogate data) to C3 shown in Fig.20(a) where the red curve represents NC from raw C4 to raw C3 and the blue curve represents NC from surrogate data of C4 to raw C3. From Fig.20(a) one can see that the surrogate data results have very small values and do not have any peak in mu rhythm. However, NC from raw C4 to raw C3 have a clear peak in mu rhythm.

This demonstrates that NC from raw C4 to raw C3 reveal the underlying true causality and it is not undesired form of bias. Similarly, we shuffle Cz, calculate NC from surrogate data of Cz to raw C3/C4, and plot Fig.20(b) and (c) from which we can draw a similar conclusion. Similar to left MI, for right MI we plot Fig.21 from which we can draw the same conclusion.

V. CONCLUSIONS

Nowadays many researchers apply various causality measures to investigate how different brain regions may causally influence on each other. GC, 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. GC wide applications have also been accompanied by criticism from different perspectives. The criticism of GC has most been centered around the philosophical debate on the relationship between GC and true causality. As its name implies, GC is not necessarily true causality [14]. In 2011, Hu et al. [16] systematically pointed out shortcomings and/or limitations of GC by using several illustrative examples and showed that GC is only a causality definition in the sense of Granger and may not necessarily reflect true causality. Hu et al. also proposed NC which is shown to be more reasonable and understandable than GC to reveal true causality. In this paper, we pointed out a fatal drawback that given a jointly regression model, in GC method one has to estimate the autoregressive model, which is a fatal drawback that given a jointly regression model, in GC only has to estimate the autoregressive model, which is equivalent to take a series of backward recursive operations which are infeasible in many irreversible chemical reaction.
models. Thus, GC method cannot be applied at all. In this case GC value by forcibly estimating the auto regressive model (i.e., taking a series of backward recursive operations) inevitably cannot reveal true causality. This fact actually demonstrates that any causality method by decomposing \( X_2 \) at present time into \( X_1, X_2, \eta_2 \) at the past time and then ignoring \( X_1 \) at the past time inevitably cannot reveal true causality from \( X_2 \) to \( X_1 \). On the other hand, by an illustrative example we gave the exact mathematical formula for GC, which is only related to some coefficients and has nothing to do with the other coefficients, and thus GC cannot reveal true causality at all since true causality underlying the joint regression model with different time-invariant coefficients surely be different.

We then applied GC and NC to MI which is an impor-
tant mental process in cognitive neuroscience and cognitive psychology and has received wide attention for a long time. However, there is few work about causal flow so far during MI based on scalp EEG. We used scalp EEG to study causal flow during MI. We are particularly interested in three regions: Cz, C3 and C4 which were shown to be optimal locations for recognizing MI states [27]. The scalp EEGs are from 9 subjects in BCI competition IV held in 2008 and provided by Graz University of Technology. We calculated GC and NC in both time and frequency domains.

In time domain, our results suggest that i) there is strong directional connectivity from Cz to C3/C4 during left and right hand MIIs based on GC and NC (see Fig.22(a)). ii) During left hand MI, there is strong directional connectivity from C4 to C3 (see Fig.22(b)) based on GC and NC. During right hand MI, there is strong directional connectivity from C3 to C4 (see Fig.22(c)). The results in frequency domain are consistent with that in time domain, especially in \( mu \) rhythm. It is noted that during MI state channel 2 with larger power in the mu band having directional causality on channel 1 with smaller power in the mu band may not be true. For example, during left hand MI we obtained directional causality from C4 to C3 for both subject 1 and subject 2. However, C4 has larger power in the mu band than C3 for subject 1 (see Fig.6(a)), and C4 has smaller power in the mu band than C3 for subject 2 (see Fig.10(a)).

There are several evidences to strongly demonstrate that the NC method is much more sensitive than GC method to reveal real causality flow among different brain regions during MI states: i) For all these subjects being right-handedness, during right hand MI the portion (78\%) of GC values from Cz to C3 being larger than that from C3 to Cz is less than the portion (85\%) of NC values from Cz to C3 being larger than that from C3 to Cz. The portion (78\%) of GC values from Cz to
C4 being larger than that from C4 to Cz is much less than the portion (96%) of NC values from Cz to C4 being larger than that from C4 to Cz. So, the NC method identifies the causal influence from Cz to C3/C4 more clearly than GC method during right hand MI. ii) During right hand MI, the portion (60%) of GC values from C3 to C4 being larger than that from C4 to C3 is much less than the portion (81%) of NC values from C3 to C4 being larger than that from C4 to C3. So, the NC method identifies the causal influence from C3 to C4 during right hand MI more clearly than GC method. iii) For the three subjects analyzed, the peaks in $\mu$ rhythm mostly appear for spectral NC results which are consistent with peaks of the power spectra of Cz, C3 and C4. However, they do not appear in spectral GC results. iv) The portion of spectral causality values between C3 and C4 have peaks in $\mu$ rhythm by the NC method is 54%. However the corresponding portion by GC method is only 32%. Thus, the NC method is much clearer to reveal the causal influence between C3 and C4 than GC method in the frequency domain.

In this paper, we only use bivariate autoregressive model to discuss causal influence among three optimal locations during MI. Since NC may be defined for multiple channels, our future work will focus on applying NC method to reveal effective connectivity structure of whole brain during MI states and/or other cognitive experimental tasks. Moreover, since the pairwise bivariate GC may fail to reveal synergetic effects among variables, the use of multivariate GC would be needed to put in evidence synergy among brain areas.

![Causality Directions](image)

Fig. 22. (a) Cz has causal influence on C3 and C4 during MI. (b) C4 has causal influence on C3 during left hand MI. (c) C3 has causal influence on C4 during right hand MI.

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